29-Years Old Woman Presenting with ST Elevation Myocardial Infarction

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Abstract

Myocardial infarction in young female is rare condition, but carries significant morbidity and mortality. The recognition management for MI in young female is challenging due to its unspecific clinical presentation and lack of guidance in the current guidelines. We reported a 29-years old woman presenting with infarction type chest pain, showing ST- elevation in the inferior leads with total AV block as complication. The risk factors in this patient were smoking, family history, cardiomyopathy, and preeclampsia history. The cause for myocardial infarction in this patient was atherosclerotic plaque rupture, but we did not manage to performed coronary intervention due to her low ventricular function and unavailability data for myocardial viability at the time of hospitalization.

Introduction

ST elevation myocardial Infarction (STEMI) still represents the leading cause of mortality and morbidity in the worldwide and is also very important in women as a cause of death and disability. Most coronary heart disease is found in the older age groups, but it also occurs in younger people, with a lower prevalence in women than men. Fortunately, its incidence is not common in patients younger than 45 years. However, the disease carries a significant morbidity, psychological effects, and financial constraints for the person and the family when it occurs at a young age especially women. Previously the MI in young female was ignored since women carry protective factors, but recently available data showed the incidence has significant mortality in this group (Vaccarino *et al.*, 2000). This problem of MI in such young age group became substantial since they are in the productive age and provide significant contribution to the community. Several studies has reported clinical profile and the outcome of STEMI in young age, with variable incidence 2-10% (Wong *et al.*, 2012).

It has not been clear yet whether the incidence of MI in young female is associated with prognosis or requires further management which is different to those older. In addiation, whether MI in young female in a representation of infarction or an acceleration process of atherosclerosis as in older population still remain questioned. However, primary and secondary prevention has definitely to be performed intensively to such young population (Alkhiary, 2011). The clinical presentation and risk factors of MI in young female are different and often cause the ignorance of this population in epidemiological research and clinical trials. In the database Get with the Guidelines CAD, it was found that the mortality rate due to STEMI in woman is twice higher and in this population women received lower aspirin and beta blocker as compared to men. In addition, there was smaller number of reperfusion therapy in this population and the reperfusion time is delayed (Wenger, 2012).

A small number of studies in women with MI, the lack of participant in clinical trials have caused discrepancy in the symptom recognition, acute management and long term treatment for women with STEMI as compared to men. However, guideline from American Heart Association on cardiovascular disease in women pointed important message on the prevention and several consideration on pregnancy, gestational diabetes, and hypertension in pregnancy as a risk factor for cardiovascular in women (Wenger, 2012). In this report, we presented a young female, 5 months post delivery who suffered from STEMI. Important message to be discussed in this report are incidence, risk factors associated with atherosclerosis, management, and prognosis.

Case

A 29 years-old female came to the emergency department of our hospital reffered by rural hospital with infarction type chest pain 3 days before admission. The pain was dull, heavy, retrosternal radiating to the back accompanied by diaphoresis and nausea. At the rural hospital she had been admitted for 2 days as having myocardial infarction

and she was reffered due to history of total AV block. There was no previous complain of angina at all.

Five months before admission, she was diagnosed as having congestive heart failure due to peripartum cardiomyopathy. This abnormal LV function has limited her for household work due to dyspnea on effort. She had 3 histories of pregnancy and delivery. There was no history of abortion. The first two deliveries were normal, while the last delivery mode was cesarean section. She denied any history of using neither hormonal nor mechanical contraception. The atherosclerotic risk factors for this patient were smoking for the last 10 years, 10 cigarettes per day and the strong family history of the father died of having heart attack at 45 years old. In addition, she had history of pre-eclampsia and peripartum cardiomyopathy (PPCM). There was no history for sing and symptoms related to autoimmune diseases.

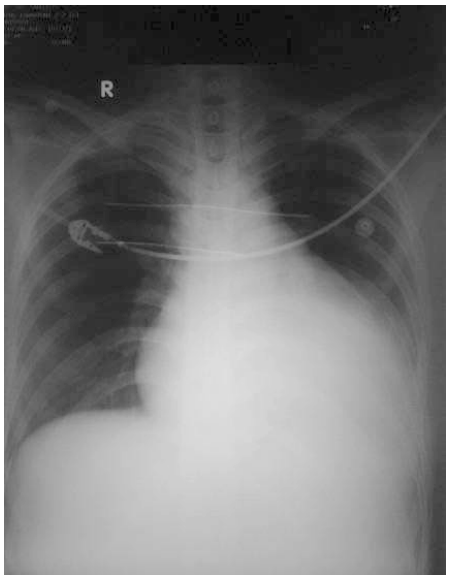

Figure 1. Chest X-ray showed enlargement of the cardiac chambers

Physical examination on arrival showed that the patient was dyspnea with stable hemodynamic signs; BP 110/70 mmHg, pulse 92x/minute, respiration rate 24x/minute, and SpO2 99%. There was increase in jugular vein pressure and enlargement of the heart with gallop S3. We did not find any murmur or other heart sound. Lung examination was normal, we found ascites and enlargement of the liver. There was no abnormality in the extremities. Chest X-ray revealed a lateral downward apex of the heart showing the enlargement of the LV. There was also sign of LA and RV enlargement (Figure 1).

ECG examination showed sinus rhythm, total AV block with ventricular rate 40x/minute and evoluted ST elevation in the inferior leads (II, III, dan avF). In our hospital, there rhythm was sinus with evoluted ST elevation in inferior leads accompanied by reciprocal ST depression in I and aVL, as well as the signs of LVH and LAH.

Laboratory examination showed increase in the cardiac enzyme; CK 2746; CKMB 623; Trop I 9,57. Due the suspicion of vasculitis, we performed an erythrocyte sedimentation rate and CRP level. The ESR was normal while the CRP showed positive result 10 mg/L. However due to its high value, we consider that this was a result of coronary thrombosis and myocardial infarction rather than vasculitis. Based on the data above, we diagnosed this patient as STEMI inferior with the suspicion of non atherosclerotic cause probable hypercoagulable state or vasospastic.

Echocardiography examination showed dilated all cardiac chambers with dyskinesis in

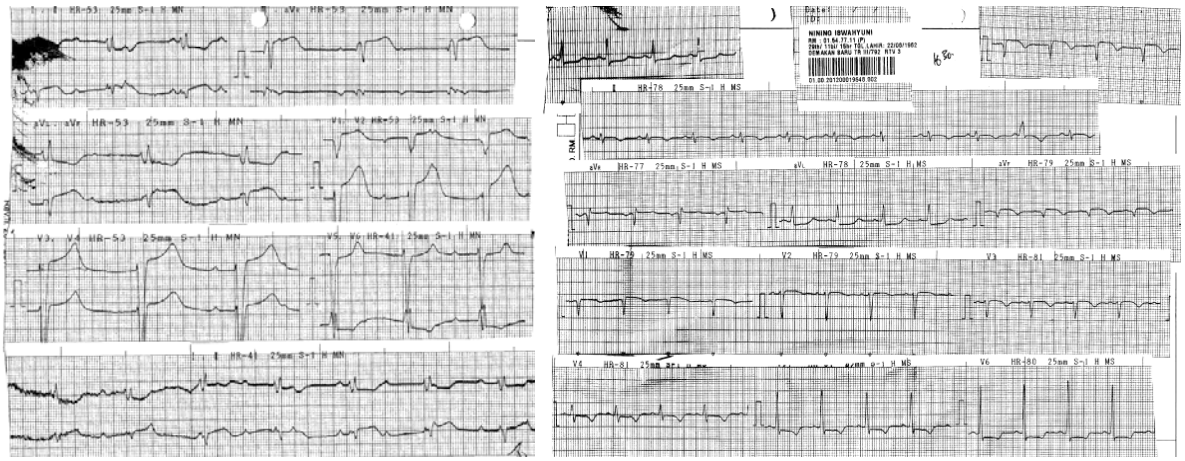

Figure 2. ECG examination in rural hospital (left) and in our hospital (right).

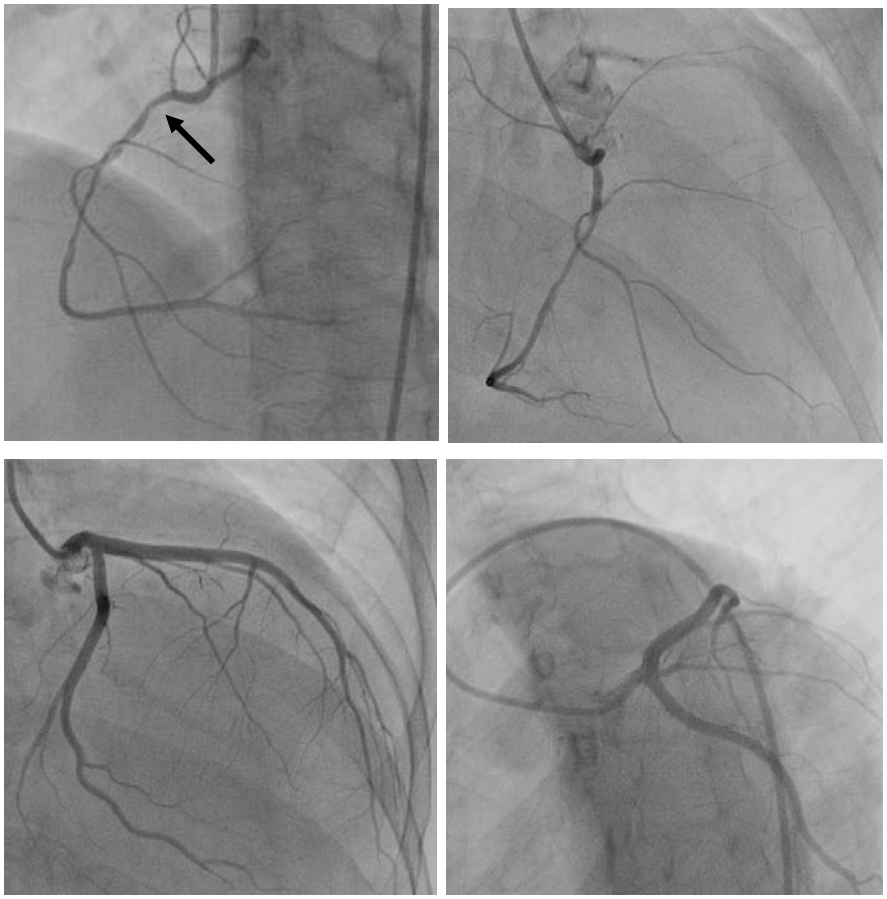

Figure 3. Coronary angiography showed 70% long stenosis in RCA (upper) and soft plaque (arrow). LCx (lower left) and LAD (lower right) were normal.

anteroseptal and hypokinesis in other segments of the wall. The l*eft ventricular end diastolic diameter* (LVIDd) was 52.4 mm and the EF was 14 – 20%. There was also decreased in RV function with Tricuspid Annular Plane Systolic Excursion (TAPSE) 6 mm. We also found mitral and tricuspid regurgitation due to chamber dilatation.

We then performed coronary angiography and showed 70% long stenosis in the right coronary artery (RCA) from proximal to mid segment. In addition in the proximal we found a 90% stenosis with suspicion of soft plaque. The left anterior descenden (LAD) and left circumflex (LCx) still within normal limit (Figure 3). We decided not to perform coronary intervention directly due to its reduced systolic function, thus the myocardial viability was necessary.

We admitted the patient in the ICCU and we administered unfractionated heparin (UFH) in addition to aspirin 1x80 mg, clopidogrel 1x75 mg, simvastatin 1x20 mg and the medication for heart low LV function which are captopril 3x25 mg and furosemide. On the day 5, we added carvedilol 1x3.125 mg as heart failure treatment. During her admission, she once suffered from a frequent ventricular extra systole (VES) which was improved with lidocaine therapy.

Patient was admitted for 7 days, and discharged after adequate mobilization and cardiac rehabilitation without any symptoms and signs of ischemia. The definite diagnosis was STEMI due to atherosclerotic plaque rupture and PPCM, and we managed her with aspirin 1x80 mg, clopidogrel 1x75 mg, simvastatin 1x20 mg, furosemid 40 mg/hari, carvedilol 1x 3.125 mg, and captopril 3x 25 mg.

Discussion

In our case we reported a 29 years-old female in the productive age who suffered from STEMI due to atherosclerotic plaque rupture. The interesting point in this case was that MI occurred in young female, in her reproductive age,

5 months after delivery. There were many reports regarding the MI in young age, however the cases in young female were rarely published. A case report showed a 25-years old female with high risk factors of smoking and metabolic syndrome suffered from STEMI (Falcone *et al.*, 2004). A study in 303 young women aged less than 40 years old, showed 5% incidence of MI in less that 25 years old. In this study, 22 out of 32 patients were screened for coronary heart disease and showed ECG revealing old myocardial infarction (Arnold dan Moodie, 1993).

In young female, the combination for risk factors hold important role for MI. In our case, the patient smokes, had strong family history, PPCM as well as pre-eclampsia history in the last pregnancy. The combination for risk factors increases the incidence of MI. One study showed that in women less than 30 years old who suffer from MI, 60% has hypercholesterolemia, increase LDL, and triglyceride. In addition, 28% had juvenile diabetes, 38% had hypertension, and 28% used oral contraception (Arnold dan Moodie, 1993). Seventy two percent young female who had MI is smoker. Thus, smoker is the strong risk factors for MI in young female. Based on Myocardial Infarction Causality (MICA), smoker women had 10 times *odd ratio* to have MI. The increase in the number of cigarette per day, the bigger risk they have. Smoking is known to decrease estrogen in women, thus pre menopause women had risk of MI with smoking (Dunn *et al.*, 1999).

Family history hold important role and positively associated with early MI in women. In study who enrolled women with MI aged 18-40 years, family risk factors contribute to 5.17 times odd ratio (Friedlander *et al.*, 2001). Our patient had strong family risk factor on the father. The role of traditional risk factors for MI in young age is lower as compared to those who is older. However, non traditional risk factors such as inflammatory gen, homosistein level, as well as intima-media thickness (IMT) shared important risk (Knoflach *et al.*, 2009). In our patient, unfortunately we did not measure the factors above.

Among reproductive factors, the association between coronary heart disease and parity showed inconsistent data. Some data showed that the parity number is associated with coronary heart disease, some is not. Young age at the first delivery has been reported to be associated with the increase in risk. In addition, women who have history of spontaneous abortion also have increase risk for MI as compared to those who do not (Bertuccio *et al.*, 2007). These also account for the history of pre-eclampsia and PPCM (Lin *et al.*, 2011). It was reported that C-reactive protein (CRP), Tumor Necrosis Factor (TNF)-α in both diseases (Fett dan Ansari, 2010). The estrogen level in women is known to have protective effect against MI. The reduced estrogen level in menopause women increases risk for MI in that population. Estrogen deficiency in pre menopause women will increase 7 times for the risk of MI (Anderson dan Pepine, 2007). In our patient we did not measured the estrogen level due to some limitation on the laboratory examination.

The variability of clinical presentation for MI in young female is challenging for doctors. The initial presentation is not specific and unclear. Infarction type chest pain is rarely significant. About 85 – 90% women showed prodromal symptoms prior to MI, however most of the patients ignore these symptoms. Unfortunately, some who seek for medical advice due to these symptoms reported unawareness from medical professional thus often misdiagnosed (Mcsweeney *et al.*, 2003). Several studies in women reported only 29.7% patients suffer from chest pain. The characteristic for chest pain in women varied from heavy, feeling numbness, burned, feeling fullness, even tickling. Other acute symptoms are breathless (58%), weakness (55%), fatigue (43%), cold sweating (39%), and headache (39%). The pain usually radiates to the back (37%) and shoulder (28%) (Mcsweeney *et al.*, 2003).

MI in young female had coronary characteristic varied from traditional atherosclerotic process to secondary due to other underlying disease. This pattern should be evaluated by coronary angiography. Morphologically, the coronary size in women is smaller as compared to men, even after adjusted with body weight. Aortic stiffness associated with age is more frequently found in diabetic women. This change also occurs in pregnancy, and may refpect some physiological changes. However, the stiffness is a pathological manifestation of vascular remodeling that should not be ignored (Anderson dan Pepine, 2007). Women who suffer from atherosclerotic plaque rupture showed different coronary characteristics. It was reported that 44% had single-vessels, 44% had multivessel disease and 13% had normal coronary vessel (Glover *et al.*, 1982). Our patient showed soft plaque in proximal RCA.

The clinical implication of MI in young female is to define the best therapeutic band intervention strategy. Previous study reported that men are more likely to receive thrombolysis, aspirin and beta blocker as compared to women, indicated that the standard strategy accordingn to guidelines has not been well implemented in women. Percutaneus intervention in women has been reported 14.2% as compared to 24.4% in men. The expected number of intervention in women is 17.4% (Anderson dan Pepine, 2007). The *under-treatment* condition in women given the high risk factors profile has increase mortality. In our case, we administered dual antiplatelet therapy and statin. We did not performe coronary intervention in our patient during hospitalization due to lack of myocardial viability data.

The differences in comorbodity among gender result in different outcome after MI. Several conditions are diabetes, post delivery state that including peripartum vascular diseaction, preeclampsia and eclampsia, polycystic ovarian syndrome, and low birth weighted baby (Anderson dan Pepine, 2007). Another study showed that female gender is an independent predictor for short term outcome after MI. In addition, the negative outcome in women post MI will increase in younger age. As compared men with similar age, young female had 25% higher mortality and this ratio will decrease with the increment of age (Andrikopoulos *et al.*, 2006).

Conclusion

We reported a 29 years-old female with inferior STEMI occurring 5 months after delivery. Although she was considered protected by estrogen mechanism, the other risk factors for MI in this patient was smoker and strong family history. The suspicion for MI in younger age should be increased especially for those who has strong risk factors. The careful examination should be performed to prevent misdiagnosis. Aggressive therapy and the guideline implementation must be adjusted similarly to women as to men to decrease mortality and negative outcome after MI.

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