Abstract. – Acute myocardial infarction (AMI) during the early postpartum period is rare but may be associated with poor maternal outcome. We report an inferior AMI in a 30-year-old woman with mitral valve replacement during early postpartum period successfully treated with tirofiban. For a patient that has got AMI due to massive thrombus and that is not suitable for percutaneous coronary intervention and has got contraindication to thrombolytic therapy, glikoprotein IIb-IIIa inhibitor (tirofiban) infusion must be kept in mind.

Key Words: Acute myocardial infarction, Early postpartum period, Tirofiban.

Introduction

Acute myocardial infarction (AMI) during the early postpartum period is rare but may be associated with poor maternal outcome. The most important risk factors are advanced maternal age and pre-existing risk factors for coronary heart disease, such as diabetes mellitus, hypertension, smoking and hypercholesterolaemia. The prevalence of ischemic heart disease was reported only in 13% of pregnant women with AMI. When coronary artery characteristics were determined (with angiography or autopsy) during pregnancy or postpartum period, atherosclerosis with or without intra-coronary thrombus was found in 40% of patients and definite or probable coronary thrombus without evidence of atherosclerotic disease was present in 8% of patients. The significant changes in the coagulation and fibrinolytic system that occur during pregnancy increase the risk for thrombosis. Furthermore, hypercoagulation is augmented at the time of separation of the placenta, which is a major source of tissue plasminogen activator (t-PA) inhibitor type 1 and 2. AMI due to extensive intra-coronary thrombus load without evidence of atherosclerotic disease in early postpartum period is a rare condition. The number of reported AMI occurring in the early postpartum period is considerably smaller. We report a case of an inferior AMI in a 30 year old woman with mitral valve replacement (MVR) during early postpartum period treated successfully with tirofiban.

Case Report

A 30-year-old woman with MVR was admitted to our Emergency Department because of sudden onset chest pain on the third day after delivery. She did not have dyspnea, syncope, haemoptysis, or fever. She gave no history of cardiovascular risk factors. Her medical history included an operation of MVR approximately ten months ago. She had used warfarin for four months; then began to take enoxaparine (2*1 SC) during pregnancy. On physical examination, blood pressure was 110/70 mmHg, heart rate 96 beats/min and respirations 21 breaths/min. Oxygen saturation determined by pulse oximetry was 98%. The heart sounds were normal, and there was no murmur, rub or gallop. Electrocardiography showed ST-segment elevation of 2 mm in leads DII, DIII, aVF and reciprocal ST depression in DI, aVL, V1-3 (Figure 1a). Acute inferior wall myocardial infarction was diagnosed. The serum creatinine kinase and troponin-I peaked at 2481 U/L and 46.2 µg/L respectively. Complete blood count and plasma glucose were normal. Her total serum cholesterol elevated at 220 mg/dL with fasting low-density lipoprotein cholesterol of 143 mg/dL. Transthoracic echocardiography revealed hypokinesis of the inferior wall, with an overall ejection fraction estimated at 45% with Simpson formula. Normal function of the mitral valve prosthesis was observed. The right ventricle appeared to have normal dimension and function. Emergency coronary angiography demonstrated extensive thrombus load in the left circumflex coronary artery (LCx) and first obtus margin branch (OM1) (Figure 2).
Postpartum AMI treated with tirofiban

Figure 1. A, Electrocardiography showing ST-segment elevation in leads DII, DIII, aVF and reciprocal ST depression in DI, aVL, V1-3. B, Emergency coronary angiography demonstrates extensive thrombus load in the left circumflex coronary artery and first obtus margin branch. (LCx; Left circumflex coronary artery; MVR; Mitral valve replacement; LAD: Left artery descendent).

1 week of labour or delivery, mortality may be as high as 45%5. AMI in these women has been associated with coronary artery spasm, hypercoagulability, and atherosclerotic heart disease. At angiography, coronary atherosclerosis with or without thrombus is identified in the majority of cases2-5. The frequency of thrombus with normal coronary arteries in diagnostic coronary angiography is 21% in pregnancy1. The mechanism leading to the development of a coronary thrombus is obscure, but may be similar to other clotting abnormalities complicating the third trimester and puerperium. Thrombus formation is the net effect of the changing in the hematologic system during pregnancy.

Criteria for diagnosis of AMI in women with pregnancy or the puerperium period are in general the same as in non-pregnant patients and consist primarily of symptoms, electrocardiographic changes, and cardiac biomarkers. However, evidence-based treatment of these patients is difficult due to the lack of data from randomized controlled trials. According to the ACC/AHA guidelines, thrombolytic therapy has a relative contraindication due to increased maternal and fetal bleeding in pregnant women and/or in the early postpartum period.6 Considering the limited information related to the efficacy and safety of thrombolysis during pregnancy/peurperium and the relatively high frequency of normal coronary anatomy found in these patients, an invasive strategy may be preferred in women presenting with AMI during pregnancy or the puerperium. Case reports of successful interventional treatment of AMI during pregnancy and postpartum period has been documented in the

Discussion

The prevalence of AMI during pregnancy and the puerperium has been determined to occur with a frequency of 1 in 10000 to 1 in 35750 deliveries. However, pregnancy has been shown to increase the risk of AMI 3- to 4-fold1. If AMI occurs within 2 weeks of labour or delivery, mortality may be as high as 45%5. AMI in these women has been associated with coronary artery spasm, hypercoagulability, and atherosclerotic heart disease. At angiography, coronary atherosclerosis with or without thrombus is identified in the majority of cases2-5. The frequency of thrombus with normal coronary arteries in diagnostic coronary angiography is 21% in pregnancy1. The mechanism leading to the development of a coronary thrombus is obscure, but may be similar to other clotting abnormalities complicating the third trimester and puerperium. Thrombus formation is the net effect of the changing in the hematologic system during pregnancy.

Criteria for diagnosis of AMI in women with pregnancy or the puerperium period are in general the same as in non-pregnant patients and consist primarily of symptoms, electrocardiographic changes, and cardiac biomarkers. However, evidence-based treatment of these patients is difficult during the follow-up period and she was discharged ten days after the admission. After six months of follow-up the patient is still asymptomatic and is carrying on her therapy with aspirin, warfarin, metoprolol and ramipril.
medical literature\textsuperscript{1,3,5}. However, some patients may not be suitable for invasive treatment options, as in our patient. Management decisions must be taken according to the individual circumstances involving each case, with risk benefit assessment, guided by standard, evidence-based therapy and case report data from the literature.

We describe an inferior AMI on the third day of the early postpartum in a 30 year old woman with MVR. In our case, coronary angiography demonstrated an extensive intracoronary thrombus load in the LCx and OM1. Pregnancy-related hematological changes and irregular use of low molecular weight heparin after the delivery probably triggered the development of massive and diffuse thrombus in our case. Because of TIMI-III flow in these coronary arteries and the relative contraindication of thrombolysis, glycoprotein IIb-IIIa inhibitor therapy was our choice of treatment.

Extensive intracoronary thrombus load without atherosclerotic disease is a rare cause of an acute coronary syndrome in women with early postpartum period. Unfortunately, there is no medical consensus on the optimal management protocol of such patients, but there is evidence that these patients have a better prognosis when referred for percutaneous coronary intervention or aggressive medical therapy. For a patient who suffers AMI due to massive coronary thrombus not suitable for percutaneous coronary intervention and possesses a contraindication to thrombolytic therapy (such as our patients), the option of glycoprotein IIb-IIIa inhibitor (tirofiban) infusion must be kept in mind. In addition, there are no studies evaluating the efficacy and safety of glycoprotein IIb-IIIa inhibitor therapy, such as tirofiban, in early postpartum period. However, the safety of tirofiban use (alone or adjunctive) in patients with AMI during the postpartum period needs to be determined via larger studies.

**Reference**

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The summary of this case report presented at ‘6th Congress of Update in Cardiology and Cardiovascular Surgery, in conjunction with 59th International Congress of the European Society for Cardiovascular Surgery (ESCVS), April 15-18, 2010, Izmir, Turkey.