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Case Report

Awake craniotomy in a patient with ejection fraction of 10%: considerations of cerebrovascular and cardiovascular physiology☆,☆☆

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Abstract A 37-year-old man with nonischemic 4-chamber dilated cardiomyopathy and low-output cardiac failure (estimated ejection fraction of 10%) underwent awake craniotomy for a low-grade oligodendroglioma resection under monitored anesthesia care. The cerebrovascular and cardiovascular physiologic challenges and our management of this patient are discussed.
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1. Introduction

Normal brain function depends upon the maintenance of adequate cerebral blood flow (CBF), which is tightly regulated by various mechanisms [1]. Each organ's perfusion, including the brain's, is a portion of cardiac output (CO) [2]. When CO is decreased, the redistribution of blood flow between various organs determines the adequacy of CBF [3-6]. Moreover, brain circulation is propelled by cerebral perfusion pressure. In a patient with low-output cardiac failure, both low CO and low blood pressure compromise brain perfusion; there is little room for error in clinical care. During awake craniotomy, one of the anesthesiologist's goals is to ensure a cognitively intact patient for neurophysiologic testing, which requires adequate CBF. It is thus instructive to examine a case, where a patient with low-output cardiac failure and an estimated ejection fraction (EF) of 10% underwent an awake craniotomy for brain tumor resection. We present considerations of cerebrovascular and cardiovascular physiology, as they apply to the management of this case.

2. Case report

A 37-year-old right handed man complained of dizziness and then "passed out" with concurrent right upper extremity tonic-clonic movements. He was found to have a wide

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complex ventricular tachycardia for which he was successfully cardioverted. Brain magnetic resonance imaging showed a large left insular mass; he was transferred to our hospital and scheduled for an awake craniotomy. His medical history was significant for a New York Heart Association class II-III nonischemic 4-chamber dilated cardiomyopathy likely due to methamphetamine abuse. Transthoracic echocardiography estimated left ventricular EF of 10% and noted severe mitral regurgitation. He was in sinus rhythm with first degree atrioventricular block and frequent premature ventricular contractions since hospital admission. His medications on transfer included furosemide, digoxin, carvedilol, lisinopril, and levetiracetam. Our cardiology consultant described him as being "at the end of the curve of his cardiac disease" and recommended automatic implantable cardioverter defibrillator placement as well as evaluation for heart transplantation after craniotomy.

In the preoperative area, his hands and feet were cold, clammy, and blue tinged. After informed consent, the patient was brought to the operating room, without automatic implantable cardioverter defibrillator placement, for a left frontotemporal craniotomy for resection of insular glioma with intraoperative electrocorticography and awake speech and motor mapping under monitored anesthesia care. External defibrillation pads were placed in addition to routine anesthesia monitors. Right radial arterial catheter and left subclavian double-lumen central venous catheter were placed with minimal sedation. The patient was then positioned in a right lateral position with the head in Mayfield pins for surgery. Supplemental oxygen was administered via nasal cannula. Sedation was adjusted according to surgical stages and accomplished with midazolam boluses (7 mg in divided doses), fentanyl boluses (50 μg), remifentanil infusion (0-0.05 μg/kg per minute), and propofol infusion (0-25 μg/kg per minute). Dobutamine infusion (2-8 μg/kg per minute) was started at the same time as sedation and was adjusted based on arterial blood pressure reading. The goal was to maintain his baseline blood pressure (95/63 mm Hg). Slow transfusion of packed red blood cells diluted with normal saline was started after skin incision. The transfusion rate was adjusted based on the estimated ongoing blood loss with the goal of maintaining hemoglobin level concentration no lower than 80% of his baseline value (17.6 g/dL). The rationale for this strategy will be explained in the discussion. The patient received 50-g intravenous mannitol. Intravascular volume replacement was managed with the goal of maintaining euvolemia. Serial arterial blood gas analysis was performed through the procedure and demonstrated minimal deviation from baseline. Throughout the case, the hemoglobin level concentration was maintained between 15.5 and 17.2 g/dL, base excess between 1 and 3 mEq/L, and lactate between 0.7 and 0.8 mmol/L. Other parameters included in blood gas analysis were unremarkable. During the early stages of the procedure, the pulse oximeter signal was consistently poor at various locations; however, this improved after the start of dobutamine infusion, volume expansion, and packed red blood cell transfusion (Figure). The patient's neurocognitive function remained intact during awake testing. The case lasted 5 hours and concluded without noticeable complications. He received a total 400-mL packed red blood cells, 500-mL 5% albumin, and 1750-mL crystalloid. The estimated blood loss was 800 mL, and urine output was 650 mL. The patient was transported to neurointensive care unit with intact neurocognitive function and stable vital signs. The final pathologic diagnosis was grade 2 oligodendroglioma. He was discharged home on postoperative day 3.

3. Discussion

We present a case in which a patient with low-output cardiac failure underwent craniotomy for tumor resection under monitored anesthesia care. Awake craniotomy is performed when surgical resection is planned near critical motor, somatosensory, or speech areas in an attempt to maximize the resection while minimizing postoperative neurologic deficits. Overall anesthetic considerations during awake craniotomy have been discussed elsewhere [7-9]. In the "typical" awake craniotomy, the major anesthetic concerns revolve around the provision of adequate anesthesia, analgesia, and anxiolysis for the beginning of the case (ie, opening) while then allowing an awake and cooperative patient during neurophysiologic testing. However, in "any" case involving severe low-output cardiac failure, hemodynamic stability and perhaps even intraoperative survival become pressing concerns. The unique challenge of this case is the maintenance of adequate cerebral perfusion and oxygenation via proper management of both cerebrovascular and cardiovascular physiology.

3.1. Cardiac output distribution to the brain: effects of failure

To understand the management of this patient, it is useful to review how CO is normally distributed and how that distribution might be altered in this patient. All organ perfusion is a portion of CO with different organs receiving different proportions of CO. Cerebral blood flow is usually approximately 12% of CO [2]. How might that distribution change in a patient with low-output cardiac failure?

Although clinicians often use EF as a shorthand summation of overall cardiac function, there is no simple relationship between EF and CO. However, based on data from the patient's echocardiogram, some estimates can be made. Left ventricular end diastolic and end systolic volumes were estimated at 346 and 310 mL, respectively, giving a stroke volume (SV) of 36 mL. The value for normal SV is often given as 70-75 mL [10]. Thus, despite an EF of 10% because of the severe chamber dilation, the patient's SV is 50% of normal. His resting heart rate in the 90s, likely a