

Reverse Takotsubo Cardiomyopathy in Peripartum Period: A Two-Case Series

Dr. Kavya. P¹ and Dr. Archana Kumari²

¹Postgraduate, Saveetha medical college and Hospital

²Assistant Professor, Saveetha medical college and Hospital

*Corresponding Author
Dr. Kavya. P

Article History

Received: 08.08.2025

Revised: 15.09.2025

Accepted: 24.10.2025

Published: 05.11.2025

Abstract: *Background:* Stress-induced (Takotsubo) and peripartum cardiomyopathies are uncommon but potentially fatal causes of acute heart failure in young women. Early recognition remains challenging, especially when presentations overlap with obstetric or surgical emergencies. *Cases:* We describe (i) a 28-year-old woman who developed reverse Takotsubo cardiomyopathy with an ejection fraction (EF) of 33% immediately after emergency ileal resection performed 15 days post-caesarean delivery, and (ii) a 28-year-old primigravida who, following a hypertensive crisis and eclamptic seizures at 33 weeks, manifested severe dilated cardiomyopathy (EF 20%) complicated by posterior reversible encephalopathy syndrome (PRES). Both patients showed near-complete functional recovery within one week (EF > 60%). *Conclusion:* The cases illustrate distinct stress-triggered myocardial phenotypes on the obstetric-surgical continuum and emphasise the value of early echocardiography, guideline-directed heart-failure therapy, and multidisciplinary care.

Keywords: Takotsubo cardiomyopathy, Peripartum cardiomyopathy, Stress-induced cardiomyopathy, Echocardiography, Heart failure.

INTRODUCTION

Pregnancy-associated heart failure encompasses two major yet mechanistically distinct syndromes—peripartum cardiomyopathy (PPCM) and stress-induced Takotsubo syndrome (TTS)—each of which can evolve rapidly from maternal or obstetric stressors and culminate in life-threatening circulatory collapse.

PPCM is defined as a new-onset reduction in left-ventricular ejection fraction (LVEF <45%) occurring between the last month of gestation and the first six months postpartum in the absence of another identifiable cause, affecting approximately 1 in 1000 – 4000 deliveries worldwide (1).

TTS, first described in Japan, manifests as acute, reversible systolic dysfunction triggered by a catecholaminergic surge after intense stress, mimicking acute coronary syndrome in presentation and biomarker profile (2). Although the prototypical “apical ballooning” pattern predominates, younger females are disproportionately represented among reverse or basal variants, in which basal and mid-ventricular segments are akinetic while the apex remains hyperdynamic (3,4).

Pregnancy creates a milieu of haemodynamic expansion, hormonal flux, oxidative stress, and inflammatory activation, which together with surgical stress or hypertension can unmask latent myocardial vulnerability. In PPCM, oxidative cleavage of prolactin into a 16-kDa anti-angiogenic fragment, compounded by genetic variants in sarcomeric or cytoskeletal proteins, leads to endothelial dysfunction and myocyte apoptosis (1,7). In TTS, intense sympathetic discharge provokes myocardial stunning through calcium overload,

microvascular spasm, and catecholamine toxicity, with perioperative stress being a recognized trigger (2,6).

Distinguishing PPCM from TTS is challenging because both present with dyspnoea, pulmonary oedema, and elevated natriuretic peptides. However, certain features help: regional wall-motion abnormalities beyond a single coronary territory (especially basal-only akinesia), modest troponin rise, and temporal association with stressors favour TTS; while diffuse hypokinesia in late pregnancy/postpartum suggests PPCM (2,4,5).

Correct diagnosis has therapeutic implications: prolactin blockade with bromocriptine and prolonged anticoagulation are advocated for PPCM (8–10), whereas β -adrenergic modulation and cautious fluids are crucial in TTS (5). European Society of Cardiology (ESC) guidelines recommend early echocardiography, serial biomarkers, and multidisciplinary care (5,9).

We present two cases that highlight divergent but overlapping phenotypes of reverse TTS and PPCM, and discuss their management in the light of current evidence.

CASE PRESENTATION

Case 1: Reverse Takotsubo Cardiomyopathy in the Postpartum Perioperative Setting

A 28-year-old woman, gravida 2 para 2, delivered by emergency caesarean section 15 days earlier, presented with abdominal pain, bilious vomiting, and distension. Examination and imaging confirmed adhesive intestinal obstruction.

She underwent emergency laparotomy with adhesiolysis and ileal resection. During extubation, she developed bradycardia (HR 38/min), hypotension (60/40 mmHg), and transient ECG changes with ST depression. Troponin I peaked at 224 ng/L. Echocardiography showed basal and mid-ventricular hypokinesia with preserved apical function (EF 33%). Coronary CT angiography excluded obstructive disease.

She was managed with cautious fluids, norepinephrine (weaned within 6 h), and later metoprolol and ivabradine. Serial echocardiography showed EF improvement to 52% by day 2 and 63% by day 6. She was discharged on oral beta-blockers and remained asymptomatic at follow-up.

Case 2: Peripartum Cardiomyopathy with PRES after Eclampsia

A 28-year-old primigravida at 33 weeks presented with eclamptic seizures and severe hypertension (BP 200/120 mmHg). Emergency caesarean section was performed, delivering a 1.9 kg infant. Postoperatively, she developed pulmonary oedema and pink frothy sputum.

Echocardiography revealed global LV hypokinesia (EF 20%). BNP was markedly elevated (7200 pg/mL), troponin normal. She was managed with diuretics, nitroglycerin, non-invasive ventilation, and later carvedilol and vasodilators.

Neurologically, she developed transient cortical blindness. MRI showed parieto-occipital vasogenic oedema consistent with PRES. She improved with BP control, magnesium, and levetiracetam. EF normalized to 62% by day 7. At 3-month review, she remained asymptomatic with complete radiological recovery.

DISCUSSION

Both cases highlight stress-induced cardiomyopathies but with distinct triggers.

Reverse TTS is more common in young women perioperatively, related to catecholamine surges and basal adrenergic receptor sensitivity (2,6). Mechanisms include catecholamine cardiotoxicity, calcium overload, and microvascular spasm.

PPCM is linked to oxidative stress-mediated cleavage of prolactin into a 16-kDa fragment, endothelial dysfunction, and genetic predispositions (1,7). Bromocriptine, by inhibiting prolactin, has shown EF improvement in randomized trials and meta-analyses (8–10).

Key discriminators:

- Wall motion: regional basal akinesia (TTS) vs global hypokinesia (PPCM).
- Troponin: higher in TTS.

- Timing: perioperative/early postpartum (TTS) vs late gestation/postpartum (PPCM).
- Coronary angiography: normal in both but essential to rule out dissection.

Treatment overlaps: both require heart-failure therapy (beta-blockers, diuretics, vasodilators, ACE inhibitors after delivery). Ivabradine is an option when rate control is needed. Anticoagulation is advised if EF <35% or thrombus is present (5). Bromocriptine is recommended for PPCM under ESC guidelines (5,9,10).

PRES complicates severe preeclampsia/eclampsia in 0.01–0.05% of pregnancies, related to endothelial dysfunction and hypertension (11). Cardiogenic factors such as low EF may exacerbate cerebral oedema. Prognosis is favourable with seizure prophylaxis and BP control (12).

Recovery is variable: 70–90% of TTS patients normalize EF in 4–8 weeks (2,3). PPCM recovery is slower; only 50–75% normalize EF within 6–12 months, with relapse risk in subsequent pregnancies if EF <55% (4). Both our patients showed rapid recovery, emphasizing the benefit of early diagnosis and therapy.

CONCLUSION

These cases underscore the dynamic cardiocerebral complications that may follow obstetric or abdominal emergencies in young women. Bedside echocardiography, vigilant haemodynamic monitoring, and adherence to ESC guidelines enabled swift recovery. Clinicians should maintain high suspicion for reversible cardiomyopathies and PRES in peripartum cardiovascular collapse.

REFERENCES

1. Sliwa K, Bauersachs J, Hilfiker-Kleiner D, et al. Current state of knowledge on aetiology, diagnosis, management, and therapy of peripartum cardiomyopathy: a position statement of the ESC Heart Failure Association. *Eur J Heart Fail.* 2018;20(7):951-967.
2. Lyon AR, Bossone E, Schneider B, et al. Pathophysiology of Takotsubo Syndrome. *J Am Coll Cardiol.* 2020;76(7):902-921.
3. Barrionuevo-Sánchez MI, et al. Failing in reverse: a case report of reverse Takotsubo syndrome. *Eur Heart J Case Rep.* 2023;7(11):ytad493.
4. Biteker M, İlhan E. Long-term outcomes in peripartum cardiomyopathy. *Open Cardiovasc Med J.* 2019;13:13-22.
5. McDonagh TA, Metra M, Adamo M, et al. 2023 ESC Guidelines for the management of cardiomyopathies. *Eur Heart J.* 2023;44(1):e1-e219.
6. Pérez-Campos Mayoral L, et al. Surgery as precipitating factor of Takotsubo cardiomyopathy: case report and literature review. *Int J Surg Case Rep.* 2023;107:108094.

7. Ware JS, Li J, Witte KK, et al. Genetic basis of peripartum cardiomyopathy. *Nat Genet.* 2016;48(12):138-141.
8. Hilfiker-Kleiner D, et al. Bromocriptine for treatment of peripartum cardiomyopathy: multicentre randomized trial. *Eur Heart J.* 2017;38(35):2671-2679.
9. Regitz-Zagrosek V, et al. ESC Guidelines on the management of cardiovascular diseases during pregnancy. *Eur Heart J.* 2018;39(34):3165-3241.
10. Hernandez-Galindo V, Chaparro S. Effects of bromocriptine in peripartum cardiomyopathy: a systematic review and meta-analysis. *Heart Fail Rev.* 2022;27(4):1141-1153.
11. Fugate JE, Rabinstein AA. Posterior reversible encephalopathy syndrome: clinical and radiological manifestations, pathophysiology, and outstanding questions. *Lancet Neurol.* 2015;14(9):914-925.
12. Teixeira B, et al. Posterior Reversible Encephalopathy Syndrome in a Pediatric Intensive Care Unit: a case series. *Cureus.* 2023;15(6):e50658.