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When is the Appropriate Time to Perform a Right-Sided Electrocardiography Examination on Syncope Patients? An Emergency Case Report

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ABSTRACT

Introduction: Syncope is a symptom of transient loss of consciousness with numerous potential causes. Identifying and prioritizing life-threatening conditions is crucial to avoid a catastrophic outcome. Here we report a young smoker patient who presented with syncope as the manifestation of an acute right ventricular myocardial infarction (RVMI) and inferior wall myocardial infarction (IWMI).

Case Description: A 46-year-old man, presented to the emergency department (ED) with syncope and chest pain. On arrival at the ED, he stated not having had episodes of chest pain before. The physical and neurologic examinations were normal. Initially, a 12-lead ECG showed no abnormalities. Right-sided ECG was conducted and there were ST elevations in lead V3R until V5R indicated RVMI. Repeated 12-lead ECG showed ST elevations in lead II, III, and avF. Treatment includes aspirin, heparin, clopidogrel, and thrombolysis. The coronary angiogram after thrombolysis demonstrated stenosis of the proximal third of the right coronary artery. Syncope was assumed to be hypoperfusion of the sinus node artery arising from the right coronary artery, causing transient sinus node dysfunction.

Discussion: Syncope could be the sole manifestation of RVMI which can develop with IWMI and be complicated by sinus node dysfunction. The younger a person starts smoking, the higher the risk for cardiovascular damage. Syncope followed by one of the cardiovascular risk factors can be considered using Right-sided ECG. A 12-lead ECG monitoring is required in all patients with syncope conditions, even without typical symptoms of acute coronary syndrome or hemodynamic instability.

Conclusion: Syncope with suspected cardiovascular involvement, Right-sided ECG can be performed. Additionally, serial ECG monitoring and prolonged ED observation are necessary for syncopal patients with suspected cardiovascular causes to ensure heart safety, especially in healthcare facilities that do not have cardiac enzyme tests.

INTISARI

Pendahuluan: Sinkop merupakan gejala kehilangan kesadaran sementara dengan berbagai potensial penyebab. Identifikasi kondisi mengancam jiwa pada kondisi ini merupakan hal penting. Studi ini bertujuan untuk melaporkan kasus pasien perokok muda dengan sinkop sebagai manifestasi dari infark miokard ventrikel kanan dan infark miokard dinding inferior.

Laporan Kasus: Laki-laki, usia 46 tahun, dating ke unit gawat darat (UGD) dengan sinkop dan nyeri dada. Saat kedatangan di UGD, pasien menyatakan bahwa dirinya tidak pernah memiliki riwayat nyeri dada. Hasil pemeriksaan fisik dan neurologis dalam batas normal. Pasien dilakukan pemeriksaan

elektrokardiografi (EKG) standar (12-lead) dan hasilnya normal. Setelah itu, EKG kanan dilakukan dan hasilnya ST elevasi di lead V3R-V5R (infark miokard ventrikel kanan). EKG standar (12-lead) diulang kembali dan hasilnya ST elevasi di lead II, III, dan aVF (infark miokard inferior). Terapi yang diberikan yaitu aspirin, heparin, clopidogrel, dan trombolisis. Pemeriksaan angiogram coroner setelah trombolisis didapatkan stenosis di sepertiga proksimal arteri koroner kanan. Sinkop pada kasus ini diasumsikan dari hipoperfusi sinus node artery yang berasal dari arteri koroner kanan, sehingga menyebabkan disfungsi sementara pada nodus sinus.

Pembahasan: Sinkop dapat menjadi manifestasi utama dari infark miokard ventrikel kanan yang dapat berkembang dengan infark miokard inferior, dan disebabkan dari disfungsi nodus sinus. Semakin muda pasien merokok, maka semakin besar risiko kerusakan kardiovaskular. Sinkop yang diikuti salah satu faktor risiko kardiovaskular dapat dipertimbangkan pemeriksaan EKG kanan. Monitoring EKG standar diperlukan pada seluruh pasien dengan kondisi pasien sinkop tanpa gejala tipikal dari sindrom koroner akut atau instabilitas hemodinamik.

Kesimpulan: Sinkop yang dicurigai keterkaitan kardiak, pemeriksaan EKG kanan dapat dilakukan. Selain itu,, monitoring EKD dan observasi perlu diperlakukan pada pasien sinkop dengan kecurigaan penyebab faktor kardiak untuk memastikan keamanan jantung, terutama pada fasilitas kesehatan tidak memiliki tes enzim jantung.

INTRODUCTION

Syncope is a symptom defined as transient loss of consciousness and postural tone, self-limited, and usually leading to fall. It affects 30 – 40% of the population and is probably conservative because many individuals experiencing syncope do not seek medical attention in a hospital or urgent care facility. While most syncopal events are innocuous, the causes of syncope are numerous and should be identified as a priority to avoid a catastrophic outcome. Here we report a young smoker patient who presented with syncope as the manifestation of an acute right ventricular myocardial infarction (RVMI) and inferior wall myocardial infarction (IWMI).

CASE PRESENTATION

A 46-year-old man, young smoker, presented to the emergency department (ED) with syncope and chest pain. On arrival at the emergency department (ED), he regained consciousness briskly within 2 minutes and he stated not having had any episodes of chest pain before. He denied having any systemic illness or recreational drug use. He smoked cigarette 1 pack per day since the age of 18 years. He had no history of epilepsy and head trauma. His family had no history of any diseases. His vital signs include blood pressure of 96/70 mmHg, pulse rate of 70 beats per minute, and respiratory rate of 16 per minute. The conjunctiva was not pale, there was no increase in jugular vein pressure, and the heart also lungs were clear to auscultation. The remaining physical and neurologic examinations were without abnormalities. The patient was conducted with 12-lead electrocardiography (ECG), and there were no abnormalities. After that, we decided to continue with Right-sided ECG and there were ST elevations in lead V3R until V5R indicated right ventricular myocardial infarction (RVMI). We conducted again with a 12-lead ECG because at first, we suspected a potential cause of syncopal event in this patient was cardiac involvement. Surprisingly, there were ST elevations in lead II, III, and aVF indicating inferior myocardial infarction.

Chest X-ray showed no abnormalities. Based on the cardiologist's advice, we decided to check routine hematology and CKMB levels. The results were hemoglobin 13,2 gr/dL, leukocyte 9,92x10^3/µL, thrombocyte 210x10^3/µL, hematocrit 40,5%, SGOT 26 U/L, SGPT 18 U/L, BUN 12,5 mg/dl, creatinine 1,15 mg/dL, blood sugar 136 mg/dL, sodium 133 mmol/L, potassium 3,60 mmol/L, chloride 100 mmol/L, dan CKMB 33,5 U/L. There was an increase in CKMB levels. The patient was moved into the intensive cardiac care unit (ICCU) with 12-lead ECG monitoring and the treatment included aspirin, heparin, clopidogrel, and thrombolysis. After the thrombolysis was given, the coronary angiogram was conducted. The coronary angiogram in this patient showed stenosis of the proximal third of the right coronary artery. The cause of syncope in this case was assumed to be hypoperfusion of the sinus node artery arising from the right coronary artery, causing transient sinus node dysfunction. On follow-up at 7 days, he was getting better through the treatments.

DISCUSSION

Here we present a young smoker male patient who developed episodes of syncope as the sole manifestation of right ventricular myocardial infarction (RVMI) which can develop with inferior wall myocardial infarction (IWMI) and be complicated by sinus node dysfunction. The causes of syncope are either cardiac (4-36%) or non-cardiac (17-52%) or remain unexplained (13-48%). Medical history is the fundamental aspect of the evaluation of a patient with transient loss of consciousness[1]. Clinical indicators that might indicate cardiogenic syncope include the existence of significant structural heart conditions, attacks during exertion or supine, preceded by palpitation or

accompanied by chest pain, and a family history of sudden cardiac death. The assessment of a syncope patient consists of medical history, physical examination, and standard 12-lead ECG. Specific ECG findings in syncope patients not only help to determine the cause of the loss of consciousness but also assist in making decisions regarding early treatment and the appropriate care plan. In some cases of syncope patients, and there is suspected cardiac involvement, right-sided ECG should be conducted. Because, in syncope, patients can develop right ventricular myocardial infarction (RVMI) (30-50%). An ischemic right ventricular can be dilated and stiff with deceased RV output, which can be impaired by LV preload, and the result is decreased cardiac output[1,2].

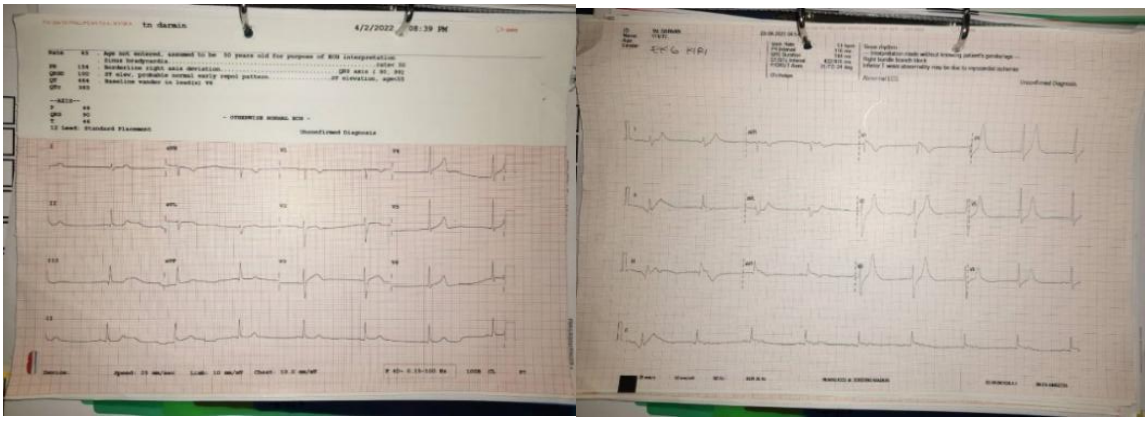

Figure 1. 12-lead ECG first arrival (right side) and repeated 12-lead ECG (left side)

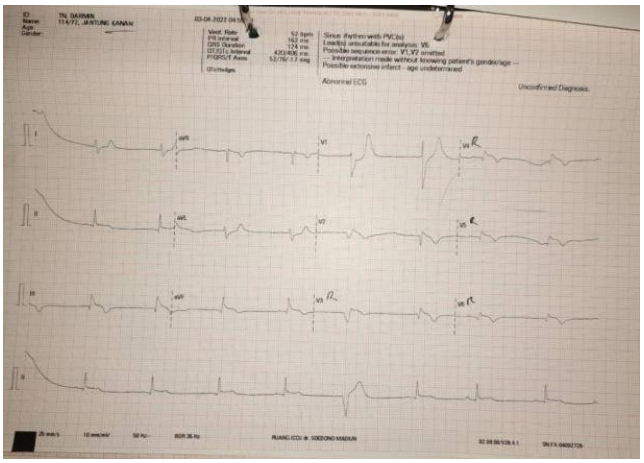

Figure 2. Right-sided electrocardiography (ECG) indicated RVMI

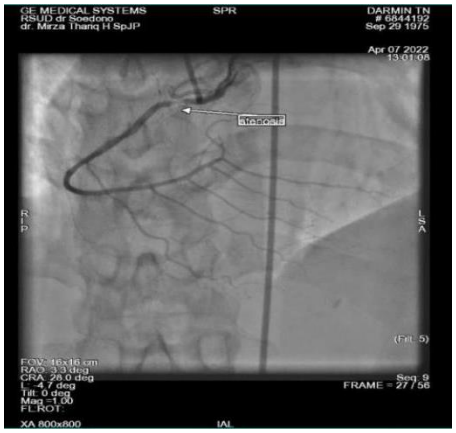

Figure 3. A Coronary Angiogram showed stenosis of the proximal third of the right coronary artery

Syncope could be the initial complaint in 5-12% of patients with acute myocardial infarction (MI). There are a few risk factors that can develop a person to acute MI, and one of them is the smoking habit. Exposure to toxic compounds contained in cigarette smoke causes free radical–mediated oxidative stress and decreased nitric oxide bioavailability resulting in decreased endothelium-dependent vasodilatation. Furthermore, smoking has been associated with increased adrenergic tone and increased coronary vasospasm. If the exposure happens for a long period, it can cause worse impairment. The younger the person to start smoking, the higher the risk for cardiovascular damage[3,4].

A coronary angiogram can be conducted in patients with coronary artery disease. In this patient, the result was stenosis of the proximal third of the right coronary artery (RCA). As we know, a proximal third of RCA is the blood source to the sinoatrial node sinus node artery which is

circulated by the sinus node artery. Stenosis of the proximal third of the right coronary artery can lead to hypoperfusion of the sinus node artery. It means, the sinoatrial cannot work properly, inability to produce an adequate heart rate like arrhythmias conditions (bradycardia or tachycardia). The final result of these conditions can lead to underperfusion in CNS which manifests in syncope[5,6].

CONCLUSION

In syncope with suspected cardiovascular involvement, a Right-sided ECG can be performed. Additionally, serial ECG monitoring and prolonged ED observation are necessary for syncopal patients with suspected cardiovascular causes to ensure heart safety, especially in healthcare facilities that do not have cardiac enzyme tests.

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Peripartum cardiomyopathy and massive tranfusion due to post partum haemorrhage: was it associated each other?

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Keywords: PPCM; massive transfussion; heart failure; cardiomyopathy

ABSTRACT

Introduction: Peripartum cardiomyopathy (PPCM) is a potentially life-threatening pregnancy-associated disease marked by left ventricular (LV) dysfunction and heart failure (HF). Clinical findings of HF are often masked by the normal physiological changes seen in pregnancy making the diagnosis challenging. Furthermore, postpartum hemorrhage followed by massive blood transfusion may masked the diagnosis of PPCM or worsen the decompensated HF.

Case Presentation: Here, we report a case of 35-year-old postpartum gemelli woman with history of massive postpartum hemorrhage due to atonia uteri and Disseminated Intravascular Coagulation (DIC) who complain of shortness of breath after received massive blood transfusion Sign of PPCM was identified by chest x ray showed right pleural effusion and early lung edema. The echocardiography showed decrease LV systolic function with ejection fraction 41% pseudonormal diastolic function and global hypokinetic. She was diagnosed with PPCM, acute lung edema, pleural effusion, and pneumonia. Patient was treated with Furosemid continuous pump, Spironolactone, Bisoprolol, Valsartan. Her dyspnea greatly decreased with diuresis and antibiotic. She was discharged with HF medication continued.

Conclusion: The presence of massive transfusion in patient with PPCM can be challenging in diagnostic of PPCM itself and also unpredictable course of decompensated HF in peripartum mothers. Due to its high mortality rate without proper treatment, prompt investigation is important in improving maternal survival.

INTISARI

Pendahuluan: Peripartum Cardiomyopathy (PPCM) adalah penyakit jantung pada kehamilan yang berpotensi mengancam nyawa ditandai dengan disfungsi ventrikel kiri (LV) dan gagal jantung (HF). Temuan klinis gagal jantung sering kali tertutupi oleh perubahan fisiologis normal yang terjadi pada kehamilan sehingga membuat diagnosis menjadi sulit. Selain itu, Haemorrhagic Post Partum (HPP) yang diikuti dengan masif transfusi dapat mempersulit diagnosis PPCM dan memperburuk kondisi gagal jantung.

Presentasi Kasus: berikut kami laporkan kasus seorang wanita hamil gemelli berusia 35 tahun pascapersalinan dengan riwayat HPP akibat atonia uteri dan Disseminated Intravascular Coagulation (DIC) yang mengeluhkan sesak napas setelah menerima massif transfusi. Diagnostic PPCM diidentifikasi melalui foto rontgen dada yang menunjukkan adanya efusi pleura kanan dan edema paru awal. Ekokardiografi menunjukkan penurunan fungsi sistolik ventrikel kiri dengan fraksi ejeksi 41%, fungsi diastolik pseudonormal, dan hipokinetik global. Pasien didiagnosis dengan PPCM, edema paru akut, efusi pleura, dan pneumonia. Pasien mendapatkan terapi infus continued pump Furosemid, Spironolakton, Bisoprolol, Valsartan. Keluhan sesak berkurang

dengan diuresis dan antibiotik. Pasien dipulangkan dan mendapatkan pengobatan gagal jantung dari Poliklinik.

Kesimpulan: Pemberian transfusi masif pada pasien dengan PPCM dapat mempersulit diagnostik PPCM sendiri dan juga klinis gagal jantung yang tidak dapat diprediksi pada pasien dengan PPCM. Karena tingginya angka kematian tanpa penanganan yang tepat, investigasi yang cepat sangat penting untuk meningkatkan

deposisi zat besi pada miokard dengan disfungsi ventrikel kiri pada thalassemia mayor termasuk dalam kategori lemah. Patofisiologi gagal jantung pada talasemia yang berfokus pada kelebihan zat besi miokard mungkin perlu dipertimbangkan kembali

INTRODUCTION

Peripartum cardiomyopathy (PPCM) is a potentially life-threatening pregnancy-associated disease marked by LV dysfunction and heart failure (HF). Clinical findings of HF are often masked by the normal physiological changes seen in pregnancy making the diagnosis challenging. Furthermore, postpartum hemorrhage followed by massive blood transfusion may be masked by the diagnosis of PPCM or worsen the decompensated HF. Risk factors of PPCM include multiparity, black race, older maternal age, pre-eclampsia, and gestational hypertensio.[1] A case control study in the United States found that, when compared to non – African Americans, African American women had a 15.7-fold higher relative risk of PPCM.[2] The incidence of PPCM in black women was 4 times higher than white women (1:1087 versus 1:4266), and the fatality rate at 5-year follow-up was also 4 times as high (24% versus 6%).[3] In South Korea, incidence of PPCM was 1 in 1741 deliveries. After excluding 20.102 patients of preceding probable HF, there were 1.384.449 deliveries in South Korea from 2010 to 2012. Among these, 795 cases had codes that defined as PPCM.[4]

Purpose of this case report is to deliver a rare case of PPCM that had relations with massive transfusion due to post partum haemorrhage. In addition, it will be presented pathogenesis, diagnosis, therapy of disease and more things that may cause the disease. Researchers expect that PPCM get more spotlight considering the clinical sign of symptoms of the disease are like the normal physiological changes of the pregnancies. It is hoped that there will be no delay in recognizing the disease and causing an increase in morbidity and mortality.

CASE PRESENTATION

A 35-year-old multigravida (G4P3A0) Indonesian female referred to the Emergency Room (ER) about giving birth her gamely pregnancy. The first baby was born spontaneous in her house one hour before. She had massive and continues bleeding during delayed the second child labour. During examination in the ER the patient was noted to be lethargy and had blood pressure (BP)100/60 mmHg, pulse rate 100 beats per minute, respiratory rate 24 breaths per minute, and oxygen saturation 95%. Hemoglobin 10.5 g/dL. The gynecology examination was found portio rupture and active bleeding with attached 3 tampons. The second child was born in hospital. She loses 750 ml of blood during labour in hospital and still has active bleeding because of atonia uteri and Disseminated Intravascular Coagulation (DIC). The blood pressure became 90/50 mmHg, a pulse rate 144 beats per minute and the patient became agitated. The therapy of obstetrician was 3000 ml of ringer lactate, whole blood transfusion 4 kolf of blood and 1000 cc of HES solution After initial transfusion, there was no sign of short of breathness but the patient still agitated and disoriented. The patient scheduled hysterectomy right away.

During hysterectomy the bleeding still active and massive, the patient had transfusion 750 ml of whole blood and 1000 ml kristaloid. After hysterectomy the bleeding from vagina and drain is still active (900 ml) Hemoglobin from complete blood count shows 8 g/dL. The obstetrician decided to reopen 3 hours after. In the Intensive care unit (ICU) after surgery patient got 5 kolf plasma transfusion and 5 kolf of thrombocyte concentrate. From ER until 6 days observation in the ICU patient got 16 kolf Whole blood, 4 kolf of Packed Red Cells, and 5 kolf of Trombocyte Concentrate transfusion. Patient was noted had DIC with Protombin time (PT) 16.3, International Normalized Ratio (INR) 1.22, Activated Partial Thromboplastin Time (aPTT) 35.5 Fibrinogen 319, D Dimer 7,777 ng/ml FEU. Patient was got observation in ward 9 days after critical condition. And discharged with no complaint, blood pressure 120/80 mmHg, a pulse rate 80 beats per minute and respiratory rate 18 breaths per minute.

10 days after discharged from hospital the patient came to the ER with complaint of shortness of breath, fever, and experienced chest pain during cough. In this patient, the increase short of breathness was associated with physical activity, sleep with more than one pillow. The complaint about 5 days and worse in these 2 days.

During examination in the ER, the patient was noted to be febrile and had a normal BP (120/80mmHg), tachycardia (112x/minute), dyspneua (27x/minute), and an oxygen saturation of 95% with 3 lpm nasal cannula. Her lungs were noted symmetrically on inspection, dim on percussion in bassal inferior and found ronchi to auscultation. Her heart rate was regular. Compete blood count shows Hb 9.1 g/dL, White blood cells 9.530, Trombocyte 439.000 Blood gas Analysis shows alkalosis respiratorik with compensation pH 7.477 PCO2 28.6 HCO3 20.7 BE -2.2. An

electrocardiogram showed a sinus tachicardia with heart rate 113 beats per minute normal frontal axis and clockwise rotation horizontal axis. Chest radiographs showed Pleural effusion on the right side and early lung oedema. The heart size was not able to be evaluated because of effusion.

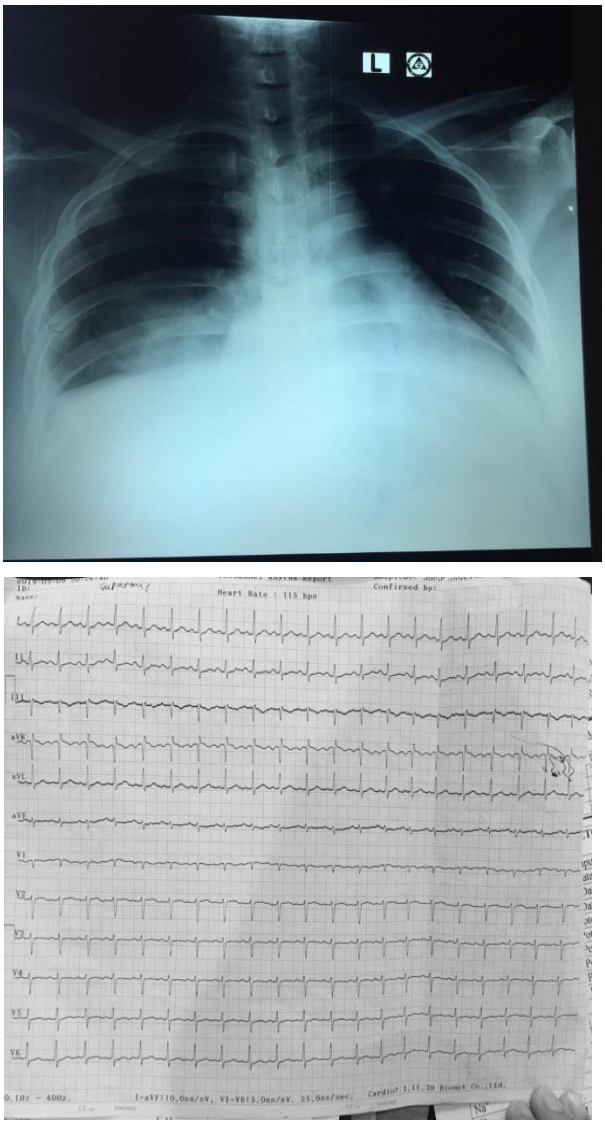

Figure 1

The patient was transferred to the Intensive Care Unit (ICU), in this case, furosemide iv 80 mg given initially, antibiotics Ceftriaxone iv 1 gram over 12 hours, Bromhexine iv 4 mg over 12 hours, Nebulizer Combivent 1 unit dose (2.5 ml) and Flixotide 2 mg over 8 hours. The patient was scheduled echocardiogram the day after.

The echocardiogram showed a left ventricle ejection fraction 41% with increasing systolic function. Diastolic function pseudonormal. Right ventricle systolic function was increased. Global hypokinetic and PH low probability. Patient had electrical imbalance hypokalaemia, which Kalium found 2.8 and hypo albumin 3.3.

Patient was diagnosed with Postpartum Cardiomyopathy, Pleural Effusion, Acute Lung Oedema and Pneumonia. Patient was treated in ICU 3 days and got additional treatment Furosemid 40 mg twice daily, Spironolacton 25 mg once daily, Bisoprolol 2.5 mg once daily, Valsartan 80 mg once daily.

The patient signs and symptomps became better over 2 days observation and treatment in the ICU. The patient was reffered to the ward. Her dyspnea greatly decreased with diuresis and antibiotic. She was discharged from the hospital 2 days later and instructed to take Spironolactone 25 mg, Bisoprolol 2.5 mg; Furosemide 40 mg and regularly control to the clinic.

DISCUSSION

Peripartum cardiomyopathy is potentially life-threatening disease that arises in the peripartum period and is idiopathic.[3] Factors such as black race, multiparity, maternal age >30 years, twin pregnancies, history of hypertension, preeclampsia, and eclampsia have been associated with higher incidence, although no causal association has been shown.[3-5] In this case, patient is multiparity (G4P3A0), age is 35-year-old, and twin pregnancies. There is no history of hypertension and preeclampsia.

The Diagnostic of PPCM are as follows: (1) development of HF in the last month of pregnancy or within 5 months after delivery; (2) LV systolic dysfunction (LV EF < 45% by echocardiography); (3) no identifiable cause for HF; and (4) no recognized heart disease before the last month of pregnancy.

Postpartum hemorrhage (PPH) remains the leading cause of maternal morbidity and mortality worldwide. Few population-based studies have examined the epidemiology of massive transfusion for PPH. One-quarter of all women receiving massive transfusion underwent a hysterectomy to control bleeding.[8] Placenta abruption, uterine arterial embolization, and peripartum hysterectomy are associated with blood loss in delivery. In these situations, massive transfusion or fluid resuscitation are usually required, and these conditions may induce HF at the time of delivery. Therefore, it is not surprising that we observed an increase in conditions associated with peripartum hemorrhage in patients with PPCM.[4]

Bosch et al show case about the patient a 27-year-old gravida 1, para 0 were admitted to hospital at a gestational age of 39+1 weeks with preeclampsia. Her blood pressure was 140/95 mmHg; postpartum, she suffered a blood loss of 1200 ml due to an atonic uterus. The total blood loss was 3000 ml for which she received transfusions of blood products. Postoperatively acute respiratory failure developed. She had a diagnosis of postpartum lung edema associated with preeclampsia and possibly a sulprostone effect. She recovered quickly and returned to the maternity ward after two days. Five days after delivery, the patient experienced dyspnoea. She was admitted to the intensive care unit with severe heart failure. The chest X-ray showed signs of pulmonary edema. Echocardiography demonstrated a dilated left ventricle with normal valves.

Based on history, physical examination, and further examination, the diagnosis of peripartum cardiomyopathy was made. Treatment included sodium and fluid restriction, ACE inhibitor and low-dose β-receptor antagonists. She was discharged after eight days. Left ventricular ejection fraction was 40 to 45%. Seven weeks postpartum the echocardiography showed a normal right and left ventricular function with no valvular abnormalities. The patient would like to become pregnant again.[11]

Massive transfusion is traditionally defined as transfusion of 10 units of packed red blood cells (PRBCs) within a 24 hours period. Massive transfusion has been used in many clinical settings, including obstetrics, gastroenterology, trauma, and the operating room. Complications include hypothermia, acid/base derangements, electrolyte abnormalities (hypocalcemia, hypomagnesemia, hypokalemia, hyperkalemia), citrate toxicity, and transfusion-associated acute lung injury.[12,13]

Effective management of PPCM reduces mortality and increases the number of women who fully recover their left ventricular function. Outcomes for subsequent pregnancies are better in women who have fully recovered cardiac function after PPCM. However, recurrence of cardiac failure is common in subsequent pregnancies — Bromocriptine, which is effective in PPCM, to treat patient.[9]

CONCLUSION

The presence of massive transfusion in patient with PPCM can be challenging in diagnostic of PPCM itself and also unpredictable course of decompensated HF in peripartum mothers. Due to its high mortality rate without proper treatment, prompt investigation is important in improving maternal survival.

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Early Surgery in Complicated Infective Endocarditis: To Accelerate Is to Be Appropriate

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Keywords: Infective Endocarditis; Early Surgery

ABSTRACT

Background: Infective endocarditis (IE) is associated with a high risk of mortality and morbidity.1,2 Surgery has been the treatment of choice for IE because of severe complications. The optimal timing of the surgery has been unclear.3,4 In particular conditions early surgery brings certain benefits, so to avoid further deterioration yet performing surgery during the active phase of IE carries significant risk.4 Identification, risk stratification and early consultation with cardiac surgeon is capital to determine the best therapeutic approach.3

Case Illustration: A 20-years-old female was admitted to emergency room with complaints of breathlessness, tachypnea, palpitation, heaviness in chest at night, and dyspnoea on exertion, had undergone an appendectomy in other hospital two weeks before with one month history of prolonged fever with no record of the highest temperature. Physical examination revealed a regular heart rate with systolic murmur sound heard on the apex. An electrocardiography (ECG) was sinus rhythm, heart rate 115x/minutes, bigeminy premature ventricular contraction (PVC). A blood examination revealed anaemia, leucocytosis, thrombocytosis, electrolyte imbalance. A transthoracic echocardiography (TTE) dilatation of left atrial and ventricle, an eccentric hypertrophic left ventricle, severe mitral regurgitation with possible vegetation on anterior and posterior mitral leaflet. Patient was diagnosed with Infective Endocarditis (IE), severe Mitral Regurgitation accompanied with acute heart failure, treated to IE protocol for 2 weeks with good response. The patient was decided to get early surgery to mitral valve repair. The result was remarkable, with subsided symptoms of heart failure and mild residual MR.

Conclusion: Infective Endocarditis along with complications need apropriate treatment plan, The decision to perform early surgery to this patient resulting favorable end, opening possibility to do it in regards of capitalizing improvement of patient's condition, shortening length of stay and cutting costs over scalable risk that present in this patient.

INTISARI

Pendahuluan: Endokarditis infektif (IE) dikaitkan dengan risiko mortalitas dan morbiditas yang tinggi.1,2 Pembedahan telah menjadi pengobatan pilihan untuk IE karena komplikasinya yang parah. Waktu optimal untuk pembedahan masih belum jelas.3,4 Dalam kondisi tertentu, pembedahan dini memberikan manfaat tertentu, sehingga untuk menghindari kerusakan lebih lanjut namun melakukan pembedahan selama fase aktif IE mempunyai risiko yang signifikan.4 Identifikasi, stratifikasi risiko, dan konsultasi dini dengan ahli jantung ahli bedah adalah modal untuk menentukan pendekatan terapi terbaik.

Laporan Kasus: Seorang wanita berusia 20 tahun dirawat di IGD dengan keluhan sesak napas, takipnea, jantung berdebar, dada terasa berat pada malam hari, dan sesak saat beraktivitas, pernah menjalani operasi usus

buntu di rumah sakit lain dua minggu sebelumnya dengan riwayat demam berkepanjangan selama satu bulan dengan tidak ada catatan suhu tertinggi. Pemeriksaan fisik menunjukkan denyut jantung teratur dengan bunyi murmur sistolik terdengar di apeks. Pemeriksaan elektrokardiografi (EKG) irama sinus, denyut jantung 115x/menit, kontraksi ventrikel prematur bigeminy (PVC). Pemeriksaan darah menunjukkan anemia, leukositosis, trombositosis, ketidakseimbangan elektrolit. Dilatasi ekokardiografi transthoracic (TTE) pada atrium dan ventrikel kiri, ventrikel kiri hipertrofik eksentrik, regurgitasi mitral parah dengan kemungkinan adanya vegetasi pada selebaran mitral anterior dan posterior. Pasien didiagnosis Endokarditis Infektif (IE), Regurgitasi Mitral berat disertai gagal jantung akut, dirawat sesuai protokol IE selama 2 minggu dengan respon baik. Pasien diputuskan untuk menjalani operasi dini untuk perbaikan katup mitral. Hasilnya luar biasa, gejala gagal jantung mereda dan sisa MR ringan.

Kesimpulan: Endokarditis Infektif beserta komplikasinya memerlukan rencana pengobatan yang tepat, Keputusan untuk melakukan pembedahan dini pada pasien ini menghasilkan hasil yang menguntungkan, membuka kemungkinan untuk melakukannya dalam hal memanfaatkan perbaikan kondisi pasien, memperpendek lama rawat inap dan memotong biaya atas risiko terukur yang ada pada pasien ini.

INTRODUCTION

Infective endocarditis (IE) is the most severe and potentially devastating complication of heart valve disease, be it native valve endocarditis (NVE), prosthetic valve endocarditis (PVE), or infection on another cardiac device that carries a considerable risk of death and morbidity despite interventions.[1,2] The incidence of IE is 3-7 cases per 100.000 persons-years and mortality rate is up to 50 %. Early surgical intervention is the best strategy to increase survival, however the optimal timing of surgery remains unclear. In the past, IE mainly affected young adults with known valve disease (mostly affected by rheumatic fever) nowadays the typical IE patient is elderly (peak incidence between 70-80 years) with valve disease of a degenerative etiology, prosthetic valve(s) or intracardiac devices including pacemaker or cardioverter defibrillator. Despite advances in diagnosis and therapeutic approach, neither the incidence nor the mortality of IE has reduced in the last 30 years and mortality remains high.

The role of Surgical treatment is required in approximately half of the patients with IE because of severe complications[3,4] Under American Heart Association, European Society of Cardiology guideline and American Association for Thoracic Surgery early surgery can be done whilst patient is in initial hospitalization without completing antimicrobial therapy with certain indications. Generally, is indicated when antibiotic treatment alone is unlikely to be curative of may be associated with persistent risk of complications.

The management of IE required the close collaboration of multidisciplinary endocarditis teams that must decide on the diagnostic approach; the appropriate initial treatment in critical phase, the detection of patients needing surgery and the timing of this intervention; and finally, the accurate selection of patients for out of hospital treatment, either at home hospitalization or with oral antibiotic treatment. Regarding surgical timing, we found no increase in mortality in patients who underwent early vs late surgery. Rapid identification of patients with IE at higher risk of mortality may offer the opportunity to change the course of disease and improve prognosis. In general, surgery is recommended in cases in which antibiotic treatment alone may not be curative or may be associated with worse outcome.

Early surgery has been the treatment of choice for IE, but its effectiveness has not been systematically and comprehensively assessed. The choice of operation time and their strength and most were supported by expert opinions or clinical experience. In general, surgical indications in patients with IE relate to heart failure or shock, evidence of risk of persistent infection and embolic risk reduction. The presence of a clear diagnosis of IE and when an indication for surgery has been established, there are no proven benefits in delaying surgery. The decisions on the timing of surgical intervention in IE are complex and depend on many clinical factors. It is generally agreed that those decisions on both the indication and timing of surgical intervention should be determined by a multispecialty team with expertise in cardiology, imaging, cardiothoracic surgery and infectious disease. The timing of surgery is based on balancing the urgency of indications for surgery versus the risks of complication occurring afterward. To delay surgery to get more antibiotics on board is likely not going to lead to any additional patient benefit. Among those factors that mitigate decisions toward early surgery are the infecting organism, the size of vegetation, the presence of perivalvular infection, embolism or heart failure, patient age, noncardiac comorbidities and available surgical expertise. Early consultation with a cardiac surgeon within 'heart team' is recommended in order to determine the best therapeutic approach.[3] Although early surgery can avoid death and severe complications, performing surgery during the active phase of IE carries significant risk.[4]

CASE PRESENTATION

A 20 – year old female was admitted to emergency room complaints of breathlessness, tachypnea, palpitation, heaviness in chest at night and dyspnoea on exertion, takikardia, DOE (+), breathlessness worsens at night. Unable to carry out any physical activity without discomfort. Symptoms of cardiac insufficiency at rest If physical activity undertaken, discomfort is increased. Presented the condition of heart failure functional class IV. Before admitted to emergency room, one month prior to hospitalization, the patient had history of intermittent fever with temperature above 38 C, was slightly decreased by the administer of antipyretic agents. The symptoms developed ever since the patient had undergone an appendectomy in other hospital two weeks before. Dyspnea was felt to worsen since the last week after appendectomy in Aulia hospital Jagakarsa.

She was suffered from loss of appetite and nausea within last week. The patient looked underweight (body weight 41 kg height 155 cm) with pale conjunctiva. Nutrition status of the patient was malnourished. On admission patient was compos mentis and had febrile with axillae temperature was 38 C, From Hemodynamic assessment patient was hypotension, (84/69) mmHg with a tacycardia, the temperature was normal, with a normal saturation. From the physical examination, no elevated jugular venous pressure, finished percussion sound of cardiac waist indicates widening of left heart border. From chest auscultation a systolic murmur grade 3/6 was heard on the apex with regular heart rhytm and rate, while the lungs sound was clear bilaterally. The abdomen was normal was soft and non-distended. She had a normal dentition with no tooth decay, no skin rash or petechiae. Based on an examination at referral hospital, patient was diagnosed with infective endocarditis and severe mitral regurgitation. An electrocardiography exam showed sinus rhythm with heart rate of 115x/minutes with bigeminy premature ventricular contraction (PVC). A complete blood count revealed an anaemia with haemoglobin and haematocrit Hb 10.6 g/dL and haematocrit 31.8% respectively, leucocytosis with white blood cell counts 20,140/µL and thrombocytosis with platelets 554,900/µL. The chemical blood test shown imbalance electrolytes; hypokalaemia potassium of 2.23 mEq/L. There were three blood cultures taken from different vein location with time difference of 30 minutes apart. The result of blood cultures then revealed to negative of bacterial growth. An obtained transthoracic echocardiography (TTE) demonstrated eccentric left ventricle hypertrophy, dilatation of both left ventricle and atrium, severe mitral regurgitation, with vegetation on anterior and posterior mitral leaflet (AML & PML). E >1.2 CM/second, MR Ero, MR VC, PV systolic reversal hard to assess. Ejection fraction 82%, MR severe. Based on the modified duke's criteria, clinical sign and symptoms of patient fulfil one major and three minor for clinical diagnosis. Patient was then diagnosed with Infective Endocarditis (IE), severe Mitral Regurgitation (MR) accompanied by acute heart failure. Chest X-Ray normal, Pro cardiac conference, to discuss uregent MV repair kiv replacement, before urgent surgery consider antibiotic for 4 weeks.

Patient was treated with 2 grams intravenous antibiotics Ceftriaxone once in a day for four weeks and 80 grams of Gentamicin twice a day for two week, 20 miligrams of furosemide three times a day for optimal heart failure therapy, along with ramipril 1.25 milligrams once daily, spironolactone 25 milligrams once daily, digoxin 0.25 milligrams once daily, 1.25 milligrams concor once daily, electrolyte correction with kcl 25 mEq in 500 cc NaCl in 24 hours, 1200 mg KSR three times a day, 500 mg NaCl capsule three times a day. There was period of fever in hospitalization, the fever subsided within two weeks after antibiotics therapy, during the following days fluid balance improved, the patient responded well to therapies as symptoms of heart failure improved, reduced white blood cells count, no embolic event, and the size of vegetation did not increase. After the infection status was diminished, heart failure status was under control, the patient was planned to get early surgery of mitral valve repair. Before the surgery proceeded, a transoesophageal echocardiography was performed which presented MR destructed P2 region with vegetation, chordae rupture, and P3 region prolapse while the edges of AML were destructed. In the surgery it was the confirmed that there were vegetation and chordae rupture in both A3 and P3. The vegetation areas were resected and cultured while the remaining valve was repaired with pericardial patch and annuloplasty ring. The administration of Ceftriaxone was then continued peri-operatively until 4 weeks. The result of mitral tissues culture was *Staphylococcus epidermidis* that resistant to Penicillin G and Ampicillin.

After undergoing a whole series of therapies and surgery, there was a significant improvement of heart failure status, before enter hospital heart failure status of patient was in functional class IV and infection was diminished, no longer complaints of breathlessness, tachypnea, palpitation, heaviness in chest at night and dyspnoea on exertion, takikardia, DOE (+), breathlessness worsen at night. A week post-surgery another TTE was obtained with result of reduced left atrial and ventricle size into normal, no remarkable left ventricle hypertrophy, with remaining MS with gradient of 5 mmHg and residual mild MR. No palpitation and other symptoms of heart failure was reported.

DISCUSSION

Endocarditis should be suspected in patients with unexplained fevers, diagnosis is made using the duke criteria, which include clinical, laboratory and echocardiographic findings. We present a case of a 20-year-old female known to have severe mitral regurgitation (MR) with a history of fever. According to the modified duke criteria, clinical sign and symptoms fulfilled one major criterion (echocardiography finding of vegetation of mitral valve) and three minor (fever of at least 38 C).

Risk factors include the presence of a prosthetic heart valve, structural or congenital heart disease, intravenous drug use and a recent history of invasive procedures.

Common blood culture isolates include staphylococcus aureus, viridans streptococcus, enterococci and coagulase negative staphylococci. Blood cultures collected from three locations of puncture at 30 minutes intervals The result of blood cultures then revealed to negative of bacterial growth. Blood culture-negative endocarditis (BCNE) may represent up to 70% of all endocarditis cases.

The patient was treated for 2 weeks given antibiotics therapy and heart failure management, infection and heart failure were under controlled and no evidence of valvular deterioration nor enlarging vegetation, alas, early surgery approach was chosen considering effective antibiotics administration and other evident indications.

Poor prognosis in patients with IE comes from complications its cause, where IE patients with congestive heart failure mortality rate as high as 50 %, thus immediate treatment plan is vital.[6] Those complications require early surgery to be done are Uncontrollable Infection, heart failure and to prevent embolization. Where reduction in mortality with surgery is highest among patients with Moderate to severe Heart Failure. AHA recommendation stated that any valvular dysfunction induced Heart failure require early surgery. Uncontrolled infection manifests in evidence of persistent infection, heart block or abscess, or resistant organism likewise S. Aureus and Fungi. Indication of preventing embolization by ESC guideline are vegetation over 10 mm or 15 mm of isolated vegetation feasibility in valve repair, or by AHA guideline that recurrent emboli, persistent vegetation despite antibiotic therapy or large mobile vegetation on native valve.[4]

Definition of early surgery are somewhat fluid, As AHA recommendation that early surgery is to be done during the first hospitalization before completion of antibiotic therapy. The necessity for potential surgical intervention decision derived from specific founding in Transthoracic Echocardiography/Transoesophageal echocardiography, the features are categorically divided to 1) Vegetation, Enlargement of size despite antimicrobial therapy, persistent, size over 10 mm in Anterior Mitral Leaflet, and multiple embolic events during 2 weeks of antimicrobial therapy. 2) Valvular dysfunction, Acute Aortic or Mitral Regurgitation with signs of Ventricular failure, Unresponsive HF despite therapy. 3) Perivalvular extension, valvular perforation, rupture or fistula, sign of new heart block and large abscess.

Under AATS guideline once surgery indication is evident surgery should not be delayed, especially after effective administration of pre operation antibiotic therapy.[1] Because the outcomes can be drastically improved as in reduction of in-hospital mortality and advancement in long-term prognosis in this often lethal disease.[6] Valve repair is performed whenever possible, particularly for the mitral valve, because repair is associated with improved long-term survival advantage compared to replacement.[5]

CONCLUSION

Infective Endocarditis along with its dire complications needs appropriate treatment plan, The decision to perform early surgery to this patient resulting favorable end, opening possibility to do it in regards of capitalizing improvement of patient's condition and shortening length of stay over scalable risk that present in this patient.

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Acute Upper Limb Ischemia in Electric Cigarette Smoker: A Case Report

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ABSTRACT

Acute limb ischemia is defined as a sudden decrease in limb perfusion with less than two weeks onset. Upper limb ischemia is less common than lower limb ischemia

A man, 36 years old, presented to the ER with paresthesia and cold sensation in his right hand ten hours before admission. Radiating pain, cough and dyspnea on effort one week before were admitted. Hypertension and diabetic mellitus history were unknown. Electric cigarette smoking history was admitted. On examination, BP 137/96, HR 125, RR 28, non-febrile, SpO2 91 room air. Impalpable radialis artery, pallor,cold palpation in right hand were found. Oxygen saturation in the right hand fingers were undetectable. Increased random blood sugar to 486. The patient was diagnosed with acute upper limb ischemia and diabetic mellitus type II. The patient was sent to RSUD Dr. Moewardi and conducted MSCT Angiography showed total stenosis from 1/3 medial to 1/3 distal dextra radialis et ulnaris artery and subtotal stenosis in subclavian et axillary artery treated by heparin, cilostazol, warfarin, and aspirin.

This patient presented to ER with classic six Ps and high risk factors of ALI (electric cigarette smoker and diabetic mellitus). MSCT Angiography then showed ALI Rutherford II

INTISARI

Iskemia tungkai akut adalah penurun fungsi tungkai secara mendadak dengan onset kurang dari dua minggu. Iskemia akut pada tungkai atas lebih jarang terjadi dibandingkan dengan tungkai bawah

Seorang laki-laki berusia 36 tahun datang dengan keluhan kesemutan di tangan kanan disertai rasa dingin sejak 10 jam terakhir didahului dengan nyeri dada menjalar, batuk, dan sesak nafas saat beraktivitas sejak satu minggu sebelumnya. Riwayat hipertensi dan diabetes melitus tidak diketahui sebelumnya dan pasien merupakan perokok aktif elektrik. Pada pemeriksaan fisik diketahui TD 137/96, Nadi 125, laju nafas 28, suhu dalam batas normal, saturasi oksigen 91%. Tangan kanan terlihat pucat dan teraba dingin, denyut nadi pada arteri radialis tidak teraba, dan saturasi pada jari tangan kanan tidak terdeteksi. Gula darah sewaktu tercatat 486. Pasien didiagnosis sebagai iskemia tungkai atas akut dan diabetes melitus. Pasien lalu dirujuk ke RSUD Dr. Moewardi untuk dilakukan CT Scan Angiografi dan ditemukan stenosis total pada 1/3 media hingga 1/3 pangkal arteri radialis dan ulnaris dan stenosis sebagian pada arteri subklavia dan aksilaris kanan. Pasien diberikan terapi oral berupa heparin, cilostazol, warfarin, dan aspirin.

Pasien datang dengan gejala klasik iskemia tungkai akut yaitu 6P dan memiliki risiko tinggi yaitu merokok elektrik dan diabetes melitus. Pada CT Scan Angiografi ditemukan iskemia tungkai atas akut tipe Rutherford II.

INTRODUCTION

Acute limb ischemia is defined as a sudden decrease in limb perfusion with the onset of symptoms of less than two weeks duration. Acute limb ischemia is a life-threatening condition caused by various etiologies including atherothrombosis and peripheral embolization[1]. Upper limb ischemia is less common than lower limb ischemia accounting for only one-fifth of all patients presenting with acute limb ischemia and relatively few cases have been reported[1,2].

The purpose of this study was to present a case of Acute Upper Limb Ischemic including its diagnosis, early management during the hospitalization in our hospital followed by other examination results data.

CASE PRESENTATION

A man, 36 years old, presented to the ER with paresthesia in his right hand ten hours before admission, with pain radiating beforehand. Cold sensation in the right hand was also acknowledged. He also admitted dyspnea on effort one week before admission accompanied with cough. Previous similar complaint, hypertension, and diabetic mellitus history were denied. Electric cigarette smoking history was admitted.

On examination, BP 137/96, HR 125, RR 28, non-febrile, SpO2 91 room air. Impalpable radialis artery, pallor and cold palpation in right hand were found. Oxygen saturation evaluation in the right-hand fingers were also undetectable. Blood examination showed increase aPTT to 211.6 and increased random blood sugar to 486. ECG showed normal sinus rhythm. Thorax rontgen showed no abnormality. The patient was diagnosed with acute upper limb ischemia suspected right brachialis artery occlusion and diabetic mellitus type II and given heparin as the initial therapy.

The patient was sent to RSUD Dr. Moewardi and conducted MSCT Angiography showed ALI Rutherford II with total stenosis from 1/3 medial to 1/3 distal dextra radialis et ulnaris artery and soft plaque with subtotal stenosis in subclavian et axillary artery treated by heparin, cilostazol, warfarin, and aspirin, Diabetic mellitus II complaint controlled by insulin.

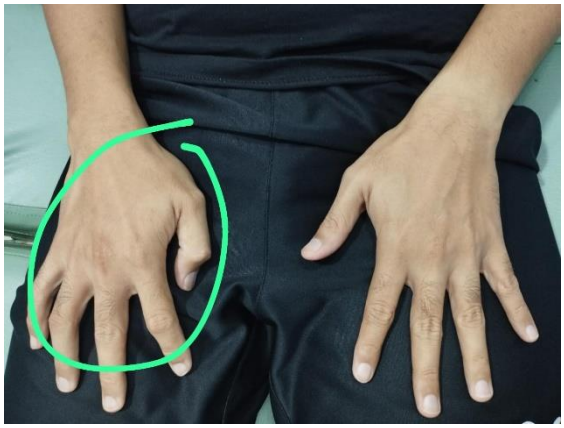

Figure 1. On admission patient's clinical presentation

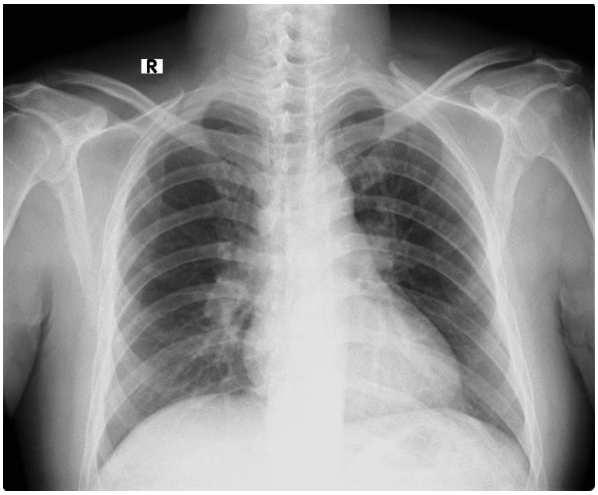

Figure 2. On admission Chest X-Ray showing no abnormality

DISCUSSION

Limb ischemia refers to symptomatic disruption of arterial blood flow to the extremities and is classified into acute and chronic limb ischemia[3]. The incidence of ALI is approximately 1.5 cases out of 10,000 people per year[4]. The upper extremity accounted for an average of 17 percent (range 7 to 32 percent) of cases of acute limb ischemia in one systematic review.

The cause, pathophysiology, and treatment of upper-extremity ischemia can be differentiated between small vessel and large vessel disease. Small vessel arteriopathies in the upper extremity lead to distal extremity and hand ischemia. These small vessel diseases include autoimmune or connective tissue disease, such as scleroderma, rheumatoid arthritis, systemic lupus, Buerger's disease (thromboangiitis obliterans), and Raynaud phenomenon. Large vessel artery disease in the upper extremity is mainly attributed to atherosclerosis. De novo atherosclerotic arterial disease in the upper extremities is associated with known risk factors, particularly diabetes and renal failure, and with peripheral arterial disease (PAD) involving lower extremities[5]. The most common location of occlusive

disease in the upper extremity is the left subclavian artery origin[1,2].

ALI is a medical emergency, and is important that the diagnosis is confirmed promptly by the classic "six Ps" (pain, pallor, pulselessness, poikilothermia [perishing with cold], paraesthesia, and paralysis) for assessing the clinical severity of ischaemia. Determination of the ankle brachial pressure index (ABI) using hand held Doppler can be predict the outcome as well as an index <0.7 is critical. However, there are no studies that support the routine use of biomarkers to predict limb salvage and survival after ALI[6]. The diagnosis is clinical and the level of occlusion can be determined by palpation of pulses. Confirmation is by arterial imaging with DUS or CTA. Arterial imaging may not be necessary before intervention for every patient with acute upper limb ischaemia. If the patient has a typical cardiac embolus (AF, short history, and normal arterial pulses elsewhere), it may be reasonable to proceed to treatment immediately if the limb is immediately threatened and if the axillary artery pulse in the upper arm is easily palpable. If the ischaemia is not typically embolic or the axillary pulse is not palpable, imaging of the proximal upper limb vessels is mandatory before treatment (in most cases a CTA). Blind embolectomy in this situation may not improve the blood flow to the hand and may simply make the ischaemia worse[3].

The therapeutic strategy will depend on type of occlusion (thrombus or embolus), location, type of conduit (artery of graft), Rutherford class, duration of ischemia, co-morbidities and therapy-related risks and outcomes. Rutherford Class I in ALI can be treated with medical therapy or revascularization electively. Class IIa can be treated with endovascular (more preferred) or surgical treatment (if ischemic symptoms occur more than 2 weeks). Class IIb is preferred with surgical revascularization. Class III is not indicated for immediate revascularization instead requires amputation[7]. In ALI, systemic anticoagulation with unfractionated heparin needs to be immediately given to decrease the risk of further clot propagation and to prevent microvascular thrombosis. An initial bolus of 100 Units/kg is given, followed by an intravenous infusion of 1000 Units/h. If urgent revascularization is not undertaken, the heparin dose should be titrated to maintain aPTT between 50 and 80 s or 2.0–3.0 times normal values. For patients with heparin allergy, direct thrombin inhibitor can be used and titrated to an aPTT of 50–80[3].

Complications among ALI patients are high and despite early revascularization, 30-day mortality and amputation rates are between 10 and 15%[8].

CONCLUSION

ALI is a life-threatening condition and is important that the diagnosis is confirmed promptly by the classic "six Ps" and Determination of the ankle brachial pressure index (ABI) using hand held Doppler. Acute limb ischemia is a life-threatening condition caused by various etiologists including atherothrombosis and peripheral embolization. This patient presented to ER with classic six Ps and has high risk factors to the occurrence of ALI which were electric cigarette consuming and diabetic mellitus. As conducted MSCT Angiography in this patient showed ALI Rutherford II, then given heparin, cilostazol, warfarin, and aspirin. Diabetic mellitus II complaint controlled by insulin. Regardless of the rare incidence, every clinician especially in the low-resource setting should be aware of this disease for potentially fatal complication as the mortality remains high, early prompt diagnosis and treatment is the key to achieve an optimal outcome.

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Tachy-Brady Syndrome in Acute Coronary Syndrome. How to Treat? A Case Report

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ABSTRACT

SND also known as SSS, is the inability of the SA node to produce an adequate rhythm as pacemaker function. SND includes various symptoms and abnormal ECG findings such as sinus bradycardia, sinus pauses/arrest, sinus exit blocks, or TBS.[1]

The patient was diagnosed with SND and unstable angina. He was hospitalized for 5 days. During admission, the patients ECG kept changing from TAVB to AF. The patient was observed intensively and was given DAPT and anticoagulant. Digoxin IV was given if tachycardia occurs and patient showed unstable signs. Patient was discharged after improvement of symptoms and was suggested for PPM.

During hospitalization, patient felt chest discomfort and palpitations. Even though the patient was diagnosed with ACS, PCI was not performed because there were no sign of infarction, which excludes ACS as its cause.4 The current treatment options for TBS involve correction of extrinsic causes, such as potassium disturbance or hypocalcemia, which in this case was excluded because of normal electrolytes. To control heart rate, atropine, isoproterenol, or temporary pacing can be given in bradyarrhytmias, while tachyarrhytmias can be managed by digoxin and propranolol.[2] During observation, ECG still showed TBS albeit the medication given. Because of this dynamics changes between TAVB and AF, the patient was suggested for PPM implantation.3

PPM implantation should be considered for SND patient who has AF associated with TAVB. Reversible and extrinsic causes need to be managed before adjusting the next treatments.

INTISARI

SND atau diketahui juga sebagai SSS, adalah ketidakmampuan nodus SA unutk memproduksi ritme yang adekuat sebagai fungsi pacemaker. SND adalah beberapa gejala dan ekg yang tidak normal termasuk sinus bradikardia, sinus arrest, sinus exit blocks atau TBS.[1]

Pasien didiagnosa sebagai SND dan angina tidak stabil. Pasien dirawat selama 5 hari. Saat perawatan, rekam jantung pasien terus berubah dari TAVB menjadi AF. Pasien dipantau secara ketat dan diberikan DAPT serta antikoagulan. Injeksi digoxin diberikan jika muncul tanda tidak stabil dan takikardia. Pasien dipulangkan setelah adanya perbaikan dari gejala dan disarankan rujuk untuk pemasangan PPM.

Saat perawatan, pasien merasakan dada tidak nyaman dan berdebar. Walaupun pasien didiagnosa ACS, PCI tidak dilakukan karena tidak adanya tanda infark, yang membuat ACS dieksklusi dari penyebab. Penanganan TBS terkait dengan koreksi dari faktor ekstrinsik sperti kelainan potassium atau hipokalsemia, yang mana pada kasus dieksklusi karen hasil elektrolit yang normal. Pada kondisi bradikaritmia dapat diberikan atropine, isoproterenol, atau pemasangan temporary pacing. Sedangkan pada kondisi takiaritmia dapat diberikan digoxin dan propranolol.[2] Pada perawatan, pasien masih

menunjukkan TBS walaupun obat sudah diberikan. Maka dari itu pasien disarankan rujuk untuk pemasangan PPM. Pemasangan PPM bisa dipertimbangkan pada pasien SND yang memiliki kaitan AF dengan TAVB. Penyebab yang reversibel serta faktor ekstrinsik harus ditangani sebelum melakukan penanganan selanjutnya

INTRODUCTION

SND also known as SSS, is the inability of the SA node to produce an adequate rhythm as pacemaker function. SND includes various symptoms and abnormal ECG findings such as sinus bradycardia, sinus pauses/arrest, sinus exit blocks, or TBS.[1]

CASE ILUSTRATION

The patient was diagnosed with SND and unstable angina. He was hospitalized for 5 days. During admission, the patients ECG kept changing from TAVB to AF. The patient was observed intensively and was given DAPT and anticoagulant. Digoxin IV was given if tachycardia occurs and patient showed unstable signs. Patient was discharged after improvement of symptoms and was suggested for PPM.

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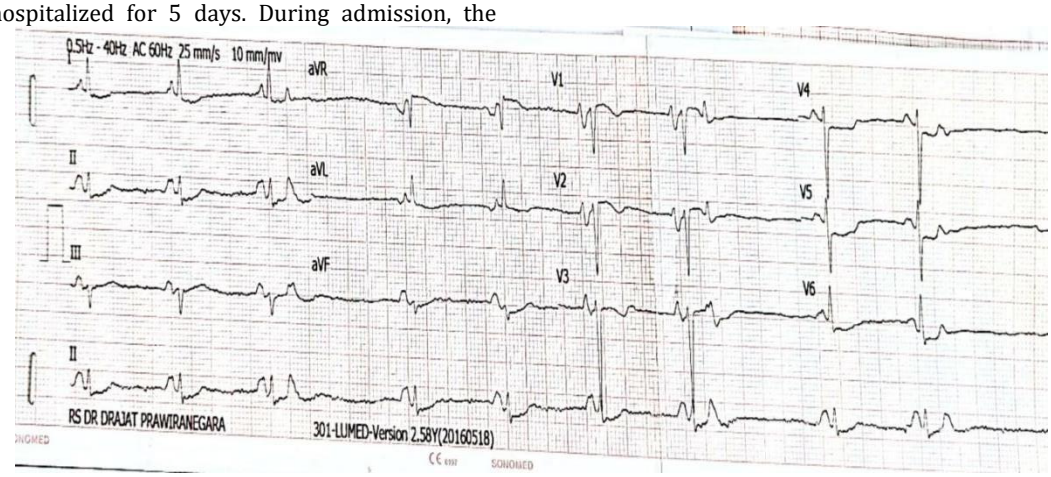

Figure 1. ECG Showing TAVB

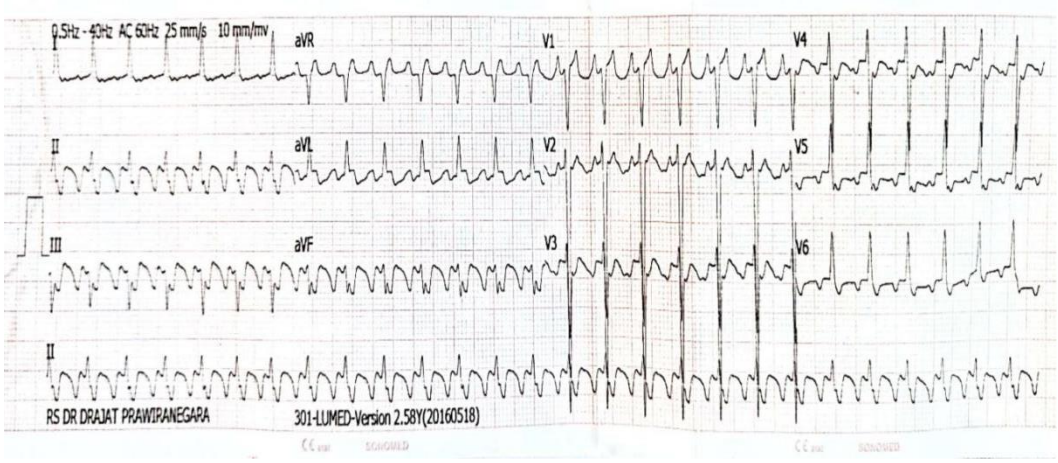

Figure 2. ECG showing AF

DISCUSSION

During hospitalization, patient felt chest discomfort and palpitations. Even though the patient was diagnosed with ACS, PCI was not performed because there were no sign of infarction, which excludes ACS as its cause.[4] The current treatment options for TBS involve correction of extrinsic causes, such as potassium disturbance or hypocalcemia, which in this case was excluded because of normal electrolytes. To control heart rate, atropine,

isoproterenol, or temporary pacing can be given in bradyarrhytmias, while tachyarrhytmias can be managed by digoxin and propranolol.[2] During observation, ECG still showed TBS albeit the medication given. Because of this dynamics changes between TAVB and AF, the patient was suggested for PPM implantation.[3]

CONCLUSION

PPM implantation should be considered for SND patient who has AF associated with TAVB. Reversible and extrinsic causes need to be managed before adjusting the next treatments

DISCLOSURES AND ETHICS

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Bromocriptine in the Therapeutic Management of Peripartum Cardiomyopathy : Case Report

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Keywords: peripartum cardiomyopathy; bromocriptine; heart failure – pregnancy; LVEF

ABSTRACT

Introduction: Peripartum cardiomyopathy (PPCM) is recognized as a major cause of pregnancy related heart failure with high morbidity and mortality

Case Illustration: A 29 years old female came to ER with complaints sudden severe shortness of breath since 1 hour before admitted. The patient had a post partum history 3 months ago by sectio caesaria for indications of preeclampsia. The Clinical examination showed BP 140/82 mmHg HR 107/ min , RR 35-38/min, SpO2 97% room air and diffuse rales in both lung . The ECG Showing presence of Sinus tachycardia and mital p waves. The Chest X ray showing presence of Cardiomegaly with Lung Edema intertitial. In Echocardiography was found dilatation of left atrium and left ventricle , LVEF 36% . The patient we treated with drip furosemide 5 mg / hour, drip Isosorbide Dinitrat 1 mg/hour , drip Dobutamine 3 mcg/ kg/ min and Bromocriptine 2,5 mg twice daily

Discussion: The etiology of PPCM remains unknown, a combined mechanism involving host susceptibility and systemic angiogenic imbalance is the pathogenesis of this disease. The proteolytic degradation of the nursing hormone prolactin generates a smaller 16 KDa prolactin molecule also known as vasoinhibin, which prevents angiogenesis and promotes cellular apoptosis. Vasoinhibin also stimulates microRNA-146a expression on endothelial cells, which subsequently drives endothelial cell injury and impairs cardiomyocyte metabolism, resulting in myocardial dysfunction. Furthermore, based on the pathological role of the 16 KDa prolactin fragment in PPCM, treatment with bromocriptine could prevent the development of PPCM by binding dopamine D2 receptors, thereby inhibiting the pituitary release of full-length prolactin. Several studies have demonstrated that bromocriptine, in combination with conventional HF therapy, appears to benefit left ventricular ejection fraction (LVEF) and maternal morbidity and mortality in women with acute PPCM

*Conclusion:*The optimal management of PPCM necessitates further research into alternative disease-specific treatments.

INTISARI

Pendahuluan: Kardiomiopati peripartum (PPCM) diketahui sebagai penyebab utama gagal jantung terkait kehamilan dengan morbiditas dan mortalitas yang tinggi.

Presentasi Kasus: Seorang perempuan berusia 29 tahun datang ke IGD dengan keluhan sesak napas berat yang tiba-tiba sejak 1 jam sebelum masuk rumah sakit. Pasien mempunyai riwayat postpartum 3 bulan yang lalu dengan operasi caesar karena indikasi preeklampsia. Pemeriksaan klinis menunjukkan TD 140/82 mmHg HR 107/menit, RR 35-38/menit, SpO2 97% udara ruangan dan ronki menyebar di kedua paru. EKG Menunjukkan adanya Sinus takikardia dan gelombang mital p. Rontgen thorax menunjukkan adanya Kardiomegali dengan intertitial Edema Paru.

Pada pemeriksaan Ekokardiografi ditemukan dilatasi atrium dan ventrikel kiri, LVEF 36% . Pasien diterapi dengan drip furosemid 5 mg/jam, drip Isosorbide Dinitrat 1 mg/jam, drip Dobutamin 3 mcg/kg/menit dan Bromokriptin 2,5 mg dua kali sehari

Diskusi: Etiologi PPCM masih belum diketahui, mekanisme gabungan yang melibatkan kerentanan pasien dan ketidakseimbangan angiogenik sistemik merupakan patogenesis penyakit ini. Degradasi proteolitik dari hormon prolaktin menghasilkan molekul prolaktin 16 KDa yang lebih kecil yang juga dikenal sebagai vasoinhibin, yang mencegah angiogenesis dan mendorong apoptosis seluler. Vasoinhibin juga menstimulasi ekspresi microRNA-146a pada sel endotel, yang selanjutnya menyebabkan cedera sel endotel dan mengganggu metabolisme kardiomiosit, sehingga mengakibatkan disfungsi miokard. Lebih lanjut, berdasarkan peran patologis dari fragmen prolaktin 16 KDa pada PPCM, pengobatan dengan bromokriptin dapat mencegah perkembangan PPCM dengan mengikat reseptor dopamin D2, sehingga menghambat pelepasan prolaktin full-length oleh hipofisis. Beberapa penelitian telah menunjukkan bahwa bromocriptine, dalam kombinasi dengan terapi gagal jantung konvensional, tampaknya memberikan manfaat pada fraksi ejeksi ventrikel kiri (LVEF) dan morbiditas dan mortalitas ibu pada wanita dengan PPCM akut.

Kesimpulan: Penatalaksanaan PPCM yang optimal memerlukan penelitian lebih lanjut mengenai pengobatan alternatif khusus terkait penyakit.

INTRODUCTION

Peripartum cardiomyopathy (PPCM) is an uncommon disease that characterized by a new onset of acute heart failure occurring among previously healthy pregnant patients between 1 month before and up to 5 months after delivery. Peripartum cardiomyopathy (PPCM) is recognized as a major cause of pregnancy related heart failure with high morbidity and mortality.[1,2]

CASE PRESENTATION

A 29 years old female came to ER with complaints sudden severe shortness of breath since 1 hour before admitted. The patient had a post partum history 3 months ago by sectio caesaria for indications of preeclampsia. The Clinical examination showed BP 140/82 mmHg pulse rate 107/ min , RR 35-38/min, SpO2 97% room air and diffuse rales in both lung . The ECG Showing presence of Sinus tachycardia and mital p waves. The Chest X ray showing presence of Cardiomegaly with Lung Edema intertitial. In Echocardiography was found dilatation of left atrium and left ventricle , LVEF 36% . The patient we treated with drip furosemide 5 mg / hour, drip Isosorbide Dinitrat 1 mg/hour , drip Dobutamine 3 mcg/ kg/ min and Bromocriptine 2,5 mg twice daily

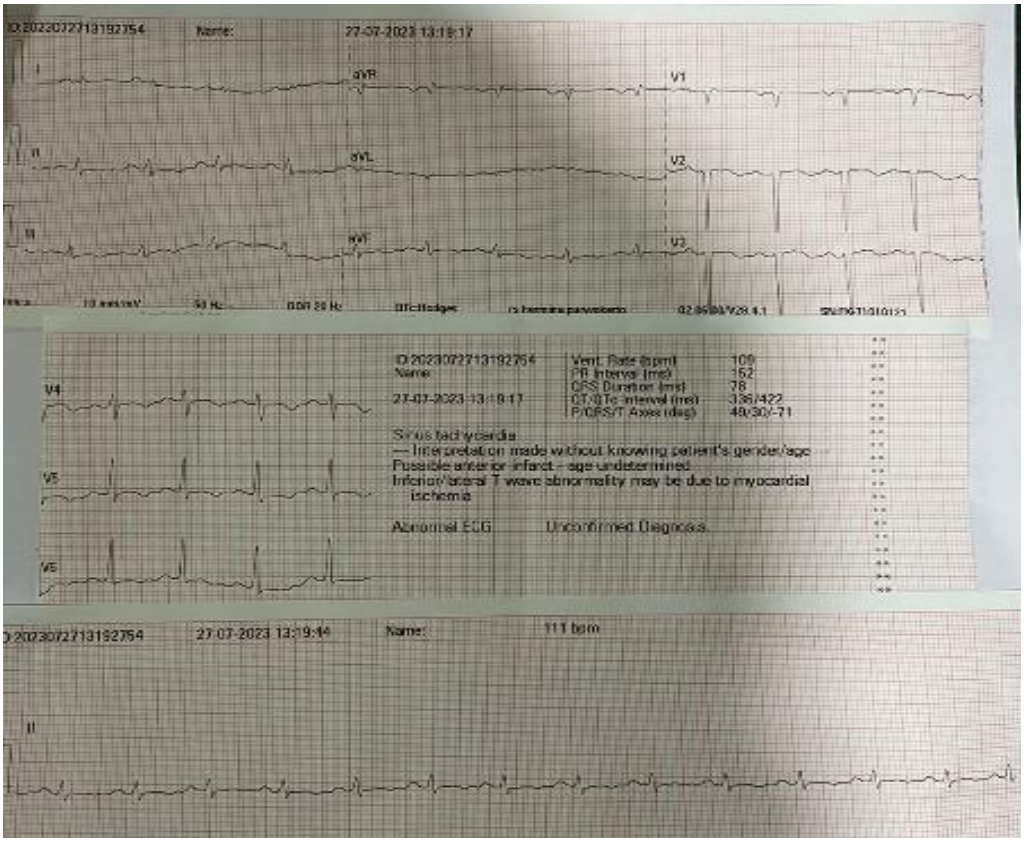

Figure 1. ECG

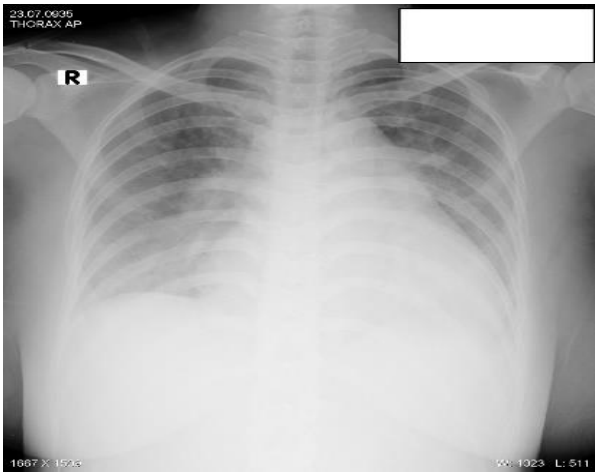

Figure 2. Rongent Thorax

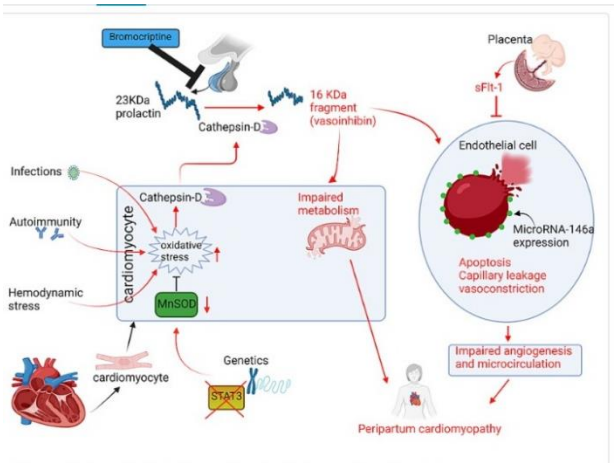

Figure 3 . The molecular pathophysiology of peripartum cardiomyopathy (PPCM)[3]

DISCUSSION

The etiology of PPCM remains unknown, a combined mechanism involving host susceptibility and systemic angiogenic imbalance is the pathogenesis of this disease.3 There are many factors contributing to PPCM include genetic mutations, decreased serum selenium, viral disorders, immune-mediated cytokines, autoimmunity, increased oxidative stress, inappropriate responses to

hemodynamic changes during pregnancy, and upregulation of angiogenesis inhibitors. General risk factors identified are hypertension, diabetes, obesity, smoking and substance abuse, African ancestry, and malnutrition. The possible pregnancy - related risk factors are pre-eclampsia, cesarean section, multi-parity, twin pregnancy, teenage pregnancy, advanced maternal age, and prolonged tocolytic therapy. There is no evidence-based disease specific therapy for PPCM.4 Recommended treatment for PPCM is similar to that of heart failure from other aetiologies based on registry data and expert opinion. Combining Bromocriptine and conventional treatment HF in the early stages has been shown to improve outcomes in patients with peripartum cardiomyopathy. Bromocriptine has been used for many years to stop lactation in postpartum women. In addition, prolactin-independent cytoprotective effects of bromocriptine were also shown in various organs including the heart. With the mechanism high levels of the nursing hormone prolactin and the production of a cleaved 16kDa N-terminal fragment of prolactin have emerged as potential key factors in the pathophysiology of PPCM.5 The proteolytic degradation of the nursing hormone prolactin generates a smaller 16 KDa prolactin molecule (16 KDa PRL), also known as vasoinhibin, which prevents angiogenesis and promotes cellular apoptosis. Vasoinhibin also stimulates microRNA-146a expression on endothelial cells, which subsequently drives endothelial cell injury and impairs cardiomyocyte metabolism, resulting in myocardial dysfunction. Furthermore, based on the pathological role of the 16 KDa prolactin fragment in this disease, treatment with bromocriptine could prevent the development of PPCM by binding dopamine D2 receptors, thereby inhibiting the pituitary release of full-length prolactin. Several studies have demonstrated that bromocriptine, in combination with conventional HF therapy, appears to benefit left ventricular ejection fraction (LVEF) and maternal morbidity and mortality in women with acute PPCM.[3]

Saint Croix et al.'s study reported that bromocriptine combined with regular HF treatment leads to an 11.37% increase in LVEF (mean difference 11.37, 95% confidence interval [CI]: 9.55-13.19; p = 0.001) after six months when compared with conventional HF treatment only. Pilot study by Sliwa K et al, reported that women receiving bromocriptine and standard HF therapy displayed significantly greater LVEF recovery (27%-58%, p = 0.012) than women receiving standard HF therapy only (27%-36%) at six months. Moreover, women on bromocriptine improved to NYHA functional class I after six months of treatment compared to those receiving standard HF therapy only, which improved to NYHA functional class II or III after six months.[6]

CONCLUSION

Here, we report the case of a patient who suddenly developed acute PPCM, we treat with combination bromocriptine and conventional treatment HF, that perhaps can contribute to improved prognosis in this still life-threatening disease. Finally, the optimal management of PPCM necessitates further research into alternative disease-specific treatments that target other molecules involved in the pathophysiological pathways of this disease. And large-scale multicenter studies with more extended follow-up periods are warranted to assess this more robustly

DISCLOSURES AND ETHICS

As a requirement of publication author(s) have provided to the publisher signed confirmation of compliance with legal and ethical obligations in: authorship, conflicts of interest, privacy and confidentiality and protection of human and animal research subjects.

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What Happens When Stemi Patients are Not Reperfused?

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Keywords: STEMI, reperfusion, PCI, fibrinolytic, outcome, survival

ABSTRACT

Background: ST-elevation myocardial infarction (STEMI) is a life-threatening coronary artery disease requiring immediate reperfusion. However, in rural areas, reperfusion therapy using percutaneous coronary intervention (PCI) or fibrinolytic treatment is not always available. This report describes a STEMI patient in the hospital without any PCI or fibrinolysis resources

Case Presentation: A 67-year-old smoker with hypertension and type 2 diabetes mellitus presented in the sub-district hospital with progressive chest pain and breathlessness since 3 hours ago. Vital signs showed increased blood pressure (BP 143/88) and slightly decreased oxygen saturation (SpO2 94%). An electrocardiograph (ECG) demonstrated inferior STEMI and sinus rhythm. The echocardiogram showed a reduced ejection fraction (EF 29.18%). The patient was treated with acetylsalicylic acid, ticagrelor, enoxaparin, furosemide, statins, and other supportive measures. ECG monitoring was performed daily. The patient suffered from total atrioventricular block (TAVB) on the first and second days of hospitalization. On the fifth day, the ECG reverted to sinus, and the patient was discharged.

Conclusion: STEMI management using pharmacological approaches is an alternative with comparable outcomes. Nevertheless, patient risk stratification and follow-up should be done to optimize care and survival.

INTISARI

Latar belakang: Infark miokard elevasi ST (STEMI) adalah penyakit arteri koroner yang mengancam jiwa yang membutuhkan reperfusi segera. Namun, di daerah pedesaan, terapi reperfusi dengan menggunakan intervensi koroner perkutan (PCI) atau pengobatan fibrinolitik tidak selalu tersedia. Laporan ini menggambarkan seorang pasien STEMI di rumah sakit tanpa sumber daya PCI atau fibrinolisis.

Presentasi kasus: Seorang perokok berusia 67 tahun dengan hipertensi dan diabetes melitus tipe 2 datang ke rumah sakit kecamatan dengan nyeri dada yang progresif dan sesak napas sejak 3 jam yang lalu. Tanda-tanda vital menunjukkan peningkatan tekanan darah (TD 143/88) dan saturasi oksigen yang sedikit menurun (SpO2 94%). Elektrokardiograf (EKG) menunjukkan STEMI inferior dan irama sinus. Ekokardiogram menunjukkan penurunan fraksi ejeksi (EF 29,18%). Pasien diobati dengan asam asetilsalisilat, ticagrelor, enoxaparin, furosemid, statin, dan tindakan suportif lainnya. Pemantauan EKG dilakukan setiap hari. Pasien menderita blok atrioventrikular total (TAVB) pada hari pertama dan kedua rawat inap. Pada hari kelima, EKG kembali ke sinus, dan pasien dipulangkan.

Kesimpulan: Penanganan STEMI menggunakan pendekatan farmakologis merupakan alternatif dengan hasil yang sebanding. Namun demikian, stratifikasi risiko pasien dan tindak lanjut harus dilakukan untuk mengoptimalkan perawatan dan kelangsungan hidup.

INTRODUCTION

ST-elevation myocardial infarction (STEMI) is a subset of acute coronary syndrome (ACS) diagnosed by history, electrocardiogram changes, and raised cardiac enzymes. The electrocardiogram pathognomonic for STEMI is the presence of new ST elevation in contiguous leads of 1 mm in limb leads or precordial leads other than V2 and V3. In V2 and V3, STEMI diagnosis is obtained when there is a 2 mm elevation in men or 1,5 mm in women. STEMI is responsible for 39% (31,653 patients) of all hospital admissions due to myocardial infarction in the UK and a non-adjusted 30-day mortality rate of 8.1% during 2013-2014. Most STEMI is caused by atherosclerotic plaque rupture with total vessel occlusion due to secondary thrombosis. Therefore, the key priority in STEMI management is the timely restoration of vessel patency to optimize myocardial salvage. The shorter the ischemic period, the higher the efficacy of the reperfusion therapy.[1,2,3]

PCI is a non-surgical, invasive procedure that relieves coronary artery occlusion and enhances the blood supply to the ischemic tissue. The system is usually done by ballooning the narrow segment (balloon angioplasty) or deploying a stent to keep the artery open (stent implantation). This procedure has been used extensively over the last 20 years, significantly reducing short-term mortality in STEMI patients. However, the efficacy of PCI is tightly linked to the interval between patient admission and balloon insertion (door-to-balloon). The recommended door-to-balloon time by the American Heart Association is less than 90 minutes. Another PCI pitfall is that it requires a specialized facility (i.e., a Catheterization lab), which is only sometimes available in hospitals in Indonesia. The door-to-balloon time limits the feasibility of transporting STEMI patients to cath-lab hospitals.[3,4]

Another type of reperfusion therapy is fibrinolytic. Streptokinase is the most widely used fibrinolytic agent, especially in economically burdened countries. Fibrinolytic agents are recommended in the American Heart Association guideline as an alternative therapy when PCI-capable hospitals are not within two hours of reach, or door-to-balloon cannot be achieved within 90 minutes.[5] However, both percutaneous coronary intervention (PCI) and fibrinolytic agents are only sometimes attainable in rural hospitals in Indonesia. This report elaborates on a STEMI patient in a C-level hospital without any PCI or fibrinolysis resources

CASE PRESENTATION

A 67-year-old male presented to the hospital three hours ago with progressive chest pain and breathlessness. The patient had a smoking history, uncontrolled hypertension, and type 2 diabetes mellitus. The man appeared weak, out of breath, and was in pain. Vital signs showed hypertension (BP 143/88), decreased oxygen saturation (SpO2 94%), HR 58 bpm, RR 26x/min, and temperature 36.1 degrees Celsius. A physical head-to-toe examination revealed rales in the bilateral lung base and epigastric tenderness. Electrocardiograph (ECG) demonstrated sinus bradycardia of 58 bpm, normal axis, ST elevation inferior leads (II, III, aVF), and reciprocal ST depression, suggesting inferior STEMI (Figure 1). The echocardiogram showed a reduced ejection fraction (EF 29.18%). Laboratory examination showed typical hematological values, increased blood glucose (229 mg/dL), and normal kidney function. The patient was diagnosed with inferior STEMI, congestive heart failure, and type 2 diabetes mellitus.

The initial management in the emergency unit was total bedrest, oxygen 2 liter/minute (nasal cannula), Ringer Lactate infusion ten drops/minute, enoxaparin IV injection 30 mg followed by subcutaneous injection 60 mg/12 hours, furosemide IV injection 20 mg/12 hours, pantoprazole IV injection 40 mg/24 hours, oral acetylsalicylic acid, oral ticagrelor, oral atorvastatin, oral rebapimide, and oral laxative agents. The patient was placed in the intensive care unit (ICU). The electrocardiogram demonstrated total atrioventricular block on the first and second days of hospitalizations (Fig 2-3). The treatment and daily ECG monitoring were continued. On the fifth day, the ECG reverted to sinus (Fig 4), and the patient was discharged.

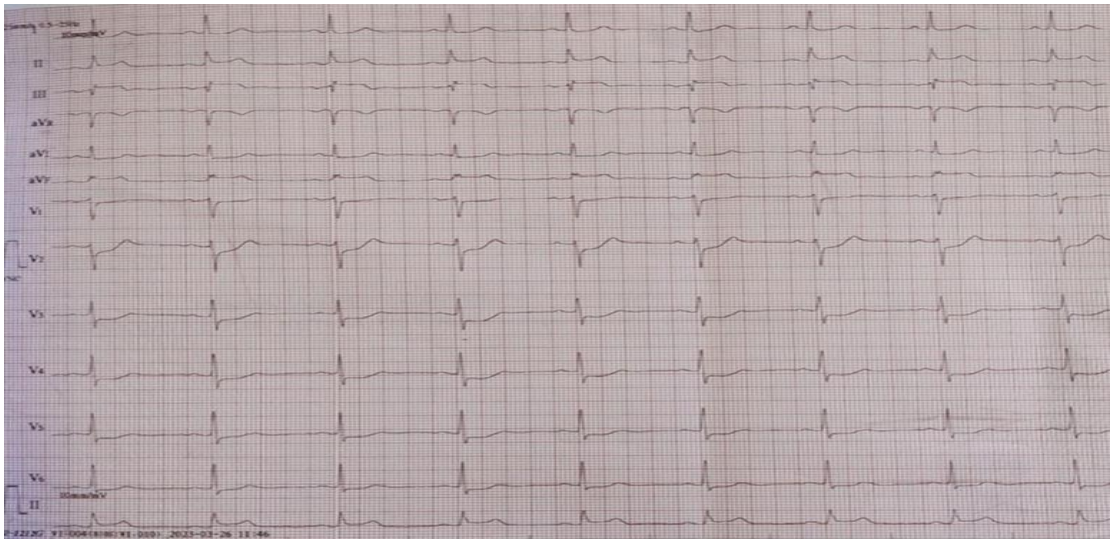

Fig 1. ECG at admission. The ECG demonstrated sinus rhythm and inferior STEM

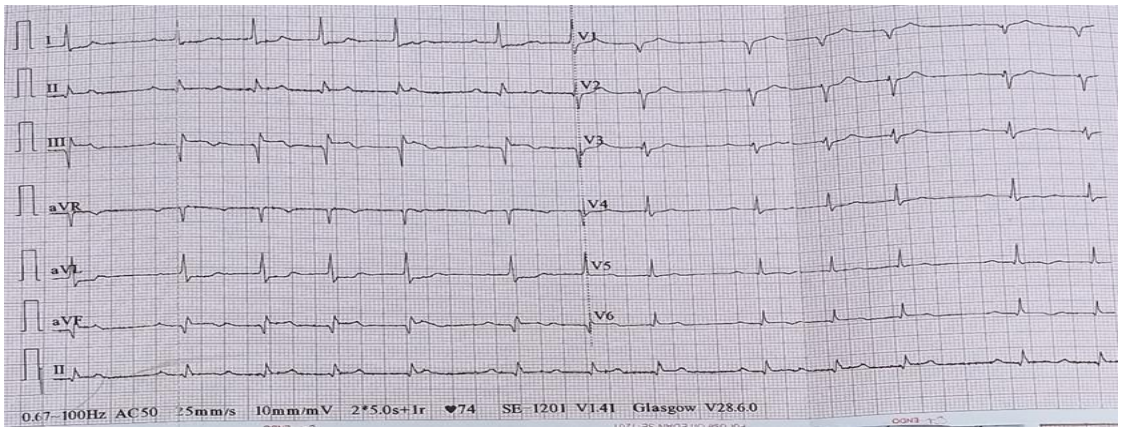

Fig 2. ECG on the first day of hospitalization. The ECG showed TAVB with junctional escape rhythm and inferior STEMI

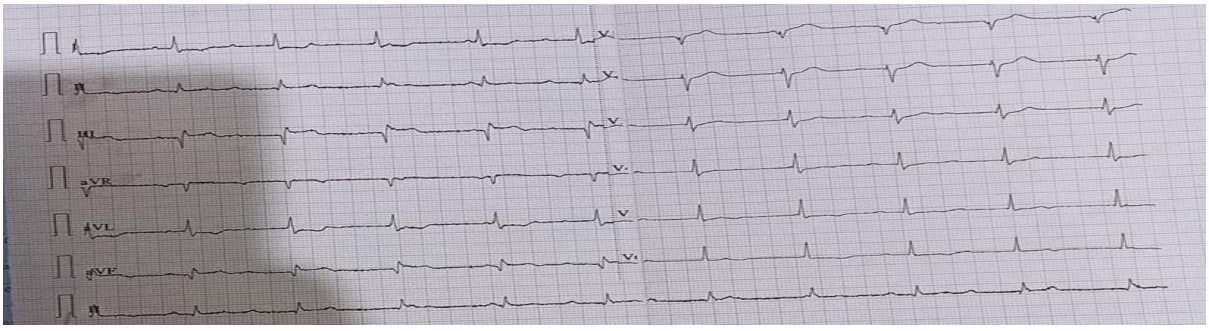

Fig 3. ECG on the second day of hospitalization. The ECG showed TAVB with junctional escape rhythm and inferior STEMI.

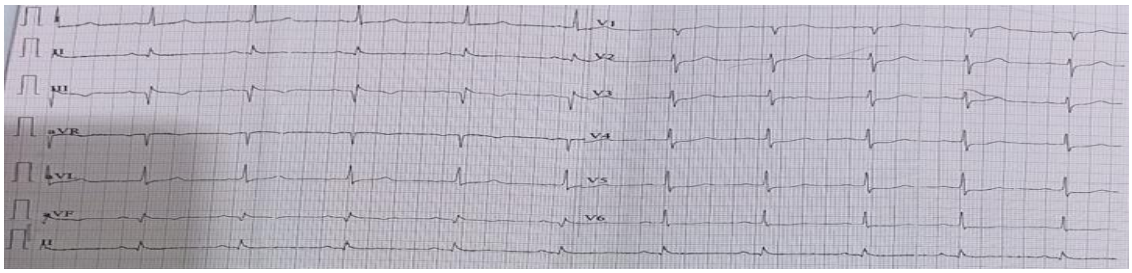

Fig 4. ECG at discharge. The ECG showed conversion to sinus rhythm.

DISCUSSION

The study aimed to report and discuss a case of STEMI patients not receiving reperfusion therapy. Reperfusion therapy remains the definitive treatment for STEMI patients due to its proven efficacy and mortality reduction. However, the need for reperfusion therapy is a problem in Indonesia and worldwide. About 37% (8,5-40%) of all STEMI patients worldwide did not receive reperfusion therapy. Specific cases were caused by coronary anatomy unsuitable for PCI (3,3%), uncontrolled comorbidities, mistakes in the diagnosis, and active bleeding.6,7 Yet, delayed presentation to hospital services was the primary cause (96.1%) in low-to-middle-income countries. In Mexico, the delayed presentation was caused by the difficulties of getting rapid medical attention, the fragmentation of medical services, and the need for more education regarding the importance of fast transport to hospitals.8 Indonesia was facing similar problems as Mexico. The non-reperfusion treatment in Indonesia was caused by a lack of resources due to geographical and structural barriers. Only 40 hospitals in 514 cities in Indonesia are PCI-capable, and most of them are located in large cities. The Jakarta Acute Coronary Syndrome (JAC) registry collected 2.103 ACS patients, including 654 acute STEMI patients admitted to the National Cardiovascular Center Harapan Kita, Jakarta, Indonesia. The study reported that 59% of patients did not receive reperfusion therapy, and almost 80% presented very late (>12 hours).[8]

Our patient was admitted with inferior STEMI and sinus bradycardia, and developed transient third-degree AV block (TAVB). TAVB is found in 8,1% of ACS patients. The risk of developing TAVB is significantly higher in STEMI than non-STEMI patients—two current hypotheses: (1) cardio-inhibitory reflex and (2) AV nodal ischemia. In STEMI patients, the ischemic left ventricular infero-posterior wall may induce vagal reflex, leading to cardio-inhibitory reflex, delayed impulse transmission from the atrium to the ventricle, and eventually TAVB. Two different rhythms can take over in TAVB. If it occurs above or at the crest of the AV node, the junctional rhythm will take over, QRS complexes will be narrow, and the rate will be 40-55 beats per minute. If the block occurs below the AV node, the ventricular rhythm will take over, instigating wide QRS and ventricular rate of 20-40 beats per minute.9,10 Sinus bradycardia is a frequent finding in 15-25% of patients with ACS. The right coronary artery perfuses the AV node, making the AV block more prominent in the inferior STEMI. According to the guideline, ACS patients with persistent or hemodynamic instability bradyarrhythmias are candidates for temporary ventricular pacing or permanent pacemakers if indicated after several days of observation. Our patient has good hemodynamic stability, and the AV block disappears after 48 hours, making the pacemaker unnecessary.[10]

Mortality and morbidity risks of STEMI patients were significantly higher among non-reperfused patients. The survival rates of non-reperfused vs. reperfused STEMI patients were 7.2% and 12.7%, respectively.7 Nevertheless, our patient recovered and was discharged, indicating the presence of other factors. Gopar-Nieto et al. found that age >65 years old (p = 0.013), systolic blood pressure <100 mmHg during admission (p<0.001), blood glucose >180 g/dL (p = 0.002), right bundle branch block (p = 0.006), and decreased glomerular filtration rate (p<0,001), and left ventricular ejection pressure below 40% (p<0.001) were associated with poor outcome in the multivariate analysis.7 Our patient was 67, hyperglycemic, and had a low ejection fraction. However, the absence of hypotension during admission, right bundle branch block, and kidney failure might contribute towards a good outcome.

Early patient presentation to the hospital influences survival as it facilitates earlier treatment. Administration of acetylsalicylic acid (p<0,001), P2Y12 inhibitor (p<0,001), heparin (p<0,001), enoxaparin (p<0,001), statins (p<0,001), intravenous nitrate (p<0,001), and intravenous/oral diuretics (p<0,001) were found to enhance survival in non-reperfused STEMI patients.7 Our patient was admitted three hours after the symptom onset and was treated with acetylsalicylic acid (aspirin), P2Y12 inhibitor (ticagrelor), enoxaparin, statins (atorvastatin), sublingual nitrates (ISDN), and intravenous diuretics (furosemide). This showed that the patient underwent adequate and appropriate medical treatment, which boosted survival without definitive reperfusion therapy.

CONCLUSION

Pharmacological approaches in STEMI patients are alternatives to reperfusion therapy with comparable outcomes. However, patient risk stratification and follow-up should be carried out to optimize care and prolong long-term survival. Further health promotion about acute coronary syndrome is also required to encourage early patient presentation to the hospital.

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A Case Report: A Challenging Diagnosis of Acute Myocardial Infarction in A Patient with Atypical Symptom and Right Bundle Branch Block

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Keywords: atypical chest pain; myocardial infarction; diagnosis

ABSTRACT

Introduction: Acute myocardial infarction (AMI) is a life-threatening condition characterized by chest pain. However, some patients do not show the classic symptoms of chest pain. This report presents a case of a patient that was diagnosed with acute myocardial infarction without typical presentation and shows how challenging it is in making a diagnosis of this patient

Case Presentation: A 70-year-old male complained of atypical chest pain. The pain was sharp, no penetration, no radiation, or diaphoresis. He just complained about dizziness during exercise. The patient was alert, the VAS score was 4 out of 10, and no signs of shocks. The first electrocardiography showed atrial fibrillation (AF) and the right bundle branch block (RBBB) pattern. The second and third electrocardiography showed only AF. During observation and therapy, the VAS score increased and electrocardiography showed ST-elevation in lead V1-V4 and also aVR. This patient was given inotropic and thrombolysis therapy since the hospital was not capable of performing percutaneous coronary intervention (PCI).

Conclusion: It was quite challenging to diagnose this case. The absence of obvious ST-T changes without any typical symptoms obscures the diagnosis. Concerning the fact that acute myocardial infarction is a life-threatening condition, physicians should pay more attention to those who come with an atypical symptom to avoid misdiagnosis and undertreatment.

INTISARI

Pendahuluan: Infark miokard akut (IMA) adalah kondisi yang mengancam jiwa yang ditandai dengan nyeri dada. Namun, beberapa pasien tidak menunjukkan gejala klasik nyeri dada. Laporan kasus ini menyajikan kasus seorang pasien yang didiagnosis menderita infark miokard akut tanpa gambaran yang khas dan menunjukkan sulitnya penegakan diagnosis pada pasien tersebut.

Presentasi Kasus: Seorang laki-laki berusia 70 tahun mengeluh nyeri dada yang tidak khas. Nyeri terasa tajam, tidak tidak tembus, tidak menjalar, dan tidak mengalami keringat dingin. Ia hanya mengeluh pening saat beraktivitas. Pasien dalam keadaan sadar, skor VAS 4 dari 10, dan tidak ada tanda-tanda syok. Pada rekaman elektrokardiografi pertama didapatkan gambaran fibrilasi atrium dan blok cabang berkas kanan. Rekaman kedua dan ketiga hanya didapatkan gambaran atrial fibrilasi. Selama observasi dan pemberian obat obatan, skor VAS pasien meningkat dan gambaran elektrokardiografi menunjukkan adanya ST elevasi pada sadapan V1-V4 dan juga aVR. Pasien ini kemudian diberikan inotropik dan dilakukan trombolisis karena rumah sakit tidak dapat melakukan intervensi koroner perkutan.

Kesimpulan: Cukup sulit untuk mendiagnosis pasien dalam kasus ini. Tidak adanya perubahan ST-T yang jelas disertai tidak adanya gejala khas berhasil mengaburkan diagnosis yang seharusnya. Mengingat fakta bahwa infark miokard akut merupakan kondisi yang mengancam jiwa, dokter harus lebih memperhatikan pasien yang datang dengan gejala atipikal untuk menghindari kesalahan diagnosis dan pengobatan yang tidak tepat.

INTRODUCTION

Acute myocardial infarction (AMI) is a life-threatening condition with a high mortality rate. The majority of cases of this illness are caused by the rupture of an atherosclerotic plaque, which leads to platelet aggregation and the creation of an intracoronary thrombus. AMI is diagnosed using two out of three diagnostic criteria. The first criterion is a symptom with a specific feature, such as retrosternal pressure radiating to the neck, mouth, or left shoulder and arm. The second criterion is the electrocardiography abnormalities (ST-T changes). The third criterion is the detection of specific serum markers of myocardial necrosis.[1]

Acute Myocardial infarction is classified with the terms ST-Elevation Myocardial Infarction (STEMI) and Non-ST-Elevation Myocardial Infarction (NSTEMI). STEMI is a spectrum of AMI that results from complete thrombus obstruction of the coronary artery, accompanied by ST-Elevation in electrocardiography (ECG) finding. NSTEMI differs from STEMI in the features of ST T alterations, which are characterized by ST depression and/or T inversion.[1]

However, some of the patients who experienced myocardial infarction or even STEMI do not show a specific appearance. Previous studies reported some cases of myocardial infarction that presented with breathlessness, dizziness, fatiguability, or syncope with no specific symptoms.[2] Another atypical presentation of myocardial infarction is RBBB finding that is recognized as one of the significant ECG patterns for occlusive myocardial infarction. The study suggested the need to pay critical attention to these patients.[3] Besides RBBB, other studies also stated that MI and AF are closely related through various mechanisms.[4]

This report presents a case of a patient that was diagnosed with acute myocardial infarction without typical presentation and shows how challenging it is in making a diagnosis of this patient.

CASE PRESENTATION

A 70-year-old male came to our emergency room with a chief complaint of atypical chest pain 3hours before arrival. His pain was sharp, no radiation, no penetration, triggered by activity, with the visual analogue scale (VAS) was 4 over 10. He also had no history of illness. He was fully alert with his blood pressure was 96/60 mmHg, his heart rate was 101 beats per minute, breaths rate was 18 breaths per minute, his body temperature was 36.6°C, and room air oxygen saturation was 98%. He also showed no sign of shock.

On his first ECG, we saw an atrial fibrillation with rapid ventricular response and a RBBB pattern. We performed a serial ECG because his symptoms and condition were not specific. The second serial ECG revealed atrial fibrillation with normal ventricular response, and the RBBB pattern vanished. His blood pressure began to decline, and his high sensitivity troponin I level was 1148 ng/dl. Even though the symptoms remained unspecific, he was diagnosed with Non-ST-Elevation Myocardial Infarction since two other criteria pointed to this diagnosis. We gave him antiplatelet, anticoagulant, and inotropic agents because the result of his fluid challenge test was inconclusive. There were no alterations in the third series ECG. His condition was clinically stable as well.

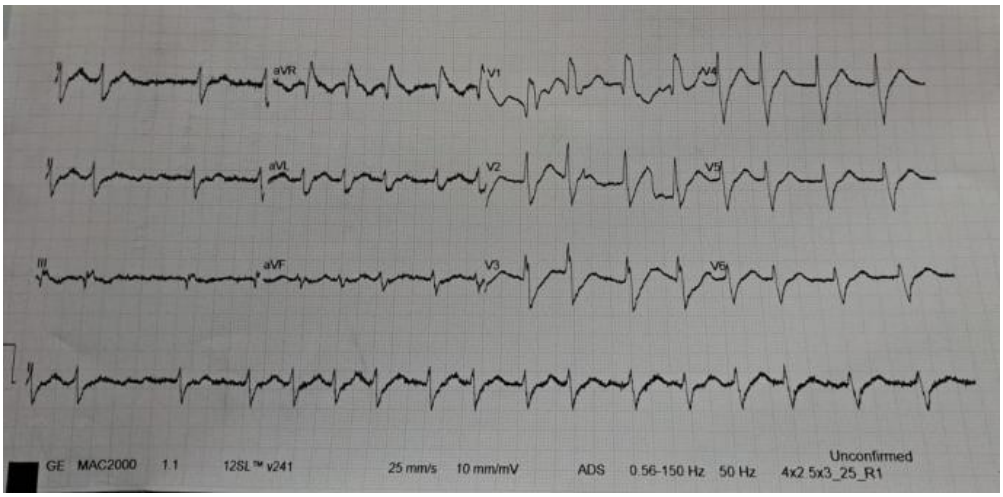

Figure 1. First ECG

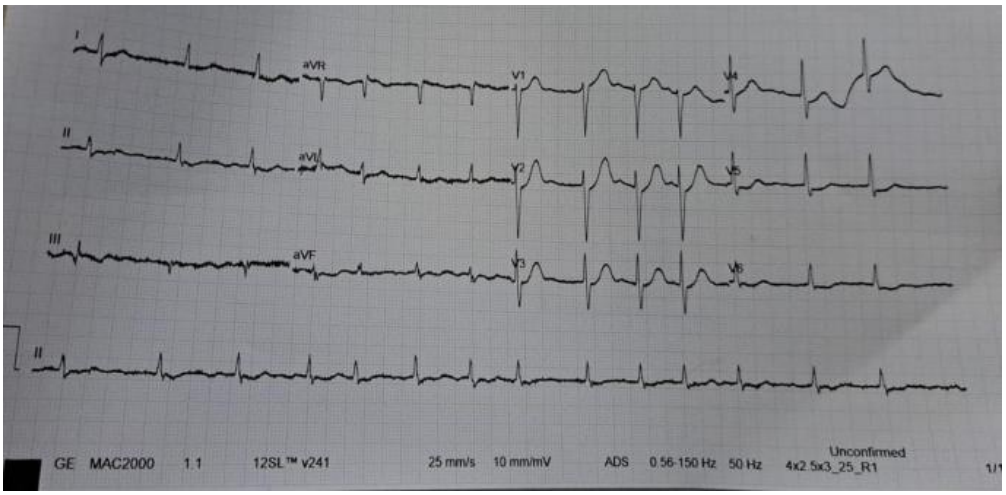

Figure 2. Second ECG

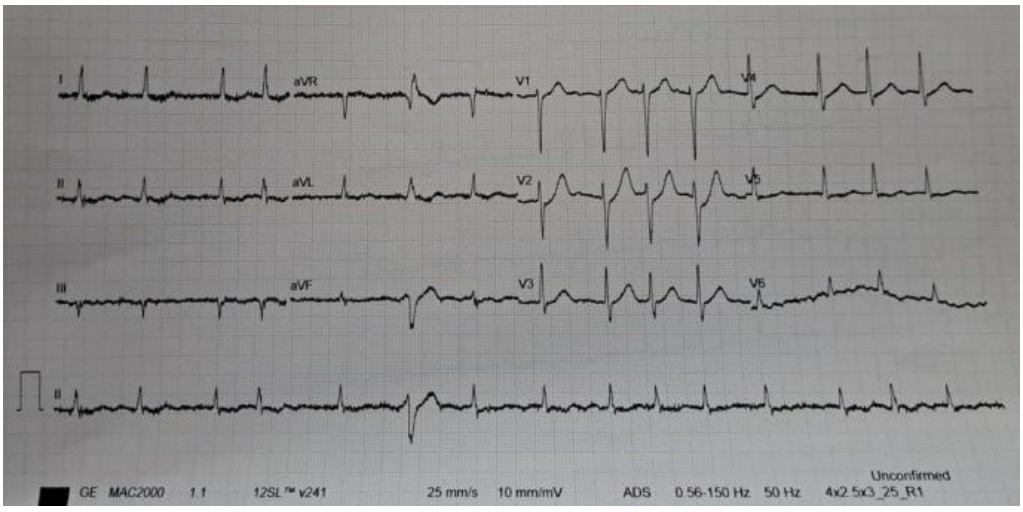

Figure 3. Third ECG

During observation, he suddenly agitated, experienced diaphoresis, his VAS score increased to 8 over 10, and his blood pressure continued to fall. We took another ECG and discovered an ST-Elevation in leads V1-V4 and aVR. He was then assessed with STEMI. We did thrombolysis and administered vasoconstrictor since our hospital was not capable of performing percutaneous coronary intervention.

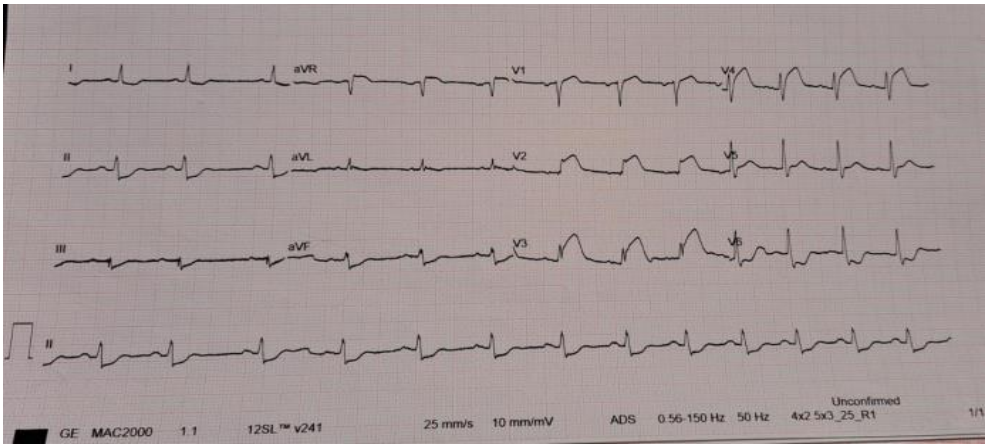

Figure 4. Fourth ECG

After completing the thrombolysis, his condition got better. There were no complications during the thrombolysis administration. His blood pressure became stable, his pain was relieved, his VAS score became 2 over 10, and his ECG showed normal sinus rhythm with significant ST-segment reduction. He was transferred to ICCU for further monitoring.

DISCUSSION

It was quite challenging to diagnose this patient. The absence of obvious ST-T changes without any typical symptoms obscures the diagnosis. The presence of AF and also RBBB on the first ECG recording also doubts in establishing the diagnosis. The clinician's skill and precision are required to diagnose this unique case.

Based on earlier research, new onset of atrial fibrillation was discovered in certain patients presenting with acute myocardial infarction. AF remains a prevalent and important consequence of AMI.5 Atrial fibrillation, on the other hand, can aggravate ischemia and cause atrial myocardial infarction (AMI) through atrial diastolic overload and increased inflammatory response.[6,7]

Besides presumably new AF, this patient also presented RBBB. Only a small percentage of a large cohort of undifferentiated patients who came with chest pain and RBBB (regardless of whether they were new or presumably new) developed acute coronary syndrome and even fewer had STEMI.[8] However, a new onset RBBB pattern in a patient with classic STEMI and ischemic symptoms should highlight the possibility of significant proximal LAD coronary blockage. It is widely recognized as one of the key ECG patterns for occlusive myocardial infarction associated with the worst prognosis and death, emphasizing the importance of paying close attention to these individuals. Given these patients' poor prognosis in the event of an AMI, it is critical to initiate reperfusion therapy as soon as possible.[3]

The patient's condition improved dramatically after the fourth evaluation. The ECG clearly showed ST elevation in leads V1-V4 and aVR. In the setting of acute coronary syndrome, ST-Elevation in aVR is frequently associated with severe disease of the left main coronary or proximal LAD artery, and doctors must be aware of this urgent state. Along with ST-Elevation in aVR, ST-Elevation in V1 with RBBB denotes an ischemic lesion in the basal septal area, which is frequently accompanied by insufficient backup circulation from the conus branch to the basal right septum.[9] This might also explain the hemodynamic instability of this patient.

CONCLUSION

Concerning the fact that acute myocardial infarction is a life-threatening condition, physicians should pay more attention to those who come with an atypical symptom to avoid misdiagnosis and undertreatment.

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Asystole, The Forgotten due to Hypokalemia

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Keywords: hypokalemia; asystole; diabetes Mellitus

ABSTRACT

Hypokalemia often causes clinical conditions, including weakness and difficulty breathing. In this case, there was a woman came with weakness and vomiting and diarrhea. Potassium measured 1.88 mEq/dL. After the measured potassium was reported, patient had seizures, then she was given an injection of diazepam to stop seizures and regained consciousness one hour after diazepam injection. Two hours after convulsions stopped, the patient wasn't breathing and no pulse was found on carotid pulse palpation. The patient was placed on a monitor and asystole. One cycle of CPR was performed, the patient responds. Then, the patient underwent electrolyte correction. One hour after regaining consciousness, the patient had asystole and CPR was performed. Less than one cycle, the patient responds. After that, the patient was treated in the ICU and corrected potassium 4 times. Potassium in patient was increased, started 1.88 at start, 2.88 after last correction

INTISARI

Hipokalemia seringkali menyebabkan keluhan klinis, diantaranya kelemahan dan kesulitan bernapas. Pada kasus ini, ada seorang wanita datang dengan kondisi lemas, muntah-muntah, dan diare. Kalium terukur 1,88 mEq/dL. Setelah kalium terlapor, pasien mengalami kejang dan diberikan suntikan diazepam untuk menghentikan kejang dan sadar kembali satu jam setelah pemberian diazepam. Dua jam setelah kejang berhenti, pasien tidak bernapas dan tidak ditemukan denyut nadi pada palpasi denyut karotis. Pasien dipasang monitor dan muncul gambaran irama asistol. Satu siklus CPR dilakukan, pasien merespons dengan baik. Kemudian, pasien menjalani koreksi elektrolit. Satu jam setelah sadar kembali, pasien mengalami asistol kebali dan dilakukan CPR. Kurang dari satu siklus, pasien kembali merespons. Setelah itu, pasien dirawat di ICU dan dikoreksi kalium sebanyak 4 kali. Kalium pada pasien meningkat, mulai 1,88 pada awal, 2,88 setelah koreksi terakhir

INTRODUCTION

Hypokalemia is defined as serum K+ levels (serum-[K+]) <3.5 mM[1]. Hypokalaemia is the most common electrolyte abnormality found in hospitalized patients and therefore represents an important cause of arrhythmias and associated mortality observed in clinical practice2. Hypokalemia often causes clinical conditions, including weakness and difficulty breathing. Low serum-[K+] might thus be a cause of arrhythmias in patients even without clinically recognized hypokalemia1. The following features are observed on the electrocardiogram (ECG) during hypokalaemia: ventricular premature complexes (VPCs), prolonged QT interval, ST segment depression and the appearance of a U wave[3]. However, often examiner forgets that hypokalemia, especially severe ones, can cause arrhythmias, one of which is asystole. Even moderate hypokalemia (2.5–3.0 mmol/l) can be highly arrhythmogenic in normal hearts. In isolated rabbit and rat hearts, we found that modestly reducing [K+]o to 2.7 mmol/l resulted in spontaneous polymorphic VT and VF in approximately over 50% of hearts studied, whereas severe hypokalemia (2.0 mmol/l) caused VF in 100%[4].

CASE PRESENTATION

A 47-year-old woman came to Emergency department with general weakness, vomiting profusely and diarrhea. Patient has history of Diabetes Mellitus and routinely uses long-acting insulin 18 units at night. Blood Sugar in this patient

measured 542 mg/dL. Advice given included using a syringe pump containing 50 units of rapid-acting insulin in 50 cc normal saline and measuring electrolytes. Due to limitations of equipment, electrolyte checking was carried out with partial reference. On admission to ward, potassium measured 1.88 mEq/dL. After the measured potassium was reported, patient had seizures, then she was given an injection of diazepam to stop seizures and regained consciousness one hour after diazepam injection. Two hours after convulsions stopped, the patient wasn't breathing and no pulse was found on carotid pulse palpation. The patient was placed on a monitor and asystole. One cycle of CPR was performed, the patient responds. Then, the patient underwent electrolyte correction. One hour after regaining consciousness, the patient had asystole again and CPR was performed. Less than one cycle, the patient responds. After that, the patient was treated in the ICU and corrected potassium 4 times, 3 times at ICU and once inward after ICU. Potassium in patient was increased, started 1.88 at start, 2,24 after first correction, 2.45 in the second one, 2,68 in the third one. After patient was stable in hemodynamic and rhythm, patient was transferred to the ward, and and the potassium correction was carried out one more time. The potassium in patient was increased to 2,88. Because there were no further complaints from the patient, including weakness, diarrhea and vomiting, the patient was discharged from the ward 12 hours after the fourth potassium correction.

DISCUSSION

Hypokalaemia is common in patients presenting with VT/VF, and those with severe hypokalaemia have found to be associated with preceding gastrointestinal illness, higher doses of diuretics, use of drugs such as anti-depressants, as well as post-operative settings[2].

The arrhythmic mechanism of hypokalemia is started from Reduced repolarization reserve predisposes the heart to EADs and EAD-mediated arrhythmias including Torsades de pointes (TdP) and polymorphic ventricular tachycardia (VT), which can degenerate to ventricular fibrillation (VF) causing sudden cardiac death[5]. Reductions in repolarization reserve prolong APD in a heterogeneous manner, both because ion channel expression is heterogeneous throughout the atria and ventricles and because EADs are intrinsically chaotic, occurring irregularly instead of reliably with every beat[6,7]. Due to the gap junction coupling in cardiac tissue that prevents adjacent myocytes from exhibiting markedly different APD, the chaotic behavior causes some regions to exhibit EADs synchronously whereas other nearby regions do not. This dynamical process has been termed “regional chaos synchronization” and generates marked dispersion of repolarization since areas with long APD due to EADs are juxtaposed next to regions much shorter APD without EADs[7,8]. Moreover, if His-Purkinje fibers or regions of myocardium with EADs reach the threshold for triggered activity, the resulting extrasystoles can propagate into recovered regions without EADs, but may block when propagating into other regions with subthreshold EADs, thereby initiating reentry. Under these conditions, reentry can have a special property called biexcitability[9]. Incomplete repolarization allows slow meandering rotors to propagate using the L-type Ca2+ current (manifesting as TdP or polymorphic VT), whereas full repolarization allows fast rotors to propagate using the Na+ current (manifesting as polymorphic VT or VF). This arrhythmia mechanism is called mixed focal-reentrant fibrillation (as opposed the multiple wavelet or mother rotor fibrillation) since the unstable rotors often self-terminate but new rotors are then initiated by ongoing EAD-mediated triggered activity arising from His-Purkinje tissue and/or ventricular myocardium[7,8].

The reduction in repolarization reserve by hypokalemia has classically been attributed to direct suppression of K+ channel conductances, but recent evidence indicates that indirect effects of hypokalemia leading to activation of late Na+ and Ca2+ currents play a key role as well[5].

Many K+ channels exhibit a strong allosteric dependence on extracellular K+ concentration [K+]o. In inward rectifier K+ channels, outward current through the channel is regulated by voltage-dependent block of the pore by cytoplasmic Mg2+ and polyamines that bind to the negative charges in the pore’s cytoplasmic vestibule and prevent passage of K+ ions. Extracellular K+ ions entering the pore from the outside electrostatically destabilize and “knock off” these blocking cations from their binding sites, restoring outward K+ flow10. Thus, even though hypokalemia hyperpolarizes EK, increasing the driving force for outward K+ flow (Em - EK), the increased stability of blocking cations in the pore decreases the conductance more powerfully, resulting in decreased outward K+ current.

The rate at which Na+-K+ ATPase transports ions depends both on the affinities of the extracellular and intracellular binding sites for Na+/K+ and Em, since the transport cycle moves one net positive charge outward[4]. Reducing [K+]o from 4.5 to 2.7 mmol/l decreases ion pumping rate by about 20% . Because Na+-K+ ATPase generates a net outward current and is inhibited by hyperpolarization, the combined effect reduced K+ binding and hyperpolarization is predicted to reduce Na+-K+ ATPase ion pumping rate by 43%11. The consequence was a slow rise in intracellular [Na+] that inhibited the ability of the Na+-Ca2+ exchanger to remove Ca2+ from the cell resulting in spontaneous diastolic Ca2+ waves[4].

Hypokalemia prolongs APD by reducing outward current through both K+ channels and Na+-K+ ATPase. The prolonged APD results in increased Ca2+ influx through Ca2+ channels. At the same time, intracellular Ca2+ removal via NCX is compromised by the elevated intracellular [Na+] from Na+-K+ ATPase inhibition. Together, these factors cause an increase in cytoplasmic [Ca2+] sufficient to activate CaMK12. When activated, CaMK phosphorylates a variety of protein targets, including Na+ channels, L-type Ca2+ channels and RyRs[13]. Na+ channel phosphorylation by CaMKII increases late Na+ current which further decreases repolarization reserve and also further exacerbates intracellular Na+ loading[14]. L-type Ca2+ channel phosphorylation by CaMKII both increases

current amplitude and slows inactivation13, increasing the Ca2+ window current that plays a critical role in EAD generation[15,16,17]. RyR phosphorylation by CaMKII increases RyR leakiness, further elevating diastolic [Ca2+] and promoting Ca2+ waves and DADs[13].

CONCLUSION

Up to 20% of patients admitted to the hospital exhibit hypokalemia18. Hypokalemia have powerful electrophysiological effects promoting cardiac arrhythmias. Hypokalemia ([K+]o<3.5 mmol/l) reduces repolarization reserve by directly inhibiting K+ channel conductances and indirectly by suppressing Na+-K+ ATPase. The latter results in intracellular Na+ and Ca2+ loading activating CaMK signaling whose targets include Na+ and Ca2+ channels, initiating positive feedback cascades that further reduce repolarization reserve to the range promoting EADs, DADs and afterdepolarization-mediated arrhythmias

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ECG Time: VT or ...? ; Abnormal ECG with loss of consciousness

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ABSTRACT

Sometimes, VT and LBBB waves are difficult to distinguish in daily practice. One of the cases that will be presented is the case of VT vs LBBB with loss of consciousness. An 82 years old woman came with declining consciousness for 6 hours before entering the hospital. Fever since last 3 days. Denied complaints of headache, vomiting, nausea and chest pain. The patient cannot mobilize properly. The patient has a history of NSTEMI 1 month ago. On the ECG, there is a picture between LBBB but the P waves are not clearly visible. The patient was given Amiodarone 150 mg bolus and there was no ECG change after 2 hours of administration. Later it was discussed, that the P wave was seen right after the T wave, so that the ECG was suspected of first degree AV block with LBBB, ST elevation in the precordial leads, with a differential diagnosis of VT. The patient is now fully conscious, but still has limitations due to the previous stroke

INTISARI

Terkadang gelombang VT dan LBBB sulit dibedakan dalam praktik sehari-hari. Salah satu kasus yang akan dipaparkan adalah kasus VT vs LBBB dengan kehilangan kesadaran. Seorang wanita berusia 82 tahun datang dengan kesadaran menurun sejak 6 jam sebelum masuk RS. Demam sejak 3 hari terakhir. Keluhan sakit kepala, muntah, mual dan nyeri dada disangkal. Pasien tidak dapat melakukan mobilisasi dengan baik. Pasien mempunyai riwayat NSTEMI 1 bulan yang lalu. Pada EKG terdapat gambaran di antara LBBB namun gelombang P tidak terlihat jelas. Pasien diberikan bolus Amiodarone 150 mg dan tidak terjadi perubahan EKG setelah 2 jam pemberian. Kemudian dibahas, bahwa gelombang P terlihat tepat setelah gelombang T, sehingga pada EKG diduga blok AV derajat I dengan LBBB, elevasi ST pada sadapan prekordial, dengan diagnosis banding VT. Pasien kini sudah sadar penuh, namun masih memiliki keterbatasan akibat serangan stroke sebelumnya.

INTRODUCTION

Sometimes, VT and LBBB waves are difficult to distinguish in daily practice. Because VT is a potentially fatal cardiac rhythm disturbance caused by electrical activation originating from an abnormal focus or electrical circuit in the myocardium of the ventricles[1,2]. LBBB is often misinterpreted as VT even though LBBB is not as deadly as VT. The clinical manifestations and consequences of VT range from syncope, cardiogenic shock, ventricular fibrillation (VF), electrical storm (ES), cardiac arrest, and Sudden Cardiac Death[3].

Left bundle branch block (LBBB) was first recorded electrocardiographically in humans in 1914[4]. Although the main features of contemporary LBBB definitions are similar (i.e. QRS prolongation, dominant S waves in lead V1 and lateral notching or slurring), differences in definitions were shown to result in significant discordance when scoring LBBB in clinical practice. Recently, the ESC proposed new electrocardiographic criteria to define LBBB. The updated ESC 2021 definition emphasises on the importance of QRS notching/slurring and delayed R-wave peak time, and provides new recommendations on ST-segment and T-wave assessment. However, how the 2021 revised definition performs in diagnosing LBBB compared with the previous ESC 2013 definition has not been addressed[5,6].

One of the cases that will be presented is the case of VT vs LBBB with loss of consciousness

CASE PRESENTATION

An 82 year old woman came with decreased consciousness since 6 hours before entering the hospital. Fever since last 3 days. Denied complaints of headache, vomiting, nausea and chest pain. The patient cannot mobilize properly. The patient has a history of NSTEMI 1 month ago. On the ECG, there is a picture (Figure 1) between LBBB but the P waves are not clearly visible. The patient was given Amiodarone 150 mg bolus and there was no ECG change after 2 hours of administration. Later it was discussed, that the P wave was seen right after the T wave, so that the ECG was suspected of first degree AV block with LBBB, ST elevation in the precordial leads, with a differential diagnosis of VT. The patient is now fully conscious, but still has limitations due to the previous stroke.

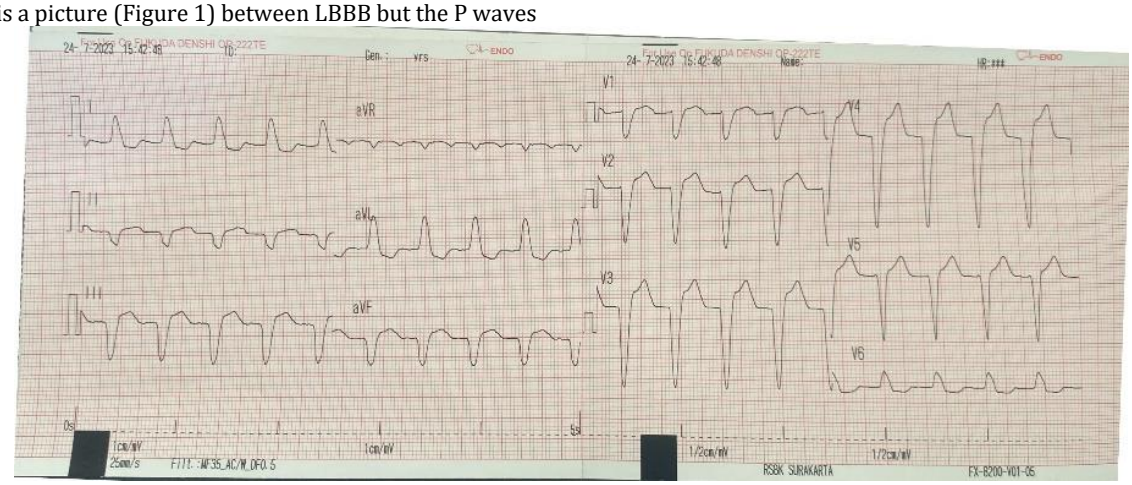

Figure 1. ECG on 82 years olds with first degree AV block with LBBB, ST elevation in the precordial leads, with a differential diagnosis of VT.

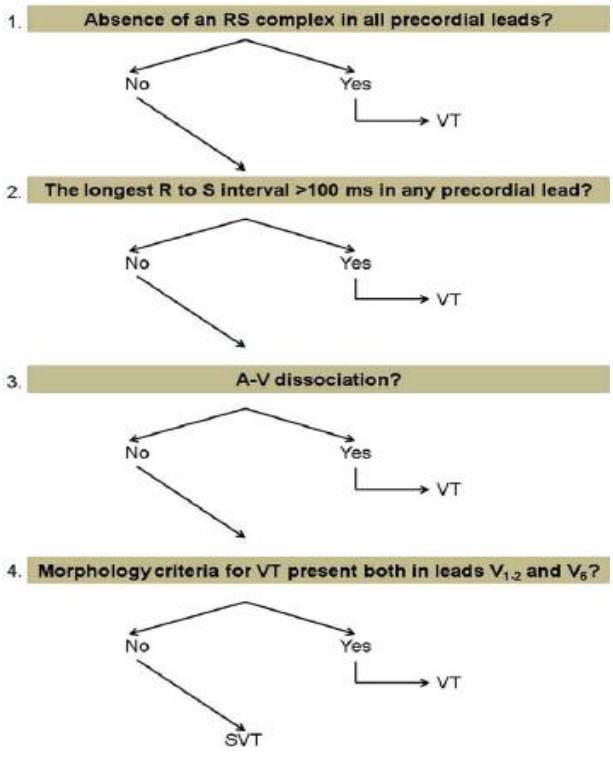

Figure 2. Brugada Algorithm[12]

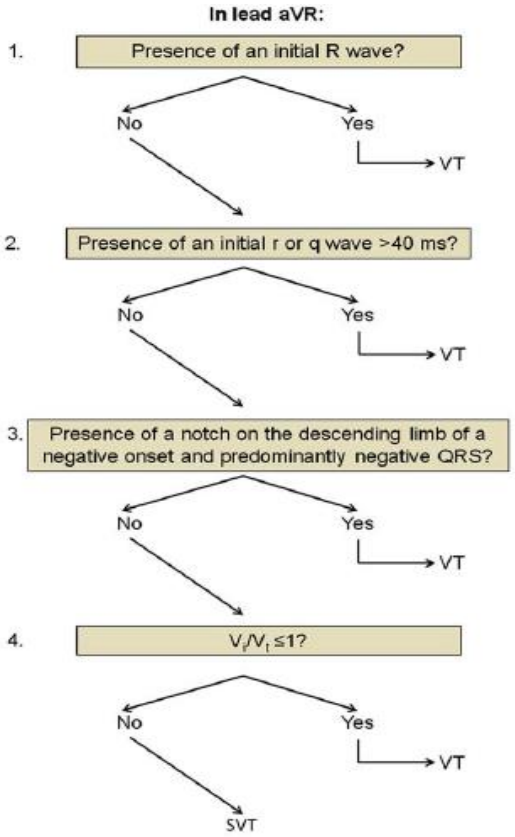

Figure 3. aVR Vereckei Algorithm7

DISCUSSION

A relatively common frustrating and anxiety-provoking situation in medical practice is the confrontation of a physician with a wide QRS complex tachycardia (WCT) ECG tracing The elucidation of the mechanism of WCT isvital not only for acute arrhythmia management, but also for the further work-up, prognosis and chronic management[7]. The differential diagnosis typically involves distinguishin between ventricular tachycardia (VT) and supraventricular tachycardia (SVT) with aberrant conduction. Of the two, VT posesa greater risk to patients. While electrophysiology study (EPS) can facilitate the diagnosisof VT, accurately distinguishing VT from WCT in acute care settings is challenging[8]. There are ECG features that are suggestive of VT as opposed to SVT, with some mechanism of QRS broadening. Several algorithms have been proposed to systematically distinguish between VT and SVT[7]. which include similarity of QRS morphology to a typical pattern of aberrant conduction: if the QRS complex has a 'typical' bundle branch or fascicular block appearance, there is a higher likelihood that the arrhythmia is an aberrantly conducted SVT. If the appearance is not consistent with any combination of bundle branch or fascicular blocks, then the diagnosis is most likely that of pre-excited SVT or VT[9].

Physical findings that indicate the presence of AV dissociation suggest VT with a very high likelihood[7]. There are several electrocardiographic and echocardiographic methods that may facilitate the detection of AV dissociation. The use of Lewis leads may improve the detection of P waves on the ECG. Lewis lead is a special bipolar chest lead with the right arm electrode applied to the right side of the sternum at the 2nd intercostal space and the left arm electrodeapplied to the right 4th intercostal space adjacent to the sternum. The recording of the tracing can be seen in lead I. Calibration should be adjusted to 1 mV=20 mm[10,11]. There is 2 methods yo distinguish VT and SVT, Brugada Algorithm dan Veckerei Algorithm.

In 1991 Brugada and coworkers[12] published a stepwise, decision-tree like algorithm in which 4 criteria for VT are sequentially considered (see Fig. 2). The first two criteria in their 4-step algorithm were new, in the 3rd and 4th step the algorithm used the old traditional criteria of AV dissociation and morphological criteria in leads V1-2 and V6. The Brugada algorithm is the most widely known and commonly used algorithm. In the first step precordial leads are assesssed for the absence of an RS complex (only the presence or absence of an RS complex is valuable for the diagnosis, QR, QRS, QS, monophasic R or rSR complexes are not considered RS complexes), which would indicate VT with a specificity of 100% and a sensitivity of 21% for VT diagnosis. In the next step, when an RS complex is present in one or more precordial leads, the longest RS interval in any precordial lead is measured (between the onset of the R wave and the nadir of the S wave). If the longest RS interval >100 ms VT is diagnosed with a reported specificity of 98% and sensitivity of 66% for VT diagnosis. If the longest RS interval is <100 ms in the 3rd step when AV dissociation is present VT diagnosis can be made with 21% sensitivity and 100% specificity. Fourth, if the RS interval <100 ms and AV dissociation cannot be detected, the traditional QRS morphology criteria in leads V1-2 and V6 are considered. When QRS morphology criteria are present both in leads V1-2 and V6 VT is diagnosed, if either the V1-2 and V6 criteria are not consistent, or none are consistent with VT, SVT-A is diagnosed by exclusion.The sensitivity and specificity of the 4th step wasn't reported. The authors prospectively analyzed 554 WCTs and reported a very high sensitivity and specificity of the 4 consecutive steps of 98.7% and 96.5% respectively..

The aVR Vereckei Algorithm[7] (Figure 3) were analyzed in lead aVR: 1) The presence of an initial R wave? 2) Presence of an initial r or q wave of >40 ms width? 3) Notching on the descending limb of a negative onset, predominantly negative QRS complex? 4) vi/vt ratio? When any of the first three criteria of the algorithm was met, a diagnosis of VT was made and the analysis was stopped at that step. In the 4th step a vi/vt <1 diagnosed VT, if vi/vt was >1 a diagnosis of SVT was made.The aVR Vereckei algorithm is based solely on the principle of differences in the direction and velocity of the initial and terminal ventricular activation during WCT due to VT and SVT. Although the aVR Vereckei algorithm does not contain any fundamentally new criteria compared with the first Vereckei algorithm, it is based on three novel concepts: 1) Selection of lead aVR exclusively for the differential di agnosis of WCTs; 2) classification of VTs into two main groups: a) VTs arising from the inferior or apical region of the ventricles yielding an initial R wave in lead aVR, b) VTs arising from other regions and lacking an initial R wave in aVR, but with slowing of the initial part of the predominantly negative QRS complex (in contrast to SVTs that show more rapid initial QRS forces); and 3) elimination of the complex morphological criteria (and the AV dissociation criterion) used by all prior algorithms and traditional criteria.

The aVR Vereckei algorithm was tested in 483 WCT tracings recorded from 313 patients. The overall test accuracy of the aVR Vereckei algorithm was similar to that of the first Vereckei algorithm and superior to that of the Brugada algorithm (91.5% vs. 90.7% and 85.5% respectively). A limitation of the aVR Vereckei algorithm similar to that of the first Vereckei and Brugada algorithms was its inability to differentiate VTs from preexcited SVTs with the possible exception of the presence of an initial R wave criterion. In fact none of the 20 preexcited SVTs that were analyzed during the study had an initial R wave in lead aVR.

ECG-based algorithms exhibit high sensitivity and moderate specificity in the diagnosis of WCT. A combination of Brugada or Vereckei-aVR algorithm may be considered to diagnose WCT[8].

CONCLUSION

If unable to differentiate between VT and non-VT, treat as VT. VT/VF is more life-threatening than other types of arrhythmias

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ST-segment elevation following electrical injury: Is it Myocardial Infarction?

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Keywords: Electrical injuries, ECG, Myocardial infarction

ABSTRACT

Introduction: Electrical injuries can cause health problems and their manifestation may resemble STEMI, with different pathomechanisms. This report describes a patient who presented with ST-segment elevation following an electrical injury.

Case presentation: A 58-year-old male with no cardiac history was admitted to the emergency department with left-sided chest pain and breathlessness after an electric shock. The patient accidentally stepped on electrical cables while working 2 hours previously and experienced a transient loss of consciousness. ECG showed ST-elevation in lead V1-V4 and cardiac troponin was elevated. The carotid Doppler and hemodynamic profile measurements were normal. The clinical manifestation and ECG findings resembled MI significantly, but the symptoms and subsequent ECG showed marked improvement in some hours without revascularization. The patient was discharged after two days of hospitalization without any complications.

Discussion: Electrical injuries are relatively infrequent but can cause mild-to-severe health problems, include cardiac problems such as arrhythmia, myocardial injury, and even death. The exact cause and mechanism of myocardial injury due to electrical injury remain unclear. However, there is a theory regarding vasospasm of the coronary artery due to electrical injury that disturbs the oxygen supply, resulting in clinical features and ECG findings of MI. Although it has similar features, this problem is transient and resolved without revascularization. Similar to our patient, the symptoms and ECG findings showed remarkable improvement after symptomatic management.

Conclusion: Electrical injuries can have symptoms and ECG findings similar to MI but with different pathomechanisms and require different treatments. Therefore, careful history-taking is required for a correct diagnosis and appropriate treatment

INTISARI

Pendahuluan: Trauma listrik dapat menyebabkan berbagai masalah kesehatan dan manifestasinya dapat menyerupai STEMI namun dengan patomekanisme yang berbeda. Laporan ini mendeskripsikan pasien nyeri dada dan sesak nafas akibat trauma listrik

Presentasi Kasus: Seorang pria berusia 58 tahun datang ke IGD dengan nyeri dada kiri dan sesak nafas setelah tersetrum listrik. Pasien tidak sengaja menginjak kabel listrik saat bekerja 2 jam yang lalu dan sempat tidak sadarkan diri. Pada EKG terlihat ST elevasi pada lead V1-V4 dan nilai troponin meningkat. Pemeriksaan doppler karotis dan profil hemodinamik menunjukkan hasil normal. Manifestasi klinis dan temuan EKG ini menyerupai infark miokard namun gejala dan EKG berikutnya menunjukkan

perbaikan yang signifikan dalam beberapa jam tanpa revaskularisasi. Pasien diperbolehkan pulang setelah 2 hari perawatan tanpa ada komplikasi.

Diskusi: Trauma listrik relatif jarang terjadi namun dapat menyebabkan masalah kesehatan yang ringan hingga berat, termasuk masalah pada jantung seperti aritmia, cedera miokard, bahkan kematian. Penyebab dan mekanisme pasti dari cedera miokard akibat trauma listrik ini masih belum jelas. Namun ada teori tentang vasospasme pada arteri koroner akibat trauma listrik yang mengganggu suplai oksigen sehingga menimbulkan gambaran klinis dan EKG yang menyerupai infark miokard. Walaupun memiliki gambaran serupa, masalah ini bersifat sementara dan dapat pulih tanpa revaskularisasi. Serupa dengan pasien kami, gejala dan EKG menunjukkan perbaikan yang bermakna setelah terapi simptomatis.

Kesimpulan: Trauma listrik dapat memiliki gejala dan temuan EKG menyerupai infark miokard, namun dengan patomekanisme yang berbeda serta membutuhkan tatalaksana yang juga berbeda. Oleh karena itu, anamnesis yang tepat dibutuhkan untuk mendapatkan diagnosis yang tepat dan tatalaksana yang sesuai.

INTRODUCTION

Electrical injuries can occur anywhere and anytime, mostly in occupational settings. Although uncommon and the actual incidence is unknown, it can cause various health problems in the human body, from simple skin injury to fatal cardiac complications. Electrical trauma can cause skin burns, tissue necrosis, cardiac abnormalities, tetanic muscle contraction, paralysis of the respiratory muscle, central and peripheral nervous systems, and internal organ damage[1].

Because the heart is the most susceptible organ to electrical injuries, it may cause cardiac problems such as arrhythmia, conduction abnormalities, and myocardial damage. The types of arrhythmias due to electrical injury include atrial tachycardia, atrial fibrillation, premature ventricular contraction (PVC), ventricular fibrillation, ventricular tachycardia, and complete heart block[2,3,4,5,6]

Electrical injuries can cause various ECG findings, including ST elevation. The purpose of this study was to report and discuss a case of electrical injury that caused clinical manifestation and ECG appearance resembling ST-elevation myocardial infarction (STEMI).

CASE PRESENTATION

A 58-year-old male was admitted to the emergency department after an electric shock from a low-voltage source that had occurred 2 h previously. The patient accidentally stepped on electrical cables on his right sole for approximately 10 s while working. The patient complained of tightening left-sided chest pain radiating to the left shoulder immediately after the electrocution. The patient also experienced transient loss of consciousness for a short time. In addition, the patient experienced breathlessness, cold sweat, blurred vision, aching in the bones, and pain in the right sole. The patient denied any history of hypertension, diabetes mellitus, heart disease, or stroke. The patient did not take any routine medication.

When examined in the emergency room, the patient was fully conscious of the Glasgow Coma Scale (GCS) 15. Vital signs were as follows: blood pressure, 125/79 mmHg; heart rate, 93 beats/min; respiratory rate, 20 breaths/min; temperature, 37 °C; and oxygen saturation, 99% on room air. General physical examinations revealed no remarkable findings. Local examination of the right sole showed a well-defined burn lesion, approximately 0.7 cm in diameter, as an entry point, but no lesion was observed as an exit point. Electrocardiography (ECG) showed ST-elevation in lead V1-V4 (Fig 1). The cardiac troponin level was increased to 32 ng/L (normal range 0,00-0,30 ng/L). Another laboratory examination, carotid Doppler and hemodynamic profile measurements were normal. The patient was diagnosed with non-ST-elevation myocardial infarction (NSTEMI) and was initially managed with aspirin 160 mg, clopidogrel 300 mg, atorvastatin 80 mg, symptomatic therapy with oxygen at 2 L/min, ranitidine, and ketorolac injection. The clinical manifestation and ECG findings resembled MI significantly, but the symptoms and subsequent ECG showed marked improvement in some hours after initial management and symptomatic therapy without revascularization (Fig 2). The patient was eventually discharged after two days of hospitalization without any complications.

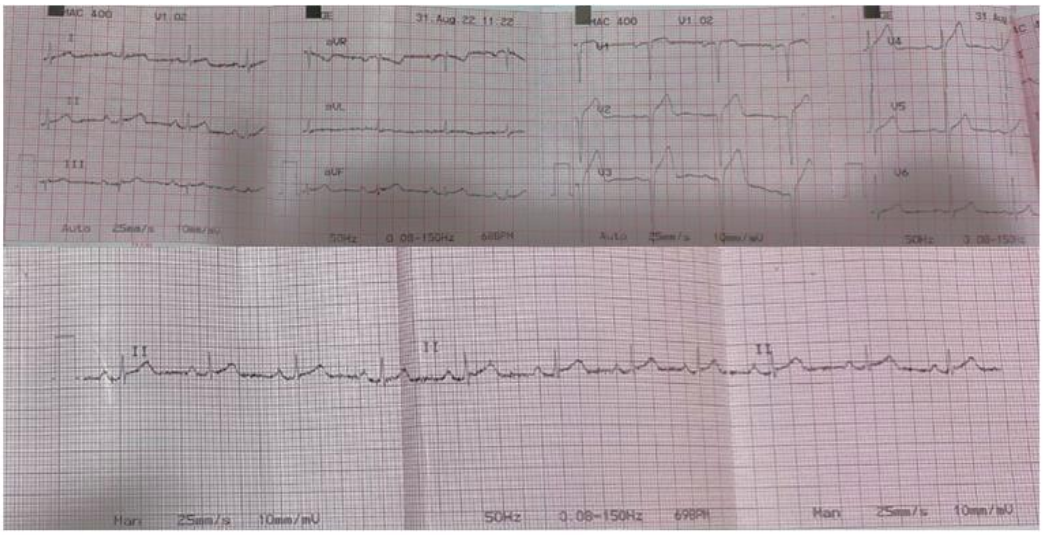

Fig 1. ECG at admission. ST elevation in lead V1-V4

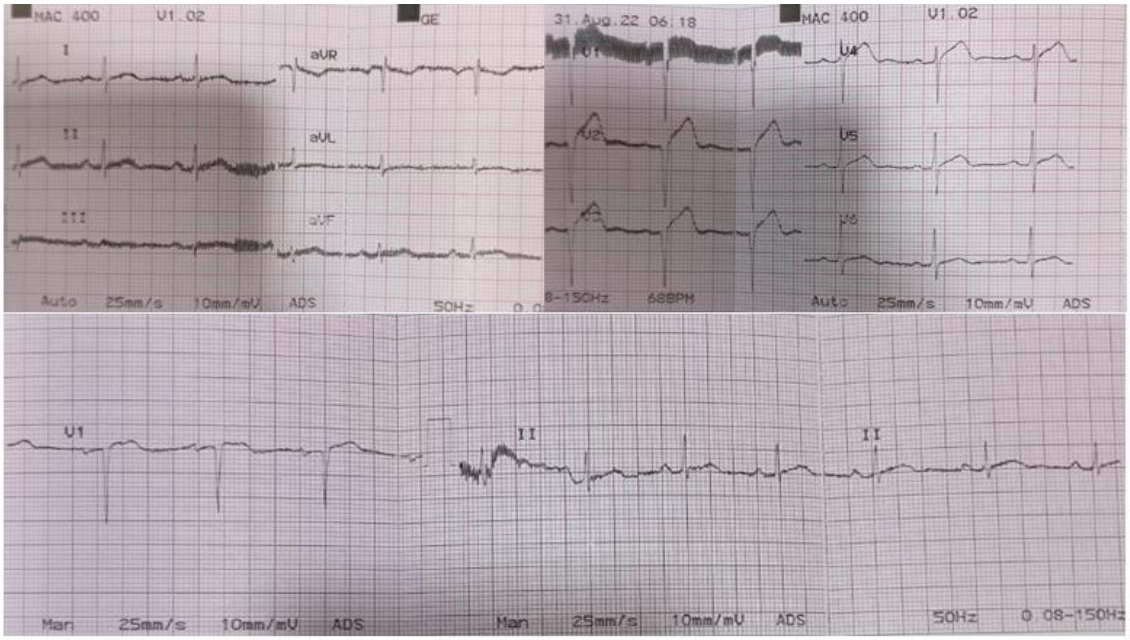

Fig 2. ECG 2 hours post initial management. Decrease ST elevation in lead V1-V4

DISCUSSION

Electrical injuries are relatively infrequent but can cause mild-to-severe health problems from mild skin burns to fatal arrhythmia. The heart is the organ most susceptible to electrical injuries and can cause cardiac problems such as arrhythmia, transient hypertension, myocardial injury, myocardial/ valvular rupture, structural changes in the coronary vessel, left ventricular dysfunction, pericardial effusion, and even death from cardiopulmonary arrest. Myocardial injury or infarction is the rare consequence of electrical injury, but it has potential and can be fatal[2,3,4,5]. Our patient reported transient loss of consciousness that was probably due to non-sustained ventricular arrhythmias that had disappeared at admission, similar to a case report by Rangaraj et al. that reported an electrocuted patient with transient brugada-type ECG pattern and brief syncopal attack[6].

Electrical injuries can cause direct damage by contact with electrical energy and indirect damage to secondary mechanical trauma due to falls, burns due to flash from the heat generated by an electrical arc, or flames from combustible materials. The severity of electrical trauma depends on the voltage, duration of contact with electric current, resistance to current flow, type of current, current

path through the body, and magnitude of energy transmitted. In some studies, abnormal ECG findings of the patient following electrical injuries were detected, ranging from a low percentage to a high percentage. The highest percentage emerged from the series of high-voltage injuries. The common ECG changes in electrical injuries are sinus tachycardia and non-specific ST segment changes, other findings including QT interval prolongation, bundle branch block, atrial and ventricular fibrillation, and atrial and ventricular premature contractions. Ventricular fibrillation is more frequent from low-voltage electrical current, whereas asystole is more common from high-voltage or direct current. Previous studies mentioned that ST elevation in the inferior is more frequent because the right coronary artery is closer to the chest wall and is more sensitive to electric current; however, in this case, the ST elevation after electric shock is on the anterior side[1,2,4,6].

There are five types of myocardial infarction: 1) spontaneous infarction related to atherothrombotic events, 2) MI secondary to increased oxygen demand or decreased oxygen supply, 3) sudden cardiac death likely due to ischemia without a biomarker, 4) MI associated with PCI/thrombosis stent, and 5) MI associated with cardiac surgery. The exact cause and mechanism of myocardial injury due to electrical injury remains unclear. However, its etiology is unrelated to coronary atherosclerosis, which can cause thrombosis, resulting in myocardial infarction. There is a theory regarding endothelial dysfunction that can cause vasospasm of the coronary artery due to electrical injury that disturbs the oxygen supply, resulting in clinical features and ECG findings of myocardial infarction. Besides that, other theories like direct thermal effect on the myocardium, ischemia secondary to arrhythmia-induced hypotension, destruction of coronary artery during cardiopulmonary resuscitation, and hypoxia due to cardiopulmonary arrest are thought to be etiology of electrocution-induced MI[2,4]

ECG changes following electrical injury are temporary and can be completely resolved in long-term survivors. Nonspecific ST-T wave abnormality after electric shock usually resolves spontaneously in a few hours. In this case, the clinical manifestations and ECG findings showed remarkable improvement after 2 h. In line with this report, a previous study by Al et al. reported that a patient with MI after electric shock had a normal coronary artery, and ECG findings were recovered after 6 hours[4]. A case report from Gursul also reported that the coronary artery of a patient with electrocution-induced MI was normal, and the ECG findings after electric shock were recovered within 100 minutes[2]. Yildiz et al also reported that patients with inferolateral MI after high voltage electric current had the ECG return to normal after 24 hours[3].

Since the possible cause of electrical injury-induced ST-segment elevation in a patient's ECG is considered to be vasospasm as a non-occlusive mechanism, not due to coronary atherosclerosis, fibrinolytic therapy should not be administered because it has no benefit in that case, and only increases the risk of side effects and medical cost[2,3]. Appropriate management of electrical injury is mostly symptomatic. The patient outcomes after electrical injury were mostly good. Ahmed et al. found that 79.4% of patients were discharged the same day, and there was no in-hospital or 30-day mortality and serious arrhythmia caused by electrical injuries[7]. However, in the case of electrical injury, cardiac monitoring and medical therapy are recommended for all patients with documented loss of consciousness, heart rhythm disorder, or ECG changes at admission, at least within 24 hours[5]. Our patient was observed in the ward for two days to follow up and look for signs of delayed complications because he experienced transient loss of consciousness and had abnormal ECG findings at admission. After 2 days of hospitalization, the patient was discharged in good health and had no complications.

CONCLUSION

Electrical injuries can have symptoms and ECG findings similar to those of STEMI but with different pathomechanisms and require different treatments. Unlike STEMI, which is due to coronary atherosclerosis, ST elevation following electrical injury is possibly due to coronary artery vasospasm. This condition is not required for fibrinolytic therapy because of its different etiologies. Therefore, in addition to cardiac monitoring for electrical injuries-induced cardiac problem, careful history taking is required for correct diagnosis and appropriate treatment to avoid overtreatment and over-medical costs.

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In-hospital Course of Acute Coronary Syndrome Patients: The Role of Active Lifestyle

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Keywords: acute coronary syndrome; inferior STEMI; AV block; active lifestyle

ABSTRACT

Introduction: Active lifestyle has been found to reduce cardiovascular events (CVE) during hospitalization and increase outcome and survival in acute coronary syndrome (ACS) patients. We report the case of a worker who presented with chest pain.

Case presentation: a 54-year-old physically active worker who was a former marathon athlete with a history of intermittent chest pain, presented to the emergency department with epigastric and substernal chest pain, vomiting, and diaphoresis. The patient has been a heavy smoker since he was a teenager. Vital signs showed bradycardia (49 bpm). ECG findings indicated sinus arrhythmia with first-degree AV block and inferior STEMI (Fig 1), and the patient was treated with thrombolytic therapy. Post-thrombolytics showed remarkable clinical and ECG improvement (Fig 2), and the patient was discharged after three days of hospitalization.

Discussion: Inferior STEMI can disturb the cardiac conduction system due to right coronary artery involvement. AV block, a complication of inferior MI, usually requires treatment according to the leading cause. Our patient returned to sinus rhythm immediately after thrombolytic therapy and recovered quickly. An active lifestyle provided by the patient contributed to better outcomes. Physically active patients had shorter lengths of stay and fewer adverse CVEs during hospitalization compared with physically inactive patients ($p<0,001$). Our patient had no CVE complications and required only three days of hospitalization

Conclusion: Active lifestyles and appropriate exercise training are protective factors that reduce the risk of STEMI and improve its prognosis. Nevertheless, cardiac screening, smoking cessation, and active lifestyle education are mandatory for ACS-risk patients and the general population.

INTISARI

Pendahuluan: Gaya hidup aktif diketahui mampu mengurangi angka kejadian kardiovaskular dan meningkatkan luaran serta kelangsungan hidup pada pasien sindrom koroner akut (SKA). Kami melaporkan kasus seorang pekerja dengan nyeri dada.

Presentasi Kasus: Seorang pria 54 tahun, pekerja aktif dan mantan atlet maraton datang ke IGD dengan keluhan nyeri dada dan ulu hati yang hilang timbul, muntah, serta keringat dingin. Pasien merupakan perokok aktif sejak remaja. Tanda vital menunjukkan bradikardia (49x/menit). Gambaran EKG menunjukkan sinus aritmia dengan AV blok derajat 1 dan STEMI inferior (Gambar 1), dan ditatalaksana dengan trombolitik. Pasca trombolitik terlihat perbaikan klinis dan EKG yang bermakna (Gambar 2), dan pasien diperbolehkan pulang setelah 3 hari perawatan.

Diskusi: STEMI inferior dapat mengganggu sistem konduksi jantung karena keterlibatan arteri koroner kanan. AV blok yang merupakan komplikasi dari STEMI inferior membutuhkan tatalaksana yang sesuai dengan penyebab utamanya. Pada pasien kami, irama jantung kembali ke irama sinus segera setelah dilakukan terapi trombolitik dan kondisinya membaik dengan cepat. Gaya hidup aktif yang selama ini dilakukan oleh pasien berkontribusi untuk prognosis yang lebih baik. Pasien yang aktif secara fisik memiliki waktu perawatan yang lebih singkat serta memiliki angka kejadian kardiovaskular yang lebih sedikit selama perawatan dibandingkan dengan pasien yang tidak aktif secara fisik (p<0,001). Pasien kami tidak memiliki komplikasi kardiovaskular selama perawatan dan hanya membutuhkan 3 hari perawatan.

Kesimpulan: Gaya hidup aktif dan latihan fisik yang sesuai merupakan faktor protektif yang mengurangi resiko terjadinya STEMI dan meningkatkan prognosisnya. Namun demikian, skrining jantung, penghentian merokok, dan edukasi gaya hidup aktif sangat diperlukan untuk pasien yang beresiko SKA maupun populasi umum.

INTRODUCTION

Heart disease remains the primary cause of death worldwide. World Health Organization said more than 17 million people died from heart and vascular disease. In Europe, ischemic heart disease causes 1,8 million people deaths per year (20% of all deaths). In Indonesia, based on Riset Kesehatan Dasar (Riskesdas), 15 out of 1000 people (2.784.064 people) had heart disease, and the prevalence increased from 0,5% in 2013 to 1.5% in 2018. Based on updated data, heart disease, especially acute coronary syndrome, is not only found in the elderly but also in adults <40 years of age, and its prevalence has been increasing over the years[1,2].

Unhealthy lifestyle which is increasingly being practiced day by day such as smoking, alcohol consumption, and sedentary lifestyle, and uncontrolled several modifiable cardiovascular risk factors such as hypertension, type 2 diabetes mellitus, obesity, and dyslipidemia contribute to increased risk of acute coronary syndrome[3].

Physical activity is known to be a protective factor of myocardial infarction. In contrast, some studies mention that high physical activity increases the risk of myocardial infarction and sudden cardiac death, especially for susceptible people[4]. Furthermore, we need to evaluate the role of physical activity as a protective factor of myocardial infarction and its contribution to a better prognosis. The purpose of this study was to report and discuss a case of inferior STEMI patient with first-degree AV block who lead an active lifestyle and its correlation with better outcomes in this patient especially during hospitalization

CASE PRESENTATION

A 54-year-old man presented to the emergency department with recurrent epigastric and substernal chest pain 2 days prior. The chest pain was dull, and the patient could not point to a specific location. The patient complained of nausea, vomiting, and cold sweats. Four weeks previously, he had experienced similar complaints but was reduced with rest and painkiller drugs. He denied a history of hypertension, diabetes mellitus, heart disease, or stroke. He also did not take any routine drugs. The patient was a physically active worker. He was also admitted to exercising frequently and was a former marathon athlete when teenager. The patient had been a heavy smoker since he was a teenager.

Vital signs were as follows: blood pressure, 116/70 mmHg; heart rate, 49 beats/min; respiratory rate, 20 breaths/min; oxygen saturation, 99% on room air; and temperature 36.5 C. Physical examinations revealed no remarkable findings. ECG showed sinus arrhythmia with first-degree AV block, ST-elevation in lead II, III, and aVF, and reciprocal change in lead I and aVL (Fig 1). The patient was diagnosed with inferior STEMI and first-degree AV block, and was given initial management with aspilet 160 mg, clopidogrel 300 mg, atorvastatin 80 mg, and revascularization with thrombolytic therapy. Post-thrombolytic therapy showed remarkable clinical and ECG improvement (Fig 2), and the patient was discharged after three days of hospitalization.

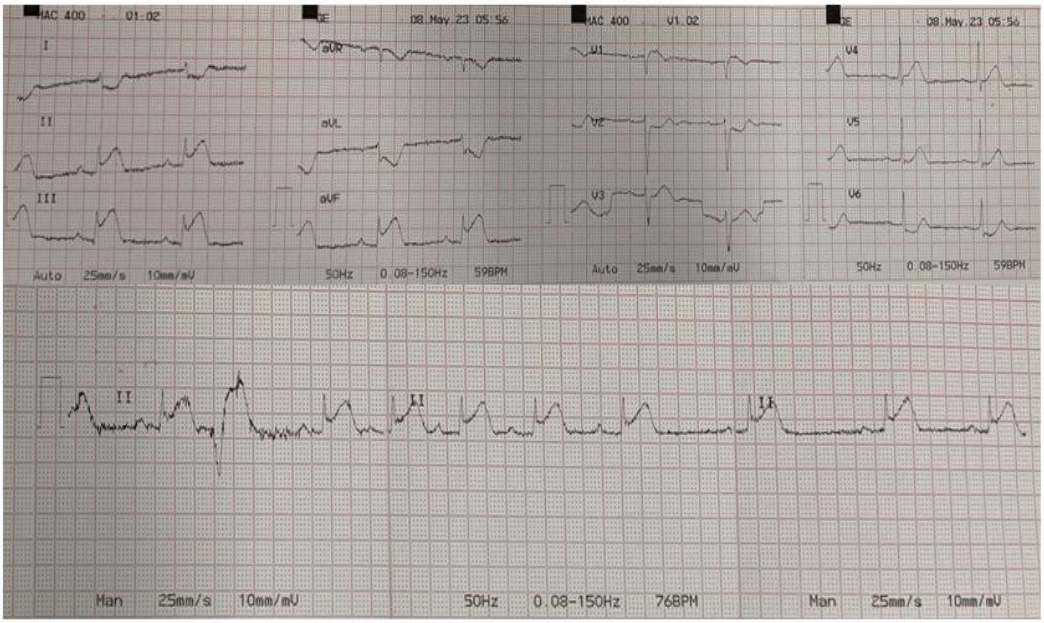

Fig 1. ECG at admission. Inferior STEMI and first degree AV block

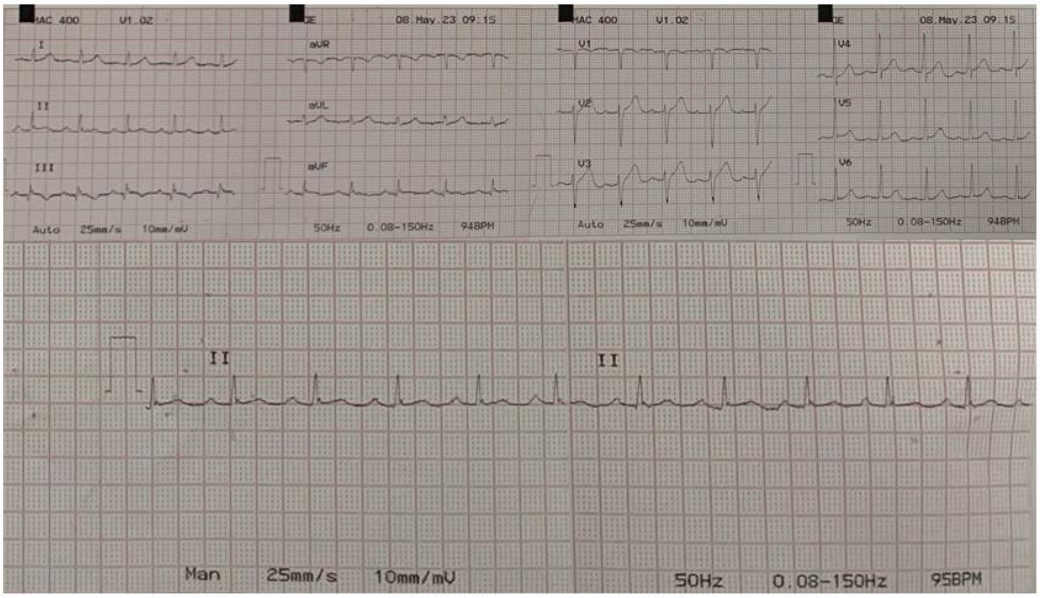

Fig 2. ECG post-thrombolytic therapy. Return to normal sinus rhytm

DISCUSSION

The incidence of STEMI has been increasing over the years. STEMI can be fatal and has complications that are associated with patient morbidity and mortality. Common complications of STEMI include hemodynamic disturbance (hypotension, heart failure, pulmonary edema, cardiogenic shock), arrhythmia (AV block, supraventricular tachycardia, ventricular tachycardia, and sinus bradycardia), mechanical complications (free wall rupture, ventricular septal rupture, and papillary muscle rupture), and pericarditis. In inferior STEMI, AV block is a common complication (reaching up to 20% of the cases) because it can disturb the cardiac conduction system, including the SA and AV nodes, due to right coronary artery involvement. Our patient was diagnosed with inferior STEMI and had a complication of first-degree AV block. Treating AV block as a complication of inferior MI usually does not require antiarrhythmic drugs, only requires treatment according to the leading cause, namely revascularization, and it can resolve spontaneously. Our patient returned to sinus

rhythm immediately after thrombolytic therapy without antiarrhythmic drugs and recovered quickly[2].

Risk factors for myocardial infarction are correlated with physical activity, smoking, alcohol consumption, dyslipidemia, diabetes mellitus, hypertension, obesity, stress, age, and sex. Almost all of the risk factors associated with atherosclerotic plaque formation, which is known to play an important role in occlusion pathogenesis, cause myocardial infarction[3]. The level of physical activity has beneficial effects on a number of CVD risk factors. Physical activity can reduce blood pressure, improve glucose tolerance, increase insulin sensitivity, reduce blood coagulability, improve artery function, and reduce atherogenic markers. Besides that, physical activity can reduce about 59-62% the risk of recurrent coronary events and mortality. In a recent cohort study, Lear et al. reported individuals with high physical activity have a 29% lower risk of MI. Peytz et al in their study reported there is an association between regular physical activity with 45% lower case fatality in MI[5,6,7].

Some studies mention that exercise can both prevent and cause acute myocardial infarction and sudden cardiac. Prospective studies have shown an association between increasing levels of regular exercise and decreasing cardiovascular event rates. Moderate exercise training has been associated with an increase in heart rate variability and baroreflex sensitivity, markers of vagal activity as an anti-fibrillatory effect and responsiveness, reduction in the incidence of obesity and diabetes, and improved lipid and blood pressure profiles, as well as reduction of acute coronary events, reduce the number of cardiac death and increased survival[8]. In a study conducted by Gorczyca et al with postmenopausal MI sufferers, it was reported that there was a reduced risk of mortality rate in patients who increased physical activity after myocardial infarction compared to patients with low physical activity[9]. Exercise training in patients with ACS can improve myocardial perfusion and reduce atherosclerosis progression, which can reduce cardiac events after acute myocardial infarction and improve the clinical outcome. Besides that, physical activity can reduce oxidative stress which contributes to the more stable plaque and cell membrane resulting in decreased frequency of arrhythmia[10].

An active lifestyle provided by the patient contributed to better outcomes. Our patient had no CVE complications during hospitalization and required only three days of hospitalization. In line with this report, a study by Jorge et al. reported an increasing trend in the frequency of CVE complications during hospitalization with decreased physical activity levels. In the active group, only 20% of patients had complications, while in the non-active group, 45% of patients had complications during hospitalization ($p<0.001$, OR 2.54). Physically active patients had shorter median lengths of stay, 6 days in the more active groups compared with 8 days in the less active groups ($p=0.011$, OR 1.15), and had fewer adverse CVEs during hospitalization, such as shock, atrial fibrillation, and acute pulmonary edema, compared with physically inactive patients ($p<0.001$). This shows that there is a strong association between being physically inactive and the frequency of CVE complications during hospitalization[10].

On the other hand, excessive physical exertion may trigger infarction and cause sudden cardiac death in several ways: 1)hemodynamic stress which can trigger the disruption of a vulnerable atherosclerotic plaque, 2) the presence of endothelial dysfunction causes vasoconstriction and may lead to increased shear forces and platelet deposition, and 3)induce a prothrombotic state[4,7,9]. Therefore, active lifestyle education and appropriate exercise training are very important for patients with cardiovascular risk factors and for the general population.

CONCLUSION

Active lifestyle and routine exercise training, apart from reducing the risk of ACS, are also associated with a better prognosis because they can reduce cardiovascular events (CVE) and increase outcomes and survival in patients with ACS. However, excessive physical activity can trigger myocardial infarction and sudden cardiac death in susceptible individuals. Therefore, in addition to cardiac screening and smoking cessation, active lifestyle education and appropriate exercise training are mandatory not only for patients with cardiovascular risk factors, but also for the general population.

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We are very grateful to the doctor in charge and all the staff at Bakti Timah Hospital, Pangkalpinang City, Bangka Belitung Province, Indonesia who have best cared for the patient.

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Symptomatic Total Atrioventricular Block in Teenager: A Rare Case

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Keywords: total atrioventricular block; teenager; symptomatic bradycardia; third-degree AV block

ABSTRACT

Introduction: Total atrioventricular block (TAVB) is a condition of severe bradycardia. It occurs due to absence of atrioventricular conduction. This condition often occurs in adults. However, cardiac conduction disorders are rare in children.

Case Presentation: A 14-year-old girl came to emergency department with syncope. She had history of chest pain on the same day. Chest pain occurred after activity and decrease with rest. She had no complaints of fever, nausea, vomiting, sweating or shortness of breath. However, she had dizziness since 4 days. There was no previous history of heart disease, including congenital heart disease. The clinical examination showed bradycardia (30 bpm) and other vital signs was normal. Heart sound was normal. The electrocardiogram showed complete AV dissociation with independent atrial and ventricular rates that indicates a third degree AV block. Laboratory findings such as routine blood tests, glucose, electrolytes, and cardiac enzymes were normal. Her chest x-ray showed no abnormal result. Atropine administration did not response. Furthermore, patient was referred immediately for cardiac pacing to prevent sudden death.

Discussion: Our patient had total atrioventricular block with symptoms and extreme low heart rates. From electrocardiogram, the supraventricular impulses is not transmitted to the ventricles. Perfusion is maintained by ineffective ventricular escape rhythm. This causes the patient to experience syncope. If prolonged, sudden cardiac death may occur. This condition is an indication for permanent pacing according to the American College of Cardiology (ACC)/American Heart Association (AHA) guidelines. Atrioventricular block in children may be caused by congenital or acquired. In our patient, we have not been able to determine the cause from brief examination, so we need further examination to determine the etiology.

Conclusion: Total atrioventricular block may occur in adolescent and can be symptomatic. Early diagnosis and appropriate management are critical. It requires urgent admission for cardiac monitoring and insertion of a cardiac pacing to prevent sudden cardiac death.

INTISARI

Pendahuluan: Blok atrioventrikular total adalah suatu kondisi bradikardia berat yang dapat terjadi karena ketiadaan konduksi atrioventrikular. Kondisi ini sering terjadi pada orang dewasa. Namun, gangguan konduksi jantung ini jarang terjadi pada anak-anak.

Laporan Kasus: Seorang anak perempuan berusia 14 tahun datang ke IGD dengan keluhan pingsan. Dia memiliki keluhan nyeri dada pada hari yang sama. Nyeri dada terjadi setelah aktivitas dan berkurang saat istirahat. Tidak ada keluhan demam, mual, muntah, keringat dingin, atau sesak. Namun, dia mengeluh pusing sejak 4 hari. Tidak ada riwayat sakit jantung sebelumnya, termasuk penyakit jantung kongenital. Pada pemeriksaan fisik didapatkan

bradikardi (30 kali per menit) dan tanda vital lain normal. Bunyi jantung normal. Pada elektrokardiogram terlihat disosiasi antrioventrikular dimana

kecepatan atrium dan ventrikel tidak berhubungan yang mengindikasikan suatu atrioventrikular blok derajat III. Pemeriksaan laboratorium seperti darah rutin, gula darah sewaktu, elektrolit, dan enzim jantung normal. Pemeriksaan rontgen dada tidak terdapat kelainan. Pemberian sulfas atropin tidak memberikan respon. Selanjutnya, pasien dirujuk segera untuk pemasangan pacu jantung untuk mencegah kematian mendadak.

Diskusi: Pasien kami mengalami blok atrioventrikular total yang disertai gejala dan detak jantung yang sangat rendah. Dari elektrokardiogram terlihat bahwa impuls supraventrikular tidak disalurkan ke ventrikel. Perfusi dipertahankan oleh ritme dari ventrikel yang tidak efektif. Hal ini yang menyebabkan pasien mengalami pingsan. Jika berlanjut dapat terjadi kematian jantung mendadak. Kondisi ini merupakan salah satu indikasi untuk dilakukan pemasangan pacu jantung permanen sesuai pedoman dari ACC/AHA. Blok atrioventrikular pada anak dapat disebabkan oleh kelainan bawaan atau didapat. Pada pasien kami, kami belum dapat menentukan penyebabnya dari pemeriksaan singkat sehingga perlu pemeriksaan lebih lanjut untuk mengetahui penyebabnya.

Kesimpulan: Blok atrioventrikular total dapat terjadi pada remaja dan dapat bergejala. Diagnosis dini dan manajemen yang tepat sangatlah penting. Pada kondisi ini memerlukan monitoring jantung ketat dan pemasangan alat pacu jantung segera untuk mencegah kematian jantung mendadak.

INTRODUCTION

Atrioventricular (AV) block is characterized by delay or interuption in the conduction of electrical signals from the atria to the ventricles. It is often caused by either anatomical or functional abnormality within the conduction system. This disruption in conduction can be temporary or permanent.[1] AV block classificate to several types, such as first-degree AV block, second-degree AV block, and third-degree AV block. First-degree AV block define as atrioventricular delay because no P wave are block and showed with a prolonged PR interval on the ECG. Second-degree AV block begins to show asynchronous of atrioventricular conduction that is recognized on the ECG with P waves that are not always followed by QRS complex. Second-degree AV block divided into two, mobitz 1 second-degree AV block and mobitz 2 second-degree AV block. Third-degree AV block which is often known as total AV block (TAVB) or complete AV block is appearing as independent contractions of the atria and ventricels in the ECG. TAVB is a condition of severe bradycardia. It occurs due to absence of atrioventricular conduction. This condition often occurs in adults. However, cardiac conduction disorders are rare in children.[2]

Atrioventricular block in children is a rare condition. Although TAVB is severest, most of cases are asymptomatic in children. From the ECG screening of school aged children (elementary and junior high school) in Japan, it was discovered that the prevalence of a third degree AV block was 2 per 100.000 children.[3] It may have been overlooked by healthcare worker because the prevalence is extremely rare or children can not communicate the symptoms that they feel. The disease is often unidentified. Sometimes, it is accidentally discovered during screening or during an examination due to another disease. Only a few patients have bradycardia-related symptoms presented as heart failure, syncope, or fatique.[4,5]

The aim from this study is to describe one of cardiac conduction disorders in children accompanied by symptoms.

CASE PRESENTATION

A 14-year-old girl patient came to emergency room due to syncope at her house 90 minutes before. She had history of chest pain on the same day, mild chest pain without radiating. Chest pain occurred after activity and decrease with rest. This condition is the first time experience for her. She had no complaints of fever, nausea, vomiting, sweating or shortness of breath. However, she had dizziness since 4 days. There was no trauma incident in this patient. There was no previous history of heart disease, including congenital heart disease. She also does not have family history with heart disease or sudden cardiac death. Her mother had no history of autoimune disease including systemic lupus erythematosus.

The patient was fully alert during examination. Her vital signs revealed a blood pressure of 110/80 mm Hg, heart rate of 30 beats per minute (bpm), temperature of 36oC, respiratory rate of 18 breaths per minute, and oxygen saturation by pulse oximetry of 99% with room air. The findings from her physical examination showed no notable abnormalities. Heart sound was normal, no murmur or gallop. Peripheral pulses was synchronous, no jugular vein distention, or no pheriperal edema. Other physical examinations including head, neurological, pulmonary, abdominal, and extremities was normal.

The electrocardiogram recorded complete AV dissociation with independent atrial and ventricular rates that indicates

a third degree AV block as shown in figure 1. Atrial rate was ~120 bpm and ventricular rate was ~30 bpm.

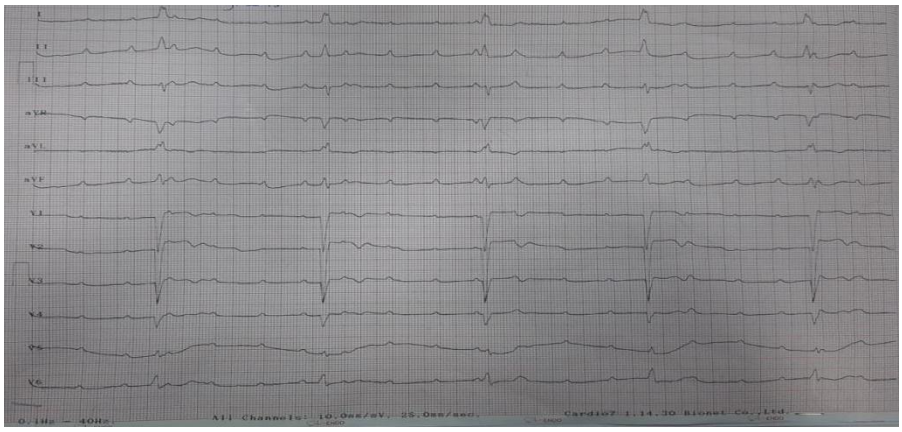

Figure 1. ECG of the patient with Total AV Block

On routine blood tests, there was only a slight increase in leukocytes level. Laboratory findings such as complete blood cell count, blood glucose level, electrolytes, and cardiac enzymes were normal as shown in table 1. Her chest x-ray showed no abnormal result (figure 2).

Table 1. Laboratory results of the patient

Laboratory Tests	Results	Note	Reference Range
Haemoglobin	12.3		11.5 – 15.5 gr/dl
Hematocrit	36.3		33.0 – 45.0 %
Thrombocyte	321		150 – 450 $10^3/\mu L$
Leucocyte	11.89	H	4.5 – 11.0 $10^3/\mu L$
Blood cell count			
Neutrophils	60.0		50 – 70 %
Lymphocytes	32.1		20 – 40 %
Monocytes	6.0		0 – 6 %
Eosinophils	1.7		0 – 4 %
Basophils	0.2		0 – 1 %
Blood glucose	72		70 – 140 mg/dL
Electrolytes			
Sodium	141.1		132 – 145 mmol/L
Potassium	3.65		3.1 – 5.1 mmol/L
Chloride	104.1		96.0 – 111.0 mmol/L
Troponin I	8.6		<2 ng/L: non AMI 2-100: Observation zone ≥100:AMI

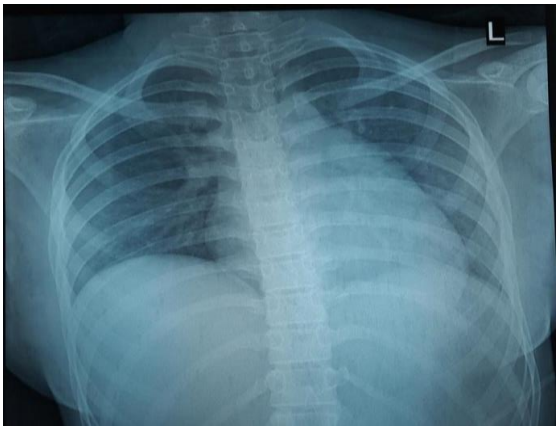

Figure 2. Chest X-ray

The patient was diagnosed with total AV block and the cause is not yet known. Atropine administration did not response. Furthermore, patient was referred immediately for cardiac pacing to prevent sudden death.

DISCUSSION

Atrioventricular block is classified according to the time of diagnosis. AV block can be diagnosed in utero, at birth, or during childhood. That is defined as congenital AV block when it was diagnosed in utero, at birth, or during neonatal period. Whereas, it was diagnosed in the first month until 18th year of life, it is defined as childhood AV block. Congenital or childhood AV block can be caused by several etiologies, either in patient with entirely normal heart structure or in patient with congenital heart disease.[6]

AV block in children is a rare condition and generally associate with congenital AV block. The prevalence of congenital heart block is only 1 every 15.000 to 20.000 live births. The most common cause of this congenital AV block is autoimune or fetal-maternal antibody associated AV block that diagnosed in the first half year of life. This condition may or may not be associated with maternal autoimune diseases such as Sjogren's syndrome or Systemic Lupus Erythematosus (SLE).[5,6,7] Transplacental

passage of maternal anti-Ro/SSA and/or anti-La/SSB auto-antibody enter to fetal circulation and trigger an inflammatory response, caused irreversible fibrosis of the cardiac conduction system, and downregulate cardiac L-type Calcium channels.5,6 Approximately 1-2% of mothers carrying anti-Ro/SSA antibodies experience the birth of children with TAVB, and there is a 12-20% chance of recurrence in their next pregnancies.[7,8] Congenital AV block Congenital AV block has a bad prognosis with a high neonatal mortality because the risk of dilated cardiomyopathy which is sometimes diagnosed late. Early diagnosis is important in this case.[5,7,9]

In a rare condition, AV block can be caused by unknown mechanism during childhood without maternal antibodies, structural heart disease, or other causes such as infection, metabolic abnormalities, or trauma. However, research about etiology and pathophysiology of this idiopathic AV block is still limited.[4,5,6]Dilated cardiomyopathy did not occur in cases unrelated to antibodies. Complication related to pacemaker is few. This condition has a better prognosis than congenital AV block if the indications for cardiac pacing are respected.[9]

AV block can cause sudden cardiac death if cardiac pacing is not given, especially in symptomatic patients. In some cases, cardiac pacing has indicated for prophylactic in patient without symptoms.[6]

Our patient had total atrioventricular block with syncope, chest pain, and dizziness as symptoms. She also has extreme low heart rates. In the electrocardiogram, the conduction from atrial to the ventricles did not found. Perfussion is maintained by ineffective ventricular escape rhythm. This causes the patient to experience syncope. If prolonged, sudden cardiac death may occur. This condition is an indication for permanent pacing according to the American College of Cardiology/American Heart Association guidelines. Atrioventricular block in children may be caused by congenital or acquired. Although, idiopathic AV block in childhood have a better outcome than congenital AV block, cardiac pacing is needed for symptomatic patient.

In our patient, we have not been able to determine the cause from brief examination. Patient did not have history of congenital heart disease, also from her family. There is also no autoimmune disease from her mother. Evidence of infections is unclear. She had never experienced trauma before. Metabolic abnormalities were also not detected. Further examination can be carried out to determine the etiology, such as echocardiogram or other laboratorium examinations. An echocardiogram is used to evaluate the present or absent of structural heart disease. Other laboratorium examinations such as Anti Nuclear Antibody (ANA) test, High sensitivity C-Reactive Protein (Hs-CRP), Thyroid Stimulating Hormone (TSH), and Free Thyroxine (FT4) is also needed. Because lack of facilities, instead of looking for the etiology, it is more important to immediately refer patients for insertion of a cardiac pacing.

CONCLUSION

Total atrioventricular block may occur in childhood. Although rare, some of the cases are symptomatic. In this case, patient have symptomatic TAVB, so early diagnosis and appropriate management are critical. It requires urgent admission for cardiac monitoring and insertion of a cardiac pacing to prevent sudden cardiac death.

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Atrial Septal Defect With Paroxysmal Atrial Tachyarrhytmia in Middle Age Soldier Patient : A Case Report

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ABSTRACT

Atrial septal defects (ASDs) are frequently asymptomatic and can remain undiagnosed until adulthood. Atrial tachyarrhythmias are not uncommon seen in patients with ASDs. Atrial fibrillation and atrial flutter are relatively rare in childhood, but become more prevalent with increasing age at time of repair or closure. The present case was an active duty 50 year old male soldier, referred to the arrhythmia division of Gatot Soebroto Army Hospital with palpitations and physical intolerance. Holter examination and electrophysiology study revealed atrial tachyarrhythmias. Transesophageal echocardiography was performed before radiofrequency catheter ablation, and unexpectedly found left to right shunt ostium secundum ASD. Right heart catheterization confirmed left to right shunt ASD with high flow-low resistance. He then underwent paroxysmal atrial tachyarrhythmias catheter ablation, followed by percutaneous transcatheter ASD closure using occluder device without fluoroscopy within six months. Both the procedures went well without any complications. His symptoms had improved during follow up, although he had episode of rapid paroxysmal atrial fibrillation on holter evaluation six months later. We conclude that ASD closure is still recommendable even in late middle age patients combined with arrhythmias management.

INTISARI

Atrial septal defek (ASD) seringkali tidak memiliki gejala, dan bisa tidak terdiagnosa sampai masa dewasa. Takiaritmia atrial banyak ditemukan pada pasien ASD. Atrial fibrilasi dan atrial flutter jarang terjadi pada masa kanak-kanak, namun akan lebih banyak terjadi seiring pertambahan usia saat dilakukannya operasi atau penutupan defek. Kasus berikut merupakan tentara aktif berusia 50 tahun, dirujuk ke divisi aritmia Rumah Sakit Pusat Angkatan Darat Gatot Soebroto dengan keluhan palpitasi dan mudah lelah. Melalui pemeriksaan holter dan *electrophysiology study* ditemukan adanya takiaritmia atrial. Pemeriksaan ekhokardiografi transeofagus dilakukan sebelum rencana ablasi kateter radiofrekuensi, dan secara tidak terduga ditemukan ASD ostium sekundum dengan pirau dari kiri ke kanan. Hal ini dikonfirmasi melalui kateterisasi jantung kanan yang memperlihatkan ASD pirau dari kiri ke kanan dengan *high flow-low resistance*. Pasien kemudian menjalani ablasi kateter atas indikasi takiaritmia atrial paroksismal, dan enam bulan kemudian dilanjutkan penutupan ASD secara transkateter perkutan dengan alat *occluder* tanpa menggunakan fluoroskopi. Kedua prosedur berlangsung lancar tanpa komplikasi. Keluhan pasien membaik pada saat kontrol, meskipun didapatkan fibrilasi atrial paroksismal pada evaluasi holter 6 bulan kemudian. Dapat disimpulkan bahwa penutupan ASD masih direkomendasikan meski pada pasien paruh baya dikombinasikan dengan manajemen aritmia.

INTRODUCTION

Atrial septal defect (ASD) represents a direct communication between right atrial (RA) and left atrial (LA) has a unique slow clinical progression. Ostium secundum type ASD (ASD II) as characterized by a communication at the level of fossa ovalis is the most frequent type, representing 80% of ASDs diagnosed.[1] Isolated ASDs represent about 7% of all cardiac anomalies and can be diagnosed at any age.[2] Patients may be asymptomatic into their fourth and fifth decade[3], and sometimes found incidentally on imaging studies. For this reason, many individuals can be undiagnosed early in life and will be able to serve in the military.

However, majority of the ASDs patient will develop symptoms including reduced functional capacity, exertional shortness of breath, and palpitations (supraventricular tachyarrhythmias), and less frequently pulmonary infections and right heart failure.[4] One of the major sources of morbidity are atrial tachyarrhythmias (ATs). ATs define as atrial fibrillation (AF), atrial flutter (AFL) and supraventricular tachycardias (SVTs). In patients above the age of 40 with unrepaired ASDs, the rate of ATs is even higher, with one study reporting the prevalence as high as 19%[5], which itself may be an underestimation.

Percutaneous closure has lately become the primary treatment option for ASD II, and according to European Society of Cardiology (ESC) guidelines, should be the therapy of choice when anatomical conditions are favorable.[1] The association between percutaneous ASD closure and atrial arrhythmias is controversial. On the one hand, reverse atrial remodeling after closure might lead to a decreased chance of supraventricular arrhythmias.[6] On the other hand, the presence of a closure device has a possible pro-arrhythmogenic effect.[7]

CASE PRESENTATION

A 50 year old male presented to the arrhythmia division of Gatot Soebroto Army Hospital for evaluation. An active duty soldier, he had noted episodic palpitations and a gradual decrease in exercise tolerance in one month. There were no chest tightness or respiratory symptoms. Physical examination revealed regular pulse 70 beats/min, blood pressure 120/70 mmHg, There was fixed splitting second heart sound, without any audible heart murmur. Patient's resting electrocardiograph (ECG) and chest X-ray posteroanterior view are shown in figures 1 and 2 respectively. Initial transthoracic echocardiography (TTE) was unremarkable. Laboratory examination was within normal limits including thyroid function. He underwent a holter examination, followed by electrophysiology (EP) study. The result was paroxysmal narrow complex tachycardia with long accessory pathway et causa atrial tachyarrhythmia (AT), abnormal sinoatrial (SA) node intrinsic function, and normal atrioventricular (AV) conduction. The patient was taken for diagnostic cardiac catheterization, coronary angiogram showed non obstructive coronary artery disease. Transesophageal echocardiography (TEE) was performed prior to the radiofrequency catheter ablation as the next intended procedure. Unexpectedly it revealed 13 mm ASD II left to right shunt (figure 3) with adequate rims size; RA and LA dilatation. Right heart catheterization (RHC) showed an oxygen step-up at the atrial level. The calculated flow ratio (FR) was 1.6, pulmonary arteriolar resistance index (PARi) was 1.8 WU, and pulmonary vascular resistance to systemic vascular resistance ratio (PVR/SVR) was 0.04.

Patient was diagnosed an ASD II left to right shunt, high flow-low resistance and paroxysmal AT. First, he had ectopic atrial arrhythmia ablation procedure. Six months later percutaneous transcatheter ASD closure using occluder device was performed without fluoroscopy. The procedures went well without any complications. According to patient, his symptoms had improved, and he could went back to work. He had holter monitor evaluation six months after the ASD closure, it showed episode of rapid paroxysmal AF. Follow up TTE examination one month and one year after the procedure showed normal heart chambers dimension without residual ASD.

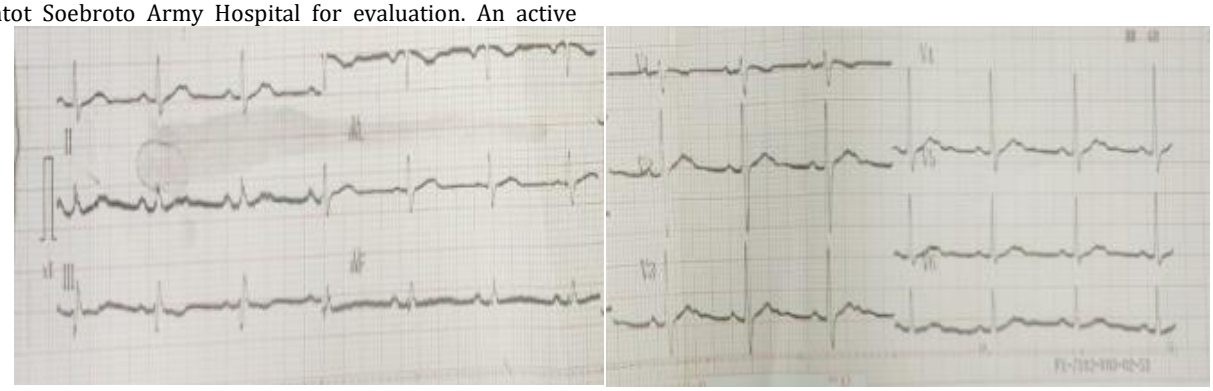

Figure 1.Normal resting ECG

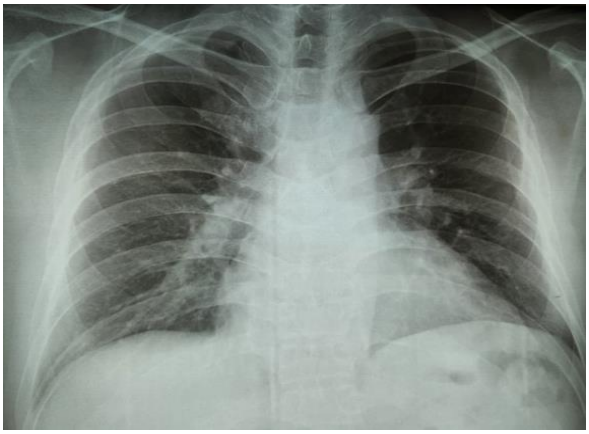

Figure 2. Chest X-ray P/A view showing mild cardiomegaly with prominent right pulmonary artery

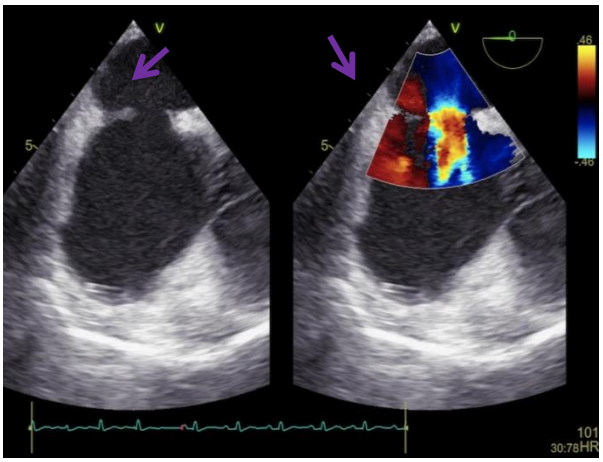

Figure 3. Transesophageal echocardiography showing secundum ASD left to right shunt

DISCUSSION

Atrial septal defect can remain undiagnosed until adulthood. ASD types include secundum ASD (80% of ASDs; located in the region of the fossa ovalis and its surrounding). The shunt volume depends on right ventricle/left ventricle (RV/LV) compliance, defect size, and LA or RA pressure. A simple ASD results in left to right shunt because of the higher compliance of the RV compared with the LV (relevant shunt in general with defect sizes ≥10 mm), and causes RV volume overload and pulmonary overcirculation.[1] A unique feature of ASD is its slow clinical progression with most children and young adults being free of symptoms, contributing to late diagnosis; hence, ASD represents the most common congenital heart disease (CHD) diagnosed in adulthood, accounting for 25–30% of new diagnoses.[8] So it is important for all cardiologists to have a solid foundation of the basic pathophysiology and management of CHD and understand when to make a referral. Besides that, as many forms of simple or maybe moderate-complexity CHD can be asymptomatic at younger age, many such individuals will be able to serve in the military.[9] When symptoms occur, patients often first notice dyspnea, fatigue, exercise intolerance, or palpitations.[10] Some patients may present with syncope or even with peripheral edema from overt right heart failure and others may develop recurrent pulmonary infections.[11] ATs, including AF and AFL, are present preoperatively in about one-fifth of adults with ASDs.[12] Our patient had only one month history of palpitations and physical intolerance.

In adults, an ASD may not be initially considered in the different diagnosis because there is considerable overlap in symptoms. TTE is one of the main intial test for the evaluation of patients with this constellation of symptoms. The guidelines recommend diagnosing an ASD by demonstration of shunting across the interatrial septum, with evaluation of the right heart and for associated abnormalities.[10] However, the interatrial communication may remain undiagnosed unless there is a high index of suspicion. As with other diagnoses, the sensitivity of echocardiography depends on the echo machine, acoustic windows, ultrasonographer, and echo reader. TEE provides higher definition visualization of the interatrial septum, it can more precisely assess the size of an ASD and guide procedural planning.[11] TEE provides a better appreciation of cardiac anatomy and hemodynamic evaluation than TTE in patients with ASD.[12] Because our patient is a male active duty officer in his fifties, he was not suspected of having a CHD and underdiagnosed in the first place.

The primary indication for ASD closure is a haemodynamically significant shunt (i.e. one that cause RA or RV enlargement), irrespective of age and symptoms, unless severe and irreversible pulmonary arterial hypertension (PAH) is present. [1,13] Available approaches to ASD II closure include percutaneous device closure and surgical closure. Surgical closure is reasonable when the anatomy of the defect is not amenable to a percutaneous approach or when concomitant tricuspid valve repair or replacement is planned. For those who have an ostium primum, sinus venosus ASD, or coronary sinus defect, surgery is the recommended technique.[11] Surgical repair has low mortality <1% in patients without significant comorbidity, and good long-term outcome when performed early (childhood, adolescence) and in the absence of pulmonary hypertension (PH).[13] A percutaneous approach is preferred when the anatomy of the defect is suitable as it avoids the need for cardiopulmonary bypass, cardioplegia, thoracotomy, sternotomy and related bleeding, or central nervous system complications, while carrying a cosmetic advantage, and also allowing a shorter hospital stay with faster rehabilitation.[11,14] A meta-analysis study suggests transcatheter ASD closure resulted safer in terms of in-hospital mortality, perioperative stroke, and post-procedural AF compared to traditional surgery.[15] Percutaneous closure of ASD II under fluoroscopic guidance is now considered a routine procedure. Studies using a variety of devices have reported good success and low complication rates in children and adults, even in the elderly. [16,17]

A low dose of radiation exposure during fluoroscopy can be achieved for transcatheter ASD closure even in complex ASDs by reduction of frame rate, avoidance of lateral view and cine acquisition, and limitation of fluoroscopic time by

avoiding unnecessary manoeuvres and using echocardiographic guidance as much as possible.[18] But it has been suggested that echocardiography alone could be used to guide device placement. TEE or TTE without fluoroscopy have been used successfully to guide peratrial or perventricular repair of ventricular septal defects.[19] Some studies have reported the use of TEE or TTE to guide percutaneous ASD closure without fluoroscopy.[19,20] The first successful transcatheter closure of ASD II using TEE fluoroscopy-free technique in Indonesia was held by Prakoso R, et al in 2018.[21] Percutaneous ASD closure under TEE guidance alone is an effective and safe procedure. Nevertheless, the distance to the mitral valve must be considered carefully because it can complicate the procedure if the distance is too short. A potentially important advantage of TEE-guided percutaneous closure over fluoroscopy-guided closure is that it avoids exposure to radiation and contrast agents. In addition to reducing the risks for the patient, TEE-guided percutaneous closure without fluoroscopy also prevents radiation to the medical staff and avoids the need for heavy lead clothing.[22]

The chronic left-to-right shunt associated with ASDs leads to increased hemodynamic load and geometric remodeling, both at a cellular and macroscopic level. This is most commonly seen in the RA and RV, but has also been described in left heart structures.[17,23] Furthermore this chronic volume stress leads to the electrical remodeling that may precipitate development of arrhythmias. Atrial myocyte electrophysiologic properties are altered, with increased intra-atrial conduction time a common finding, likely from combination of interstitial fibrosis and chamber enlargement.[24,25] Sinus node conduction properties may also be as altered, even in the pre-operative state.[25,26] ATs are commonly seen in patients with ASDs, regardless of ASD type. AFL and AF are relatively rare in childhood, but become more prevalent with increasing age at time of repair or closure. [17] AFL and AF in patients with ASDs may be treated in similar fashion to the general population, with appropriate consideration for rhythm control strategies with anti-arrhythmic medications and electrical cardioversion as indicated.[23] Appropriate anti-coagulation guidelines should also be followed.[27] All patients with symptoms consistent with potential arrhythmias should be referred for EP assessment prior to ASD closure, and assessed with at least a 24-hour Holter ECG monitoring. If indicated, any EP study with or without ablation must be performed before device implantation as this will make access to the LA more complicated afterwards, although still feasible.[28]

Closure of an existing ASD, in isolation, is generally insufficient to abolish an existing AT and catheter ablation should be considered before defect closure.[29] Ablation procedures have inconsistent medium-term results in patients with documented atrial arrhythmia prior to device closure with about 50% having symptomatic arrhythmia on follow-up.[30] However, this should not preclude ablation procedures wherever possible**.** Surgical treatment of ASD, which had been the only treatment method for more than 45 years, may be associated with the occurrence of rhythm disorders such as AF or SVT, although some authors noted a reduction in supraventricular arrhythmic burden after closure.[31] As a treatment option, percutaneous ASD II closure is also asscociate with this. A prospective study showed transcatheter closure of ASD II does not reduce arrhythmia that appears prior to ASD closure.[32] It is associated with a transient increase in supraventricular premature beats and a small risk of AV conduction abnormalities and paroxysmal AF in early follow-up. Larger device size and longer procedure time are associated with increased risk of supraventricular arrhythmia on early follow-up.[33]

Atrial septal defect closure after the age of 40 years appears not to affect the frequency of arrhythmia development during follow-up. However, the patient's morbidity benefits from closure at any age (exercise capacity, shortness of breath, right heart failure), particularly when it can be done by catheter intervention.[32] The remodelling process and associated increase in cardiopulmonary function commence immediately after closure and continue for several years.[34] Decreased RV volume improves ventricular interaction and LV filling. Subsequent increase in LV stroke volume and cardiac output is probably the main mechanism behind the improvement of exercise capacity after closure. These effects occur in patients of all ages, both symptomatic and asymptomatic.[35] This supports timely closure of sizeable ASD II, regardless of age and symptoms.[36] Patients who have had percutaneous ASD device closure should have an TTE performed at 24 hours to assess for device malposition, residual shunt, and pericardial effusion. Repeat TTE is recommended at 3, 6, and 12 months. A routine clinical follow-up and TTE should be done every 1 to 3 years thereafter. [37] Following closure of ASD, other considerations arise for evaluation and treatment of ATs. Incidence of ATs is decreased post-closure, but recurrence rate may still be significant, particularly in patients who underwent ASD closure at older age, had larger shunts, or with other comorbidities.[23,25,30] It is therefore advisable to conduct a thorough follow-up after ASD II closure, including ECG monitoring, especially in the early post-procedural period.[33]

In this case report, our patient had arrhythmia catheter ablation after was uncovered AT on holter examination and EP study. He had percutaneous transcatheter ASD II closure without fluoroscopy procedures six months later, because of many considerations which had been mention above. He stated a physical improvement after the procedures, and was able to carry out activities as before. However, this case report had a limitation because the absence of an objective assessment for patient's quality of life. We didn't perform the 6-minute walking test (6MWT) as an assessment of the functional capacity or other cardiopulmonary exercise test. Because he is still at risk of having heart rhythm disturbances in the future, he should have a thorough follow-up periodically.

CONCLUSION

Atrial septal defect as a common congenital heart disease in adult is still undersuspicious and can remain

undiagnosed. Early diagnosis and follow-up of ASDs offers the best opportunity to avoid late complications.

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A Case Report of Atrial Fibrillation in Cases of Hypertensive Emergencies and Acute Decompensated Heart Failure : What is The Most Common Cause?

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ABSTRACT

Introduction: Atrial fibrillation (AF) is a persistent supraventricular tachyarrhythmia caused by irregular electrical activity that causes palpitations, dyspnea, fatigue, and chest pain. It is associated with hypertensive emergencies and acute decompensated heart failure (ADHF). In patients with ADHF, AF affects 20%-35% of patients at clinical presentation. In the recent decade, hypertensive emergencies have increased five times among AF hospitalizations, and hospital admissions for AF have climbed by 66% because of the aging population, chronic disease, and technological developments.

Case Presentation: An 84-year-old man arrived at ER of RS Akademik UGM, Yogyakarta complaining of severe shortness of breath for the past three days. Based on anamnesis and medical assessment, the initial diagnosis was ADHF forester II, hypertensive emergencies and atrial fibrillation with normo-ventricular Response (AFnVR).

Conclusion: A review of prior studies found that the predominant cause of AF was hypertension. In this case, hypertensive emergencies was the root cause of AF. Uncontrolled hypertension such as hypertensive emergencies causes structural and electrical anomalies in the heart, which causes Atrial fibrillation.

INTISARI

Pendahuluan: Fibrilasi Atrium (AF) adalah takiaritmia supraventrikular persisten yang disebabkan oleh aktivitas listrik yang tidak teratur yang menyebabkan palpitasi, dispnea, kelelahan, dan nyeri dada. Hal ini terkait dengan keadaan darurat hipertensi dan gagal jantung dekompensasi akut (ADHF). Pada pasien dengan ADHF, AF mempengaruhi 20%-35% pasien pada presentasi klinis. Dalam dekade terakhir, keadaan darurat hipertensi telah meningkat lima kali lipat di antara rawat inap AF, dan penerimaan rumah sakit untuk AF telah meningkat 66% karena populasi yang menua, penyakit kronis, dan perkembangan teknologi.

Presentasi Kasus: Seorang pria berusia 84 tahun tiba di UGD RS Akademik UGM, Yogyakarta dengan keluhan sesak napas yang parah selama tiga hari terakhir. Berdasarkan anamnesis dan pemeriksaan fisik, diagnosis awal adalah ADHF forester II, hipertensi emergensi dan atrial fibrilasi dengan normo-ventricular Response (AFnVR).

Kesimpulan: Sebuah tinjauan terhadap penelitian sebelumnya menemukan bahwa penyebab utama AF adalah hipertensi. Dalam kasus ini, keadaan darurat hipertensi adalah akar penyebab AF. Hipertensi yang tidak terkontrol seperti keadaan darurat hipertensi menyebabkan anomali struktural dan elektrikal pada jantung, yang menyebabkan fibrilasi atrium.

INTRODUCTION

Atrial fibrillation (AF) is a common sustained arrhythmia that causes palpitations, dyspnea, fatigue and chest pain. It is associated with hypertensive emergencies and acute decompensated heart failure. Over the last 20 years, hospital admissions for AF have increased by 66%, and this trend is likely to continue rasing.[1,4]

In 2009, 20% to 35% of patients hospitalized with ADHF had AF at clinical presentation, and there was a significant increasing trend of monotonous hypertensive emergencies per 1000 AF hospitalizations from 2005 to 2015.[2,3] This case report aims to find the main causes of AF events in patients with ADHF and Emerging Hypertension theoretically based on recent research.

CASE PRESENTATION

An 84-year-old man, experiencing severe shortness of breath for three days, preferred to sit up while sleeping. The patient's family reported that he was diagnosed with hypertension in 2018 during BPH treatment, but did not have any further control.

The patient's vital signs showed 210/112 mmHg, irregular pulse rate with 94x/minute, breathing rate 28x/minute, and temperature 36.6 ℃. SpO_2 show 92% in room air.

The lung examination showed decreased vesicular breath sounds in the right lung with wet crackles (+/+) in 2/3 lung bases bilaterally. Cardiac examination revealed irregular heart sounds with a pansystolic murmur in the SIC 5 area between the left midclavicular line and the left parasternalis line.

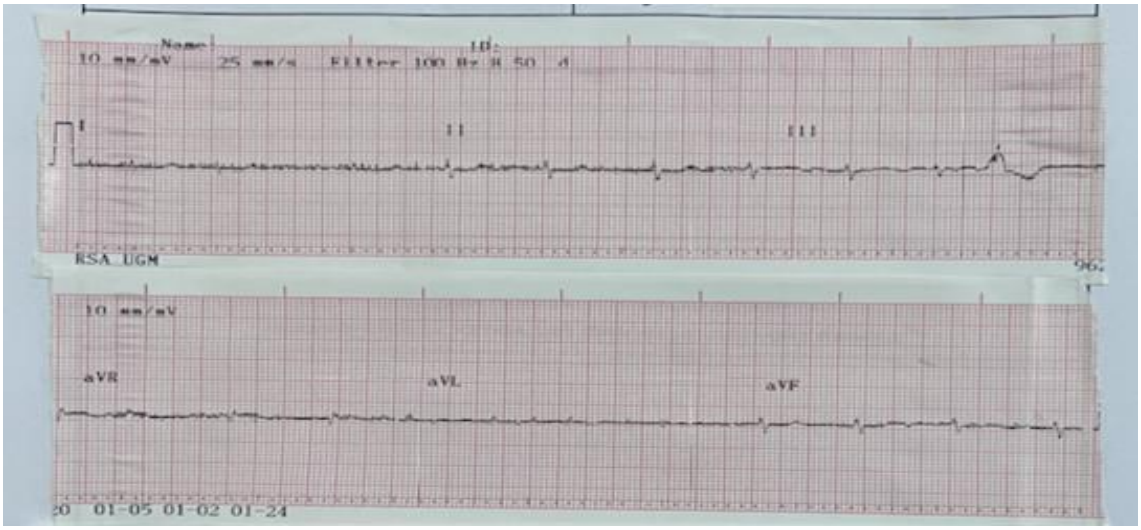
Abdominal examination showed within normal limit. Extremity xamination appear normal with no deformities and abnormalities, warm acral (+/+), CRT < 2 seconds. The volume status and pump problem are both satisfactory, and Forrester is classed as 2 (warm and wet).

Significant Lab results showed a change in platelet results were 141 million/uL which decreased from normal and increased NT-Pro BNP 4477 ρg/ml.

The EKG revealed Atrial Fibrillation Rhythm, HR 76x/minute, Normoaxis, LVH, PVC in Lead III, V3, V4. The Thorax X-ray revealed cardiomegaly, aortosclerosis, pulmonary edema, and dextra pleural effusion.

Echocardiography examination results showed concentric LVH, LA dilated, LV systolic function normal with LV EF 63% (Teich), normokinetic, LV diastolic dysfunction could not be assessed, RV systolic function normal, MR mild, TR moderate intermediate probability of Pulmonary Hypertension, LV SEC (+).

The patient's diagnosis was Acute Decompensated Heart Failure (ADHF) Forester II, Hypertensive Emergencies and Atrial Fibrillation with normo-Ventricular Response (AFnVR) based on anamnesis and medical assessment.



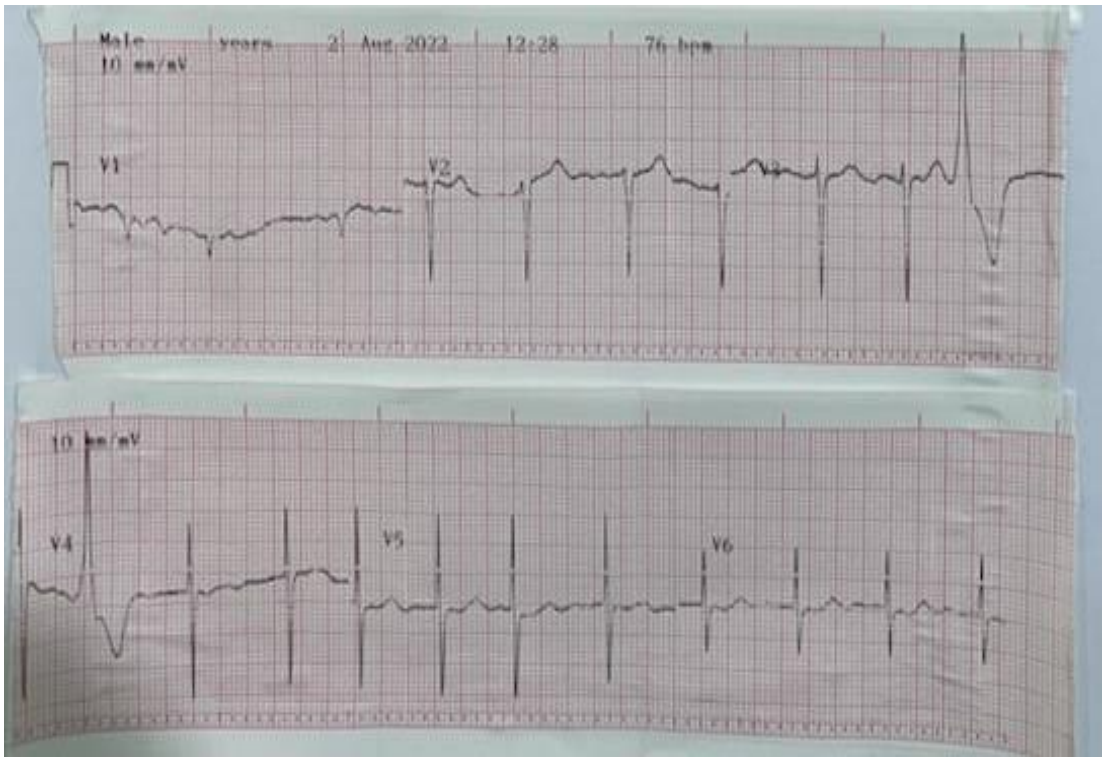


Figure 1. shows a photograph of the patient's ECG readings with the initial S. 84 years old

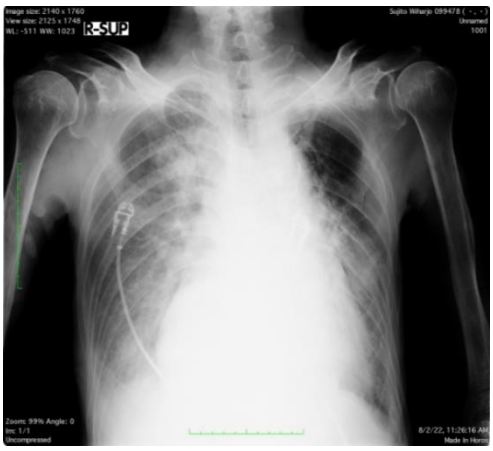


Figure 2. Photo of PA/AP Thorax X-ray Adult initial S. 84 years old

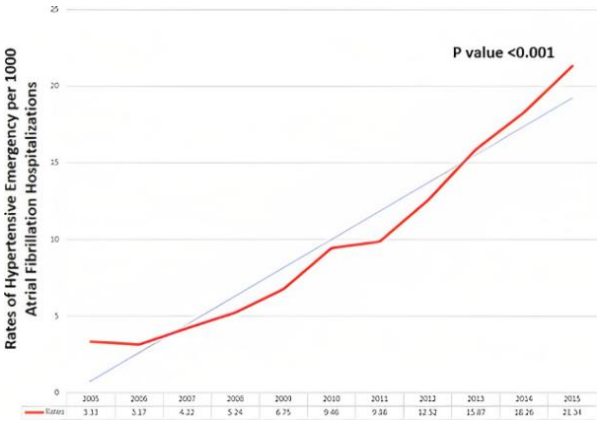


Figure 3. Rates of Hypertensive Emergencies per Atrial fibrillation mechanisms.[3]

DISCUSSION

The study involved 4,988,269 AF patients aged 18+ from 2005-2015, with 49,423 of these hospitalizations having hypertensive emergencies. Over the past decade, hypertensive emergencies have increased 5 times among AF hospitalizations.[3]

Hypertension causes atrial fibrillation through patho-physiological processes, including hypertensive urgency, emergency situations, elevated systemic pressures, and structural remodeling in the left atrium, making it more sensitive to atrial fibrillation. Atrial stretch from increased ventricular filling pressures and atrial fibrosis are all part of this remodeling.. Autonomic dysfunction and an increased renin angiotensin aldosterone system (RAAS) contribute to fibrillation.[5]

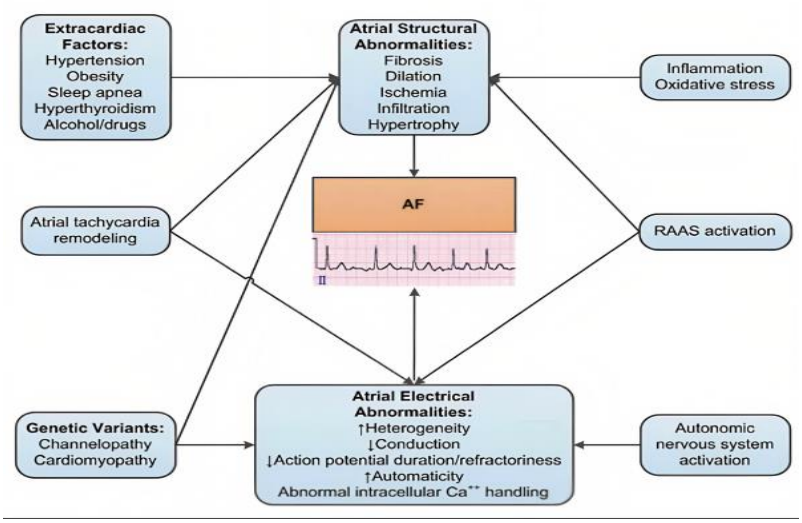


Figure 4. Atrial fibrillation mechanisms.[5]

AF is a common and poor prognosis in ADHF patients, with prevalence varying by country. In Pakistan, it was 38.1%, while in the US, it was 20%-35% at admission. In 2014, AF was the primary cause of 600,000 ED visits, 450,000 hospitalizations, and 22,000 deaths. In Beijing, the prevalence increased with age, reaching 4.87% in patients with hypertension and coronary artery disease (HT-CAD).[2-8]

These studies showed that there is a link between AF and heart failure as well as hypertension. So, what is the root cause AF in this patient ? According to the patient's previous medical history, the patient was diagnosed with hypertension in 2018 but did not receive any further therapy.

This is consistent with a 2022 study in China, in which researchers collected data from 2015 to 2019 on AF patients from 238 hospitals in China and discovered 60,390 people were hospitalized with AF, with 66.1% suffering from hypertension, particularly hypertensive emergencies episodes.[9]

CONCLUSION

There is a link between atrial fibrillation and hypertensive emergencies as well as ADHF. A review of previous studies revealed that hypertension was the primary cause that responsible for the occurrence of anatomical and electrical abnormalities in the heart, which cause Atrial fibrillation.

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Severe cefalgia and ischemic stroke in young male patient. When should a cardiologist think of Patent Foramen Ovale related cause? : A Case Report

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ABSTRACT

Introduction: Paradoxical embolism stroke is rare and uncommon case. Patent Foramen Ovale (PFO) is a condition that foramen ovale remains open and mostly asymptomatic. In normal condition clot breaks free and travels to the right heart then continue to the lung, but in someone with a PFO, the clot could pass through the hole and cause stroke.
Case Presentation: A 20 years old male came to ER with chief complain of severe cephalgia, tremor at both hands and chest pain. Cardiac physical examination, chest X-Ray, and ECG within normal limits, infarct in bilateral thalamus and cerebral oedema on head CT scan examination, PFO 3 mm from echocardiography. Patient on antiplatelet therapy and planned to refer for PFO closure procedure when on outpatient clinic.
Conclusion: Paradoxycal embolism stroke is rare and uncommon in young adult patients, as a cardiologist we should think PFO as one of the possible etiology. A good examination to diagnose an optimal collaborative management can prevent secondary complication to the patient.

INTISARI

Pendahuluan: Stroke emboli paradoksal adalah kasus yang jarang terjadi dan tidak umum. Patent Foramen Ovale (PFO) adalah suatu kondisi di mana foramen ovale tetap terbuka dan sebagian besar tidak menunjukkan gejala. Pada kondisi normal, gumpalan darah akan terlepas dan mengalir ke jantung kanan kemudian berlanjut ke paru-paru, tetapi pada seseorang dengan PFO, gumpalan darah dapat melewati lubang tersebut dan menyebabkan stroke.
Presentasi Kasus: Seorang laki-laki berusia 20 tahun datang ke UGD dengan keluhan utama cephalgia berat, tremor pada kedua tangan dan nyeri dada. Pemeriksaan fisik jantung, rontgen dada, dan EKG dalam batas normal, infark pada thalamus bilateral dan oedema serebral pada pemeriksaan CT scan kepala, PFO 3 mm pada ekokardiografi. Pasien menjalani terapi antiplatelet dan direncanakan untuk dirujuk untuk prosedur penutupan PFO saat rawat jalan.
Kesimpulan: Stroke emboli paradoksal jarang terjadi pada pasien dewasa muda, sebagai ahli jantung kita harus memikirkan PFO sebagai salah satu etiologi yang mungkin terjadi. Pemeriksaan yang baik untuk mendiagnosis dan manajemen kolaboratif yang optimal dapat mencegah komplikasi sekunder pada pasien.

INTRODUCTION

The patent foramen ovale (PFO) is a congenital cardiac anomaly characterized by the presence of a tiny aperture in the interatrial septum, enabling the passage of blood from the right atrium to the left atrium. Patent foramen ovale is observed in approximately 27% of the overall population, however it typically remains asymptomatic and does not engender any discernible complications[1]. Nevertheless, there are instances where PFO might be linked to

paradoxical embolism, a phenomenon characterized by the migration of venous thrombi or other embolic material via the PFO and into the systemic circulation. This condition has the potential to result in ischemic stroke, particularly among those in the young adult age group[2].

The precise pathophysiological mechanism by which a PFO contributes to the occurrence of stroke remains incompletely elucidated; nonetheless, multiple conjectures have been put out in this regard. One notable aspect is that the presence of a PFO contributes to the development of venous thrombi by inducing a low-pressure region inside the right atrium and modifying the dynamics of blood flow[3]. Another factor to consider is that PFO can elevate the risk of paradoxical embolism by facilitating temporary right-to-left shunts in situations that result in increased right atrial pressure, such as coughing, sneezing, or performing the Valsalva maneuver[4].

The diagnosis and management of PFO-related stroke are challenging, as it is difficult to distinguish between incidental and causal PFO. This case report will discuss a case presentation of a PFO causing a stroke in a young patient to illustrate when a clinician should suspect a PFO as the cause when finding a young patient with an ischemic stroke.

CASE PRESENTATION

We discuss the case of a 20-year-old patient who was taken to the emergency room with a history of rapid onset of severe headache, bilateral hand tremors, and chest discomfort. The patient had no previous medical or surgical history, no family history of stroke or cardiac disease, and no history of smoking, alcohol, or drug use. The patient denied any recent trauma, infection, or travel. On physical examination, the patient was alerted and have fully oriented, with normal vital signs and have some neurological deficits. In pain examination the patient has pain numeric rating scale (NRS) scored eight out of ten. The patient heart auscultation was normal, with no murmurs or gallops. The patient chest X-ray, laboratory examination and electrocardiogram (ECG) were unremarkable.

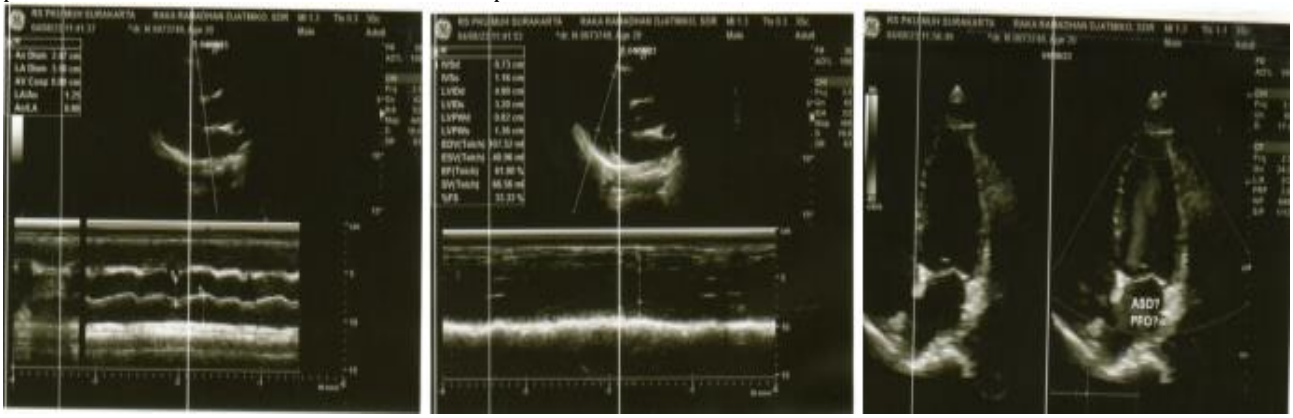

Figure 1. Echocardiography Result

Further examination using non-contrast computed tomography (CT) scan of the head revealed acute ischemic infarcts in both thalami, diffuse cerebral oedema with meningoencephalitis at left temporal lobe, and left maxillaries sinusitis. A echocardiography was done (Figure 1.), the result showed the patent have foramen ovale (PFO) measuring 3 mm in diameter, with no evidence of functional abnormalities in left and right ventricle (Table 1.). The patient was diagnosed with cryptogenic stroke due to paradoxical embolism through PFO and started on antiplatelet medication using daily dose of 80 mg aspirin and corticosteroid to control the brain oedema. The patient has already discharge from the hospital without any persistent symptom. The patient was scheduled to undergo PFO closure procedure on outpatient clinic after the patient's condition has improved and is ready for the procedure.

Table 1. Echocardiography Result

Measurements		Result	Normal Value
Aorta	Root diameter	25	20-39
Left Atrium	Dimension	31	15-40
	Ratio LA/Ao	1.25	1.1
Right Ventricle	Dimension	-	<30
Heart Function	EF (teich)	62%	53-77
	Ratio IVS/PW	-	<1.3
	EPSS	4	<10
	MVA	-	>3cm^2
Left Ventricle	LVIDd	48	35-52
	LVIDs	32	26-36
	IVSd	7	7.0-11
	IVSs	12	-
	IVS Fract T	-	>30
	LVPWd	8	7.0-11
	LVPWs	13	-
	PW Fract T	-	>30

DISCUSSION

Paradoxical embolism is a relatively infrequent and atypical etiology of stroke, yet its potential ramifications, particularly among younger individuals, should not be

underestimated. The PFO is a prevalent congenital cardiac abnormality that facilitates the passage of blood from the right atrium to the left atrium, effectively circumventing the pulmonary circulation. Patent foramen ovale typically presents as an asymptomatic condition and generally does not necessitate intervention, unless it is accompanied by paradoxical embolism. Paradoxical embolism refers to the migration of venous thrombi or other embolic material through the PFO into the systemic circulation. This condition has the potential to result in ischemic stroke, transient ischemic attack (TIA), or other instances of organ infarction[5].

The occurrence rate of PFO in the overall population is approximately 25% however, it has a higher incidence among individuals with cryptogenic stroke, with rates reaching up to 40%. Patients with extensive shunts, atrial septal aneurysm, or other prothrombotic conditions are at an increased risk of paradoxical embolism[6]. The identification of stroke associated to PFO presents difficulties, necessitating a heightened level of suspicion and a thorough assessment involving transesophageal echocardiography (TEE) with contrast and provocative techniques, such as Valsalva or coughing maneuvers. The optimal treatment approach for mitigating the risk of stroke recurrence in individuals who have experienced a stroke associated to PFO remains a topic of debate within the medical community. However, new clinical studies and meta-analyses have provided evidence supporting the superiority of percutaneous device closure compared to pharmacological therapy, particularly among patients below the age of 60[7]. Although surgical approach still pursued in certain condition of PFO that is larger than 25 mm, have already experienced failure of PFO closure beforehand, or having difficulty for percutaneous closure.

Nevertheless, there remain several unanswered matters and constraints pertaining to the therapy of stroke associated with PFO. The best antithrombotic regimen following PFO closure remains a topic of debate, as differing research papers have presented conflicting recommendations. Some studies propose that antiplatelet therapy in isolation may be adequate, while others advocate for a combination of dual antiplatelet therapy or anticoagulation for a specified duration. Furthermore, the function of PFO closure in elderly patients (>60 years old) remains uncertain due to the lack of definitive evidence. This ambiguity arises from the possibility that these individuals may have alternative factors contributing to their stroke risk, such as atherosclerosis or atrial fibrillation.[8] Furthermore, percutaneous device closure presents several technical problems and consequences, including but not limited to device malpositioning, residual shunt, device erosion or infection, atrial fibrillation, and thrombus development.[9]

Hence, the determination to undertake PFO closure necessitates a personalized approach, contingent upon a meticulous evaluation of the patient's attributes, stroke manifestations, and PFO shape. It is recommended to employ a multidisciplinary team strategy that includes neurologists, cardiologists, and interventionalists in order to enhance the management of stroke cases due to PFO[10]. Additionally, it is imperative to conduct additional research in order to effectively address the existing knowledge gaps and enhance patient outcomes in cases of stroke associated with PFO.

CONCLUSION

Paradoxycal embolism stroke is rare and uncommon in young adult patients, as a cardiologist we should think PFO as one of the possible etiologies. A good examination to diagnose an optimal collaborative management can prevent secondary complication to the patient.

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Atrial Flutter with 5:1 Conduction High Degree AV Block Developing Into Complete Heart Block in Inferoposterior STEMI: A Rare Case Report

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ABSTRACT

Introduction: Inferoposterior STEMI occurs when the right coronary artery becomes occluded, leading to myocardial oxygen deprivation. This can result in conduction disturbances, including atrial flutter, which is rare in this context. We present a case of atrial flutter progressing to complete heart block in an inferoposterior STEMI, highlighting the limited literature on this phenomenon.

Case Presentation: A 68-year-old male with a history of ischemic stroke, uncontrolled hypertension, and type 2 diabetes presented with atypical chest pain, dyspnea, malaise, nausea, and vomiting. Vital signs showed initial blood pressure of 160/80 mmHg, which later decreased to 104/68 mmHg. Heart rate was 48 bpm, respiratory rate was 24 breaths per minute, and oxygen saturation was 84%. ECG revealed ST elevation in inferior leads, reciprocal ST depression in high lateral leads, ST depression in anteroseptal leads, sawtooth P waves indicating atrial flutter, and high degree AV block with a 5:1 conduction ratio. Troponin-I level was elevated to 28012.8 ng/L, confirming inferoposterior STEMI. A complete heart block subsequently developed.

Conclusion: A comprehensive investigation of all factors contributing to electrical abnormalities is crucial. Further research is needed to deepen our understanding of the underlying pathophysiology of atrial flutter in the context of inferoposterior STEMI.

INTISARI

Pendahuluan: STEMI inferoposterior terjadi ketika arteri koroner kanan tersumbat dan menyebabkan kurangnya pasokan oksigen terhadap otot jantung. Kondisi ini dapat menyebabkan gangguan konduksi listrik jantung, yang salah satunya adalah fluter atrium, yang jarang terjadi dalam konteksi ini. Pada kasus ini, kami menyajikan sebuah fenomena fluter atrium yang berkembang menjadi blok atrioventrikular total pada pasien dengan STEMI inferioposterior, serta menyoroti terbatasnya literatur mengenai fenomena ini.

Presentasi Kasus: Seorang pria berusia 68 tahun dengan riwayat stroke iskemik, hipertensi yang tidak terkontrol, dan diabtetes tipe 2 datang dengan nyeri dada atipikal, sesak napas, lesu, mual, dan muntah. Tanda-tanda vital menunjukkan tekanan darah awal sebesar 160/80 mmHg, yang kemudian turun menjadi 104/68 mmHg. Denyut jantung pasien adalah 48 bpm, laju pernapasan 24 napas per menit, serta saturasi oksigen sebesar 84%. Pemeriksaan elektrokardiografi (EKG) menunjukkan elevasi segmen ST pada derivasi inferior, depresi segmen ST resiprokal pada derivasi lateral tinggi, depresi segment ST pada derivasi anteroseptal, gelombang P yang berbentuk seperti gerigi, mengindikasikan adanya fluter atrium, serta blok atrioventrikular derajat tinggi dengan rasio konduksi 5:1. Kadar troponin-I

yang ditemukan meningkat, yakni 28012,8 ng/L, mengkonfirmasi diagnosa STEMI inferoposterior. Pada evaluasi EKG, didapatkan blok jantung total.

Kesimpulan: Fluter atrium pada kasus STEMI inferoposterior dapat terjadi secara akut, atau akibat adanya sirkuit reentrant yang sudah ada sebelumnya, dan tereksitasi akibat kurangnya pasokan oksigen pada jaringan. Penelitian lebih lanjut diperlukan untuk mendalami patofisiologi dasar dari terjadinya fluter atrium pada kasus STEMI inferoposterior. Pemeriksaan menyeluruh terhadap semua faktor yang berkontribusi pada kelainan akitivitas listrik jantung sangatlah penting.

INTRODUCTION

Atrial flutter is a type of tachyarrhythmia resulting from reentry electrical circuit in the atria, causing a fast atrial depolarization rate, typically between 260 to 300 beats per minute, and subsequent impaired cardiac filling and pump function. In typical atrial flutter, this reentry circuit centers around the tricuspid valve annulus, with the electrical signal traveling in a counter-clockwise direction.[1] Conversely, atypical atrial flutter involves a reentry circuit around the superior vena cava or the pulmonary vein. Patients with atrial flutter commonly experience symptoms such as palpitations, dizziness, shortness of breath, and, at times, syncope. Of greater concern is the potential for stroke, with an associated prevalence of 4.1%.[1,2]

Several underlying conditions are associated with the incidence of atrial flutter, including silent coronary artery disease, chronic ventricular failure leading to atrial enlargement, valvular heart diseases, particularly affecting the mitral and tricuspid valves, and a history of cardiac surgeries.[3] Pulmonary diseases, which can induce atrial enlargement and strain, are also linked to atrial flutter.[3] While atrial flutter can result from coronary artery disease, its occurrence in the setting of acute myocardial infarction is rare and not yet fully elucidated.[4]

In this case report, we present a compelling clinical scenario involving atrial flutter and high-degree atrioventricular (AV) block occurring acutely in response to inferoposterior ST-elevation myocardial infarction (STEMI). Our primary objective is to provide physicians with a comprehensive illustration of the clinical course, diagnostic challenges, and therapeutic interventions that can be applied in healthcare settings with limited resources and facilities. Additionally, we hope that this case will stimulate further research into the mechanisms underlying atrial flutter in the context of inferoposterior STEMI, ultimately contributing to a more profound understanding of this complex condition.

CASE PRESENTATION

A 68-year-old male was admitted to our emergency room, presenting with a spectrum of symptoms, including atypical chest pain, dyspnea, malaise, nausea, and vomiting. His medical history was notable, featuring a prior ischemic stroke, uncontrolled hypertension, and type 2 diabetes. The patient had no documented history of chest pain or palpitations.

Upon initial assessment, his vital signs revealed an initial blood pressure of 160/80 mmHg, which subsequently regressed to 104/68 mmHg during the course of his presentation. His heart rate was recorded at a 48 beats per minute, and his respiratory rate was 24 breaths per minute. Furthermore, his oxygen saturation level was a concerning 84%.

The electrocardiographic (ECG) findings upon admission were striking. They revealed marked ST-segment elevation in the inferoposterior leads, concomitant with reciprocal ST depression in anterolateral leads. Adding to the complexity of the case, there was a distinct presence of sawtooth P waves, characteristic of typical clockwise atrial flutter with an atrial rate of 230 and a constant 5:1 atrioventricular (AV) conduction ratio, resulting in a ventricular rate of 48 (Figure 1A). The diagnosis was firmly supported by a troponin-I level of 28012.8 ng/L, definitively confirming the diagnosis of inferoposterior ST-segment elevation myocardial infarction (STEMI).

During meticulous monitoring by our medical team, and concurrently while awaiting the finalization of the referral process, a profound and concerning development emerged during a 30-minutes ECG evaluation. A complete heart block with junctional escape subsequently unfolded, further intensifying the intricate of the clinical scenario (Figure 1B).
Given the gravity of the situation, the patient was promptly referred for revascularization intervention to address the underlying coronary artery occlusion along with the placement of a temporary pacemaker.

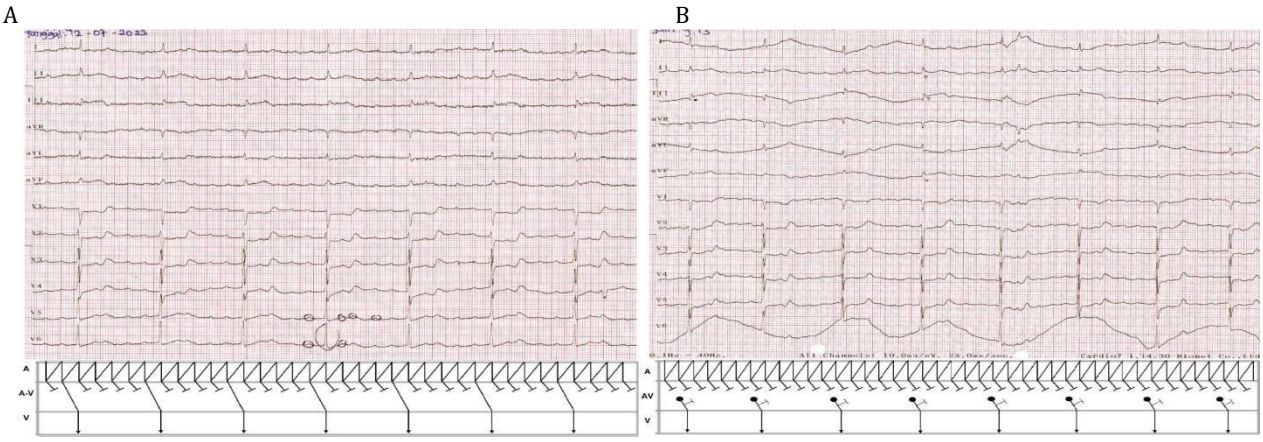

Figure 1. Electrocardiogram with ladder diagram showing an inferoposterior STEMI with atrial flutter and constant 5:1atrioventricular (AV) conduction (A). Inferoposterior STEMI with atrial flutter and complete heart block with junctional escape (B).

DISCUSSION

This case report highlights a remarkable occurrence wherein atrial flutter, in conjunction with a high-degree atrioventricular block presenting a 5:1 conduction pattern, progressed into a complete atrioventricular block in a patient diagnosed with inferoposterior ST-elevation myocardial infarction (STEMI). While atrioventricular block is a well-recognized complication in inferoposterior STEMI cases, the presence of atrial flutter in this context is exceptionally rare. In patients experiencing inferoposterior STEMI, the occlusion of the right coronary artery leads to inadequate perfusion of the inferior and posterior regions of the heart, particularly impacting the right ventricle and the atrioventricular node.[4,5] However, the precise relationship between the occlusion of this coronary artery and the acute development of atrial flutter remains a subject of ongoing investigation.[4]

One intriguing question that arises is whether the occluded right coronary artery can lead to atrial infarction and subsequently trigger atrial flutter. Notably, a case reported by Fujiwara et al. in 2022 demonstrated the occurrence of atrial tachycardia related to atrial infarction, which was successfully managed with catheter ablation.[4] One hypothesis put forth by Avula et al. suggests that the imbalance of nitroso-redox in the ischemic zone may lead to the production of reactive oxygen species (ROS).[6] Another possibility is that acute atrial scarring and fibrosis resulting from extensive areas of cardiomyocyte loss may underlie the occurrence of atrial tachycardia.[7] In relation to this case, we are prompted to explore whether these mechanisms could provide an explanation. The characteristic atrial flutter pattern observed on the ECG indicates that the macro reentry circuit exists around the tricuspid valve annulus, which might be hypoperfused due to the occluded right coronary artery.[3]

A limitation of this case report is that it originated from a healthcare facility with limited resources, where coronary angiography and electrophysiological studies were not conducted. Nonetheless, we aspire that this case report will stimulate further research into the mechanisms of atrial flutter in the context of STEMI, as a deeper understanding of these mechanisms could significantly enhance the management of these complex cases."

CONCLUSION

In conclusion, this case presents an intriguing occurrence of atrial flutter, along with high degree 5:1 AV block, developing into total AV block in inferoposterior STEMI. Though we hypothesize that the myocardial loss due to inferoposterior STEMI might induce the production of ROS, myocardial scarring, fibrosis, and later remodelling, resulting in a macro reentrant circuit, the mechanism of atrial flutter in STEMI is not yet understood, and the literature is very limited. Despite limitations of this case report due to limited resource in our healthcare facility, we hope that this report will stimulate further research into the intricate mechanisms of atrial flutter in the context of STEMI, ultimately advancing the understanding and management of these complex cases.

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Cardiac Involvement of Leptospirosis Presenting with Atrial Fibrillation

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Keywords: atrial fibrillation; leptospirosis; myopericarditis

ABSTRACT

Cardiac involvement of leptospirosis varies from arrhythmias, myocarditis, cardiogenic shock, even non-specific electrocardiographic abnormalities. A 64-year-old male farmer came to the emergency room complaining seven days of fever, dyspnea, and calf tenderness. He presented with icteric and conjunctival suffusion. Echocardiography revealed pericardial effusion. Troponin I was recorded at 0.03 ng/dl, leptospirosis IgM was positive, accompanied with severe thrombocytopenia, leukocytosis, high level of transaminases, creatinine, and BUN. During hospitalization, he developed stable atrial fibrillation with rapid ventricular response, with normal previous electrocardiography. Amiodarone successfully converted the rhythm to sinus. He received potassium diclofenac, tapered dose of intravenous methylprednisolone, antibiotics, dialysis, and supportive therapy. Leptospirosis has biphasic clinical presentation, the septicemic phase followed by the immune phase when myocarditis mostly occurs. Lymphocytes and plasma cell infiltration, petechial hemorrhages, mononuclear infiltration in the epicardium, pericardial effusions, and coronary arteritis were found in interstitial myocarditis during leptospirosis.

INTISARI

Presentasi keterlibatan jantung pada leptospirosis bervariasi, berupa aritmia, miokarditis, syok kardiogenik, bahkan perubahan tidak spesifik pada elektrokardiografi. Seorang petani laki-laki berusia 64 tahun datang ke IGD dengan keluhan demam, sesak nafas, dan nyeri betis selama tujuh hari. Tampak konjungtiva kemerahan dan tubuh ikterik. Ekokardiografi menunjukkan efusi perikardial. Troponin I tercatat 0,03 ng/dl, IgM leptospirosis positif, disertai trombositopenia berat, leukositosis, kadar transaminase, kreatinin, dan BUN yang tinggi. Dalam perawatan di rumah sakit, ia mengalami fibrilasi atrium yang stabil dengan respons ventrikel yang cepat, dengan elektrokardiografi sebelumnya yang normal. Amiodarone berhasil mengubah ritme menjadi sinus. Pasien mendapatkan terapi potasium diklofenak, metilprednisolon intravena dosis rendah, antibiotik, dialisis, dan terapi suportif. Leptospirosis memiliki gambaran klinis bifasik, fase septikemia diikuti fase imun dimana miokarditis umumnya terjadi. Infiltrasi limfosit dan sel plasma, perdarahan, infiltrasi mononuklear di epikardium, efusi perikardial, dan arteritis koroner ditemukan pada miokarditis interstisial yang disebabkan oleh leptospirosis. Steroid diduga meningkatkan kemungkinan pemulihan dengan mengurangi cedera miokard dan edema melalui peran anti-inflamasi.

INTRODUCTION

Leptospirosis is spirochaetal zoonosis disease with high morbidity and mortality, especially in resource-poor countries.[1] Globally, leptospirosis has caused 1.03 million cases with 59.900 deaths annually.[1] Furthermore, as climate change leads to flooding and heavy rainfall, leptospirosis has been re-emerging recently, causing outbreaks particularly in tropical regions.[2] In 2019, Indonesia reported 920 cases of leptospirosis followed by 122 deaths.[3] However this was

likely under-reported as the estimated annual morbidity caused by leptospirosis was 39.2 per 100.000 people.[1]

The high mortality in leptospirosis is mostly due to multiple organ damage such as kidney, liver, lung, and cardiac.[4] Cardiac involvement of leptospirosis can be cardiac failure, myocarditis, pericarditis, even non-specific electrocardiographic change.[5] The most common electrocardiographic alterations are sinus tachycardia, with the most common arrhythmia is atrial fibrillation, conduction problem and alteration of ventricular repolarization.[6,7,8] On the other side, myocarditis is potentially under-diagnosed due to unspecified clinical findings and limited diagnostic testing.[9] A histopathological analysis of 24 deceased patients dying with leptospirosis reported myocarditis was found in 96% of cases.[5] Myocarditis in fulminant leptospirosis may be common, even though not highly reported.

We report a case of fulminant leptospirosis with probable myopericarditis presenting with atrial fibrillation. We aim to contribute valuable insights to the existing knowledge about leptospirosis.

CASE PRESENTATION

A 64-year-old, male, farmer came to the emergency room complaining seven days of fever, dyspnea, and calf tenderness. He presented with prominent icteric and conjunctival suffusion. On examination, his blood pressure was 150/80 mmHg, 38.9 celsius degree, and pulse rate 109 bpm (Figure 1). Chest x-ray disclosed normal cardiac size (Figure 2). Leptospirosis IgM was positive, accompanied with metabolic alkalosis, severe thrombocytopenia, leukocytosis, high level of transaminases, creatinine, and BUN (Table 1). On the second day, he was transferred to intensive care due to shock with blood pressure 75/40 mmHg and received vasopressin 0.04 units/min. The vasopressin was discontinued the following day when his blood pressures returned to normal. Two days later, he developed stable atrial fibrillation with rapid ventricular response (Figure 3). Amiodarone successfully converted the rhythm to sinus. Echocardiography revealed 63% ejection fraction, normal cardiac chamber size, and minimal pericardial effusion (Figure 4). Troponin I was recorded at 0.03 ng/dl. He received potassium diclofenac, tapered dose of intravenous methylprednisolone, antibiotics, dialysis, and supportive therapy. On the 12th day, he was discharged without any event.

Table 1. Biochemistry evaluation during hospitalization

	D1	D2	D3	D4	D5	D12
Leptospirosis	IgM positive					
Creatinine	2.26	2.63		1.70	1.36	1.70
Ureum	82.8	168.0		167.7	126.4	50.5
Natrium	114	121			140	
Kalium	3.90	4.20			3.20	
Chloride	89.0	97.0			111.0	
Hemoglobin	9.3			9.8	9.3	
Leukocytes	24.890			16.400	22.380	11.400
Neutrofil segment	88%			91%	90%	
Hematocrit	26.9			27	25.2	
Trombocyte	44.000			37000	68.000	
PH	7.480		7.320			
PCO2	18		24			
PO2	92		173			
HCO3	13		12.3			
BE	-10.5		-13.8			
TCO2	13.6		13			
SO2	97		99			
SGOT				106		
SGPT				240		
Troponin I					0.03	

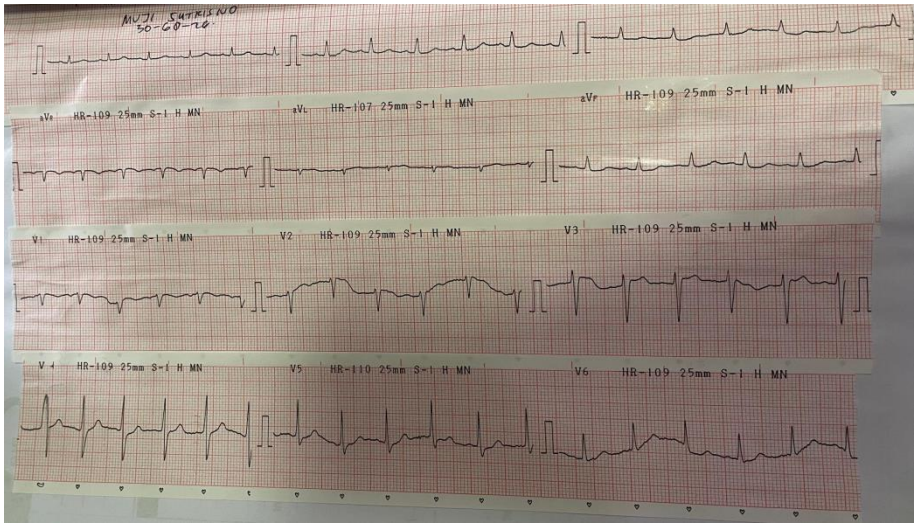

Figure 1. Electrocardiography in the emergency room showing sinus tachycardia

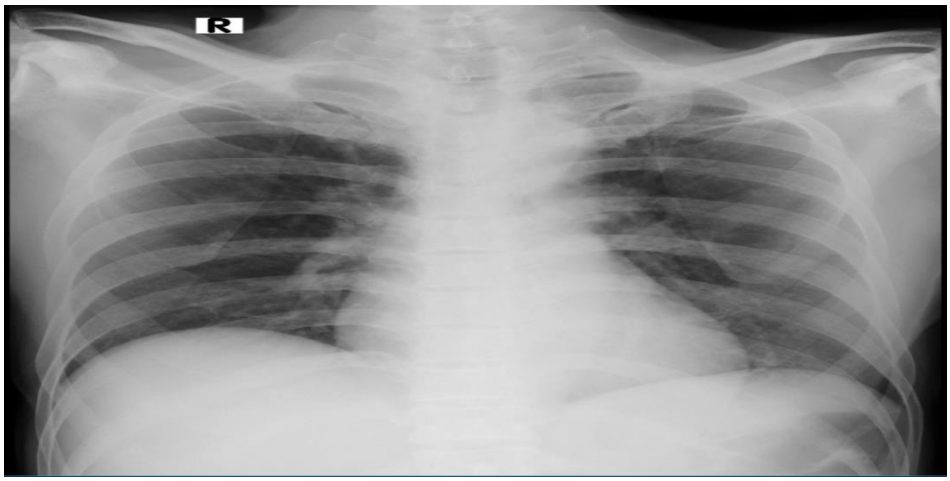

Figure 2. Chest X-ray on admission showing normal result

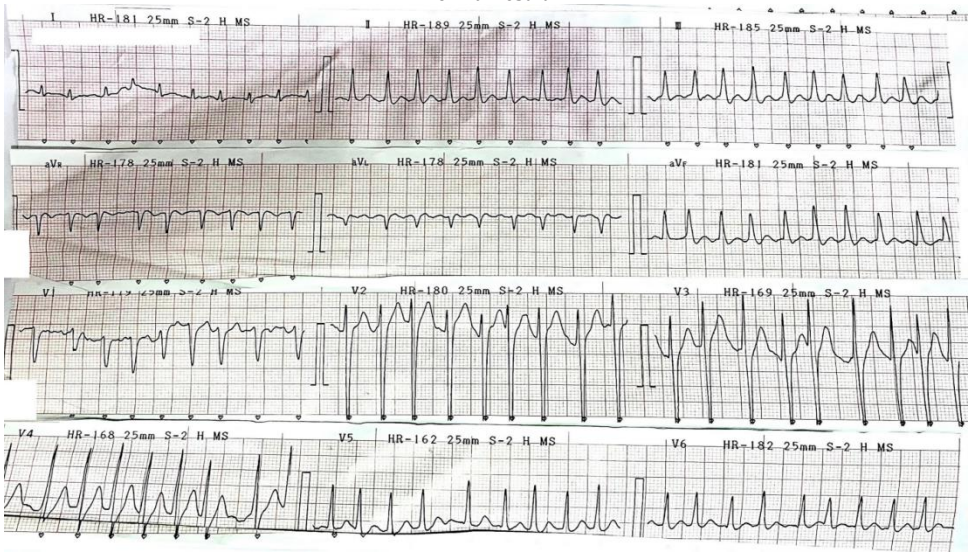

Figure 3. ECG during hospitalization disclosed atrial fibrillation withnrapid ventricular response

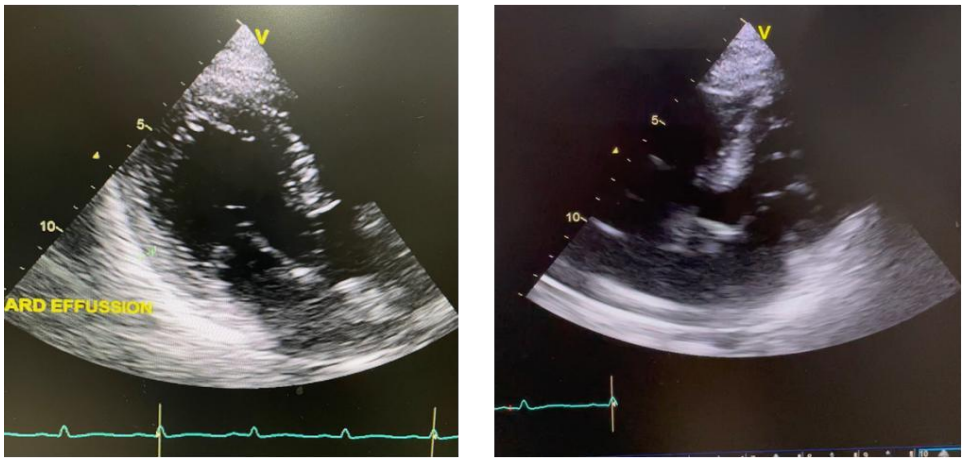

Figure 4. Echocardiography showed minimal pericardial effusion

DISCUSSION

Leptospirosis has a biphasic clinical phase in some patients.[10] The initial phase is the septicemic phase presenting with non-specific acute febrile illness.[10] Calf tenderness, myalgia, and conjunctival suffusion are common but not always shown.[11] The second phase is the immunogenic phase, occurring >7 days of symptoms onset, characterized by detection of leptospiral in urine and IgM positive from blood examination.[10,11] Organ damage occurred immunologically mediated and severe complication is expected, including myocarditis.

Myocarditis is thoroughly-documented features of leptospirosis.[5] However, definitive diagnosis of myocarditis based on histopathological, immunological and immunohistochemical criteria is not widely available and not practical. Commonly available diagnostic tests that are widely available include electrocardiography and echocardiography. Electrocardiography results of myocarditis are mostly abnormal. Tachycardia, bradycardia, conduction problem, and arrhythmia may be shown.[12] Echocardiography remains one of standard diagnostic testing despite providing broad information.[13] Echocardiography can display pericardial effusion, segmental hypokinesia, increasing wall thickness, and diastolic dysfunction even in normal ejection fraction stage.[13,14] In the meantime, high-sensitivity troponin and CK-MB are recommended to detect myocardial necrosis.[13] Cardiac MRI can reveal the magnitude of inflammation and fibrosis.[13] In this case, we could not proceed the cardiac MRI and histological study due to lack of resources in the hospital.

Postmortem histopathological studies revealed interstitial myocarditis with infiltration of lymphocytes and plasma cells.[15] Additionally, infiltration of mononuclear concurrent with petechial hemorrhage were found in the epicardium of leptospirosis patients.[5,16] Previous studies showed that hemorrhagic in epicardium was more significant compared to endocardium, with no gross evidence of endocarditis.[5,15] Immunological process causing vascular injury is postulated to be the principal mechanism.[4,5,16,17] This case highlights the magnitude of severe leptospirosis involving cardiac. Myocarditis may be common even though underreported. Vascular injury is postulated to be the principal mechanism. Early detection of cardiac involvement of leptospirosis is needed, therefore cardiac monitoring is suggested during hospitalization.

CONCLUSION

This case highlights the magnitude of severe leptospirosis involving cardiac. Myocarditis may be common even though underreported. Vascular injury is postulated to be the principal mechanism. Early detection of cardiac involvement of leptospirosis is needed, therefore cardiac monitoring is suggested during hospitalization.

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