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Neurogenic stunned myocardium complicated by aspiration pneumonia: A case report

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Abstract
Neurogenic stunned myocardium (NSM) is seen in cases of Neurological damage (stroke, bleed, seizures, hypoxia) which leads to neurological driven catecholamine rise which causes stress cardiomyopathy. ECG & 2D ECHO shows changes like cardiac strain and Low EF, making it confused with Myocardial infarction. Cardiac markers are generally elevated. There are no clear guidelines and treatment protocols available. Prognosis depends on the severity of neurological event and other comorbidities. In this article we are presenting a case of young boy ending up with Neurogenic stunned myocardium and death after a seizure episode.

Keywords: Neurogenic stunned myocardium, aspiration pneumonia, hypoxia, seizure

Introduction
Neurogenic stunned myocardium and Takotsubo cardiomyopathy are two phenotypes of Stress cardiomyopathy. Etiology is different in both conditions however they share similar pathogenesis. NSM is more associated with conditions like Stroke and SAH. Other rare conditions that can cause NSM are Brain trauma, Epilepsy and Reversible posterior encephalopathy. ANS damage and sympathoadrenergic hyperactivity, catecholamine surge leading to cardiac dysfunction is the mechanism of pathogenesis. Cardiac biomarkers and ECG, Echocardiography are useful diagnostic modalities. Treatment guidelines are not standardized. Prognosis and outcomes depend on primary neurological event. In this article we are focusing on a young epilepsy patient who developed NSM and succumbed to his illness.

Case presentation
Index case is a 13yr old male with childhood history of seizures on regular Anti epilepsy medication. Patient had visited a water sports theme park for school excursion. He had developed one episode of seizure followed by loss of consciousness. He was initially managed by paramedical personnel at water theme park with oral levetiracetam syrup. He had 1 episode of vomiting.

Patient was referred to our center for further management. Patient was received in our ER 1 hour after seizure episode. His initial examination findings were Unrecordable BP, Pulse rate 114 bpm, respiratory rate 40cpm, Spo2 was not recordable. Patient was irritable and not obeying commands. Chest examination showed bilateral diffuse coarse crepitations. Neurology opinion was taken. Plan was to do MRI Brain and EEG. ECG was done which showed Sinus tachycardia, ST elevation in Lead I, AVL and ST depression in lead 2, 3, AVF & V2 to V6. (Fig 1) 2D ECHO was done which showed EF=35%, Global hypokinesia more at mid & apical region, Dilated LV/RA/RV, Grade3 Diastolic dysfunction.

In view of Low GCS and severe respiratory distress and Cardiac failure patient was intubated and started on Mechanical ventilator support and vasopressors, broad spectrum antibiotics were initiated. ABG analysis done immediately after intubation showed pH 6.94, Pco2- 49.4, Po2- 62.5, HCO3- 8.6, Lactates 11.7, mixed picture of Severe Metabolic & Lactic acidosis. His Blood investigations showed Hb- 12.8 gm/dl, TLC- 24500 cells/microliter, Platelet count 5.24 lacs/microliter, Serum calcium 8mg/dl, serum magnesium 2.5mg/dl, Blood urea- 21mg/dl, serum creatinine- 1.2mg/dl. Urine examination showed Albumin 2+, Sugars 1+, Pus cells 4-6/hpf.

Troponin levels were raised (0.57ng/ml). CPK-MB levels were raised (10.1ng/ml), NT pro BNP levels were 104 pg/ml. Cardiologist opinion was taken. Diagnosis of Neurogenic stunned myocardium was made.

Patient was shifted to Radiology for imaging. HRCT Chest showed Bilateral symmetrical consolidatory collapse seen involving posterior segments of bilateral lower lobes with features of air bronchogram & adjacent hazy GGO's (Aspiration pneumonitis), Multi focal fluffy areas of GGO's with basal collapse/ consolidation changes noted in bilateral lung fields, predominantly along peri hilar location and both lower lobes (Pulmonary edema). Diffuse ground glass & confluent airspace densities noted involving both lung fields with interlobular septal thickening. (Fig 2, 3) MRI Cervical spine was done to look for any cervical cord injuries which was normal. (Fig 4) CT Brain (Non-Contrast) was done which showed Effaced sulcal spaces with slit like ventricles suggesting of Mild cerebral edema. (Fig 5, 6) MRI Brain was planned to rule out Hypoxia related encephalopathy, however patient developed Ventricular Tachyarrhythmias for which patient was shifted back to ICU and started on antiarrhythmic medications.

Patient had developed recurrent episodes of Ventricular tachyarrhythmia and had cardiac arrest, followed by CPR and return of spontaneous circulation. Patient vasopressor support also increased and he had prolonged Hypotension and Hypoxia. 14 hours after time of admission patient had again tachyarrhythmia followed by bradycardia and succumbed to his illness and declared dead even after resuscitation efforts.

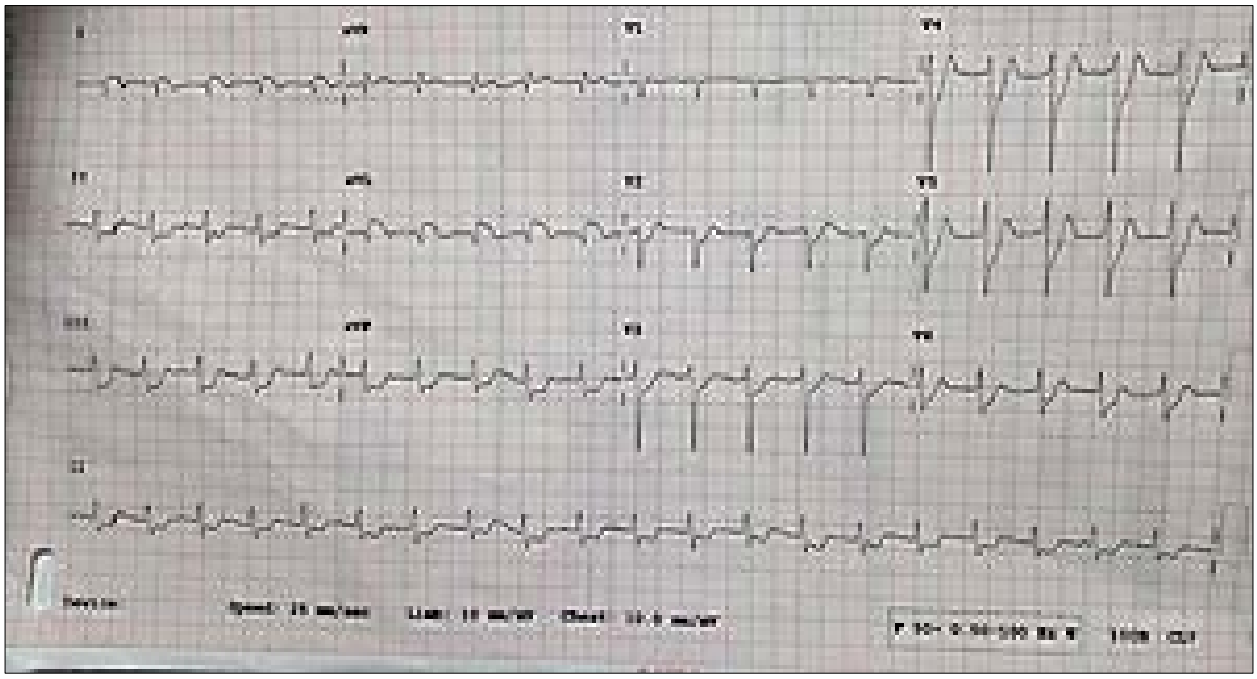

Fig 1: ECG showing ST elevation in lead I, AVL. ST depression in lead II, III, AVF, V2-V6

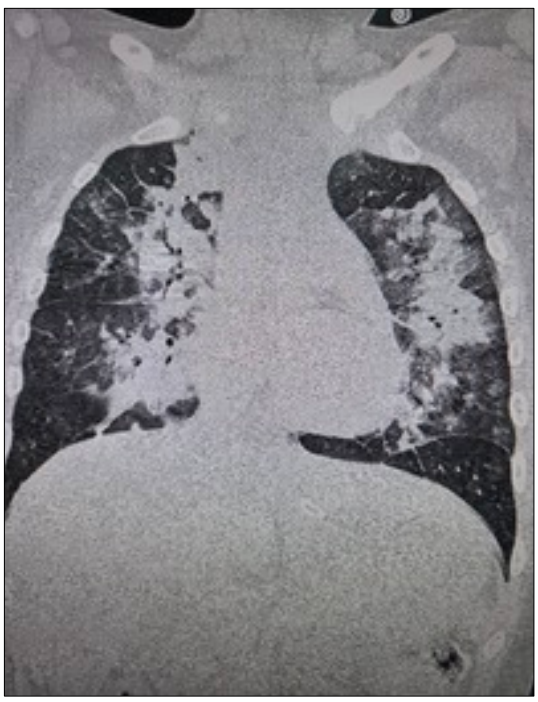

Fig 2: Coronal plane HRCT Chest

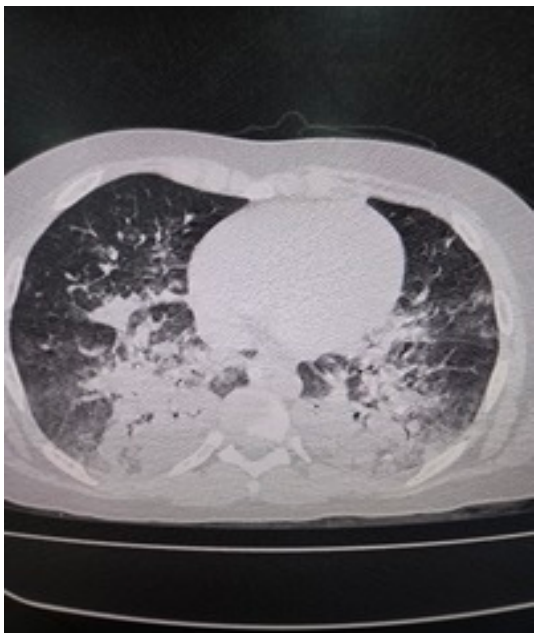

Fig 3: Axial plane HRCT Chest showing Bilateral central GGO's seen in bilateral peri hilar regions and lower lobes (Pulmonary edema with Aspiration Pneumonia)

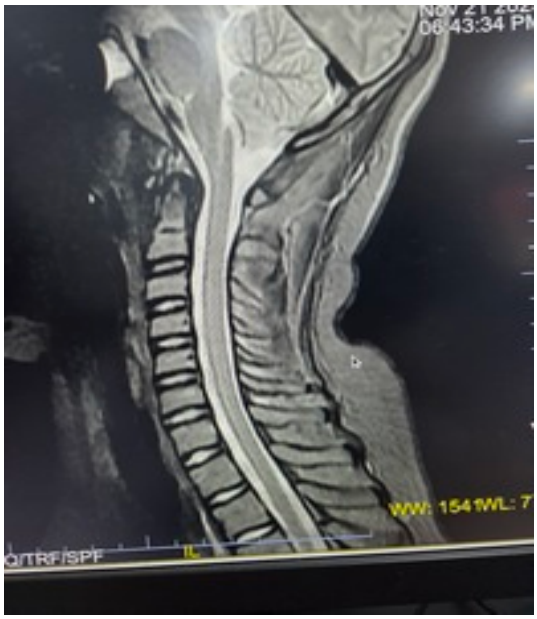

Fig 4: Sagittal STIR MRI Cervical spine which appears normal.

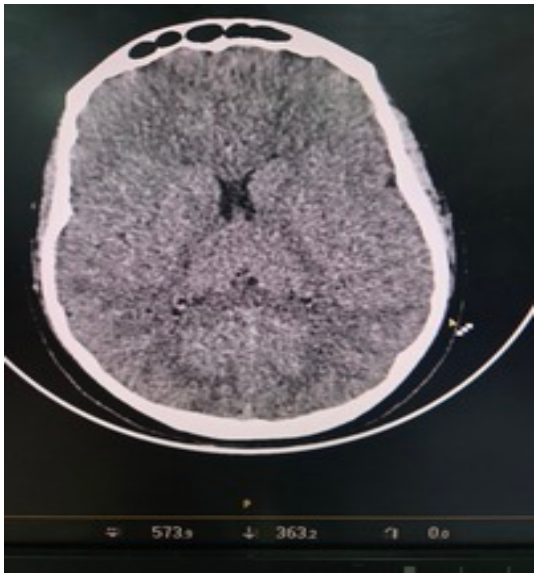

Fig 5: Axial plane CT Brain

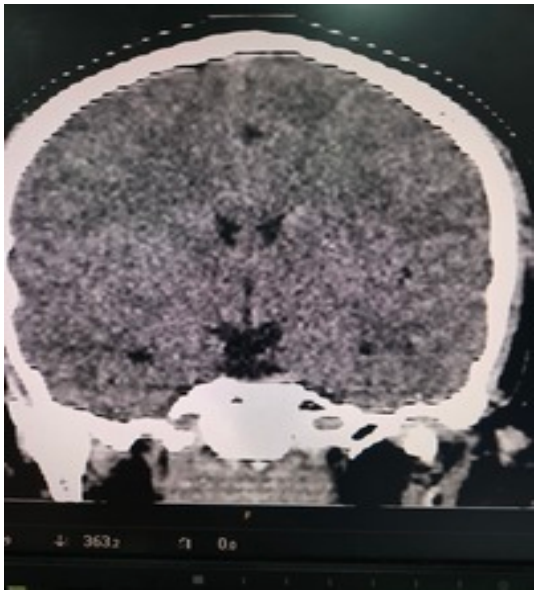

Fig 6: Coronal plane CT Brain showing effaced sulcal spaces with slit like ventricles suggesting Mild cerebral edema

Discussion

Stress cardiomyopathy has 2 phenotypes NSM and TTS. Earliest recorded association between cerebrovascular accidents and cardiac abnormalities in ECG was described by George Burch in 1954 [1]. Suto *et al* in 1990 described similar clinical features as seen in MI in cases with normal coronary angiography, reversible ventricular dysfunction with apical ballooning [2]. In 1982 Braunwald and Kloner gave concept of Stunned myocardium as reversible post ischemic contractile dysfunction accompanied by biochemical dysfunction [3].

NSM is better studied in patients with stroke and is referred as "Stroke Heart syndrome" [4]. It is also seen more often associated with SAH [5]. NSM is also seen in other neurological conditions like Brain trauma, Epilepsy and reversible posterior encephalopathy [6-8]. NSM is characterized by acute reversible ventricular myocardial dysfunction following neurological event which causes damage to Autonomic nerve centers, sympathoadrenergic hyperactivity, catecholamine surge and its cardiovascular effects.

Pathophysiology of NSM & TTS is similar. It includes i) Primary injury ii) Nerve centers involvement, beta adrenergic axis, sympathetic hyperstimulation and catecholamine release iii) cardiac response to excessive catecholamine leading to myocardial ischemia and ventricular dysfunction. In NSM the stressors are neurological events which lead to injury of Insular cortex which is involved in cardiac function control and sympathetic adrenergic tone [9, 10]. Injury to Insular cortex and parietal lobe leads to catecholamine release. Locus coeruleus which receives afferents from hypothalamus is main site of catecholamine synthesis in brain. CNS activation leads to ANS hyperactivity leading to hyper catecholaminaemia [11, 12]. Increase in catecholamines and adrenergic hyper stimulation causes endothelial dysfunction, microvascular spasm, micro thrombosis, increased contractility leading to myocardial ischemia and acute LV dysfunction [13, 14]. Reperfusion changes that accompany reduction of vasospasm and endothelial dysfunction contribute to Contraction band necrosis and cardiomyocyte function impairment [15].

Clinical features in NSM are mostly asymptomatic, sometimes have cardiac arrhythmias and signs & symptoms of Acute heart failure. ECG shows changes such as QT prolongation, ST elevation, ST depression, Arrhythmias, Atrial fibrillation and U waves. Index case had ST depression and ST elevation in ECG. Echocardiography features in SCM include akinesia/dyskinesia in apical ballooning, midventricular, inverted basal, focal and biventricular pattern. Index case had mid & apical region hypokinesia. Cardiac biomarkers (troponin, NT pro BNP) are usually elevated as is seen in our index case. Increase in hs Troponin levels is moderate in TTS & NSM and is disproportionate to magnitude and type of ECG abnormalities, as seen in our index patient. Cardiac MRI and Coronary angiography is generally advised in patients where there is a diagnostic dilemma between SCM and MI.

Treatment of SCM is not standardized. Risk stratification is made based on ventricular dysfunction, arrhythmias and comorbid conditions. In mild cases ECG and 2D ECHO findings are generally reversible not requiring any specific therapy. Beta blockers can be given in cases of adrenergic hyperstimulation. In higher risk cases treatment choice is

based on type of complications. Calcium channel blockers associated to have faster recovery. Heart failure in SCM is similar as in other heart failure cases. In cases where hemodynamic instability is there agents like dobutamine and levosimendan can be considered [16]. NSM associated with atherosclerotic event requires long term statins, antiplatelet drugs and neurological rehabilitation. In NSM generally disease course is benign. However outcomes might vary according to severity of primary neurology event. Moderate elevations of troponin levels are generally associated with poor prognosis including death [17, 18]. Index case also had troponin elevation with severe changes in ECG, 2D ECHO and had complication secondary to Aspiration pneumonia along with Heart failure. He also had early signs of Hypoxic brain injury.

Conclusion
In cases where cardiac abnormalities follow a neurological event like Stroke, SAH, Seizure or Hypoxia, NSM should be kept as a possibility. ECG & 2D ECHO are helpful in diagnosis. Cardiac enzymes are generally elevated. NSM is easily confused with Myocardial infarction. Treatment guidelines are not clearly stated for this condition, it is managed in lines of Stress cardiomyopathy. Outcomes depends on comorbidities, primary neurological condition and cardiac recovery.

Declarations
Abbreviations: Neurogenic Stunned Myocardium = NSM, Autonomic nervous system= ANS, TTS = Takotsubo syndrome, MI = Myocardial infarction, SCM= Stress Cardiomyopathy.

Ethics Approval and Consent to participate
Not Available

Consent for Publication
Informed Consent was obtained from the subject parents.

Availability of Data and Materials
Data is contained within the article.

Conflict of Interest
Authors declare no conflict of interest.

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