An Adult Female with Obstructive Hydrocephalus who Presented with Neurogenic Stunned Myocardium during a Neuroendoscopic Procedure: A Case Report

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ABSTRACT

Neurogenic stunned myocardium (NSM) is a condition wherein a neurologic pathology results in myocardial injury. This is a case of an adult patient who developed intraoperative NSM presenting as hypertension and tachycardia progressing to supraventricular tachycardia and ST-elevation during an emergency endoscopic septostomy, biopsy and ventricular peritoneal shunt (VPS) insertion. It is important for the anesthesiologist to be familiar with this condition to ensure prompt recognition and management as well as to avoid significant morbidity and mortality.

Keywords: endoscopic surgical procedures, cardiac arrhythmias, supraventricular tachycardia, myocardial stunning, obstructive hydrocephalus

INTRODUCTION

Neurogenic stunned myocardium (NSM) can be defined as a myocardial injury resulting from a neurological insult which if left unaddressed can result in significant morbidity and mortality.[1] Its clinical presentation is similar to myocardial infarction in that its features include ECG wave changes, elevated troponin I levels, and left ventricular dysfunction.[1] It is difficult to differentiate the two disease entities without additional testing, especially in a neurosurgical emergency, such as severe increased intracranial pressure in obstructive hydrocephalus. In such cases, the anesthesiologist should be wary of a possible cardiac event. While most cases of NSM is associated with subarachnoid hemorrhage, this case presents a patient who developed NSM during a neuroendoscopic procedure for an underlying condition of obstructive hydrocephalus.

CASE PRESENTATION

Preoperative Course

A 52-year-old 50 kg right-handed female without known comorbidities presented with acute decreased sensorium after previously being able to independently perform activities of daily living. The patient had a two-week history of holocranial headache. It was later associated with increased sleeping time, lack of verbal output and blank stare episodes. A cranial CT scan performed at a Level 2 center allegedly showed an intracranial mass prompting referral to this tertiary institution.

The patient was received at the ER stuporous, with minimal verbal output and not in cardiorespiratory distress. Vital signs were BP 140/100 mmHg, HR 88 bpm, RR 20

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cpm, SpO_2 100% in room air. Cardiac examination showed adynamic precordium, normal rate, regular rhythm and no appreciable murmurs. On neurologic examination, she had a GCS score of 7 ($E_1V_2M_4$), negative response to visual threat, pupils that were briskly reactive to light, no facial asymmetry, supple neck and normal deep tendon reflexes. Assessment was encephalopathy from a central neurological cause, likely an intracranial mass. Medical decompression with mannitol and dexamethasone as well as seizure prophylaxis with levetiracetam were immediately instituted. This resulted in minimal improvement in sensorium necessitating intubation for airway protection.

Complete blood count, serum ferritin, random blood sugar, liver function tests, and serum creatinine results were within normal limits. 12-lead ECG showed sinus rhythm while chest radiography was suggestive of pneumonia and cardiomegaly. No hormone studies were done at this time. Once stabilized, she was referred to Neurology and Neurosurgery services. Cranial CT scan showed a sellar-suprasellar mass with right frontal lobe upward extension with internal calcifications with mass effect seen as a compression of third and right lateral ventricle with right midline shift (Figures 1 and 2). Bony lytic changes, vascular and optic nerve/tract involvement, obstructive hydrocephalus, and possible descending transtentorial herniation were also seen (Figure 3). Despite medical intervention, she deteriorated with a GCS score of 5 ($E_1V_1M_3$) requiring emergency endoscopic septostomy, biopsy and VPS insertion.

Intraoperative Course

The patient was received intubated with BP of 137/94 mmHg, HR 87 bpm, SpO_2 100% (FiO_2: 1.0) and GCS score of 7 ($E_1V_TM_5$). ECG in the monitor showed sinus rhythm. After confirmation of proper endotracheal tube (ETT) placement, fentanyl 50 mcg and atracurium 40 mg were given intravenously. Anesthesia was maintained with sevoflurane 2 vol%. Vital signs range were BP 100-120/60-70 mmHg, HR 90s and end-tidal CO_2 31-35 mmHg. No significant hemodynamic changes occurred during the burr hole craniotomy and insertion of endoscope phase of the surgery.

Upon introduction of the irrigation fluid, there was note of a rapid increase in BP (170/100 mmHg) and HR (150s). Highly considering inadequate anesthetic depth, additional fentanyl 50 mcg was administered and sevoflurane concentration was increased from 2 vol% to 3 vol%. ECG monitor at that point showed supraventricular tachycardia (SVT). The neurosurgeon was immediately informed of this development. The endoscope was instantly pulled out. Following removal of endoscope, blood pressure and heart

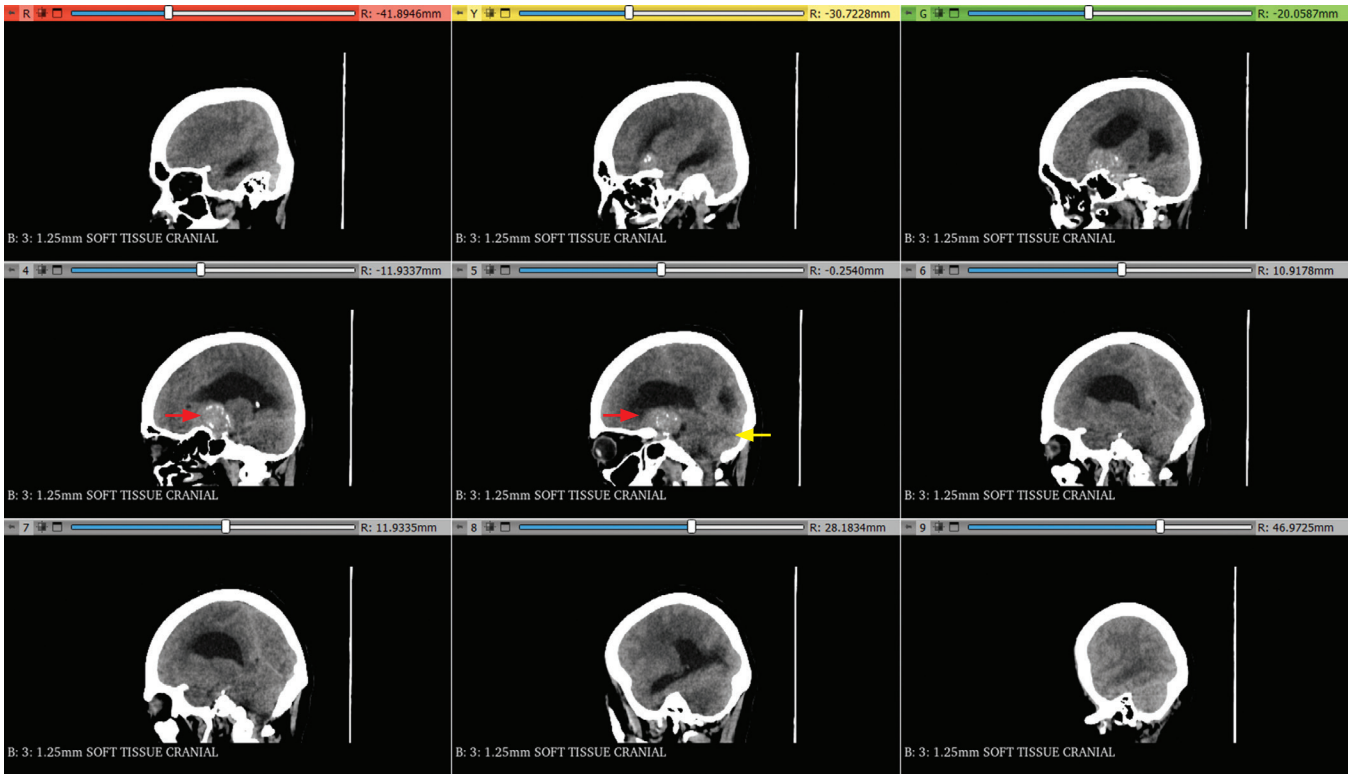

Figure 1. Sagittal view cranial CT scan with contrast. Note the 3.1 x 4.6 x 5 cm mass marked by red arrows in the sellar-suprasellar region. The mass is intimately related to the caudate nucleus and thalamus. Yellow arrow marks the midbrain being pushed inferiorly and posteriorly. No normal pituitary gland can be visualized. Diffuse cerebro-cerebellar edema can be observed.

rate reverted to baseline levels within a minute. ECG tracings on the monitor showed regular sinus rhythm but with new-onset ST-segment elevation.

Given the emergent nature of the procedure and normalization of hemodynamic parameters with endoscope withdrawal, the operation was continued. Irrigation fluid speed was decreased by the neurosurgeon on reinsertion of the endoscope. The patient was maintained on sevoflurane 2 vol% throughout the rest of the procedure. There were no further episodes of SVT observed. No cardiac medications were administered. Paracetamol 600 mg and tranexamic acid 1 gram were given intravenously. Following successful completion of septostomy, biopsy and VPS insertion, the patient was transferred to the post-operative care unit (PACU) with BP 138/88 mmHg, HR 100 bpm (regular sinus rhythm), SpO_2 of 100% and GCS score 7 ($E_1V_TM_5$).

Post-operative Course

The patient was maintained on mechanical ventilation. Levetiracetam, dexamethasone and mannitol were continued. Post-operative cranial CT scan revealed proper placement of the VPS with significant decrease in degree of obstructive hydrocephalus (Figure 4).

Differentials for the intraoperative arrhythmia were investigated. Hyperthyroidism workup showed normal FT4 levels with low TSH and T3, suggesting subclinical hyperthyroidism. HbA1c of 6.1 indicated prediabetes. Normal procalcitonin ruled out sepsis while an elevated white blood cell count (18 x 10^9/L) indicated an inflammatory response. An increase in serum ferritin to 485 ng/ml was suggestive of acute myocardial infarction.

Post-operative chest x-ray did not show pulmonary congestion but indicated beginning atelectasis due to endobronchial placement of the ETT, thus, the ETT was adjusted accordingly. A 12-lead ECG showed new-onset anterior and high lateral wall ischemia prompting cardiology referral.

On the 2nd post-operative day, the patient was noted to be oriented and able to follow commands. Vital signs range were BP 120/80 to 130/80 mmHg and HR 60-70 bpm. Breath sounds were clear and heart tones were distinct with a regular rhythm. 2D-echocardiogram was delayed due to the queue of patients scheduled. The patient remained closely monitored. On the 3rd post-operative day, the ST-segment elevation was no longer be seen in the ECG monitor.

On the 4th post-operative day, 2D-echocardiogram showed concentric remodeling with adequate wall motion, normal contractility, preserved global systolic function, no doppler evidence of diastolic dysfunction, normal dimension of all chambers and normal pulmonary artery pressure. Troponin levels were ordered but unfortunately no results could be retrieved. The patient remained hemodynamically

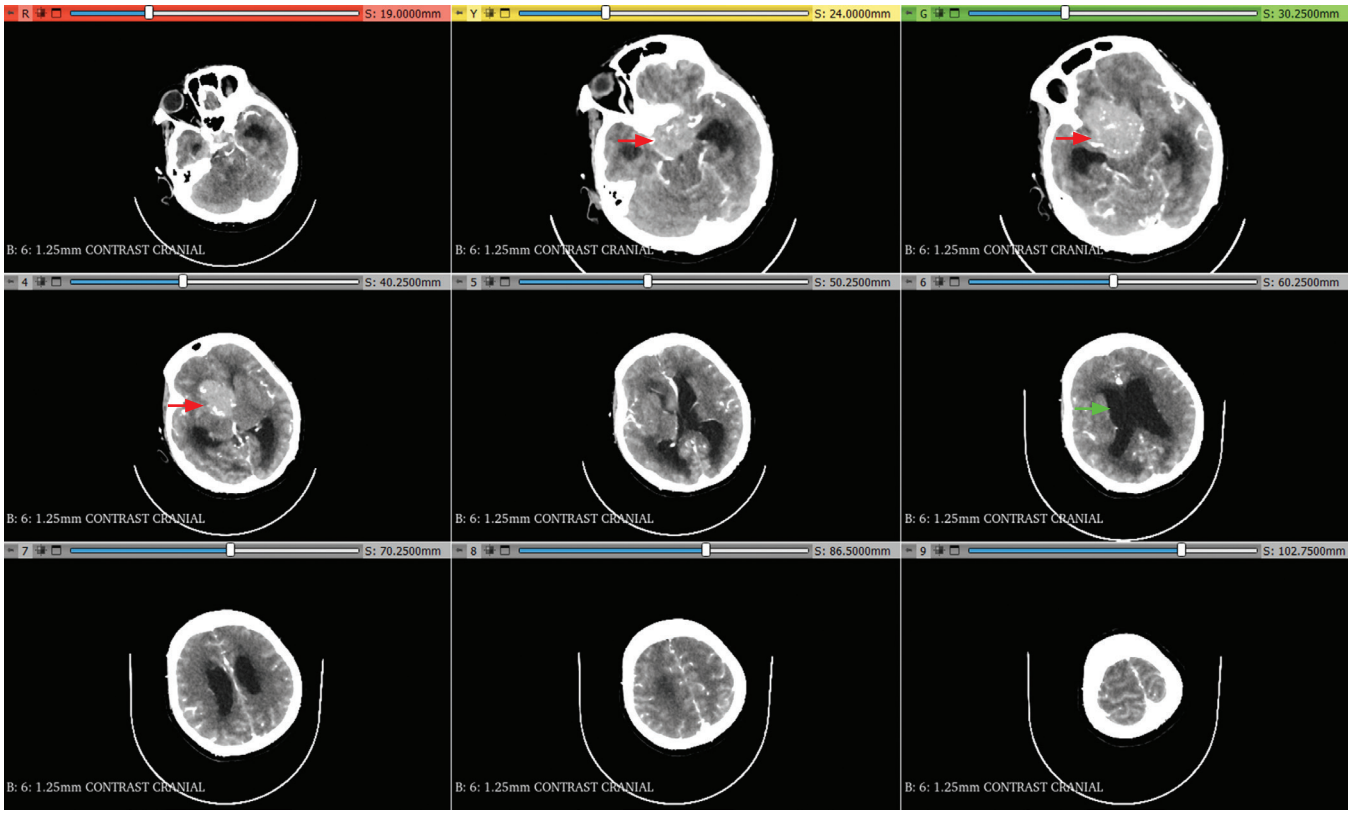

Figure 2. Axial view cranial CT scan with contrast. Sellar-suprasellar mass marked by red arrows. Dilated lateral ventricles due to compression of the 4th ventricle and cerebral aqueduct marked by green arrow.

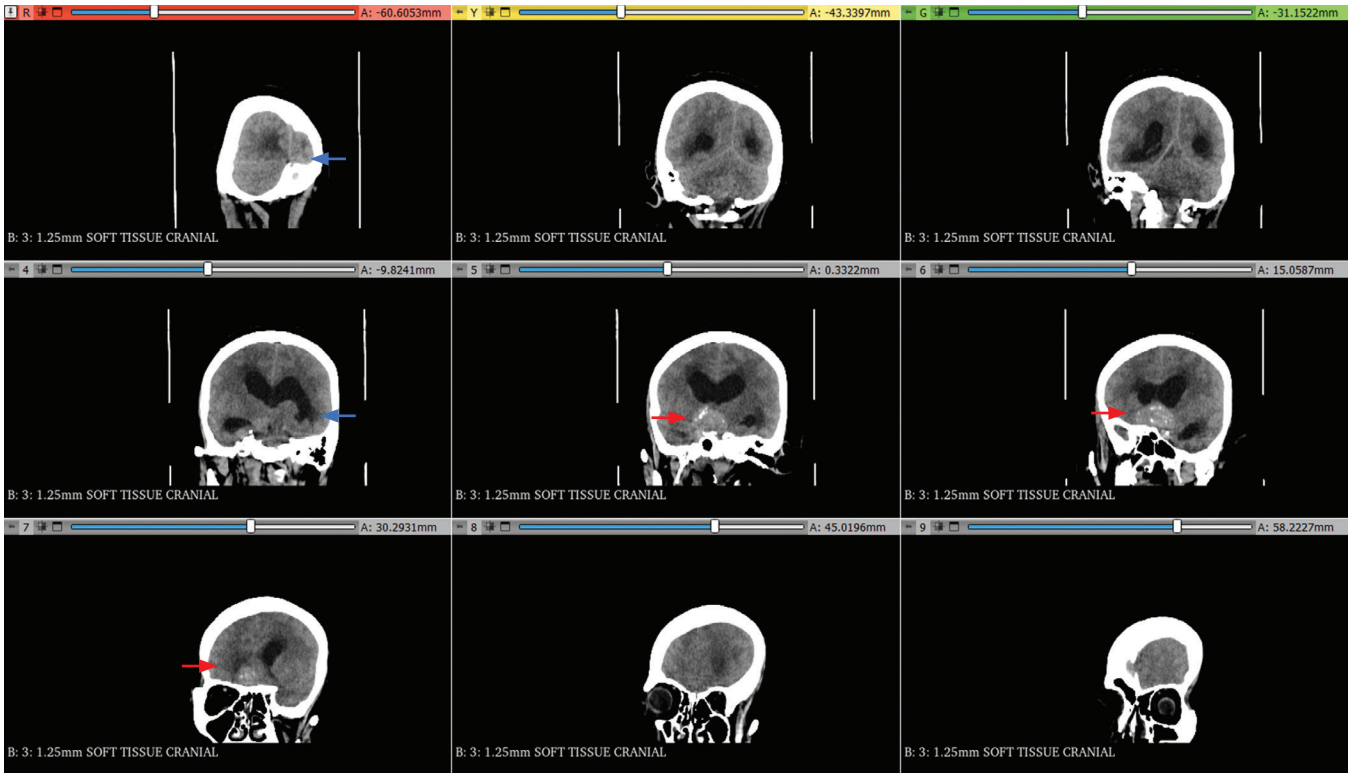

Figure 3. Coronal view cranial CT scan with contrast. Sellar supra-sellar mass marked by red arrows. Possible transtentorial herniation marked by blue arrows.

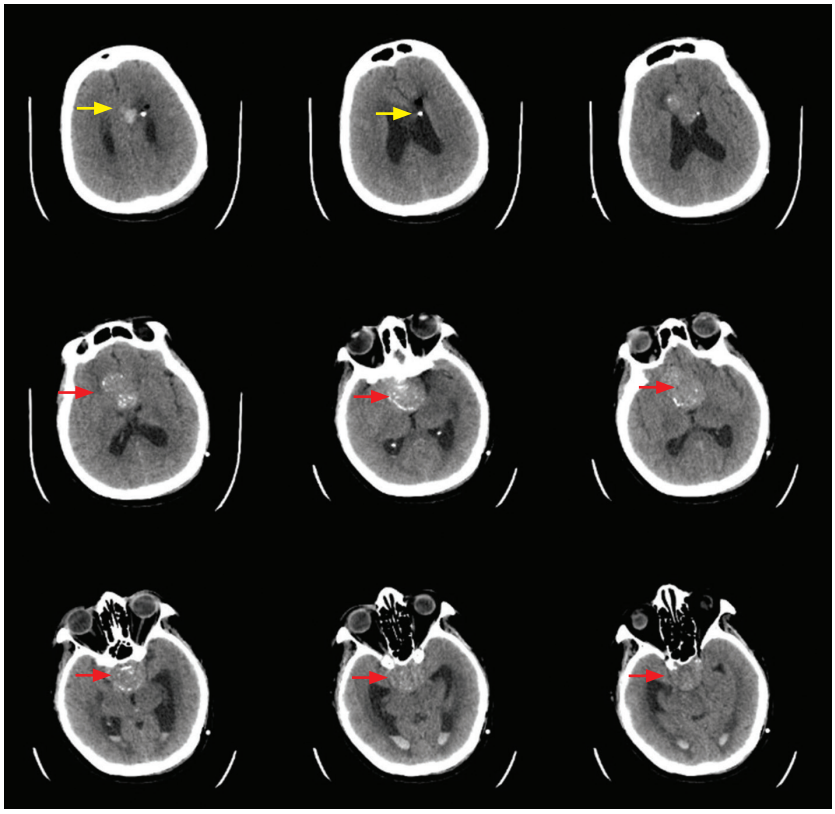

Figure 4. Post-operative transverse CT scan showing a 3.6 x 4.1 x 5.0 cm sellar-suprasellar mass, most likely a craniopharyngioma marked by red arrows. Ventriculoperitoneal shunt seen at the left frontal horn marked by the yellow arrows. Improved findings via a decrease in the dilation of the lateral horns can be noted.

and neurologically stable and was subsequently extubated successfully. There was no reccurrence of cardiac arrythmias.

Although improved, the patient developed right sided weakness (3/5) requiring referral to Rehabilitation medicine. The cardiologist was not able to identify a primary cardiac problem and did not prescribe any medication. The patient did not consent to a craniotomy and excision of intracranial tumor.

On the 10th post-operative day, she was discharged awake, oriented, conversant and able to follow commands. Subsequent follow-up consultations with Neurology service were done on an outpatient basis.

DISCUSSION

NSM is a sudden transient reversible cardiac dysfunction precipitated by severe neurological insult in the setting of a normal cardiovascular system.[1-3] Although it is more commonly associated with subarachnoid hemorrhage, it can also occur in patients with hemorrhagic or ischemic stroke, traumatic brain injury, status epilepticus, CNS infection and acute hydrocephalus.[1-4]

The mechanism of NSM has not been clearly defined. However, the prevailing theory is that a catecholamine surge follows an insult to areas of the brain responsible for the autonomic system, particularly the insula.[5] The insular cortex regulates blood pressure and heart rate through both excitatory and inhibitory baroreceptive neurotransmitters. Stimulation of the left insula can cause vasodepression and bradycardia while ischemia has been shown to produce decreased parasympathetic activity and increased vascular tone. On the other hand, stimulation of the right insula elicits activity that can cause tachycardia and hypertension whereas ischemia results in decreased sympathetic activity. Overall, lesions in the insula have been associated with increased cardiac morbidity and mortality.[6] Myocardial injury can occur from this surge through: 1) coronary vasospasm, 2) uncompensated increase in myocardial demand, leading to ischemia, or 3) excessive stimulation of beta-adrenergic receptors leading to increased calcium channel opening and depleted ATP leading to myocardial cell death.[1,6]

Case reports showing NSM in the setting of acute hydrocephalus has raised the possible role of the location of the lesion and acute increases in intracranial pressure (ICP) in the development of NSM.[4,7-9] Compression of the hypothalamus and elevated ICP stimulate the vasomotor centers leading to autonomic sympathetic discharge resulting to either stunned myocardium with or without concomitant pulmonary edema.[7] This mechanism is postulated to cause sudden cardiac death among patients with 3rd ventricular colloid cyst wherein the proximity of the 3rd ventricle to the hypothalamus either leads to direct mass compressive effect on the hypothalamus or produces complete blockade of CSF flow leading to a cascade of fatal cardiac and respiratory events.[10-12]

The presence of a significant pre-existing increased ICP which cannot be managed adequately medically rendered this patient susceptible to NSM if further increase in ICP occurs. In this case, it was the rapid introduction of irrigation fluid via the neuroendoscope which precipitated the event presenting as marked hypertension, tachycardia and arrhythmia. This increase in ICP could have been further compounded by the dose-dependent vasodilatory effect of sevoflurane which was initially increased in an effort to deepen the anesthetic depth.

NSM has been reported to occur intraoperatively during an endoscopic shunt revision on a 10-year-old with congenital hydrocephalus wherein rapid influx of irrigating fluid with accidental occlusion of the drainage port resulted in marked hypertension (255/155 mmHg) and tachycardia (138 bpm).[13] A review article stated that a sudden increase in ICP due to high-speed irrigation or obstruction of fluid outflow during neuroendoscopic procedures are primary causes for cardiovascular responses including tachycardia and hypertension.[14]

While there are no formal diagnostic criteria for NSM, widely-used indicators include ECG changes (QT prolongation, T-wave inversion and ST-segment changes), decreased cardiac function, elevated troponin levels and ventricular dysfunction.[1,5] As such, it presents similarly with myocardial infarction and can be hard to differentiate. What sets NSM apart is the reversibility of these observed findings if the neurologic event is corrected promptly.[3]

The patient presented with sinus tachycardia which later progressed to supraventricular tachycardia despite interventions to deepen the anesthetic level. It easily reverted back to sinus tachycardia after the endoscope was removed which afforded decrease in ICP by allowing outflow of fluid through the craniotomy site. The goal at that point was to address the underlying neurologic process which was the hydrocephalus-induced intracranial hypertension while at the same time decreasing afterload and maintaining cardiac contractility with careful monitoring to avoid compromising cerebral hemodynamics. Most anesthetic agents have been shown to decrease sympathetic activity which in turn leads to decreased heart rate and arterial blood pressure as well as provides vasodilation.[15,16] Direct acting agents like nitroglycerin and verapamil can be used to provide coronary vasodilation while esmolol has been used to decrease cardiac workload.[17]

Noted new-onset ST elevation did not result in hypotension and resolved spontaneously on the 3rd post-operative day making it compatible with NSM rather than a primary coronary event. Unfortunately, no troponin level test results could be retrieved to document the myocardial injury and the 2D echocardiography could not be performed earlier to document any ventricular dysfunction coinciding with the acute hemodynamic changes.

NSM is reversible and treatment is primarily supportive in nature.[4,7-9,13] However, if not recognized and managed

appropriately, it can be fatal.[18] Patients with underlying acute neurologic injury should have cardiac evaluation upon admission and must be monitored closely as early recognition and management of NSM leads to a favorable outcome.

CONCLUSION

NSM can occur even in minimally invasive neurosurgery. Rapid introduction of irrigation fluid via a neuroendoscope in a patient with a pre-existing significant elevated ICP can precipitate NSM. The surgical team must be immediately informed to rule out a surgical cause of intraoperative sudden significant hemodynamic changes. Management is primarily supportive while simultaneously addressing the neurologic injury.

Declaration of Patient Consent

The authors certify that they have obtained appropriate patient consent forms granting use of the patient's images and clinical information for publication. The patient's authorized representative understands that the patient's name and initials will not be published and due efforts will be made to conceal her identity, but anonymity cannot be guaranteed.

Statement of Authorship

MLM participated in the acquisition of data, drafting and revising both the original and final manuscripts, and approved the final submitted case report; KCS participated in the acquisition of data, revising the original and final manuscripts, and approved the final submitted case report.

Author Disclosure

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