 ISSN 0022–9032

CLINICAL VIGNETTE

Acute coronary artery thrombosis during the postpartum period complicated by cardiogenic shock and AH1N1 infection

Ostra zakrzepica tętnicy wieńcowej w okresie połogu powikłana wstrząsem kardiogennym oraz infekcją AH1N1

Łukasz Lewicki[1], Aneta Stróżyk[2], Miłosz Jaguszewski[2], Wiesław Puchalski[2], Andrzej Rynkiewicz[2], Radosław Targoński[1]

[1]Department of Invasive Cardiology, Pomeranian Cardiology Centres, Wejherowo, Poland
[2]First Department of Cardiology, Medical University of Gdansk, Gdansk, Poland

A 25 year-old woman during the postpartum period with no cardiovascular history was admitted to the coronary care unit suffering from acute angina of 3 hours' duration. An ECG displayed ST segment elevation in the anterolateral leads (Fig. 1). Coronary angiography revealed thrombotic occlusion of the left anterior descending coronary artery (LAD) (Fig. 2A). Percutaneous coronary intervention was performed coupled with thrombectomy with a good angiographic result in LAD, however with distal embolisation of the circumflex artery (Fig. 2B). An echocardiogram showed severe left ventricular (LV) dysfunction with LV ejection fraction of 20%. Hypercoagulability tests, including lupus anticoagulant, anticardiolipin antibodies IgA, IgG and IgM, antithrombin III, protein C and S, Factor V Leiden, prothrombin G20210A, as well as partial thromboplastin time, excluded antiphospholipid syndrome. Nor was an abnormal homocysteine level detectable. However, further diagnostics documented the homozygotic mutation for C677TT in the homocysteine metabolic chain enzyme literally in methylenetetrahydrofolate reductase (MTHFR). On the third day of hospitalisation, the patient developed Acute Respiratory Distress Syndrome and cardiogenic shock. The laboratory findings evidenced AH1N1 virus infection. After many weeks of management, the patient recovered and remained asymptomatic. Acute myocardial infarction (MI) during the postpartum period is rare and has been shown to be associated with a poor outcome. A large body of literature has reported different pathogenic mechanisms such as atherosclerosis, vasospasm, coronary dissection secondary to hormonal changes and thrombosis. The LAD seems to be the most frequently involved. Pregnancy is thought to be a hypercoagulable state due to increased levels of procoagulant factors with simultaneously decreased levels of anticoagulants and fibrinolytic activity. Coronary thrombosis during pregnancy due to decreased concentration of t-PA and protein S has been previously reported. Our patient carried the homozygotic mutation for C677TT of MTHFR. This can result in a thermolabile variant of the enzyme with about 50% of its residual activity and predisposes to moderate hyperhomocysteinaemia, not clearly associated with atherothrombotic disease. However, some studies have shown the MTHFR C677TT genotype to be responsible for a more than 3-fold increase in the risk of coronary artery disease and ischaemic stroke, whereas others have questioned this association. We conclude that this polymorphism is, at most, a modest risk factor for arterial thrombosis. The MTHFR C677TT mutation has been also found among patients with venous thrombosis. Foliate deficiency in young women may precede hyperhomocysteinaemia, especially when the MTHFR 677TT mutation is detected. Therefore it could be the most relevant risk factor for MI. Here we must emphasise that our patient was administered folate due to pregnancy and its blood level was within the normal range values. The contribution of genetic thrombophilia markers to arterial thrombotic disease remains unclear. Here we present a unique case of a woman in the postpartum period who carried the MTHFR C677TT mutation and was diagnosed with coronary artery thrombosis. This suggests that although this polymorphism alone is likely to be a poor risk factor for atherothrombosis, it might turn out to be crucial in the presence of acquired temporary risk factors.

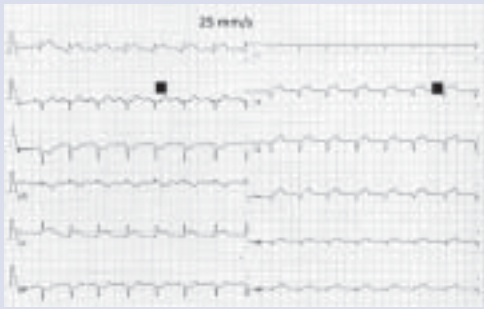

Figure 1. ECG: normal sinus rhythm with heart rhythm 100/min, ST segment elevation in leads I, aVL, V_2–V_6

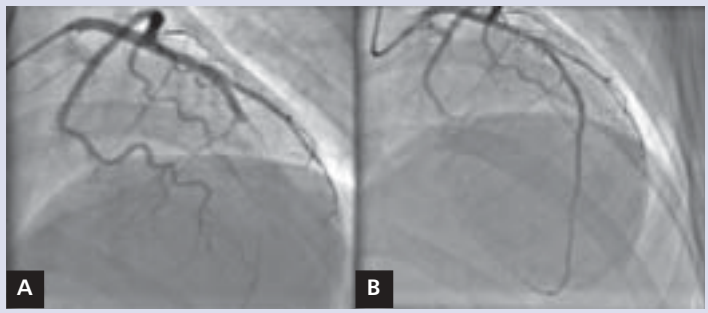

Figure 2. AP cranial view; **A.** Thrombotic occlusion of left anterior descending artery in its middle part; **B.** The infarct related artery with restored TIMI 3 flow; distal embolisation of left circumflex artery

Address for correspondence:
Łukasz Lewicki, MD, PhD, Department of Invasive Cardiology, Pomeranian Cardiology Centres, ul. Jagalskiego 10, 84–200 Wejherowo, Poland, e-mail: luklewicki@gmail.com
Received: 02.07.2012 **Accepted:** 18.07.2012

Conflict of interest: *none declared*