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Case report

Remote epidural haematoma and severe basal ganglia oedema complicating the removal of a central neurocytoma in the lateral ventricle: A case report and lessons learned

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1. Introduction

Tumours of the lateral ventricle are common in general neurosurgical practice with a reported incidence of 0.81-1.6% [1]. The majority of these lesions are benign or low grade and can be removed successfully through carefully planned surgical approaches. However, even for experienced surgeons, the surgical removal of these tumours remains a challenge and may occasionally cause severe neurological morbidity or even death. In the present report, we described a unique case of a lateral ventricular neurocytoma that simultaneously developed two rare but dreaded complications in the form of a remote epidural haematoma and severe basal ganglia oedema following surgical removal of the tumour using a frontal transcortical approach. The patient had an eventful clinical course, but a satisfactory outcome was achieved. The mechanism by which remote postoperative EDHs develops is unclear, but several hypotheses have been proposed. In our case, the loss of a substantial CSF volume during surgery, which caused a sudden drop in intracranial pressure, may have played a significant role in the pathophysiological development of the remote EDH. The involvement of the thalamostriate vein during surgery and the

2. Case report

A 45-year-old female was admitted to the hospital with a 6-month history of a progressive frontal headache and right limb weakness. The clinical examination demonstrated mild right hemiparesis (grade 4/5) and no other abnormalities. Magnetic resonance imaging disclosed a $4.0 \, \text{cm} \times 2.5 \, \text{cm}$ occupying lesion with a hypointense signal on T1-weighted images and heterogeneous enhancement after gadolinium infusion. The mass was located in the body of the left lateral ventricle and obstructed the foramen of Monro with ventricular dilatation (Fig. 1A). The patient underwent surgery to remove the tumour via a frontal transcortical approach. Intraoperatively, the mass appeared to be fragile and was attached to the ventricle wall and fed by numerous small vessels. A complete microsurgical excision of the tumour was achieved in a piecemeal fashion. Then a temporary external ventricular drain (EVD) was placed to assist with clearance of blood in the CSF and prevent postoperative hydrocephalus.

Seven hours after the surgery, before the patient awoke from anaesthesia, we performed a CT scan of the head. The CT scan revealed a large epidural haematoma in the left parietooccipital

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subsequent deep vein thrombosis may have caused the severe basal ganglia oedema. In this paper, we will discuss the pathogenesis and management aspects of the complications and the lessons that we learned from this case.

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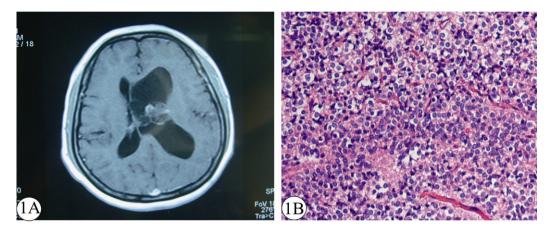


Fig. 1. (A) A preoperative MRI showing a 4×2.5 cm mass located in the body of the left lateral ventricle near the foramen of Monro. The mass caused displacement of the septum pellucidum and obstructive unilateral hydrocephalus. The lesion is solid and lobulated and shows mild heterogeneous enhancement after contrast administration. (B) The postoperative histopathologic examination (HE \times 100) revealed that the tumour was a neurocytoma.

region, which was remote from the site of the craniotomy (Fig. 2A). The patient was immediately returned to the operating theatre, and the epidural haematoma was evacuated through a left parietoccipital craniotomy (Fig. 2B). Following the second surgery, the patient awoke with slight disorientation and mildly increased right hemiplegia.

However, the patient developed a second complication. On postoperative day 2, the patient became drowsy with sudden focal sign deterioration and then developed generalised tonic–clonic seizures. An urgent CT showed severe oedema of the left basal ganglia region with a 1.5 cm midline shift to the right (Fig. 3A). The patient was taken to the operating theatre, and a left decompressive craniectomy was performed. The patient recovered gradually. One month after the third surgery, she was discharged and referred to another hospital for further adjuvant therapy. The clinical examination demonstrated slight disorientation in time and place and right hemiparesis (grade 2/5 in the arm and grade 4/5 in the leg). Subsequent CT scans revealed complete excision of the tumour and the EDH. The left basal ganglia oedema had also apparently improved (Fig. 3B). The tumour was histologically diagnosed as a central neurocytoma (Fig. 1B).

3. Discussion

Postoperative EDH is a serious and well-recognised complication of intracranial surgery that usually occurs at the site of the operation. However, a remote EDH, which occurs distant to

the site of the craniotomy, is relatively rare. Various hypotheses regarding the pathophysiology of this entity have been put forward. Because patients with remote EDHs tend to be young and have long-standing lesions with pre-existing hydrocephalus and the haematoma mostly follows neurosurgical procedures that involve opening the ventricular system or cerebrospinal fluid (CSF) cisterns, the loss of a substantial volume of CSF during surgery appears to be central to the pathophysiological development of a remote EDH [2]. Most researchers have postulated that a sudden reduction in ICP at the time of a craniotomy, tumour removal or ventricular drainage may cause traction on the dural bridging veins, which in turn may strip the dura from the inner table of the skull, resulting in extradural vessel haemorrhaging. This would cause further dural detachment with haematoma expansion [3]. Other reports have described cases of postoperative remote EDHs that may have resulted from coagulopathy or the use of pins for rigid fixation during surgery. In our case, the external ventricular drainage catheter was closed and had not yet been connected to the drainage bag in the first few hours after the surgery, so overdrainage after surgery was firstly ruled out as a cause of the EDH. Furthermore, the patient's coagulation profiles were all within the normal range during and after surgery, the location of the EDH at the left parietooccipital region was not adjacent to a pin site. During the craniotomy to evacuate the EDH, no fracture line was observed on the exposed bone, and the pins had not penetrated the inner cortex. Thus, it is difficult to attribute this EDH to pin-induced complications. Therefore, we believe that lowering the ventricular pressure

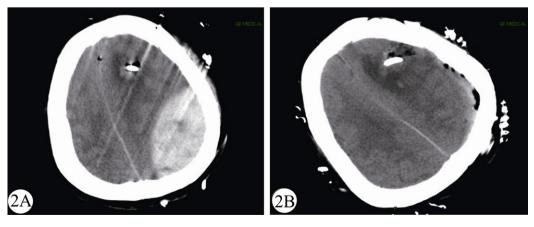


Fig. 2. (A) A postoperative CT scan demonstrating a large left parietooccipital acute epidural haematoma. (B) Repeat CT scans revealed complete evacuation of the EDH and no fresh lesion.

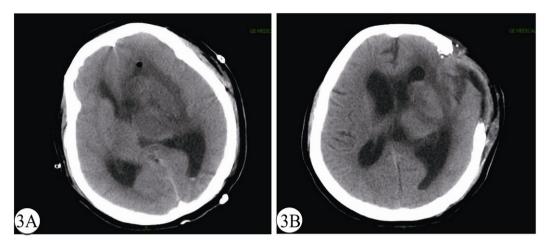


Fig. 3. (A) An urgent CT scan showed severe oedema of the left basal ganglia region with a 1.5 cm midline shift to the right. (B) Subsequent postoperative CT scans revealed that the left basal ganglia oedema had apparently improved, and the tumour was completely resected.

using a ventricular tap during the operation may have played a significant role in the formation of the remote EDH. No single preoperative or intraoperative factor can reliably predict the occurrence of remote site haemorrhages. It is important that we learn from this case that progressively decreasing ICP may prevent a remote EDH and that this hazardous complication can carry an excellent prognosis if detected early and treated promptly.

Another rare but dreaded complication mentioned in our case is severe basal ganglia oedema, which we speculate was caused by the involvement of the thalamostriate vein during surgery and the subsequent deep vein thrombosis. In our case, the tumour was located in the body of the lateral ventricle and attached to eloquent structures around the foramen of Monro. Therefore, it was difficult to remove the tumour from the lateral ventricle wall without interfering with the deep draining veins, and this may have caused the basal ganglia oedema. In addition, we identified some penetrating arteries supplying the tumour from thalamus and the basal ganglia region. These arteries were sacrificed during tumour removal. The expansion of thrombosis and vasospasm also directly influenced the blood supply to the thalamus and the basal ganglia region. The consequence of deep vein thrombosis is difficult to ascertain in an individual patient and can result in very different clinical courses [4]. In our case, the patient presented with a sudden deterioration of the focal signs and consciousness disturbances on postoperative day 2, and the prompt decompressive craniectomy prevented the patient from becoming comatose or dying from a cerebral herniation led by a midline shift if untreated.

A central neurocytoma, which is usually located in the lateral ventricles, is a rare type of neuronal tumour with benign histology and biological behaviour. Whenever possible, the standard initial treatment for a central neurocytoma is a complete resection. However, central neurocytomas tend to be radiosensitive, and gamma

knife radiosurgery has been shown to be effective against postoperative residual tumours or local recurrences of central neurocytomas [5]. Therefore, more care should be taken to preserve the eloquent structures near the tumour, and a subtotal resection is occasionally preferred.

4. Conclusion

Remote EDHs and severe basal ganglia oedema represent rare but potentially fatal complications following the surgical removal of lateral ventricle tumours. The early detection and prompt treatment of these conditions may greatly improve the outcome. We strongly advise that sudden reductions in ICP be avoided and that more care be taken to preserve the eloquent structures at the time of ventricular tumour removal to reduce the risk of such dreaded complications.

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