REVIEW ARTICLE - BRAIN INJURY

Treatment of traumatic acute posterior fossa subdural hematoma: report of four cases with systematic review and management algorithm

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Abstract

Background Traumatic posterior fossa subdural hematomas (SDHs) are rare lesions. Despite improvements in intensive care and surgical management of traumatic brain injuries over the last decades, the outcome for posterior fossa subdural hematomas remains poor.

Methods We conduct a retrospective study over a 2-year period of patients sustaining traumatic brain injury and posterior fossa SDH. Additionally, a systematic review of case series published to date was performed.

Results The incidence of posterior fossa SDH was 0,01 % (4/326). All patients in this current series had poor prognosis. Three out of four exhibited ischemic/edema lesions in postoperative CT scans leading to fourth ventricle effacement and persistent brainstem compression. Our literature review retrieved 57 patients from only seven case series. Unfavorable outcomes were seen in 63 % of patients.

Conclusions Our data and data from the literature do not provide sufficient evidence to establish an optimal treatment strategy for posterior fossa SDH. However, based on lessons learned with these four cases, together with results from review of the literature, we propose an algorithm for the management of this rare condition.

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Introduction

Traumatic posterior fossa subdural hematomas (SDHs) are rare lesions in adults, accounting for less than 3 % of head injuries [4, 8, 9, 15]. Patients with this condition have a poor prognosis and a high rate of mortality [9]. The management of these lesions is more complicated than that of their supratentorial counterparts because of the occult nature of clinical findings and lack of intracranial pressure monitoring methods for posterior fossa SDHs. Furthermore, the surgical decision is often clouded by difficulty in distinguishing whether the unconscious state of the patient is due to mass effect of the posterior fossa SDH versus the severity of the traumatic brain injury. Adding to the complexity of these factors, the rarity of these lesions has impaired efforts to assemble consensus and guidelines for the management of posterior fossa SDHs [8, 10]. In this report, we describe four patients with traumatic posterior fossa SDHs treated with a unilateral posterior fossa craniectomy. We discuss treatment options and propose an algorithm for the management of traumatic posterior fossa SDHs.

Methods

Over the 2-year period from January 2008 to February 2010, four patients presenting with posterior fossa traumatic SDHs were retrospectively identified from the database of all patients admitted to a single Center of Trauma Level 1. Clinical data were collected from the medical records and the radiographic findings were reviewed in three patients from the information

manager system Philips' iSite PACS (Philips Eletronics, USA, 2006), available in the institution since 2008. Radiographic data, including the volume and mass effect of the SDH, presence of a skull fracture overlying the hematoma, course of the fracture, and associated brain injury, were reviewed. In one patient the radiographic data (preoperative CT scan) was available in plain film. The SDH volume was measured by the empirical formula of volume (0.5 height x depth x length) on the basis of distance measurements of the depth and length on the CT slice having the largest area of the clot [26]. Patient outcome was obtained 6–8 months after injury and classified according to the Glasgow outcome score [17].

In addition, we systematically searched the PubMed database up to May 2012 for publications on TBI patients with posterior fossa SDHs using the following search terms and combinations: "acute subdural hematoma", "posterior fossa", "cerebral decompression", "brain decompression," "craniotomy", and "decompressive craniectomy". For all studies identified, the "Related Articles" feature in PubMed was used to locate additional relevant studies. From the collected studies, publications were included for review if they described three or more traumatic posterior fossa SDHs, managed either operatively or non-operatively. There were no age limits for inclusion. We excluded non-traumatic posterior fossa SDH, penetrating injuries, and articles not written in English, French, or Japanese.

Two observers (R.L.O.A and W.S.P) independently reviewed the results of the Pubmed search for articles to include and discrepancies were resolved by consensus. Data extracted were age, gender, mechanism of injury, Glasgow coma score at admission, neurological examinations, indications of neurological worsening, CT neuroimaging, hospital course and management. CT imaging characteristics collected included width of the posterior fossa SDH, compression and shift of the fourth ventricle, indications of hydrocephalus, associated skull fracture, and the presence of additional associated traumatic lesions. Patient management information collected included use of external ventricular drainage, surgical procedure performed, and need for permanent shunt diversion of CSF. When available, the patient's Glasgow outcome scale (GOS) was recorded. When the GOS was not reported in the published literature, the observers reconstructed the score, if it could be clearly deducted from the case description. In such instances, the result was dichotomized as a favorable (GOS score of 4 or 5) or unfavorable outcome (GOS score of 1, 2, or 3).

Results

From January 2008 to February 2010, 326 patients were operated on for TBI at our institution. Of these patients, four (0.01 %) had posterior fossa SDH. Three patients were male and one was female (age range, 22–57 year; mean age,

39.3 year). The clinical and radiological findings of this current series are summarized in Table 1.

All patients were adults and sustained a trauma mechanism of direct impact to the occipital bone. Although one patient had been admitted with GCS score 12, she had a sudden deterioration to coma prior to surgery. The median GCS prior to surgery was 6.2 (range, 4–8). All patients had associated lesions and radiographic signs of mass effect. Occipital bone fractures overlying the hematoma were seen in three of the four patients, but none had evident venous sinus injury. The median hematoma volume was 17.9 ml (range, 8–41 ml). Unilateral occipital craniectomy was the cornerstone of treatment in our series and external ventricular drainage was placed in two patients. Postoperative CT scans were available in three patients. Cerebellar ischemic lesions, fourth ventricle compression and obliteration of the quadrigeminal cistern were seen in the postoperative CT scan of these patients. All patients had unfavourable outcomes.

Our literature search strategy yielded 216 citations from which seven publications comprising 57 patients fulfilled the predefined inclusion criteria [4, 9, 11, 13, 19, 29, 30]. Table 2 summarizes the clinical data and radiological findings of these case series reported in the literature. We evaluated the data of the 57 described patients. In brief, 24 (56 %) of 52 patients, in whom Glasgow Coma Scale (GCS) was available, were admitted to hospital with a score less than 8. In total, 77 % of patients had associated intracranial lesions, other than fracture, with the most common findings being cerebellar contusions, frontal contusions, and hydrocephalus. The surgical treatment most commonly performed for patients in these published reports was suboccipital craniectomy [3, 8, 28]. Unfortunately, it was not possible to establish from these literature reports whether the craniectomy was unilateral or bilateral or if an external ventricular drain or shunt was used. Unfavorable outcomes were recorded in 63 % of patients.

Summary of cases

Case 1

A 57-year-old woman experienced blunt head trauma after a motor vehicle accident. The patient was admitted to our hospital with a GCS score of 12. She had no systemic injuries. She quickly showed neurological deterioration in the emergency department from GCS 12–8. Prior to being intubated, the pupils were isochoric, both reacting to light and corneal and pharyngeal reflexes were intact. CT scan of the head showed a linear occipital fracture and a right posterior fossa acute SDH measuring 41 ml in volume. The fourth ventricle and CSF cisterns were obliterated. A computed tomography angiogram (CTA) confirmed a normal vasculature with no evidence of dissection or atherosclerosis. The patient underwent a unilateral posterior fossa craniectomy, with complete evacuation of the hematoma.

Table 1 Summary data of patients

Patient	Age (years), Gender (Male/Female)	Mechanism	GCS	Associated lesions	Time of TBI to surgery (hours)	Operative technique	Second surgery	Outcome (GOS)
1	57, F	Pedestrian versus Auto	12	Frontal contusion, hydrocephalus, occipital fracture	1	EVD, unilateral suboccipital craniectomy	Expansion of suboccipital craniectomy	2
2	54, M	Assault	6	SAH, cerebellar contusion, hydrocephalus, displaced occipital fracture	2	EVD, unilateral suboccipital craniectomy	Ventriculo-peritoneal shunt	2
3	24, M	Fall 10 m	7	Acute epidural hematoma	14	Unilateral suboccipital craniectomy	None	3
4	22, M	Fall	4	Frontal contusion, cerebellar contusion, occipital fracture	3	Unilateral suboccipital craniectomy	None	1

Table 2 Summary of case series published of head-injured patients and traumatic posterior fossa subdural hematoma

Author (year)	Number of patients	GCS < 9	Age (y); gender (M:F)	Mechanism	Coup site	Posterior fossa SDH width	Treatment	Associated lesions	Unfavourable outcome - GOS 1,2,3 (%)
Fukumitsu et al. (1976)	5	NA	≤15 (3) >15 (2); 5M:0F	MVA (3), Fall (2)	Occipital (4); NA(1)	NA	Craniectomy (4) Conservative (1)	Cerebellar injury (4), supratentorial SDH (3)	80 %
Tsai et al. (1980)	6	2	≤ 15 (4) > 15 (2); 5M:1F	NA	NA	NA	Craniectomy (5) Conservative (1)	Cerebellar injury (2), hydrocephalus (1)	50 %
Hamasaki et al. (1987)	4	3	≤15 (1) > 15 (3); 2M:2F	NA	Occipital (4)	NA	Craniectomy (3) Conservative (1)	Cerebellar injury (3)	100 %
Borzone et al. (1995)	3	2	>15 (3); 3M:0F	NA	Occipital (3)	> 1 cm (2)	Unilateral craniectomy (3)	Frontal contusion (3), posterior fossa EDH (2)	33 %
Karasawa et al. (1997)	5	NA	>15 (5); 2M:3F	MVA (2), Fall (3)	Occipital (3)	NA	Conservative (5)	Supratentorial lesion (1)	20 %
d'Avella et al. (2003)	24	12	9–78 (mean age, 39.4); 18M:6F	MVA (12), Fall (12)	NA	> 1 cm (24)	Unilateral or Midline based Craniectomy (19), Ventriculostomy alone (1), Conservative (2)	Cerebellar injury(13), supratentorial lesion (4), hydrocephalus (11)	58 %
Takeuchi et al. (2012)	10	5	≤15 (1) >15 (9); 7M:3F	MVA (3), Fall (6), Hit (1)	NA	1 cm (2)	Conservative (9), craniectomy(1)	Supratentorial SDH (6), supratentorial contusion (5), cerebellar injury (3),	90 %

NA unspecified, *MVA* motor vehicle accident

A postoperative CT scan showed the presence of cerebellar edema and hydrocephalus. An extraventricular drain was placed to treat the hydrocephalus and the patient was re-operated upon to extend the unilateral suboccipital decompressive craniectomy to an expansive bilateral suboccipital decompression (Fig. 1). Late postoperative CT scan depicted a hypodense lesion in the right cerebellar hemisphere. At a subsequent follow-up of 6 months, the patient had a GOS score of 2.

Case 2

A 54-year-old-man with assault to the right parietal region was emergently intubated upon admission to the emergency department. His GCS after intubation was 6 and pupils were sluggishly reactive to light. CT scan of the head showed an acute SDH of the posterior fossa of 9 ml, associated with an extensive displaced occipital fracture, cerebellar contusion, traumatic subarachnoid hemorrhage and moderate hydrocephalus. The fourth ventricle was effaced but still open. CTA showed no vascular lesions. A unilateral suboccipital craniectomy was performed, and the hematoma was evacuated. An external ventricular drain was placed in the right frontal horn of the lateral ventricle. Postoperatively, the patient was CSF diversion dependent and required a ventriculoperitoneal

shunt. Late postoperative CT scan showed bilateral cerebellar hypodensity lesions. At a subsequent follow-up of 8 months, the patient had a GOS score of 2.

Case 3

A 24-year-old man experienced blunt head trauma after a 7-m fall from a building. The patient was admitted in coma and had a GCS score of 7 with mechanical ventilation. He had external signs of right temporal and occipital impact. Both pupils were isochoric and reactive to light. An assessment using the Advanced Trauma Life Support protocol indicated that the patient's other systems were stable. A head CT scan revealed an acute epidural hematoma (EDH) in the right middle cranial fossa, which was associated with a temporal fracture and a small left posterior fossa subdural hematoma without signs of an occipital fracture. The patient underwent an urgent craniotomy and evacuation of the epidural hematoma without complications. Post-operatively, a repeat CT scan showed an increase in the posterior fossa subdural hematoma (hematoma volume 13.2 ml) with compressive effect. We performed a unilateral suboccipital craniectomy to evacuate the subdural hematoma and placed an extraventricular drain to monitor his intracranial pressure (Fig. 2). Further CT imaging after this second surgery

Fig. 1 Patient 1: a Preoperative CT scan showing a right posterior fossa SDH with obliteration of the fourth ventricle and CSF cisterns b) Associated CT findings include a small frontal contusion. c Postoperative CT scan depicting hypodensity in the cerebellum suggestive of ischemia and/or edema with mass effect evidenced by persistent effacement of the fourth ventricle and CSF cisterns. The patient underwent re-operation with extension of the decompression to a bilateral suboccipital craniectomy. d A postoperative CT scan after this second surgery shows persistent mass effect in the posterior fossa despite a wide decompression of the posterior fossa
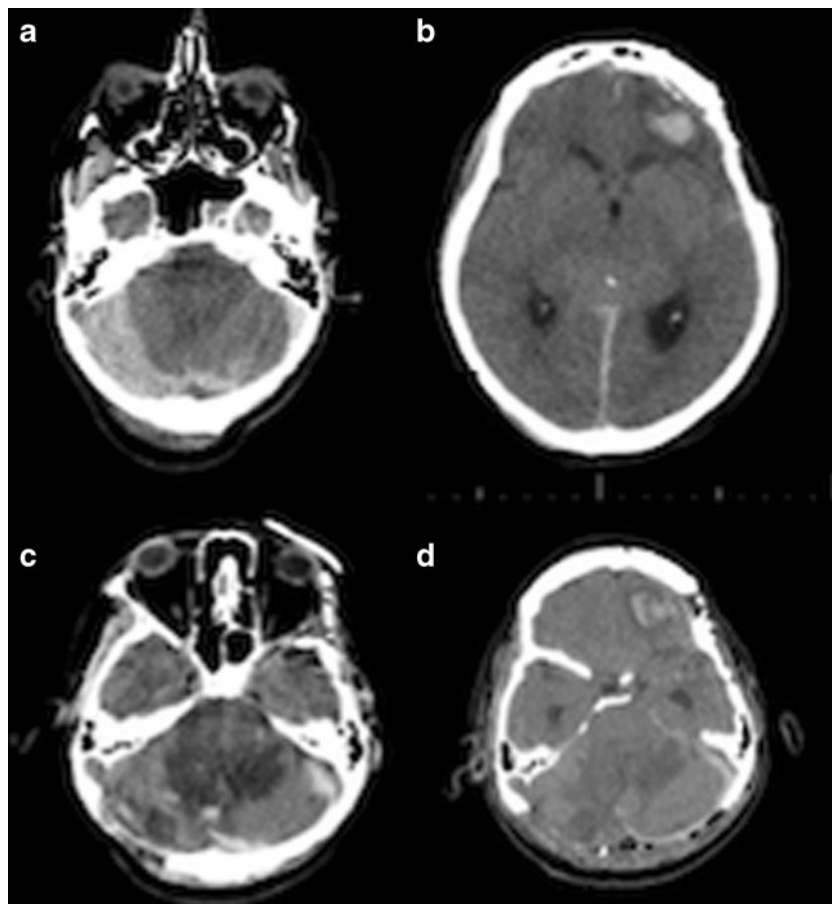
Fig. 2 Patient 3: **a** CT scan showing a right temporal epidural hematoma (EDH) and a laminar posterior fossa SDH (*arrow*) next to the left sigmoid sinus. **b** Post-operative CT scan 8 h after evacuation of the EDH showed expansion of the posterior fossa hematoma. **c** Even after evacuation of the posterior fossa SDH, through a unilateral suboccipital craniectomy, the fourth ventricle remains compressed. No other surgical treatment was adopted

showed good evacuation of the posterior fossa SDH. There was improvement in the degree of mass effect in the posterior fossa, but a hypodense lesion consistent with ischemia or edema was evident within the left cerebellar hemisphere. At late follow-up the patient had a GOS score of 3.

Case 4

A 22-year old man was admitted with occipital trauma after a fall down stairs. He was intubated during transfer to our Level 1 trauma center. Although he had no systemic lesions, he had GCS score of 4 with isochoric and reactive pupils and no other clinical signs of brain stem compression. The CT scan of the brain revealed a left posterior fossa SDH (8.4 ml), a left cerebellar hematoma (12.6 ml) and a right frontal hematoma (2.5 cm in diameter). There were signs of significant mass effect in the posterior fossa including compression of fourth ventricle and obliteration of the tentorial cisterns, consistent with upward herniation. The patient underwent a left suboccipital craniectomy with evacuation of the posterior fossa subdural and cerebellar hematomas and a right frontal craniotomy to evacuate the frontal hematoma. The patient progressed to brain death early in his post-operative course.

Discussion

Our experience of poor outcomes in all four patients treated by unilateral suboccipital decompressive craniectomy is consistent with similar observations in the literature. This led us to critically review the literature with attention to analysis of the treatment and outcome of patients with traumatic posterior fossa SDHs and to propose a treatment algorithm for these lesions.

Patients with posterior fossa SDH usually have a history of trauma to the occipital region. Most patients with posterior fossa SDH exhibit an occipital fracture suggesting a direct impact [4, 9, 29]. Just one of our patients had no fractures in occipital region, but all had external signs of impact to this area. Fracture with laceration of the cerebellar cortex seems to

be the main mechanism involved in posterior fossa SDH formation [29]. In addition, posterior fossa SDHs may arise secondary to cerebellar contusional hematoma with extravasation into the subdural space, as was observed in one of our patients.

Patients with posterior fossa SDHs usually have significant depression of consciousness [4, 9, 30]. Mass effect from the SDH may compress the brainstem and cause acute hydrocephalus; consequently, the state of consciousness and respiration may suddenly deteriorate [4, 28, 29]. Borzone et al. observed that neurological deterioration occurs primarily within the first 9 h after injury [4]. In this current series, all patients were comatose when operated on, but only one patient presented a brief lucid interval (30 min) before entering a coma.

We believe that effacement of the basal cisterns, compression and shift of the fourth ventricle, associated cerebellar hematomas, and hydrocephalus are correlated with patient outcome. Karasawa et al. recorded 100 % mortality in patients with posterior fossa injury that presented with intracerebellar contusions associated with hydrocephalus [19]. In our series, all patients had a compressed fourth ventricle, with two patients requiring further operation because of persistent compression (patient 1) and hydrocephalus (patients 1 and 2). Postoperative CT scan is rarely discussed in the literature in the context of outcome. Despite prompt evacuation of the posterior fossa SDH, all patients with postoperative head CT available exhibited persistent radiological findings of mass effect and developed cerebellar hypodense lesions suggesting ischemia and/or edema.

Treatment

Conservative management is generally accepted in patients who are not in a coma and have a hematoma thickness of less than 1 cm [4, 9, 29]. Surgical evacuation should be considered in patients having a subdural hematoma thickness of more than 1 cm or signs of mass effect as compression of fourth ventricle, obliteration of the tentorial cisterns, and tonsils in or below the foramen magnum [9, 29]. Even in patients with no

neurological deficit, surgical evacuation is prompted by radiological signs of a mass effect on the posterior fossa, such as fourth ventricle distortion, compression of the brainstem, or effacement of the surrounding basal and tentorial cisterns [6, 15]. Regarding the surgical technique, we found that suboccipital craniectomy with hematoma evacuation was the treatment most commonly performed; however, the use of a single burr hole was described as an option in selected cases in which patients exhibited unstable vital signs [22].

Outcome

In contrast to the prognosis of posterior fossa epidural hematomas [3, 20], the outcome of posterior fossa SDH is frequently unfavorable, with a mortality range of 33 %–100 % [4, 9, 30]. The main prognostic factor in patients with posterior fossa SDH is the clinical condition of the patient at the time of surgery [9, 12]. Patients exhibiting acute subdural hematoma in the posterior cranial fossa and who were alert or only had mild disturbance of consciousness prior to surgery, usually achieve a good recovery [8, 28, 30, 32]. Multivariate analysis in a study by d'Avella et al. showed that GCS admission scores were an independent prognostic factor [9]. For instance, the risk of a poor outcome was 19 times greater in patients with GCS scores of less than 8 than in those with scores of 8 or more [9]. Consistent with the report of d'Avella, all of our patients presented with severe TBI prior to surgical decompression and outcome was poor in all cases.

Cerebellar mass effect

The presence of cerebellar mass effect can lead to brainstem compression and coma. The main issue is that most patients sustaining posterior fossa SDH are comatose, making clinical evaluation challenging. As many patients can have associated supratentorial lesions and hydrocephalus, clinical signs of brainstem compression are often nonspecific and difficult to elucidate. A Babinski sign or diminished/absent corneal reflex may raise clinical suspicion of brainstem compression. Turning to the radiological signs of posterior fossa mass effect, reliable findings include compression or effacement of the fourth ventricle and ambient cisterns. Concomitantly, hydrocephalus is usually also present. For didactic purposes, we considered just the radiological signs as criteria for "signs of mass effect".

Treatment algorithm

The morbidity and mortality of posterior fossa SDHs remains high and there are no guidelines for treatment, specific to these lesions. Based on our experience and review of the literature, we propose an algorithm for the management of posterior fossa SDHs (Fig. 3). We believe that treatment of posterior

fossa acute SDHs should be guided by the clinical and radiological findings. We further believe that comatose patients or those exhibiting signs of mass effect on CT scan should undergo a bilateral decompressive craniectomy. This surgery should be incorporated into the routine management of patients with severe injury to the posterior fossa. In contrast, we cannot infer for sure that an earlier bilateral decompressive would bring further benefit. However, findings of persistent cerebellar edema in postoperative CT scans (three out of four patients) despite prompt hematoma evacuation led us to think that gaining much additional space should be the goal as the primary surgery, due to the possibility of brain swelling. In this setting, cerebellar stroke could be used as a model. Although still a matter of debate [16, 18], most of cerebellar stroke surgical protocols include bilateral decompression, with or without ventriculostomy placement, as the primary surgery [1, 7, 27] even in cases of unilateral infarct. The C1 laminectomy to optimize cerebellar decompression can be included according to the surgeon's preference [2, 16].

Most authors agree that a conservative treatment should be adopted in patients with posterior fossa SDH of thickness less than 10 mm [4, 9, 29]. In one study, four of six patients with hematoma thickness less than 10 mm and with GCS more than 8 had spontaneous resorption of their hematoma [9]. All had good outcomes. In three series it was possible to measure the thickness of the SDHs [4, 9, 30]. Together with the current study, only 6 (25 %) out of 24 patients sustaining SDH thickness greater or equal to 10 mm were not in coma and, just two patients (8,3 %) were not submitted to surgery [4, 9, 30]. 25 % of these patients had favourable outcome. Therefore, although the whole picture of the patient must be taken into account, we suggest 10 mm and signs of mass effect on CT scan as the critical thresholds for surgery.

Due to the possibility of abrupt neurological deterioration, and based on this current series, we recommend that patients who are not referred to surgery, be monitored in the ICU. Head-up position, judicious analgesia and a mean arterial pressure maintained close to normal levels are the goals for the clinical treatment in awake patients. A second CT scan should be performed within 6 h or if clinical worsening occurs. Comatose patients should have their ICP monitored and treated according to the current guidelines.

Currently, surgical recommendations for posterior fossa mass lesions are mass effect on CT scan or neurological dysfunction or deterioration referable to the lesion [5]. Regarding the use of suboccipital craniotomy or craniectomy after subdural hematoma evacuation, there is no optimal surgical technique [16, 18]. In cases of supratentorial acute SDH, the decision of whether to replace the bone is based on intraoperative findings, preoperative GCS, values of intracranial pressure monitoring, and radiological findings suggesting swelling [14, 21]. Such criteria are not developed for posterior fossa SDHs, and most authors agree that craniectomy, instead of craniotomy, is the

Fig. 3 Recommended treatment algorithm for patients with traumatic posterior fossa subdural hematoma. *SDH* subdural; *GCS* Glasgow coma scale; *ICU* Intensive care unit; *MAP* mean arterial pressure; *ICP* intracranial pressure; signs of mass effect: fourth ventricle compressed or completely effaced, basal cistern compressed or absent, or hydrocephalus. *Consider insertion of external ventricular drainage when hydrocephalus is present
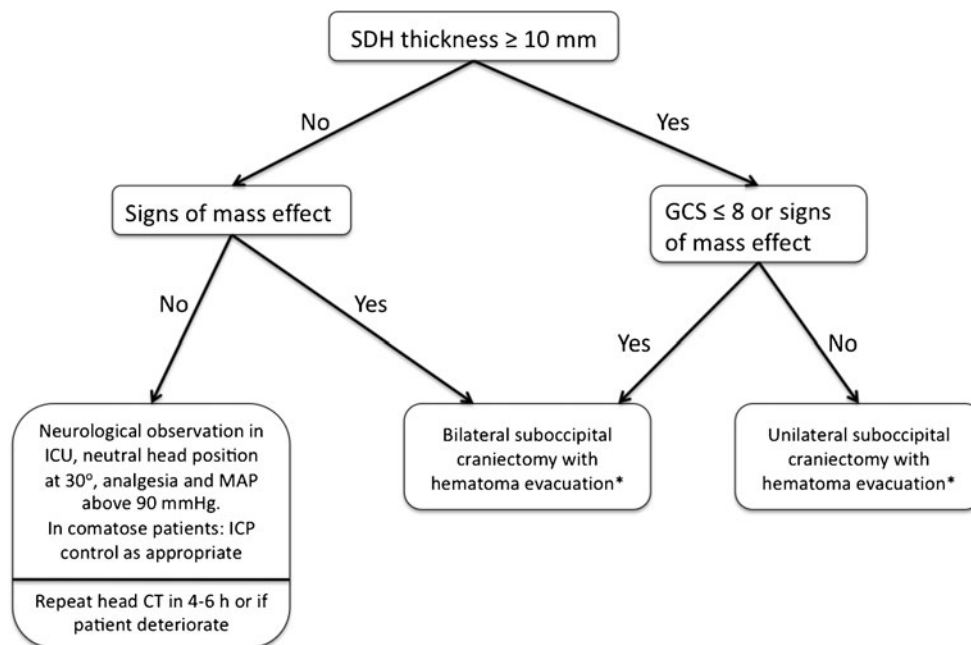
safer surgical treatment. The argument is that the posterior fossa has less room to accommodate any postoperative oozing, bleeding, or swelling [5]. Moreover, cosmetic issues are not a major problem in this context. There were no reports of complications regarding the use of suboccipital craniectomy in the literature studies we analyzed. However, posterior fossa dural reconstruction can be challenging. Some authors report a high incidence of postoperative cerebrospinal fluid (CSF) leak, pseudomeningocele, and wound reclosures when comparing suboccipital craniectomy to craniotomy [24]. Also, hydrocephalus is a frequent finding in patients with posterior fossa SDHs [8, 29], which increases the risk of CSF leak [23, 25]. Therefore, autologous or collagen matrix duraplasty with a multilayered closure, together with a low threshold for extraventricular drainage of CSF, is recommended during surgery for posterior fossa SDH evacuation.

Our literature review found that the majority of patients with posterior fossa SDHs were treated with a suboccipital craniectomy, but the investigators did not specify whether the decompression was unilateral or bilateral, or if the arch of the first vertebra was removed or not [4, 9, 13, 29, 30]. Patients not in coma and without signs of mass effect, but wherein the size of the SDH meets threshold criteria for evacuation, may undergo a unilateral decompression (Fig. 3). In the current series, all patients were submitted to unilateral craniectomy and, those who had postoperative CT scan available, all exhibited sustained signs of mass effect and hypodensity lesions in postoperative CT scans. We believe that swelling and/or ischemic lesions of the cerebellum, which may lead to hydrocephalus and compression of the brain stem, play a crucial role in patient prognosis. Moreover, ICP monitoring of posterior fossa mass lesions is challenging, and even

conscious patients can suddenly deteriorate [31]. Taking these into account, we believe that the primary surgical procedure should be a bilateral decompressive craniectomy in all patients with a GCS score less than 9 or if the initial CT scan already depicts signs of posterior fossa mass effect. As discussed above, patients may develop cerebellar swelling or ischemic damage, which may lead to persistent brain stem compression. Therefore, for comatose patients (GCS ≤ 8) with signs of mass effect on CT, we propose that a rational initial approach should entail a bilateral decompressive craniectomy (DC) with duraplasty such as that performed for posterior fossa ischemic stroke [6]. Additionally, the resection of the arch of the first vertebra may be performed at the surgeon's discretion and is recommended to optimize decompression if tonsillar herniation is apparent [2, 16]. Also, the insertion of an external ventricular drainage should always be considered in cases of hydrocephalus.

Limitations of the study

A number of caveats need to be noted regarding the present study. It is a retrospective study from a single center with a small number of patients. All patients were operated in coma and there was no control group for comparison. Moreover, CTA or other vascular study was not performed in all patients to rule out arterial ischemia or venous vascular injury due to sinus thrombosis. Also, no patients were submitted to bilateral suboccipital craniectomy as the primary surgery to support the hypothesis that this approach can avoid postoperative hypodensities, brainstem compression, and improve outcome. Moreover, few cases in the literature describe whether the

decompression was unilateral or bilateral. Therefore, our data and data from the literature do not provide sufficient evidence to establish an optimal treatment strategy in this setting. Additionally, the proposed algorithm is based exclusively on literature evidence from case reports and case series. Even with the limitations of these small studies, the literature suggests that rapid hematoma evacuation may be beneficial for patients sustaining hematomas greater than 10 mm in thickness or if there are signs of mass effect on CT scan. Finally, given the low incidence of posterior fossa SDHs, only a coordinated multicentric effort can definitely establish the best standard of care for these rare traumatic lesions.

Conflicts of interest None.

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