

Acute myocardial infarction in a young pregnant woman

Genç bir gebede akut miyokart infarktüsü

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Introduction

Myocardial infarction (MI) is very rare during pregnancy (1/10000), happens mostly during the third trimester and puerperium and mortality rates are high (19-21 %) (1). In most cases the diagnosis was made postmortem (2). Mostly, vasospasm with pregnancy induced hypercoagulable state, which is potentiated by exogenous factors -like progesterogens and smoking.

Case report

A 24-year-old woman was admitted to hospital with chest pain in her 18th week of pregnancy. She had had two abortions during the last two years and was receiving hydroxyprogesterone caproate treatment. Electrocardiogram revealed ST elevation in leads DI, aVL, V2-V6 and ST depression in DII, DIII and aVF. Physical examination was normal except tachycardia. Echocardiography showed akinesia of anterior and apical segments and ejection fraction was 25%. Emergent coronary angiography showed a total occlusion of proximal left anterior descending artery (LAD) with heavy thrombus burden. Circumflex and right coronary arteries were normal. After balloon dilatations, a 3.0x32 mm heparin coated Jostent (Jomed GmbH, Rangendingen, Germany) was implanted in proximal LAD within 3 hours after the onset of symptoms. Although a TIMI 2-3 flow with no residual stenosis was achieved, she became hypotensive, showed signs of progressive LV dysfunction and died despite pharmacological and mechanical ventilatory support. Blood biochemistry revealed high levels of total cholesterol (283 mg/dl with HDL 65 and LDL 160 mg/dl) and triglyceride (287 mg/dl), mild anemia (Hb 10g/dl) with high leucocyte (52000) and thrombocyte (450000) counts.

Discussion

Incidence of classical risk factors like hypertension, smoking, family history and hyperlipidemia among peri/ante partum MI were reported as 21%, 6%, 32% and 12%, respectively (2). In pregnancy profound alterations occur in the coagulation and fibrinolytic systems; increase in clotting factors (II, VII, VIII, IX and X), fibrinogen levels, platelet turnover, decrease in deformability of erythrocytes and reduction in func-

tional protein S levels. These changes result in a gradual decrease in fibrinolytic activity. Smoking further increases risk for thrombosis (3).

Preeclampsia may also play a role in the ischemic syndromes; both by causing an increase in workload and inducing the dissection of coronaries. Incidence of preeclampsia among pregnant women with MI is 10% (2).

Badui et al. (4) reported vasospasm with hypercoagulable state as the most common cause of MI during pregnancy (4). Roth and Elkayam (3) reported atherosclerosis (43%) as the primary cause, 21% coronary thrombus without atherosclerosis, 16% coronary dissection and 29% normal coronaries. Kulka et al. (5) reported a woman with MI during caesarean section with angiographically normal coronaries; an intravascular ultrasound study demonstrated an atheroma with ruptured fibrous cap.

In our case, coronary angiography showed no atherosclerotic changes but predominantly thrombus formation in LAD. High lipid levels were the only risk factor for MI in our patient and progesterone use might have induced an increase in plasminogen activator inhibitor levels and vasospasm promoting thrombus formation. Reports about progesterogens are controversial and mostly derived from postmenopausal replacement and/or oral contraceptive studies, so they cannot be adopted directly for pregnant patients. Nakagawa et al. (6) reported of MI in a 54-year old women receiving medroxyprogesterone therapy. Medroxyprogesteronacetat may antagonize the anti-atherosclerotic effects of estradiol (7). However, an international, multicenter study indicates that there is little or no increased risk of stroke, venous thromboembolism or MI associated with progesterogens (8).

Ladner et al. (9) has found a lower mortality than previously reported (7.3% vs. 19-21%) despite the increased incidence of peripartum MI between 1991 and 2000. They reported that chronic hypertension, diabetes and advanced maternal age were the independent predictors of MI. James et al. (10) researched the national databases between 2000 and 2002 in US and found that the mean age was 33 years for those with acute MI and 27 years for those without (10). They found a higher incidence and lower mortality (5.1%) than Ladner's analysis. They think that this increase may have been due to higher number of older pregnant women than before and widespread use of troponins for diagnosis. In their study the significant risk factors were: advanced age, black race,

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hypertension, trombophilia, anemia, diabetes mellitus, smoking and preeclampsia.

Conclusion

Acute MI during pregnancy has high maternal and fetal mortality rates and in most cases vasospasm, hypercoagulable state and additional exogenous factors (e.g. progestogens and smoking) may be the underlying mechanism.

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Hepatopulmonary syndrome associated with Budd-Chiari syndrome

Budd-Chiari sendromu ile birliktelik gösteren hepatopulmoner sendrom

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Introduction

Budd-Chiari Syndrome (BCS) is a rare cause of portal hypertension. Occlusion of inferior vena cava (IVC) or hepatic vein (HV) causes BCS and leads to centrilobular congestion and necrosis of liver. We present a cyanotic patient with BCS associating with hepatopulmonary syndrome (HPS).

Case Report

A 15-year-old girl admitted with dyspnea and cyanosis. She was referred to our clinic with the prediagnosis of congenital heart disease. On examination, there was cyanosis of the skin and mucosa; clubbing of the fingers and toes (Fig.1) and a grade 2/6 systolic murmur. Electrocardiography showed right axis deviation. Chest X-ray and thoracic computed tomography findings are presented in Figure 2. The pulmonary veins in the lower parts of the lungs were prominent and enlarged.

Her laboratory results revealed; hemoglobin, 17.9 g/dl; platelets, 120000/mm³, white blood cell count, 5000/mm³. Blood glucose level was 73 mg/dl, alanine aminotransferase, 29 U/L; aspartate aminotransfer-

ase, 38 U/L; gamma-glutamyl transferase, 52 U/L; alkaline phosphatase, 490 U/L; total protein, 7.6 mg/dl; albumin, 3.9 mg/dl; total bilirubin, 3.32 mg/dl; direct bilirubin, 1.02 mg/dl. Arterial blood gas analysis revealed;



Figure 1. Cyanosis and clubbing of the fingers are shown in the figure

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