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Management of Acute Elevation of Intracranial Pressure during Hepatic Transplantation

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This report concerns a patient in Stage 4 hepatic coma, according to the criteria described by Brems *et al.*,[1] who presents for liver transplantation. The major reported cause of mortality associated with the management of patients in Stage 4 hepatic coma is increased intracranial pressure (ICP) as a result of cerebral edema.[2] Direct ICP monitoring, while still controversial, is recommended by some authors to more effectively manage changes in ICP.[3–5] Perioperative use of direct ICP monitoring with the ability to drain cerebrospinal fluid (CSF) may reduce the morbidity and mortality rates associated with unrecognized prolonged elevation of ICP. The following case report illustrates the successful management of one such patient.

REPORT OF A CASE

The patient is a 28-yr-old woman with a 10-yr history of chronic active hepatitis of autoimmune origin. She had a history of multiple esophageal and gastric variceal bleeding episodes. A portacaval shunt was constructed 1 week prior to the liver transplant using the right internal jugular vein. The patient became encephalopathic 3 days after placement of the shunt and was transferred to our institution for emergency liver transplantation. The patient was comatose, her trachea intubated, and at first would respond minimally to deep noxious stimuli (Stage 3). The patient was subsequently classified as Stage 4 when she no longer showed any response to noxious stimuli. Computerized tomography demonstrated cerebral swelling without intracranial hemorrhage. Electroencephalography revealed high amplitude delta waves with no focal or lateralizing alterations. Following neurologic and neurosurgical evaluation, the decision was made to place an ICP monitor. The coagulopathy was corrected by administration of 4 units of fresh-frozen plasma, and then a ventriculostomy catheter was inserted into a lateral ventricle and connected to a closed, sterile monitoring system. This is a relatively simple apparatus that directly measures intraventricular pressure and also allows CSF to be drained quantitatively from the ventricles without violating the integrity of the system. A radial artery line and a fiberoptic flow-directed pulmonary artery catheter were also placed at that time. The patient was returned to the intensive care unit (ICU) where hyperventilation, optimal head positioning, iv mannitol, and steroids were used in an attempt to maintain the ICP at less than 15 mmHg. Dialysis or plasmapheresis was not used. The patient had no signs of renal impairment during this hospitalization. If, despite these measures, the ICP exceeded 15 mmHg, an appropriate amount of CSF was drained from the ventriculostomy catheter. The patient was returned to the operating room for liver transplantation 8 h following the insertion of the ventriculostomy catheter.

The patient arrived in the operating room without any apparent change in neurologic status. Anesthesia was induced and maintained with iv sufentanil, isoflurane in 50% air/oxygen mixture, and iv pancuronium. Continuous monitoring included ECG (leads II and V_5), radial artery pressure, pulmonary artery pressure and oxygen saturation and temperature, central venous pressure, ICP, and inspired and exhaled respiratory gases. The patient was hemodynamically stable, but despite the use of hyperventilation, steroids, mannitol, and diuretics, still required intermittent drainage of CSF throughout the surgery to maintain the ICP between 7 and 15 mmHg. Approximately 4 h after the beginning of the operation, venovenous bypass was instituted.

Upon completion of vascular anastomoses to the newly transplanted liver, blood flow was reestablished by unclamping the vena cava and the portal vein. Following revascularization of the liver, the patient became hemodynamically unstable. The mean systemic pressure decreased to 45 mmHg and was treated by intravascular fluid replacement, calcium chloride, and iv vasopressors (epinephrine). Several minutes thereafter the ICP increased from 7 to 24 mmHg. Immediately 10 ml of CSF was drained *via* the ventriculostomy catheter. With drainage of CSF the ICP fell to 12 mmHg and gradually stabilized at 7 mmHg. The ICP was maintained in the range of 7–12 mmHg for the remainder of the procedure by the standard measures of treating intracranial hypertension mentioned above and by drainage of CSF as needed. The cerebral perfusion pressure decreased below 40 mmHg on two occasions, both occurring after reperfusion of the new liver: when the ICP rose to 24 mmHg and when the arterial blood pressure fell to 45 mmHg.

The case proceeded uneventfully, and the patient was taken to the ICU at the end of the procedure. She began to respond appropriately 18 h after arrival in the ICU, and her trachea was extubated on the second postoperative day. She was alert and oriented with no apparent neurologic deficits. The ICP monitor was removed 48 h following extubation. She continued to do well and left the hospital 8 weeks from the date of her transplant.

DISCUSSION

The anesthetic management of a patient undergoing hepatic transplantation may present a formidable challenge. There have been numerous reports that have outlined the problems encountered during this procedure.[6,7] Most patients present with major hematologic, hemodynamic, and metabolic abnormalities. Extensive monitoring

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of these patients intraoperatively is essential. This case report deals specifically with one type of patient presenting for liver transplantation: the patient in Stage 4 coma who is usually in fulminant hepatic failure.

The etiology of the encephalopathy and coma in these patients has been attributed to the accumulation of toxic substances in the circulation that may act by damaging the blood brain barrier and impairing neuronal function.[3] Some of these substances include ammonia, fatty acids, and phenols. Many investigators propose that a principal cause of death in patients with fulminant hepatic failure is cerebral edema and increased ICP.[8] One review of 32 patients who died of hepatic necrosis found a 50% incidence of cerebral edema at autopsy.[9]

Direct monitoring of ICP should permit earlier detection of abnormal ICP and enable treatment to be started at a potentially reversible stage. This is particularly important in liver transplantation because these patients may be more susceptible to sudden changes in ICP because of frequent changes in fluid balance, venous pressures, and overall hemodynamic instability.[10] The presence of a potentially damaged blood brain barrier also makes these patients more prone to changes in ICP. The goal of treatment is the maintenance of cerebral perfusion pressure above 40 mmHg as computed from the mean arterial pressure, CVP, and ICP.[2]

The treatment of elevated ICP is varied and for the most part only a temporizing measure. Treatment can include the use of steroids, hyperventilation, osmotic diuretics, lactulose to reduce ammonia production, and plasmapheresis or dialysis to remove toxic substances from the circulation.[3] Steroids may be effective in treating focal brain edema; however, their effectiveness in generalized cerebral edema is less certain.[11] Hyperventilation reduces cerebral blood flow and ICP only transiently.[3] Early and rapid administration of mannitol has been shown to decrease ICP. This form of therapy must be used with caution in patients with renal impairment.[3] Mannitol may also cause a paradoxical elevation in ICP.[11] Barbiturates have been advocated in cases of elevated ICP. Care must be used when administering barbiturates to liver transplant patients where the potential for hemodynamic instability is present. The anesthetic agents of choice for patients with elevated ICP are varied and may include thiopental, narcotics, benzodiazepines, muscle relaxants such as pancuronium, and an inhalational agent such as isoflurane. There is however no one single optimum approach, and the anesthetic agents and anesthetic technique used must be tailored to each individual patient.[12]

Placement of an ICP monitor is an invasive procedure that requires expert surgical technique, especially in these patients who may have a significant coagulopathy. All patients should therefore be given blood component therapy as needed, based on coagulation studies, prior to insertion of the ICP monitor. There are a variety of different techniques that may be employed to estimate ICP including subdural, extradural, and intraventricular monitoring systems. The intraventricular catheter, which is the most invasive of these techniques, is the best measure of ICP and at the same time permits drainage of CSF when conventional therapy is not effective. The other techniques, although less invasive and with lower risks of bleeding, are not as accurate and do not allow for drainage of CSF. The catheter system described in this article allows drainage of CSF in a sterile and closed fashion. There are certain risks involved in inserting an ICP monitor in these patients including bleeding and infection. Correction of the coagulopathy, broad spectrum antibiotic coverage, as well as continued use of available standard therapies to decrease ICP such as hyperventilation, steroids, mannitol, head positioning, and barbiturates, prior to insertion of the monitor would decrease the risks associated with this procedure.

In this case report we describe a situation that has not been presented previously. Upon reperfusion of the newly transplanted liver the patient's ICP rose from 7 to 24 mmHg within 35 s. The patient was already being hyperventilated and had received steroids, mannitol, and diuretics. The only immediately effective treatment was to drain the CSF *via* the ventriculostomy catheter. This resulted in a rapid decrease of the ICP to baseline levels. Hemodynamic instability commonly occurs at the time of reperfusion. This together with the effects of the requisite therapy (neosynephrine, calcium chloride, epinephrine, and rapid blood administration) may result in dramatic increases in ICP in those patients with cerebral edema and decreased intracranial compliance.

The potential risk of coning or of even more severe cerebral edema in this setting is great. It is for this reason that the use of a ventriculostomy catheter to monitor ICP, and particularly to drain CSF, may offer a significant addition to the perioperative management of liver transplant patients in Stage 4 hepatic coma, who also have evidence of cerebral edema and raised intracranial pressure. The goal of this therapy is to reduce the morbidity and mortality rates associated with increased ICP in this patient population.

References

1. Brems JJ, Hiatt JR, Ramming KP, Quinones-Baldrich WJ, Busutti RN: Fulminant hepatic failure: The role of liver transplantation as primary therapy. Am J Surg 154:137–140, 1987
2. Corall I, Williams R: Management of liver failure. Br J Anaesth 58:234–245, 1986
3. Ede RJ, Williams R: Hepatic encephalopathy and cerebral edema. Semin Liver Dis 6:107–118, 1986
4. Bowers SA, Marshall LF: Outcome in 200 consecutive cases of severe head injury treated in San Diego County: A prospective analysis. Neurosurgery 6:237–242, 1980

5. Stuart GG, Merry GS, Smith JA, Yelland DN: Severe head injury managed without intracranial pressure monitoring. J Neurosurg 59:601–605, 1983

6. Carmichael FJ, Lindop MJ, Forman JV: Anesthesia for hepatic transplantation: Cardiovascular and metabolic alterations and their management. Anesth Analg 64:108–116, 1985

7. Kang YG, Gelman S: Anesthesia for liver transplantation, Anesthesia for Organ Transplantation. Edited by Gelman S. Philadelphia, W. B. Saunders, 1987, pp 139–185

8. Hanid MA, Davies M, Mellon PJ, Silk DBA, Strunin L, McCabe JJ, Williams R: Clinical monitoring of intracranial pressure in fulminant hepatic necrosis. Gut 21:866–869, 1980

9. Ware AJ, D'Agostino AN, Combes B: Cerebral edema: A major complication of massive hepatic necrosis. Gastroenterology 61: 877–884, 1971

10. Paulsen AW, Valek TR, Blessing WS, Johnson DD, Parks RI, Pyron JT, Ramsay MA, Simpson BR, Swygert TH, Walling PT, Klintmalm G: Hemodynamics during liver transplantation with veno-venous bypass. Transplant Proc 19:2417–2419, 1987

11. Canalese J, Gimson AES, Davis C, Mellon PJ, Davis M, Williams R: Controlled trial of dexamethasone and mannitol for the cerebral edema of fulminant hepatic failure. Gut 23:625–629, 1982

12. Shapiro HM: Anesthesia, Neurosurgical Anesthesia and Intracranial Hypertension. Edited by Miller RD. New York, Churchill Livingstone, 1986, pp 1592–1593

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Treatment of Isorhythmic A-V Dissociation during General Anesthesia with Propranolol

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Isorhythmic A-V dissociation is a common cardiac dysrhythmia during anesthesia that usually occurs in patients anesthetized with one of the potent volatile anesthetics.[1–3] With continuous monitoring of the electrocardiogram, the P-R interval is observed to gradually shorten as the P wave approaches the QRS complex and then disappears within it. The QRS morphology does not change, and the SA node and AV node continue to depolarize independently of each other but at similar rates.[4,5] The P wave usually remains hidden within the QRS complex giving the appearance of an A-V junctional rhythm, but it may be observed to immediately follow it or to move in and out of it. When observed the P wave morphology is not altered, unlike in true A-V junctional rhythms where the atria are depolarized from the A-V node, usually causing inverted P waves in leads II, III, and AVF.[6]

Isorhythmic A-V dissociation is usually tolerated without hemodynamic compromise and its diagnosis is frequently missed. In healthy patients anesthetized with halothane, isorhythmic A-V dissociation has been found to cause a 17% decrease in systolic blood pressure[7] and a 14% decrease in mean arterial blood pressure[8] with a relatively constant heart rate. Isorhythmic A-V dissociation can, however, occasionally result in a significant decrease in arterial blood pressure.[9] In patients with compromised cardiac function the loss of the normal atrial contribution to left ventricular filling can significantly reduce cardiac output.

Decreasing the inhaled anesthetic concentration can convert isorhythmic A-V dissociation to sinus rhythm.[7] Previous clinical reports have demonstrated that both atropine[9] and succinylcholine[10] may be effective treatments in some cases of A-V junctional rhythm during anesthesia. The incidence of A-V junctional rhythms in patients anesthetized with fentanyl and nitrous oxide was reduced by pretreatment with practolol.[11] Intravenous propranolol was reported to successfully convert an apparent A-V junctional rhythm to a sinus rhythm in one patient.[12] Some of these rhythm disturbances were probably isorthymic A-V dissociation.[3,13]

Four cases of patients who developed isorthythmic A-V dissociation during cardiac surgery are presented. In each case the P wave was observed to gradually approach the QRS complex and then disappear within it. The administration of propranolol was successful in treating each episode.

CASE REPORTS

Case 1. A 74-yr-old man with class IV angina, a history of palpitations, a previous myocardial infarction (MI), and a traumatic right lower leg amputation was admitted to the hospital for evaluation of his angina. His electrocardiogram (ECG) showed a normal sinus rhythm at a rate of 75 beats/min and an old inferior MI. Cardiac catheterization revealed significant three vessel coronary artery disease, an ejection fraction of 55%, and aortic stenosis with a pressure gradient of 40 mmHg across the aortic valve. His preoperative medications, including 30 mg of diltiazem and 20 mg of isorbid dinitrate every 6 h, were continued to the time of surgery. He was scheduled for coronary revascularization

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