

Short Report

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Not your usual post-partum: A case of reversible cerebral Vasoconstriction syndrome and Takotsubo cardiomyopathy

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Abstract

A young lady with a recent caesarean section presented with thunderclap headache and was diagnosed to have Reversible Cerebral Vasoconstriction Syndrome (RCVS). Soon after, she had tachycardia with troponin elevation. Cardiac imaging confirmed a diagnosis of Takotsubo Cardiomyopathy (TCM). RCVS and TCM have been reported separately in post-partum women but rarely occur together. Their hypothesized pathophysiology's have significant overlay, for instance sympathetic hyperstimulation from catecholamine release. Both conditions usually have good clinical outcomes, but some have in-hospital complications or residual neurological or cardiovascular deficits. Both conditions usually have good clinical outcomes, but some have in-hospital complications or residual neurological or cardiovascular deficits.

Keywords: Reversible Cerebral Vasoconstriction Syndrome; Takotsubo cardiomyopathy; Post-partum; Catecholamine excess.

Abbreviations: RCVS: Reversible Cerebral Vasoconstriction Syndrome; TCM: Takotsubo Cardiomyopathy; EF: Ejection Fraction; TTE: Transthoracic Echocardiogram; CoW: Circle of Willis; CT: Computed Tomography.

Introduction

Reversible Cerebral Vasoconstriction Syndrome (RCVS) and Takotsubo Cardiomyopathy (TCM) have been reported separately in post-partum women [1-3] but only one case report of RCVS and TCM was found simultaneously post-partum. The hypothesized pathophysiology's of RCVS and TCM have significant overlay [4]. Estrogen levels decrease post-partum with the expulsion of placenta, which is further suppressed by breastfeeding. This leads to increased sympathetic nervous activity and decreased vasodilation and microvascular blood flow through both endothelium-dependent and-independent mechanisms [2-5]. Mental and physical stress experienced by the mother can

also lead to sympathetic hyperstimulation from catecholamine release, endothelial dysfunction, and oxidative stress [6]. This causes transient dysfunction and vasoconstriction in both cerebrovascular and coronary artery vascular tone leading to the clinical manifestations of RCVS and TCM [6,7]. In patients who have persistent symptoms despite analgesia and primary headache prophylaxis or red flag symptoms, evaluation of non-primary headaches should be considered. Both conditions usually have good clinical outcomes, but a percentage have in-hospital complications or residual neurological or cardiovascular deficits. As such, clinicians should counsel patients appropriately and monitor longitudinally for resolution [6,7].

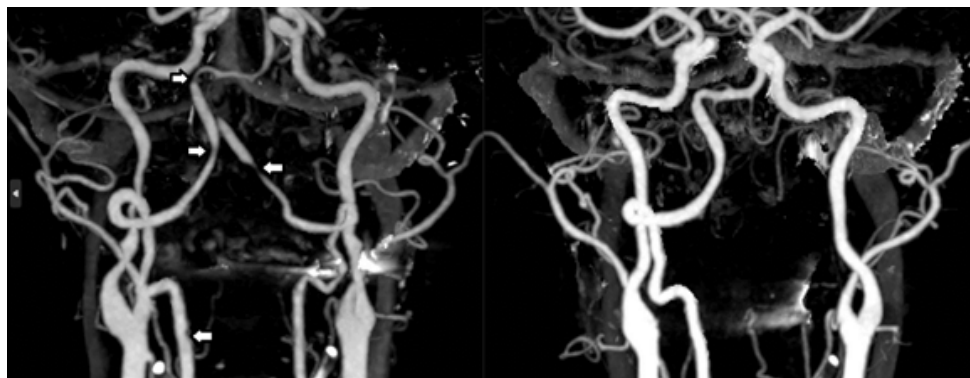


Figure 1: (A) Computed tomography of circle of Willis showing multifocal narrowing (white arrows) on presentation and (B) at 2 months.

Case presentation

A 42-year-old lady presented to the emergency department with two episodes of thunderclap headache after delivering her first child via caesarean section under spinal anaesthesia 13 days earlier. There was hypertension on postpartum day 1 which normalised, and she was discharged well on postpartum day 4. The first headache occurred while straining during defaecation. Described as the worst headache of her life, it was accompanied by phonophobia but no nausea, fever, neck stiffness, or visual changes. A similar headache occurred 7 days later. Her symptoms persisted despite regular Paracetamol, Indomethacin, Propranolol and Amitriptyline. In the emergency room, her blood pressure was 174/88 mmHg but normalised with analgesia and other vital signs were normal. Glasgow coma scale was 15. Clinical examination was normal without signs of meningism or papilloedema. Investigations including full blood count, renal and liver function tests were unremarkable. Urinalysis was negative for protein. Computed Tomography (CT) venogram and contrasted magnetic resonance imaging of the brain were normal. Lumbar puncture did not reveal xanthochromia or infection. CT carotid arteries and circle of Willis (CoW) showed multifocal luminal narrowings of varying severity (Figure 1A). Autoimmune screen was negative. She was diagnosed with postpartum RCVS and started on Verapamil 40 mg twice daily.

During admission, she developed asymptomatic tachycardia (120 beats/minute). High sensitivity Troponin I was 124.9 pg/mL (<15.6 pg/mL) with subsequent rise and fall (135.9 pg/mL to 100 pg/mL). Electrocardiogram did not show acute changes. D-dimer was elevated at 1.97 µg/mL (<0.5 µg/mL), and NT-proBNP at 284 pg/mL (<100 pg/mL). Transthoracic echocardiogram (TTE) showed an ejection fraction (EF) of 49% with basal hypokinesia and apical sparing. CT coronary arteries and pulmonary angiogram were normal. She was diagnosed with postpartum TCM. Bisoprolol 2.5 mg daily and Lisinopril 5mg daily were started and breastfeeding stopped. An important differential diagnosis of postpartum headache to consider is a vascular event such as a subarachnoid hemorrhage, cerebral venous thrombosis, and cervical artery dissection. Infective causes such as meningitis or encephalitis should also be considered, especially since she had a Cesarean section under spinal anaesthesia. Pre-eclampsia is also a possibility in view of hypertension at both postpartum day 1 as well as in the emergency department. The differential diagnosis of multifocal cerebral artery narrowings include intracranial vasculitis, moyamoya disease,

and intracranial atherosclerosis. Differential diagnosis of sinus tachycardia with raised troponins and BNP includes acute coronary syndrome and pulmonary embolism. The patient was discharged after 9 days with a reduction in headache severity and frequency. She was appreciative of the care provided to her and grateful that there were diagnoses to explain her symptoms. In the outpatient setting, lisinopril was weaned to 2.5 mg daily and eventually stopped 2 weeks later in view of postural dizziness from tight blood pressure control. Interval CT CoW angiogram showed interval resolution of stenoses at 2 months, consistent with the diagnosis of RCVS (Figure 1B). Bisoprolol was stopped after cardiac function normalised 3 months later-TTE showed an EF of 62% without regional wall motion abnormalities.

Conclusion

Both RCVS and TCM share a common pathophysiological pathway of catecholamine excess and increased sympathetic activity. In pregnant and postpartum patients who present with symptoms suggestive of these uncommon conditions, it would be important to consider them as differential diagnoses.

References

1. Sanchez-Amaya DJ, Lopez-Lizarraga MA, Gutierrez Castañeda M, Araiza-Garaygordobil D, Arias-Mendoza A. Reverse Takotsubo Cardiomyopathy During Immediate Post-partum: A Case Report. *Cureus*, 2023; 15(3): 36700.
2. Tzerefos S, Aloizou D, Nikolakopoulou S, Aloizos S. Takotsubo Syndrome: Differences between Peripartum Period and General Population. *Healthcare (Basel)*. 2024; 12(16): 1602.
3. Laeeq R, Berman JS, Khalid U, Lakkis NM, Tabbaa R. Reversible Cerebral Vasoconstriction Syndrome Associated with Coronary Artery Vasospasm. *Tex Heart Inst J*. 2019; 46(2): 139-142.
4. Enderton EL, Cardwell MS. Postpartum takotsubo cardiomyopathy with reversible cerebral vasoconstriction syndrome: A case report *Case Rep Perinat Med*. 2013; 2(1-2): 21-24.
5. Mónica Brauer M, Smith PG. Estrogen and female reproductive tract innervation: cellular and molecular mechanisms of autonomic neuroplasticity. *Auton Neurosci*. 2015; 187: 1-17.
6. Singhal AB. Reversible cerebral vasoconstriction syndrome: A review of pathogenesis, clinical presentation, and treatment. *Int J Stroke*. 2023; 18(10): 1151-1160.
7. Singh T, Khan H, Gamble DT, et al. Takotsubo Syndrome: Pathophysiology, Emerging Concepts, and Clinical Implications. *Circulation*. 2022; 145(13): 1002-1019.