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# Stress cardiomyopathy in young woman after Cesarean section with SARS-CoV 2 infection

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**Abstract.** In this report, we present a case of a 42-year-old patient who underwent a Caesarean section at 33 weeks of pregnancy. The patient complained about persistent chest pain radiating to upper extremities. The objective findings were unremarkable, the ECG showed a sinus rhythm with an appropriate heart rate, negative T waves in V1-V3. Cardiac markers were elevated with negative dynamic change. As part of the differential diagnosis, a CT pulmonary angiography and a CT aortography were performed and excluded embolization to the pulmonary artery and acute aortic syndrome. However, there was an incidental finding of interstitial pulmonary oedema. Subsequent echocardiographic examination showed left ventricular regional wall movement abnormalities of interventricular septum, borderline reduction of left ventricular ejection fraction, and a smaller pericardial effusion, without tamponade. Consequently, the patient was referred to the cardiac centre for selective coronarography, which showed normal coronary arteries. Left ventriculography revealed regional kinetic disorders consistent with midventricular stress cardiomyopathy. A COVID-19 antigen test was performed, which yielded a positive result. The patient was without chest pain, showed no signs of congestion and did not require any heart failure treatment as her clinical status had spontaneously improved. Fig. 5, Ref. 3, on-line full text (Free, PDF) www.cardiologyletters.sk

**Key words**: acute cardiac injury – cardiomyopathy – myocarditis – cardiac specific markers

Stress cardiomyopathy (SCM) is a transient form of non-ischemic myocardial dysfunction that presents with symptoms similar to an acute myocardial infarction (AMI), but without evidence of obstructive coronary artery disease. SCM is frequently associated with a trigger event such as physical or emotional stress, which is believed to lead to excessive catecholamine release and subsequent myocardial stunning. The association between COVID-19 infection and SCM has been previously reported in literature (1). Here, we present a case of a 42-year-old woman with chest pain following Caesarean section, with an incidental finding of interstitial pulmonary oedema and abnormalities of the left ventricle, consistent with an atypical form of stress cardiomyopathy.

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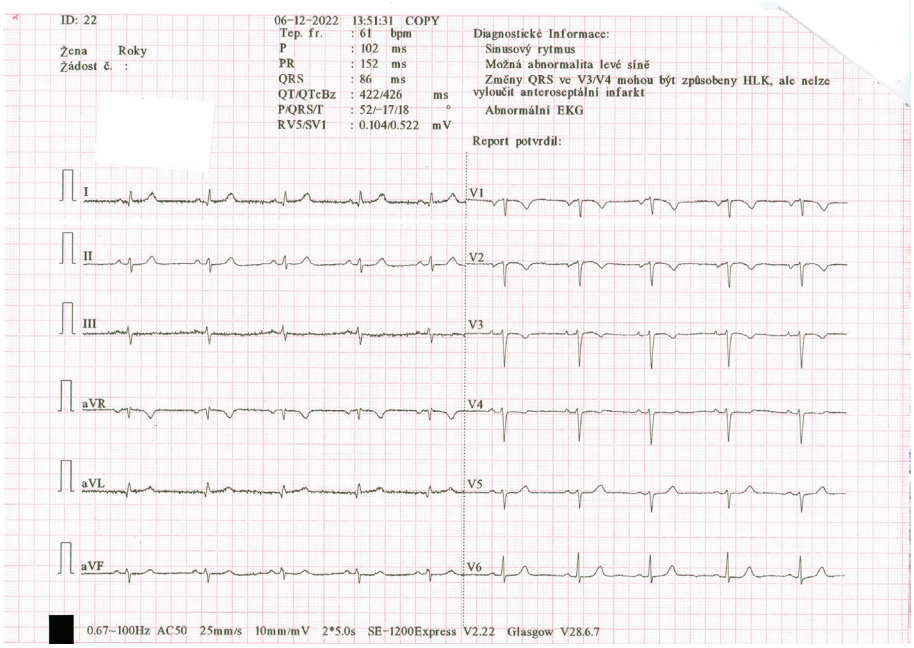


**Figure 1 Initial patient's ECG showing negative T waves in leads V1-V3, biphasic T wave in V4**

## Case presentation

A 42-year-old female without previous medical history was admitted to the hospital at 33 weeks of gestation due to preterm labour. Several hours after the delivery by Caesarean section (indicated due to failure to progress), the patient was examined by an internal medicine doctor for newly developed persistent chest pain radiating to both upper extremities.

While the objective findings were unremarkable, the electrocardiogram (ECG) showed sinus rhythm with an appropriate heart rate, negative T waves in V1-V3, biphasic T wave in V4 and poor R-wave progression (**Figure 1**). Initially, the patient was hypertensive (160/110 mmHg); the blood pressure was later normalized after the administration of analgesia. Cardiac specific markers were elevated – with hs-cTnT initially 688.1 ng/l, and 622.2 ng/l after 3-hour follow-up NTproBNP was 2880 ng/l. The patient was at first evaluated for a possible case of an acute coronary syndrome, spontaneous coronary artery dissection, acute aortic syndrome, pulmonary embolism, myocarditis, peripartum cardiomyopathy or an atypical form of stress cardiomyopathy. CT pulmonary angiography and CT aortography were performed to exclude pulmonary embolism (PE) and acute aortic syndrome (AAS). The CT examination revealed incidental interstitial pulmonary oedema, small bilateral fluidothorax (up to 10 mm) and a pericardial effusion (up to 15 mm) without PE or AAS (**Figure 2**). Subsequent echocardiography showed regional wall motion abnormalities (WMA) in the interventricular septal area, borderline reduced left

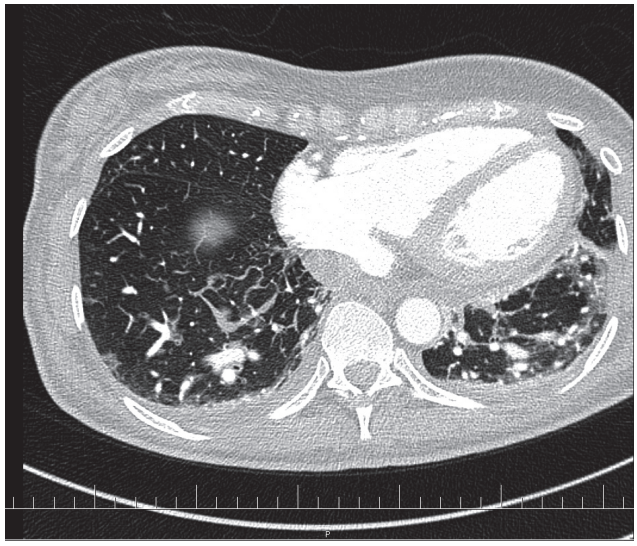

**Figure 2 Patient's initial CT showing interstitial pulmonary oedema**

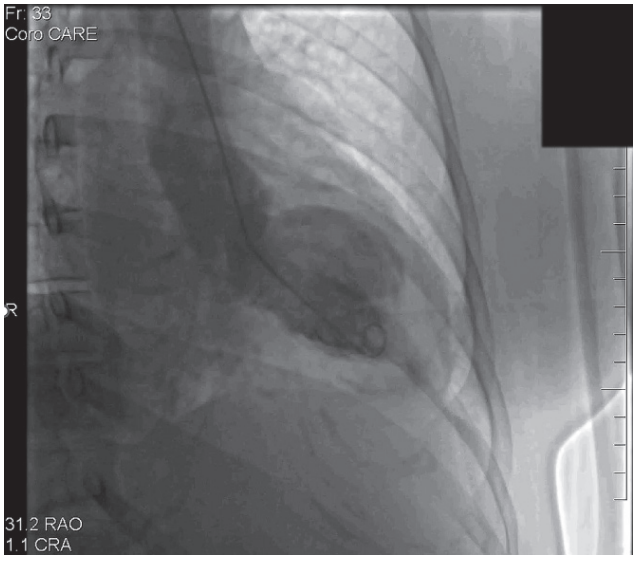


**Figure 3 Patient's ventriculography showing regional wall motion abnormalities in systole corresponding to midventricular SCM**

ventricular ejection fraction and a smaller pericardial effusion without tamponade.

The patient was transferred to the ICU and later referred to the cardiac centre for selective coronary angiography, which showed negative findings in the coronary arteries, thus excluding acute coronary syndrome and spontaneous coronary artery dissection. Left ventriculography showed regional kinetic disorders compatible with SCM of the midventricular type (**Figure 3**) and the performing cardiologist recommended a cardiac MR to confirm the diagnosis. After the patient was transferred back to the gynaecology ward, a COVID-19 antigen screening test was performed with a positive result. The course of SARS-CoV2 infection was asymptomatic with only transient elevation of inflammatory markers (CRP up to 55.21 mg/l), without leucocytosis.

A follow-up echocardiography was performed 10 days later, which revealed normal left ventricular ejection fraction and adequate diastolic function. However, prominent regional WMA were still detectable: particularly pronounced was hypokinesis in midsegment of the interventricular septum (IVS), anterior and anterolateral wall. Slight hypokinesis was also observed in the remaining segments of the IVS, anterior and anterolateral walls, in the mid segment of the posterior wall, and in the mid segment of the posterolateral wall. Additionally, a small separation of the pericardial layers was identified – possibly a discrete pericardial effusion. Cardiac MR was performed with delay due to the isolation forced by COVID-19 and revealed minor late gadolinium enhancement in the pericardium, which may indicate a possible case of resolved stress cardiomyopathy (SCM).

## Management and outcome

The patient was treated symptomatically without specific treatment for SCM and showed spontaneous improvement in clinical status during observation. Follow-up laboratory results showed decreased hs-cTnT of 21.27 ng/l and NTproBNP of 959.6 ng/l (**Figure 4**). A fol-

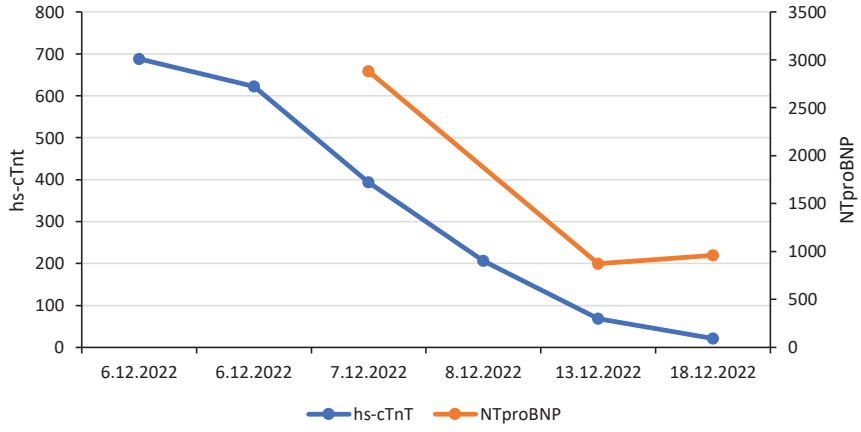


**Figure 4 A graph showing high sensitive Troponin T and NTproBNP changes during hospitalisation**

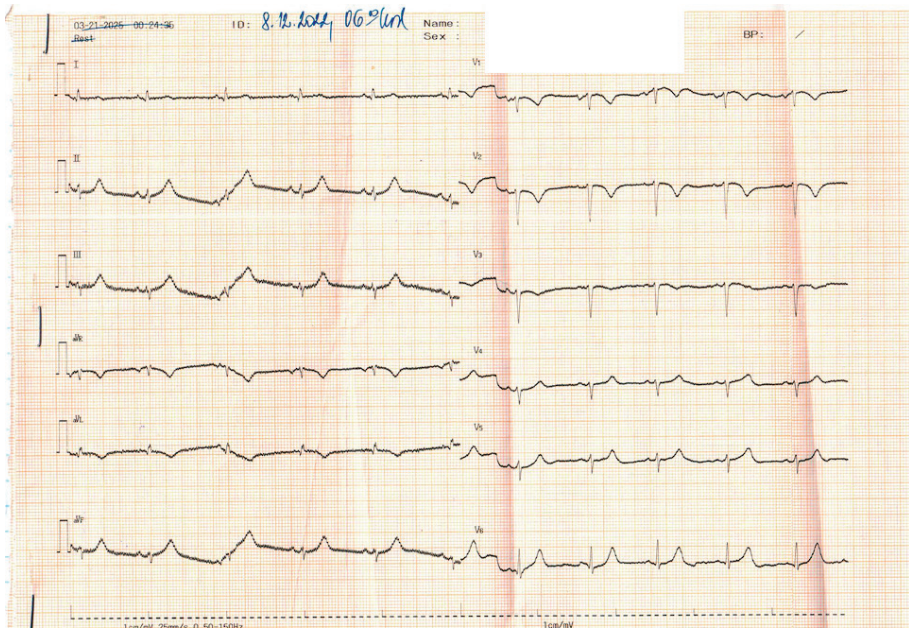


**Figure 5 Later ECG showing partial normalisation of T waves in V3 and V4**

low-up ECGs showed partial normalisation of T waves in V3 and V4 (**Figure 5**), and the patient was discharged asymptomatic without any further complications.

## Discussion

Considering that the results of electrocardiography, repeated echocardiography, selective coronarography, left ventriculography and CMR were all consistent with SCM, the diagnosis of stress cardiomyopathy of midventricular type was established. In differential diagnosis we excluded acute coronary syndrome, spontaneous coronary artery dissection, acute aortic syndrome and pulmonary embolism.

Stress cardiomyopathy is an acute reversible form of heart failure that occurs in response to intense emotional or physical stress. It presents with clinical features that resemble acute coronary syndrome, such as chest pain, dyspnea, and electrocardiographic changes. However, unlike acute coronary syndrome, stress cardiomyopathy does not involve significant coronary artery obstruction or myocardial necrosis and typically resolves within several weeks. The condition is also referred to as takotsubo syndrome (2). It predominantly affects postmenopausal women and can be precipitated by various psychological or physiological stressors, such as bereavement, fear, anger, or infection. The pathophysiology of stress cardiomyopathy is not fully elucidated, but it is known to involve excessive catecholamine release that can lead to coronary microvascular dysfunction or direct myocardial toxicity (3).

This case highlights the importance of considering stress cardiomyopathy as a possible cause of chest pain even in younger patients without a history of coronary heart disease, especially in the setting of ongoing COVID-19. It also underscores the significance of cardiac specific markers, CT imaging, echocardiography, and selective coronary angiography in the diagnosis of SCM. Further research is needed to explore the association between COVID-19 and SCM and to identify the optimal management strategies for patients with this condition.

In this case, COVID-19 was asymptomatic, with only transient elevation of inflammatory markers without leucocytosis. It is possible that the patient's SCM was triggered by the stress of the Caesarean section, with the possible contribution of underlying COVID-19. The management of SCM involves supportive care and treatment of the underlying trigger event.

In conclusion, SCM should be considered as a potential differential diagnosis in patients with COVID-19

infection who present with chest pain and elevated cardiac specific markers with other possible stress factors. Timely diagnosis and management of SCM can improve patient outcomes and prevent unnecessary interventions.

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