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CLINICAL REPORT

Acute intraoperative neurogenic myocardial stunning during intracranial endoscopic fenestration and shunt revision in a pediatric patient

Kristen Elizabeth Dragan[1] · William D. Patten[2] · Osama M. Elzamzamy[1] · Ahmed Fikry Attaallah[1]

Abstract Neurogenic stunned myocardium (NSM) is syndrome of myocardial dysfunction following an acute neurological insult. We report a case of NSM that occurred intraoperatively in a pediatric patient undergoing endoscopic fenestration and shunt revision. Accidental outflow occlusion of irrigation fluid and ventricular distension resulted in an acute increase in heart rate and arterial blood pressure. Subsequently, the patient developed stunned myocardium with global myocardial hypokinesia and pulmonary edema. She was promptly treated intraoperatively then admitted to the pediatric intensive care unit with resolution of her symptoms within 12 h. She was later discharged to home on the fourth postoperative day. In the current endoscopic era, this report highlights the possibility of intraoperative NSM and neurogenic pulmonary edema in the pediatric population. Early detection and treatment with a team approach help to achieve optimal control of this life-threatening condition and improve the outcome.

Introduction

Neurogenic stunned myocardium (NSM) is a syndrome of cardiac dysfunction after a neurological insult [1]. It is usually observed after aneurysmal subarachnoid hemorrhage in adults, but is increasingly being reported after other neurological events such as acute hydrocephalus [2, 3]. There are fewer reports of NSM in the pediatric population. The proposed underlying pathophysiological mechanism of NSM is believed to be a sudden increase in intracranial pressure and/or a decrease in the hypothalamus and medulla perfusion, leading to a sympathetic surge causing weakened cardiac contractility and possibly direct myocardial damage [1]. Observed cardiac changes following neurologic injury include electrocardiographic changes, evidence of myocardial necrosis, and systolic and diastolic dysfunction of the left ventricle [1–3].

Case description

History and examination

Our patient is a 10-year-old, 50-kg female with congenital hydrocephalus and previous ventriculoperitoneal (VP) shunt placement who presented with recent onset of headaches that were relieved while lying supine. She initially had a VP shunt placed shortly after birth and had undergone four revisions since that time. Her brain MRI showed ventricular collapse suggestive of cerebrospinal fluid over-drainage by the shunt system.

✉ Ahmed Fikry Attaallah
attaallaha@wvuhealthcare.com

Kristen Elizabeth Dragan
dragank@wvuhealthcare.com

William D. Patten
wpatten@hsc.wvu.edu

Osama M. Elzamzamy
zamzamyo@wvuhealthcare.com

1 Department of Anesthesiology, West Virginia University, School of Medicine, 1 Medical Center, PO Box 8255, Morgantown, WV 26506, USA

2 Department of Pediatrics, Section of Critical Care Medicine, West Virginia University, Morgantown, WV, USA

Surgical events

She was scheduled for endoscopic shunt revision. Anesthesia was induced with 8 % sevoflurane and endotracheal intubation was uneventful. Anesthesia was maintained with sevoflurane 2.5 %, remifentanil 0.05 mcg/kg/min, and boluses of rocuronium. An arterial catheter was placed and the patient's blood pressure was maintained within a narrow range of her baseline pressure (100/60 mmHg) with a heart rate of 70–80 beats/min. Shortly after the cerebral endoscopy was started, the patient became acutely tachycardic and hypertensive with a maximum blood pressure of 255/155 mmHg (mean arterial pressure 197 mmHg) and a maximum heart rate of 138 beats/min (Fig. 1). The surgeon reported that this response may be due to excess irrigation fluid stretching the cerebral ventricles. He unintentionally allowed irrigation fluid through the endoscope while the drainage port was occluded. He immediately decompressed the ventricles, and the anesthesiology team treated the patient by increasing the inhalational anesthetic concentration and by administering a 200-mcg bolus of remifentanil and a 50-mg bolus of esmolol. After 5 min, the patient's blood pressure returned to baseline and the tachycardia improved but remained in the low 100s (Fig. 1). We noted that the patient's peak inspiratory pressure slightly increased (her baseline peak inspiratory pressure was 27 cmH_2O, which then increased to 31 cmH_2O); however, her oxygen saturation remained at 100 % on FiO_2 of 0.5. She remained hemodynamically stable and the surgery was resumed. After completion of surgery and removal of the drapes, a frothy pink fluid was noticed in the endotracheal tube. Approximately 200 mL were suctioned from the patient's airway, and we decided that she would remain intubated, sedated, and mechanically ventilated.

Postoperative course

The patient was transported to the pediatric intensive care unit (PICU). Her initial ventilator settings were pressure-regulated volume control (PRVC): tidal volume = 6 mL/kg, FiO_2 = 40 %, positive end expiratory pressure (PEEP) = 10 cmH_2O, respiratory rate = 12, and pressure support (PS) = 10 cmH_2O. A postoperative electrocardiogram (EKG) was obtained, and she was found to have ST elevations in all leads and QT prolongation (Fig. 2). A chest X-ray showed a prominent interstitial pattern and hazy lung opacities, suggesting early pulmonary edema (Fig. 3). A transthoracic echocardiogram demonstrated increased right ventricular pressures of 29 mm Hg and a reduced left ventricular ejection fraction of 49 %. Furosemide 5 mg was given intravenously. Several hours later, the patient's tachycardia resolved and the EKG returned to normal. She also had decreased haziness and improved
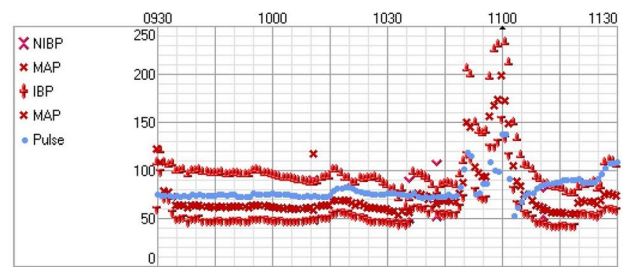

Fig. 1 Intraoperative traces for blood pressure and heart rate during the neurogenic hyperadrenergic response

aeration on subsequent chest radiographs. The FiO_2 and the PEEP were decreased to 30 % and 6 cmH_2O, and later she was weaned to continuous positive airway pressure (CPAP) ventilation. Endotracheal extubation was performed on the evening of the surgery. The patient remained stable from a respiratory and cardiovascular standpoint following extubation, and she was later discharged to home on the fourth postoperative day.

Discussion

A few authors have previously reported that acute brain injury may be associated with autonomic imbalance with subsequent catecholamine release [1–5]. Excessive catecholamines result in neurogenic stunned myocardium, which involves weakened cardiac contractility, pulmonary edema, arrhythmias, and direct myocyte damage [1, 5]. A catecholamine surge can also lead to increased pulmonary vascular resistance, increased pulmonary hydrostatic pressure, and increased capillary permeability, which worsens the pulmonary edema [4, 6].

As endoscopic neurosurgery procedures gain popularity, their complications are likely to become more common. In this case, we report acute intraoperative NSM in a pediatric patient due to a faulty irrigation system setup which permitted rapid inflow of the irrigation fluid while the egress flow was restricted. In the pediatric population, which is associated with a relatively small intracranial space, vigilance is essential to avoid this fatal complication.

While tachycardia and hypertension may be initially interpreted as light anesthesia or painful stimulus, proper communication with the surgical team to identify a potential surgical cause is imperative. Initial management must be focused on treating the underlying neurologic process, which can usually be rapidly corrected with direct decompression of the ventricles [1, 3, 5, 6]. Meanwhile, supportive care should be initiated, aiming at reducing afterload and maintaining cardiac contractility with careful monitoring to avoid compromising cerebral hemodynamics [1,

Vent. rate   81   BPM   ** ** ** ** * Pediatric ECG Analysis * ** ** ** **
PR interval   134 ms   Normal sinus rhythm
QRS duration   84   ms   ST elevation, consider early repolarization, pericarditis, or injury
QT/QTc   406/471 ms Borderline Prolonged QT
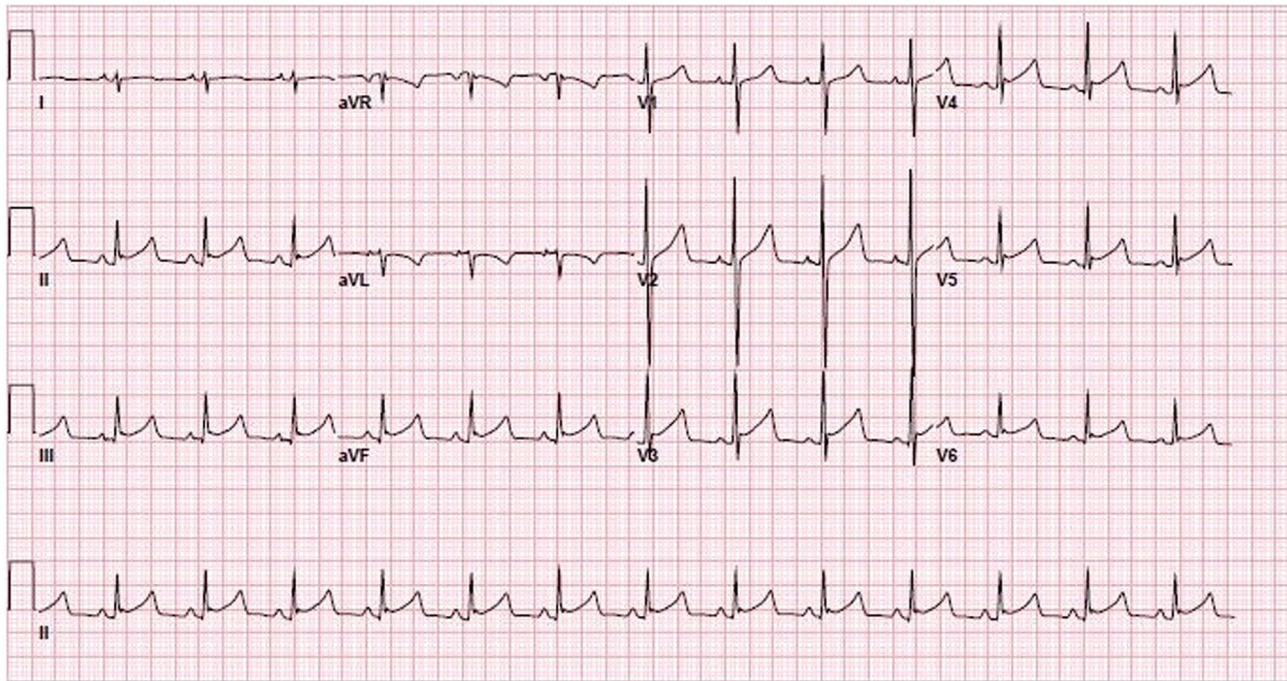P-R-T axes   59 92 82

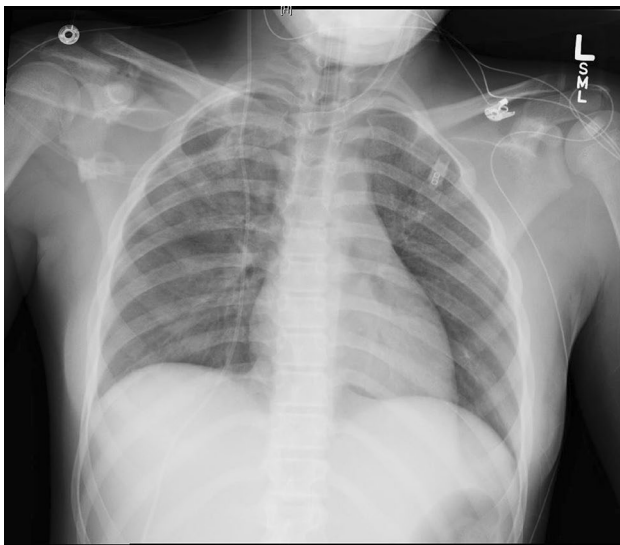**Fig. 2** Postoperative EKG showing diffuse ST elevation and QT prolongation

Fig. 3 Postoperative chest radiograph showing prominent interstitium and hazy lung parenchyma bilaterally, suggestive of pulmonary edema

5]. While short-acting direct vasodilators are the first-line treatment agents for a hyperadrenergic state [7, 8], our initial management was to increase the inhalational anesthetic and administer a bolus of opioids to decrease the response to sympathetic stimulation and produce vasodilation. After vasodilation had been achieved, as evidenced by a decrease in blood pressure, a short-acting beta-blocker (esmolol) was given to lower the heart rate [9, 10].

Neurogenic stunned myocardium and pulmonary edema can be fatal, but recovery is rapid and early with prompt recognition and treatment [1, 3, 5, 6]. Our patient's abnormal physiologic response was quickly recognized due to appropriate intraoperative monitoring and communication. A team approach to this life-threatening condition helped our patient to achieve the optimal outcome, and she was fortunate to have a fast and full recovery.

Compliance with ethical standards

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