

## Henoch Schonlein Purpura and Auto-Immune Hemolytic Anemia Induced Cardiac Dilatation

A.P. Sunjaya<sup>1,\*</sup>, M. Galastri<sup>2</sup>

<sup>1</sup>Faculty of Medicine, Tarumanagara University Jakarta, Indonesia

<sup>2</sup>Department of Internal Medicine, Ciawi General Hospital, West Java, Indonesia

\*correspondence: anthony@doctors.web.id

### ABSTRACT

**Background:** Henoch Schonlein Purpura (HSP) is an uncommon leukocytoclastic vasculitis with over 90% of cases affecting children and only about 3.4 cases per million in adults. Cardiac involvement is not recognized as a feature of HSP with fewer than 15 cases reported in literature and mostly described to have fatal outcomes. We present an unusual case of HSP with autoimmune haemolytic anemia and cardiac dilatation wherein the patient was successful managed.

**Case report:** A 38 year old female presented to the emergency department with a 1 week history of progressive general weakness and nausea as well as intermittent fever over the past 4 months. This was accompanied by shortness of breath and dyspnoea on effort. Examination of the patient demonstrated conjunctival pallor, an enlarged heart boundary on percussion and a painless palpable purpuric eruption over the lower extremities. Blood work revealed anemia, normal thrombocyte levels, elevated erythrocyte sedimentation rate, elevated creatinine levels, hyponatremia and hypokalemia. Peripheral blood morphology showed hypochromic, anisocytosis, microcytic haemoglobin with ovalocyte, elliptocyte and tear drop cells. Further testing revealed a positive direct Coomb's Test. Chest X-Ray was performed on the patient which showed cardiomegaly and pulmonary congestion while electrocardiography did not find any remarkable findings. Further echocardiographic testing was performed which revealed left atrial dilatation, ejection fraction of 63%, E/A ratio >1, DT 257 and mild mitral regurgitation.

**Conclusion:** Though HSP primarily presents with abdominal pain and in the pediatric population, it must be considered among adults with purpuras especially in the extremities. Purpura biopsy and IgA blood test can be used to confirm the diagnosis. As HSP has been reported to be related to autoimmunity, patients with HSP may present with other autoimmune diseases such as AIHA in this patient. While uncommon, cardiac comorbidities among HSP patients must also be assessed as late treatment worsens prognosis. Further studies are required to determine the association and pathophysiologic mechanism underlying cardiac disorders with HSP.

**Keywords:** adult onset henoch schonlein purpura; autoimmune haemolytic anemia; cardiac dilatation

## Acute Myocardial Infarction with Non-Obstructive Coronary Arteries

F. Caesario\*, A. Wibisono

dr. Iskak General Hospital, Tulungagung, East Java, Indonesia

\*correspondence: fahrezacaesario@yahoo.com

### ABSTRACT

**Background:** ST-elevation Myocardial Infarction (STEMI) commonly occurs when a coronary artery becomes totally occluded by a blood-clot disrupting blood flow to the myocardium. However, there is a condition where the common etiology is not present, thus called Myocardial Infarction with Non-Obstructive Coronary Arteries (MINOCA).

**Case Description:** A forty-nine years old caucasian male came to emergency department with about two hour typical chest pain with cold sweating then diagnosed with anteroseptal STEMI. Patient had no history of chest pain, hypertension, diabetes, smoking cessation, or alcohol abuse, but there were some history of syncope and shortness of breath after exercise. Physical examination showed blood pressure 123/80, heart rate 97 beats per minute, heart thrill with a grade IV/VI crescendo-decrescendo systolic rumbling murmur that radiated to the carotis. The ECG showed significant ST elevation at aVR, V1-V2 with hyperacute T at V3-V4 and reciprocal at inferior lead, consistent with anteroseptal myocardial infarction. Laboratory testing showed cardiac Hs-Troponin I elevation at 4841,3 ng/L. In the cath-lab, coronary angiography showed normal coronary vessel with TIMI 3 flow, and the working diagnosis changed to MINOCA. Bedside echocardiographic examination showed AV jet velocity 4,5 m/s; AVA planimetry area 0,674 cm<sup>2</sup>; AV mean PG 51.9 mmHg, we assume that the elevation of ST segment and cardiac troponin is may from myocardial oxygen demand-supply diproportion due to coronary microvascular dysfunction from the aortic stenosis problem. The patient then treated conservatively with aspirin, atorvastatin, bisoprolol and stabilized for the chest pain, daily follow up showed resolving of ST elevation and the chest pain dissolved. After 4 days observation, patient out-hospitalized with planning of follow up with a non-invasive dobutamine-stress echocardiography test and education for aortic valve repairment. Unfortunately the patient decease before the test held.

**Conclusion:** MINOCA is a working diagnosis when there is no evidence of coronary artery obstruction in a clinically diagnosed STEMI patient. Further examination is required to define the main etiology. Stress-test echocardiography or stress-test CMR may be held if the suggestive origin is microvascular coronary dysfunction.

**Keywords:** STEMI; MINOCA; aortic stenosis

## Mitral Valve Repair Induced Hemolytic Anemia: A Case Report

A.P. Sunjaya<sup>1,\*</sup>, C. Tarigan<sup>2</sup>, C.I. Rahayu<sup>3</sup>

<sup>1</sup>Faculty of Medicine, Tarumanagara University, Jakarta, Indonesia

<sup>2</sup>Department of Internal Medicine, Ciawi General Hospital, West Java, Indonesia

<sup>3</sup>Department of Cardiology, Ciawi General Hospital, West Java, Indonesia

\*correspondence: anthonyjsy@yahoo.com

### ABSTRACT

**Background:** Haemolytic anemia is a rarely reported complication of mitral valve repair. Although occurring to a lesser degree than in patients with prosthetic mitral valve replacement, valvular repair also leads to the introduction of prosthetic material required for anuloplasty. As a result, following repair certain patterns of regurgitant flow disturbances associated with high shear stress may occur which is responsible for haemolysis.

**Case Illustration:** A 32-year old male presented with to the outpatient department with a 1-month history of progressive lethargy, jaundice and dyspnoea-on-effort. The patient had a prior history of mitral valve repair 2 years ago due to mitral valve regurgitation caused by rheumatic-heart-disease and without apparent complications post-surgery. Following surgery the patient was prescribed with blood-thinners but have stopped consuming them 3 months post-surgery due to non-compliance. He had a history of being admitted and receiving transfusion in a different hospital a year ago for anemia. Examination of the patient demonstrated scleral icterus, conjunctival pallor, grade 4/6 pansystolic murmur over the apex and an enlarged left-heart boundary on percussion. Blood work revealed anemia, negative direct Coomb's test and hyperbillirubinemia. Peripheral blood smear revealed poikilocytosis, spherocytosis and fragmented red blood cells. Patient was given packed-red-cell transfusion and referred to an interventional cardiologist wherein subsequently mitral valve repair was again performed.

**Conclusion:** Though a rare occurrence, haemolytic anemia must be taken into account in patients with a history mitral valve repair as haemolysis may emerge immediately or even years following mitral valve repair even in patients with mild regurgitation.

**Keywords:** mechanical haemolytic anemia; mitral valve repair

## **Acute Myocardial Infarction Complicated with Ventricular Septal Rupture and Cardiogenic Shock: A Case Report**

**M.A. Mukti<sup>1,\*</sup>, I.M. Loebis<sup>2</sup>, G.I. Hadiyat<sup>2</sup>**

<sup>1</sup>General Practitioner at Hasna Medika Heart Hospital, Cirebon, Indonesia

<sup>2</sup>Cardiologist at Hasna Medika Heart Hospital, Cirebon, Indonesia

\*correspondence: aliy\_dr@yahoo.com

### **ABSTRACT**

Ventricular Septal Rupture (VSR) is a rare but fatal complication of Acute Myocardial Infarction (AMI) with mortality that ranges from 41% to 80%. The Incidence of VSR after reperfusion therapies became the standard of practice in the treatment of AMI only 0.17-0.31%. We report a case of 62-year-old man came to ER complaining of typical chest pain since one day before. He was conscious but look in extreme pain. He has histories of hypertension and smoking 1 pack of cigarettes/day. Examination showed tachycardia, HR 105 beats/minute; BP 80/60 mmHg. ECG showed sinus tachycardia and ST segment elevation in V1-V4. Laboratory test showed increase Troponin result and Leukocytosis. Echocardiography showed akinetic anteroseptal wall, thin wall, and ventricular septal rupture at apical anteroseptal wall. LMWH and Inotropic drugs continue to delivered but patient couldn't make it in day 5 of care. As shown in the SHOCK trial and validated by GUSTO-I and APEX-AMI, VSR typically occurs much earlier ranging from 8-24 hours after AMI. The improved early detection of VSR may be the result of other factors including near universal access to Echocardiography and alterations in tissue pathology as a result of reperfusion injury combined with fibrinolysis. Current guidelines from ACCF/AHA recommend emergent surgical repair regardless of hemodynamic stability at the time of diagnosis. Improving early detection of VSR is important to determine complication of AMI and despite the timing of VSR repair and perioperative therapeutic management remains controversial, recommendation emergent surgical repair regardless of hemodynamic stability at the time of diagnosis is necessary.

**Keyword:** acute myocardial infarction; ventricular septal rupture; cardiogenic shock

## Readmission Following Primary Percutaneous Coronary Intervention for ST-Segment Elevation Myocardial Infarction

A. Kristyagita<sup>1,\*</sup>, B.B. Siswanto<sup>2</sup>, A. Sain<sup>2</sup>

<sup>1</sup>RSUD dr. M. Ashari, Pemalang, Indonesia

<sup>2</sup>Department of Cardiology and Vascular Medicine, Faculty of Medicine, Universitas Indonesia, Jakarta, Indonesia

\*correspondence: akristyagita@yahoo.com

### ABSTRACT

**Background:** Despite the increasing rate of primary PCI (PPCI), the prevalence of heart failure (HF) readmission is still high. Appropriate medications are strongly recommended in the post myocardial-infarction (MI) period. Previous controlled studies have shown that beta blocker can reduce HF mortality and readmission and is recommended as secondary prevention.

**Objectives:** To present a case of readmission following PPCI for ST-segment elevation MI (STEMI) due to the suboptimal post-procedural therapy.

**Case illustration:** A 49-year-old man came to emergency department (ED) of National Cardiovascular Center Harapan Kita (NCCHK) with the chief complaint of dyspnea a week after PPCI at previous hospital, accompanied by angina three days before admission. He was discharged from the previous hospital with suboptimal discharge-drugs. His CAD risk factor: hypertension and heavy-smoker. Physical examination revealed bibasilar rales of the lung. His ECG showed inverted T-waves in  $V_2$ - $V_6$ , as well as pathological Q-waves and slight ST-segment elevation in  $V_1$ - $V_6$ . His CXR showed cardiomegaly with congestive appearance. Laboratory findings showed hyponatremia and hypokalemia. The patient was diagnosed as ADHF due to old anterior MI, UAP, hypertension, hyponatremia, and hypokalemia. The discharge medications from the previous hospital did not include beta blocker.

**Discussion:** Readmission rate of HF is still high. An optimal medications post-MI using beta blocker and other guideline-directed drugs is needed to reduce HF mortality and readmission. Studies have shown that beta blocker can improve cardiac function, as well as reduce the HF symptoms. In this case, lack of appropriate medications made the patient readmitted.

**Summary:** A man was readmitted with ADHF and UAP after undergoing PPCI at another hospital without receiving beta blocker at discharge. The optimal HF medications—comprising diuretics, angiotensin converting enzyme (ACE) inhibitor, and beta blockers, with/without mineralocorticoid receptor antagonist—are mandatory, even after PPCI.

**Keywords:** heart failure; optimal medications; beta blocker; readmission; myocardial infarction

## Worsening AV Block Due To ST-Elevation Myocardial Infarction

I.V. Muhaka<sup>1,\*</sup>, A. Ardian<sup>2</sup>, Y. Restuti<sup>3</sup>

<sup>1</sup>General Practitioner of RSUD Tebet, Jakarta, Indonesia;

<sup>2</sup>Internist of RSUD Tebet, Jakarta, Indonesia

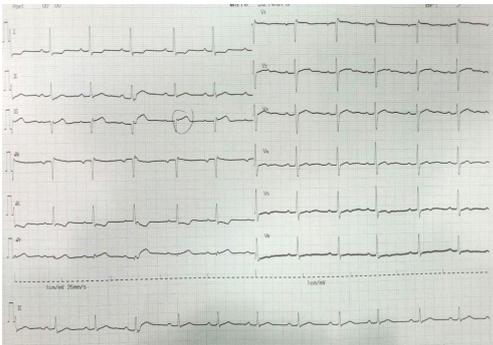
<sup>3</sup>RSUD Tebet, Jakarta, Indonesia.

correspondence: ivanvienozamuhaka@yahoo.com

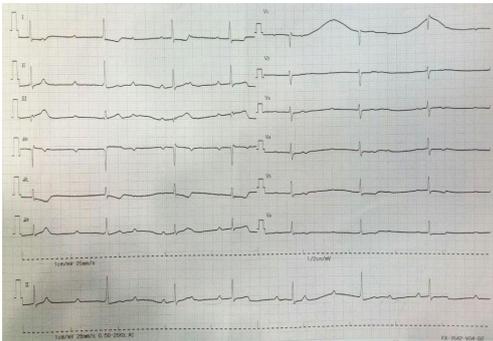
### ABSTRACT

**Introduction:** Acute myocardial infarction (AMI) happens due to occlusion of coronary arteries. One of its complications is AV block.

**Case presentation:** 51 y.o male, uncontrolled hypertension, came to ER with chest pain since 3 hours, radiating to back, cold sweating and nausea. VAS were 7/10. BP were 160/100. Physical examinations and laboratory test were normal but there was no facility for cardiac marker.



ECG showed first degree AV block and ST elevation on III and avF. Initial therapy were oxygen, DAPT, nitrate, ACEi, statin, and heparin.



Follow-up ECG showed TAVB. The Coroangiography at tertiary hospital showed 80 % stenosis at RCA. He was subjected to POBA PPCI. The patient has no symptoms with normal ECG until now.

**Discussion:** The AV block happens due to heart ischemic related to the stenotic location which he underwent from time to time and got worse when he had STEMI.

**Conclusion:** TAVB in this case is the complication of AMI.

**Keywords:** AMI; AV block; TAVB; PCI

## Coronary Artery Ectasia of Three Vessels in Patients with Stable Angina: A Rare Case Report

N.H. Karunawan<sup>1,\*</sup>, D.T.W. Nugroho<sup>2</sup>

<sup>1</sup>General Practitioner, Wahidin Sudiro Husodo Hospital, Mojokerto, Indonesia

<sup>2</sup>Cardiologist, Wahidin Sudiro Husodo Hospital, Mojokerto, Indonesia

\*correspondence: niyatahananta@yahoo.co.id

### ABSTRACT

**Introduction:** Coronary artery ectasia (CAE) is dilatation of an arterial segment to a diameter at least 1.5 times that of the adjacent normal coronary artery. Coronary artery ectasia (CAE) is rare case, Its prevalence ranges from 1.2%-4.9%. Clinical presentation and management of CAE are not well defined due to limited experience.

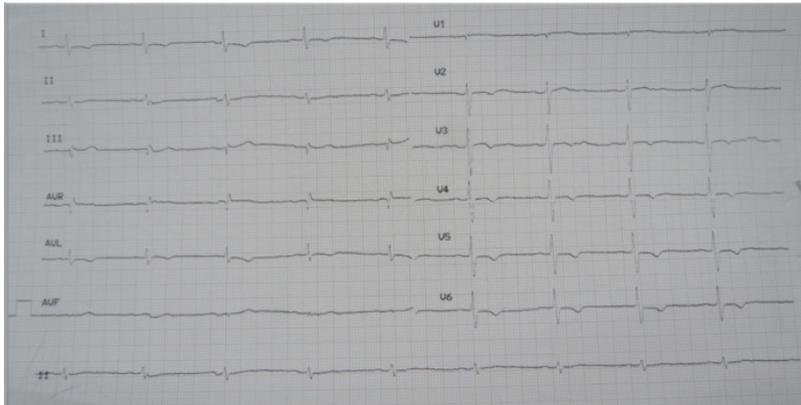
**Case Report:** A 54 year-old man was presented with history of NSTEMI which was managed with medical treatment in previous hospital. Patients had complain with symptom of chest pain, which was exacerbated in activity and recovered with rest. The Patient had history of syncope third time when he standing too long. Patient was diagnosed NSTEMI 3 month ago and was planned coronary angiography. Cardiovascular risk factors of patient were found hypertension, smoking, and obesity. Blood pressure 170/80, heart rate 84 bpm, respiration rate 22 SpO<sub>2</sub> 98%. On Physical examination was normal. ECG showed T inversion in lead V1-V6, I, aVL and Rontgen thorax was normal. Coronary angiography showed coronary artery ectasia of RCA, LCX, LAD with slow flow (TIMI-2). Patient was given Valsartan 1x160mg, Nifedipine-GITS 1x30mg, HCT 1x2.5 mg, Trimetazidine-Dihydrochloride 1x1, Aspirin 1x100mg, Clopidogrel 1x75 mg.

**Discussion:** The cause of CAE is predominantly atherosclerotic, other causes include kawasaki disease, infection, autoimmune disease, dissection, trauma, congenital malformation, and idiopathic. CAE is more common in males. Hypertension and smoking is risk factor. The majority of the patients with CAE are asymptomatic but CAE may present with angina pectoris stable, myocardial infarction, sudden death or complications. CAE may be detected by coronary angiography, computed tomography, magnetic resonance imaging but coronary angiography remains the gold standar. Our patient presented with chest pain, a diagnostic coronary angiography was done straightaway. There is still no guideline for management of CAE. Treatment options include risk factor modifications for coronary artery disease (CAD), antiischemic therapy, anti-platelet, chronic anticoagulation, percutaneous and/or surgical coronary vascularisation techniques.

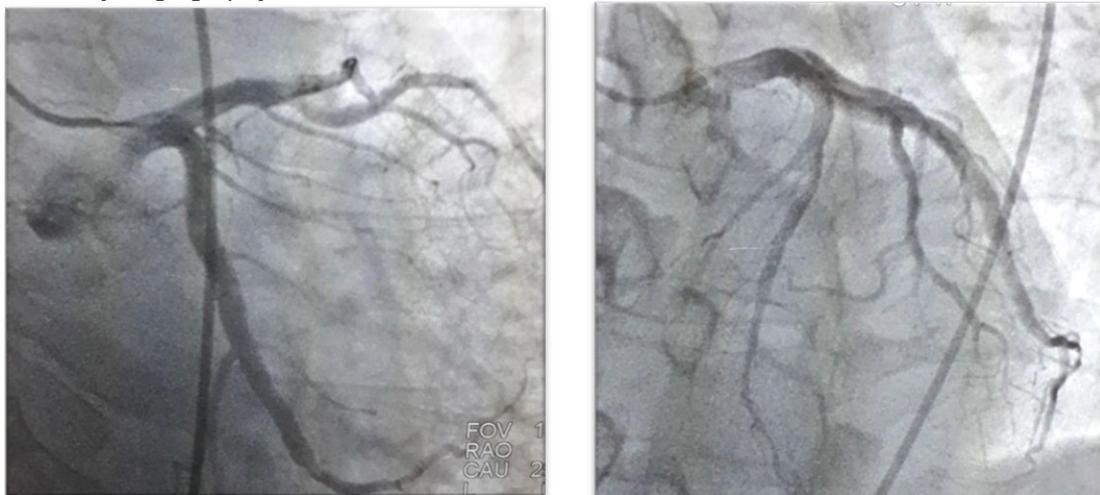
**Conclusion:** Stable angina is the most common presentation in patients with CAE. The Gold standard to diagnose of ectasia is coronary angiography and management is similar to that of CAD with few exceptions

**Keywords:** coronary artery ectasia; stable angina; coronary artery disease; rare case

*Electrocardiogram*



*Coronary Angiography*



## Thyroid Heart Disease: Manifestation and Pathophysiological Mechanism

Fikri<sup>1,\*</sup>, R. Soerarso<sup>1</sup>, A. Kristyagita<sup>2</sup>, A. Sain<sup>1</sup>

<sup>1</sup>Department of Cardiology and Vascular Medicine, Faculty of Medicine, Universitas Indonesia, Jakarta, Indonesia

<sup>2</sup>RSUD dr. M. Ashari, Pemalang, Indonesia

\*correspondence: fikriyt@gmail.com

### ABSTRACT

**Introduction:** The thyroid hormones affect many organs, especially the heart. Hyperthyroidism increases risk of cardiovascular (CV) mortality. Its cardiac manifestations are associated with T3-related-genes transcriptional changes. Hyperthyroidism affects myocytes and hemodynamic. Reviewing its pathophysiology at cellular level is important to fully understand those hemodynamic changes. The prevalence of hyperthyroidism with heart disease in NCCHK tends to decrease from 69 (in 2011) to 44 (in 2015). This case report aims to present THD manifestation and discuss its pathophysiology.

**Case Presentation:** A 45-year-old woman was referred to NCCHK with worsening shortness of breath since one day pre-admission. She got tired easily and had leg swelling during activities since six months pre-admission. Physical examination at ER revealed tachycardia, hypertension, exophthalmos, struma, elevated JVP, bibasilar-lung rales, tremor, and pitting edema of legs. Her ECG showed AFRVR; her CXR showed cardiomegaly and congestion; and her laboratory test showed hypokalemia. She was diagnosed with ADHF et causa HHD, AFRVR, and hypokalemia. During hospitalization, thyroid-function test showed hyperthyroidism (low TSH with elevated FT4); TTE revealed good biventricular systolic function, mild LVH, moderate-severe TR, intermediate probability of PH, and no thrombus; and thyroid ultrasonography showed bilateral struma-nodosa. Optimal HF-therapy and antihyperthyroidism drug were given.

**Discussion:** The prevalence of hyperthyroidism in Indonesian-females is greater than in males'. Genomic and non-genomic mechanisms determine T3 effects on CV through its action on nuclear receptors and ion channels. Thyroid hormones were regulated by TSH. They induce vasodilatation, decreased diastolic pressure, and  $\beta_1$ -receptors expression. Vasodilatation and activated RAA system increase preload. Increased cardiac output is the result of increased stroke-volume and HR. Manifestation of THD include hypertension, arrhythmias, and HF.

**Conclusion:** We have reported a case of THD showing the important thyroid-hormones-CV-system-relationship. Holistic management-approach encompassing basic clinical-cardiology skill is needed.

**Keywords:** hyperthyroidism; thyroid heart disease; hypertension; atrial fibrillation; heart failure

## Myocardial Infarction with Normal Coronary in Patient with Tombstone Electrocardiography: A Case Report

N.R. Sutrisno\*, A. Astuti, A.F. Yahya, J.W. Martha, M.R. Akbar

Department of Cardiology and Vascular Medicine, Faculty of Medicine, Universitas Padjadjaran, Dr. Hasan Sadikin General Hospital, Bandung, Indonesia

\*correspondence: natayaregiza01@gmail.com

### ABSTRACT

**Introduction:** Tombstone electrocardiography (ECG) is an ST-elevation myocardial infarction (STEMI) with prominent ST elevation indicating severe stenosis of left anterior descendant (LAD) artery which associated with severe myocardial damage and poor outcome left ventricular dysfunction. We reported a case of tombstone ECG with normal coronary arteries.

**Case Presentation:** Forty year-old male was referred to ER with chief complain of chest pain 19 hours before admission. He had fever for 1 week and paroxysmal nocturnal dyspnea. Mitral regurgitation murmur and fine rales were found on physical examination. The ECG showed an extensive anterior wall STEMI with elevated troponin I (>10 ng/dl). The patient underwent fibrinolytic at 1 hour onset, but the ECG did not show any ST segment resolution. Angiography revealed normal coronary arteries. Transthoracic echocardiography showed dilated left atrium; ejection Fraction of 50%; hypokinetic at base to mid anteroseptal wall; prolapsed PML, severe MR, suspected vegetation at PML; and mild pericardial effusion. The patient was diagnosed with clinically suspected myopericarditis, acute heart failure, and MVP with suspected infective endocarditis. The patient was treated with aspirin 750 mg three times daily and intravenous furosemide. But unfortunately, he passed away the following day due to refractory cardiogenic shock.

**Discussion:** Tombstone ECG was previously observed in 10-26.1% of STEMI patients. It had high positive predictive accuracy and specificity for LAD stenosis. In our patient, coronary angiography did not show evidence of LAD stenosis. This shows that tombstone is not always caused by LAD stenosis, but represent extensive anterior myocardial damage.

**Conclusion:** Tombstone ECG is not always associated with coronary event and can occur in myocardial infarction with normal coronary arteries which represent extensive damage of anterior myocardium.

**Keywords:** tomsbtone ECG; normal coronary arteries; myopericarditis

## **Intravascular Ultrasound (IVUS) Guided Angiography Percutaneous Coronary Intervention (PCI) Through Left Distal Transradial Access on Young Woman with ST-Elevation Myocardial Infarction (STEMI): A Case Report**

**G.N.I. Putri, B.B. Tiksnadi, A.F. Yahya, J.W. Martha, M.R. Akbar**

Department of Cardiology and Vascular Medicine, Universitas Padjadjaran, Dr. Hasan Sadikin General Hospital, Bandung, Indonesia

\*correspondence: geminastitiip@gmail.com

### **ABSTRACT**

**Introduction:** IVUS is a catheter-based imaging modality that provides high-resolution cross-sectional images of the coronary artery, enabling measurements of vessel areas more accurately.

**Case Presentation:** A 28 years old, female, was admitted to emergency with chest pain 3 days before admission. The risk factor for coronary artery disease (CAD) are hypertension, dyslipidemia, passive smoker and using hormonal contraception. Electrocardiography (ECG) showed ST elevation at lead II, III, aVF, V7-V9, V4R-V5R. Laboratory finding showed Troponin I >10ng/dL. She was diagnosed with STEMI inferoposterior-right ventricle wall, Killip I. She underwent coronary angiography which showed diffuse critical stenosis in proximal RCA. The data from IVUS was shown atherofibrotic plaque with minimum lumen area (MLA) 2.4 mm<sup>2</sup> and reference diameter 4 mm<sup>2</sup>. Direct stenting was conducted in proximal-mid RCA. Post-stenting IVUS evaluation showed apposition of stent, so post dilatation ballooning performed. There were no complications after the PCI and showed TIMI flow 3.

**Discussion:** The most common cause of ACS in young women is coronary dissection and spasm that can be accurately seen by IVUS. IVUS accurately measure MLA, better than angiography. Eight percent of angiographic finding in young women was atherosclerotic plaques which was found in this patient. The plaque composition seen by IVUS help the interventionists to decide the PCI strategy used.

**Conclusion:** ACS in very young adult aged less than 35 years is uncommon. Besides atherosclerotic CAD, non-atherosclerotic or hypercoagulability should be considered. IVUS result also showed her fibrotic lesions which is less than 8% in the woman population.

**Keywords:** AMI; young women; IVUS

## **Acute Myocardial Infarction with Complication Acute Pericarditis in Young Male with Nephrotic Syndrome: A Case Report**

**M. Jamilah\*, B.B. Tiksnadi, T.A. Sihite, J.W. Martha, M.R. Akbar**

Department of Cardiology and Vascular Medicine, Faculty of Medicine, Universitas Padjadjaran, Bandung, Indonesia

\*correspondence: maryam88jamilah@gmail.com

### **ABSTRACT**

**Background:** Arterial thrombosis is an uncommon but serious complication of nephrotic syndrome (NS). Several cases suggested increasing of the ischemic cardiovascular event in NS, but reports of coronary thrombosis are extremely rare.

**Case Presentation:** A 22-years-old male, diagnosed with NS 12 years ago, came into emergency with pleuritic chest pain since 2 hours before admission. It was preceded retrosternal chest pain radiating to the back and sweating at 36 hours before. Physical examination showed pericardial friction rub. Electrocardiogram (ECG) showed an evolution of inferior-posterior ST-elevation. Echocardiogram showed abnormal regional wall motion at inferior, inferoseptal, and inferolateral-wall with circumferential mild pericardial effusion. Laboratorium results revealed thrombocytosis, elevated Troponin I, hypoalbuminemia, proteinuria, elevated plasma fibrinogen, and D-Dimer. Coronary angiography revealed subtotal thrombotic occlusion at the distal right coronary artery (RCA). Angiography after thrombosuction still revealed a thrombus with a mild atherosclerotic lesion. The patient undergoes implantation 2 bioactive stents at distal RCA to proximal right posterolateral branch.

**Discussion:** The main possible role for acute myocardial infarction (AMI) in this patient was arterial thrombosis. Hypercoagulable state along with hypoalbuminemia and thrombocytosis in this patient suggesting a causative relationship between coronary thrombosis and NS. The presence of a mild atherosclerotic lesion in this patient suggests accelerated atherosclerosis that caused by dyslipidemia, endothelial dysfunction, and prolonged steroid therapy. Acute pericarditis in this patient possibly was post MI pericarditis (PMIP). PMIP incidence was rare and its especially occurred in patient with young age, had less hypertension, higher cardiac biomarker, reduced LVEF and had longer time to reperfusion.

**Conclusion:** AMI due to arterial thrombosis can be attributed to a hypercoagulable state resulting from the NS. Beside hypercoagulability, accelerated atherosclerosis might play a role in the ischemic heart disease in NS patients.

**Keywords:** acute myocardial infarction; nephrotic syndrome; hypercoagulable state; pericarditis

## Contrast-Induced Nephropathy After Percutaneous Coronary Intervention : Serial Case Reports

E.D.N. Rahmawati<sup>1,\*</sup>, M.T. Ismail<sup>2</sup>, N. Taufiq<sup>2</sup>

<sup>1</sup>Resident of Department of Cardiology and Vascular Medicine, Faculty of Medicine, Public Health and Nursing, Universitas Gadjah Mada – Dr. Sardjito Hospital, Yogyakarta, Indonesia

<sup>2</sup>Staff of Department of Cardiology and Vascular Medicine, Faculty of Medicine, Public Health and Nursing, Universitas Gadjah Mada – Dr. Sardjito Hospital, Yogyakarta, Indonesia

\*correspondence: evitadevi.dr@gmail.com

### ABSTRACT

**Background:** Contrast-induced nephropathy (CIN), also known as contrast-induced acute kidney injury is an iatrogenic renal injury follows after intravascular administration of radio-opaque contrast media (CM). CIN responsible for a third of all hospital-acquired acute kidney injury (AKI). Improvement of imaging methods and interventional procedures cause significantly increased exposure to CM that increases the number of CIN. CIN incidence in normal patients is about 3.3-8% and increase in patients with underlying renal disease, 12-20%. Primary percutaneous coronary intervention makes better outcome of cardiovascular but increases incidence of CIN.

**Case:** First case was a 61 years-old male admitted to our hospital with chief complain of syncope, dizziness with cold sweating. Patient had no history of heart disease but he had diabetes type 2 last 2 years. Physical examination revealed bradycardia 40 bpm that confirmed with his ECG that showed total AV block QRS rate 40 bpm. Patient was diagnosed with NSTEMI with TAVB so this patient was immediately undergo urgent PCI and temporary pacemaker and implanted 2 stent in right coronary artery with 35 ml of low osmolar agent CM. Baseline serum creatinin (SCr) was 2.2 mg/dL then followed up time by time and increasing everyday. Dialysis was performed in third day, but SCr not significantly decrease and going up in the next day. In the 8<sup>th</sup> day after performed 3<sup>rd</sup> dialysis, SCr was still too high and patient passed away because of his deterioration of kidney function and uncontrolled secondary infection in his lungs and urinary tract. The second case was 78 years-old male admitted to our hospital with angina pectoris stabil that planned to elective PCI. His CV risk factors were ex-smoker and hypertension. His physical examination was within normal limit with his baseline SCr was 1.75 mg/dL. He got hydration overnight before performed PCI and continue 24 hours after exposure of low osmolar CM. first day after PCI his SCr was increase almost 2x from baseline, but second day after 24 hours hydration, his SCr back to his baseline without dialysis.

**Conclusion:** CIN is usually preventable and reversible, but underlying disease (acute coronary syndrome vs stable ischemic heart disease) can affect prognosis. So screening is very important. Use isoosmolar or low osmolar agent with minimal volume is suggested. Hydration in all high risk patient will undergo percutaneous coronary intervention is advised.

**Keywords:** contrast-induced nephropathy; contrast media; PCI; dialysis; hydration

## **Rheumatic Heart Disease with ST Elevation Myocard Infarct : A Case Report**

**E.S. Tirta<sup>1,\*</sup>, M.A. Mukti<sup>1</sup>, S.F. Nadya<sup>1</sup>, I.M. Loebis<sup>2</sup>, G.I.Hadiyat<sup>2</sup>**

<sup>1</sup>General practitioner at Hasna Medika Heart Hospital, Cirebon, Indonesia

<sup>2</sup>Cardiologist at Hasna Medika Heart Hospital, Cirebon, Indonesia

\*correspondence: aliy\_dr@yahoo.com

### **ABSTRACT**

Rheumatic Fever is the dominant cause of Valvular Heart Disease. It is a disease of childhood that leading to an inflammatory reaction that involves many organs, including heart. Embolic Acute Myocardial Infarction is an infrequent cause of Acute Coronary Syndrome, but the association between Valvular Heart Disease and Coronary involvement is well known. We report a case of 59 years old man came to ER complaining of central chest pain four hour earlier. Examination showed tachycardia HR 80 beats/minute; BP 120/70 mmHg. ECG showed ST segment elevation in I, AVL, V2-V6 and Normo Ventricular Response Atrial Fibrillation. Cardiac Troponin was increase in Laboratory test. In Echocardiography, there was a reduce in ejection fraction with anterior wall hypokinetic, severe mitral stenosis and dilated left atrium. Presumptive diagnosis was Acute anterior extensive Myocardial Infarction in Rheumatic Heart Disease. Aspirin, Clopidogrel, and subcutaneous LMWH was given to the patient. After given a sublingual ISDN, his chest pain was reduce upon transfer to Coronary Care Unit to have an early Percutaneous Coronary Intervention. A total occlusion in the Osteal Left Anterior Descending coronary artery was identified. A Bare Metal Stent applied at osteal to proximal LAD coronary artery. Patient was discharged with tripple therapy (Aspirin, Clopidogrel and Warfarin). Correlating these findings we presume an Embolic STEMI probably from a left atrial thrombus, associated with severe MS and Rheumatic Heart Disease. For the next case, Transoesophageal Echocardiography should be done to confirm trombus in left atrial appendage.

**keywords:** rheumatic heart disease; atrial fibrillation; embolic acute myocardial infarction

## **Epicardial Ventricular Tachycardia as Complication in Transmural Myocardial Infarction with Acute Pericarditis: A Case Report**

**D. Listina\*, T.I. Dewi, M. Iqbal, J.W. Martha, M.R. Akbar**

Department of Cardiology and Vascular Medicine, Faculty of Medicine, Universitas Padjajaran, Hasan Sadikin General Hospital Bandung

\*correspondence: dedeslistina@gmail.com

### **ABSTRACT**

**Background:** Most ventricular tachycardia (VT) in transmural myocardial infarction (MI) arose from endocardium, VT from epicardium was rare. Early recognition and appropriate management epicardial VT was essential to prevent further complication.

**Case Presentation:** A 58 years old, male, presented to emergency department with ST-elevation MI infero postero lateral wall, with onset 48 hours. He complained pleuritic chest pain on the fourth day of hospitalization. He was diagnosed acute pericarditis. Echocardiography showed mild reduced LV systolic function with regional wall motion abnormality, and circumferential pericardial effusion (0.6 cm). On the seventh day of hospitalization, he had VT episode that spontaneously resolved. His electrolytes results were normal. ECG showed monomorphic VT with pseudo-delta wave in precordial lead and q wave in lead I. The origin of VT was from left basal lateral. He was given amiodarone 150 mg intravenously followed by 1mg/minute. Angiography revealed CAD 3 VD with total occlusion in proximal left circumflex (LCx) artery. He was implanted 2 stents in LCx. He was discharged without any complications.

**Discussion:** Ventricular tachycardia in this patient was scar related of transmural myocardial infarction with extension to epicardial. Electrocardiography recognition of epicardial VT was pseudodelta wave in precordial lead and q wave in lead I. Pericarditis indicates extensive infarction and leads to higher incidence to VT.

**Conclusion:** Determining origin of VT was important because it would help us giving treatment accurately. Post-infarction-angina pericarditis will make us more cautious of higher incidence in VT. Further electrophysiology study and endo-epicardial ablation will be needed in this patient if the VT is recurring.

**Keyword:** epicardial ventricular tachycardia, pericarditis, transmural myocardial infarction