Thrombolytic Therapy After Cardiopulmonary Resuscitation In Acute St-Segment Elevation Myocardial Infarction: "Take It Or Leave It"

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INTRODUCTION: Patients with cardiac arrest can obtain recovery of spontaneous circulation (ROSC) after effective cardiopulmonary resuscitation (CPR). However, thrombolysis after CPR in acute STEMI is still controversial, and guidelines do not consistently recommend it because of the risk of bleeding.
CASE PRESENTATION: We report a 70 years old female patient who came to the emergency room with complaints of epigastric pain, nausea, vomiting and cold sweat, since 1 hour before admission. The ECG
shows a junctional rhythm, with heart rate of 47 beats/min, and ST segment elevation in leads II, III, aVF. Immediately after the ECG recording, the patient suddenly became unconscious and had no pulse. The ECG monitor showing ventricular fibrillation and CPR was performed immediately with defibrillation according to the ACLS algorithm. Intubation was also performed to maintain the airway and ROSC was achieved in less than 10 minutes after CPR. The patient was immediately transferred to the ICCU and given thrombolytic therapy using streptokinase. After thrombolysis, the ST segment gradually decrease and the rhythm return into sinus rhythm. The following day, the patient's condition gradually improved and the ventilator was removed. During hospitalization, there were no bleeding complications or recurrent cardiac arrest.
DISCUSSION: Thrombolysis has become the first-choice treatment of acute myocardial infarction in non- PCI centers. However, the increased risk of bleeding makes CPR a relative contraindication for thrombolytic therapy, especially for prolonged CPR. In this case, CPR was immediately performed after cardiac arrest and ROSC was achieved in less than 10 minutes. Thrombolysis was successful without any bleeding
complications. Rapid thrombolysis will result in a better prognosis if ROSC can be achieved within 10 minutes.
CONCLUSION: In patients with cardiac arrest due to STEMI, it may be acceptable to use thrombolysis as a reperfusion strategy. This applies especially in hospitals where Primary PCI is not available.
Keywords: *Thrombolytic; Cardiopulmonary Resuscitation; Cardiac Arrest; Bleeding; STEMI*

Diffuse “Shark Fin” St Elevation In Elderly Woman With Suspected Perimyocarditis Mimicking Acute Myocardial Infarction: A Case Report

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INTRODUCTION: "Shark Fin" ST elevation is often a sign of severe transmural ischemia resulting from acute coronary occlusion. Although this pattern is usually associated with acute myocardial infarction, this condition indicates ongoing myocardial injury that can be caused by a variety of etiologies. Perimyocarditis often causes symptoms, such as chest pain, ST segment elevation and elevated cardiac enzymes.
CASE PRESENTATION: We report a 64-year-old female who was referred from a private hospital with complaints of nausea, vomiting and chest pain. ECG showed diffuse ‘shark fin pattern’ ST-elevation in leads V1-V6, II, and aVF. Laboratory tests revealed leukocytosis (AL 31,900), renal insufficiency (creatinin 9.0 mg/dL), potassium 5.9 mEq/L, and positive troponin I. Based on the symptom, ECG changes and positif cardiac enzymes, a diagnosis of acute extensive anterior et inferior STEMI was made with perimyocarditis as a differential diagnosis. The patient refused to be referred, so it was decided to do thrombolysis. After thrombolysis, the chest pain was slightly resolved without a significant decrease in ST-segment elevation. The patient was admitted to the ICCU and received unfractionated heparin and also anti-inflammatory therapy. On the 3rd day of treatment, the ECG changed dramatically, where the ‘shark-fin’ ST-elevation disappeared, without any Q wave formation or T waves changes. Transthoracic echocardiogram showed thickened heart wall with mild global hypokinetic (EF 52%) and minimal pericardial effusion.
DISCUSSION: This case demonstrates that perimyocarditis can present with chest pain, ECG changes and elevated cardiac enzymes that mimic acute myocardial infarction. Coronary angiography and cardiovascular magnetic resonance (CMR) can be used to differentiate between STEMI and perimyocarditis. Even though the examination could not be performed in the patient, improvement in the ECG and patient's symptoms following anti-inflammatory therapy suggests an underlying inflammatory process and further supports the diagnosis of perimyocarditis.
CONCLUSION: Acute presentation of perimyocarditis could resemble STEMI, potentially misdirecting the therapeutic decision.
Keywords: Shark Fin ST Elevation; Perimyocarditis; STEMI

Wide Complex Tachycardia Due to Hypocalcaemia in Acute Heart Failure Patient: A Case Report

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INTRODUCTION: Hypocalcemia can cause ST segment modification and QT interval prolongation. In severe conditions, the predisposition will become life-threatening ventricular arrhythmias1.
CASE PRESENTATION: A 67-year-old woman presented to ER with shortness of breath. She had a prior history of CHF and CAD. Her blood pressure was 170/80 mmHg, HR 121 bpm, RR 24 rpm. The ECG showed sinus rhythm, LBBB, and LVH. Thorax X-ray showed acute lung edema and cardiomegaly. She was hospitalized with a furosemide pump of 5mg/hour and continued with a bolus of 20 mg daily on the next day. On the 4th day, the patient developed progressive dyspnea and palpitation. Her blood pressure was 80/60 mmHg, HR 140 bpm, RR 28 rpm, SpO2 95%. ECG evaluation showed wide complex tachycardia. She got amiodarone pump 150 mg for 6 hours and dobutamine pump 5 mg/hour. Laboratory examination found a decrease in ionized calcium levels from 4.4 to 3.3 mg/dL. Bolus 2 gr calcium gluconate was given immediately and then continued with oral administration of 1 g calcium carbonate per day. ECG at 24 hours showed sinus rhythm. The patient fully recovered and was dismissed in a medical ward after a few days.
DISCUSSION: Hypocalcaemia predisposes to arrhythmias by affecting both depolarisation and repolarisation of cardiac myocytes. Decreases in calcium allow increased sodium passage and lower the depolarization threshold, causing greater myocardial irritability2. Furosemide increases renal calcium excretion3. The patient in this case got a furosemide bolus with a prior history of prolonged use of furosemide because of CHF.
CONCLUSION: Severe hypocalcemia is not very common in heart failure and finding the true cause can be troublesome. Electrolyte monitoring should be carried out for patients receiving furosemide therapy.
Keywords: Widecomplextachycardia; Hypocalcemia; Acuteheartfailure

When is The Appropriate Time to Perform A Right-Sided Electrocardiography Examination on Syncope Patients?: An Emergency Case Report

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INTRODUCTION: Syncope is a symptom of transient loss of consciousness with numerous potential causes. Identifying and prioritizing life-threatening conditions is crucial to avoid a catastrophic outcome. Here we report a young smoker patient who presented with syncope as the manifestation of an acute right ventricular myocardial infarction (RVMI) and inferior wall myocardial infarction (IWMI).
CASE PRESENTATION: A 46-year-old man, presented to the emergency department (ED) with syncope and chest pain. On arrival at the ED, he stated not having had episodes of chest pain before. The physical and neurologic examinations were normal. Initially, a 12-lead ECG showed no abnormalities. Right-sided ECG was conducted, and there were ST elevations in deep Q wave and ST elevation in lead V3R until V5R, indicating RVMI. Repeated 12-lead ECG shows ST elevation in lead II, III, and avF. Treatment includes aspirin, heparin, clopidogrel, and thrombolysis. The coronary angiogram after thrombolysis demonstrated stenosis of the proximal third of the right coronary artery. Syncope was assumed to be hypoperfusion of the sinus node artery arising from the right coronary artery, causing transient sinus node dysfunction.
DISCUSSION: Syncope could be the sole manifestation of RVMI which can develop with IWMI and be complicated by sinus node dysfunction. Sympathetic withdrawal is one of the syncope mechanisms in patients with acute MI. The younger person starts smoking, the higher risk for cardiovascular damage.
Syncope followed by one of the cardiovascular risk factors can be considered using Right-sided ECG. A 12- lead ECG monitoring is required in all patients with syncope conditions, even without typical symptoms of acute coronary syndrome or hemodynamic instability.
CONCLUSION: Syncope with suspected cardiovascular involvement, Right-sided ECG can be performed. Additionally, serial ECG monitoring and prolonged ED observation are necessary for syncopal patients with suspected cardiovascular causes to ensure heart safety, especially in healthcare facilities that do not have cardiac enzyme tests.
Keywords: Syncope; Acute myocardial infarction; ECG; Sick Sinus Syndrome; Emergency Department

Diagnostic Challenges in A Rare Case of Simultaneous/Synchronous Cardio-Cerebral Infarction with Right Bundle Branch Block as the Masquerade: A Case Report

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INTRODUCTION: Concurrent cardio-cerebral-infarction(CCI), a rare condition defined as simultaneous occlusions in the cerebrovascular and coronary vessels, has high-mortality-rate but limited literature. We aim to present a challenging case of CCI which the ECG-features of Myocardial-Infarction were masked by bundle-branch-block(BBB).
CASE PRESENTATION: A 59-year-old male, referred to Emergency-Department with acute-onset left- sided-weakness of 2-hours'-duration before admission. Additional-complaint was dizziness and denied any chest-discomfort, palpitations or dyspnea. Past-medical-history was significant for Coronary-Artery-Disease in 2022, for which he refused PCI at-that-moment, moreover he was no under any routine-medications. ECG showed incomplete-RBBB with OMI-inferior. Laboratory-workups were within normal-limits, whereas radiology-exam showed cardiomegaly-pulmonary edema and acute-stroke large-vessel-occlusion. However, while doing cerebral-CT-Angiography, he had diaphoresis, dyspnea and increased work-of-breathing. ECG was immediately-repeated, nevertheless the result showed similar-images as the first-ECG, then the-patient was planned for serial-ECG. Surprisingly, on the 4thserial-ECG, there were differences in ST-segment and later Troponin-T showed elevation (332ng/L). The patient was assessed with NSTEMI and ischemic stroke (Simultaneous-Cardio-Cerebral-Infarction).
DISCUSSION: The patient's-characteristics in our hospital is similar with existing literature, which stated that the mean-age-specific rates was 59 –y/o with predominantly in men(90%). Life-threatening-MI might be missed when BBB is present. According to previous study, RBBB can mask minor ST-segment. New onset RBBB is likely caused by proximal occlusion of the LAD. Ibekwe et al, stated 4 major pathophysiologic mechanisms of CCI and the most likely underlying mechanism in our patient is due to cardio-embolic. While myocardial infarction and stroke share similar pathophysiology, their treatment options vary greatly. From several previous studies, proposed to administer IV-tPA(0.9mg/kgBB) then proceed to Percutaneous- Transluminal-Coronary-Angioplasty-and-Stenting(PTCAS) if indicated for ACS.
CONCLUSION: Cardio-cerebral-infarction is uncommon and rare with devastating clinical scenario; therefore, further research is still opened to be done. In addition, when the ECG-changes, clinicians need to pay attention to—and neither underestimate nor ignore—the presence of RBBB because it may contribute to life-threatening occlusion of the left-coronary-artery.
Keywords: Cardio-Cerebral; Infarction; RBBB; Emboli

Congestive Heart Failure Due To An Intracardiac "Yo-Yo Ball": A Case Report

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INTRODUCTION: Cardiac myxoma is the most common primary tumour of the heart. This tumor is rare but mostly benign, which has a stalk attached to the heart wall, like a yo-yo ball. Local obstruction to atrial outflow can present with dyspnea, orthopnea, syncope, and sudden death in rarely case. We report a case of congestive heart failure in a female due to a Left Atrial Myxoma.
CASE PRESENTATION: We present a case of a 56-year-old female who presented with a history of congestive heart failure. The patient came to the heart clinic of Cilacap General Hospital complaining of palpitation, shortness of breath and paroxysmal nocturnal dyspnea. ECG examination showed sinus tachycardia with heart rate of 120 bpm. From the echocardiography examination, a yo-yo ball-like appearance was obtained in the left atrium (diameter 33 x 50 mm). The mass attached to the interatrial septum, moving to and fro into the left atrium cavity and sometimes protruding into the left ventricle across the mitral valve. Mild to moderate mitral valve regurgitation, moderate tricuspid valve regurgitation and pericardial effusion were also found. She was given standard treatment for heart failure and then immediately referred to Dr. Sardjito General Hospital for surgical resection.
DISCUSSION: The clinical presentation of cardiac myxoma varies, from asymptomatic to a classic triad of symptoms, including obstructive, embolic, and constitutional symptoms. Atrial myxoma can interfere the mitral valve function, causing regurgitation or functional stenosis. Myxoma can also affect the myocardium directly, which may lead to myocardial dysfunction, arrhythmias, or pericardial effusion.
CONCLUSION: Primary cardiac tumors are rare and mostly benign, with 50% of cases being cardiac myxoma. Obstruction of blood flow in the heart will cause symptoms of heart failure. Surgical resection is the treatment of choice in cases of cardiac myxoma.
Keywords: *Cardiac Myxoma; Heart Failure; Echocardiography; Obstruction; Surgical Resection*

A Neglected Ventricular Septal Defect (VSD) Case Developing to An Eisenmenger's Syndrome Coincide with The Left Ventricular (LV) Thrombus in Child Having Down Syndrome : What is The Missing Piece?

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INTRODUCTION: Down syndrome (DS) is a chromosomal abnormality that most common present with 16 per 10000 live births. These children with DS are likely to have congenital heart disease (CHD) as much 40 to 50 times.
CASE PRESENTATION: A male patient aged 15 years old came to the emergency department (ED) as the parents complained worsened dyspnea since 3 days accompanied by cough and bluish of fingers and mouth observed 7 days before the chief complaint. It is known from the anamnesis that patient has cardiovascular problem, been consulted to type A hospital and had been planed to be operated. Because of the dental problem, the management planning discontinued by the parents. The physical examination revealed decreased oxygen saturation 67% per room air, clubbing fingers, pansystolic murmur at the apex and rales. Electrocardiogram (ECG) showed right axis deviation, pulmonale P and hypertrophy of LV, from the echocardiography we obtained 14-16 mm perimembranous VSD with right-to-left (R-L) shunt with LV thrombus, ejection fraction (EF) was 40%. The patient treated with diuretics, prostaglandin I2 (PGI2) and heparin.
DISCUSSION: The second most common CHD in DS is VSD in most study, VSD results in left-to-right (L- R) shunt with extra pulmonary blood flow. The chronic L-R shunt can elevate the pulmonary vascular resistance which leads to a reversed shunt and causing the systemic cyanosis, moreover the children with DS are at risk for about 20-fold to having pulmonary hypertension (PH). Furthermore, the R-L shunt directing to a hyper-viscosity and blood stasis that activates the coagulation. No therapies could reverse the syndrome, though pulmonary vasodilator provide the symptoms relief.
CONCLUSION: It was irony that the parents ceased treatment plan for about 7 years because they seen that the patient have no complaints for his condition. It disclosed to all health care providers that education and follow up are mandatory.
Keywords: Down syndrome ; congenital heart disease ; VSD ; eisenmenger's syndrome

Peripartum Cardiomyopathy and Massive Tranfusion due to Post Partum Haemorrhage: was it associated each other?

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INTRODUCTION: Peripartum cardiomyopathy (PPCM) is a life-threatening pregnancy-associated disease marked by LV dysfunction and heart failure (HF). Clinical findings of HF are often masked by the normal physiological changes seen in pregnancy making the diagnosis challenging. Furthermore, postpartum hemorrhage followed by massive blood transfusion may masked the diagnosis of PPCM or worsen the decompensated HF.
CASE PRESENTATION: a 35-year-old postpartum gemelli woman with history of massive postpartum hemorrhage due to atonia uteri and Disseminated Intravascular Coagulation complained shortness of breath after received massive blood transfusion. Sign of PPCM was identified by chest x ray showed right pleural effusion and early lung edema. The echocardiography showed decrease LV systolic function with EF 41% pseudonormal diastolic function and global hypokinetic. She was diagnosed with PPCM, acute lung edema, pleural effusion, and pneumonia. Patient was treated with Furosemid continuous pump, Spironolactone, Bisoprolol, Valsartan. Her dyspnea greatly decrased with diuresis and antibiotic. She was discharged with HF medication continued.
DISCUSSION: The cause of Post partum haemorrhage with massive transfusion was uterine atony. Placenta abruption, uterine arterial embolization, and peripartum hysterectomy are associated with blood loss in delivery. In these situations, massive transfusions are usually required, and these conditions induce HF at the time of delivery1. There is very seldom a study explained PPCM causing by massive transfussion. Moriyama et al reporting peripartum cardiomyopathi related to DIC and hellp syndrome2. Monitoring before and 24 h after delivery is important because it's the period of the most prominent hemodynamic changes.
Understanding of physiological hemodynamic changes during pregnancy is importance for interpretation of hemodynamic parameters of PPCM6.
CONCLUSION: The presence of massive transfusion in patient with PPCM can be challenging in diagnostic of PPCM itself and also unpredictable course of decompensated HF in peripartum mothers. Due to its high mortality rate without proper treatment, prompt investigation is important in improving maternal survival.
Keywords: PPCM; transfusion; partum; haemorrhage; cardiomyopathy, DIC,

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

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INTRODUCTION: Paradoxical embolism stroke is rare and uncommon case. Patent Foramen Ovale (PFO) is a condition that foramen ovale remains open and mostly asymptomatic. In normal condition clot breaks free and travels to the right heart then continue to the lung, but in someone with a PFO, the clot could pass through the hole and cause stroke.
CASE PRESENTATION: A 20 y.o. male came to ER with chief complain of severe cefalgia, tremor at both hands and chest pain. Cardiac physical examination, chest X-Ray, and ECG within normal limits, infarct in bilateral thalamus and cerebral edema on head ct scan examination, PFO 3 mm from echocardiography.
Patient on antiplatelet therapy and planned to refer for PFO closure procedure when on outpatient clinic.
DISCUSSION: Paradoxical embolism stroke is rare and uncommon case. PFO is a condition that foramen ovale remains open, occurs in about 1 in 4 people, mostly asymptomatic and rarely requires treatments. It's usually univestigated unless a patient is having symptoms like severe cefalgia, TIA or stroke before age 55. Young patients who had a paradoxycal thromboemboli stroke, 30-40% had a PFO and seem to be at higher risk for recurrent stroke (as high as 15% per year). In adults, treatment for PFO is mainly indicated in a patient with strokes. Medical therapy includes antithrombotic medications (aspirin), percutaneous device closure of PFO is superior to medical therapy in reducing the risk for recurrence in patients less than 60 y.o. Although surgical approach pursued in the condition of PFO that is larger than 25 mm, failure of PFO device closure, difficult for percutaneous closure.
CONCLUSION: Paradoxycal embolism stroke is rare and uncommon in young adult patients, as a cardiologist we should think PFO as one of the possible etiology. A good examination to diagnose an optimal collaborative management can prevent secondary complication to the patient.
Keywords: Patent Foramen Ovale; paradoxial embolism stroke

Cardiac Involvement of Leptospirosis Presenting with Atrial Fibrillation

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INTRODUCTION: Cardiac involvement of leptospirosis is potentially fatal. The manifestations vary from arrhythmias, myopericarditis, cardiogenic shock even non-specific electrocardiographic abnormalities.
However, the pathophysiology is poorly understood and the extent of the issue is under-reported. We report a case of a fulminant leptospirosis with probable myopericarditis treated with NSAID and tapered low dose methylprednisolone.
CASE PRESENTATION: A 64-year-old male farmer came to emergency room complaining seven-day history of fever, dyspnea, and calf tenderness. He presented with icteric and conjunctival suffusion. On his fourth day of hospitalization, he developed stable atrial fibrillation with rapid ventricular response, with normal previous electrocardiography. His heart rate was 178 bpm, with 120/74 mmHg blood pressure on vasopressor, the oxygen saturation was 99% on nasal cannula. Amiodarone was administered, successfully converting the rhythm to sinus. Echocardiography revealed 63% ejection fraction, normal cardiac chamber size, and minimal pericardial effusion concluded possible acute pericarditis. Troponin I was recorded at 0.03 ng/dl, leptospirosis IgM was positive, accompanied with severe thrombocytopenia, leukocytosis, high level of transaminases, creatinine, and BUN. He received potassium diclofenac, tapered dose of intravenous methylprednisolone, antibiotics, dialysis, and supportive therapy.
DISCUSSION: Leptospirosis has biphasic clinical presentation. The initial days represents the septicemic phase. The immune phase, marked by antibody and cytokine production along with leptospires detected in the urine, is when multi organ dysfunction involved. Myopericarditis frequently appears during the immunogenic phase. Lymphocytes and plasma cells infiltration, petechial hemorrhages, mononuclear infiltration in the epicardium, pericardial effusions, and coronary arteritis were found in interstitial myocarditis during leptospirosis. Steroids have been hypothesized to enhance the chance of recovery by decrease myocardial injury and oedema through their anti-inflammatory role.
CONCLUSION: Cardiac involvement of leptospirosis should be investigated because its potential impact on the cardiovascular system. Further studies on combination of NSAID and low dose corticosteroid in fulminant leptospirosis with probable myopericarditis are needed.
Keywords: Atrial fibrillation; leptospirosis; myopericarditis