Acute postpartum dyspnea: is it a simple or a complicated item?

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ABSTRACT

Background: Peripartum cardiomyopathy (PPCM), like spontaneous coronary artery dissection (SCAD), is a disease that affect healthy women and occur in the days before and after delivery.

Case Presentation: We discuss the case of a 26-year-old woman, already a mother of a boy, who gave birth to two healthy twin girls by cesarean section. Two hours after delivery, the woman suddenly developed acute dyspnea and chest pain, which had angina pectoris features and was permanent. Her electrocardiogram showed sinus tachycardia at 125 beats per minute, with no specific STsegment deviation at 12 precordial (on the ECG), but also right and posterior leads. Echocardiography showed a significant reduction in left ventricular systolic performance, with an estimated LVEF (Left Ventricular Ejection Fraction) of approximately 25%-30%, and significant hypokinesis in the mid-distal part of the interventricular septum and the apex of the left ventricle. Her blood examination revealed an increase in troponin-T high sensitivity (0.245 ng/ml), with a mild parallel increase in creatine phosphokinase, as well as the MB (isoenzyme of CPK, the most specific indicator for the myocardial infarction diagnosis) form (383 and 52 IU/I, respectively). The NT-pro BNP (N-terminal pro Brain Natriuretic Peptide) was mildly increased, too, at 1280 pg/ml. The chest pain remained until the next 36 hours when it gradually disappeared, as the dyspnea, too. She was completely recovered until the 10th day of hospitalization. The patient underwent coronary computed tomography angiography, which showed 0 Agatston coronary artery calcium score units. However, in the middle of the left anterior descending (LAD) artery, after the protrusion of the second diagonal branch, an eccentric 7 mm non-calcified lesion was observed, which caused mild stenosis (1%-24%).

Conclusion: PPCM and SCAD are related to young healthy women and occur in the peripartum period. However, in some cases, their clinical picture may overlap, making the diagnosis challenging.

Keywords: Peripartum cardiomyopathy, spontaneous coronary dissection, differential diagnosis.

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Background

Peripartum cardiomyopathy (PPCM), like spontaneous coronary artery dissection (SCAD), is a rare disease that affects healthy women and occurs on the days before and after delivery. Although the pathophysiological mechanism [1] is different between the two (autoimmunity, myocarditis, etc. vs. spontaneous dissection), as is their treatment, nevertheless their clinical picture may present common points that create confusion at the time of diagnosis. Spontaneous dissection of the coronary arteries [2] is a rare cause of acute coronary syndrome in the general population. It usually affects women during pregnancy or more often 3 months postpartum.

The case of a woman who initially appeared to be PPCM is described. However, the clinical picture leads to a differential diagnosis.

Case Presentation

A 26-year-old woman, already the mother of a boy, gave birth to two healthy twin girls by cesarean section. During

pregnancy, the woman did not present any complications. Two hours after delivery, the woman suddenly developed acute dyspnea and chest pain, which had angina pectoris features and was permanent. There was a reduction in her oxygen saturation to 76%, with a parallel decrease in the partial pressure of oxygen, while crackles were heard when listening to her lungs. From the auscultation of the heart, it was found a mild blowing holosystolic murmur, which was heard best at the apex. Her blood pressure was normal (110/67 mmHg), while her electrocardiogram (Figure 1) showed sinus tachycardia at 125 beats per minute, with no specific ST-segment deviation at 12 precordial, but also at right and posterior leads. It was only observed a remarkable OTc (OT segment on the ECG corrected) prolongation, at 707 ms. From the chest X-ray, there were no noticeable signs of congestion, nor pleural effusion. She was admitted to the intensive care unit and immediately underwent non-invasive ventilation, as well as intravenous diuresis, as well as b-blocker therapy, to slow down her heart rate.

Echocardiography showed a significant reduction in left ventricular systolic performance, with an estimated LVEF of approximately 25%-30%, and significant hypokinesis in the mid-distal part of the interventricular septum and the apex of the left ventricle (Figure 2). Her blood examination revealed an increase in troponin-T high sensitivity (0.245 ng/ml), with a mild parallel increase in creatine phosphokinase, as well as the MB isoenzyme (383 and 52 IU/l, respectively). The NT-pro BNP was mildly increased, too, at 1,280 pg/ ml. The chest pain remained until the next 36 hours when it gradually disappeared, as the dyspnea, too. Laboratory tests gradually returned to normal, while left ventricular ejection fraction improved on the third day (40%-45%), and it was completely recovered until the 10th day of hospitalization. The patient underwent coronary computed tomography angiography, which showed 0 Agatston coronary artery calcium score units. However, in the middle of the LAD artery, after the protrusion of the second diagonal branch, an eccentric 7 mm non-calcified lesion was observed, which caused mild stenosis (1%-24%) (Figure 3).

After 3 months the ECHO (Echocardiogram) parameters were improved. Clinically she was doing well with no symptoms (Figure 4).

Discussion

Pregnancy is one of the causative factors of the occurrence of SCAD in young women. It usually occurs during the first month after delivery and most of them in the 1st week [3]. It usually affects the performance of the left ventricle, while angiographically usually there is a multivessel disease. It usually appears to be associated with hormone fluctuations during pregnancy, as well as with oxytocin release in breastfeeding mothers. It also seems to be associated with the onset of eclampsia or pre-eclampsia, but also with the occurrence of PPCM [4]. Hypertensive disorders, as well as pre-eclampsia, may share common pathogenetic mechanisms, in terms of small vessels, with PPCM, but also with SCAD. In addition, SCAD appears to be associated with extracoronary vascular abnormalities, as well as fibromuscular dysplasia [5].

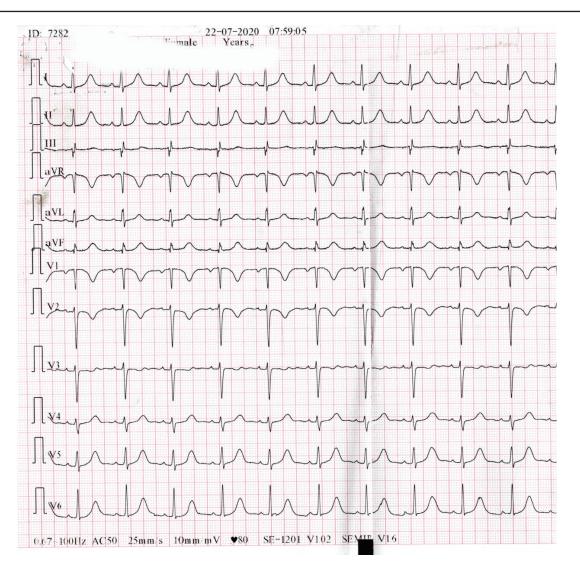


Figure 1. Electrocardiogram showed sinus tachycardia at 125 beats per minute, with no specific ST-segment deviation at 12 precordial, but also at right and posterior leads. It was only observed a remarkable QTc prolongation, at 707 msec.

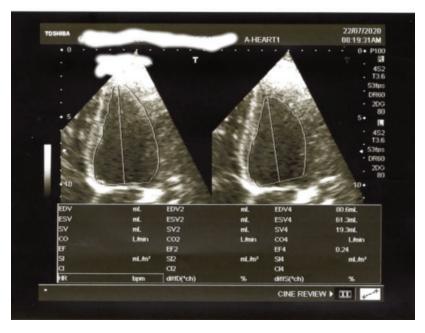


Figure 2. Significant reduction in left ventricular systolic performance, with an estimated LVEF of approximately 25%-30%, and significant hypokinesis in the mid-distal part of the interventricular septum and the apex of the left ventricle.

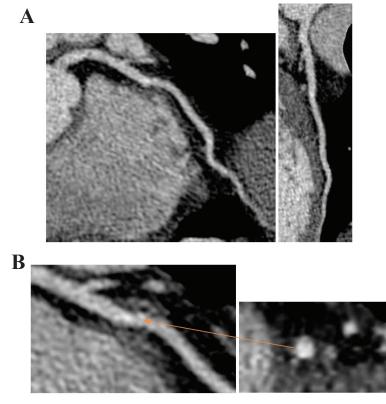


Figure 3. (A) The lower third of the LAD artery is inspected without significant lesions. (B) In the middle of LAD, after the protrusion of the second diagonal branch, an eccentric 7 mm non-calcified lesion was observed, which caused mild stenosis (1%-24%).

On the other hand, PPCM is another entity of pregnancy-related heart diseases. It occurs in the first postpartum months. Its onset can be subacute, with mild initial symptoms, but it can also begin as an acute heart failure [6]. It is a condition that presupposes left ventricular dysfunction, with left ventricular ejection fraction <45%, with or without impairment of the left ventricular cavity. Many times, differential diagnosis can include dilated cardiomyopathy, valvular heart disease, hypertensive disorders, pulmonary embolism, or even acute coronary syndrome [7].

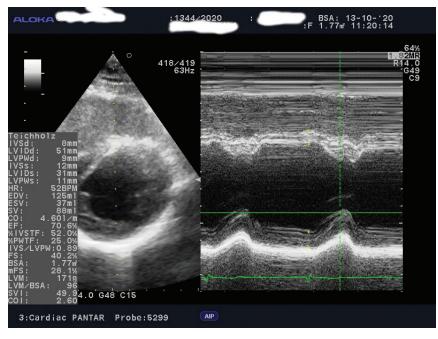


Figure 4. After 3 months the ejection fraction was improved to 70%.

Either of these two entities can be potentially fatal to the patient's life. Typically, SCAD may be dangerous if manifested as an ST-elevation myocardial infarction and involves the left main coronary artery or the orifices of the large epicardial vessels, or even if it is complicated with a cardiogenic shock [8]. PPCM, on the other hand, usually has a lower mortality rate, but may be associated with long-term use of cardiovascular medication, persistent hypertension, or the presence of arrhythmias [9].

The effect of necrosis that will be caused by the SCAD in the myocardium, has to do with the extent of the damaged myocardium. Other times, it may lead to a small increase in troponin, in the setting of a non-ST-elevation myocardial infarction, or even it may involve a large area of the myocardium and lead to death [10]. Also, the increase in the levels of troponin in the occurrence of PPCM predicted the persistence of left ventricular dysfunction, with an ejection fraction <50%, as well as an unfavorable prognosis in the next 6 months [11]. As far the electrocardiographic changes, that might be identified during an episode of PPCM, tachycardia and QTc prolongation >460 ms might have significant prognostic value, although modestly correlated with left ventricle's dimensions and stroke volume index [12].

SCAD and PPCM have many differences, especially in the pathogenetic background, but at the same time, they present some common characteristics, as far the clinical manifestations, which may overlap, and may create doubts regarding the initial diagnosis in an acute-onset episode of dyspnea and thoracic pain, in a young healthy woman [13].

In our case, given that the clinical evolution was very similar to that of PPCM, however, the sudden onset and intense presence of precardiac pain for two days, rise and fall of troponin, the lack of a predisposing factor for coronary heart disease, and this finding in the tomography, that could be a dissection flap, implies for the existence of a possible SCAD.

Conclusion

PPCM and SCAD are related to young healthy women and occur in the peripartum period. However, in some cases, their clinical picture may overlap, making the diagnosis challenging.

What is new?

PPCM, like SCAD, is a rare disease that affects healthy women and occurs in the days before and after delivery. Coronary computed tomography angiography is a contribution to the final diagnosis and management.

List of Abbreviation

ECHO	Echocardiogram
LVEF	Left Ventricular Ejection Fraction
MB	isoenzyme of Creatinine Phosphokinase (CPK) the
	most specific indicator for the myocardial infarc-
	tion diagnosis.
NT-pro BNP	N-terminal pro Brain Natriuretic Peptide
PPCM	Peripartum Cardiomyopathy
QTc	segment on the ECG, corrected according to the
	Bazzet's formula.
SCAD	Spontaneous Coronary Artery Dissection
ST	segment on the ECG

Conflict of interests

The authors declare that there is no conflict of interests regarding the publication of this case report.

Funding

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Consent for publication

Written informed consent was taken from the patient.

Ethical approval

Ethical approval is not required at our institution for publishing an anonymous case report.

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References

- Baris L, Cornette J, Johnson MR, Sliwa K, Roos-Hesselink JW. Peripartum cardiomyopathy: disease or syndrome? Heart. 2019;105(5):357–62. https://doi.org/10.1136/ heartjnl-2019-315060
- Vogiatzis I, Hadjimiltiades S, Sachpekidis V, Parcharidis G. Spontaneous coronary artery dissection and acute myocardial infarction during pregnancy. Hellenic J Cardiol. 2010;51(1):74–80.
- Tweet MS, Hayes SN, Codsi E, Gulati R, Rose CH, Best PJM. Spontaneous coronary artery dissection associated with pregnancy. J Am Coll Cardiol. 2017;70(4):426–35. https:// doi.org/10.1016/j.jacc.2017.05.055
- Parikh P, Blauwet L. Peripartum cardiomyopathy and preeclampsia: overlapping diseases of pregnancy. Curr Hypertens Rep. 2018;20(8):69. https://doi.org/10.1007/ s11906-018-0868-9
- Prasad M, Tweet MS, Hayes SN, Leng S, Liang JJ, Eleid MF, et al. Prevalence of extracoronary vascular abnormalities and fibromuscular dysplasia in patients with spontaneous coronary artery dissection. Am J Cardiol. 2015;115(12):1672–7. https://doi.org/10.1016/j. amjcard.2015.03.011

- Patel H, Berg M, Barasa A, Begley C, Schaufelberger M. Symptoms in women with peripartum cardiomyopathy: a mixed method study. Midwifery. 2016;32:14–20. https:// doi.org/10.1016/j.midw.2015.10.001
- Ricci F, De Innocentiis C, Verrengia E, Ceriello L, Mantini C, Pietrangelo C, et al. The role of multimodality cardiovascular imaging in peripartum cardiomyopathy. Front Cardiovasc Med. 2020;7:4. https://doi.org/10.3389/ fcvm.2020.00004
- Saw J, Humphries K, Aymong E, Sedlak T, Prakash R, Starovoytov A, et al. Spontaneous coronary artery dissection: clinical outcomes and risk of recurrence. J Am Coll Cardiol. 2017;70(9):1148–58. https://doi.org/10.1016/j. jacc.2017.06.053
- Moulig V, Pfeffer TJ, Ricke-Hoch M, Schlothauer S, Koenig T, Schwab J, et al. Long-term follow-up in peripartum cardiomyopathy patients with contemporary treatment: low mortality, high cardiac recovery, but significant cardiovascular co-morbidities. Eur J Heart Fail. 2019;21(12):1534– 42. https://doi.org/10.1002/ejhf.1624
- Aprigliano G, Palloshi A, Morici N, Ferraresi R, Bianchi M, Anzuini A. Acute coronary syndrome in pre- and post-partum women - a review. Interv Cardiol. 2013;8(1):8–13. https://doi.org/10.15420/icr.2013.8.1.8
- 11. Arany Z. Understanding peripartum cardiomyopathy. Annu Rev Med. 2018;69:165–76. https://doi. org/10.1146/annurev-med-041316-090545
- Karaye KM, Lindmark K, Henein MY. Electrocardiographic predictors of peripartum cardiomyopathy. Cardiovasc J Afr. 2016;27(2):66–70. https://doi.org/10.5830/ CVJA-2015-092
- Lee R, Carr D. Pregnancy-associated spontaneous coronary artery dissection (PASCAD): an etiology for chest pain in the young peripartum patient. CJEM. 2018;20(S2):S64– 9. https://doi.org/10.1017/cem.2018.9

Summary of the case

1	Patient (gender, age)	Female, 26 years	
2	Final diagnosis	Peripartum myocardiopathy	
3	Symptoms	Acute dyspnea and chest pain, which had angina pectoris features and was permanent	
4	Medications	Nitrates, diuresis, ACE inhibitors	
5	Clinical procedure	ECHO, coronary computed tomography angiography	
6	Specialty	Cardiology	