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Case Report

Non-invasive cardiac output monitoring in brain trauma–induced takotsubo: A case report

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Abstract

To report a case of neurogenic stress cardiomyopathy post-trauma, managed using Non-Invasive Cardiac Output Monitor (NICOM)-guided conservative therapy. A middle-aged patient with traumatic subdural and subarachnoid hemorrhages developed hemodynamic instability. ECG showed QT prolongation; echocardiography revealed apical ballooning. We used NICOM which indicated low cardiac output (3.5 L/min). Fluids and inotropes were adjusted based on NICOM trends. The patient showed gradual hemodynamic and cardiac recovery. NICOM guided safe de-escalation of inotropes. Echocardiography normalized, and the patient was discharged.
NICOM can be used for precise, non-invasive hemodynamic monitoring, aiding recovery in stress cardiomyopathy.

Keywords: Traumatic brain injury, Takotsubo cardiomyopathy, Stress cardiomyopathy, Non-invasive cardiac output monitoring, Hemodynamic instability, Apical ballooning, Neurocritical care.

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1. Introduction

Stress cardiomyopathy, commonly referred to as Takotsubo cardiomyopathy (TCM), is characterized by a transient regional impairment of left ventricular systolic function. Clinically, it mimics acute coronary syndrome but occurs in the absence of obstructive coronary artery disease. It is classically precipitated by sudden emotional or physical stress, with neurocritical insults such as aneurysmal subarachnoid hemorrhage[1] being well-recognized precipitants. However, its occurrence following traumatic brain injury is less commonly reported.[2] The management of stress cardiomyopathy in patients with neurotrauma is particularly challenging because of the delicate balance required between optimizing cerebral perfusion pressure and supporting myocardial function.[3] In such scenarios, continuous, real-time hemodynamic assessment is essential. Traditional invasive monitoring techniques, while effective, carry procedural risks including infection, thrombosis, and haemorrhage. Non-invasive cardiac output monitoring (NICOM) provides an attractive alternative, offering dynamic cardiac performance data without the complications associated with invasive devices. This report presents a case where NICOM played a important role in guiding conservative hemodynamic management in a patient with stress cardiomyopathy complicating traumatic brain injury.

2. Case Report

A middle-aged male was admitted to the Trauma Centre ICU, AIIMS New Delhi, following a fall from the first floor on May 28, 2023. Initially managed at a peripheral hospital, he was referred for suspected intracranial bleeding. On arrival, primary survey revealed a patent airway, spontaneous breathing (18/min), SpO_2 99%, pulse 91/min, BP 115/64 mmHg, and a GCS of E4V4M5. Chest auscultation was clear, and pupils were equal and reactive.

Focused sonography (FAST) and a pneumo-scan were unremarkable. Non-contrast CT head showed subdural hemorrhage (SDH) and sulcal subarachnoid hemorrhage (SAH). The patient was managed conservatively with oxygen, intravenous fluids, and antibiotics, and shifted to the neuro-ICU.

Immediately after receiving from emergency, the patient developed hemodynamic instability with hypotension initiating noradrenaline infusion. Condition was not improved which led to Electrocardiogram (ECG) revealing QT interval prolongation and T wave inversions in leads V4 to V6 (**Figure 1**a). A bedside transthoracic echocardiogram demonstrated characteristic apical ballooning of the left ventricle with mid and apical regional wall motion abnormalities (RWMA) (Video 1a, b), with Ejection fraction of 20-30% (**Figure 2**a), cardiac output of 3.4 L/min (**Figure 2b**), without evidence of left ventricular outflow tract obstruction (LVOTO), valvular dysfunction, or B-lines on lung ultrasound. Interestingly, Troponin I levels were slightly high non suggestive of myocardial infarction.

After ECHO, a provisional diagnosis of TCM was established and Noradrenaline was stopped and levosimendan was started. To guide hemodynamic management, Non-invasive cardiac output monitoring (NICOM) was instituted early, revealing an initial cardiac output of 3.5 litters per minute (**Figure 2c**). NICOM parameters were continuously monitored and used to titrate ionotropic support, adjust systemic vascular resistance, and carefully manage fluid therapy. A strategy of cautious fluid restriction was adopted to avoid exacerbating intracranial pressure as well as cardiac failure, while maintaining adequate cerebral perfusion guided by real-time trends in stroke volume index and systemic vascular resistance index. Over the following days, progressive clinical improvement was noted with normalization of NICOM-derived hemodynamic parameters, gradual tapering and cessation of inotropes, and improvement in echocardiographic as well as ECG findings (**Figure 1b**). The patient stabilized hemodynamically, recovered well neurologically, and was subsequently discharged in a stable condition without residual deficits.

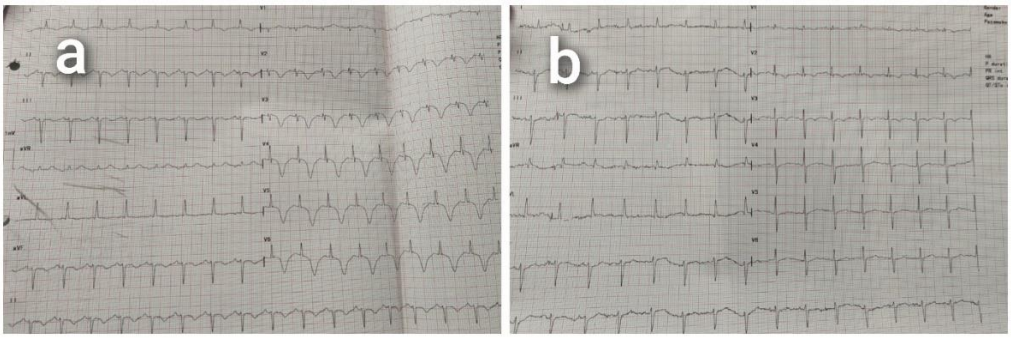

Figure 1: (a): Initial ECG showing T wave inversion in lead V1-V4; **(b)**: Post recovery ECG with normal lead findings

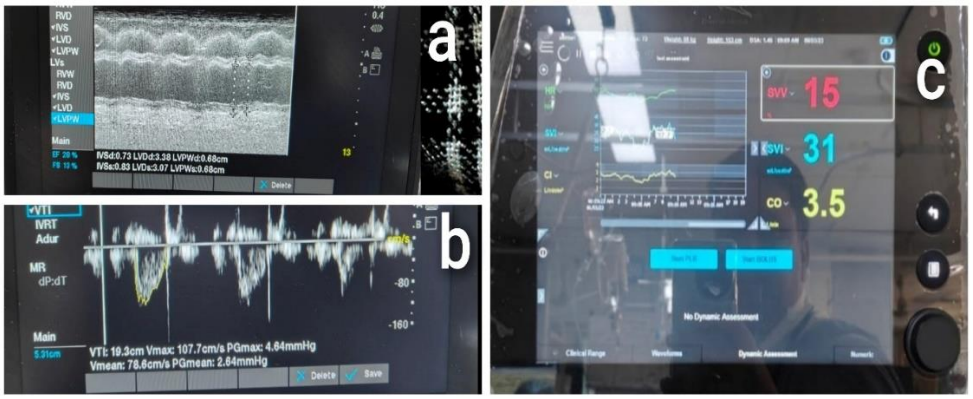

Figure 2: (a): LVEF 28%; **(b)**: 2D ECHO showing VTI of 19.3 for which cardiac output of the patient was calculated as 3.4 L/min; **(c)**: NICOM showing cardiac output of 3.5 L/min (yellow) and Stroke volume index of 31 ml/m^2 (blue)

3. Discussion

Cardiac dysfunction is a recognized complication of traumatic brain injury (TBI). A retrospective study reported myocardial dysfunction in 22% of isolated TBI cases, with 12% showing reduced ejection fraction and 17.5% having regional wall motion abnormalities.[4] TCM in TBI is rare, with only 17 cases reported,[5] and a large database study even suggested a negative association between TBI and TCM.[6]

This case highlights the complicated pathophysiological relationship between acute neurogenic stress and myocardial dysfunction, a phenomenon well-described in the context of aneurysmal subarachnoid hemorrhage but less frequently recognized following traumatic brain injury.[2] It is driven by a catecholamine surge resulting in myocardial stunning. In this patient, the development of hypotension and echocardiographic evidence of stress cardiomyopathy, raised troponin I following traumatic SDH and SAH complicated the already precarious balance of ensuring adequate cerebral perfusion while avoiding myocardial overload.[7] As there was no LVOTO conservative management (levosimendan) to maintain hemodynamic was the main goal of the management.[8] Traditional management strategies in such cases typically involve cautious use of ionotropic support and judicious fluid therapy. However, titrating these interventions without continuous hemodynamic feedback increases the risk of either under-resuscitation or volume overload.

In this scenario, NICOM proved invaluable by providing continuous, non-invasive, real-time data on cardiac output, stroke volume index, and systemic vascular resistance index. These parameters allowed precise adjustments in inotropic therapy and fluid management tailored to the patient's evolving condition. Unlike invasive monitoring systems, NICOM posed no additional procedural risks, an important consideration in neurotrauma patients where minimizing infection risk and avoiding coagulopathy-related complications is crucial. While the use of NICOM in stress cardiomyopathy in perioperative period has been described in literature[9], its application in trauma-induced stress cardiomyopathy is absent. This case supports NICOM's practical utility in neurocritical care, especially in scenarios demanding a delicate hemodynamic balance.

In this case, NICOM allowed:

1. Continuous, non-invasive tracking of cardiac output, SVI, and SVRI.
2. Timely adjustments in ionotropic therapy.
3. Safe fluid titration avoiding volume overload.
4. Individualized hemodynamic optimization without invasive lines.

4. Conclusion

Non-invasive cardiac output monitoring (NICOM) is an invaluable adjunct in the conservative management of stress cardiomyopathy complicating traumatic brain injury. It provides real-time, risk-free hemodynamic data that guide precise ionotropic and fluid therapy, ensuring both myocardial recovery and optimal cerebral perfusion.

Declaration of Patient Consent

Informed patient consent was obtained for publication of clinical information.

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Nil.

Conflict of Interest

Nil.

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