"Neurogenic stunned myocardium": A rare but real possibility in a paediatric patient

Acute non-traumatic brain injury precipitating cardiac dysfunction is not a common occurrence in children. Neurogenic stunned myocardium (NSM) and Takotsubo cardiomyopathy (TC) are interchangeably used to describe the stress cardiomyopathy.[1,2] TC predominantly causes mid-segment hypokinesia with apical ballooning, whereas NSM presents as a basal hypokinesia.[3] Both forms occur due to excessive catecholamine release. NSM has been described in many conditions such as stroke, subarachnoid haemorrhage, traumatic brain injury, status epilepticus and hydrocephalus.[4-6]

A 7-year-old female child, with no known comorbidities, presented to the emergency department with complaints of progressive headache and vomiting

of 7 days duration. She had no focal neurological deficits, obeying commands. Plain computed tomography (CT) of the head revealed a left parasagittal intracerebral haematoma, compressing the frontal horn of the left lateral ventricle. Her routine blood investigations and chest x-ray were unremarkable. The child was managed conservatively for her raised intracranial pressure (ICP) with intravenous mannitol 20% and dexamethasone. Intravenous phenytoin was started for seizure prophylaxis. On the second day of admission, she developed three episodes of generalised tonic-clonic seizures and became unresponsive. The airway was secured and antioedema and antiseizure prophylaxis continued.

A repeat plain CT of the head showed an increase in the size of the haematoma with impending uncal herniation. A decision was made to surgically evacuate the haematoma.

General anaesthesia was induced after connecting standard monitors, with intravenous thiopentone 80 mg intravenous fentanyl 30 mcg and intravenous atracurium 10 mg, and the trachea was intubated. The baseline vitals were found to be within normal range except for a sinus tachycardia. Anaesthesia was maintained with 0.8 MAC sevoflurane in 50% air oxygen mixture with ventilation adjusted to an endtidal CO_2 of 35 mm Hg. A few minutes following induction, the heart rate increased from a baseline value of 142-188 bpm. The blood pressure dropped from 84/38 to 40/20 mm Hg. An arterial catheter was placed in the left radial artery for invasive blood pressure monitoring. Fluid deficit was suspected and 100 mL crystalloid bolus was given. However, there was no improvement in blood pressure. A second fluid bolus was given with two boluses of mephentermine 3 mg. As the blood pressure did not improve, norepinephrine infusion was started at a dose of 5 mcg/kg/min. Simultaneously, a transthoracic echocardiographic evaluation of the heart was done on the OR table under the surgical drapes [Video 1]. The subcostal window was accessible as it showed a hypokinetic left ventricle (LV), hypokinesia being predominant in the mid-segment and basal regions, with normal contractility of the LV Apex [Figure 1]. NSM was suspected and dobutamine 5 mcg/kg/min was added to noradrenaline. Her tachycardia persisted but the blood pressure improved to 70/40 mm Hg. As her baseline haemoglobin was 9.3 g/dl, intraoperative blood loss (180 ml) was replaced with equal volumes of packed red blood cells (pRBC). In addition to blood, the patient received 40 ml/h of crystalloid as maintenance fluid during the surgery which lasted for 4 h. After surgery, the patient was shifted to the intensive care unit (ICU) for elective ventilation.

ECG showed sinus tachycardia [Figure 2]. In the ICU, the blood pressure gradually rose to 90/40 mm Hg. Dobutamine and noradrenaline were tapered over the next 3 days and stopped. Daily screening with echocardiography showed a progressive improvement in LV contractile function with a normal cardiac contractility by day 6. Cardiac troponins was not measured due to non-availability.The patient was gradually weaned off from ventilatory support by 18th POD.

In the present case, a sudden rise in ICP due to rebleed into a possible pre-existing lesion and seizures could have precipitated the NSM following anaesthetic induction leading to myocardial depression and reduction in systemic vascular resistance, and resultant hypotension. Repeated fluid administration in this condition could have worsened her cardiac condition. In previously published literature on children, the majority had lesions in the brainstem, cerebellum and midbrain and rarely thalamus.[3,6,7]

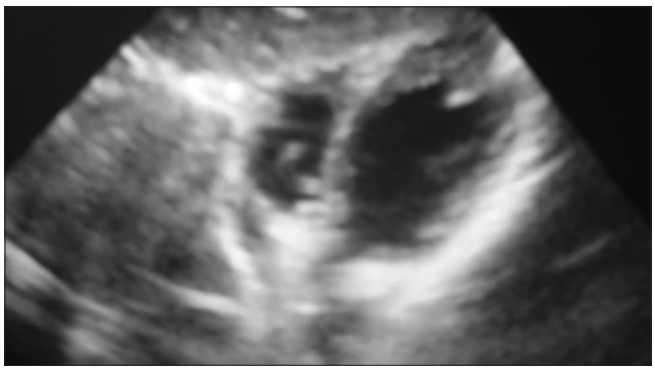

Figure 1: (Original) intraoperative echo four-chamber subcoastal view showing hypokinetic LV

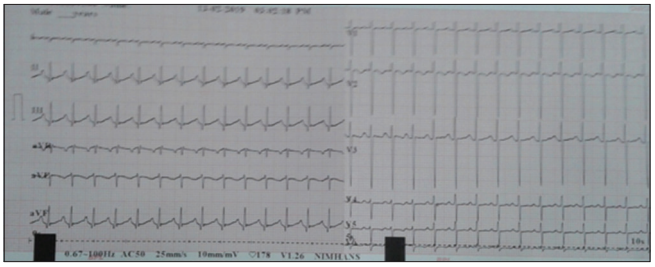

Figure 2: (Original) ECG immediate postoperative showing sinus tachycardia

The reversibility of the myocardial dysfunction with supportive therapy suggests the diagnosis of NSM rather than structural heart disease or coronary artery disease. Early detection and good supportive care is the key to recovery in these children.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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