[PP-26]
Persistent Atrial Tachycardia Mimickly Like Atrioventicular Nodal Reentrant Tachycardia: A Case Report

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ABSTRACT

Atrial tachycardia are relatively rare and only comprise about 10% of all Supraventricular Tachycardia cases. Atrial tachycardia may be caused by digoxin toxicity, structural abnormalities such as atriotomy scars, catecholamines, heart failure, or chronic obstructive pulmonary disease.[1,3] A patient who is experiencing an Atrial Tachycardia usually characterized by rapid pulse rate and on 12-lead Electrocardiogram showed abnormal P morphology, inverted P in lide II, III, avf.[4] The Symptoms range from irritability , palpitations, shortness of breath, fatigue, chest pain and syncope.[3,5] In many cases, it is quite difficult to distinguish atrial tachycardia and others supraventricular tachycardia. Like in this case we report on a 57 year-old man with atrial tachycardia mimicking AV nodal reentry tachycardia, he has been suffering chest pain since 3 days before he came to our institution, he was also feeling fatique. From vital sign we found that the patient was having uncontrol hipertention, his electrocardiogram showed supraventricular tachycardia with heart rate was 187 beats per minute without clearly seen p wave. The tachycardia was diagnosed as persistent atrial tachycardia because this rhythm was suddenly back after the patient got anti-arrhythmic medication, amiodaron. We found this patient blood pressure was drop after he got infusion amidaron and also complained about chest pain, caused by that condition, the patient got fluid challenge with NaCl 0.9% 250cc and the patient's blood pressure back to the normal after the patient got the treatment. During the observation held in the emergency room, the patient's monitoring ECG showed the rhythm of patient's heart back to atrial tachycardia with heart beat around 160-180 beat per second, but other vital sign was in normal limit. The patient was taken care in ICCU in order to get intensive care and closed monitoring of hemodinamic.

Keywords: Atrial tachycardia; electrocardiogram; P wave morphologi; AV Nodal re-entry tachycardia; supravricular tachycardi

[PP-27]
Will Pulmonary Hypertension Protect the Worsening of Ventricular Septal Rupture? : A Case Report

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ABSTRACT

Introduction: Ventricular septal rupture (VSR) is a condition due to perforation of the interventricular septum, thus forming a shunt between the two ventricles. VSR is a rare but lethal complication of acute myocardial infarction (AMI). As acute reperfusion strategies for AMI have evolved, VSR has become increasingly rare. However, the mortality rate due to these complication is still very high.
Most of VSR will result in severe hemodynamic perturbation. Rupture causes left-to-right shunt accompanied by volume overload of the right ventricle, increased pulmonary blood flow, and secondary volume overload of the left atrium and left ventricle. The ongoing process will cause the left ventricular systolic function to decrease. The body will perform negative feedback by increasing systemic vascular resistance through peripheral vasoconstriction. Over time, the left ventricle may experience pump failure so the systolic pressure will decrease.
On the other hand, pulmonary hypertension (PH) plays an important role in increasing morbidity and mortality. In the case of VSR, the high pulmonary pressure was thought to be protective towards those hemodynamic consequences.
Case Illustration: 62-year-old active heavy smoker male was brought to the Emergency Room due to typical angina. He was diagnosed with acute anterior STEMI Killip III and fibrinolytic was administered, which came out failed. On the same day, his clinical and hemodynamic presentation was declined as VSR complication was found. Echocardiography confirmed 2-3 mm of bidirectional shunt VSR with high pulmonary pressure (estimated mPAP 40 mmHg). Patient was stabilized only using inotropic agent and discharged on the day 6 with optimal standard heart failure therapy. After 4 months of follow up, there was no worsening symptom or functional class.
Discussion: A survival rate of patients with VSR without surgical intervention is very low. Nevertheless, few cases had ever been reported to survive for more than 5 years. Rothfled et al in 1972 also ever reported a patient with AMI complicated by VSR who survived for up to 17 years due to high pulmonary artery resistance. The patient had eisenmenger syndrome with severe pulmonary hypertension. The left-to-right shunt had undergone reversion. However, this slight changes may reduce the RV overload, decreasing the left-to-right shunt, and minimizing exessive pulmonary blood flow. Thus prevent rapid deterioration of hemodynamic even without support from intra aortic balloon pump (IABP).
Summary: This is a case of acute STEMI with VSR that happened on prior PH patient. The PH shows its protection value by making slight changes that may reduce the RV overload, decreasing the left-to-right shunt, and minimizing exessive pulmonary blood flow. Because of this mechanism, this patient had more stable haemodynamic and thus better survival.

Keywords: Ventricular Septal Rupture; Pulmonary Hypertension; stable hemodynamic; survival.

[PP-28]
Role Of Low-Dose Dopamine In Management Therapy Of Heart Failure With Renal Dysfunction: A Case Series

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ABSTRACT

Introduction: *Dopamine is an inotropic agent that has vasodilatory effects at low doses. A "renal dose"* of dopamine is often used to increase renal blood flow and increase creatinine clearance; however, data on the magnitude of effect and site of action in patients with heart failure are rare. Decreased renal function during hospitalization in patients admitted for heart failure (HF) is common and has been associated with unfavorable outcomes, especially heart failure in patients accompanied by renal failure.

Case I: A 70 years old woman, was hospitalize in ICU room with chronic heart failure (CHF) and chronic kidney disease (CKD) post hemodialysis. The chief complaint was dispneu, vital sign show BP: 130/80mmhg HR: 85bpm RR: 30x/m, blood test day 1 post hemodialysis showed creatinine: 2,86mg/dL. Monitoring urine output obtain result 1500cc/day (after being given furosemide 40mg). Dopamine 1mcg/kg/m added to this patient. Blood test day 2 showed creatinine: 1,66 mg/dL and urine output result 2400cc/day.

Case II: A 73 years old man, was admitted to ED room with chief complaint of chest pain and breathless since one day before hospital admission. Patient was diagnosed with CHF e.c old myocard infarct. Blood test day 1 showed creatinine: 2,1mg/dL and urine output result 800cc/day (after being given furosemide 40mg). Dopamine 1mcg/kg/m added to this patient. Blood test day 2 showed creatinine: 1,9mg/dL and urine output result 1200cc/day.

Case III: A 35 years old woman, was diagnosed with CHF with CKD. Blood test day 1 showed creatinine: 30,8mg/dL and urine output result 7200cc/day (after being given furosemide 40mg). Dopamine 1mcg/kg/m added to this patient. Blood test day 2 showed creatinine: 20,1mg/dL and urine output result 4200cc/day.

Discussion: Dopamine is associated with an increase in renal blood flow in patients with heart failure. This effect is due to dilation of both the large conductance and small resistance renal blood vessels. In the first case showed that the use of dopamine with a dose of 1mcg/kg/m has an effect on increasing urine output and decreasing creatinine blood levels. In the second case showed that the use of dopamine did not show significant results in a decreasing creatinine blood levels but showed an increase in urinary output. The third case did not show increasing of urinary output but significantly dereasing a creatinine blood levels.

Conclusion: Low doses of dopamine in this study still have an effect on patient with heart failure accompanied with decrease of renal function.

Keywords: Heart failure; low-dose-dopamine; renal dysfunction

[PP-29]
Libman-Sacks Endocarditis: Case Report of 38 Year-Old Female with Systemic Lupus Erythematosus

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ABSTRACT

Background : In negative blood culture infective endocarditis, one should be considered is Libman-Sacks endocarditis. It is cardiac presentation of systemic lupus erythematosus (SLE) which characterized by sterile vegetation and no sign of bacterial infection. We report a case who was firstly diagnosed as infective endocarditis but after several investigation she was diagnosed as Libman-Sacks endocarditis.
Case : A 38-year old female admitted to our hospital with chief complain of dyspnea and from echocardiography result was found vegetation on anterior mitral leaflet, severe mitral regurgitation and valve thickening. She had been diagnosed as definite infective endocarditis that fulfilled 1 major criteria and 4 minor criteria of Modified Duke's criteria for infective endocarditis. She got empirical antibiotics for her disease but showed no responses to the therapy. Investigation of other possibilities was conducted. One we thought was autoimmune disease such as SLE. Based on American College of Rheumatology criteria, this patient had 7 signs and ANA test positive. Other data strongly tend to be definite SLE. CRP was normal, blood culture was negative, procalsitonin was normal that indicates no sign of bacterial infection. Then patient was given steroid, immunosupresant agent and also antibiotic, she improved well and discharge after 15 days hospitalization. After 6 weeks treatment of steroid and immunosupresant, her clinical symptoms and echocardiography evaluation result were satisfying.
Conclusion : It highlights the challenges in clinically differentiating infective endocarditis and Libman-Sacks endocarditis. Prompt diagnosis and treatment may improve symptoms and quality of life.

Keywords : Libman-Sacks endocarditis; SLE; autoimmune; vegetation; mitral regurgitation

[PP-30]
Aortic Regurgitation in Patients with Tetralogy of Fallot

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ABSTRACT

Background : Tetralogy of Fallot (ToF) is one of the most common congenital heart disorders (CHDs). This condition is classified as a cyanotic heart disorder beacause tetralogy of fallot results in an inadequate flow of blood to the lungs (right- to - left shunt). Tetralogy of fallots results from a single developmental defect : an abnormal anterior and cephalad displacement of the infundibular (outflow tract) portion of the interventricular system . As a consequence , four anomalies arise : (1) a VSD caused by malalignment of interventricular septum, (2) subvalvular pulmonic stenosis because of obstruction from the infundibular septum, (3) an overriding aorta that receives blood from both ventricles, (4) right ventricular hypertrophy owing to the high pressure load placed on the RV by the pulmonic stenosis. Tetralogy of fallot occurs in 5 of 10,000 live births, and is often associated with other cardiac defects , including a right – sided aortic arch (25% of patients), ASD (10% of patients), and less often, anomalous origin of the left coronary artery. In ToF, the overriding aorta results in long – standing volume overload of the ascending aorta. It may cause aortic root dilatation and develops an aortic regurgitation. Aortic regurgitation is an abnormality in which blood from the aorta regurgitates into the left ventricle during diastole. If it is left untreated , severe acute AR causes a greatly increased Left ventricular – end – diastolic pressure leading to elevated left atrial and pulmonary pressure with resulting pulmonary edema, as well as decreased coronary perfusion gradients that potentially can cause myocardial ischemia and even sudden cardiac death.
Case : A 15 – year – old boy came to the emergency room due to cyanotic spells. It has already happened since he was a little kid. This got worsened often when he got tired and as a result, his lips and finger tips turned cyanotic. He also had a history of yellowish productive cough for about one week. This often happened recurrently with either clear or coloured sputum. His parents only took him to alternative medicine practice and bought over - the - counter medications. They only went to see the real doctor once. His respiratory rate during admission was 22 times per minute, the body temperature was $36,5^0$C, and other vital signs were normal. The physical examination showed clubbing fingers, cyanotic lips, tongue, and fingertips . The cardiac examinations showed 3/6 systolic murmur at the left sternal border, 3 / 6 systolic murmur at the tricuspid region, 3/6 systolic murmur at the pulmonic region, and 3/4 diastolic murmur at the aortic region . The ECG findings showed sinus rhytm, Right axis deviation, and right ventricle hypertrophy.The X- ray showed cardiomegaly and the echocardiography examination concluded Tetralogy of Fallot , MAPCAs, Moderate Aortic Regurgitation, and Mild Tricuspid Regurgitation. The laboratory finding showed leukocytosis. Based on all the examinations the diagnosis was Tetralogy of Fallot.
Conclusion : Clinicians should be aware that aortic regurgitation potentially occurs in patients with Tetralogy of Fallot. It is essential to make an early diagnosis in order to treat the disease and prevent further complications.

Keywords : Cyanotic Congenital Heart Disorders; Tetralogy of Fallot; Aortic Regurgitation

[PP-31]
Arterial Switch Operation in Childhood for the Patients with Transposition of Great Arteries

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ABSTRACT

Introduction: Transposition of great arteries (TGA) is the most common cause of cyanosis in neonates. In this malformation, ventricles are connected with transposed great-arteries; the aorta arises from the right ventricle and the pulmonary artery arises from the left ventricle (LV) with or without ventricular septal defect (VSD). Early detection and surgical management of TGA must be performed due to the high mortality rate if not treated properly. This case report aims to discuss the timing of surgery and clinical outcomes of patients with TGA—with or without VSD—undergoing arterial switch operation (ASO) in childhood.
Case Presentation: The first case is a 3-year-old boy with TGA, intact interventricular-septum (IVS), atrial septal defect (ASD), and pulmonary hypertension (PH). The second case is a 2-year-5-month-old boy with TGA, VSD, and PH. ASO and surgical corrections for other cardiac malformations were performed for both patients and resulted in different clinical outcomes. In the first case, the post-surgical outcome was good, no complication was found. In the second case, there were complications i.e. persistent PH, episodes of PH crises, and prolonged pleural effusion.
Discussion: In TGA, connection between systemic- and pulmonary-circulation is a *sine-qua-non*. Both patients have adequate connection (large ASD and VSD), so they can survive. ASO is the procedure of choice to completely repair TGA due to its good long-term outcome and is ideally done in the first month of life. Post-operative risk for TGA-with-intact-IVS increases along with the reduction of LV function. For TGA with VSD, the best time for performing ASO and VSD closure is < 3 month of life. Difference in outcome is determined by duration of exposure to the pulmonary blood flow.
Conclusion: We have reported two cases of TGA with PH accompanied by different cardiac-malformation. Both patients underwent ASO resulting in different post-surgical clinical outcome.

Keywords: transposition of great arteries; management; surgery; arterial switch operation; post-surgical outcome

[PP-32]
Acute Iatrogenic Cardiac Tamponade After Temporary Pacemaker Reposition

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ABSTRACT

Introduction: Cardiac tamponade is a life-threatening emergency condition, defined as an immediate or slow compression of the heart caused by an abnormally increased pericardial fluid in the pericardial sac. Iatrogeny is becoming one of the main causes of cardiac tamponade, because of the increasing development of invasive procedures, together with the use of potent antithrombotic drugs. Cardiac tamponade due to implantation of a pacemaker is a relatively rare complication, occurred only around 0.1 - 1.1% of cases for pacemakers. Cardiac tamponade is not only caused by pacemaker lead causing perforation of the myocardial wall, but also caused by the extraction of a pacemaker lead. Beck's triad is a classic presentation of patients with cardiac tamponade. But Beck's triad rarely appears simultaneously, and usually only appears shortly before cardiac arrest.
Case Presentation: An underweight 61-year-old male with STEMI inferior et dextra KILLIP I TIMI Risk 2/14 Grace Score 119 onset of 12 hours with total AV-block. Patient was successfully performed Primary PCI and **T**emporary **P**ace**m**aker (TPM) implantation. But at the third day of treatment in the ICCU room, patient suffered acute cardiac tamponade complications after TPM repositioning. Therefore, patient had bradycardia and **P**ulseless **E**lectrical **A**ctivity (PEA), and then CPR was performed with simultaneous emergency pericardiocentesis. The patient was discharge from the ICCU three days later with stable hemodynamic conditions without significant complaints.
Discussion: A patient with a rapidly accumulating pericardial effusion may present with severe respiratory distress, agitation, tachycardia, and hypotension, followed by quick progression to obtundation, bradycardia, and PEA. The risk factors for perforation in the intracardiac processes are fixation of active electrodes (especially helical type), transvenous pacing along with corticosteroid therapy or anticoagulants, female and **B**ody **M**ass **I**ndex (BMI) below 20 Kg/m2. Acute pericardial fluid collection is not detected on chest X-ray unless more than 200 ml of fluid has accumulated. There is no absolute contraindication to pericardiocentesis in cardiac tamponade patients with unstable conditions where emergency pericardiocentesis can save patient's life. The risk of heart perforation due to lead TPM may be prevented by placing lead on the RV septal wall rather than the apex wall or RV free wall.
Conclusion: Cardiac tamponade due to complications of TPM repositioning is a life-threatening emergency. Being familiar with the symptoms and early detection of cardiac tamponade is important to save patient's life. The risk of cardiac tamponade may be prevented by pacing the RV septum rather than the apex or free wall.

Keywords: Cardiac tamponade; temporary pacemaker; pericardiocentesis

[PP-33]
Giant T Wave Inversion in Emergency Departement : Cardiac or Non Cardiac

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ABSTRACT

Introduction: T wave is a period of ventricular repolarization on an electrocardiogram. T wave abnormalities on ECG often provide valuable clues to the pathology underlying heart disease or systemic syndrome. Giant T wave Inversions defined in 1979 by Yamaguchi et al as a negative T wave with an amplitude of more than 10 mm (1 mV) has been associated with a variety of clinical conditions.
Case: A 59-year-old female patient came to the Emergecy Room with headache, spinning and felt powerless. The patient had syncope at home about 1 hour before being taken to the hospital. The patient had experience partial seizure throughout the body for about 2 minutes before syncope. The patient experiences syncope for approximately 10 minutes. The patient did not have a history of previous similar illnesses, no history of hypertension or hypotension. There is no history of hereditary disease. There are no similar complaints in the family. On physical examination, it was found *compos mentis* with stable hemodynamics, no signs for heart disease. Neither lateralization of cranial nerves nor limbs were obtained. From the ECG examination, the Giant T inverted captured in precordial leads V1-V6. Patient was managed with the Transcient Loss of Consciousness and then observed in the regular ward.
Discussion: Overview of the above case TLoC with ECG picture Giant T inverted V1-V6 is often difficult to distinguish between cases of heart disease or neurologic disease. The giant T-wave inversion appears as a manifestation of ventricular repolarization abnormalities, and are associated with various clinical conditions such as myocardial infarction, pericarditis, hypertrophic cardiomyopathy, central nervous system diseases, electrolyte imbalance (potassium or calcium deficiency), LQTS, or drug effects. TLoC can occur due to an underlying cardiological problem. Referral for cardiovascular assessment within 24 hours is recommended if we found ECG abnormality, heart failure (history or physical signs), TLoC occurs during exercise, family history of sudden cardiac death in people aged <40 years and/or an inherited cardiac condition,new or unexplained breathlessness, a heart murmur. From some guidelines, it is stated that patients suspected of having TLoC with changes in the ECG picture should be sent to the cardiologist to determine whether the cause of TLoC is from heart disease or neurological disease. Some further examinations have been carried out and do not indicate the presence of heart disease.
Conclussion: Syncope which is accompanied by ECG changes is not always caused by heart disease. This can occur in neurological diseases or unexplained. Specific guidance is given for appropriate further examination, depending on the cause of the suspicion. In this case, Giant T wave inversion are most likely caused by neurological disease.

Keywords : Neurologic disease; Trancient Loss of Conciousness; Giant T wave inversion

[PP-34]
Cardiac Arrytmia and Pulmonary Edema in Thyroid Crisis Precipitated by Trauma :a Case Report – Diagnostic Challenge for Emergency Doctors

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ABSTRACT

Introduction: Thyroid crisis is acute life-threatening condition, representing extreme manifestation of thyrotoxicosis. This case is rare, about 1-2 % thyrotoxicosis manifest as thyroid crisis. The mortality rate is high, about 10 – 30%, mostly due to cardiorespiratory failure. Clinicians should be aware that thyroid crisis can be the first presentation of thyrotoxicosis, and precipitated by trauma.
Case presentation: We report a case of a patient with thyroid crisis exhibiting symptoms that similar with symptoms of traumatic injury. A 30-year-old motorcyclist woman was brought to our emergency department by motorcycle, after experiencing a traffic accident without wearing a helmet, with laceration and abrasion wound on her face and limb. She was agitated and diaphoretic. On admission, her vital signs are respiratory rates 45; pulse 166; temperature 37 °C, and blood pressure 170/100. Primary and secondary survey was done in suspicion of traumatic bleeding. Chest auscultation and x ray reveals pulmonary edema. Further examination reveals bilateral anterior neck mass. Her ECG record was showing atrial tachycardia. Burch Wartofsky score was 70, suggestive ongoing thyroid crisis.
Discussion: The diagnosis was delayed because similarities of symptoms between traumatic hypovolemic shock, and intracranial hemorrhage, but this patient was experiencing trauma-induced thyroid crisis. Treatment with anti-thyroid medication and cardiopulmonary support leads to great outcome. Management of pulmonary edema and heart rate control was important, to prevent mortality caused by cardiorespiratory failure. The use of diuretics and beta blocker in thyroid crisis should be monitored closely, especially patient with heart failure Killip 3-4 stages.
Conclusion: Careful history taking and physical examination play major role for every clinician who works in emergency department to diagnose thyroid crisis precipitated by trauma. Beta blocker could be use to minimize sympathomimetic symptoms, however caution should be taken in patients with decompensated heart failure.

Keywords: arrhythmia; thyroid; crisis; trauma; precipitated

[PP-35]
A Case Report of Anemia Induce ST Segment Changes in Acute Coronary Syndrome: Should we do aggressive treatment or not?

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ABSTRACT

According to the World Health Organization (WHO), anemia is defined as hemoglobin (Hb) levels <12.0 g/dL in women and <13.0 g/dL in men. Anemia is very common in congestive heart failure (CHF) and coronary heart disease (CHD) patients. Anemia in the setting of acute coronary syndrome (ACS) has an established adverse and poor prognostic value. Anemia has been shown to be an independent predictor of short and long term mortality. Its impact on outcome appears to be independent of underlying causative factors most often multiple – and time of onset. Anemia treatment strategies include red blood cell transfusions. We reported a case of Severe anemia in Patient with Acute Coronary Syndrome, which both of them can make ST Segment changes in ECG and also have a significant impact for the treatment. This case suggest the need of proper diagnosis and proper treatment in ACS, including adequate transfusion.

[PP-36]
Transient ST Elevation: Is It STEMI or NSTEMI?

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ABSTRACT

Acute myocardial infarction (AMI) is one of the leading causes of death worldwide. The mortality in AMI patients is influenced by many factors, among them is time delay to treatment. Prompt diagnosis by general practitioner as a first medical contact is important to reduce mortality. But, diagnosis of AMI could be a challenge. We report a case of atypical chest pain in DM patient and ECG showed transient ST elevation. By that, we think some differential diagnosis: NSTEMI with transient ST elevation or STEMI with a possibility of autolysis.

A 51-year-old male was referred from primary health care with inferior STEMI. He felt discomfort on epigastric region and his jaw 2,5 hours before. He has DM and he's smoking 1 pack per day. He had stable hemodynamic and normal physical examination. ECG taken in primary health care showed sinus bradycardia, HR 50 bpm, and ST elevation in inferior lead. ECG evaluation in hospital (taken 2 hours after) showed sinus rhythm, HR 68 bpm with no ST elevation. Laboratory result for troponin I, routine blood test, liver function, renal function and electrolyte were within normal limit. Our working diagnosis was NSTEMI with transient ST elevation, Killip class 1 with differential diagnosis of inferior STEMI. Patient was hospitalized and given tab clopidogrel 300 mg followed by 1x75 mg, aspirin 320 mg followed by 1x80 mg, fondaparinux 1x2,5 mg s.c for five days, atorvastatin 1x40 mg, captopril 3x12,5 mg. ECG evaluation the day after showed sinus rhythm, HR 55 bpm, and q wave in lead III, AvF. Troponin I evaluation showed an elevation >10mg/dL.

Thus, with the presence of evolution in ECG the final diagnosis of this patient was inferior STEMI, Killip class 1.

Keywords: Acute myocardial infarction; transient ST elevation

[PP-37]
Case Report: Post Myocardium Infarction Ventricular Septal Rupture in Hospital without Cardiothoracic Surgery Facility

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ABSTRACT

Background: Ventricular septal rupture (VSR) is a rare but lethal complication of acute myocardial infraction (AMI). The event occurs 2-8 days after an infarction and often precipitates cardiogenic shock. Maintaining hemodynamic status is crucial until definitive therapy is conducted.
Case report: A 75 year old male complained progressive shortness of breath since 5 days ago which was presented by epigastric pain at early onset. Therefore he was not diagnose on the time and VSR occured. In early physical examination, the blood pressure was 100/70 mmHg and the heart rate 116 beats/min. A 4/5 systolic murmur appeared on apex. ECG showed high elevation of ST segment in V2-V6 and pathological Q waves in V1-V5 both in precordial leads. Echocardiography showed obviously VSR. The patient was treated with diuretic, statin, antiplatelet, anticoagulan, beta blocker and angiotensin reseptor blocker (ARB). After ensuring that patient had a stable hemodynamic, than the patient was referred to the hospital which had catheterization laboratory facilities and cardiothoracic surgeon.
Discussion: The key to managing VSR is a fast and precise diagnosis, aggressive hemodynamic stabilization including oxygenation, medical therapy, mechanical, and angiographic.. In addition to medical therapy, Intra aortic baloon counter pulsation (IABP) was recommended to improve hemodynamic status before definite operation is taken. Coronary artery bypass grafting (CABG) is the treatment of choice. Therapy by closing the defect through transchateter as an alternative to therapy.

Keywords: ventricular septal rupture; post myocardium infarction; cardiothoracic surgery

[PP-38]
Congestive Heart Failure in Acute Rheumatic Fever patient with Severe Carditis

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ABSTRACT

Acute Rheumatic Fever (ARF) remains a major burden disease in developing countries including Indonesia. The annual incidence varies from <0.5/100.000 in highly developed countries to >100/100.000 in developing countries. The latest data revealed prevalence of ARF in Indonesia is 0,3-0,8 per 1000 school age children and high mortality rate that count 8,4% per year. The index of missdiagnosed ARF cases in health care providers is high due to various clinical presentation that can mimic other conditions. The missdiagnosed ARF cases lead to improper treatment of the first episode, that cause recurrences of ARF. Recurrences ARF develop into progressive valvular damage, with between 50-75% of cases progressing to Rheumatic Heart Disease (RHD) then arrhythmias and heart failure as the complications. The revised Jones criteria including the echocardiography in diagnosis of ARF carditis can reduce the missdiagnoses, but it is still a challenge for physician in developing countries to solve the lack of expertise and instruments for working up the diagnosis. We reported a 19-years old woman with congestive heart failure due to severe carditis as a clinical manifestation of ARF with 9-months follow up (november 2017-august 2018). She has a history of shortness of breath, intermittent fevers, fatigue, migratory joint pain, but no history of pharyngitis. The workups for her sign and symptoms revealed the diagnosis is an acute rheumatic fever with severe carditis which is have severe mitral valve regurgitation and pericardial effusion. Furthermore patient was treated with phenoxymethylpenicillin for secondary prophylaxis and she was started on salicylate acid and glucocorticoids for severe carditis. She was followed in a long term periods of follow up to evaluate the progressivity of the disease. In 9 months, the symptoms were disappeared, chest radiography presented cardiomegaly was back to the normal size, and the echocardiography revealed function of heart valves was improved.

Keywords : Acute Rheumatic Fever; Severe Carditis; Congestive Heart Failure

[PP-39]
Successful Vaginal Delivery in Primigravida with Uncorrected Pentalogy of Fallot

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ABSTRACT

Pentalogy of Fallot is a cyanotic congenital heart disease that has guarded prognosis without surgical intervention in infancy. Women with uncorrected defects rarely survive into childbearing age and pregnancy in this group is associated with a high rate of perinatal loss. Physiological cardiovascular changes in pregnancy can lead to maternal haemodynamic instability with subsequent adverse cardiac sequelae with or without fetal decompensation. Optimum management and pregnancy outcomes in mother with uncorrected Pentalogy of Fallot have not been more described in the literature. We describe a successful case of vaginal delivery an affected woman who has not undergone surgical repair. Her pregnancy progressed without any adverse cardiopulmonary complications. Her vaginal delivery and postpartum recovery were favourable, with successful birth of healthy babies at 40 weeks and 2 days. This case emphasises the importance of a multidisciplinary team, especially of obstetricians with expertise in high-risk pregnancies, adult congenital heart disease cardiologists and anaesthesiologist.

Keywords: Pentalogy of Fallot; vaginal delivery; full term pregnancy

[PP-40]
Dilated Cardiomyopathy in Children with Severe Cogestif Heart Failure

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ABSTRACT

Dilated Cardiomyopathy (DCM) is the most common form of the cardiomyopathies in children among hiperthropic Cardiomyopathy and restrictive Cardiomyopathy that accounting for a significant cause of morbidity and mortality as well as a common indication for heart transplant. Although dilated cardiomyopathy is a common indication for Cardiac transplantation, there are still treatable causes of dilated cardiomyopathy for decrease the morbidity. The incidence of Dilated Cardiomyopathy in developed countries is 0.57–1.23 per 100,000 per year in children younger than 18 years an in Indonesia the prevalence are unknown. We reported a 6- years with the siymptomps are fatigue, weakness, shortness of breath, associated with progressive dyspnoea for almost 2 week and peripheral edema. She had no previous medical history. No family history of cardiomyopathy was reported. Chest radiography reveals cardiomegaly. Echocardiography showed global hypokinetic, dilatation Left Ventikel,. Left Ventikel Ejection Frection (LVeF) 30.4%. Furthermore patient was treated with diuretics, inotropic agent and ace-inhibitor. The Dilated Cardiomyopathy symptoms can be vague but improved prognosis and outcomes require prompt diagnosis and appropriate management

Keywords : Cardiomyopathy; Children; Cardiac failure; Cardiac transplantation

[PP-41]
Hyperthyroidism-Induced Cardiomyopathy

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ABSTRACT

Background: Thyroid hormones have a significant impact on cardiac function and structure. Excess thyroid hormone affects cardiovascular hemodynamics, leading to high-output heart failure and—in late stages—dilated cardiomyopathy, an uncommon, yet well recognized, manifestation of thyrotoxicosis.
Objectives: To present and discuss two cases of long-standing hyperthyroidism with unusual manifestation of heart failure with reduced ejection fraction, the possible underlying mechanism, and the management.
Case illustration: A 32-year-old woman and a 62-year-old man came to the emergency department of National Cardiovascular Center Harapan Kita, both with worsening heart failure and preceding signs and symptoms of hyperthyroidism. They were also found to have low ejection-fractions and clinical signs suggesting cardiomyopathy. The patients were then managed with heart failure treatments and antihyperthyroid drugs resulting in improvements of their clinical condition.
Summary: We have reported two cases of patients with obvious clinical sign and symptoms of heart failure and hyperthyroidism. It is very probably because prolonged exposure of high thyroid hormone levels to the heart that the manifestation of heart failure occurred in these patients. Therefore, evaluation of thyroid level is important because most of the hyperthyroidism cases are reversible.

Keywords: hyperthyroidism; cardiomyopathy; thyroid heart disease; heart failure; low ejection fraction

[PP-42]
Progressive Pulmonary Hypertension on a Teenager : A Case Report of Rapid Right Ventricular Failure in Newly Diagnosed Patient

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ABSTRACT

Background: Pulmonary hypertension (PH) is an incapacitating disease with poor survival and high economic burden. Right ventricular (RV) failure believed as one of the cause of death beside sudden death and extra cardiac events. Study has shown that newly diagnosed patients have higher risk of disease progression and worse prognosis. After the diagnosis recognised, the rapid deterioration of the RV may be the next threat the physician must resolve.
Case description: A 16 years old boy came to emergency room with shortness of breath several hours before admission. Physical examination revealed tachypnea, tachycardia, normal blood pressure with oxygen saturation 93%. There were gallop and bilateral ronchi on auscultation, ascites, hepatomegaly, clubbing finger and also leg edema. Electrocardiogram showed RV hypertrophy. Thorax X-ray result was cardiomegaly with cardiothoracic ratio (CTR) 70%. Laboratory result was anemia and hypoalbumin. Previously he had history of complete lung tuberculosis treatment. Approximately 2 months before the admission, he had admitted at the hospital twice with similar complain. Thorax X-ray 2 months before showed emphysematous lung with CTR 45 % then rapidly transformed into CTR 65% a month after. Echocardiography showed right atrium and RV dilatation, paradoxical interventricular septum, mild pulmonal valve regurgitation, severe tricuspid valve regurgitation suggesting high probability of PH. Diuretics, aldosterone antagonist, venodilator, and phosphodiesterase type 5 inhibitor were given to improve the condition. He was moved to ICU and then his condition was deteriorating rapidly until declared dead on the 11th day.
Conclusion: PH may cause devastating effect. The outcomes depend on the clinical evidence of RV failure, etiology, functional class, hemodynamic, biomarker, echocardiographic findings and progressivity. Not only an early diagnosis is needed but also a proper treatment to resolve the rapid progression of the disease.

Keywords: pulmonary hypertension; right ventricular failure; right ventricular dilatation

[PP-43]
Role of Cardiac-Ct Imaging in Characterization of Dextrocardia : A Case Report

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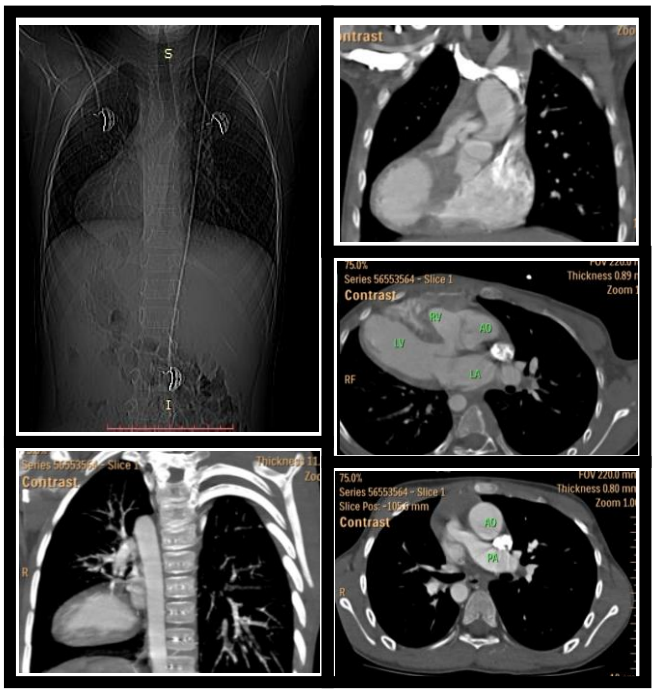
ABSTRACT

Dextrocardia is a cardiac congenital malposition which the major axis of the heart directed to the right and may associated with other congenital cardiact defect. Since it common to have more complex defect, it's necessary to analysis the structure of the heart in sequence through cardiac CT. Here we report a 9 years old boy, came with history of shortness of breath and dextrocardia according to physical examination. The echocardiography show dextrocardia mirror image, double outlet right ventricel, transposition great arteries, large VSD and PS severe. Because of his complex cardiac structure, cardiac-CT has been performed. The result showed dextrocardia with situs inversus totalis which is corresponded to visceral status. Sequence cardiac structure analysis was performed to identify any anomaly of the heart. Atrial septal defect and ventricular septal defect were observed. The connection of atrials and ventricles is concordancely related. However, the connection between ventricles and great arteries is discordant meaning left ventricle were connected to pulmonary artery while right ventricle is connected to aorta. Furthermore, none of any extracardiac shunt was found through examination. The current case is presented in order to remind the application of cardiac-CT imaging in patient with dextrocardia to have advance analysis for proper management.

Image:



[PP-44]
Is It Necessary? A Pacemaker For Total AV Block Case Report : A 56 Years Old Man with Acute Myocardial Infarction of Inferior Wall Killip I with Total AV Block

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ABSTRACT

The presence of atrioventricular (AV) block as a part of conduction defects complicating myocardial infarction (MI) is relatively frequent and is associated with increased short and long term mortality rates, and atrioventricular block is a common one in inferior MI1. In the thrombolytic era, the overall incidence was 6.9% and more than a half was a total AV block2.

A 56 years old man came to the emergency room (ER) with a chief complain of typical chest pain for angina onset 5 hours before admitted accompanied by sweating. From physical examination the blood pressure was 104/63mmHg, heart rate 42 beats per minute, respiration rate 20 times per minute and oxygen saturation was 99% in room air. There was no murmur from cor examination, no rales from pulmo examination, and there was no extremity edema. Electrocardiography (ECG) showed ST elevation at lead II, II, and aVF, total AV block with P-P rate 83 beats per minute and R-R rate 45 beats per minute. From the laboratory findings showed haemoglobin 14900 g/dl, hematocrite 43,6%, leucocyte 12500/ul, thrombocyte 399000/ul, ureum 21,40 mg/dl, creatinin 1,53mg/dl, random blood glucose 120mg/dl, sodium 137,9 mmol/L, potassium 4,35 mmol/L, chloride 105,9 mmol/L, and hs-troponin >40.0000mg/l. Working diagnose for this patient was acute myocardial infarction of inferior wall, killip I and total AV block. The patient was given oxygen, acetylsalicylic acid 320 mg, clopidogrel 300 mg, injection of atropine sulfate 0.5 mg, dopamin 5mcg/body weight/minute and continued with streptokinase 1.5 million units administered intravenous for 60 minutes, and it was a success. After 60 minutes of streptokinase there was a decreasing of ST segment elevation and no chest pain. The post streptokinase ECG showed a sinus rhytm 100 beats per minute and acute MI of inferior wall.

The blood supply to AV node and the inferoposterior surface of the heart share the same arterial territory, which was 90% by right coronary artery (RCA) and 10% by left circumflex (LCx)3. Irreversible AV node block due to necrosis associated with inferior MI that persist beyond 48-72 hours should raise the suspicion of damage to AV node4. As in this patient, total AV block complicating the inferior MI caused by an occlusion probably at RCA. After received fibrinolytic, the occlusion at RCA faded and it also recovered blood flow through the AV node converted the total AV block into sinus rhytm.

A 56 years old man with inferior MI killip I complicated by total AV block came to ER within window period for fibrinolytic therapy. After 60 minutes received streptokinase therapy it was met a success criteria. And how about the total AV block ? it was gone.

Keywords : Total AV Block; Acute Myocardial Infarction; Fibrinolytic; Pacemaker; Right Coronary Artery

Images:

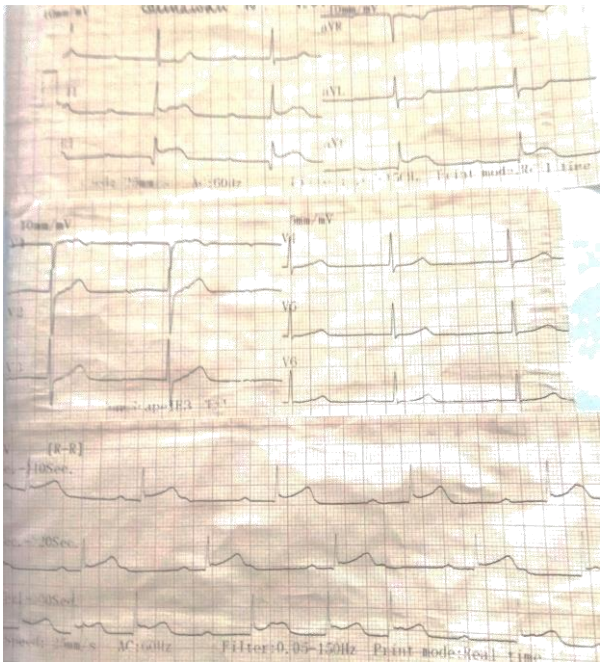

Figure 1. Pre Streptokinase ECG shows ST elevation of lead II, III, aVF and Total AV block P-P rate 83 bpm, R-R rate 45 bpm.

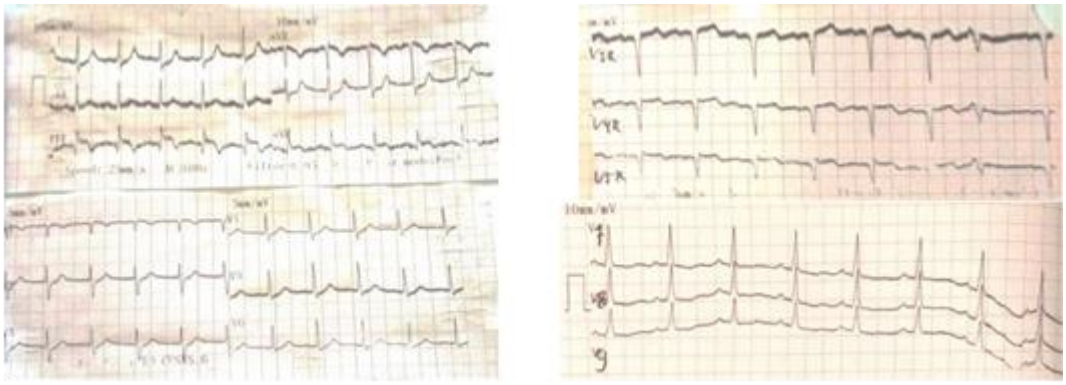

Figure 2. 60 minutes Post Streptokinase ECG shows Sinus Rhytm 100 bpm and ST elevation of lead II, III, aVF.