Emergent craniotomy for SAH with cerebral-induced cardiac ischemia
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Case Presentation

55 year old previously healthy female with a large SAH (HH 4) presented emergently to the OR for aneurysmal clipping for three cerebral aneurysms. In the ICU she had no hemodynamic instability but her EKG had changed from NSR to cerebral T wave abnormalities. Close attention to the patient's hemodynamics and carefully emergently to the OR for aneurysm al clipping for three cerebral aneurysms. Intracranial pressure (ICP) and intracranial hemodynamics were utilized. Cranial pinning was done with boluses of nor epinephrine and propofol boluses ensured hemodynamically was maintained at approximately 60mmHg. Mannitol 75g was given at ICU she had no hemodynamic instability but her EKG had changed from NSR to cerebral T wave abnormalities. Ischemic changes such as ST and T wave changes correlate with the male gender and worse one year functional outcome.

Transcranial Doppler the next day showed moderate to severe left MCA vasospasm. Troponins continued to trend down and T wave inversions. Cardiac ischemia is well described in patients with SAH. Ischemic changes to the patient ECG as well as their cardiac function. These ischemic ECG and functional changes are not typically explained by underlying coronary disease or thrombosis.

Abnormalities in ECG tracings of rhythm and morphology are seen in 50-80% of patients with SAH. They usually appear within 48 hours and normalize in 6 weeks. The most common ECG changes are repolarization abnormalities and changes of in the T wave morphology and ST segment. The large upright T waves "cerebral T waves" are well described in patients with SAH. Ischemic changes such as ST and T wave changes correlate with the male gender and worse one year functional outcome.

Prolonged QTC is frequently seen and has been shown to correlate with the female gender and use of propofol. Cardiac dysrhythmias may also occur and can lead to life threatening dysrhythmias usually during the first 48 hours after SAH. Troponin elevation is also frequently seen and is related to the severity of the intracranial hemorrhage. Patients with elevated troponin I concentrations are more likely to have ECG abnormalities and clinical evidence of left ventricular dysfunction.

The sub endocardial injury can manifest itself as a stress cardiomyopathy with cardiomyopathy appearance. This ventricular dysfunction is seen in up to 33% of patients after SAH is associated with pulmonary edema, intra atrial thrombus and embolic stroke.

Discussion

Anesthetic management

Care of the patient with cerebral induced cardiac ischemia starts when possible, with pre-operative evaluation of the patient’s baseline medical history and cardiac history. Review of the patient’s ECG pre and post SAH will allow for additional preparation for possible arrhythmias and changes that may be seen intraoperatively.

Transcranial Doppler can provide valuable information on the patient’s systolic function and possible need for inotropic support intraoperatively. Discussion with the neurosurgical team about blood pressure goals and fluid status in context of the patient with ischemic cardiomyopathy help plan an appropriate anesthetic. Any decision to postpone surgery for further cardiac evaluation must be balanced against the risk of re-bleeding and vasospasm. Most of these patients will not benefit from cardiac interrogation in the Cath lab as the cause of their ischemic changes is not driven by coronary atherosclerosis.

Intraoperatively all of the principles of neuro anesthesia remain unchanged. Invasive arterial monitoring is essential for CO2 management and meticulous blood pressure control. IV access capable of rapid transfusion is necessary to manage possible intraoperative rupture. Patients with cardiac dysrhythmias, in addition to cerebrally induced cardiomyopathies require additional attention to monitor for possible malignant arrhythmias. Vasoactive medications may be required and antiarrhythmic should be available. Post operatively the use of beta blockade for cardio protection given the catecholamine induced nature of the cardiac dysfunction can be considered. However patients’ with SAH may require vasopressors and possible inotropes for hypotension and cardiac failure and beta blockers may need to be withheld.

In summary understanding the pathophysiology of cerebral induced cardiac ischemia in patients with SAH will assist the anesthesiologist in optimal intraoperative care.

References:


