

Case Report

Takotsubo Cardiomyopathy in A 23 Months-Old Following Traumatic Brain Injury

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Abstract

Takotsubo cardiomyopathy is not infrequently encountered in adult populations, but has rarely been described in pediatric patients. Takotsubo cardiomyopathy is a form of stress-related cardiomyopathy, that usually occurs after significant emotional or physical stressors leading to autonomic storm, although this is not required for diagnosis. It is indistinguishable from an acute myocardial infarction and often times resolves without intervention. We report a case of a 23-month-old male who suffered traumatic brain injury, and Takotsubo cardiomyopathy 11 days after the initial injury. We propose either hypoglycemia or cessation of sedation as the possible cause of Takotsubo cardiomyopathy.

Keywords

- Takotsubo cardiomyopathy
- Brain injuries
- Pediatrics

ABBREVIATIONS

CK - Creatinine Kinase; **CK-MB** - Creatinine Kinase-MB isoenzyme ; **CT** - Computed Tomography ; **EKG** - Electrocardiogram ; **GCS** - Glasgow Coma Scale ; **LV** - Left Ventricle ; **MRI** - Magnetic Resonance Imaging

INTRODUCTION

Takotsubo cardiomyopathy is a form of stress-related cardiomyopathy, that has been well-described in adults, with only a few cases reported in the pediatric population [1-3]. Stress-related cardiomyopathy is thought to occur in response to catecholamine surges secondary to a life-threatening stress causing myocardial toxicity [4]. There have been multiple theories proposed as to the cause of stress-related cardiomyopathy however the exact mechanism is not fully understood. Nevertheless, the electrocardiogram (EKG) changes associated with this phenomenon essentially mimic that of an acute myocardial infarction, and are associated with mild elevation of cardiac enzymes [4-6]. It is important to recognize stress-related cardiomyopathy or in this case Takotsubo cardiomyopathy, as a possible diagnosis in a critically ill child with significant risk factors.

CASE PRESENTATION

A 23-month-old male with no significant past medical history was admitted to our pediatric intensive care unit after falling from a 3rd floor balcony. Initial computed tomography (CT) scan revealed a large skull base fracture involving the clivus and extending to the anterior cranial fossa and cribriform plate, as well as subarachnoid hemorrhage with intraventricular, subdural

and parenchymal components. The patient was intubated for a Glasgow coma scale (GCS) of 7, and had an external ventricular drain and intracranial pressure bolt placed upon admission. The MRI performed on hospital day five revealed areas of infarction in the brainstem, as well as in the hemispheric white matter, likely secondary to post traumatic evolution of his injury. His intracranial pressure remained stable and sedation as well as neuromuscular blockade was discontinued along with the hypertonic saline infusion. The patient remained mechanically ventilated, but had minimal oxygen requirements.

On hospital day twelve, he was hypoglycemic with a serum glucose of 33mg/dL and received an intravenous bolus of glucose with subsequent correction. Shortly thereafter, the patient demonstrated changes in his heart rhythm on telemetry monitoring (Figure 1). An electrocardiogram performed during the event showed diffuse ST segment elevation, not shown on his previous electrocardiogram (Figure 2). At the time of this acute change in heart rhythm, he was being cooled for a temperature of 38.6 °C and had a heart rate of 160 bpm, and a blood pressure of 80/41 mmHg without evidence of acute changes in intracranial pressure. Electrolytes obtained demonstrated ongoing hyponatremia (sodium of 166mmol/L), a result of hyperosmolar therapy. The remainder of his electrolytes and blood gas results were normal. Cardiac enzymes revealed a mildly elevated troponin at 0.10ng/ml (range <0.06ng/ml) which peaked at 0.11ng/ml at 7 hours. Creatine kinase (CK) and Creatine kinase- MB isoenzyme (CK-MB) were 134U/L and 4.49ng/ml respectively. Echocardiogram showed mild dilatation of the left ventricle (LV) with moderate global hypokinesia and depressed function (Figure 3). Of note, the interventricular septum was

more severely affected than the LV free wall. The EKG normalized within the next 24 hours and the cardiac enzymes returned to baseline at approximately 80 hours post event (Figure 4). The child recovered from the cardiac event without requiring any interventions.

DISCUSSION

Stress-induced cardiomyopathy is a disease entity that is not frequently encountered in pediatrics. It encompasses several types including Takotsubo cardiomyopathy, acute LV dysfunction associated with subarachnoid hemorrhage, acute LV dysfunction associated with pheochromocytoma, or exogenous catecholamine administration, and acute LV dysfunction in the critically ill [4].

In 2010 the Mayo Clinic proposed four diagnostic criteria, all of which must be present for diagnosis of Takotsubo cardiomyopathy [7]. These criteria are : 1) transient hypokinesis, akinesis or

dyskinesis of the left ventricular mid segments with or without apical involvement; the regional wall motion abnormalities extend beyond a single epicardial vascular distribution; stressful trigger is often present, but not always present, 2) absence of obstructive coronary disease or angiographic evidence of acute plaque rupture, 3) new electrocardiographic abnormalities, or modest elevation in cardiac troponin, and 4) absence of pheochromocytoma, and myocarditis [7].

Our patient had risk factors for developing stress-related cardiomyopathy. He had sustained significant traumatic brain injury as a result of his fall. However, it is unclear as to the exact inciting event for his acute EKG changes 12 days later. In cases of autonomic storm or catecholamine storm, symptoms can be seen after weeks to months from the initial injury [8], although typical neurogenic stress cardiomyopathy after subarachnoid hemorrhage has been shown to develop within hours of aneurysmal rupture [9]. On the other hand, there are some

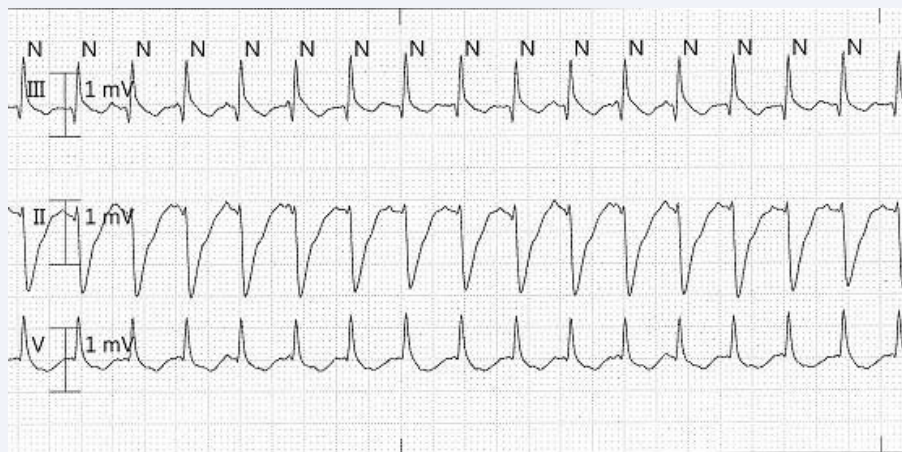


Figure 1 EKG obtained during the event on telemetry monitor.

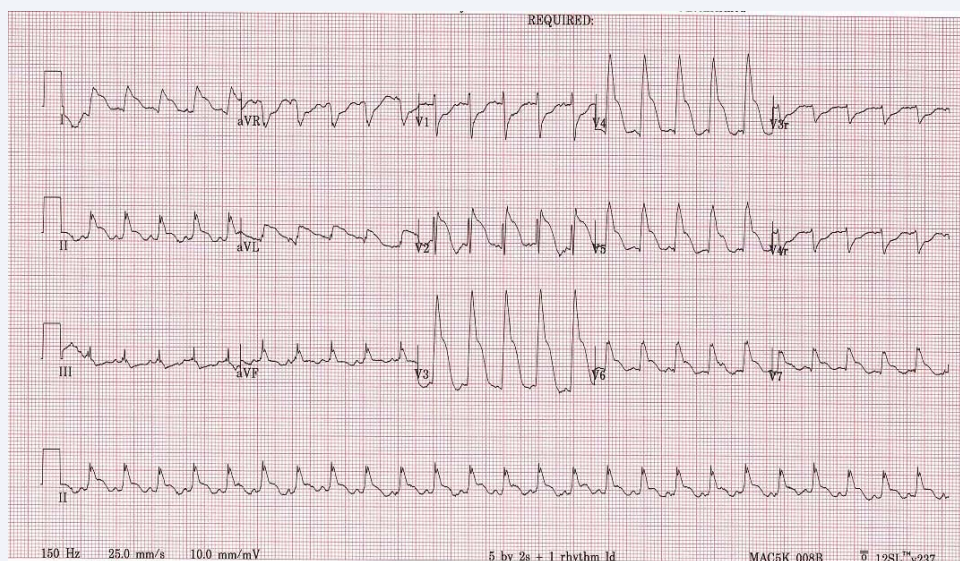


Figure 2 EKG obtained during the event (Hospital day 12) Sinus Tachycardia. Elevation segments in all leads, possible ischemic changes. Borderline prolonged QT.

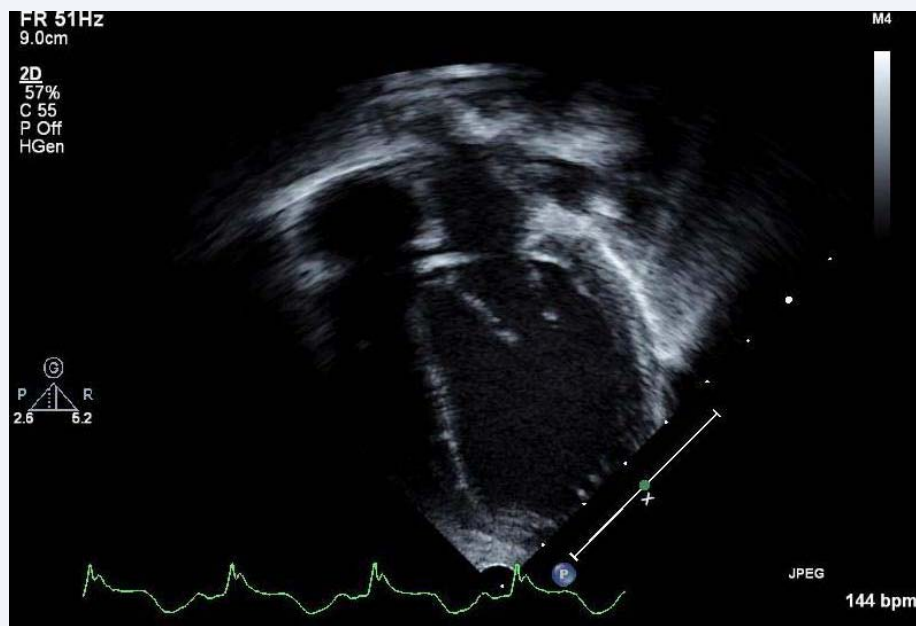


Figure 3 Mild dilatation of the left ventricle (LV) with moderate global hypokinesia and depressed function. Shortening Fraction 30%.

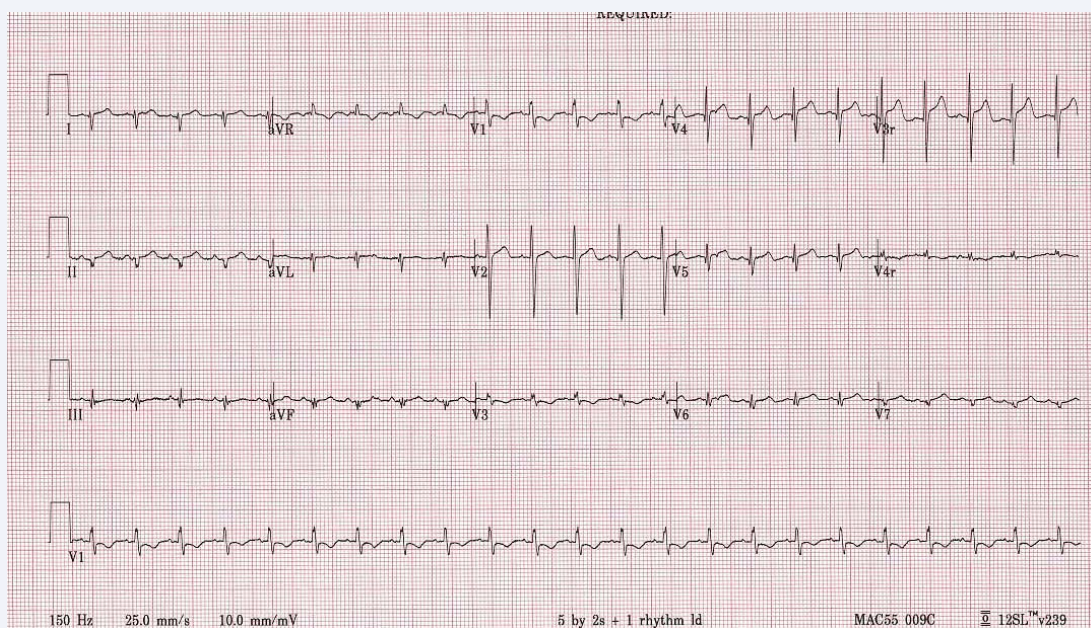


Figure 4 EKG obtained following day (Hospital day 13) Sinus Tachycardia. Low voltage.

case reports describing Takotsubo cardiomyopathy secondary to hypoglycemia [10]. We can postulate that the hypoglycemia he developed could have caused a rapid surge of catecholamine leading to stress-induced Takotsubo cardiomyopathy. It is also possible that the decreased sedation over the few days prior to this event left the patient vulnerable to autonomic storming that can be seen after traumatic brain injury.

There are three proposed mechanism for stress-related cardiomyopathy, 1) ischemic myocardial stunning due to epicardial coronary spasm, 2) acute coronary microvascular

dysfunction and 3) catecholamine-mediated direct myocardial injury. Amongst the proposed causes, catecholamine-mediated direct myocardial injury is the most widely accepted [4]. The catecholamine surge increases intracellular calcium, and overdrives oxygen-derived free radicals which cause direct myocardial toxicity that can lead to myocardial dysfunction. This mechanism could explain why infants and children experience fewer stress-related cardiomyopathies as they tend to have lower intracellular calcium storage. Further research may help elucidate this issue.

It is also important to note that once Takotsubo cardiomyopathy is suspected in a child, generally only symptomatic treatment is necessary and virtually all the cases are completely reversible. In some patients, particularly older adults, pulmonary edema, cardiogenic shock and arrhythmia can be significant in the acute phase [7].

Takotsubo cardiomyopathy may potentially occur in any patient admitted to the pediatric intensive care unit, as admission to an intensive care unit itself is a significant stressor. Therefore it is important to note that any acute EKG changes that occur in such settings require prompt attention and evaluation, but may not always warrant any intervention, especially when the patient is hemodynamically stable.

CONFLICT OF INTEREST

Jahee Hong, Lily B. Glater-Welt, and Linda B. Siegel have no potential conflicts of interest to disclose.

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