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Neurogenic Stunned Myocardium: An Unusual Cause Of Shock In Brain Injury

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Abstract
Neurogenic stunned myocardium (NSM) is a transient cardiac dysfunction associated with acute central nervous system (CNS) injury, characterized by cardiac enzyme elevation ,electrocardiographic changes , and regional or global kinetic wall motion abnormalities in absence of significant coronary artery disease. It presents a diagnostic challenge due to its resemblance to Takotsubo cardiomyopathy and acute coronary syndrome. Here we report a case of a 9-year-old male with severe traumatic brain injury who developed cardiogenic shock post-surgery, ultimately diagnosed with NSM. Even with maximum hemodynamic support, the child didn't survived for 48 hours. This case shows the importance of recognizing NSM early to improve the patient outcomes. A comprehensive review of pathophysiology, diagnostic modalities, clinical presentations, , and the management strategies is provided to enhance understanding of the condition.

Keywords: cardiogenic shock, , subarachnoid haemorrhage, traumatic brain injury , catecholamine surge, stunned myocardium, , neurogenic

Introduction

Neurogenic stunned myocardium (NSM) is a form of stress induced cardiomyopathy that occurs secondary to acute CNS injury. It was first described in subarachnoid haemorrhage , NSM has since been identified in other CNS pathologies, including cerebral infarction, cerebral haemorrhage, traumatic brain injury (TBI), and epilepsy [1,2]. It manifests with ECG abnormalities and transient left ventricular (LV) dysfunction, echocardiographic evidence of regional wall motion dysfunction, and elevated troponin levels often mimicking ACS or Takotsubo cardiomyopathy [3].
NSM occurs due to increased catecholamine release that leads to direct myocardial toxicity, microvascular dysfunction and transient myocardial dysfunction. The incidence of NSM in patients along with SAH has been found to be 30-33%, while in cases with TBI, the incidence remains under recognized due to the overlapping of clinical presentations [4,5]. This paper discusses a rare case of NSM following severe TBI and shows the necessity of increased awareness and early intervention in the patients of NSM.

Case Presentation A 9-year-old male child was admitted to our hospital following an road traffic accident the child presented with unconsciousness, multiple scalp lacerations, and severe head trauma. Initial NCCT of the brain revealed a depressed skull fracture with was associated with subarachnoid haemorrhage. Glasgow Coma Scale (GCS) of the patient was 5/15, which indicated severe neurological impairment. Emergency neurosurgical intervention was done to relieve intracranial pressure and repair the skull defect.
Postoperatively, the child developed cardiogenic shock of uncertain etiology, characterized by hypotension (Blood Pressure(BP): 70/40 mmHg), tachycardia (heart rate(HR): 140 bpm), and reduced

cardiac output. Despite adequate fluid resuscitation, the patient remained hemodynamically unstable. ECG was normal. Serum troponin levels were significantly elevated (5.2 ng/mL; normal <0.04 ng/mL), suggesting myocardial injury. Echocardiography revealed regional wall motion abnormalities with LV hypokinesia.
Septic shock and hypovolemic shock were ruled out based upon clinical and laboratory findings. The absence of the primary cardiac pathology and the presence of a recent CNS insult led to the diagnosis of NSM. Despite aggressive supportive care with vasopressors (norepinephrine and dobutamine) and mechanical ventilation, the patient could not be revived and continued to deteriorate, ultimately leading to multi-organ dysfunction and unfortunately succumb to illness within 48 hours of admission .

Discussion NSM results from an excessive catecholamine surge secondary to autonomic dysregulation following acute CNS injury. The primary mechanisms involved include:

Mechanism	Description
Catecholamine toxicity	Direct myocardial injury due to excessive catecholamine release, leading to apoptosis and necrosis.
Microvascular dysfunction	Vasoconstriction and endothelial damage impair myocardial perfusion, leading to ischemia.
Sympathetic overstimulation	Overactivation of the autonomic nervous system causes excessive beta-adrenergic stimulation, leading to calcium overload and myocardial contractile dysfunction.

In clinical practice, differentiating NSM from ACS and Takotsubo cardiomyopathy is difficult. While Takotsubo cardiomyopathy presents with similar ECG and echocardiographic features, it predominantly affects postmenopausal women and is stress-induced rather than CNS-mediated [9]. Unlike ACS, NSM exhibits normal coronary arteries on angiography, making invasive cardiac evaluation unnecessary in most cases.

Diagnostic Approach: A systematic approach is essential for diagnosing NSM in the setting of acute brain injury. Key diagnostic criteria include:
• History of acute cns insult (e.g., SAH, TBI, stroke)
• Cardiogenic shock or unexplained LV dysfunction
• Elevated cardiac biomarkers (troponin, CK-MB)
• Echocardiographic findings of regional/global LV dysfunction
• Absence of coronary artery disease on ct angiography

Management Strategies: There is no definitive treatment for NSM; management remains largely supportive.
• **Hemodynamic stabilization** – Inotropic agents (dobutamine, milrinone) and vasopressors (norepinephrine) may be required to maintain adequate cardiac output.
• **Beta-blockade** – Selective beta-blockers (e.g., esmolol) may be beneficial in reducing excessive catecholaminergic stimulation while maintaining cardiac function.
• **Mechanical ventilation** – Required in cases with respiratory failure due to concurrent neurogenic pulmonary edema.
• **Intracranial pressure management** – Optimizing cerebral perfusion pressure is essential in cases with severe CNS injury to mitigate secondary injury effects.
Despite advancements in supportive care, NSM carries a high mortality risk, particularly in severe TBI cases with profound autonomic dysfunction. The prognosis depends on the severity of CNS injury

and the degree of myocardial dysfunction. Early identification and prompt initiation of supportive therapy are crucial in improving patient outcomes.

Conclusion – it is very important to consider NSM in a patient with neurologic pathology and unexplained cardiogenic shock when hypovolemic shock is ruled out . early recognition of this condition is important to improve the clinical outcomes in these patients.

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