



HHS Public Access

Author manuscript

A A Case Rep. Author manuscript; available in PMC 2017 January 01.

Published in final edited form as:

A A Case Rep. 2016 January 1; 6(1): 3–5. doi:10.1213/XAA.0000000000000207.

Prolonged Cardiac Dysfunction After Intraparenchymal Hemorrhage and Neurogenic Stunned Myocardium

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Abstract

Cardiac dysfunction occurring secondary to neurologic disease, termed neurogenic stunned myocardium, is an incompletely understood phenomenon that has been described after several distinct neurologic processes. We present a case of neurogenic stunned myocardium, discovered intraoperatively after anesthetic induction, in a patient who presented to our operating room with a recent intraparenchymal hemorrhage. We discuss the longitudinal cardiac functional course after neurogenic stunned myocardium. Lastly, we discuss the pathophysiology of neurogenic stunned myocardium, as well as its implications for anesthesiologists caring for neurosurgical patients.

Introduction

Cardiac dysfunction occurring secondary to neurologic injury is incompletely understood. The development of acute heart failure after a neurologic insult has been described most thoroughly after subarachnoid hemorrhage,¹ but has been reported in association with nearly all types of neurologic insults including traumatic brain injury, intraparenchymal hemorrhage, encephalitis, epileptic seizures, electroconvulsive therapy, spinal cord injury, and hydrocephalus.²

Characterization of neurogenic stunned myocardium suggests a pathophysiology that is distinct from typical ischemic heart disease and heart failure.³ Furthermore, time to normalization of cardiac function remains unclear.

We present the clinical course and 1-year functional follow up of a patient diagnosed with pontomedullary junction intraparenchymal hemorrhage, intraoperative hypotension, and

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The authors declare no conflict of interest.

prolonged cardiac dysfunction. We also discuss brain-heart interactions and their importance to the anesthesiologist delivering perioperative care for these patients.

We made multiple attempts to contact the patient but were not successful; following our local protocol, our IRB determined that approval was not required.

Case Description

A 43 year-old man with a medical history of hypertension and methamphetamine use presented to the Emergency Department with an acute onset of emesis, dysarthria, and diffuse paresthesias. Initial head computed tomography scan revealed a 12 x 10 mm focus of intraparenchymal hemorrhage near the midline ponto-medullary junction. At the time of admission, his arterial blood pressure was 210/130 mmHg. A 12-lead electrocardiogram (ECG) revealed a normal sinus rhythm, but was notable for signs of left atrial and left ventricular enlargement, a nonspecific repolarization abnormality, and a prolonged corrected QT interval (QTc = 529 milliseconds). Physical examination revealed clear lung fields, no murmurs or pathologic heart sounds, and no signs of jugular venous distension or peripheral edema. The admission chest radiograph was normal. We initiated pressure control with a nicardipine infusion and seizure prophylaxis with levetiracetam. The patient remained neurologically stable, hemodynamically stable, and was admitted to the Neurocritical Care Unit for further monitoring. Diagnostic cerebral angiogram performed on the first hospital day identified a cavernous malformation in association with the patient's intraparenchymal hemorrhage. Resection of the cavernoma via right frontotemporal craniotomy was planned.

On the day of resection (hospital day 4), repeat magnetic resonance imaging confirmed stable appearance of the intraparenchymal hemorrhage. The nicardipine infusion had been weaned off. Clinical examination revealed no changes in the patient's neurologic symptoms. Preoperatively, the patient's hematocrit was within normal limits, arterial blood pressure was 132/97 mmHg, heart rate was 96 bpm, and oxygen saturation was 99% on room air. The cardiopulmonary physical examination was unremarkable. The patient was premedicated with midazolam 2 mg IV. Standard ASA monitors were placed. Additional neurophysiologic monitoring after induction included multichannel analog electroencephalogram. Sensory, motor, auditory, and cranial nerve VII evoked potential monitors were planned.

Intravascular access included 3 large-bore peripheral IV catheters and a right internal jugular central venous catheter, with an arterial line placed after induction. General anesthesia was induced with IV fentanyl 200 mcg, lidocaine 50 mg, remifentanil 0.05 mg and propofol 150 mg. Neuromuscular blockade was accomplished with rocuronium 50 mg for tracheal intubation, and general anesthesia was maintained with isoflurane < 1 minimum alveolar concentration. The anesthetic plan called for a conversion to total IV anesthesia (with propofol and remifentanil infusions) before the commencement of neuromonitoring.

Within 10 minutes of induction, the patient's arterial blood pressure decreased to 85/55 mmHg, and was refractory to a 200 mg IV bolus dose of phenylephrine. An IV phenylephrine infusion was initiated at a rate of 25 mcg/min, and 800 mL of Plasma-Lyte was infused. Intraoperative laboratory examination revealed a normal hematocrit and stable acid-base status. Over the next hour, 3 additional IV 200 mcg boluses of phenylephrine were

administered, and the phenylephrine infusion rate was increased to 90 mcg/min. The patient remained hypotensive at 70/50 mmHg, prompting the performance of an intraoperative transesophageal echocardiogram. This revealed global hypokinesis of the left ventricle, a qualitative left ventricular ejection fraction of less than 20%, and central mitral regurgitation (Figure 1 and Videos 1-4). A chest radiograph revealed no new significant changes. A norepinephrine infusion at 0.5 mcg/min was initiated for inotropic hemodynamic support, and adequate arterial blood pressure control was achieved. The case was aborted due to the hemodynamic instability.

After normal troponin levels and absence of any new ECG changes from admission (including no new ST segment changes) excluded an acute myocardial infarction, the constellation of echocardiography findings in the setting of a recent intraparenchymal hemorrhage was ascribed to the diagnosis of neurogenic stunned myocardium. Given the relative contraindication of his recent neurologic hemorrhage coupled with the clinical presentation and the patient's relatively young age, the suspicion for ischemic heart disease was low and the cardiology team elected not to perform a cardiac catheterization.

Within 24 hours after postoperative intensive care unit admission, the patient was weaned off vasopressors and his trachea was extubated. A transthoracic echocardiogram was performed 6 days later and showed an ejection fraction of 19%, with no significant improvement compared with the intraoperative study. Therefore, surgery was again delayed, and the patient's heart failure was managed medically with an angiotensin-converting enzyme inhibitor and beta-blockade (with the dose slowly increased after continued confirmation of hemodynamic stability). On hospital day # 14, the patient was discharged in stable neurologic condition to a skilled nursing facility.

A repeat transthoracic echocardiography performed 45 days after the initial surgery attempt demonstrated global hypokinesis and an ejection fraction of 38%, representing sufficient improvement from the prior echocardiogram to clear the patient for a repeat surgical attempt. Resection of the cavernoma was re-attempted 77 days after the original operation. At the time of the induction of general anesthesia with propofol and fentanyl, there was a modest decrease in arterial blood pressure from 140/100 to 97/65, which responded adequately to a phenylephrine infusion of 20 – 40 mcg/min. The phenylephrine infusion was discontinued after 1 hour and 45 minutes, and the arterial blood pressure remained normal for the remainder of the procedure. His postoperative course was complicated by new-onset seizures requiring medical therapy. The patient was discharged on postoperative day 20. The patient had continued follow-up with the cardiology service after being discharged. Transthoracic echocardiography performed 10 months after the initial intraoperative echocardiogram showed improved left ventricular systolic and diastolic function, with an ejection fraction of 47%.

Discussion

Data on the long-term cardiac function after neurogenic stunned myocardium are scarce. Our case adds to the limited literature on the longitudinal cardiac functional course of

neurogenic stunned myocardium. Our case highlights the relevance of brain-heart interactions to the clinical practice of anesthesiology.

Possible causes of intraoperative hypotension in our case included anesthetic-induced hypotension, methamphetamine-associated cardiomyopathy, acute myocardial infarction (MI), and neurogenic stunned myocardium. Given the long-term persistence of cardiac dysfunction after the intraoperative course, we considered anesthetic-induced hypotension an unlikely explanation. The patient's methamphetamine use is an interesting variable to consider, because acute methamphetamine-associated cardiomyopathy is a rare and poorly understood entity.⁴ The patient did not have any pre-hemorrhage diagnostic studies (such as a pre-hemorrhage ECG to establish whether the admission ECG findings were acute or chronic). However, evidence against methamphetamine-associated cardiomyopathy included the lack of any heart failure symptoms before hospitalization and the lack of improvement in cardiac function after methamphetamine withdrawal.

The absence of elevated cardiac biomarkers or new-onset ECG changes argues against an acute MI explaining the patient's clinical picture. The regional wall motion abnormalities observed in our patient's echocardiograms were global, and not attributable to a single coronary artery territory. This pattern is consistent with observations in neurogenic stunned myocardium, where the pattern of regional wall motion abnormality extends beyond the distribution of a single epicardial vessel.⁵ In a study comparing patients with cardiac dysfunction after subarachnoid hemorrhage to historic myocardial infarction controls with similar left ventricular dysfunction, it was determined that an ejection fraction of <40% and cardiac troponin I <2.8 ng/ml are more consistent with neurogenic stunned myocardium rather than acute MI.⁶

Cardiac necrosis in neurogenic stunned myocardium mirrors the distribution of adrenergic receptors in the myocardium, and has been shown to be most severe near nerve terminals in the endocardium with less severity in the epicardium.^{7,8} Correlation with adrenergic receptor distribution fits with the widely accepted hypothesis that catecholamine-mediated direct myocardial injury underlies the pathophysiology of neurogenic stunned myocardium. Furthermore, basic science studies have revealed cardiopulmonary dysfunction after induced lesions throughout the brain and including the brainstem,⁹ similar to the anatomic region of injury in our patient. Briefly, neurologic injury triggers a stress response that leads to elevated catecholamine levels both systemically via the hypothalamic-pituitary-adrenal axis and directly through norepinephrine release through nerve terminals at the myocardial endplate. Excessive prolonged activation of beta-1-adrenergic receptors leads to cyclic adenosine monophosphate-mediated calcium overload and rapid depletion of adenosine triphosphate. This sequence leads to the classic finding of contraction-band necrosis, characterized by hypercontracted sarcomeres, interstitial mononuclear infiltrate, and irregular cross-band formations.¹⁰

The temporal pattern of cardiac dysfunction in this case is particularly noteworthy since the patient had no history of cardiac symptoms before hospitalization. Severe cardiac dysfunction persisted at least 6 days after it was initially diagnosed intraoperatively. Significant but incomplete recovery of systolic function was observed at 6 weeks. Further

improvement of cardiac function with a suboptimal ejection fraction of 47% was seen at 10 months. Complete recovery of ventricular function in Takotsubo cardiomyopathy typically occurs over a 1 to 3 month period,¹¹ and among 48 patients with subarachnoid hemorrhage-induced cardiac dysfunction, 25% experienced complete normalization within 8 days and an additional 41% showed at least partial recovery². In our patient, there was a definite trend of continued improvement in systolic and contractile left ventricular function up to 10 months after the initial insult, which is longer than previously reported.

In summary, the central event in our case was refractory hypotension after induction of anesthesia in a patient with an intraparenchymal hemorrhage, with clinical and echocardiographic findings consistent with a diagnosis of neurogenic stunned myocardium. Longitudinal transthoracic echocardiography revealed a slow improvement in cardiac function over the year after diagnosis. Anesthesiologists should be aware of the phenomenon of neurogenic stunned myocardium and be prepared to identify and treat refractory hypotension during anesthesia. Further study is necessary to characterize the clinical course and risk factors for neurogenic stunned myocardium after intraparenchymal hemorrhage, and to determine the role of echocardiography in detecting subclinical cardiac dysfunction after neurologic injury.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments

Funding: NIH National Research Service Award T32 GM086270

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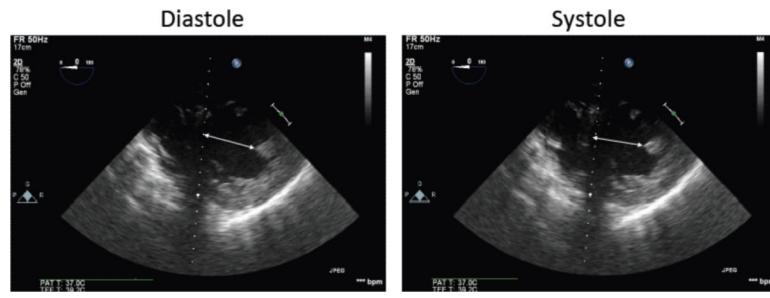


Figure 1.

Intraoperative transesophageal echocardiogram (transgastric, short-axis, midpapillary view) demonstrating severely diminished left ventricular contractile function.