

Original Article

Emergency decompressive craniectomy after removal of convexity meningiomas

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Abstract

Background: Convexity meningiomas are benign brain tumors that are amenable to complete surgical resection and are associated with a low complication rate. The aim of this study was to identify factors that result in acute postoperative neurological worsening after the removal of convexity meningiomas.

Methods: Clinical evaluation and neuroradiological analysis of patients who underwent removal of a supratentorial convexity meningioma were reviewed. Patients were selected when their postoperative course was complicated by acute neurological deterioration requiring decompressive craniectomy.

Results: Six patients (mean age: 43.3 years) underwent surgical removal of a supratentorial convexity meningioma. Brain shift (mean: 9.9 mm) was evident on preoperative imaging due to lesions of varying size and perilesional edema. At various times postoperatively, patient consciousness worsened (up to decerebrate posture) with contralateral paresis and pupillary anisocoria. Computed tomography revealed no postoperative hematoma, however, did indicate increased brain edema and ventricular shift (mean: 12 mm). Emergency decompressive craniectomy and brief ventilator assistance were performed in all patients. Ischemia of the ipsilateral posterior cerebral artery occurred in 3 patients and hydrocephalus occurred in 2 patients. Outcome was good in 2, fair in 2, 1 patient had severe disability, and 1 patient died after 8 months.

Conclusions: Brain shift on preoperative imaging is a substantial risk factor for postoperative neurological worsening in young adult patients after the removal of convexity meningiomas. Emergency decompressive craniectomy must be considered because it is effective in most cases. Other than consciousness impairment, there is no reliable clinical landmark to guide the decision to perform decompressive craniectomy; however, brain ischemia may have already occurred.

Key Words: Brain, craniectomy, meningioma, shift, ventricular system

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INTRODUCTION

Decompressive craniectomy is a function and life-saving procedure for patients with severe head injury, hemorrhagic or malignant ischemic stroke, and infectious diseases of the brain. These pathological events may lead to intracranial hypertension, brain swelling, and possible secondary diffuse ischemic damage that could cause brain death. Convexity meningiomas are a group of benign brain tumors that are amenable to complete surgical resection and are associated with a low complication rate.^[1,5,9,10] However, in some cases, the postoperative course can be complicated by serious brain swelling.^[3,7,8] Here, we report a group of patients in which severe brain edema after surgical removal of a convexity meningioma was responsible for acute neurological deterioration that resolved after emergency decompressive craniectomy.

PATIENTS AND METHODS

Between January 2010 and April 2015, 123 consecutive patients underwent resection of a supratentorial convexity meningioma in our institutions. In six cases (4.8%), the postoperative course was complicated by acute or fast neurological decline that was unresponsive to maximized corticosteroid and osmotic therapies: Betamethasone 4 mg IV, mannitol 18% IV, 100 ml three times and six times daily, respectively. Patients in whom ischemia occurred as an intraoperative vessel injury were excluded from subsequent analysis. Preoperative and postoperative clinical courses as well as brain imaging were analyzed. Patients provided written informed consent to the surgical procedure and to the eventual publication of any accompanying image.

RESULTS

The 6 selected patients underwent surgical removal of a convexity meningioma (mean age: 43.3 years)

[Table 1]. Symptoms included progressive headache ($n = 6$), faciobrachial hemiparesis ($n = 2$), altered consciousness ($n = 2$), gait unsteadiness ($n = 2$), and blurred vision ($n = 1$). One patient harbored contemporary multiple infratentorial and supratentorial cavernous angiomas, one of which was responsible for a previous cerebellar hemorrhage. In 3 patients, the lesion was located in the frontotemporal region and in the other 3 patients it was in the frontal convexity [Figure 1]. The lesion was small (≤ 2 cm) in 1 patient, medium-sized (≤ 5 cm) in 2 patients, and large (≥ 5 cm) in 3 patients. In all cases, brain edema was evident on preoperative magnetic resonance imaging and was responsible for varying degrees of brain shift (mean shift: 9.9 mm). Frontal ($n = 3$) and frontotemporal ($n = 3$) approaches were performed, including the dural edges into which the meningiomas extended. There were no reports of vascular injury intraoperatively, and venous outflow around the tumor was carefully preserved.

In the awakening phase, 1 patient displayed decerebrate posture and homolateral anisocoria, which prompted immediate reopening of the craniotomy site. Due to serious brain swelling, the edges of the craniotomy were enlarged and duraplasty was carried out. While the other 5 patients were under corticosteroid and osmotic therapies, fast progressive neurological impairment

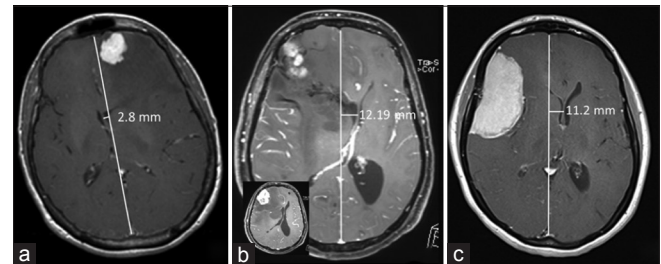


Figure 1: Magnetic resonance imaging of three patients with convexity meningioma and brain shift (a-c). Lesions vary in size; even small lesions can cause substantial brain shifts

Table 1: Patient Characteristics

Sex	Age (years)	Location	Preoperative midline shift (mm)	Postoperative midline shift (mm)	Hours between neurological decline and decompressive craniectomy	Midline shift (mm) after decompressive craniectomy	Complication related to intracranial hypertension	Outcome (Glasgow Outcome Scale-E xtended)
M	52	frontotemporal	17.74	17.92	48	6.4	Hydrocephalus	Death after 8 months
F	44	frontotemporal	11.21	14.18	6	3.3	Posterior cerebral artery ischemia	7
M	49	frontotemporal	3.0	9.4	72	2.5	None	8
M	28	frontal	12.50	Not applicable	0	0	None	8
M	46	frontal	12.19	12.43	48	6.6	Posterior cerebral artery ischemia, hydrocephalus	5
F	41	frontal	2.8	6.1	48	0	Posterior cerebral artery ischemia	7

(worsening of consciousness and decerebrate posture in 1 patient, worsening of consciousness with contralateral motor impairment in 4 patients, and homolateral anisocoria in 3 patients) that was unresponsive to maximized medical therapies occurred 6–72 h after surgery (mean time to occurrence: 37 h). Computed tomography (CT) indicated that brain shift had increased by varying degrees in all 5 patients (mean shift: 12 mm) due to brain edema; no postoperative hematomas were evident, however, small intracavitary blood collection occurred in 2 patients, presumably as a secondary effect of the hindrance of venous outflow due to high intracranial pressure [Figure 2]. Emergency decompressive craniectomy with enlargement of craniotomy edges and duraplasty was accomplished followed by a short period of ventilatory assistance (24–48 h in all but 1 patient). Restoration of the pupillary isocoria was the first immediate clinical effect after decompressive craniectomy. Early postoperative CT demonstrated a substantial reduction in brain shift in all patients (mean: 3.1 mm), however, ipsilateral posterior cerebral artery ischemia was present in three patients [Figure 3]. After several days, hydrocephalus was identified in 2 patients, who subsequently received a ventriculoperitoneal shunt. Histological examination indicated meningothelial meningioma (World Health Organization grade I) in all patients.

Cranioplasty was performed in all patients at 3–16 weeks (mean: 8 weeks) without complications. The 90-day mortality rate was 0%, however, 1 patient died after 8 months due to infectious disease and multiple organ failure. In other patients, at a mean follow-up of 14 months, the Glasgow Outcome Scores-Extended ranged from 5 to 8.

DISCUSSION

Serious neurological worsening can occur early after the surgical removal of a benign convexity meningioma. However, this complication has not received much

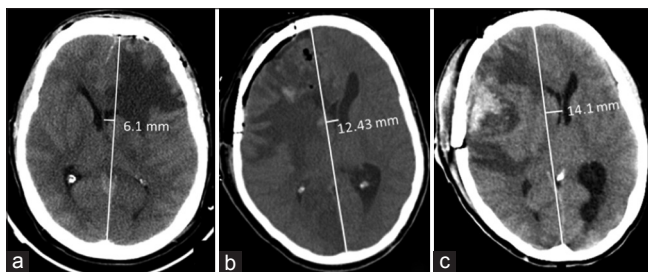


Figure 2: Emergent postoperative computed tomography revealed increased ventricular shift. Patients are shown left to right, as in Figure 1 (a-c). Small intracavitary blood collection was evident, presumably due to the hindrance of venous outflow for high intracranial pressure (c). Note the posterior cerebral ischemia homolateral to the surgical territory in the center case (b)

attention in the literature. Here, clinical impairment in the early postoperative course was unexpected (in 1 patient it occurred immediately after surgical treatment), insidious, and very serious. It is plausible that surgical treatment and unwanted concomitant retraction injury presumably exacerbated pre-existing brain edema and ventricular shift, as demonstrated by control CT in the present investigation.

The preoperative mean midline shift in additional 111 preoperative images available in our retrospective study of convexity meningiomas was 6.2 mm and in the postoperative period was 7.3 mm. Although these results are not statistically significant, patients who experienced diminished consciousness had mean preoperative and postoperative midline shifts that were greater than that for patients who showed a normal postoperative clinical course.

Venous injury during surgical resection and postoperative hematoma were excluded. A surgeon should be aware that this fast neurological worsening is not related to the size of the meningioma because even a small convexity meningioma with marked edema can elicit this treacherous complication.

Clinical decline 24–48 h after uneventful meningioma resection is mentioned for lesions of the posterior cranial fossa.^[2] Patients become progressively sleepier and CT reveals brain edema that fills the residual space from the previous meningioma. In these patients, increasing osmotic and corticosteroid therapy alone can be successful. In some cases, endotracheal reintubation and ventilatory assistance should be employed to counteract further brain swelling caused by breathing difficulties, however, this therapeutic choice may be hindered by intensive-care complications.

Here, our decision to remove the bone flap and to enlarge the bone edges for decompressive purposes appeared natural and mandatory in order to reverse the severe neurological worsening and marked ventricular shift on imaging, as is employed in patients suffering stroke or brain injury. An embarrassing dilemma is the lack of a

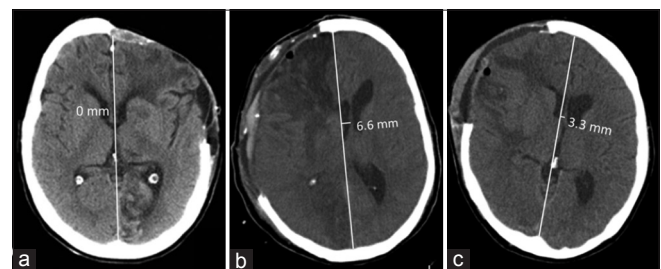


Figure 3: Computed tomography of the patients in Figures 1-2 early after emergency decompressive craniectomy. Ventricular shift is reduced (b,c) or resolved (a), although all cases exhibit posterior cerebral ischemia homolateral to the surgical territory

firm indicator, other than the level of consciousness, that surgical decompression must be accomplished because an ischemic event in the posterior circulation may have already occurred. In all but 2 patients, decompressive craniectomy was implemented without modifying the skin incision; brain swelling prompted us to enlarge the bone margins as much as possible and to add a dural patch. This surgical choice immediately normalized the patients' pupillary diameter and reduced ventricular shift on CT [Figure 3]. In the majority of our cases, these clinical and radiological improvements were followed by restoration of consciousness after a short period of ventilatory assistance.

Ischemia in the territory of the posterior cerebral artery in 3 patients caused harsh intracranial hypertension and reduced cerebral blood flow, which we counteracted with decompressive craniectomy. The young adult age of our patients is notable.

In our clinical study of 6 patients, we observed a mean age 43.3 years. The remaining 117 patients harboring a convexity meningioma, and with an uncomplicated postoperative course, had a mean age of 57.3 years (range: 29–84 years). Due to the small sample size, this difference was not statistically significant.

The removal of convexity meningiomas with pre-existing edema yielded ventricular shift up to 10 mm in these young brains; young brains are less tolerant of intracranial mass effects than aged brains. Thus, the removal of convexity meningiomas must be considered highly risky in these patients.

In a previous series of 100 patients with a parasagittal meningioma invading the superior sagittal sinus, 3 patients (3%) died, presumably due to brain swelling.^[6] In the study, the author suggested that "in the case of severe bilateral peritumoral edema ... we recommend having a plan to leave out the bone flap after tumor resection."^[6] In another previous large series of patients who underwent removal of an intracranial meningioma, 6 patients experienced postoperative deterioration due to extensive brain edema of unknown etiology.^[3] In 3 of these patients, decompressive surgery was performed with favorable results. Positive outcome was previously reported in a case of large olfactory groove meningioma in which the Glasgow Coma Score deteriorated and the pupils were dilated and fixed.^[4] After failed medical management via mannitol and hyperventilation, bifrontal decompressive craniectomy resolved the severe clinical compromise. In 1 case of atypical meningioma of the skull base, emergency large decompressive craniectomy was performed due to rapidly progressing impaired consciousness.^[12] The patient immediately recovered consciousness and 1 month later underwent radical tumor resection. The relevance of peritumoral edema and prognosis in surgery for intracranial meningioma has been

emphasized previously, and decompressive craniectomy is indicated as a successful procedure.^[3,11] Surveillance of consciousness was mandatory in our 6 cases, and when medical therapies failed to stabilize the neurological condition, prompt surgical decompression was performed.

In conclusion, young adult patients with convexity meningiomas of various sizes associated with brain edema and ventricular shift up to 10 mm can deteriorate early after surgical removal of these lesions. When medical therapy fails, emergency decompressive craniectomy must be accomplished as early as possible to save the patient's neurological functions and life.

Patient consent

We have obtained fully informed, voluntary and written consent to publish from each patient whose case is described in the article.

All authors certify that they have NO affiliations with or involvement in any organization or entity with any financial interest (such as honoraria; educational; participation in speakers' bureaus; membership, employment, consultancies, stock ownership, or other equity interest; and expert testimony or patent-licensing arrangements), or non-financial interest (such as personal or professional relationships, affiliations, knowledge, or beliefs) in the subject matter or materials discussed in this manuscript.

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Conflicts of interest

There are no conflicts of interest.

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Commentary

It has been my practice to leave the bone flap out in meningiomas, particularly if they are large and long standing, as they develop further swelling and can act as a mass. Just because it is a meningioma is not reason enough to replace the bone flap. One must think of the post-operative course, which inevitably produces edema. In large tumors, that can result in the complications you report. Usually, after leaving the flap out, the post-op course is

more normal and the flap can be replaced later. You will find on repeat CTS that the edema is slow to resolve, so it may take up to two months to replace the bone flap.

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