

Echocardiography showed a significant reduction in left ventricular systolic performance, with an estimated LVEF of approximately 25%-30%, and significant hypokinesia 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].

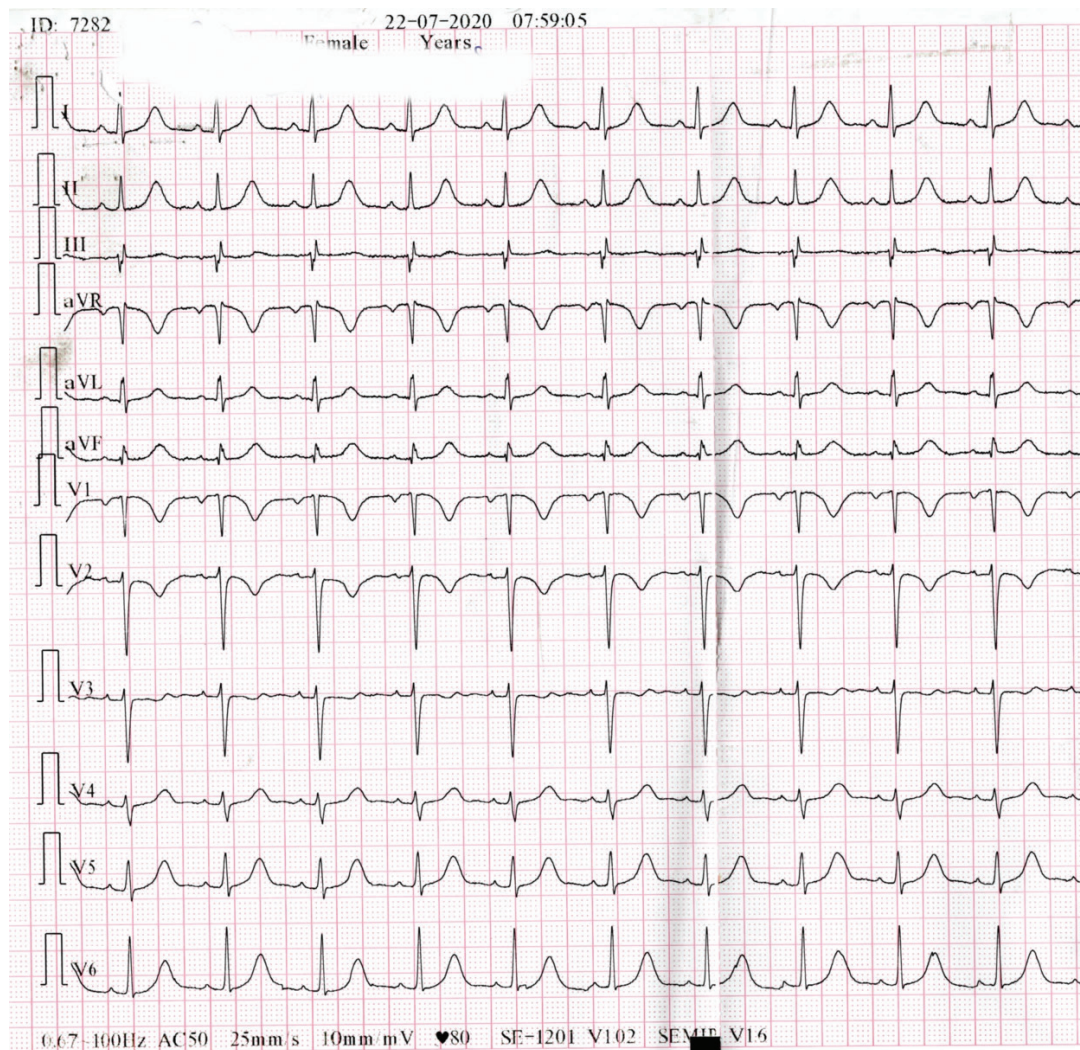


Figure 1. Electrocardiogram showed sinus tachycardia at 125 beats per minute, with no specific ST-segment deviation at 12 precordial, but also at right and posterior leads. It was only observed a remarkable QTc prolongation, at 707 msec.

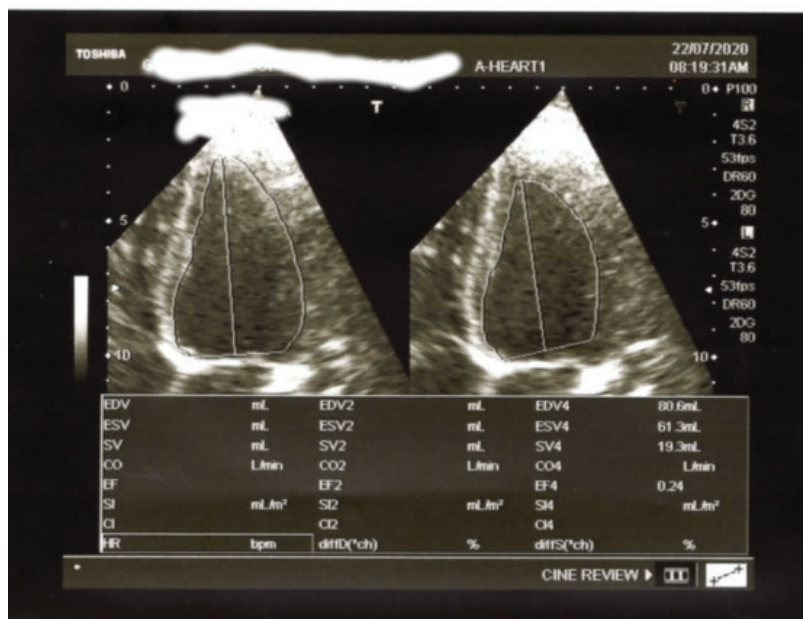


Figure 2. Significant reduction in left ventricular systolic performance, with an estimated LVEF of approximately 25%-30%, and significant hypokinesis in the mid-distal part of the interventricular septum and the apex of the left ventricle.

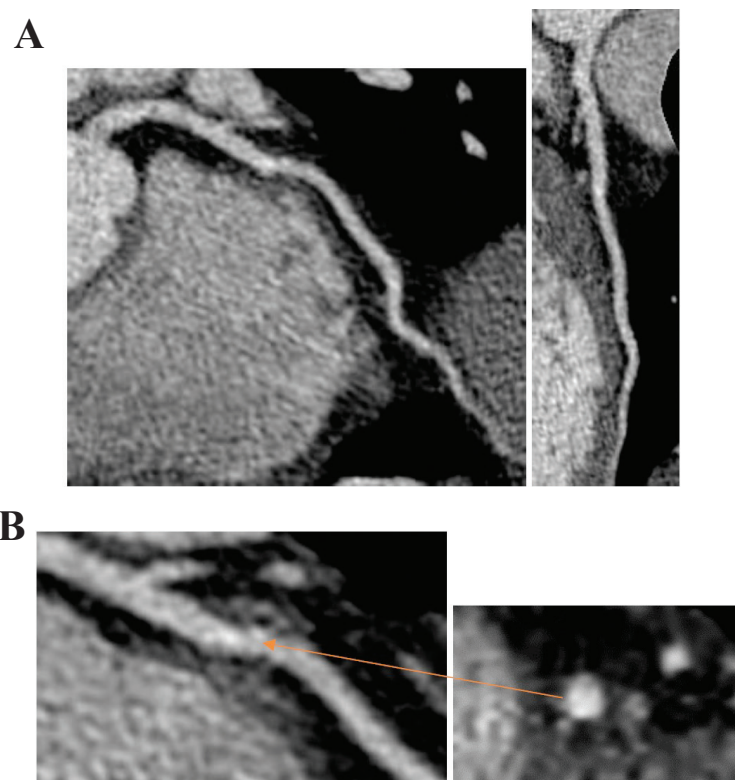


Figure 3. (A) The lower third of the LAD artery is inspected without significant lesions. (B) In the middle of LAD, after the protrusion of the second diagonal branch, an eccentric 7 mm non-calcified lesion was observed, which caused mild stenosis (1%-24%).

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].

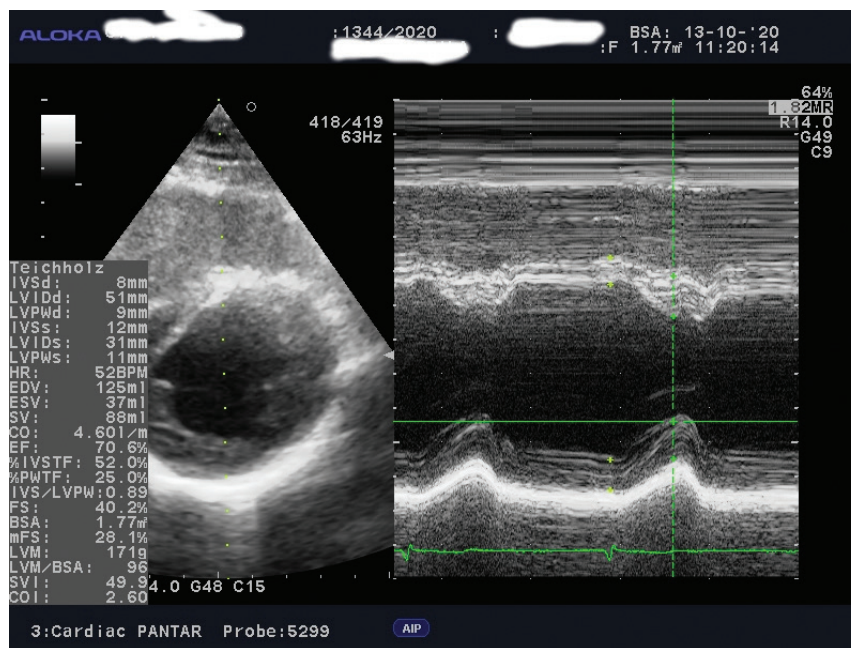


Figure 4. After 3 months the ejection fraction was improved to 70%.

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 precordial 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 infarction diagnosis.
NT-pro BNP	N-terminal pro Brain Natriuretic Peptide
PPCM	Peripartum Cardiomyopathy
QTc	segment on the ECG, corrected according to the Bazett'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.

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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

1. 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>
2. 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.
3. Tweet MS, Hayes SN, Codsí 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>
4. 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>
5. 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>
6. 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>
7. 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>
8. 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>
9. 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>
10. Aprigliano G, Pallosi 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>
12. 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>
13. 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