Acute Open Brain Herniation During Elective Tumour Resection

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Citation

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Abstract

We report a case of a 35-year-old female patient undergoing elective surgery for excision of a frontal meningioma. Intraoperatively, the patient had an acute brain swelling after a near total excision of the tumour. All possible measures were taken to reduce the swelling. All possible causes of this complication were considered and ruled out. The management of the case is discussed.

INTRODUCTION

Malignant brain edema is not an uncommon occurrence in the neurosurgical operating room and can turn a meticulous operation into a life-threatening emergency. Open brain herniation is more commonly seen during intracranial procedures being conducted for head injury, as compared to elective neurosurgical operations. In the case of the latter, brain edema may be due to subarachnoid haemorrhage (SAH) secondary to aneurysm rupture or intraventricular haemorrhage during resection of brain tumors[1]. The brain may suddenly swell uncontrollably and inexplicably, so much so, that the surgical procedure may have to be abandoned due to lack of access to the intracranial operating site. This can result in devastating consequences, with permanent postoperative neurological sequelae. We encountered acute intraoperative brain swelling in a patient undergoing elective surgery for excision of a frontal meningioma. The possible cause and management is discussed.

CASE HISTORY

A 35-year-old female, weighing 105 kilograms was admitted to our hospital with the presenting complaint of one episode of generalised seizures. Her medical history was remarkable for poliomyelitis in childhood, which had produced left lower limb weakness. All routine preoperative investigations were within normal limits. Magnetic resonance imaging revealed a non-homogenous hyperintense lesion in the left frontal cortex, enhancing irregularly with contrast, which was associated with peri-lesional edema. Based on these findings, a diagnosis of left frontal glioma was made. An elective frontal craniotomy and tumor decompression was planned. Inside the operation theatre, continuous monitoring of electrocardiograph, non-invasive blood pressure and pulse oximetry were instituted (Philips Intellivue~Anesthesia). General anaesthesia was induced with fentanyl 100 mcg and thiopentone sodium 300 mg. Vecuronium 8 mg was given to facilitate tracheal intubation with cuffed orotracheal tube. Anaesthesia was maintained with O2, N2O (40:60), isoflurane, fentanyl and vecuronium. Intraoperative monitoring also included heart rate, intra-arterial blood pressure, CVP, end-tidal carbon dioxide, peak airway pressure, urine output, and temperature. The position of the patient was supine with a head-up tilt of about 15 degrees. The head was placed in the neutral position.

After craniotomy, the lesion was found to be just below the surface, moderately vascular and with a well-defined margin of cleavage. A near total excision of the tumor was completed within one hour of skin incision. Suddenly the surgeons noticed extrusion of the brain beyond the craniotomy margins. The anaesthesiologists were informed. The position of the head was checked to rule out neck vein compression. Measures to reduce brain edema were taken which included thiopentone 100 mg, mannitol 140 gm, methylprednisolone 2 gm, and hyperventilation to reduce the end-tidal carbon dioxide from 30 mmHg to 26 mmHg. Inhalational anaesthetic and nitrous oxide were discontinued and total intravenous anaesthesia was started using propofol infusion @ 50 mcg/ kg/min. Meanwhile the surgeons failed to locate any actively bleeding site, which was confirmed by intraoperative ultrasonography. A single lumen central

venous line was then placed in the right basilic vein to monitor the central venous pressure, which was found to be 10 mmHg. Despite the above measures, there was only a minimal reduction in brain swelling. It was then decided to de-bulk the brain and partial frontal lobectomy was done. Throughout the surgery, the central venous pressure was maintained between 8 and 10 mmHg. The total fluid input and urine output were 3 litres and 2.5 litres respectively. The blood loss during the surgery was 700 ml approximately. At the end of surgery, neuro-muscular block was not reversed and patient shifted for computerized Tomography. The scan revealed diffuse subarachnoid bleed involving the supratentorial compartment with mild ventriculomegaly. The patient was then transferred to the neurosurgical ICU and put on ventilatory support. Four hours later, after spontaneous neuromuscular reversal, the trachea was extubated. At that time, although higher mental functions were normal, a left hemiparesis was present which persisted till the time of discharge. A CT – angiography done on the 2 nd postoperative day revealed diffuse subarachnoid bleed involving the supratentorial compartment. There was no evidence of obvious aneurysm or vascular malformation. On histopathological examination, the tumour was found to be an atypical meningioma. A digital subtraction angiography could not be carried out, as consent was denied.

DISCUSSION

The exact mechanism for the occurrence of acute and overt intraoperative brain herniation during elective neurosurgery is not known. A combination of acute intracranial hypertension caused by either subarachnoid or intraventricular haemorrhage together with hyperaemia, rather than brain edema, has been proposed as the primary cause. Cerebral hyperaemia may be the result of either the subarachnoid or intraventricular haemorrhage bolus into the CSF space or indirectly by the SAH activating various neurovascular reflex mechanisms[1,2,3]. It is postulated that excitation of medullary or pontine nuclei, perhaps due to brainstem microcirculatory disturbances caused by the SAH, results in neurogenic vasodilatation and hyperaemia [1, 4].

We encountered sudden intraoperative brain swelling following excision of a tumour, which was moderately vascular. In such an emergent situation, anaesthetic as well as intracranial causes need to be rapidly checked and ruled out, as persistence of brain swelling could lead to irreversible neurological damage and even rupture of the brain[1, 5]. Possible anaesthesia related aetiological factors like hypercarbia, hypoxia, overhydration and faulty positioning of the head leading to impaired cerebral venous drainage were first ruled out. Simultaneously, an attempt was made by the surgeon to rule out haemorrhage into the depths of the tumour cavity. Other situations in which brain swelling could occur include excision of vascular tumours, tumours involving deep venous structures, evolving distant haematoma [6] and rupture of aneurysms with subarachnoid haemorrhage [1,7].

However, intraoperative sonography as well as post-operative CT scan ruled out any major intra-tumoural bleeds. The latter, however, revealed the presence of subarachnoid blood in the supratentorial region associated with mild hydrocephalus, which could have been the cause of brain swelling. Another cause for open brain herniation may be brain shrinkage and shift consequent upon tumour decompression that may tear draining veins leading to contralateral subdural haematoma. Ohigashi et al reported intraoperative massive bleeding and sudden brain swelling in their case report in which the bleeders were the diploic veins and the complication occurred immediately at craniotomy [8].

Management of intraoperative brain herniation is directed towards reducing cerebral blood volume, improving cerebral oxygenation and reducing the effects of brain swelling. Since the brain bulk in such a situation is primarily due to hyperaemia, both pharmacological as well as non-pharmacological measures may be effective. We instituted hyperventilation as one of the therapies to reduce brain swelling. This strategy, however, carries the potential risk of reducing cerebral oxygenation by excessive reduction of cerebral blood flow in the normal brain as well. Therefore, its use has been recommended for short periods only. Moreover, there is no direct evidence that hyperventilation improves outcome in acute intraoperative cerebral edema. Although barbiturates have been used in the treatment of raised ICP and in intraoperative brain edema, it has not resulted in any obvious benefit [1] nor is its effect on outcome uniform [9]. In the series reported by Whittle et al [1], despite using thiopentone in all their patients, there was no marked improvement in brain swelling and the scalp had to be closed on the swollen brain. Besides, administration of high dose barbiturates for prolonged periods, as required in patients with brain swelling, can lead to delayed awakening, immunosuppression and electrolyte imbalance. In our patient, we discontinued all inhalational agents and resorted to total intravenous anaesthesia with propofol infusion. The global effects of propofol include a reduction in cerebral metabolic rate and CBF with concomitant reduction in ICP

and MAP. Its cellular effects include glutamate receptor antagonism at the NMDA receptor and GABA receptor agonism [10]. These actions support its ability to reduce release of excitotoxic factors secondary to cerebral ischaemia, which can occur as a consequence of brain herniation. Beneficial neuroprotective properties have also been demonstrated in animal studies[11,12]. If similar effects can be proven in clinical studies, it would be superior to barbiturates, as it does not present problems associated with high dose or prolonged barbiturate therapy. The use of a short-acting drug like propofol may be beneficial in this regard and may have facilitated early weaning from ventilatory support in our patient.

The surgical management of open brain herniation is controversial. In a series of patients who developed open brain herniation after elective neurosurgery, the brain had to be manually confined to effect scalp closure, which may have to be done in a "floating fashion"[1]. In our patient, since intraoperative sonography did not reveal any significant haematoma in the tumour bed, a lobectomy was performed to prevent rupture of the herniating brain. This is a matter of controversy, as some authors are of the opinion that resection of normal brain tissue by the surgeon faced with intraoperative brain herniation is not really justified, as it could result in a major loss of neural function. Despite the catastrophic nature of the event, the overall outcome in patients with intraoperative brain herniation due to extra-axial subarachnoid or intraventricular haemorrhage is better as compared to brain herniation secondary to intraparenchymal haemorrhage or diffuse cerebral edema. In a series of 7 patients who developed intraoperative brain edema, out of which, five had rupture of cerebral aneurysm, the authors mention expeditious abandonment of the procedure with closure of the cranium contributing to the satisfactory clinical outcome [1]. Although our patient was successfully weaned off the ventilator in the immediate postoperative period, she had hemiparesis, which could be attributed to the resection of brain done to decompress the swelling.

In summary, we report a case of intraoperative brain swelling in a patient undergoing excision of a brain tumour where the exact cause could not be ascertained. We believe that diffuse subarachnoid bleed along with ventriculomegaly could have contributed to brain swelling, although its aetiology could not be established. Despite the fact that the extreme step of performing a lobectomy had to be undertaken to restore cerebral homeostasis, timely institution of pharmacological as well as non-pharmacological measures could have favourably affected the outcome of this patient. In this regard, institution of propofol infusion may have the advantage of not only decreasing cerebral blood flow and cerebral metabolism, but may also have helped to prevent ischaemia-induced release of excitotoxic factors as well as affording neuroprotection. Moreover, it facilitated early recovery of the patient, which helped in early neurological assessment, and subsequent weaning of the patient from mechanical ventilation. Thus, the rapidity with which sudden intraoperative open brain herniation is recognized by the surgeon, conveyed to the anaesthesiologist and treatment strategies instituted determines the outcome of the patient. In this respect, a close coordination between the neurosurgeon and the anaesthesiologist is essential. The favorable outcome in this patient may have also been partly due to the extra-axial nature of the bleed, which is generally associated with a better prognosis.

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References

1. Whittle I R, Vishwanathan R. Acute intraoperative brain herniation during elective neurosurgery: Pathophysiology and management considerations. J Neurol Neurosurg Psychiatry 1996; 61: 584-90
2. Dorsch N, Branston N, Symon L, Jakubowsky J. Intracranial pressure in experimental subarachnoid haemorrhage. In: Hoff JT, Betz AI, eds. Intracranial pressure VII. Berlin, Springer Verlag, 1989; 715-18
3. Nornes H. The role of intracranial pressure in the arrest of haemorrhage in patients with ruptured intracranial aneurysm. J Neurosurg 1973; 39: 226-34
4. Hashimoto M, Higashi S, Kogure Y et al. Pressure waves and brain stem microcirculatory disturbance following experimental subarachnoid haemorrhage. In: Hoff JT, Betz AI, eds. Intracranial pressure VII. Berlin, Springer Verlag, 1989; 748-53
5. Reilly PL Intra-operative brain swelling (editorial) Br J Neurosurg 1990; 4(1): 3-4
6. Medina M, Melcarne A, Musso C, Ettorre F. Acute brain swelling during removal of supratentorial cystic lesion caused by contralateral extradural hematoma: case report. Surg Neurol 1997; 47(5): 428-31
7. Pickard JD, Lovick AHJ, Read DH. Evidence for brain engorgement as the initial cause of brain swelling following intraoperative aneurysm rupture in man after subarachnoid haemorrhage. Acta Physiol Scand 1986; 172 (suppl 552): 94-95
8. Ohigashi Y, Tanabe A. A huge frontal meningioma associated with intraoperative massive bleeding and severe brain swelling: case report. J Clin Neurosci 2001; 8(suppl): 54-58

9. Chee VWT, Lew TWK. Acute intraoperative cerebral oedema: Are current therapies evidence based? Anaesth Intensive Care 2003; 31: 309-315
10. Orser BA, Bertlik M, Wang LY, MacDonald JF. Inhibition by propofol(2,6 di isopropylphenol) of the N-methyl-D-aspartate subtype glutamate receptor in cultured hippocampal neurons. Br J Pharmacol 1995; 116: 1761-1768
11. Peters CE, Korcok J,Gelb AW, Wilson JX. Anesthetic concentrations of propofol protect against oxidative stress in primary astrocyte cultures: comparison with hypothermia. Anesthesiology 2001; 94: 313-321
12. Pittman JE, Sheng H, Pearlstein R, Brinkhous A, Dexter F, Warner DS. Comparison of the effects of propofol and pentobarbital on neurologic outcome and cerebral infarct size after temporary focal Ischemia in the rat. Anesthesiology 1998; 89: 798-799

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