When Left ventricle Acutely "crashes" and "recovers" but the troponin stays Silent!

Quand le ventricule gauche « s'effondre » et « récupère » avec des troponines silencieuses !

Naziha Turki [1,2], Emna Derbel [1,2], Hela Fendri[2,3], Elleuch Wafa[1,2], Rania Hammami [1,2]

1. *Military University Hospital of Sfax , Cardiology Department, Sfax, Tunisia*
2. *University of Sfax, Faculty of Medicine of Sfax , Sfax, Tunisia*
3. *Hedi Chaker Hospital, Radiology Department, Sfax, Tunisia*

Summary

Acute reversible left ventricular dysfunction most commonly occurs in the setting of acute coronary syndrome or acute myocarditis and is generally accompanied by elevated troponin levels. We reported the case of a female patient presenting with loss of consciousness, retrosternal chest pain and anterior ST segment depression. High-sensitivity troponin levels remained negative on serial measurements. Echocardiography revealed Segmental wall motion abnormalities with left ventricular dysfunction (LVEF =45%). Coronary angiography showed no significant obstructive coronary disease. Cardiac magnetic resonance imaging demonstrated myocardial edema without late gadolinium enhancement. A total recovery of the LV function was obtained under heart failure medical treatment, the diagnosis of Takutsubo cardiomyopathy (TCM) was retained. This case highlights that negative troponin levels do not exclude TCM and underscores the crucial role of cardiac magnetic resonance imaging in the diagnostic workup.

Keywords

Takotsubo cardiomyopathy; Coronary vasospasm; Troponin-negative; Acute left ventricular dysfunction; Cardiac MRI

Résumé

La dysfonction aiguë et réversible du ventricule gauche survient le plus souvent dans le contexte d'un syndrome coronarien aigu ou d'une myocardite aiguë et s'accompagne généralement d'une élévation de la troponine. Nous rapportons le cas d'une patiente se présentant avec une perte de connaissance transitoire, une douleur thoracique rétrosternale et un sous-décalage du segment ST en antérieur. Les taux de troponine ultrasensible sont restés négatifs. L'échocardiographie a montré des troubles segmentaires de la cinétique avec une dysfonction du ventricule gauche (FEVG = 45 %). La coronarographie n'a objectivé aucune sténose coronaire significatives. L'IRM cardiaque a mis en évidence un œdème myocardique sans rehaussement tardif au gadolinium. Une récupération complète de la fonction ventriculaire gauche a été obtenue sous traitement médical de l'insuffisance cardiaque, permettant de retenir le diagnostic de cardiomyopathie de Takotsubo (TCM). Ce cas souligne que des taux de troponine négatifs n'excluent pas le diagnostic de TCM et met en évidence le rôle crucial de l'IRM cardiaque dans la démarche diagnostique.

Mots-clés

Cardiomyopathie de Takotsubo ; Spasme coronaire ; Dysfonction ventriculaire gauche aiguë ; Troponine normale ; IRM cardiaque

Correspondance

Naziha Turki

Military university hospital of Sfax, Cardiology department, Sfax, Tunisia

Email: nazihaturki@yahoo.fr,

INTRODUCTION

Acute transient LV dysfunction is commonly attributed to acute coronary syndromes or myocarditis. Takotsubo cardiomyopathy (TCM) accounts for up to 2–5%(1)of INOCA presentations and remains diagnostically challenging, TCM affects predominantly postmenopausal women. It's characterized by ventricular dysfunction in the absence of obstructive coronary artery disease. It may be triggered by an acute medical illness or intense physical or emotional stress (2). TCM usually associated with moderate elevation of cardiac biomarkers, particularly cardiac troponins. However, rare atypical cases with normal troponin levels have been reported, making diagnosis challenging. Cardiac magnetic resonance imaging (CMR) plays a key role in confirming the diagnosis by demonstrating myocardial edema without late gadolinium enhancement(3). We reported a rare case of troponin-negative Takotsubo syndrome confirmed by CMR, highlighting the importance of multimodality imaging in atypical presentations.

CASE PRESENTATION

A 59-year-old woman with history of migraine, dyslipidemia on dietary management, no history of smoking, and no other cardiovascular risk factors, presented with nocturnal retrosternal chest pain and a 30-minute transient loss of consciousness without tonic-clonic movements or urinary incontinence. In the emergency department, she was hemodynamically stable, conscious, and asymptomatic. The first electrocardiography showed ST segment depression with T negative waves in the anteroseptal territory and QTc at the upper limit of normal (450 ms) (figure 1), so the patient was admitted to the cardiology unit.

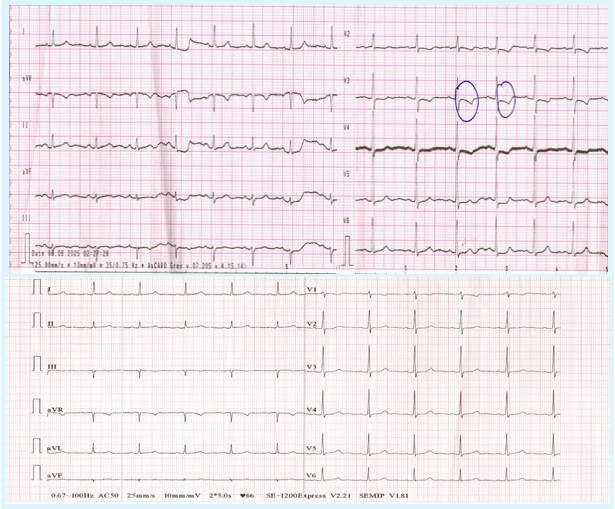

Figure 1. Evolution of electrocardiographic changes during hospitalization

On admission, the patient was free of chest pain, a second electrocardiography was realized showing regression of the ST segment depression but QTc remained borderline (449 ms).

Serial high-sensitivity troponin measurements at 3-hour intervals from 1 hour after symptom onset remained negative. NT-pro BNP was also normal. The rest of the laboratory workup was unremarkable, including potassium, calcium, and magnesium levels

Transthoracic echocardiography revealed a severe anteroseptal and anterior wall hypokinesia associated with moderate anterolateral hypokinesia. The ejection fraction was 45%, LV global longitudinal strain was impaired (–10%), predominantly in the apical segments (figure 2), with an S' velocity of 7 cm/s. No other abnormalities were detected in the echocardiography.

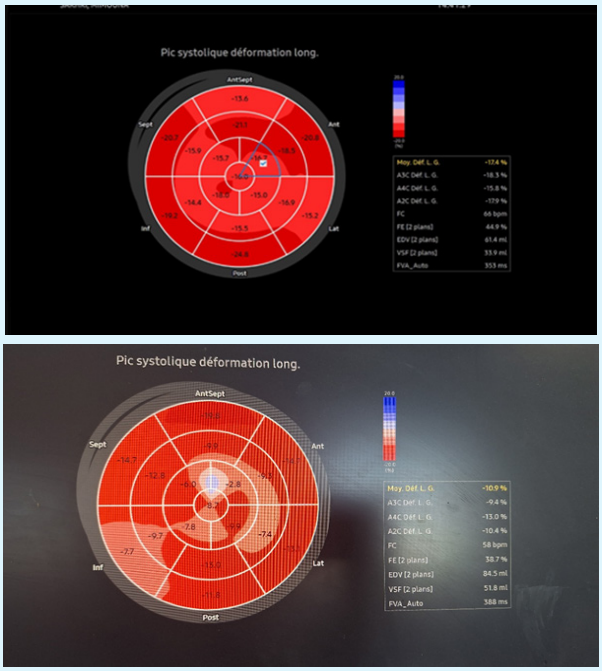

Figure 2. Recovery of left ventricular systolic function assessed by strain imaging

Given the chest pain and echocardiographic abnormalities, coronary angiography was performed within 24 hours. It revealed a 50% stenosis of a diagonal branch and the rest of the coronary tree was infiltrated without significant stenosis.

Given the clinical presentation consistent with INOCA, further evaluation with cardiac magnetic resonance (CMR) imaging was performed to differentiate between coronary vasospasm and Takotsubo syndrome. CMR was obtained

on the second day of hospitalization, after initiation of medical therapy including an angiotensin-converting enzyme inhibitor and a beta-blocker. CMR demonstrated left ventricular dysfunction with an ejection fraction of 48 %, hypokinesia of the anteroseptal and the apical left ventricular walls. There was myocardial edema involving the segment 14 on T2-weighted imaging, without evidence of late gadolinium enhancement, excluding myocardial infarction and myocarditis (figure 3).

Further questioning revealed a history of emotional stress related to the death of a cousin.

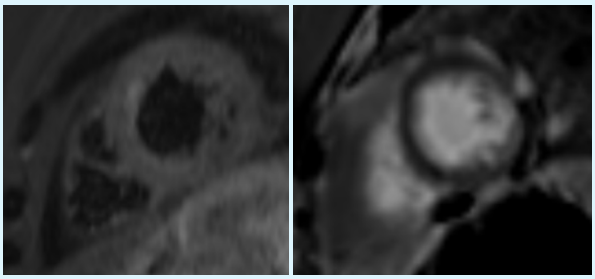

Figure 3. Cardiac MR imaging showing myocardial oedema

A-Subendocardial hypersignal of the anteroseptal-apical wall of the LV on STIR sequence

B-Absence of myocardial enhancement

As part of the diagnostic workup, the InterTAK Diagnostic Score (tableau 1) was calculated and showed a score of 64 indicating an intermediate probability of TCM. The TS scoring system (4,5) which is composed of 10 points was 8 further supporting the diagnosis of TCM over vasospasm.

Based on the clinical presentation, imaging findings, and triggering emotional stress, the diagnosis of Takotsubo syndrome was established. Treatment with a beta-blocker and an ACE inhibitor was initiated.

At four-month follow-up, left ventricular function had fully recovered, with homogeneous wall motion and normalized strain (figure 2).

Table 1. Comparison between takotsubo syndrome and coronary vasospasm

Feature	Takotsubo Cardiomyopathy (TCM)	Coronary Vasospasm	Patient Findings
Sex	Predominantly female	Both sexes; less female-predominant	Female
Smoking	Not defining	Risk factor	Non-smoker
Trigger	**Emotional or physical stress**	Often spontaneous, nocturnal	Nocturnal chest pain; emotional trigger (bereavement)
Coronary arteries	Usually normal	Usually normal	50% diagonal branch, others normal
Troponin	**Mildly elevated or normal**	Normal if brief ischemia; elevated if prolonged	Negative on 3 serial ultrasensitive measurements
ECG	ST-segment elevation/ depression, T-wave inversion, QTc prolongation	Transient ST changes; usually normal QTc	ST depression, prolonged QTc
Echocardiography	**Segmental wall-motion abnormalities, LVEF ≤40–50%, reduced GLS**	May show segmental abnormalities	Segmental wall-motion abnormalities; LVEF 45%; GLS -13%
Cardiac MRI	Myocardial edema, no LGE	May show edema if prolonged ischemia	Myocardial edema without LGE
Recovery	**Usually complete**	Usually complete if no infarction	Full recovery at 4 months
Mechanism	Catecholamine-mediated myocardial stunning	Transient coronary constriction	Stress-related TCM pattern rather than isolated vasospasm

DISCUSSION

The diagnosis of acute transient left ventricular dysfunction remains challenging, as these syndromes frequently mimic acute coronary syndromes (ACS) or myocarditis, particularly in atypical presentations. Among patients presenting with chest pain and non-obstructive coronary arteries (INOCA), Takotsubo cardiomyopathy (TCM) accounts for approximately 1–3% of suspected ACS cases, and up to 5–6% in women presenting with ST-segment elevation(1), underscoring its clinical relevance in this setting.

The pathophysiology of TCM is multifactorial. Emotional or physical stress triggers an acute catecholamine surge, leading to a combination of multivessel epicardial coronary spasm, direct catecholamine-mediated myocardial toxicity, microvascular dysfunction, and metabolic switching from fatty acid to glucose oxidation(6).

In the classical apical variant, these mechanisms result in akinesia of the mid and apical left ventricular segments associated with basal hyperkinesis, producing the characteristic "apical ballooning" appearance. Regional differences in adrenergic receptor density and sympathetic innervation have been proposed to explain this typical distribution of wall-motion abnormalities(1).

In the present case, the identification of a clear emotional trigger—bereavement following the death of a close relative—further supports the diagnosis. Emotional stress is a well-established precipitating factor for TCM and reinforces the central role of neurohumoral mechanisms in transient myocardial stunning and reversible ventricular dysfunction(4).

From a clinical perspective, differentiating Takotsubo cardiomyopathy from coronary vasospasm remains a major diagnostic challenge, as both conditions may present with acute chest pain, ECG changes, and transient left ventricular dysfunction (tableau 3). However, several features favored TCM in this patient. In TCM, regional wall-motion abnormalities typically extend beyond a single coronary territory, most commonly involving the mid and apical segments, whereas coronary vasospasm usually produces dysfunction confined to the territory of the spastic artery(7). A vasospasm severe enough to cause extensive apical and anterior akinesia would generally involve the mid left anterior descending artery and would be expected to result in troponin elevation. Although provocative testing remains the gold standard for diagnosing coronary spasm, it was not performed in this patient.

In our patient, the presence of an emotional trigger, female sex, absence of conventional cardiovascular risk factors, QTc prolongation, oriented the diagnosis toward Takotsubo cardiomyopathy.

Diagnostic scores also are useful tools for guiding diagnostic orientation. The InterTAK Diagnostic Score (tableau 1) was 64, corresponding to an intermediate probability of TCM. In addition, the Takotsubo Syndrome (TS) scoring system, a simplified 10-point score recently validated in 2025, was applied(4,5). A TS score ≥ 8 has demonstrated excellent discriminative ability for differentiating TCM from ST-segment elevation myocardial infarction. In our patient, the TS score was 8, further reinforcing the diagnosis.

The absence of troponin elevation represents another important diagnostic consideration. In most reported cases, TCM is associated with mild to moderate troponin elevation reflecting limited myocardial injury(8). However, troponin-negative presentations, although rare, have been described. This phenomenon is thought to reflect predominant myocardial edema without irreversible necrosis, particularly in early or less extensive forms of the disease(9).

This atypical presentation highlights that normal troponin levels do not exclude Takotsubo syndrome and emphasizes that reliance on biomarkers alone may be misleading and that multimodality imaging plays a crucial role in such scenarios (8).

In this context, cardiac magnetic resonance (CMR) plays a pivotal role in the diagnostic work-up, particularly in atypical or troponin-negative presentations. CMR allows comprehensive tissue characterization, with typical findings in TCM including myocardial edema on T2-weighted imaging or T2 mapping, in the absence of late gadolinium enhancement (LGE)(3). The lack of LGE is crucial to exclude myocardial infarction and myocarditis, both of which are characterized by irreversible myocardial injury. In our patient, CMR demonstrated myocardial edema without LGE, thereby confirming the diagnosis of TCM and excluding alternative causes of acute myocardial dysfunction.

The prognosis of TCM is generally favorable, with recovery of left ventricular function occurring within weeks to months(8,10). In this patient, four-month follow-up echocardiography showed complete normalization of left ventricular systolic function, with homogeneous wall motion and normalization of global longitudinal strain, highlighting the usefulness of strain analysis in documenting full myocardial recovery beyond left ventricular ejection fraction alone

CONCLUSION

This case illustrates a rare presentation of Takotsubo syndrome with normal cardiac troponin levels, confirmed by cardiac magnetic resonance imaging. Clinicians should be aware that the absence of biomarker elevation does not exclude Takotsubo syndrome. CMR plays a pivotal role in establishing the diagnosis, particularly in atypical cases, and should be considered when coronary findings and biomarkers fail to explain the clinical presentation.

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