CASE STUDY

CONTENTS

Acute coronary syndrome in post-partum period: challenge for differential diagnosis and proper management

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ABSTRACT

The patient was presented with chest pain, new ECG changes indicating ischemia, an increase in troponin and NT pro BNP, elevated D-dimer levels, and wall motion abnormalities on an Echo. A chest computed tomography angiography did not show pulmonary embolism but revealed a sac-like aneurysm of the left anterior descending artery (LAD). The coronary angiogram confirmed an aneurysm of the LAD in 6-7 segments, along with an extended 80% stenosis of the a. intermedia. The interventional cardiology team concluded that the patient required coronary artery bypass grafting (CABG) due to extensive vessel damage. She underwent CABG three months after acute coronary syndrome in an experienced center.

KEY WORDS: Acute coronary syndrome, coronary artery aneurysm, post partum period

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INTRODUCTION

Despite significant improvement in cardiovascular outcomes in the nonpregnant population, maternal mortality due to cardiac disease has not decreased in almost 20 years, especially for acute coronary syndrome (ACS)[1]. Although ACS is rare during pregnancy and postpartum period, it still increases the risk of acute myocardial infarction (AMI) 3- to 4-fold [2]. It accounted for more than 20% of all cardiac deaths in the latest UK triannual enquiry [3]. AMI is often caused by atherosclerotic coronary artery disease (CAD). This risk increases with factors such as smoking, older maternal age, hypertension, diabetes mellitus, obesity, and dyslipidemia. [4,5]. It is expected that the incidence of ACS will increase in the coming years because the pregnant population is becoming older, more overweight, and with more medical co-morbidities [2]. The cause of the higher susceptibility to ACS during pregnancy or early postpartum period is not entirely clear, but it may be due to hormonal influence (especially oestrogen and progesterone), an increase in vessel shear stress caused by pregnancy and labour, as well as hypercoagulability that persists for weeks after delivery [6].

The causes of ACS are more often non-atherosclerotic, including coronary artery dissection, coronary artery spasm, and coronary thrombosis [7]. Chest pain during pregnancy and in the postpartum period can have a wide range of possible causes, but ACS should be considered as one of them. The similarity between pregnancy-related symptoms and those of ACS may lead to a delayed diagnosis. Additionally, ECG changes during pregnancy and postpartum period, which involve inverted T-waves, can further complicate the diagnosis. However, it is worth noting that serum troponins are typically not affected, although they may be found in cases of pre-eclampsia [8].

CASE REPORT

A 35-year-old female presented to our department approximately two weeks postpartum after a term, singleton vaginal delivery with a chief complaint of a squeezing chest discomfort, radiating to both arms, started after physical activity. Associated symptoms were shortness of breath and palpitations at rest. She had no known risk factors for atherosclerotic disease, smoking, alcohol or drugs abuse and no history of any connective tissue disorder or infectious diseases. The course of her pregnancy had been uncomplicated.

On admissiom her respiratory rate was 22/min, heart rate – 90/min, blood pressure – 140/90 mm of Hg, oxygen saturation – 94 % on room air, temp. – 36.6°C, BMI – 28 kg/m2. Chest auscultation did not reveal any obvious abnormal heart murmurs. ECG demonstrated ST segment elevation in II, III, AVF and ST segment depression with T wave inversion in V2-V3 leads (Fig.1). Bedside transthoracic echocardiography showed intra-ventricular septal dyssynchrony and hypokinesia of basal and middle segments of anterior wall of left ventricle with preserved ejection fraction 55 %. Cardiac biomarkers were found to be elevated on the day of admission: high-sensitivity troponin T – 334 ng/l (0-24.5

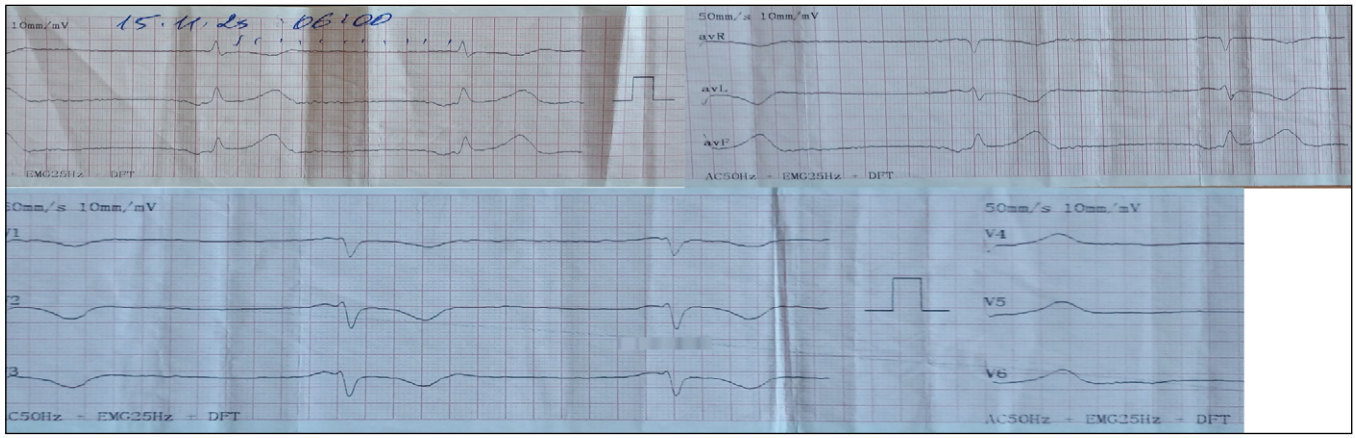

Fig. 1. Initial ECG.

ng/l), NT pro BNP – 700.3 pg/ml (0-125 pg/ml), D-dimer – 3.25 mcg/ml (< 0.5 mkg/ml), C reactive protein 18.92 mg/ml (< 5.0 mg/ml).

The main differential diagnosis were ACS, pulmonary embolism, myocarditis. A chest computed tomography angiography (CTA) did not reveal pulmonary embolism but showed sac-like aneurysm of left anterior descendent artery (LAD) 13 mm in diameter (Fig.2, Fig.3). Coronary angiogram revealed aneurysm of LAD in 6-7 segments, extended 80 % stenosis of a. intermedia and 40 % stenosis of right coronary artery (RCA) in 1 segment (Fig.4).

Upon consultation with cardiothoracic surgery, the interventional cardiology team determined that the patient required coronary artery bypass grafting due to extensive vessel damage. The breastfeeding was discontinued, and she was started on a double anti-platelet therapy.(aspirin 100 mg and clopidogrel 75 mg), enoxaparin 0.6 ml twice daily, nebivolol 2,5 mg, enalapril 5 mg and rozuvastatin 10 mg. In 2 weeks ECG after the event showed resolution of MI pattern (Fig.5). She underwent CABG in 3 monthes after ACS in experienced centre. During follow up the patient remains asymptomatic.

The presence of coronary artery aneurysm (CAA) or ectasia has been associated with poor long-term outcomes irrespective of the presence of concomitant atherosclerotic coronary artery disease [10]. Clinical presentations range from incidental finding on cardiac imaging to ACS [11]. The pathogenesis of CAA is not clear. However, there can be associations between certain risk factors in particular:

- an individual genetic susceptibility in patients with congenital CAA [12].
- systemic connective tissue diseases with vasculitis (e.g., Kawasaki disease, Marfan, and so on)
- atherosclerosis lesion
- iatrogenic CAA caused by intracoronary manipulation (PCI, brachytherapy etc)
- post-infectious CAAs due to infectious direct wall invasion or immune complex deposition [10].

Thrombosis and distal embolization are the most likely causes of an ACS-like presentation in patients with CAA but without underlying coronary artery obstruction or atherosclerosis. Thrombosis usually develops in the aneurysm, which is often asymptomatic. Patients start experiencing symptoms when the thrombus either spreads and blocks the coronary artery, usually proximal to the aneurysm, or breaks apart and gets lodged in a distal artery causing an embolic phenomenon [13].

Coronary aneurysm can be a result of obstructive ischemic coronary artery disease with decreased flow volume. This can lead to exercise-induced myocardial ischemia [14]. The coronary flow reserve is also significantly reduced in coronary aneurysms [15].

Dilatation of the coronary arteries can lead to disturbed coronary flow, which in turn can increase blood viscosity and activate coagulation. This pathophysiological element may cause thrombotic occlusion of ectatic coronary arteries. Another possible mechanism is the elevated level of inflammatory cytokines in CAE subjects, which can activate the coagulation cascade and increase the risk of acute coronary events. [16].

Matrix metalloproteinases (MMPs) have been shown to be involved in the pathogenesis of CAA formation by causing increased proteolysis of extracellular matrix proteins [17]. The MMP-3 5A allele is linked with higher promoter activity for transcription of the gene, and is more frequently seen in patients with CAA and atherosclerosis compared to patients with only coronary atherosclerosis.[18, 20]. The presence of CAA can predict future cardiac events in patients with acute MI. As a result, these patients might benefit from a pharmacological approach to controlling the coagulation cascade [19].

IMAGING OF CAA

Coronary angiography is considered the most reliable diagnostic tool for the detection of CAA. It is also useful

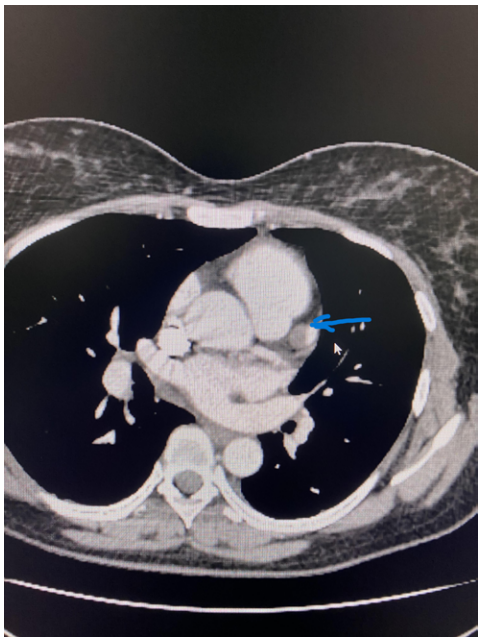

Fig. 2. CT angiography 1.

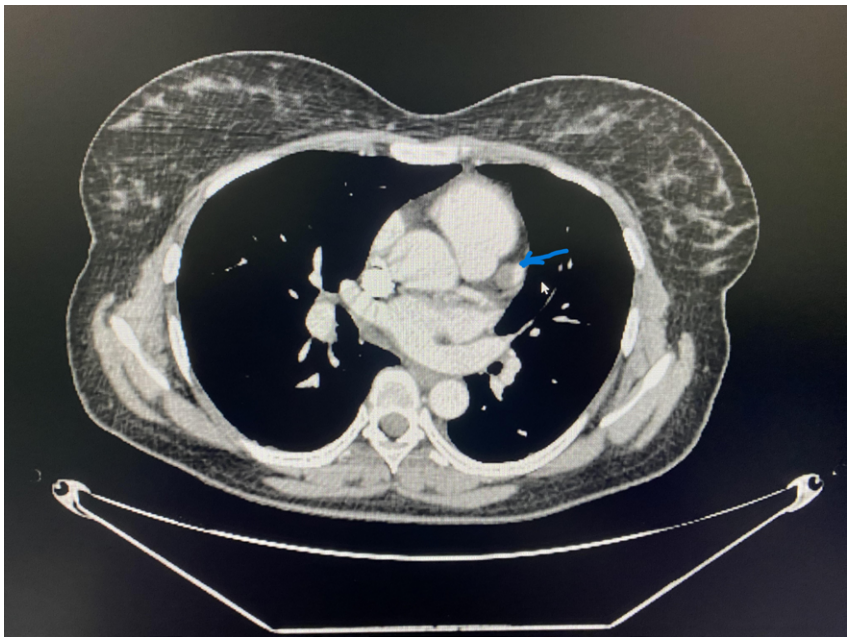

Fig. 3. CT angiography 2.

in determining the optimal strategy for surgical resection. Other imaging modalities include transthoracic echocardiography, ECG-gated CT angiography, MRI and/or MR angiography, and angiographic cardiac catheterization [21]. Proper imaging helps to evaluate aneurysm shape and structure, its morphology (fusiform or saccular), diameter, wall calcification, luminal thrombosis, presence of associated stenosis, origin and termination, monitoring of growth rate, exclude potential complications, myocardial perfusion abnormalities, fistula formation, extrinsic mass compression, rupture and hemopericardium [21]. CT coronary angiography is a highly sensitive tool for detecting CAAs and provides clear visualization of the coronary lumen. It highlights intraluminal thrombi. In our case, it was a superior imaging revealed the cause of ACS in our patient. CT coronary angiography yields a versatile post-processing: maximum intensity projections, curved multiplanar reformations and 3D volume rendering clearly identify the anatomical relationships of the aneurysm with the surrounding structures [20, 22]. Intravascular ultrasound (IVUS) is a highly reliable technique used to produce clear images of the coronary arteries. It provides information about the composition of the lumen and arterial wall structure, making it an effective tool in differentiating between true and false aneurysms caused by plaque rupture. IVUS is considered the "gold standard" technique for this purpose [20, 23].

TREATMENT MANAGEMENT OF CAA

The treatment of CAA involves medical management, surgical resection, and stent placement. However, the appropriate treatment for CAAs depends on the specific clinical situation. Currently, there are no randomized trials to evaluate different management strategies and their outcomes in these patients. Due to a high risk of thromboembolism, antiplatelet and anticoagulant medications should be administered [20]. In our case, the patient received enoxaparin, aspirin and clopidogrel at hospital and was discharged with dual antiplatelet therapy.

Excessive TGF-β and metalloproteinase have been linked to the development and progression of CAAs. Angiotensin II type-1 receptor antagonists can inhibit TGF-β [24]. Statins have been shown to inhibit the secretion of metalloproteinases 1, 2, 3, and 9 from macrophages and vascular smooth muscle cells [20]. Therefore, these drugs may be helpful in treating and preventing the progression of CAA. However, there are currently no long-term randomized data available.

In patients with ACS due to CAA culprit, the emphasis is to restore flow. Percutaneous coronary intervention of an aneurysmal/ectatic culprit vessel had lower procedural success and a higher incidence of no-reflow and distal embolization [10]. In cases where PCI is not a viable option for symptomatic patients, it is recommended to perform surgical excision or ligation of CAA along with bypass grafting of the affected coronary arteries [20, 25]. The surgical treatment of CAA is indicated in cases of severe coronary artery disease, when the aneurysms are located near the bifurcation of large branches, when there is evidence of emboli from the aneurysm to the distal coronary bed resulting in myocardial ischemia, when there is progressive enlargement of a CAA documented by serial angiographic measurements, and when there are complications such as fistula formation, compression of cardiac chambers, or giant

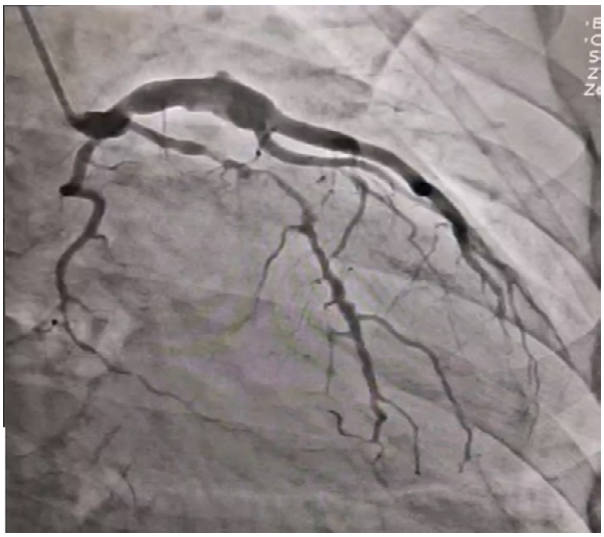

Fig. 4. Coronarography.

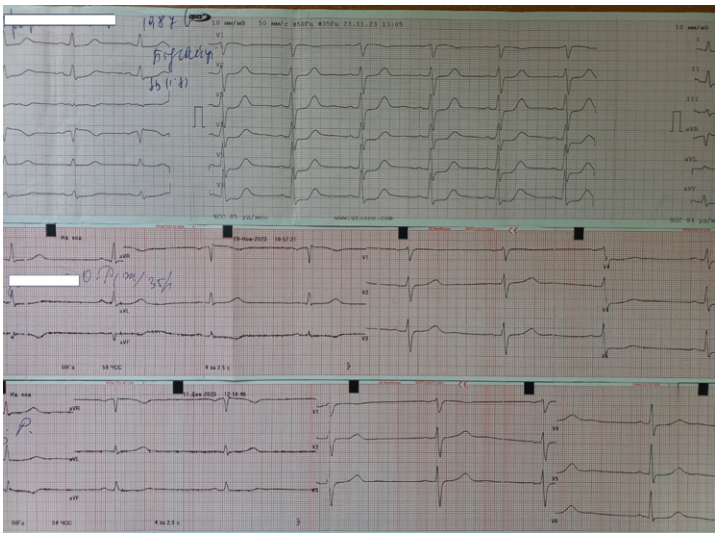

Fig. 5. Follow up ECG.

CAA (dilatation exceeding the reference vessel diameter by more than four times) [20].

CONCLUSIONS

Chest pain during pregnancy or in the postpartum period prompts a broad spectrum of differential diagnoses, and ACS should always be considered. CAA is a rare disorder, which can be presented as a random findings on cardiac imaging or ACS with life-threatening consequences. During the early postpartum period managing of CAA is still a challenge. It's crucial to know variants of clinical presentation, perform accurate imaging assessments and access interventional or surgical treatment to achieve optimal results. To improve patient care and outcomes, it's essential to conduct prospective studies and maintain registries.

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CONFLICT OF INTEREST

The Authors declare no conflict of interest

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