Atrial Fibrillation with Total Atrioventricular (AV) Block to Sinus Tachycardia: Miraculously Converted in a Non-Capable Transcutaneous Pacemaker Center

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INTRODUCTION: Right coronary artery occlusions can cause inferior wall myocardial infarction (MI), which can interfere with AV node conduction. MI induced conductivity changes in the myocardial electric potential frequently cause arrhythmias. Therefore, atrial fibrillation with total atrioventricular (AV) block can coexist in some cases.
CASE PRESENTATION: A 50-year-old male admitted to the ER with chest pain, cold sweat, and nausea. Patient's history were uncontrolled hypertension, diabetes mellitus, and smoker. At first, patient was presented with atrial fibrillation with normal ventricular response (AFNVR) heart rate 95. In 30 minutes, ECG evaluation showed AFNVR with STEMI inferior, posterior, and dextra. Later, he fell into hypotension and bradycardia with heart rate 37 the ECG showed atrial fibrillation with total AV block. Patient was given fluid challenge 200cc, sulfate atropine total 3-gram, syringe pump norepinephrine and dobutamine. Patients rate got better gradually and set with dobutamine 3 mcg, heart rate reached 138 the ECG showed sinus tachycardia.
DISCUSSION: If arterial perfusion is restored quickly, the Total AV block may improve. When atropine is unsuccessful, an intravenous infusion of dopamine, dobutamine, or epinephrine may be used to enhance atrioventricular conduction, raise ventricular rate, and alleviate symptoms.
CONCLUSION: Total atrioventricular (AV) block is one of MI complications. Therefore atrial fibrillation with total atrioventricular (AV) block can coexist in this case as the patient's base rhythm already atrial fibrillation. Initial treatment is needed to enhance atrioventricular conduction and alleviate patient's symptoms.
Keywords: Atrial Fibrillation; Total Atrioventricular Block; Total AV Block

Cardiomyopathy Induced By Septicemia: A Case Report And Literature Review

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INTRODUCTION: Sepsis induced cardiomyopathy is likely underdiagnosed and has mortality implications. Current evidence supports echocardiography to identify decreased contractility irrespective of left ventricular ejection fraction for diagnosis that patient. Patient predominantly encountered in the intensive care unit and its prevalence in septic patients ranges from 10 to 70%
CASE PRESENTATION: A 70-year-old woman was brought to the hospital because of difficult in breathing. Her family said that the patient didn't have an appetite. Patient also has a chronic coughing(+) with diabetes mellitus type 2 . There are no episodes of fever, nausea, or vomitus. On general condition, the GCS pasien was compos mentis, E3V5M6. The vital signs was BP 138/79, T0: 360C, RR 24, HR: 89, and O2 saturation was 87% on room air. Blood sugar level by finger stick detected low. On physical examination, stridor(+), Lung: wheezing(+/+), rales (+/+),S1 and S2 heart sounds were normal, cyanosis in extremities (+/+), pitting oedema(+/+). CBC shows AL 4.73; AT 108; Hb 13; Albumin 3.1; AST 48; ALT 32. Chest X- Ray showed cardiomegaly. USG in EM room shows poor contractility with non-collapsible IVC. Patient was diagnose cardiomyopathy induced by septicemia. Patient was hospitalized in ICU setting, treatment included oksigenation, SABA, fluid resuscitation, inotropic drugs, hypoglicemia correction, and anti-inflammatory drugs.
DISCUSSION: Several ultrasound-guided protocols have been developed over the years to guide fluid resuscitation according to fluid responsiveness, with the aim of helping clinicians choose the right approach to balance hemodynamics and the risk of excessive fluid intake. But still have an issue about efficacy of dynamic measures of fluid responsiveness in shock to guide fluid resuscitation and improve patient outcomes as compared to usual care.
CONCLUSION: Clinicals should consider the diagnosis in patients with sepsis associated organ dysfunction and shock requiring vasopressor therapy. Echocardiography is currently gold standard for diagnosis and evaluation.
Keywords: cardiomyopathy; septicemia; echocardiography; fluid resuscitation; inotropes

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

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INTRODUCTION: Acute limb ischemia is defined as a sudden decrease in limb perfusion with the onset of symptoms of less than two weeks duration. Upper limb ischemia is less common than lower limb ischemia.
CASE PRESENTATION: 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. Blood examination showed increase aPTT to 211.6 and increased random blood sugar to 486. ECG showed normal sinus rhythm. 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.
DISCUSSION: Limb ischemia refers to symptomatic disruption of arterial blood flow to the extremities. Diagnosis of ALI can be confirmed by the classic “six Ps” (pain, pallor, pulselessness, poikilothermia, paraesthesia, and paralysis) and ankle brachial pressure index (ABI) by using Doppler (Björck, 2020). The upper extremity accounted for 17 percent of cases of acute limb ischemia. Thromboembolism is the most common cause of acute upper extremity ischemia. Atherosclerotic arterial disease in the upper extremities is associated with risk factors such as diabetes and renal failure (Andersen,2013)
CONCLUSION: 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
Keywords: Acute limb ischemia; Upper limb ischemia; Lower limb ischemia

An Occurence Of Sinus Node Dysfunction In An NSTEACS Patient: Is It A Cause-Effect Case Or Two Different Ongoing Diseases?

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INTRODUCTION: Tachy-brady syndrome refers to one of Sinus Node Dysfunction (SND) subtype in which there's alternating bradycardia with paroxysmal tachycardia. Ischemia is believed to be one of intrinsic causes of sinus node dysfunction.
CASE PRESENTATION: A 68-year-old male came to ER with chest pain, reverberated to his left arm. He had 3 episodes of syncope at home preceded by dizziness, nausea and vomiting. He had a history of inferior STEMI with successful fibrinolytic in 2022. Since then he got checked up regularly with a cardiologist and took Bisoprolol, Aspilet, Simvastatin, Nitrogliserin, and Miniaspi. At ER, he was compos mentis, his vital sign showed 153/104 in blood pressure, and slow heart beat of 39 bpm. His ECG showed extreme sinus bradycardia and inferior OMI, hence he was put on a bedsite monitor. During observation, his bedsite monitor showed alternating rhythm from bradycardia with paroxysmal tachycardia, accompanied by sinus pauses. The patient was then diagnosed with NSTEACS with suspected sinus node dysfunction. He was admitted to IMCC for heparinization. During his stay, due to his ongoing chest pain despite optimal therapy, he was referred for coroangiography. His coroangiography showed a 3-vessel-disease with chronic total occlusion in his right coronary artery.
DISCUSSION: Sinus node (SN) is perfused by the sinoatrial nodal artery which arises either from the right coronary artery (60%) or the left circumflex artery (40%). Chronic SN ischemia caused by occlusion of the SN artery can impair its function. Beside ischemia, SND can be due to intrinsic or extrinsic cause (neurocardiogenic reflexes, enhanced vagal tone, hypoxia, hypercapnia, increased intracranial pressure, hypothyroidism, hyperkalemia or drugs).
CONCLUSION: The patient had both CAD3VD and SND at the same time. Elimination/treatment of one of the cause of SND, in this case ischemia, may determine whether this SND is a cause-effect case or two different ongoing diseases.
Keywords: Sinus Node Dysfunction, NSTEACS, ECG

Myocardial Injury Related by Vivax Malaria: A Case Report

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INTRODUCTION: In Indonesia, cardiac manifestation in vivax malaria is very rare reported. We report a case of patient with vivax malaria induce myocardial injury.
CASE PRESENTATION: A male 22 years old with no significant past medical history who came to emergency department complaining chest pain and heavy to breath for four hours before came to the hospital. The pain was increased by exercise and decreased by rest. He also had high fever associated chills and headache over three days. His vital sign showed normal. The ECG showed nonspecific ST-T changes in leads II, III, aVF. Creatine kinase (CK-MB) was elevated 51.5 ng/mL. Transthoracic echocardiography showed ejection fraction was 61%, normokinetic and either systolic or diastolic function was normal.
Peripheral blood smear showed Plasmodium vivax. We treated the patient with dihydroartemisinin- piperaquine (DHP) 3 tablets per day for three days and primaquine once a day for 14 days. After three days of treatment, he had no chest pain and CK-MB result was 0.59 ng/mL
DISCUSSION: The typical manifestations of malaria are fever, chill, headache, nausea, vomiting, diarrhea but during the course of severe malaria, it affects different organ system of the body. Possible hypothesis of pathophysiology is included imbalanced proinflammatory cytokine response and/or erythrocyte sequestration by increased cytoadherence to endothelium.
CONCLUSION: Even though cardiac manifestation of malaria is rare, as a tropical country and endemic for malaria, we have to consider of cardiac complication when patients have chest pain and malaria. By reducing level of parasitaemia, the cardiac symptom can be treated.
Keywords: myocardial injury; chest pain; malaria vivax

Progressive Clinical Course of Patients with Hypertrophic Obstructive Cardiomyopathy

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INTRODUCTION: Hypertrophic cardiomyopathy (HCM) is a common genetic disorder characterized by asymmetrical left ventricular (LV) hypertrophy with no secondary causes. When left ventricle outflow tract (LVOT) obstruction is present, it's referred to hypertrophic obstructive cardiomyopathy (HOCM). The diagnosis of HOCM is challenging due to its broad symptoms, from asymptomatic to sudden death.
CASE PRESENTATION: A 49-years old male was admitted to hospital with urinary calculus. However, the patient revealed history of valvular heart disease 8 years prior but did not perform routine visits. Dyspnea is experienced only in strenuous activity. Physical examinations revealed pansystolic murmur at the 2nd intercostal space right parasternal border worsening with Valsalva maneuver and at the apex.
Echocardiography revealed dilated left atrium (LA), asymmetrical LV hypertrophy, LV diastolic dysfunction, LVOT obstruction with systolic anterior motion of the mitral valve, moderate aortic regurgitation (AR) and severe mitral regurgitation (MR). Diagnosis of HOCM was made, and patient was routinely prescribed with bisoprolol which relieved his symptoms. Sixteen-months after first admission, patient was readmitted to the hospital with complaints of palpitations and progressive dyspnea from minor physical activity.
DISCUSSION: Dyspnea in HCOM is mainly due to diastolic dysfunction and from LVOT obstruction. Obstruction severity is influenced by decreasing ventricular volume, increased contractility, reduced in preload. Thus, valsalva maneuvers, nitroglycerin and furosemide administration can provoke obstruction. The murmur of HCOM becomes louder with valsalva maneuver, important sign to differentiate from aortic stenosis. Diagnosis is done in presence of LV end diastolic wall thickness >13 mm on echocardiogram. Beta blockers remain the cornerstone of therapy to decrease LVOT obstruction. Definite therapy for advanced stage diseases including septal myomectomy or alcoholic septal ablation.
CONCLUSION: HOCM is a rare case and needs special examination to diagnose, and special treatment. Identifying the disease and a good medical record are needed to ensure better management of the patient.
Keywords: hypertrophic obstructive cardiomyopathy; dyspnea; left ventricular outflow tract; echocardiogram

Occupational Cardiology Approach in Factory Worker with Infective Endocarditis: Case Report and Literature Review

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INTRODUCTION: Infective endocarditis (IE) is a rare but potentially life-threatening multisystem disease that results from infection of the endocardial surface of the heart. IE presents a critical challenge to clinical management, particularly in occupational settings. This case report discusses the management of IE in a factory worker, integrating principles from occupational cardiology to address the cardiac condition, the potential impact of occupational factors on disease progression, and determining the appropriateness and timing of return to work.
CASE PRESENTATION: Here we report a case of a 32 years old male presented with history of fever, fatigue, and malaise. Physical examination revealed a new-onset heart murmur. Vegetation in mitral valve was founded with echocardiography examination. Despite prompt initiation of appropriate antibiotics, the patient developed congestive heart failure. The patient's occupation as a factory worker raised questions about potential occupational exposures contributing to the development of IE.
DISCUSSION: A comprehensive literature evaluation identified limited evidence linking occupational risks to the risk of IE. However, it highlighted the importance of precise personal hygiene and infection control practices in high-risk environments. Occupational exposures such as exposure to chemicals, metal dust, biological agents and physical stress may potentially contribute to the development of bacteremia, and may increase the risk of IE in certain individuals. Employing principles from both cardiology and occupational medicine, management plan was formulated including medical stabilization, functional capacity assessment, psychosocial support, workplace modifications, and education and lifestyle adaptation.
CONCLUSION: Physicians should consider the possibilty of occupational contribution and occupational approach in evaluating and managing patients with infective endocarditis in certain populations.
Keywords: infective endocarditis ; occupational cardiology ; factory worker ; occupational exposures ; management approach

A Rare Case Report: A 32 Years Old Man with Type 1 Brugada Pattern

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INTRODUCTION: Brugada syndrome (BrS) is hereditary arrhythmogenic disease characterized by ST- segment elevation in right precordial leads (V1-V3) and a high incidence of sudden cardiac death (SCD) with structurally normal hearts. Patients exhibiting the Brugada pattern also at a higher risk of ventricular tachycardia and ventricular fibrillation, potentially leading to SCD. The Brugada pattern can resolve upon correction of the reversible underlying cause.
CASE PRESENTATION: A 32 years old man came to ER with complaints of dyspnea, fever, and cough for 3 days. Patient was alert and normal physical examination except tachycardia (120) and fever (38.5). Chest
x-ray found bronchopneumonia. Blood test found lymphocytes decreased (17.0), monocytes increased (11.5) and hypokalemia (2.9). ECG examination showed coved ST-segment elevation on right precordial (V1-V2). Echocardiographic result: concentric remodeling, EF 67% global normokinetic. Neither genetic testing for BrS nor provocative challenge with sodium channel blockers was performed. Patient had a history of struck by lightning in 2001.
DISCUSSION: This patient diagnosed with bronchopneumonia, hypokalemia, and Brugada pattern type I. The ECG manifestations of BrS are often concealed and can be unmasked by a febrile state and hypokalemia (in this case). The patient's metabolic abnormalities were corrected (K: 3.6) and symptoms improve but the ECG evaluation after 24 hours showed no significant changes. Due to neither syncope episode nor family history of SCD found, it is too early concluding this as BrS. We postulate that electrocution patient's history may contribute to the existing Brugada pattern. Next ECG follow-up should perform to confirm whether Brugada pattern resolves
CONCLUSION: This report serves to bring attention to this important association and inform physicians on the potential development of Brugada pattern caused by these triggers. Further investigations like provocative challenge with sodium channel blockers need to be considered.
Keywords: brugada syndrome; brugada pattern; sudden cardiac death; fever; hypokalemia

Cor Pulmonale In Elderly Woman With Acute Exacerbation Of Chronic Obstructive Pulmonary Disease

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INTRODUCTION: Cor pulmonale is a condition characterized by structural and functional abnormalities of the right ventricle secondary to pulmonary hypertension (PH) caused by respiratory disorders. Pulmonary Hypertension results in right ventricular enlargement and may lead to right heart failure (RHF). Chronic obstructive pulmonary disease (COPD) is the most common cause of cor pulmonale.

CASE PRESENTATION: A 61-year-old woman came to emergency room with complaints of shortness of breath with a history of COPD. Physical examination revealed tachypnea, leg swelling and mild ascites. Her blood pressure, pulse and oxygen saturation were 106/74 mmHg, 97 bpm, and 72% respectively. Pansystolic murmur at the left lower sternal border as well as whistling-like breath sounds were heard on auscultation.

Electrocardiogram (ECG) revealed sinus rhythm with right axis deviation, incomplete RBBB, and right ventricular hypertrophy. Chest X-ray showed cardiomegaly and early pulmonary oedema. Echocardiogram showed RV dilatation with TAPSE 15 mm, moderate tricuspid valve regurgitation, and intermediate probability of PH. The patient received oxygen supplementation using non-rebreathing mask and standard treatment for acute exacerbation of COPD, along with furosemide and digoxin for the cor pulmonale.

DISCUSSION: Mild-to-moderate PH is a common complication of COPD and associated with increased risks of exacerbation. Patients with COPD typically present to the emergency department with complaints related to acute exacerbation. However, in this case, the patient also presented with manifestations of RHF, such as ankle oedema and mild ascites. Right heart catheterization is the gold standard for diagnosing cor pulmonale, unfortunately this examination cannot be performed in our hospital. However, from the physical examination and echocardiography examination, supports the diagnosis of cor pulmonale.

CONCLUSION: In chronic respiratory disease, PH is of mild to moderate degree, but it may worsen during acute exacerbations of the disease. Some patients experience episodes of RHF during exacerbations of the disease accompanied by a worsening of PH.

Keywords: Cor pulmonale; COPD; Pulmonary Hypertension; Right Heart Failure

Successful Fibrinolytic Strategy in Patient with Myocardial Infarction Inferior Wall Who Refuse Primary Percutaneous Intervention

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INTRODUCTION: Fibrinolytic, also known as thrombolytic, is a medication to manage intravascular clots by dissolving blood clots and often used as treatment of myocardial infarction in absence of catheterization laboratory to perform primary percutaneous intervention (pPCI). However, fibrinolytic has been widely neglected because of its side effects.
CASE PRESENTATION: A 40 year old male came to our Emergency Room with chest pain 2 hours prior to admission, short of breath on exertion and history as a smoker. The electrocardiography (ECG) disclosed heart rate of 68 bpm, sinus arrhythmia, and inferior acute myocardial infarction. A Catheterization laboratory was indicated, but the patient and family refused to undergo pPCI because they were terrified. We offered
second option to undergo a fibrinolytic procedure with the risks and they consented. We checked the fibrinolytic checklist and there were no contraindications. We proceeded with close monitoring for any major advance cardiovascular event. After it was done, the current ECG showed remarkable reduction amplitude ST elevation in leads II, III, aVF and reciprocal leads showed obscure ST depression. He felt significant improvement of chest pain, and there was a reduction of ST elevation >50%. Then he was transferred to the ICU for further observation.
DISCUSSION: Due to thesatisfactory results of PCI, fibrinolytic has been largely neglected. Guidelines recommend pPCI as the preferred reperfusion method. However, many patients still refuse to undergo PCI because they are hindered by knowledge, beliefs, customs, culture and many more. Thus, fibrinolytics can be included and become an option with regard to the checklist for contraindications of fibrinolytics. With close monitoring, fulfillment of therapy according to guidelines, fibrinolytics can provide satisfactory results and have a good prognosis.
CONCLUSION: Fibrinolytic could still be an option for myocardial infarction STEMI patients who refuse pPCI under close monitoring and comply with the guideline and still have a good prognosis outcome.
Keywords: STEMI; Successful Fibrinolytic; Myocardial Infarction

Unraveling Wellen's Syndrome: A Critical ECG Indicator of Cardiac Threats

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INTRODUCTION: Wellen's syndrome is critical electrocardiographic finding associated with myocardial infarction involving the left anterior descending artery (LAD). It may develop to life-threatening condition.
CASE PRESENTATION: A 55-year-old patient came with left chest pain 10 days prior to admission radiating to the back and experienced major chest pain 30 minutes PTA. History of hypertension and smoking. Patient had a similar complaint 1 year ago, suggested to install a heart "ring" but refused, treated conservatively by cardiologist. In ER, BP of 130/78 mmHg, HR 81 bpm, RR 18x/m and saturation 99%. He said he felt fine when arrived at the hospital. Electrocardiogram showed sinus rhythm, biphasic T waves with positive initial part and negative terminal part in leads V2 and V3 indicating Wellen's syndrome type A. No elevation of cardiac enzymes was found. He was taken for a coronary angiography and revealed proximal total occlusion of the left anterior descending artery (LAD). One stent was placed with positive outcome.
Patient referred to the ICU and discharged 3 days later in a stable pain-free condition.
DISCUSSION: A study demonstrated 75% of patients with Wellen's syndrome experienced extensive anterior wall myocardial infarction within a few weeks PTA. It suggests complete or near-complete occlusion in the lad. The presentation is transient, lasting short duration, recognition and timely diagnosis crucial.
Therefore, anamnesis, physical examination and characteristic ECG changes and subsequent intervention, such as coronary angiography and revascularization are essential to prevent further cardiac damage and improve outcomes. Early identification and appropriate management can significantly impact the prognosis of individuals at risk of developing LAD-related heart complications.
CONCLUSION: Anamnesis, physical examination and ECG interpretation especially in patients with complaints of chest pain, specific or not are crucial. Accuracy in ECG readings will have an impact on better patient outcomes according to the therapy.
Keywords: Wellen's syndrome; Electrocardiogram; Myocardial Infarction

Wellens Syndrome, when is the best time to have reperfusion?

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INTRODUCTION: Wellen's Syndrome is one of the ECG abnormalities that occur in acute coronary syndrome, characterized by T wave abnormalities (T biphasic or T inversions). The association with the incidence of critical stenosis of the coronary arteries makes it important to choose the need for immediate reperfusion or start with pharmacotherapy first in a non-PCI center hospital.
CASE PRESENTATION: A 53 years old man, he has a history of uncontrolled hypertension and is a smoker, with the main complaint of recurrent left chest pain. It's described as hot and feels crushed. During the physical examination, the awareness is compos mentis, blood pressure 165/80 mmHg, pulse 70 x/minute, temperature 36.6°C, SpO2 99%, and weight 65 kg. There are no abnormalities in the head-to-toe examination. However, the results of the ECG examination showed an inversion of T waves in leads V2-V4 while the hs-Troponin I enzyme examination showed an increase of 16969.2 ng/L. During treatment, the patient received anticoagulant, antiplatelet, statin, and antihypertensive therapy.
DISCUSSION: The characteristic of Wellen's syndrome is an abnormal appearance of the T wave (T biphasic or T inverted) seen in leads V2-V3. It mostly indicates stenosis of the coronary artery in the proximal left anterior descending (LAD). Several studies reported that definitive therapy for this case requires reperfusion to reduce the incidence of occlusion. Patients with or without a history of angina but who have an ECG with suspected Wellens syndrome are advised to undergo reperfusion therapy as soon as possible and not recommended for another cardiac testing because of the increased risk of sudden cardiac death. Multiple antiplatelet administration (acetylsalicylic acid and clopidogrel), thrombolysis, and statins did not reduce the risk of complications and death.
CONCLUSION: Immediate reperfusion in patients with Wellen's syndrome on the ECG is more advisable than pharmacotherapy to reduce occlusion and reduce the risk of sudden death.
Keywords: Wellens Syndrome; Reperfusi; ECG

Early Surgery In Complicated Infective Endocarditis: To Accelerate Is To Be Appropriate

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INTRODUCTION: Infective endocarditis (IE) is associated with a high risk of mortality and morbidity. Surgery has been the treatment of choice for IE because of severe complications. The optimal timing of the surgery has been unclear. 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.
CASE PRESENTATION: 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 with one month history of prolonged fever. 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.
DISCUSSION: As AHA recommendation that early surgery are to be done during the first hospitalization before completion of antibiotic therapy. the outcomes can be drastically improved as in reduction of in- hospital mortality and advancement in long-term prognosis in this often lethal disease.
CONCLUSION: 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.
Keywords: Infective endocarditis','Early surgery'.

A Rare Case of Atrial Fibrillation with Regular Ventricular Rhythm. Co-Existing Atrial Fibrillation and Total AV Block: Diagnosis and Management. A Case Report

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INTRODUCTION: AF and TAVB are arrhythmias that can be caused by common aetiologies. Only a limited number of cases with both arrhythmias have been reported. It is essential to distinguish both entities from the electrocardiography features and determine their cause.
CASE PRESENTATION: A 70-year old man came to the ER with shortness of breath and swollen legs since 2 days prior to admission. Patient had history of diabetes and heart failure. From auscultation, his heart rate was irregular bradycardia of 38 bpm. ECG showed TAVB. During observation at the ICCU, ECG highlighted an absence of P wave and regular ventricular rhythm, consistent with the diagnosis of TAVB and AF. Patient was also diagnosed with ADHF. Treatment given were aimed only to reduce congestion of heart failure. There were no change in ECG, albeit given dopamine infusion. Patient was discharged after symptoms were relieved and suggested for PPM implantation.
DISCUSSION: Determining the cause of TAVB and AF is vital to initiate treatment. Ischemic heart disease was excluded because of normal troponin level. Other reversible causes were also excluded, such as electrolyte disturbance, infection and medication. Paroxysmal AF happened later during admission as an effect of patient's structural heart damage associated with CAD, HF, and diabetes mellitus which was why warfarin was given but not anti-arrhythmic as it could exacerbate the AV conduction delay. TAVB in our patient with AF was concluded as a result of chronic idiopathic fibrosis and sclerosis of the conduction system, found in Lenègre-Lev disease, commonly occuring as part of the heart aging process in elderly, which indicated the need of PPM implantation.
CONCLUSION: TAVB and AF can exist together. Early diagnosis is crucial to prevent adverse cardiovascular event, including heart failure and sudden cardiac death. Reversible causes need to be excluded before PPM implantation is considered.
Keywords: Atrial Fibrillation, Atrioventricular Block, Pacemaker

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

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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
CASE PRESENTATION: 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.
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. 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. 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.
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.
Keywords: Sick Sinus Syndrome, Atrial Fibrillation, Atrioventricular Block, Pacemaker

Not Just An Ordinary Headache In Patient With St Elevation Myocardial Infarction Undergone Thrombolytic Therapy: A Case Report From Suburbs Hospital

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INTRODUCTION: ST segment elevation myocardial infarction (STEMI) is a syndrome characterized by myocardial ischaemia and persistent ST segment elevation. Reperfusion therapy (PCI/thrombolytic) must be initiated as soon as possible.
CASE PRESENTATION: A 66-year-old woman came with chief complaint diffuse chest discomfort since 10.00 am. She came to ER at 12.40 am with good condition. Electrocardiogram revealed sinus rhythm 79 bpm with ST elevation in the anterolateral lead. Chest x-ray revealed cardiomegaly with stage 1 pulmonary oedema. Leucocyte was slightly elevated and mild hyperglycaemia. Cardiac marker examinations weren't available. The patient was diagnosed with Acute Anterolateral Myocardial Infarction. Initial treatments were given. Because emergent percutaneous coronary intervention wasn't available, we used intravenous thrombolytic agent. Streptokinase (25 minutes after being diagnosed) and heparin afterwards were administered. There were no absolute contraindications for thrombolytic therapy. After two days, she had a severe headache. We found anisocoric pupils without debility. Head CT showed intracerebral haemorrhage.
DISCUSSION: The patient was diagnosed with Acute Anterolateral Myocardial Infarction. Initial therapy was administered. Reperfusion therapy needs to be initiated as soon as possible. Primary PCI cannot be performed within 120 minutes from STEMI diagnosis (referral system) and there was no contraindications to thrombolytic. We gave Streptokinase 1.500.000 unit (25 minutes after being diagnosed). Patient had failed thrombolytic. Afer two days, the patient had a headache that could not be relieved with analgetics. However, surprisingly a pain in her head did not recede but it was not accompanied by extremity weakness. Head CT revealed Intracranial haemmorhage.
CONCLUSION: Headache is not just a simple complaint. Even though no contraindication to fibrinolytic, the risk of intracerebral haemorrhage is still present. Awareness should be given because it can be a big problem, thus anticoagulant and DAPT therapy should be postponed. Nevertheless, reperfusion therapy in this case thrombolytic therapy should be given as soon as possible in STEMI.
Keywords: Acute myocardial infarction; STEMI; thrombolytic therapy; intracerebral haemorrhage

A 3-Month-Old Boy With Ventricular Septal Defect (VSD) and Pneumonia

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INTRODUCTION: This defect arises congenitally as a result of the interventricular septum not closing completely during embryonic development. The diagnosis of VSD is made by history taking, physical examination, and supporting examinations such as thorax radiology and electrocardiogram. However, echocardiography now plays a very important role in helping to make the diagnosis. A good knowledge of the anatomy of the interventricular septum and the embryology of how it is formed is necessary.
CASE PRESENTATION: A 3 month old male brought to the hospital with complains of rapid breathing, the child is sucking ang coughing. On physical examination, it was found that the pulse was 155 x/m,RR 65 x/m, SpO2 95 % BW 3300 mg, with a general condition that seemed tight, on physical examination of the chest, it was found that there were substernal retractions, in the heart there was an additional sound murmur. The patient was then evaluated with echocardiography, on the post catheter echo examination, a large muscular VSD and PDA were found with CoA not allowing closure with a device and Pulmonary hypertension flow low resistance. This patient was diagnosed with suspected congenital heart disease and pneumonia
DISCUSSION: Children with small ventricular septal defect (VSD) are asymptomatic and have an excellent long-term prognosis. Neither medical nor surgical therapy is indicated. Prophylactic antibiotic therapy against endocarditis is no longer indicated in the majority of cases. Uncontrolled CHF with growth failure and recurrent respiratory tract infections is an indication for immediate surgical repair. Neither the age nor the size of the patient is a barrier in considering surgery.
CONCLUSION: Knowledge of septal anatomy is also necessary in understanding the clinical classification of VSD as well as its management which is determined by the size of the defect, location of the defect, and hemodynamic disturbances between the pulmonary and systemic vasculature.
Keywords: Ventricular septal defect (VSD), echocardiography, CHF.

Sudden Cardiogenic Shock in Atrial Fibrillation Patient with Severe Hypoglycemia: A Case Report

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INTRODUCTION: Cardiogenic Shock (CS) and Atrial Fibrillation (AF) are common in Emergency Department (ED) settings. CS incidence in atrial fibrillation and diabetes globally recognized between 30- 32% in total patients. In this case we will talk about the correlation between Sudden CS in AF with severe hypoglycemia (SH) patient.
CASE PRESENTATION: A 45-year-old female patient was admitted to Al-Islam Hospital after experiencing increased shortness of breath and loss of consciousness during previous hour. She arrived in ED with syncope, mottled skin, and cyanosis. The patient had no prior history of CV disease but has Type II Diabetes. Monitor showed an AF RVR rate 208 x/min, RR 48 x/min, temperature 35,80 C, saturation 57%, with increase JVP and rhales in both hemithorax. Electrical synchronized cardioversion 50J then delivered and the rhythm was converted to sinus 115x/min. Patient then being intubated on ventilator. Laboratory tests found blood glucose of 3 mg/dl ; Leukocyte 11.720 mg/dl, with creatinine serum 1,9 mg/dl. Other blood tests are within normal limits. Chest x-ray showed cardiomegaly. Echocardiography showed dilatation of LA, reduced LVEF 46% and moderate MS. The patient was diagnosed with HF, CAD, ALO, RF, AF RVR (post Cardioversion), AKI and SH
DISCUSSION: SH in this patient may be associated with incidence of AF leading to CS. The mechanism that could happen is cardiac autonomic nerve dysfunction that causing inhomogeneous atrial depolarization. Action potentials dynamic marked by prolonged QTc due to swift potassium changes in SH causing abnormal cardiac repolarization led to AF. These changes increase the risk of AF RVR led to CS. Coroangiography examination is needed for further examination.
CONCLUSION: Atrial Fibrillation with severe hypoglycemia has been led to various cardiac dysfunction, including cardiogenic shock. Early detection and management supported by prompt treatment may results with better outcome in the critically ill patients particularly in ED or intensive care unit.
Keywords: Cardiogenic Shock; Atrial Fibrillation; Severe Hypoglycemia; Emergency Department; Case Report

A Rare Presentation Of Guillain-Barré Syndrome After Myocardial Infarction Treated By Percutaneous Coronary Intervention (PCI)

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INTRODUCTION: Guillain-Barre syndrome (GBS) is an acute polyneuropathy that is mostly preceded by an infection. The incidence of GBS following myocardial infection occurs infrequently, and its occurrence after percutaneous coronary intervention (PCI) is extremely rare. Due to the high mortality of myocardial infarction (MI), disability, and complication of GBS, early detection and intervention is necessary.
CASE PRESENTATION: a-55-years old male was transferred to our hospital with typical chest pain companied by diaphoresis 5 hours before. He has a history of type 2 diabetes and was an active smoker. The ECG showed persistent ST elevation along with Q-pathology on V1-V4 and presume new ST elevation on lead V5-V6, I, AVL. The angiography was performed and found the total occlusion in the proximal LAD, 99% stenosis in proximal LCX, and 60% stenosis in RCA. He got 1 DES implantation in LAD and POBA in LCX. About 1 hour after the PCI, he complained of a numbness sensation in his limbs followed by ascending paralysis. We consult the neurologist soon, the day after the EMG was performed and the result comes with an Acute Inflammatory Demyelinating Polyneuropathy (AIDP) type Acute Motoric Sensory Axonal Neuropathy (AMSAN). He was diagnosed with GBS and is being treated with Intravenous Immunoglobulin (IVIG). Unfortunately, he doesn't respond to the treatment and got intubated. On the 10th day, his condition got worse, and died due to multiorgan failure related to septic shock.
DISCUSSION: the mechanism of GBS after MI and after surgical procedures was still unclear. It was suggested that cardiomyocytes and stroma fragments in infraction release damage-related mode molecules to activate the innate immune system and inflammatory signaling pathways which generates a strong inflammatory response. Additionally, it was suspected that cardiac remodeling related to MI and reperfusion injury during heart procedure may trigger immune-mediated peripheral nerve demyelination through similar inflammatory responses.
CONCLUSION: MI and/or PCI may be a trigger of immune-mediated response and cause severe complications such as GBS.
Keywords: Guillain-Barre Syndrome; MI; PCI

Challenging Cases Of Thrombolysis After Cardiopulmonary Resuscitation In Patients With Acute And Extensive Anterior Stemi In Non-Pci Capable Centre: A Case Report

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INTRODUCTION: Thrombolysis after cardiopulmonary resucitation in patients with acute STEMI is controversial.
CASE PRESENTATION: A 53-years-old man presented with a typical chest pain since 3 hours prior admission. The ECG showed ST segment elevation in lead V1-V6. After initial STEMI therapy, the patient had seizures leading to unconsciousness. The ECG showed a polymorphic VT without pulse. CPR was performed and 200 joule defibrillation was administered immediately. After 2 minutes, polymorphic VT persisted, so another 200 joule of defibrillation was given. The patient returned to sinus rhythm with hemodynamic instability. The patient did not have absolute contraindications to thrombolysis. Streptokinase 1,5 million units was administered. Unfortunately, thrombolysis failed. The family refused to be referred for rescue PCI. The patient underwent conservative therapy for 7 days and luckily the patient survived. Three days after discharge, echocardiographic results showed mid-anteroseptal hypokinetic and decreased LVEF (52%).
DISCUSSION: After ROSC the patients experienced cardiogenic shock. Norepinefrin was administered. A Primary PCI strategy is recommended in patients with resucitated cardiac arrest and an ECG consistent with STEMI. However, due to Non-PCI Capable Centre, thrombolysis was chosen. A meta-analysis showed that there were no significant differences of neurological recovery and length of stay between cardiac arrest patient who received PCI compared to thrombolytic after circulation restoration.
CONCLUSION: Thrombolysis may be used to treat patients with ROSC in acute STEMI.
Keywords: STEMI, ROSC, Thrombolysis

Symptomatic Total Atrioventricular Block in Teenager: A Rare Case

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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 complete blood cell count, glucose, electrolytes, and cardiac enzymes were normal. Her chest x-ray showed cardiomegaly. 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/American Heart Association 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.
Keywords: Total atrioventricular block, teenager, symptomatic bradicardia

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

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INTRODUCTION: 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

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 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

DISCUSSION: The VT wave on the ECG is a particular difficulty for emergency doctors, cardiologists and internal medicine specialists when diagnosing patients. The diagnosis of VT or tachycardia with LBBB can be made using the Brugada algorithm. On close observation, there is a P wave that appears just after the T wave, so we assume that the patient is still in sinus rhythm. However, due to the prolonged PR interval, it
was concluded that the patient was in first degree AV block. In the precordial leads, there was ST elevation in the anterior leads. So, concluded if there was first degree AV block with LBBB and ST elevation in the precordial leads,

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

Keywords: Ventricular Tachycardia ; First Degree AV Block ; Left Bundle Branch Block

A-69-year-old with Recurrent Extreme Bradycardia Post Temporary Pacemaker Placement

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INTRODUCTION: Extreme bradycardia is a state of HR <50x/minute that may be accompanied by or without symptoms. It can be life-threatening. This case aims to report recurrent extreme bradycardia post temporary pacemaker placement.
CASE PRESENTATION: A 69-year-old woman emerged to the Jantung Tasikmalaya Hospital with fatigue and headache. History of patient's pharmacological treatment was rhytmonerm and bisoprolol. On examination, patient was alert, with blood pressure was 98/56 mmHg, heart rate was 28 beats per minutes, respiratory rate was 20 breaths per minutes, temperature was 36,2 degrees Celsius, and 93% oxygen saturation on room air. On physical examination obtained normal heart sound. Electrocardiography revealed extreme bradycardia. Echocardiography revealed atrial fibrillation, heart failure with ejection fraction 25- 44%, and coronary artery disease. Patient's management included temporary pacemaker placement. After temporary pacemaker placement, heart rate was 71 beats per minutes, and electrocardiography showed atrial fibrillation with slow ventricular respond. Due evaluation, heart rate was 19 beats per minutes if temporary pacemaker was off. Patient was transferred to Hasan Sadikin Hospital for permanent pacemaker placement for further management.
DISCUSSION: The patient had a history of treatment with rhytmonorm and bisoprolol since two years ago. Patients with long-term bisoprolol treatment can cause extreme bradycardia with a prevalence of 54% and 62% with rhytmonorm treatment due to changes in the structure of the heart.1,2 Conditions of extreme bradycardia induced by bisoprolol and rhytmonorm drugs have not been effective with temporary pacemaker treatment.3 So, in this case, the patient needs a permanent pacemaker for effective long-term management.
CONCLUSION: Prolonged bisoprolol and rhytmonorm treatment can cause recurrent extreme bradycardia, then require permanent pacemaker management.
Keywords: Recurrent extreme bradycardia;bisoprolol;rhytmonorm

A 54-Year-Old Woman With Pericardial Effusion Impending Tamponade And Left Pleural Effusion: A Case Report

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INTRODUCTION: Pericardial effusion is an accumulation fluid within the pericardial space and need emergent treatment. It carries high mortality rate. In this case, we try to describe that intervention was best management.
CASE PRESENTATION: A 54-year-old woman comes to emergency room with shortness of breath for three days and getting worse. Dyspnea on effort, chest discomfort were found. She had uncontrolled hypertension since 5 years ago. The patient was alert, hypertension, dsypnea, no palpitations, and normal temperature. Oxygen saturation was 91% room air. Physical examination showed an increase in jugular venous pressure, muffled heart sounds, decreased vesicular breath sound on the left lower lung, and rales was heard at the right lung. ECG examination was sinus rhytm with low-voltage. Chest X-Ray examination showed cardiomegaly, bronchopneumonia, left pleural effusion. Echocardiography showed normal chambers with preserved LV function and moderate to severe pericardial effusion with RA collapse. Pericardiocentesis was performed in the catheterization laboratory, 250 cc of xantrochrome fluid were obtained and drained about 300 cc in 8 hours. Moreover, a pleural puncture was performed and obtained 800 cc of serohemorrhagic fluid. The cytological examination for both fluid was negative for malignancy.
DISCUSSION: Pericardial effusions arise from diverse etiologies such as infection, malignancy or idiopathic. The severe ones could be life-threatening condition. Majority of patients with pericardial effusions had left-sided pleural disease caused by malignancy. In this case, we suspect it was caused by an infection that required further examination. Prognosis of pericardial effusion is essentially related to its etiology, volume of effusion, and treatment. About 83% patients recovered after pericardiocentesis. The majority of pericardial effusion will resolve with treatment of the underlying pleural disease.
CONCLUSION: Early diagnosis and intervention management are best for this life-threatening condition and also evaluation of underlying disease.
Keywords: Pericardial effusion; pleural effusion

Case Report: A Challenging Diagnosis of Acute Myocardial Infarction in A Patient with Atypical Chest Pain Presenting Paroxysmal Atrial Fibrillation and New Onset of Right Bundle Branch Block

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INTRODUCTION: Acute myocardial infarction (AMI) is a life-threatening condition that commonly characterized with chest pain. However, some patients do not show classic symptoms of chest pain.
CASE PRESENTATION: A 70 year-old male came to ER with chest pain 3-hour earlier. The pain was sharp, triggered by activity, not penetrating or radiating, and no diaphoresis. He was alert, BP 90/60 mmHg, HR 110 bpm, VAS 4, SpO2 98%, and no signs of shocks. First ECG showed AFRVR with RBBB. Second and third ECG showed only AFNVR. Troponin was 1148 ng/dl. We assessed with NSTEMI and gave him antithrombotic. During observation, VAS increased to 8, and ECG showed ST-elevation in anterior lead. We administered inotropic and thrombolysis therapy, but his BP continued to fall. We combined nor-epinephrine with vasopressor since the fluid challenge showed inconclusive result. After completed thrombolysis, VAS decreased to 4, ST-segment was lower, and BP 127/80 mmHg. He was transferred to ICCU for further monitoring
DISCUSSION: It was quite challenging to diagnose this patient. At first, we found no obvious ST-T changes with no typical symptom from this patient. There were just AF with RBBB. This phenomenon can be explained by prior studies that AF can induce AMI through inflammation and atrial diastolic overload, whereas rapid heart rate of AF leads to increase in oxygen demand and worsen ischemia. Newly onset RBBB itself is a consequence of the current anterior MI. It is related to the proximal LAD occlusion with compromise circulation in the septal arteries supplying the bundle branches. This also explain the presence of ST-elevation in the last ECG.
CONCLUSION: Atypical chest pain is a symptom that should get extra attention to avoid misdiagnosed and undertreated since it can be a life-threatening condition and difficult to diagnose, especially those which present new onset of AF and RBBB
Keywords: Atypical chest pain, Miagnosis, Myocardial infarction

Recurrent Ventricular Tachycardia And Ventricular Fibrillation Due To Severe Hypokalemia

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INTRODUCTION: Hypokalemia is present when serum levels of potassium are lower than normal. Hypokalemia can have cardiovascular effect leading to electrocardiographic (ECG) changes, cardiac arrhythmias and heart failure. Sometimes, severe hypokalemia (<2.5 mmol/L) is life-threatening and may lead to cardiac arrest.
CASE PRESENTATION: A woman, 54-year old patient presented to the emergency department with complains of dyspneu dan chest pain since 3 days ago. She was diagnosed with Acute Decompesated Heart Failure Forester II and Non ST-Elevation Myocardial Infarction (NSTEMI champit). The patient was then treated with drugs, loop diuretics and unfractionated heparin. On the 2nd day of hospitalization the patient had a cardiac arrest with ventricular tachycardia mode, with no complain of chest pain before, resuscitation and defibrillation was performed and the patient was return of spontaneus circulation (ROSC). ECG rechecked showed prolonged QT interval with no changes of ST-T segment and potassium was urgently checked which showed severe hypokalemia (K: 2.4 mmol/L from 3.6 mmol/L before). Intravenous supplemental potassium cloride was administered and potassium level increased to 3.4 mmol/L, but six hours later, the patient had another cardiac arrest with ventricular tachycardia/ventricular fibrillation mode leading to death.
DISCUSSION: Hypokalemia is associated with abnormal cardiac rhythm. The main etiologies of hypokalemia is classified into 3 groups: increased loss (renal or gastrointestinal), intracellular shift (due to medications or hormonal abnormalities), and reduced K+ intake. Characteristic ECG changes of hypokalemia include changes in T wave morphology (flattening and inversion of T waves) in mild hypokalemia, followed by prolonged QT interval, prominent U wave, and mild ST segment depression in severe cases. Moreover, hypokalemia can present with various patterns of arrhythmia such as premature ventricular complexes, atrial fibrillation/flutter, supraventricular tachycardia, and, in the worst cases, torsade de pointes, ventricular tachycardia, and ventricular fibrillation, which can be life-threatening.
CONCLUSION: Patient with hypokalemia may have recurrent ventricular tachycardia/ventricular fibrillation.
Keywords: Hypokalemia; ventricular tachycardia; ventricular fibrillation; ADHF Forester II; NSTEMI champit; prolonged QT interval; return of spontaneus circulation.

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

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INTRODUCTION: Active lifestyle has been found to reduce cardiovascular events (CVE) and increase outcome and survival in acute coronary syndrome (ACS) patients. We report the case of a worker who presented with chest pain and no history of cardiovascular disease.
CASE PRESENTATION: a 54-year-old physically active worker who was a former marathon athlete with a history of intermittent chest pain came 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 a slow heart rate (49 bpm). 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 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, including SA and AV nodes, 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, such as shock,
atrial fibrillation, and acute pulmonary edema, compared with physically inactive patients ($p<0,001$). Our patient had no CVE complications during hospitalization and required only three days of hospitalization.
CONCLUSION: Smoking is a significant cardiovascular risk factor among the general population. Appropriate exercise training is a protective factor that improves the prognosis of STEMI patients. Nevertheless, cardiac screening, smoking cessation, and active lifestyle education are mandatory for reducing the risk of ACS.
Keywords: Acute coronary syndrome; inferior STEMI; AV block; active lifestyle; smoking

Asystole, The Forgotten due to Hypokalemia

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INTRODUCTION: Hypokalemia often causes clinical conditions, including weakness and difficulty breathing. However, often examiner forgets that hypokalemia, especially severe ones, can cause arrhythmias, one of which is asystole.
CASE PRESENTATION: A 47-year-old woman came with weakness and vomiting and diarrhea. Patient has history of Diabetes Mellitus and routinely uses long-acting insulin 18 units at night. Advice given included using a syringe pump containing rapid-acting insulin in normal saline, and measuring electrolytes. Due to limitations of equipment, electrolyte checking is 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 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.
DISCUSSION: Hypokalemia causes hyperpolarization of resting membrane, inhibition of Na+-K+-ATPase and suppresses the conduction of potassium channels which results in lengthening of the duration of the action potential. The prolonged APD results in increased Ca2+ influxs. 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 CaMK. The role of CaMK activation was critical, since blocking CaMK with KN-93 completely prevented VT/VF in both myocytes
CONCLUSION: Hypokalemia if not handled properly, can cause asystole Keywords: Hypokalemia; Asystole; Diabetes Mellitus

Ischemic Stroke and ECG Changes, Is There Any Relation?

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INTRODUCTION: Ischemic stroke results from cerebral deficient blood supply caused by cerebral vascular thrombosis, cerebral embolism, or other cause. It is often associated with secondary heart impairment, which can further aggravate cerebral primary infarct lesions, even induce sudden death. There are several studies about ECG changes observed in patients with ischemic stroke. Ischemic stroke may be associated with ECG changes such as prolonged QT interval, ST-segment depression, T-wave inversion or abnormal U waves.
CASE PRESENTATION: A 81-years-old woman with history of hypertension but without known of pre- existing heart disease came to the emergency room with sudden confusion and agitation. During examination, the patient had a blood pressure of 186/75 mmHg, a pulse rate of 70 bpm, a respiratory rate of 20/minute, and oxygen saturation of 98% on room air with left limb hemiparesis. 12-leads ECG showed sinus rhythm with rSR-wave in lead V1-V2 and T-wave inversion. Cerebral computed tomography (CT) imaging identified multiple ischemic areas in crus anterior to the internal capsule left and right, lentiform nucleus left and right, corona radiata left and right, centrum semiovale left and right, and pons midline . The patient was discharge after 7 days, with moderate mental confusion.
DISCUSSION: ECG changes in ischemic stroke patients still require further research. Ischemic stroke may be associated with ECG changes such as prolonged QT interval, ST-segment depression, T-wave inversion or abnormal U waves. The precise mechanism has not been yet identified, but changes are usually transient.
Some studies suggested that most of the patients with ischemic stroke had no specific ECG changes, and ECG abnormalities may often resulting from related factors such as primary cardiac diseases, age, or physiological state
CONCLUSION: Although there are differences in research results, , identification of this unusual ECG patterns may help to preventing and treating cardiovascular complications in patients with ischemic stroke
Keywords: ischemic stroke; ECG changes; T-wave inversion

Successful Management of Malignant Arrhythmia Complication on Peripartum Cardiomyopathy at District Hospital

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INTRODUCTION: Pregnancy is associated with an increased incidence of arrhythmias. Peripartum cardiomyopathy (PPCM) present with symptoms and signs of heart failure, thromboembolism or arrhythmia. Supraventricular tachycardia (SVT) is one of the most common arrhythmias during pregnancy with prevalence of 24 per 100,000 admissions. Immediate and intensive intervention is needed in this situation.
CASE PRESENTATION: A 24 years-old woman with preeclampsia history presented sudden severe respiratory distress with SVT and hypertension after 7 days of spontaneous labour. At the time of admission, she was unconscious with oxygen saturation 76% room air. Physical examination findings rales on both chest. Initial chest X-ray presented reticular pattern on both lung with paracardial consolidation suggestive of pulmonary edema. The blood gas analytic showed lethal respiratory acidosis with pH 7.12 and pCO2 70 mmHg. After immediate synchronized cardioversion was performed, the rhythm turned to atrial tachycardia HR 160bpm. Echocardiogram in intensive care unit revealed peripartum dilated cardiomyopathy with left cavities dilatation. The management was to stabilize the patient using amiodarone in syringe pump, potent diuretics, and digitalis administration. After 2 days, the 12-leads ECG showed normal sinus rhythm rate 96bpm, the oxygen saturation was increase and remain stable. Blood gas study were repeated with pH 7.48 and pCO2 27 mmHg. After the patient's condition was stable, amiodarone, captopril, spironolactone, were given as arrythmia and cardiomyopathy therapy. She was extubated on day 3 and discharged on day 7.
DISCUSSION: Patients with PPCM often have rhythm disturbances. Indeed, malignant arrhythmias contribute significantly to sudden cardiac death in this population. Electrical cardioversion is a reasonable option at all stages of pregnancy when arrhythmias are associated with hemodynamic instability.
CONCLUSION: PPCM with any arrhythmia had greater frequencies of in-hospital death. In case of hemodynamic instability, urgent electrical cardioversion can be safely performed.
Keywords: Arrythmia; PPCM; cardioversion

ASD with paroxysmal atrial tachyarrhytmia in middle age soldier patient : A case report

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INTRODUCTION: ASD are frequently asymptomatic and can remain undiagnosed until adulthood. Atrial fibrillation and atrial flutter become more prevalent with increasing age at time of repair or closure
CASE PRESENTATION: A 50 year old male soldier, referred to 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. RHC 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.
DISCUSSION: ASD can remain undiagnosed until adulthood. When symptoms occur, patients often first notice dyspnea, fatigue, exercise intolerance, or palpitations. The primary indication for ASD closure is a haemodynamically significant shunt (i.e. one that cause RA or RV enlargement). Percutaneous closure of ASD II under fluoroscopic guidance is now considered a routine procedure. TEE-guided percutaneous closure without fluoroscopy prevents radiation to the medical staff. Chronic volume stress in ASD leads to electrical remodeling that may precipitate development of arrhythmias. Closure of an existing ASD, in isolation, is generally insufficient to abolish an existing AT and catheter ablation should be considered before defect closure. ASD closure after the age of 40 years appears not to affect the frequency of arrhythmia development during follow-up. It is advisable to conduct a thorough follow-up after ASD II closure.
CONCLUSION: ASD as a common CHD in adult is still undersuspicious and can remain undiagnosed. Early diagnosis and follow-up of ASDs offers the best opportunity to avoid late complications
Keywords: Secundum ASD; paroxysmal atrial tachyarrhytmia; radiofrequency catheter ablation; transcatheter ASD closure without fluoroscopy

A Closer Look at Thoracic Aortic Aneurysm: Could Previous History of Primary Lung Tuberculosis be the Culprit?

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INTRODUCTION: Tuberculous aortic aneurysm (TAA) is a rare cardiovascular complication of tuberculosis (TB) first described in 1882. It carries a high risk of sudden aortic rupture if untreated and occurs in less than 1% of cases, often indicating disseminated TB. Physicians in TB endemic regions like Indonesia should be vigilant for TBAA in patients with a history of lung tuberculosis symptoms and an aneurysm. Aneurysms of the ascending aorta present a unique challenge as they remain asymptomatic until complications arise.
CASE PRESENTATION: The case study of a 65-year-old female with TBAA symptoms revealed palpitations, prolonged epigastric pain, weight loss, difficulty swallowing, and a raspy voice. Diagnostic tests confirmed an aneurysm in the thoracic ascending artery, and treatment with Bisoprolol resulted in improved symptoms.
DISCUSSION: TBAA is an extension of the tuberculosis (TB) infection. It occurs in the walls of the small pulmonary and meningeal arteries from the neighboring or contiguous inflammatory foci. It often causes aneurysms in the tuberculous cavities and meninges. The progression of infection occurs from the lungs to various parts of the body. Clinical features of TBAA vary greatly, ranging from asymptomatic aneurysm with or without constitutional symptoms like pulsatile or palpable mass, chest pain, dysphagia, hoarseness, abdominal pain, back pain, frank rupture, bleeding, and shock. Due to the wide variations in symptoms, the investigation becomes a major problem.
CONCLUSION: Clinical features of TAA are highly variable, ranging from asymptomatic with or without constitutional symptoms. The diagnosis of tuberculous aortitis is very challenging and the diagnosis without histology is further challenging. Active tuberculosis in any other organ or past history of tuberculosis may direct the clinician towards this very rare diagnosis. Development of diagnostic criteria for tuberculous aortitis based on the clinical findings and imaging may be a future research area.
Keywords: aortic aneurysm; ascending aortic aneurysm; Tuberculous aortic aneurysm;

Bromocriptine in the Therapeutic Management of Peripartum Cardiomyopathy: Case Report

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INTRODUCTION: Peripartum cardiomyopathy (PPCM) is recognized as a major cause of pregnancy related heart failure with high morbidity and mortality
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 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.
Keywords: Peripartum Cardiomyopathy, Bromocriptine, Heart Failure – Pregnancy, LVEF

Myocardial Injury Related by Vivax Malaria: A Case Report

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INTRODUCTION: In Indonesia, cardiac manifestation in vivax malaria is very rare reported. We report a case of patient with vivax malaria induce myocardial injury.
CASE PRESENTATION: A male 22 years old with no significant past medical history who came to emergency department complaining chest pain and heavy to breath for four hours before came to the
hospital. The pain was increased by exercise and decreased by rest. He also had high fever associated chills and headache over three days. His vital sign showed normal. The ECG showed nonspecific ST-T changes in leads II, III, aVF. Creatine kinase (CK-MB) was elevated 51.5 ng/mL. Transthoracic echocardiography
showed ejection fraction was 61%, normokinetic and either systolic or diastolic function was normal. Peripheral blood smear showed Plasmodium vivax. We treated the patient with dihydroartemisinin- piperaquine (DHP) 3 tablets per day for three days and primaquine once a day for 14 days. After three days of treatment, he had no chest pain and CK-MB result was 0.59 ng/mL
DISCUSSION: The typical manifestations of malaria are fever, chill, headache, nausea, vomiting, diarrhea but during the course of severe malaria, it affects different organ system of the body. Possible hypothesis of pathophysiology is included imbalanced proinflammatory cytokine response and/or erythrocyte sequestration by increased cytoadherence to endothelium.
CONCLUSION: Even though cardiac manifestation of malaria is rare, as a tropical country and endemic for malaria, we have to consider of cardiac complication when patients have chest pain and malaria. By reducing level of parasitaemia, the cardiac symptom can be treated.
Keywords: myocardial injury; chest pain; malaria vivax

ST-segment elevation following electrical injury: Is it ST-elevation myocardial infarction?

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INTRODUCTION: Electrical injuries can cause health problems, and their manifestation may resemble other diseases, such as ST Elevation Myocardial Infarction (STEMI), with different pathomechanisms. This report describes a patient who presented with chest pain and breathlessness 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 before and experienced a transient loss of consciousness. Electrocardiography (ECG) showed ST-elevation in lead V1-V4. The carotid Doppler and hemodynamic profile measurements were normal. The clinical manifestation and ECG findings resembled STEMI significantly, but the symptoms and subsequent ECG showed marked improvement in some hours without revascularization measures. The patient was eventually discharged after two days of hospitalization without any complications.
DISCUSSION: Electrical injuries are relatively infrequent but can cause mild-to-severe health problems. Electrical injuries can cause cardiac problems such as heart rhythm disturbance, 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 vasoconstriction of the coronary artery due to electrical injury that disturbs the oxygen supply, resulting in clinical features and ECG findings of myocardial infarction. Although it has similar features, this problem is transient and resolved without revascularization methods. Similar to our patient, the symptoms and ECG findings showed a remarkable improvement after symptomatic management
CONCLUSION: Electrical injuries can have symptoms and ECG findings similar to STEMI, but with different pathomechanisms and require different treatments. Therefore, careful history-taking is required for a correct diagnosis and appropriate treatment.
Keywords: Electrical injuries; ECG; ST-elevation myocardial infarction

Can Wellens Syndrome Resolve without PCI?

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INTRODUCTION: The recommended treatment of Wellens syndrome is percutaneous coronary intervention (PCI) to reduce the risk of anterior ST-elevation myocardial infarction (STEMI). However, PCI is unavailable in sub-district (type C) general hospitals. Therefore, we would like to assess the outcome of a patient with Wellens syndrome treated only with a pharmacological approach.
CASE PRESENTATION: A 69-year-old patient with a history of intermittent chest pain was admitted for generalized weakness and hypertensive crisis. Electrocardiograph (ECG) examination demonstrated T wave inversion in lead V2-V6, suggesting Wellens syndrome (Fig 1). Echocardiogram showed grade I diastolic dysfunction with normal ejection fraction (EF 54.71%). The patient was administered aspirin, clopidogrel, and nitrates in the Intensive Care Unit (ICU). The patient showed marked improvement and was discharged
after six days of hospitalization. One week after discharge, the follow-up in the cardiac polyclinic revealed a normal ECG (Fig 2).
DISCUSSION: Our patient did not receive PCI treatment but did not develop anterior STEMI. Another study reported that 25% of patients with Wellens syndrome did not develop anterior STEMI. The fair outcome of our patient was likely to be contributed to the timely diagnosis and intervention (i.e
administration of antianginal drugs in the absence of chest pain). Furthermore, a study elaborated that PCI is the mainstay treatment, but aspirin, clopidogrel, and nitrates remained an effective essential treatment that
could be implemented in the case of a lack of PCI resources. Nevertheless, our study could not adequately exclude pseudo-Wellens syndrome.
CONCLUSION: The patient with Wellens syndrome may survive without PCI treatment. However, intense monitoring during hospitalization and follow-up after discharge is mandatory to observe the clinical course and risk of STEMI development.
Keywords: Wellens syndrome; PCI; percutaneous coronary intervention; outcome

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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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).
DISCUSSION: The clinical presentation of an atrial fibrillation patient who came with hypertensive emergencies and ADHF is discussed in this case. A 2020 investigation discovered 4,988,269 AF patients aged 18 and up, with 49,423 patients experiencing hypertensive emergencies. According to data from 2021 in Pakistan, the prevalence of AF in people with ADHF was 38.1%. Another study conducted in the United States discovered that 20%-35% of patients with ADHF had AF at the time of admission, and one out of every three episodes of AF was acute. So, what is the root cause AF in this patient?
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.
Keywords: Atrial fibrillation; Hypertension; Hypertensive Emergencies; Heart Failure; Acute Decompensated Heart Failure

A Rare Presentation Of Acute Myocardial Infarction In A Elder Patient: Shifting St Elevation On The Initial Electrocardiogram From Anteroseptal To Inferior Wall Infarction: A Case Report

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INTRODUCTION: ST segment changes and their distribution are commonly used as predictors of the culprit vessel during an acute myocardial infarction. ST segment changes in electrocardiographic during acute myocardial infarction cannot always be explained by logical sequelae of the injury current, vessel anatomy, and irrigation territory. We report a rare case of acute myocardial infarction with shifting ST elevation on the initial electrocardiogram (ECG).
CASE PRESENTATION: A 63-year-old smoker male with a medical history of hypertension and diabetes mellitus presented to our hospital, referred to by the clinic with infarction-type chest pain 2 hours before admission. His BP was 158/86. The physical examination within normal. The initial electrocardiogram (ECG) in the clinic showed ST-segment elevation in anterior leads (V1-4). A repeat electrocardiogram in the emergency room showed significant ST elevation in the inferior lead (II, III, aVF) with the resolution of ST elevation in anterior leads. His Troponin I was 3449 ng/ml. The patient immediately underwent fibrinolytic reperfusion with streptokinase. Follow-up echocardiogram showed hypokinetic in inferior wall and EF 59%.
DISCUSSION: Shifting anterior ST elevation during acute Inferior myocardial infarction is unusual. In this case, the initial ECG was shifting from anterior wall myocardial infarction to ST elevation in inferior leads consistent with the resolution of anterior wall ST elevation. Wraparound LAD (Type III variant) supplies blood anteriorly and posterior inferiorly to the myocardium. In several studies shifting inferior and anterior ST elevation has been reported due to a distal occlusion of a "wrapped" left anterior descending artery (LAD) to its first diagonal branch. The shifting is due to a smaller mass of the ischemic inferior myocardium, creating reciprocal changes and combined with the anterior wall's transmural ischemic myocardium.
CONCLUSION: Shifting anterior ST elevation during acute inferior myocardial infarction was a rare presentation. This condition due to occlusion of wrap around LAD to D1 branch.
Keywords: Shifting STEMI, LAD Occlusion, Wraparound LAD, Anterior STEMI, Inferior STEMI

What happens when STEMI patients are not reperfused?

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INTRODUCTION: ST-elevation myocardial infarct (STEMI) is a life-threatening coronary artery disease requiring immediate reperfusion. However, reperfusion therapy using percutaneous coronary intervention (PCI) or fibrinolytic treatment is not always available in rural areas. This report describes a STEMI patient in the hospital without any PCI or fibrinolysis resources.
CASE PRESENTATION: A 67-year-old smoker with a history of 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%).
Electrocardiograph (ECG) demonstrated inferior STEMI and sinus rhythm (Fig 1). The echocardiogram showed a reduced ejection fraction (EF 29.18%). The patient was treated with aspirin, clopidogrel, fondaparinux, and nitrates, then daily ECG monitoring was performed. The patient suffered from total atrioventricular block (TAVB) on the first and second days of hospitalization (Fig 2-3). On the fifth day, the symptoms resolved, the ECG reverted to sinus (Fig 4), and the patient was discharged.
DISCUSSION: In Indonesia, only 40 hospitals in 514 cities in Indonesia are PCI-capable. Non-reperfused STEMI patients had a lower survival rate (7.2% vs 12.7%), and are prone to complications, such as conduction defects. Our patient presented with inferior STEMI and sinus rhythm and subsequently developed transient TAVB. Factors affecting the outcome of STEMI patients included age, low systolic blood pressure, right bundle branch block (RBBB), high blood glucose, decreased glomerular filtration rate, and left ventricular ejection pressure. Our patient was elderly and had hyperglycemia and low ejection fraction.
However, the absence of hypotension, RBBB, and renal dysfunction might contribute to an excellent outcome for the patient.
CONCLUSION: STEMI management using pharmacological approaches is an alternative with comparable outcomes. Nevertheless, patient risk stratification should still be done to mitigate the condition and optimize care.
Keywords: STEMI; reperfusion; PCI; fibrinolytic; outcome; survival

Concurrent Cardiac Anomalies in a Newborn with Possible Congenital Syphilis: A Case Report from Type C General Hospital

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INTRODUCTION: Congenital syphilis (CS) can lead to severe complications and is rarely associated with congenital heart disease. This report discusses a unique newborn case exhibiting both possible CS and cardiac anomalies.
CASE PRESENTATION: A male infant was delivered via cesarean section by a 28-year-old mother diagnosed with secondary syphilis during her third trimester (TPHA titer: 1:2560; VDRL titer: 1:1), due to a lack of antenatal screening and examination. The mother's treatment was initiated inadequately with clindamycin. Although the delivery was uneventful and the baby showed no typical CS signs, a continuous murmur with systolic accentuation was observed at birth. Echocardiography revealed a large ASD measuring 7 mm, a large PDA measuring 5.1 mm with a left-to-right shunt, right ventricular dominance, and trivial tricuspid regurgitation. Despite these findings, overall cardiac function was normal (EF: 83%, globally normal kinetic). Laboratory confirmation for CS yielded reactive TPHA (titer 1:2560) and reactive VDRL (titer 1:1). The infant experienced desaturation on the second hospital day but significantly improved over the following week. Procaine penicillin was administered on the third day for potential CS management, and follow-up evaluations were scheduled to monitor cardiac anomalies and CS progression.
DISCUSSION: Although rare, cardiac anomalies can occur in CS cases. Prior research has noted instances of septal defects, myocarditis, syphilitic aortitis, and pulmonary artery anomalies, with 60% of cases asymptomatic at birth. This could be attributed to the cardiac tropism of Treponema pallidum. Neonatal infections, including syphilis during pregnancy, can potentially lead to cardiac anomalies at birth, warranting further investigation. Management strategies encompass early diagnosis, imaging studies, laboratory tests to confirm syphilis, and appropriate treatment as per established guidelines.
CONCLUSION: Prompt diagnosis and appropriate management of congenital syphilis are essential to prevent adverse outcomes and future permanent deformities. When necessary, referral to specialized healthcare facilities plays a crucial role in achieving optimal outcomes.
Keywords: Congenital syphilis; congenital heart disease; newborn

Comprehensive Approach in Diagnosing Patent Foramen Ovale in Young Adult with Embolic Stroke of Undetermined Source (ESUS) in Indonesia: A Challenging Neuro- Cardiology Case Report

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INTRODUCTION: The concept of embolic stroke of undetermined source (ESUS) was introduced by the cryptogenic stroke/ESUS international working group in 2014, with approximately 15–20% of patients with ESUS are young patients. Underlying pathologies of stroke of unknown cause are multiple, including Patent Foramen Ovale (PFO). We aim to present a comprehensive approach in diagnosing PFO from anamnesis through supporting examinations in young adult with cryptogenic stroke.
CASE PRESENTATION: A-27-years-old-woman, presented to Emergency-Department National-Brain- Center-Hospital with sudden decreased level of consciousness. She was in relatively good health without past medical history except for chronic complaints of intermittent-migraine and limbs-numbness. The-patient had GlasgowComaScale of E4M5V4, meningeal-sign(+), Babinski-reflex-dextra(+) and right-lateralization, whereas other-systems were normal. Laboratory-workups were within normal-limits. Head CT-without- contrast, CTA and MRI was done which displayed subacute-cortical-infarct(middle cerebral artery territory). The-working-diagnosis was embolic stroke with unknown origin, RoPE-score 9. Digital-Subtraction- Angiography(DSA) and BubbleTest-Echocardiography was performed that shown total-occlusion of left-M1 and passing-microbubbles through tunnels(grade3/4), respectively. Moreover, based on PASCAL- classification-system, the patient was in PROBABLE category. The patient was later planned for PFO- closure with dual anti platelet therapy.
DISCUSSION: Based on characteristics, concordance was observed with previous study which stated that the most common Potential-Embolic-Sources(PES) in young patients was PFO/ASA; the rate of other-PES was low(2–7%); ESUS in older-patients was observed-mainly in male(58%) which atrial-cardiopathy as PES. Chronic-intermittent-migraine was found in our patient that not-resolved with medication; PFO was associated with symptoms such as migraine, with the hypothesis, Paradoxical-Embolism which lead to tiny- brain-infarctions, triggering low-perfusion/cortical-spreading-depression, may cause migraine attack. RoPE- Score identifies cryptogenic-stroke who are likely to have pathogenic PFO rather than incidental (cut-off score6). Whereas, PASCAL-classification predict the likelihood of PFO as the-associated-stroke-causal.
CONCLUSION: PFO should not be overlooked when exploring cryptogenic-stroke at young-age. RoPE- score(simple scoring) is imperative as a guidance in diagnosing PFO. Therefore, not only pay attention to serious complications (stroke/TIA) but also related symptoms such as migraine.
Keywords: ESUS ; Patent Foramen Ovale ; Young Adult ; Cryptogenic Stroke;

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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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.
DISCUSSION: Atrial flutter with AV nodal block in inferoposterior STEMI is rare. The patient's history of uncontrolled hypertension and type 2 diabetes suggests a pre-existing atrial flutter. The occlusion of the proximal right coronary artery may trigger pre-existing reentrant circuits, leading to atrial flutter.
Alternatively, atrial flutter could be a direct consequence of the myocardial infarction. AV nodal block commonly occurs in inferoposterior STEMI. The combination of atrial flutter and AV nodal block has a worse prognosis, necessitating prompt identification and treatment.
CONCLUSION: Managing inferoposterior STEMI is challenging due to potential electrical disturbances. Although less common, atrial flutter can occur in specific cases. Thus, 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.
Keywords: Atrial flutter; AV nodal block; myocardial infarction; right coronary artery

Differentiating Diffuse Subendocardial Ischemia with De Winter T Wave on Electrocardiography to Enhance Optimal Reperfusion Therapy Decision: A Case Report

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INTRODUCTION: The de Winter T wave pattern is equivalent to anterior ST-elevation myocardial infarction (STEMI) and can be challenging to distinguish from diffuse subendocardial ischemia or non-ST elevation myocardial infarction (NSTEMI). Accurate differentiation is crucial for selecting the appropriate reperfusion therapy.
CASE PRESENTATION: A 57-year-old male presented with sudden chest pain, dyspnea, and nausea. Initial ECG findings suggested the de Winter T wave pattern with widesperead upsloping ST depression in precordial lead, tall T waves, ST elevation in aVR. But, further examination revealed T waves of insufficient amplitude. Considering the patient's medical history of diabetes and hypertension, and the elevated troponin- I level, the diagnosis was revised to NSTEMI.
DISCUSSION: This case involved a perplexing ECG pattern, straddling subendocardial ischemia and the de Winter T wave pattern. T wave amplitude played a pivotal role in diagnosis. While a tall T wave (≥10 mm) indicates the de Winter pattern, this patient exhibited an average T wave amplitude of 7 mm. Misidentifying subendocardial ischemia may trigger unnecessary catheterization, while misdiagnosing the de Winter pattern delays reperfusion therapy and jeopardizes patient outcomes.
CONCLUSION: When encountering acute coronary syndrome with ST depression but no ST elevation, it is crucial not to dismiss the possibility of STEMI. Considering the de Winter T wave pattern is essential.
However, careful evaluation of T wave morphology and amplitude is paramount to differentiate between myocardial infarction and subendocardial ischemia accurately. In this case, insufficient T wave amplitude led to a revised diagnosis of NSTEMI. This underscores the importance of differentiating between the de Winter pattern and subendocardial ischemia to select appropriate reperfusion therapy promptly.
Keywords: de Winter T wave pattern; subendocardial ischemia; NSTEMI; acute coronary syndrome

Factors of Early Recurrent Myocardial Infarction on Survivor of Myocardial Infarction after Successful Primary Percutaneous Coronary Intervention: A Case Report

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INTRODUCTION: Survivors of acute myocardial infarction (AMI) have a risk of recurrent infarction after discharge. There are causes of recurrent acute myocardial infarction (RMI) related to features of the plaque, features of the blood (thrombogenesis), and features of the patient. The majority of early RMI will occur within two weeks after patient discharge with mortality rates approximately 50% within 5 years.
CASE PRESENTATION: A 44-year-old female patient came to our emergency room with epigastric pain region radiating to substernal area, and diaphoresis since 2 hours ago. Nine days prior, she was diagnosed with post ROSC mode VT-VF on extensive anterior STEMI Killip IV in our hospital, she was then referred to PCI procedure. The patient had several risk factors such as hypertension, familial history of heart attack, and dyslipidemia. At the ER, the patient blood pressure was 106/62 mmHg, and heart rate of 76 bpm. ECG examination showed T inverted with ST-T changes, while the laboratory results showed elevation of cardiac marker. The patient was diagnosed with NSTEMI Killip I with dynamic ST changes, CHF low EF, IHD, history of CAD1VD PCI 1 DES in LAD with the indication of extensive anterior STEMI Killip I with VT/VF. The patient was given oxygen therapy, 3 tablets of CPGs and aspilets, and bolus of 3000 U heparin (drip 6 cc/hours). Referral to the higher center was then done for evaluation of NSTEMI with a history of PCI to Coroangiography examination.
DISCUSSION: Independent predictors of RMI after successful PCI are diabetes mellitus, renal dysfunction, atypical chest pain and multivessel disease, and factors within 1 year are: Killip class III or IV, hypertension, and statin use.
CONCLUSION: Patients with risk factors for AMI recurrence should undergo active surveillance and management of modifiable risk factors to improve their cardiac function and quality of life.
Keywords: Recurrent Myocardial Infarction; Acute coronary syndrome; Acute myocardial infarction

Cardiac Involvement in Diphtheria: Can ECG Changes be An Early Sign of Diphtheria Myocarditis?

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INTRODUCTION: Cardiac involvement in diphtheria is diverse but most commonly characterized by myocardial dysfunction, arrhythmias, pericarditis and endocarditis (1). ECG abnormalities in diphtheria myocarditis include AV conduction disturbances, bundle branch block, ST depression, T-wave inversion, or some combination.
CASE PRESENTATION: We report a 42-year-old male patient who came with complaints of fever and chills that fluctuated for about 2 weeks before admission. Fever accompanied by painful swallowing. From the physical examination, we found the BP was 142/78 mmHg, the heart rate was 104 bpm, and the temperature was 37.8 C. On orofacial examination, we found tonsil hypertrophy, hyperemia, and exudate.No palpable neck lymph nodes.The leukocyte count was 25.500/uL. From the microbiological examination of the throat swab, we found C.diphtheriae.We found a NSR with a heart rate of 98 bpm and normoaxis on the first ECG.An ECG evaluation was performed on the sixth day of admission, showed sinus bradycardia with a heart rate of 45 bpm and early repolarization in leads II, III, and avF.From the echocardiography examination, we found a normal cardiac chamber, normal LV systolic function, normal RV function, normal diastolic function, and normal valves.There was no pericardial effusion.
DISCUSSION: Myocarditis has been reported in 19–68% of cases with diphtheria. However, a significant proportion of these individuals will just present with ECG changes without any symptoms of failure or shock
(3) Besides STc and bradycardia, AV blocks, bundle branch blocks (BBB), ST-T changes, T wave inversion and ventricular/atrial ectopics are seen in varying frequencies in patients with myocarditis (4). In our patient we found sinus bradycardia with early repollarization.
CONCLUSION: We report a clinically suspected case of diphtheria myocarditis. Echocardiography, together with serial ECGs and clinical monitoring, should be used in the treatment of all hospitalized diphtheria patients to look for signs of myocarditis and enable timely intervention to mitigate severe and life- threatening consequences.
Keywords: Clinically suspected myocarditis; bradycardia; early repolarization; diphteria myocarditis