CASE STUDY

CONTENTS

Spontaneous coronary artery dissection following induced vaginal delivery

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ABSTRACT

Cardiovascular diseases represent a significant threat to the health and life of women during pregnancy and the postpartum period, accounting for 13% of all maternal deaths and constituting the third most common cause of perinatal mortality. A particular cause of myocardial infarction in young women, including pregnant patients, is spontaneous coronary artery dissection (SCAD).The occurrence of spontaneous coronary artery dissection may be preceded by triggering factors such as intense physical exertion, intense Valsalva manoeuvres, emotional or physical stress, labour, as well as exposure to exogenous hormones and β hCG. Despite the increasing number of reported cases, the true incidence of pregnancy related SCAD remains unknown. The presented clinical case describes an acute coronary syndrome in a 32-year-old woman in the early postpartum period, secondary to spontaneous coronary artery dissection. The patient was qualified for urgent coronary angiography, which revealed critical, ninety-percent stenosis of the left anterior descending coronary artery (LAD). Following percutaneous transluminal coronary angioplasty, effective myocardial reperfusion was achieved. Young women with low cardiovascular risk and without clinically significant risk factors may develop acute myocardial ischaemia.

KEY WORDS: acute myocardial ischaemia, spontaneous coronary artery dissection, postpartum period

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INTRODUCTION

Cardiovascular diseases represent a significant threat to the health and life of women during pregnancy and the postpartum period, accounting for 13% of all maternal deaths and constituting the third most common cause of perinatal mortality [1]. One of the most serious cardiovascular complications is myocardial infarction. According to the European Society of Cardiology, and in line with the "Fourth Universal Definition of Myocardial Infarction" (2018), myocardial infarction is defined as the presence of acute myocardial injury confirmed by abnormal biochemical markers in a clinical setting consistent with acute myocardial ischaemia [2]. A particular and diagnostically challenging cause of myocardial infarction in young women, including pregnant patients, is spontaneous coronary artery dissection (SCAD). By definition, spontaneous dissection of an epicardial coronary artery is not associated with atherosclerosis or injury [3]. The incidence of SCAD still remains underestimated; however, it is estimated to account for 1-4% of acute coronary syndrome cases [3]. The most frequently identified risk factor is fibromuscular dysplasia (FMD) - a nonatherosclerotic, noninflammatory vascular disease characterised by abnormal arterial wall growth. FMD is diagnosed in up to 72% of SCAD cases [4]. The occurrence of spontaneous coronary artery dissection may be preceded by triggering factors such as intense physical exertion, intense Valsalva manoeuvres, emotional or physical stress, labour, as well as exposure to exogenous hormones and βhCG. Despite the increasing number of reported cases, the true incidence of pregnancyrelated SCAD remains unknown. A review of the existing literature indicates 510 described cases of this complication up to 2023 [5]. Given the serious clinical consequences and diagnostictherapeutic challenges, SCAD in the peripartum period deserves particular attention.

CASE REPORT

We present the case of a 32-year-old patient admitted electively to the Pregnancy Pathology Unit due to gestational diabetes treated with diet at 39+0 weeks of

Fig. 1. Follow up electrocardiogram showing Pardee wave in precordial leads
Source: Own materials

gestation. The pregnancy was additionally complicated by well-controlled hypothyroidism. Her obstetric history included one uncomplicated vaginal delivery three years earlier. The patient was qualified for induction of labour with 5 units of oxytocin. Labour analgesia was provided with remifentanil at a rate of 0.1 mg/hour. The first stage of labour lasted 1 hour and 45 minutes, and the second stage lasted 6 minutes. A live term male infant was delivered, with a birth weight of 3200 g and an Apgar score of 10. Due to incomplete delivery of the placenta, uterine curettage was performed. A vaginal mucosal tear was sutured. Peripartum blood loss was estimated at 300 ml.

Twelve hours after delivery, the patient began reporting chest pain, nausea, and vomiting. Additional diagnostics revealed ST-segment elevation in leads V5 - V6 on electrocardiography (ECG) and elevated high-sensitivity troponin T (hsTnT) levels of 18 ng/l, forming the basis for diagnosing acute myocardial injury. A followup ECG demonstrated evolution of changes into Pardee waves over the anterior wall leads and elevated N-terminal pro-B-type natriuretic peptide (NT pro-BNP) levels of 208 pg/ml (Fig. 1).

Computed tomography angiography of the chest excluded pulmonary embolism. The patient was transferred to the Cardiology Clinic for further management, where she was qualified for urgent coronary angiography. This revealed critical, ninety-percent stenosis of the left anterior descending coronary artery (LAD) in segment s7 with a contrast defect suggesting thrombus. The vessel lumen was significantly constricted, with possible stenosis in segment s8 **(**Fig. 2, 3). Percutaneous transluminal coronary angioplasty (PTCA) was performed without complications, with implantation of five drug-eluting stents (DES), covering the dissection proximal to the stents and achieving reperfusion with optimal residual stenosis (Fig. 4).

Myocardial infarction secondary to spontaneous coronary artery dissection was suspected. After PTCA, persistent distal vasospasm was observed without visible dissection at the distal stent edges. Dual antiplatelet therapy (DAPT) after percutaneous coronary intervention (PCI) was initiated in accordance with current guidelines, using ticagrelor and acetylsalicylic acid (ASA). Effective myocardial reperfusion was achieved three hours after symptom onset. Pharmacological suppression of lactation with cabergoline was necessary to initiate optimal treatment. On the first day after PCI, transthoracic echocardiography (TEE) revealed akinesia of the apex of the heart and adjacent segments of the

Fig. 2. Coronary angiography – LAD dissection in s7 with critical stenosis
Source: Own materials

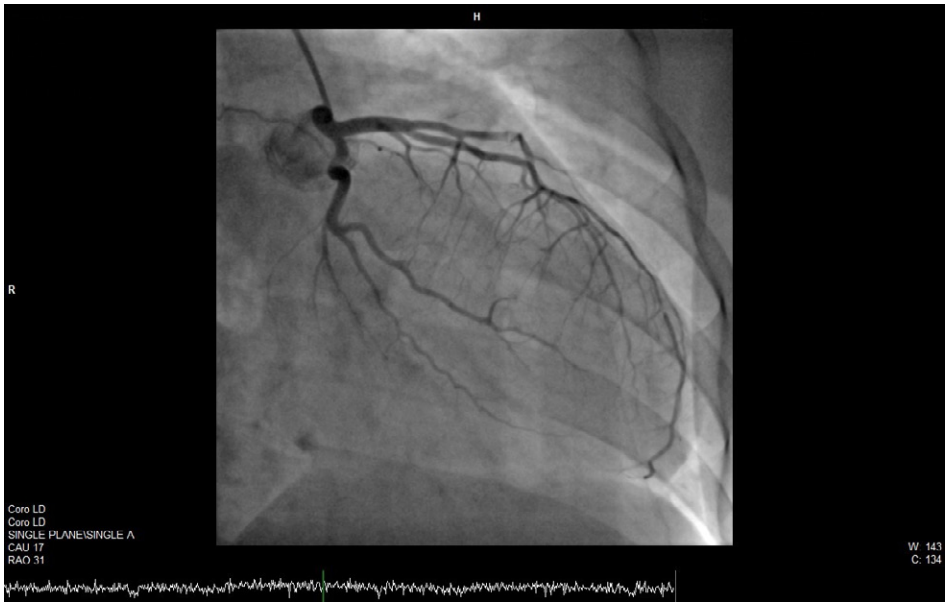

Fig. 3. Coronary angiography – LAD dissection in s7 with critical stenosis
Source: Own materials

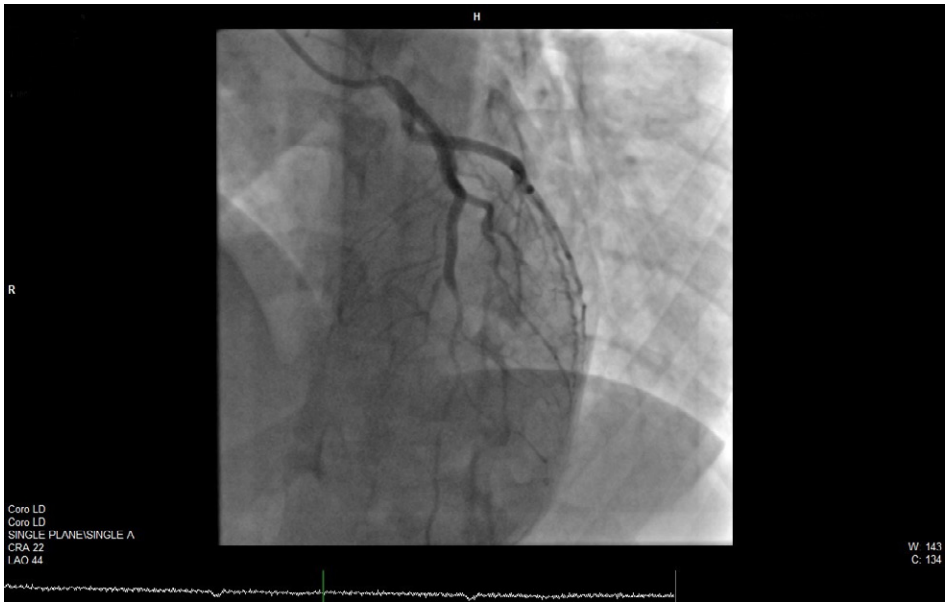

Fig. 4. Coronary angiography after percutaneous coronary intervention
Source: Own materials

anterior, inferior, and interventricular septal walls, as well as the midsegments of the anterior wall and septum, with hyperkinesia of the remaining left ventricular segments. Left ventricular ejection fraction (LVEF) was estimated at 48%.

Additionally, separation of pericardial layers was noted (in parasternal long-axis view, PLAX) up to 2 mm behind the posterior wall of the left ventricle, up to 5 mm at the apex, and in the subcostal view anterior to the right ventricle. Cardiac chamber dimensions and wall thickness remained unchanged. Follow-up hsTnT levels increased to 5583 ng/l. Mixed hyperlipidaemia was diagnosed, with elevated lowdensity lipoprotein cholesterol (LDL-C) of 152 mg/dl and triglycerides (TG) of 190 mg/dl. In subsequent days of hospitalization, significant progression of pericardial effusion was observed, up to 6 mm in PLAX behind the posterior wall of the left ventricle and 10 mm at the apex, without signs of cardiac chamber compression.

Treatment for pericarditis was initiated with non-steroidal anti-inflammatory drugs and colchicine, in accordance with current guidelines [7], resulting in reduction of effusion in followup TEE. Due to suspicion of myocardial infarction secondary to spontaneous coronary artery dissection and further propagation of dissection during the procedure, followup coronary angiography was performed, confirming optimal PTCA outcome, excluding DES thrombosis, and demonstrating proper healing of dissection at distal stent edges without impaired flow and with vessel lumen relaxation. The patient was qualified for further conservative management. She has remained under cardiology outpatient followup for one year, with no further cardiac events.

DISCUSSION

Among pregnant and postpartum women, myocardial infarction (PAMI) is a significant cause of morbidity and mortality. Its incidence is difficult to estimate. Based on a 2017 metaanalysis by Gibson et al., PAMI occurs at a rate of 3.34 per 100,000 pregnancies, with a maternal mortality rate of 0.20 per 100,000 pregnancies.[8] Acute myocardial infarction (AMI) occurs more frequently in pregnant women than in non-pregnant women of reproductive age, with hormonal and haemodynamic changes typical of pregnant patients considered the primary contributors. Additional risk factors include maternal age, hypertension, gestational diabetes, smoking, and thrombophilia [9, 10]. Spontaneous coronary artery dissection is recognised as the most common cause of myocardial infarction in pregnant and postpartum women, despite its rarity in the general population [3, 5, 11, 12]. This may be related to hormonal changes occurring during pregnancy, particularly in the peripartum period, which affect oestrogen and progesterone receptors in coronary vessels. These changes may weaken the arterial wall and lead to vessel injury, resulting in clinical symptoms [3, 11].

Two types of SCAD are distinguished. Spontaneous coronary artery dissection occurring during pregnancy or within 12 weeks postpartum is defined as pregnancy-associated SCAD (P-SCAD). In contrast, spontaneous coronary artery dissection unrelated to pregnancy (NP- SCAD) may occur at any time in a woman's life [5]. In the presented case, apart from gestational diabetes, no identifiable risk factors for acute myocardial ischaemia were present. The patient was young and had no pre-existing comorbidities. It is possible that the cumulative hormonal changes of a second pregnancy contributed to weakening of the coronary arterial wall and the onset of myocardial ischaemia. The occurrence of P-SCAD may be associated with very high progesterone levels and rapid hormonal fluctuations in the peripartum period [5, 13]. Additionally, both physical and emotional stress associated with labour may further contribute to AMI. The timing of symptom onset in this case confirms that the early postpartum period is associated with increased risk of complications, particularly when pregnancy is completed by caesarean section or when labour is complicated by postpartum haemorrhage. Most acute cardiac events associated with P-SCAD described in the literature occurred in the third trimester or within 30 days postpartum [5, 14]. According to Saw et al., as many as 64% of patients with coronary artery dissection developed symptoms immediately before delivery or within one week postpartum [15]. Due to a nonspecific chest or back pain and to the young age and absence of typical risk factors, acute myocardial ischaemia may not be initially suspected in patients with SCAD. Symptoms such as chest or back pain in pregnant or postpartum women should prompt clinical vigilance, and ECG should be performed. In this case, ECG demonstrated ST-segment elevation in leads V5 - V6, and troponin T levels were elevated. In an analysis of 120 P-SCAD cases by Havakuk et al., 75.5% of patients presented STsegment elevation myocardial infarction, and 72% of SCAD cases were located in the left anterior descending artery [16]. A more common ST-segment elevation in ECG is caused by more frequent changes in left coronary artery, which may result in more extensive myocardial injury [17]. In the presented case, critical occlusion of the left anterior descending artery was diagnosed during urgent coronary angiography, and five stents were implanted. Coronary angiography remains the gold standard for diagnosing coronary artery disease, with the additional advantage of enabling therapeutic interventions such

as angioplasty or stenting. Intravascular ultrasound or optical coherence tomography may be used during angiography for more precise diagnosis, particularly when SCAD is suspected [18–20]. Appropriate diagnostic planning and timely treatment in this case enabled rapid reperfusion of the occluded vessel. SCAD management includes conservative (watch-fulwaiting) strategies, pharmacotherapy as an adjunct to conservative management, revascularisation, or coronary stenting. Stenting is recommended in cases of significantly occluded coronary arteries, haemodynamic instability, persistent chest pain, or persistent ST-segment elevation [5]. Unfortunately, in SCAD, stenting may extend the dissection, impair healing, or contribute to vessel occlusion [3, 21]. It should also be noted that patients with P-SCAD are at higher risk of treatment failure and complications such as coronary artery bypass grafting (CABG), cardiogenic shock, mechanical circulatory support, or maternal death [5, 16]. Therefore, appropriate treatment selection and long-term follow- up are crucial. In the presented case, early pericarditis developed after acute myocardial ischaemia, as pericardium responded to standard therapy. No longterm complications occurred, and echocardiography demonstrated a left ventricular ejection fraction above 48%. The patient remains under long-term follow-up.

CONCLUSIONS

Young women with low cardiovascular risk and without clinically significant risk factors may, under the influence of multiple factors inherent to the peripartum period, develop acute myocardial ischaemia. Women in the postpartum period are particularly exposed to the risk of spontaneous coronary artery dissection due to intense physical exertion, emotional stress, and Valsalva manoeuvres during labour. The presented case highlights the need for close monitoring of maternal wellbeing in the early postpartum period and prompt response to any concerning symptoms. It also indicates the importance of close interdisciplinary collaboration to ensure the highest standard of perinatal care.

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CONFLICT OF INTEREST

The Authors declare no conflict of interest

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