Проблеми екології та медицини

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CLINICAL CASE/КЛІНІЧНИЙ ВИПАДОК

ACUTE MYOCARDIAL INFARCTION IN A PUERPERA: A CASE REPORT

[3]Fylenko B. M., [3]Roiko N. V., [1]Khazanov O. P., [2]Romaniuk A. M., [3]Cherniak V. V.

[1]Poltava Regional Bureau of Autopsy, Poltava, Ukraine
[2]Sumy State University, Sumy, Ukraine
[3]Poltava State Medical University, Poltava, Ukraine

Address for correspondence:
B. Fylenko, 23, Shevchenko Str., Poltava State Medical University, Poltava, Ukraine, 36011

E-mail: borysfylenko@gmail.com

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We report a lethal case of acute myocardial infarction in a puerpera. Cardiovascular diseases rank as the leading causes of mortality during pregnancy and the postpartum period, with myocardial infarction (MI) being a rare yet frequently fatal occurrence. Research indicates that MI typically manifests between 6 to 12 weeks postpartum, though instances may also occur at later stages.

The reported case is interesting in terms of the mismatch between the timing of clinical symptoms of acute coronary syndrome (less than 24 hours) in the puerpera and alterations in myocardial infarction (2-3 days), which was diagnosed morphologically. Therefore, we searched for possible causes that led to the clinical and morphological changes described above. At first glance, the main cause of myocardial infarction development is coronary artery dissection with the formation of hematoma in the wall of the blood vessel, which narrows its lumen and leads to necrosis of the heart muscle. A brief analysis of the case has established that ischemic damage to the myocardium preceded the development of spontaneous coronary artery dissection and could be caused by one of the factors or a combination of them: coronary artery spasm caused by ergometrine and anemia due to uterine atony-related postpartum hemorrhage. The formation of intramural hematoma of the vascular wall, which narrowed the lumen of the coronary artery, could be the result of the prescription of antithrombotic therapy, which complicated the health status of the woman and led to death. When providing medical care to puerperas with postpartum hemorrhage, it is necessary to take into account the risk for cardiovascular complications that could develop due to anemia or administration of ergometrine.

Key words: ergometrine, thrombolytics, coronary angiography, spontaneous coronary artery dissection.

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Introduction

Cardiovascular diseases rank as the leading causes of mortality during pregnancy and the postpartum period, with myocardial infarction (MI) being a rare yet frequently fatal occurrence. Research indicates that MI typically manifests between 6 to 12 weeks postpartum, though instances may also occur at later stages [1].

Beyond pregnancy as an independent factor for myocardial infarction (MI) development, specific conditions such as preeclampsia, eclampsia, thrombophilia, blood transfusion, and spontaneous coronary artery dissection (SCAD) during pregnancy elevate the incidence of MI by 3-4 times compared to non-pregnant women of reproductive age. Meanwhile, in the latter group, smoking, hypertension, diabetes mellitus, obesity, and atherosclerosis persist as the primary risk factors [2, 3].

Case report

A 30-year-old female patient, D., underwent a 10-hour treatment for acute myocardial infarction at the regional hospital. Past medical history revealed that 4 days before hospitalization, the patient gave birth to a child at 36 weeks of gestation. It was the second childbirth, complicated by the 2nd degree of perineal tear and hypotonic hemorrhage in the early postpartum period. The medical intervention included manual revision of the uterine cavity walls, removal of blood clots, and intravenous administration of ergometrine, tranexamic acid, and oxytocin in accordance with treatment protocols. Subsequent to these measures, the uterus contracted, and hemorrhage ceased, resulting in a total blood loss of 650 ml. The patient was discharged in satisfactory condition on the fourth day.

On her way home, she experienced sudden retrosternal pain, weakness, and shortness of breath. Seeking medical assistance from a family doctor, she was subsequently referred to the regional hospital with a diagnosis of "acute coronary syndrome."

Upon admission to the hospital, the patient underwent comprehensive clinical, laboratory, and instrumental examinations. Chest CT revealed indications of congestion in the lesser circulation, minor pulmonary hypertension, bilateral hydrothorax of small volume, and a relative enlargement of the left atrium.

The laboratory tests showed RBC – 2.79 × 10^{12}/L, HGB – 86 g/L, WBC – 12.6×10^{9}/L, total protein – 38 g/L, fibrinogen – 5.2 g/L, the Quick Time (prothrombin index) – 181.9%, D-dimer – 1.10 μg/ml; Troponin T – 2.0 ng/ml (normal range <0.03 ng/ml); aspartate aminotransferase – 257 U/l. Other blood indices were unchanged.

ECG showed sinus rhythm, 70 beats per minute, the normal position of the electrical axis of the heart, and elevation of the ST-segment in II, III, and avF leads.

The echocardiogram (EchoCG) disclosed diminished contractility in the left ventricle (LV) with a left ventricular ejection fraction (EF) of 44%. Hypokinesis was noted in the middle and lower thirds of the interventricular septum, as well as in the apex and lower third of the posterior wall of the left ventricle. Additionally, a slight enlargement of the LV cavity was observed. The examination revealed relative grade I-II regurgitation of the mitral and tricuspid valves, along with indications of pulmonary hypertension.

The patient underwent coronary angiography, revealing unimpeded patency in the coronary arteries. Spasm was observed in the proximal left and right coronary arteries, subsequently alleviated through the intra-arterial administration of nitroglycerin.

The findings of the clinical, laboratory, instrumental tests and examination by a cardiologist were considered and the diagnosis was made: Acute Q-wave circular myocardial infarction, type II; grade II regurgitation of the mitral and tricuspid valves; pulmonary hypertension (systolic pressure in the pulmonary artery 33 mm Hg); heart failure IIa with moderately reduced LV ejection fraction (EF 44%), functional class III; stage III arterial hypertension, the risk is very high. Day 4 of the postpartum period; uterine subinvolution, lochiometra.

Despite the treatment provided according to the established diagnosis (use of Isosorbide dinitrate, Ringer's solution, Clopidogrel, Magnicor, Nadroparin, Pantoprazole, Magnesium sulfate, Gecodes, Bisoprolol, Meropenem, Metronidazole) the patient's clinical status continued to decline and she died within 15 hours after the onset of the first symptoms of the disease.

The autopsy and histological examination of the necropsy material has established that the patient D. developed inter vivos acute transmural myocardial infarction of the anterior-membranous-apical part of the left ventricle in the postpartum period (approximately within the period of 2-3 days on morphological grounds). Microscopically, it was manifested by dystrophic and necrotic alterations of cardiomyocytes with their fragmentation, loss of transverse striation, karyolysis, and diffuse leukocyte infiltration of these areas (Fig. 1a, 1b).

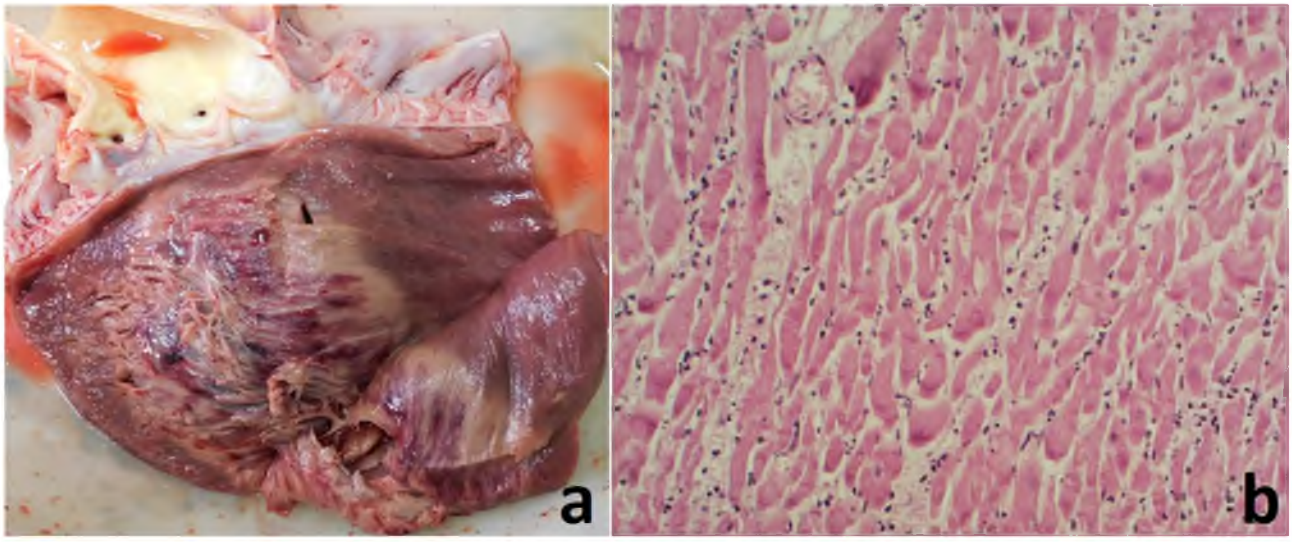

Fig. 1. Morphological alterations in myocardial infarction in the puerpera D.:
a – macroscopic picture; b – microscopically (H&E stain, ×100 magnification).

It has been established that the probable cause of myocardial infarction was the dissection of the wall of the left coronary artery and the left anterior descending artery between the media and adventitia with the formation of intravascular hematoma, which significantly narrowed the lumen of these arteries. Adventitia showed plethora of blood vessels and moderate infiltration by lymphocytes and plasma cells (Fig. 2).

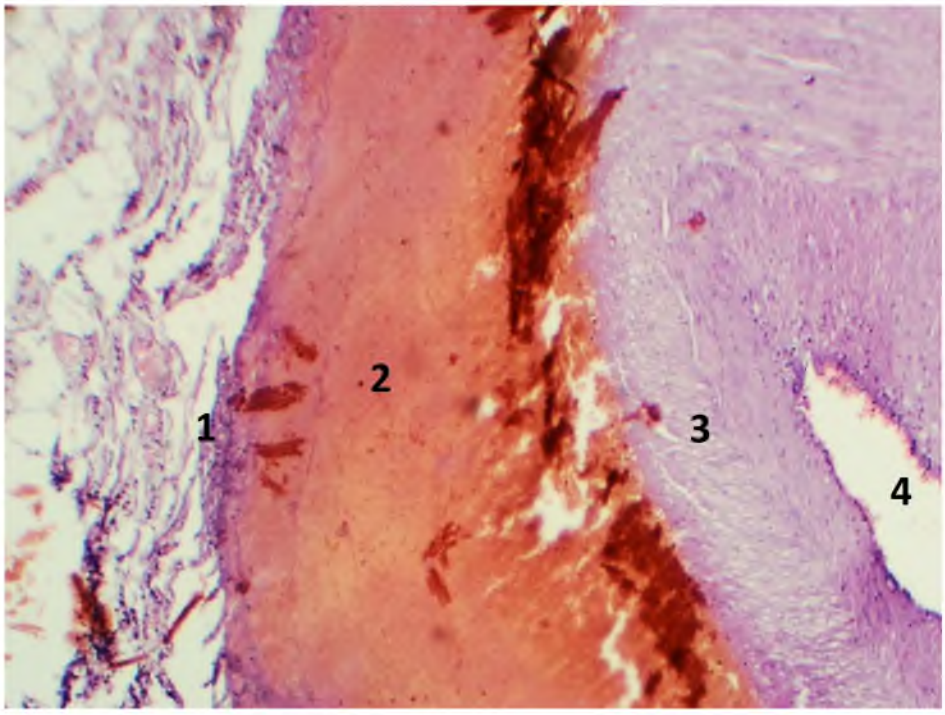

Fig. 2. Dissection of the wall of the left coronary artery in the puerpera D.
with myocardial infarction (H&E stain, ×40 magnification):
1 – adventitia, 2 – hematoma, 3 – media of the coronary artery; 4 – the lumen of the coronary artery.

It has been found that myocardial infarction was complicated with right-sided (500 ml of clear fluid in the pleural cavity) and left-sided (200 ml of fluid) hydrothorax, pulmonary edema, cerebral edema, and necrotic nephrosis.

Discussion

Current medical science is concerned with the study of morphological features of the heart and its lesions in pathology, as well as the analysis of etiopathogenetic parallels in the development of diseases, both in typical and atypical manifestations of the disease

[4]. The reported case is interesting in terms of the mismatch between the timing of clinical symptoms of acute coronary syndrome (less than 24 hours) in the puerpera and alterations in MI (2-3 days), which was diagnosed morphologically. Therefore, we searched for possible causes that led to the clinical and morphological changes described above.

At first glance, the main cause of myocardial infarction development is coronary artery dissection with the formation of hematoma in the wall of the blood vessel, which narrows its lumen and leads to necrosis of the heart muscle.

Publications report that SCAD most often develops in the left anterior descending coronary artery (72% of cases) and less often in the main trunk of the left coronary artery (36%), right coronary artery (20%), circumflex artery (4%) and 40% of SCAD cases occur simultaneously in several arteries. SCAD is most often accompanied by the development of MI with elevation of the ST segment in the anterior part of the left ventricular wall. The relationship between SCAD and pregnancy has not been fully studied to date. It is hypothesized that hormonal changes that occur during this period affect the normal structure and composition of vascular connective tissue, which is the background for the dissection of their walls under the influence of hemodynamic disorders in pregnant women, as well as other factors [5,6,7].

However, the analysis of the reported case has shown that SCAD could not be the cause of MI, since coronary angiography revealed no alterations that would indicate narrowing of the wall of the left coronary artery and left anterior descending artery, and only spasm of the proximal left and right coronary arteries was observed that was arrested by administration of nitroglycerin. Apparently, transmural myocardial infarction can be developed during spontaneous or forced spasms of the proximal coronary arteries or microcirculatory blood vessels, characterized by the development of MI without coronary obstruction with ST-segment abnormalities on the ECG [8]. In this case, coronary spasm could be caused by the administration of ergometrine, which is a second-line uterotonic after oxytocin in atonic uterine hemorrhage. Administration of this medication and its analogues is accompanied by the risk for the development of serious side effects on the cardiovascular system, including in young women, who do not have any risk factors for cardiovascular diseases. Moreover, hypoxic myocardial infarction in puerperas might be developed as a result of postpartum hemorrhage, leading to anemia and decreased circulating blood volume. Anemia and signs of hypovolemic shock may be unrecognizable because visual assessment of blood loss is not objective [9].

In the reported case, coronary artery dissection might be a secondary process that was developed as a complication of antithrombotic therapy and administration of heparin during coronary angiography. An insignificant number of publications report that thrombolytics, anticoagulants, and antiplatelets, prescribed to patients, can lead to the development of SCAD or the progression of arterial dissection until its rupture [7]. Since the hematoma, found during the pathomorphological examination, was localized between the adventitia and the media, it can be assumed that its formation is caused by spontaneous hemorrhage from the vasa vasorum. The presence of cellular inflammatory infiltrate in the surrounding tissues indicates its inter vivos formation [10].

A brief analysis of the reported case has established that ischemic damage to the heart muscle preceded the development of spontaneous coronary artery dissection and could be caused by one of the following factors or a combination of them: coronary artery spasm caused by ergometrine and anemia due to uterine atony-related postpartum hemorrhage, hypovolemia. The formation of intramural hematoma of the vascular wall, which narrowed the lumen of the coronary artery, could be the result of the prescription of antithrombotic therapy, which complicated the health status of the woman and led to death.

Conclusion

When providing medical care to puerperas with postpartum hemorrhage, it is necessary to take into account the risk for cardiovascular complications that could develop due to anemia or administration of ergometrine. Further search for causes, the study of pathogenetic links, and the improvement of diagnostic criteria for myocardial infarction in puerperas will contribute to better survival and prognosis.

ORCID authors:

Fylenko B. M.
https://orcid.org/0000-0002-8659-2267
Roiko N. V.
https://orcid.org/0000-0001-7478-0773
Khazanov O. P.
https://orcid.org/0009-0003-5664-9722
Romaniuk A. M.
https://orcid.org/0000-0003-2560-1382
Cherniak V. V.
https://orcid.org/0000-0003-4594-565X

Authors contributions:

Fylenko B. M.[DF]
Roiko N. V.[AF]
Khazanov O. P.[BF]
Romaniuk A. M.[EF]
Cherniak V. V.[CF]

A – conception and design of the study; **B** – data collection; **C** – data analysis and interpretation; **D** – writing the article; **E** – revising the article; **F** – final approval of the article

Conflict of interest:

The authors declare no conflict of interest

Ethical approval:

Ethics approval and concent to participate the study was approved by the Committee on Bioethics and Ethical Issues of Poltava State Medical University (Minutes No. 221 of 22.11.2023).

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ГОСТРИЙ ІНФАРКТ МІОКАРДА У ПОРОДІЛЛІ: ВИПАДОК З ПРАКТИКИ

[3]Филенко Б.М., [3]Ройко Н.В., [1]Хазанов О.П., [2]Романюк А.М., [3]Черняк В.В.

[1]Poltava Regional Bureau of Autopsy, Poltava, Ukraine

[2]Sumy State University, Sumy, Ukraine

[3]Полтавський державний медичний університет

Ми повідомляємо про летальний випадок гострого інфаркту міокарда у породіллі. Серцево-судинні захворювання є найпоширенішою причиною смерті під час вагітності та в післяпологовому періоді, а інфаркт міокарда – рідкісний випадок, але часто летальний. Повідомляється, що інфаркт міокарда найчастіше розвивається у період від 6 до 12 тижнів після пологів, хоча може виникнути і пізніше.

Описаний випадок цікавий з точки зору невідповідності між термінами клінічних симптомів гострого коронарного синдрому (менше 24 годин) у породіллі та змін при інфаркті міокарда (2-3 дні), який діагностовано морфологічно. Тому ми шукали можливі причини, що призвели до клініко-морфологічних змін, описаних вище. На перший погляд, основною причиною розвитку інфаркту міокарда є розшарування коронарної артерії з утворенням гематоми в стінці кровоносної судини, яка звузила її просвіт і призвела до некрозу серцевого м'яза. Короткий аналіз випадку встановив, що ішемічне ураження міокарда передувало розвитку спонтанного розшарування коронарної артерії та могло бути спричинене одним із факторів або їх комбінацією: спазмом коронарної артерії, спричиненим ергометрином, та анемією, спричиненою атонією матки з кровотечею у післяпологовому періоді. Утворення інтрамуральної гематоми судинної стінки, яка звузила просвіт коронарної артерії, могло бути наслідком призначення антитромботичної терапії, що ускладнило стан здоров'я жінки та призвело до смерті. При наданні медичної допомоги породіллям з післяпологовою кровотечею необхідно враховувати ризик розвитку серцево-судинних ускладнень, які можуть розвинутися внаслідок анемії або введення ергометрину.

Ключові слова: ергометрин, тромболітики, коронарографія, спонтанне розшарування коронарної артерії.