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Multiple supratentorial intraparenchymal hemorrhage after posterior fossa surgery

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Abstract

Background: The intraparenchymal supratentorial hemorrhages after interventions of the posterior fossa is a very rare complication, with very little literature and its precise incidence is unknown (range of 0.4–1.6%). It possesses potentially an etiology diverse from that associated with other postoperative bleeding.

Case Description: A white, 23-year-old female, with no history of coagulation disorders or other diseases, was referred to our hospital with a large ependymoma, which extended from the floor of the fourth ventricle, emerged from the foramen of Magendie and descended to the C2 level. The patient was submitted to surgical treatment and during resection of the lesion, when near the vagal trigone, the patient presented great pressure lability. In the immediate postoperative period, the patient did not have a level of consciousness sufficient to tolerate extubation. Brain computed tomography (CT) was carried out, which showed multiple supratentorial hemorrhages. On the ninth day of the postoperative period, there was a sudden neurological worsening and anisocoria. A new brain CT was carried out [Figure 4], which demonstrated a diffuse cerebral edema. In spite of the introduction of clinical measures for the control of diffuse cerebral edema, the patient evolved to brain death.

Conclusions: The principal measures in the management of these cases include early diagnosis, detection of possible coagulation disorders, continual monitoring, and maintenance of adequate cerebral perfusion. Surgical treatment is recommended in cases of the presence of mass effect or diffuse edema not yielding to clinical treatment. High rates of mortality and morbidity are observed.

Key Words: Ependymoma, hemorrhage, posterior fossa

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INTRODUCTION

Postoperative intracerebral hemorrhage remote from the operative site is a rare occurrence in the neurosurgical field, especially rare when it occurs in the supratentorial compartment after interventions to the posterior fossa. Its occurrence is associated with great morbidity and mortality.[1,5,13]

Haynes *et al.*[5] believe that the incidence of supratentorial hemorrhage in procedures in the posterior fossa is 0.6%.

In this work, we report the case of a patient submitted to resection of an ependymoma in the floor of the fourth ventricle who presented multiple supratentorial hemorrhages after surgery. We undertook further a review of the literature on the epidemiology, treatment, and clinical evolution of patients with supratentorial hemorrhages after interventions in the posterior fossa.

CASE REPORT

A white, 23-year-old female, with no history of coagulation disorders or other diseases, was referred to our hospital with a history of neck pain that commenced 1 year ago, and about 6 months ago evolved with a loss of power and multiple falling episodes.

The general physical examination was normal. In the neurological examination, the patient was completely lucid, tetraparetic, 4/5 on the right and 3/5 on the left and hyperreflexia in the 4 members.

Magnetic resonance imaging (MRI) demonstrated the existence of a large lesion, suggestive of ependymoma, which extended from the floor of the fourth ventricle, emerged from the foramen of Magendie and descended to the C2 level [Figure 1]. No other alteration was observed in the complementary preoperative examinations.

The patient was submitted to median suboccipital craniotomy and C1 laminectomy in the park bench position with the left side down [Figure 2]. The procedure was performed with continual electrophysiological monitoring, which showed no evidence of abnormalities over the whole surgery. During resection of the lesion, adhering to the floor of the fourth ventricle, near the vagal trigone, the patient presented great pressure lability, with rapid pressure elevation, blood pressure 230/120 mmHg. After intensive anesthetic management, the lability was corrected and the tumor completely removed without other complications.

As a routine in our department, in the immediate postoperative period, we tried to wake up the patient and extubate. However, the patient did not have a level of consciousness sufficient to tolerate extubation, and was maintained under mechanical ventilation. Therefore a brain computed tomography (CT) was carried out immediately after surgery [Figure 3], which showed multiple supratentorial hemorrhages. The patient evolved with good neurological response, opening her eyes when called and obeying commands. Considering the progressive clinical recovery, the absence of lesions of significant mass and the presence of multiple bleeding focuses, a conservative treatment of the bleeding was chosen. Postoperative exams did not demonstrate any signs of coagulation disorders.

The patient evolved in the days following with spontaneous opening of the eyes, and obeying commands, but continued in mechanical ventilation as she did not protect the airway effectively. Vasoactive drugs (norepinephrine) were introduced to maintain the mean arterial pressure (MAP) >60 mmHg, as pressure lability continued to be present in the postoperative period, with adequate control initially.

On the fourth day after surgery, the patient presented symptoms of pneumonia and was treated with antibiotics with satisfactory response. The patient presented good parameters at the physiological monitoring with electrocardiogram (ECG), respiratory rate, invasive blood pressure (IBP), body temperature, arterial hemoglobin oxygen saturation (SpO_2); and adequate intravascular volume. However, on the ninth day of the postoperative period, there was a sudden neurological worsening and anisocoria. A new brain CT was carried out [Figure 4], which demonstrated a diffuse cerebral edema. In spite of the introduction of clinical measures for the control of diffuse cerebral edema, the patient evolved to a condition of brain death.

A pathological anatomical examination confirmed the hypothesis of ependymoma.

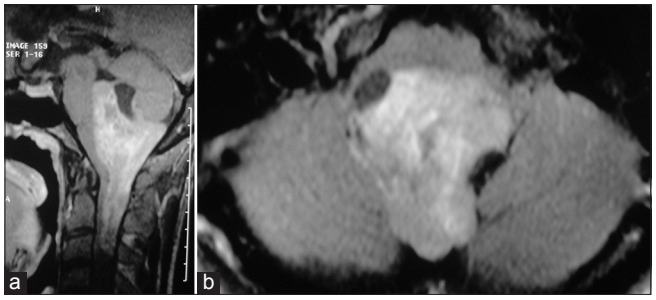

Figure 1: MRI (a) Sagittal T2-weighted MRI showing a large posterior fossa ependymoma, extending from the fourth ventricle to the C2 level. (b) Axial T1-weighted MRI with contrast, showing a fourth ventricle ependymoma passing by the foramen of Margendie

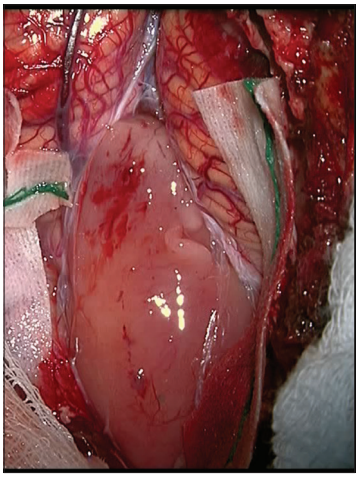

Figure 2: Aspect of the tumor after craniotomy and laminectomy, suggestive of ependymoma

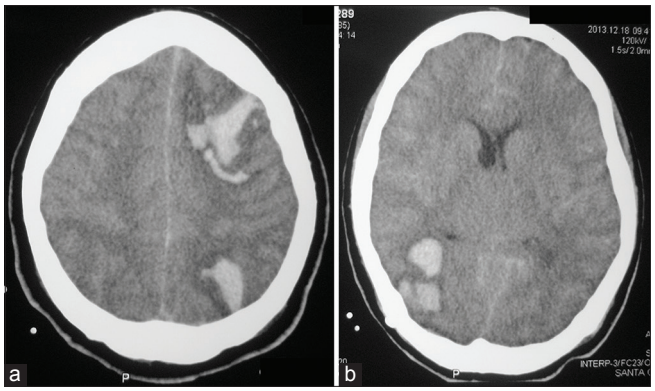

Figure 3: Computed tomography. (a) Image showing frontal and parietal left hemisphere hemorrhage. (b) Image showing occipital right hemisphere hemorrhage

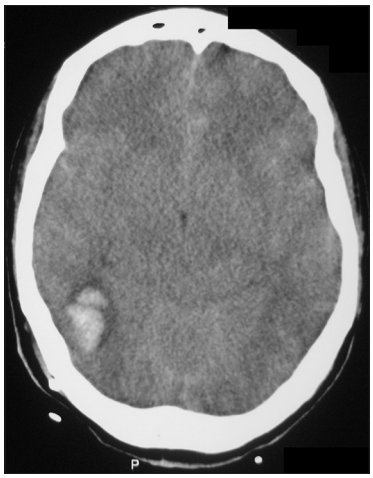

Figure 4: Computed tomography showing diffuse brain edema and basal cistern effacement

DISCUSSION

Postoperative hemorrhages remote from the craniotomy site can occur after supra or infratentorial interventions. They are rare complications and normally associated to great morbidity and mortality.[2] Different types of supratentorial bleeding after posterior fossa surgery have already been reported, such as epidural hematoma,[10,16] subdural hematoma,[6] subarachnoid hemorrhage,[1] intraparenchymal hemorrhage [Table 1], and ventricular hemorrhage.[5] Another type of supratentorial hemorrhage after infratentorial intervention was reported by Tondon *et al.*,[13] such as a case of left parietal-occipital hematoma observed 5 days after the resection of a extensive meningioma; and another case of hematoma in the left basal ganglia caused by a tumor in this region; both the patients having coagulation disorders.

The intraparenchymal supratentorial hemorrhages after interventions of the posterior fossa possess potentially an etiology diverse from that associated with other postoperative bleeding [Table 1]. It is a rare complication, with very little literature and its precise incidence is unknown. Haines *et al.*[5] reported 4 (0.48%) cases in 825 patients operated on the posterior fossa; Standefer *et al.*[12] reported 2 (0.4%) cases in 488 patients; and Harder *et al.*[6] reported 3 (1.6%) cases in 187 patients; hence the incidence is in the range of 0.4–1.6%.

In accordance with our review, there are 18 cases of supratentorial intraparenchymal hemorrhage after interventions to the posterior fossa, published in the literature. The majority of patients are females, 13/16 (81.2%) and middle-aged, mean age: 53.7 years old. Only 1/15 (6.6%) of the cases present associated coagulation disorders. In the majority of cases, the intervention to the posterior fossa was for tumor resection 13/18 (72.2%); Meningioma 5/13 (38.4%); acoustic neuroma 3/13 (23%); hemangioblastoma 2/13 (15.3%). The majority of patients were submitted to the surgery in the sitting position, 14/18 (77.7%). The diagnosis of distant hematoma occurred during the first hours after the procedure in 12/14 cases (85.7%).

There is no predominance of a specific location as focus for hemorrhages. Multiple bleeding focuses were observed in five patients (27.7%), and one of them presented a coagulation disorder.

The majority of patients evolved to death, 8/16 (50%). Severe deficiency, including hemiparesis and/or visual loss, was present in 4/16 (25%) of the cases, moderate deficiency in 3/16 (18.7%) and light deficiency in 1/16 (6.2%).

The case reported is the only one to present multiple bleeding sites soon after the surgery. In the case introduced by Vrettou *et al.*,[14] in which the patient presented Factor XIII deficiency, there was initially a single bleeding and after the decompressive craniectomy new focuses arose.

In the majority of cases, the diagnosis of the bleeding was performed within a few hours of the surgery, which suggests that this complication occurred during or soon after the surgery.

Coagulation disorders can be the cause of this intraparenchymal bleeding at a distance, as in the case introduced by Vettrou *et al.*[14] nevertheless, in our case all the coagulation examinations were within the standards of normality, discarding this possibility.

In the absence of predisposing factors, there exist some theories that attempt to explain the pathophysiology of the supratentorial intraparenchymal bleeding after surgery to the posterior fossa, including:[6,13,14] The sitting position, hypertension, and disorder of the venous drainage. The sitting position can reduce the arterial blood flow, causing ischemia and, after the return to the supine position, the hyperperfusion can lead to hemorrhaging in the cerebral tissue previously affected

Table 1: Summary of 18 cases of supratentorial intraparenchymal hemorrhage after posterior fossa surgery

Author	Year	Sex	Age	Coagulation disorder	Posterior fossa approach	Surgical position	Posterior fossa pathology	Location of hemorrhage	Hematoma diagnosis	Outcome
Haines *et al.*[5]	1978	F	65	No	Right retromastoid craniectomy	Sitting	Trigeminal neuralgia	Right occipital	Within hours	Moderate
Haines *et al.*[5]	1978	F	55	No	Right retromastoid craniectomy	Sitting	Glossopharyngeal neuralgia	Righ basal ganglia	Within hours	Death
Haines *et al.*[5]	1978	F	41	No	Right retromastoid craniectomy	Sitting	Atypical trigeminal neuralgia	Right fronto-parietal	Within hours	Moderate
Haines *et al.*[5]	1978	F	64	No	Right retromastoid craniectomy	Sitting	Anesthesis dolorosa	Left frontal	Within hours	Mild
Cartier-Giroux *et al.*[4]	1980	F	55	No	Suboccipital craniectomy	Sitting	Breast cancer	Right basal ganglia	Day 3	Death
Waga *et al.*[15]	1983	M	39	No	Subocciptal + C1-C3 laminectomy	Prone	Syrinx	Bilateral parietal	Within hours	Death
Standefer *et al.*[12]	1984	NA	55	No	Subocciptal + C1-C3 laminectomy	Sitting	Parotid cancer	Temporal	NA	Death
Standefer *et al.*[12]	1984	NA	59	No	Suboccipital	Sitting	Meningioma	Basal ganglia	Day 7	Death
Harders *et al.*[6]	1985	F	44	NA	Suboccipital	Sitting	Meningioma	Left fronto-parietal	NA	Severe
Harders *et al.*[6]	1985	F	51	NA	Suboccipital	Sitting	Acoustic neuroma	Bilateral fronto-temporal	NA	Death
Harders *et al.*[6]	1985	M	58	NA	Suboccipital	Sitting	Meningioma	Left parietal	NA	Moderate
Seiler *et al.*[11]	1986	F	66	No	Right suboccipital	Lateral	Acoustic neuroma	Right parietal	Within hours	Severe
Seiler *et al.*[11]	1986	F	64	No	Right suboccipital	Sitting	Meningioma	Left parietal and right basal ganglia	Within hours	Death
Seiler *et al.*[11]	1986	F	59	No	Right suboccipital	Sitting	Acoustic neuroma	Right parietal occipital	Within hours	Severe
Bucciero *et al.*[3]	1991	M	46	No	Suboccipital craniectomy	Sitting	Hemangiolastoma	Left temporo-parietal	Within hours	NA
Kalkan *et al.*[8]	2006	F	63	No	Suboccipital craniectomy	Sitting	Meningioma	Right fronto-parietal	Within hours	NA
Vrettou *et al.*[14]	2010	F	60	Factor XIII deficiency	Posterior fossa craniotomy	Prone	Cerebellar hemangioblastoma	Frontal lobe hematoma with severe intraventricular hemorrhage After craniectomy: New bleeding areas in the right frontal, occipital and parietal lobes, as well as subarachnoid hemorrhage	Within hours	Severe
Albuquerque *et al.*	2014	F	23	No	Subocciptal midline craniotomy+C1 laminectomy	Lateral	Ependimoma	Left frontal and parietal and right occipital intraparenchymal hematoma	Within hours	Death

All cases of epidural hematoma, subdural hematoma, subarachnoid hemorrhage and intraventricular hemorrhage are excluded in the table. The paper of Kalfas *et al.*[7] was excluded in the table because of lack of data. The paper of Tondon *et al.*[13] was excluded in the table because the two cases presented were not intraparenchymal hemorrhage. One case was a hemorrhage in a previous surgical bed and the other case was a hemorrhage inside a second tumor. M: Male, F: Female, CP: Cerebello-pontine, NF: Neurofibromatosis, NA: Not available

by ischemia. Also described is the occurrence of subdural hematoma after surgical interventions in the sitting position, possibly due to rupture of cortical bridging veins. Through the same mechanism, subcortical veins can burst and cause intraparenchymal hemorrhage.[8,13] The majority of cases described in the literature were done in the sitting position. Accordingly, considering that the case here described was performed in the park bench position, it is improbable that there is an association between the bleeding and the positioning chosen for this case. Neither does the bleeding seem to have arisen out of venous drainage disorders, as there was no significant venous or venous sinus injury during the surgery that could justify the multiple points of bleeding.

Ample drainage of cerebrospinal fluid (CSF) in patients with lesions of the posterior fossa, submitted to surgery in the sitting position, may be associated with supratentorial bleeding because of the rapid reduction of the intracranial pressure. Such fact could cause rupture in the ventricular ependymal wall.[4] Such a hypothesis

does not adequately explain the alterations observed in this case as the hemorrhages presented do not possess a periventricular character.

As introduced by Cartier-Giroux *et al.*,[4] the multiple hematomas were potentially secondary to a rapid elevation of the arterial pressure during the tumor resection next to the fourth ventricle, possibly by the manipulation of medullary vasomotor centers (possibly the dorsal nucleus of the vagus).

Peri-hemorrhagic edema may be separated into an acute phase (0–48 h) and a delayed phase (maximal edema at the end of week two). Second, delayed processes start a few days after ictus with an activation of the coagulation cascade, thrombin production, and erythrocyte/hemolysis products inciting inflammation resembling a mixed picture of cytotoxic and vasogenic edema. Our patient presented a delayed deterioration probably secondary do peri-hemorrhagic edema.[9] We believe that it leads to an increase of mass lesion and intracranial pressure, which consecutively decrease in cerebral perfusion, secondary brain damage and fatal outcome.

Intraparenchymal supratentorial hematomas after interventions of the posterior fossa where the patient does not awaken adequately or develops a new neurological deficiency should be suspect. The management varies case by case, always remembering, however, coagulation disorders and rigorous pressure control. Surgical treatment is reserved for lesions with mass effect and midline deviation. Bilateral decompressive craniectomy can be realized as an alternative for the treatment of diffuse cerebral edema not yielding to clinical treatment. Clinical treatment is based on the control of pressure lability, monitoring intracranial pressure in cases where there is a lowering of the level of consciousness, maintenance of adequate cerebral perfusion rate and the identification and treatment of potential coagulation disorders.

CONCLUSION

Supratentorial hemorrhage after resection of lesions in the posterior fossa is a rare phenomenon. It is potentially associated with ample drainage of CSF and the sitting position, which can generate excessive reduction of intracranial pressure and bleeding through injury to small bridging veins; and pressure lability during the removal of tumors next to the floor of the fourth ventricle. High rates of mortality and morbidity are observed. The principal measures in the management of these cases include early diagnosis, detection of possible coagulation disorders, continual monitoring and maintenance of adequate cerebral perfusion. Surgical treatment is recommended in cases of the presence of mass effect or diffuse edema not yielding to clinical treatment.

REFERENCES

1. Agrawal A, Kakani A, Ray K. Extensive supratentorial hemorrhages following posterior fossa meningioma surgery. J Surg Tech Case Rep 2010;2:87-9.
2. Brisman MH, Bederson JB, Sen CN, Germano IM, Moore F, Post KD. Intracerebral hemorrhage occurring remote from the craniotomy site. Neurosurgery 1996;39:1114-21.
3. Bucciero A, Quaglietta P, Vizioli L. Supratentorial intracerebral hemorrhage after posterior fossa surgery. Case report. J Neurosurg Sci 1991;35:221-4.
4. Cartier-Giroux J, Mohr G, Sautreaux JL. Supratentorial hemorrhage of hypertensive origin during operation: An unusual complication of surgery on the posterior fossa in the sitting position. Neurochirurgie 1980;26:291-4.
5. Haines SJ, Maroon JC, Jannetta PJ. Supratentorial intracerebral hemorrhage following posterior fossa surgery. J Neurosurg 1978;49:881-6.
6. Harders A, Gilsbach J, Weigel K. Supratentorial space-occupying lesions following infratentorial surgery: Early diagnosis and treatment. Acta Neurochir (Wien) 1985;74:57-60.
7. Kalfas IH, Little JR. Postoperative hemorrhage: A survey of 4992 intracranial procedures. Neurosurgery 1988;23:343-7.
8. Kalkan E, Eser O. Supratentorial intracerebral hemorrhage following posterior fossa operation. Neurol India 2006;54:220-1.
9. Kuramatsu JB, Huttner HB, Schwab S. Advances in the management of intracerebral hemorrhage. J Neural Transm 2013;120 Suppl 1:S35-41.
10. Pandey P, Madhugiri VS, Sattur MG, Devi B I. Remote supratentorial extradural hematoma following posterior fossasurgery. Childs Nerv Syst 2008;24:851-4.
11. Seiler RW, Zurbrugg HR. Supratentorial intracerebral hemorrhage after posterior fossa operation. Neurosurgery 1986;18:472-4.
12. Standefer M, Bay JW, Trusso R. The sitting position in neurosurgery: A retrospective analysis of 488 cases. Neurosurgery 1984;14:649-58.
13. Tondon A, Mahapatra AK. Superatentorial intracerebral hemorrhage following infratentorial surgery. J Clin Neurosci 2004;11:762-5.
14. Vrettou CS, Stavrinou LC, Halikias S, Kyriakopoulou M, Kollias S, Stranjalis G, Koutsoukou A. Factor XIII deficiency as a potential cause of supratentorial hemorrhage after posterior fossa surgery. Acta Neurochir (Wien) 2010;152:529-32.
15. Waga S, Shimosaka S, Sakakura M. Intracerebral hemorrhage remote from the site of the initial neurosurgical procedure. Neurosurgery 1983;13:662-5.
16. Wolfsberger S, Gruber A, Czech T. Multiple supratentorial epidural hematomas after posterior fossa surgery. Neurosurg Rev 2004;27:128-32.

Commentary

I agree with the authors that their use of a park bench position makes the more common explanations of supratentorial intracerebral hematoma following posterior fossa decompression, as being related to intraoperative patient position, unlikely. This is therefore a particularly interesting paper. Documentation is good. Hyper transient vasomotor instability in the face of work on the floor of the fourth ventricle is also a possibility. This can be missed during transfers, when monitoring can take a back seat to other pressing issues. Most modern monitors have a "trending" capability, which sometimes allows late review of time-based physiological parameters for clarification of the origin of problems.

Speculation on the etiology of the late deterioration is reasonable, albeit unproven. Norepinephrine may also have the capacity to mask an indolent progressive intravascular hypovolemia, particularly in the absence of central monitoring in the face of potential septic corporal transmural volume loss, which might exacerbate vasospasm. I have therefore preferred the use of hyperosmolar volume expansion coupled with beta-blockade as necessary, though the risk of rebound late swelling is to be guarded against, and is also a risk.

Finally, in at least one personal case (not published to date), basilar artery kinking has been documented, following supratentorial swelling late after subarachnoid hemorrhage, following deterioration and just prior to that patient's acute demise. Pressure differential from infratentorial decompression could potentially also lead to this basilar apex kinking, and therefore cause the same supratentorial ischemic compromise, though in this case this is pure speculation.

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