Hydrocephalus Following Giant Transosseous Vertex Meningioma Resection

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Abstract

Introduction Meningiomas are among the most common primary intracranial tumors. While well-described, there is limited information on the outcomes and consequences following treatment of giant-sized vertex-based meningiomas. These meningiomas have specific risks and potential complications due to their size, location, and involvement with extracalvarial soft tissue and dural sinuses. Herein, we present four giant-sized vertex transosseous meningioma cases with involvement and occlusion of the sagittal sinus, that postoperatively developed external hydrocephalus and ultimately required shunting.

Methods A retrospective chart review identified patients with large vertex meningiomas that were: (1) large (>6 cm) with hemispheric (no skull base) location, (2) involvement of the superior sagittal sinus resulting in complete sinus occlusion, (3) involvement of dura resulting in a large duraplasty area, (4) transosseous involvement requiring a 5 cm or larger craniectomy for resection of invaded calvarial bone.

Results Tumors were resected in all four cases, with all patients subsequently developing external hydrocephalus which required shunting within 2 weeks to 6 months postsurgery.

Conclusion We believe this may be the first report of the development of hydrocephalus following surgical resection of these large lesions. Based on our observations, we propose that a combination of superior sagittal sinus occlusion and changes in brain elasticity and compliance affect the brain’s CSF absorptive capacity, which ultimately lead to hydrocephalus development. We suggest that neurosurgeons be aware that postoperative hydrocephalus can quickly develop following treatment of giant-sized vertex-based meningiomas, and that correction of hydrocephalus with shunting can readily be achieved.

Keywords ► transosseous meningiomas ► hydrocephalus ► shunt ► vertex meningioma ► postoperative hydrocephalus ► tumor resection

Introduction

Meningiomas are among the common brain tumors, accounting for approximately one-third of all primary intracranial tumors.¹ Previous studies have described the wide variety of locations where these neoplasms arise, with the most common in parasagittal and cerebral convexity locations.² While usually benign, meningiomas can become highly invasive and involve the intraosseous and extracalvarial spaces. While there have been several prior reports of large transosseous and extracalvarial meningiomas, there is relatively limited discussion of the surgical repercussions from resecting these meningiomas.³⁻⁵ We have found no prior reports of postoperative hydrocephalus that required CSF diversion in this tumor population. Herein, we report a
series of four patients that presented with giant-sized (defined as greater than 6 cm) vertex transosseous meningiomas. All four patients underwent gross total resections, including resections of the involved calvarial bone, dura, and superior sagittal sinus (SSS). In all four cases, despite watertight closure of the dura with pericranium (if available) or dural substitutes, and despite temporary CSF diversion, each patient postoperatively developed significant external hydrocephalus that ultimately required shunting in the weeks or months following tumor resection (Table 1).

### Materials and Methods

A retrospective chart review was performed following approval from the University of Wisconsin Health Sciences Institutional Review Board. The criteria to identify specific meningioma case were as follows: (1) large size (>6 cm) with hemispheric location (no skull base location), (2) involvement of the SSS which resulted in complete occlusion of the sinus, (3) involvement of dura which resulted in a large duraplasty area, (4) transosseous involvement which

<table>
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<th>Case no.</th>
<th>Age (y) at time of meningioma resection</th>
<th>Pathology</th>
<th>Tumor size (cm)</th>
<th>Time to presentation of hydrocephalus following resection (mo)</th>
<th>Shunt type placed</th>
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<td>SGPS</td>
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<td>10 × 4</td>
<td>4</td>
<td>SGPS, VPS</td>
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Abbreviations: CSF, cerebrospinal fluid; EPS, epidural peritoneal shunt; SGPS, subgaleal-peritoneal shunt; VPS, ventriculoperitoneal shunt.

Note: Tumor sizes are the anterior–posterior by the rostral-caudal dimension.

Fig. 1 (A–C) Preoperative MRI imaging of Case #1 in axial, coronal, and sagittal views, respectively. (D–F) Postoperative CT scan in axial, coronal, and sagittal views demonstrating hydrocephalus. (G–I) Post-ventriculoperitoneal shunt placement CT scan in axial, coronal, and sagittal views. Note resultant improvement in external hydrocephalus denoted by the arrows. CT, computed tomography; MRI, magnetic resonance imaging.
resulted in 5 cm or greater craniectomy for resection of invaded calvarial bone.

**Results**

A total of four cases meeting the inclusion criteria were identified (Table 1).

**Case Series**

**Case 1**

A 24-year-old male presented with gradual worsening of vision in his right eye. He described experiencing a central loss of vision which gradually widened and eventually began to involve the left eye. This prompted him to be evaluated by an ophthalmologist, who found a bilateral papilledema. A brain MRI demonstrated a 10-cm dural-based, extra-axial lesion on the bifrontal vertex that invaded bone (Fig. 1A–C). The patient underwent a bilateral craniectomy with gross total resection. Of note, the SSS was occluded preoperatively and was resected along with the involved dura. The SSS was identified as being occluded during a formal preoperative cerebral angiogram. Since a significant area of the pericranium was invaded by the tumor, a water-tight duraplasty with dural substitute (sutureable DuraGen, Integra Lifesciences, Plainsboro, New Jersey, United States) was performed. Pathology demonstrated a WHO grade I meningioma. The patient’s postoperative course was complicated by seizures, which were controlled with antiepileptic medication. Postoperatively, his visual symptoms improved. He was discharged to in-patient rehabilitation. However, on postoperative day 18, he returned with a sizeable subgaleal fluid collection and severe headaches (Fig. 1D–F). The pseudomeningocele was drained multiple times with lumbar drain placement, and compressed with dressings. However, this ultimately failed. The patient was then treated by placing a ventriculoperitoneal shunt (using a programmable Medtronic Strata II valve, set to 1.0) (Fig. 1G–I), which significantly improved his symptoms and external hydrocephalus. The patient eventually had a synthetic bone cranioplasty with a KLS-Martin polyether ether ketone (PEEK) plate with no complications. He remained free of recurrence at 3-months follow-up. No further follow-up could be done due to his relocation to another state.

**Case 2**

A 70-year-old male presented with slurred speech and numbness in his left arm. He had a 5-year history of a right frontoparietal intracranial lesion with extensive involvement of the SSS and calvarial bone (Fig. 2A–C). He had

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**Fig. 2 (A–C)** Preoperative MRI imaging of Case #2 in axial, coronal, and sagittal views, respectively. (D, E) Postoperative CT scan in axial and coronal views demonstrating hydrocephalus. (F, G) Postsubgaleal peritoneal shunt placement CT scan in axial and coronal views. Note resultant improvement in hydrocephalus denoted by the arrows. CT, computed tomography; MRI, magnetic resonance imaging.
attempted to hide his lesion and delay treatment until he experienced an acute left-sided numbness and slurred speech, which led to hospital admission and ultimately treatment. The remainder of his physical examination was unremarkable. Preoperatively, he underwent endovascular embolization of arterial feeders including the right superficial temporal artery (STA), the middle meningeal artery (MMA), the occipital artery (OA), and the left STA. A formal preoperative cerebral angiogram performed in the same session as the embolization demonstrated that the tumor had obliterated the anterior half of the SSS. The patient ultimately underwent a craniectomy and gross total resection, including resection of the involved bone and dura. Duraplasty was then performed with the pericranium, and a cranioplasty was done with PEEK material. Pathology demonstrated a WHO grade II atypical meningioma. Approximately 6 months following his surgery, the patient experienced worsening weakness in all extremities. A CT of the head demonstrated a large pseudomeningocele causing brain compression (Fig. 2D–E). The patient required subgaleal-peritoneal shunt placement (using a programmable Medtronic Strata valve, set to 1.0) that ultimately improved his external hydrocephalus (Fig. 2F–G). After completing adjunctive radiation therapy, the patient remains free of recurrence over 4 years.

Case 3
This patient was a 67-year-old male who had been followed since the 1990s for a large transosseous meningioma of the vertex. The tumor was palpable and visible externally, and had been growing steadily over the past few years. The lesion was approximately 10 cm in diameter, had obliterated the SSS, and extended into the subcutaneous scalp (Fig. 3A–C). Despite knowing of the tumor for nearly a decade, the patient had previously declined surgery. A year before surgical resection, he began to experience left-sided hemiparesis which eventually progressed, requiring him to be wheelchair bound. This convinced the patient to proceed with surgery. The remainder of his physical exam at presentation for surgery consisted of slight left facial droop, three-fifths left-sided strength, and increased deep tendon reflexes. Surgery was preceded by endovascular tumor embolization via the right MMA, STA, OA, and left STA. A formal preoperative cerebral angiogram also confirmed SSS occlusion in the middle third. The patient eventually underwent bilateral frontoparietal craniectomies for gross total tumor resection. Also, the involved dura, bone, falx, and SSS were resected. A large duraplasty was performed using a dural substitute, since pericranium was involved with the tumor. Pathology revealed a WHO grade II atypical meningioma. Approximately 3 months after cranioplasty, the patient developed a

Fig. 3  (A–C) MRI imaging of Case #3 in axial, coronal, and sagittal views, respectively. (D–F) Postoperative MRI imaging demonstrating development of external hydrocephalus with significant subgaleal component. (G–I) Postsubgaleal to peritoneal shunt placement MRI imaging. Note resultant improvement in hydrocephalus denoted by the arrows. CT, computed tomography; MRI, magnetic resonance imaging.
sizeable subgaleal fluid collection around his operative site (►Fig. 3D–F). Nonpermanent CSF diversion via a lumbar drain was attempted but failed. Despite intensive drainage, the fluid continued to re-accumulate. Ultimately, the patient required a subgaleal-peritoneal shunt (using a programmable Medtronic Strata valve, set to 0.5) to be placed, which resulted in improved external hydrocephalus (►Fig. 3G–I). The patient declined to receive any adjunctive treatment after surgery. He returned to the clinic with the exposed shunt system, which was then removed. Although he was advised to return for shunt replacement after completing his antibiotic course he became lost to follow-up. The patient has very recently seen his primary care provider in clinic and has a recurrence of his external hydrocephalus with evidence of a CSF leak. Despite reaching out to the patient and his primary care provider, the patient is declining all further neurosurgical interventions.

**Case 4**

A 54-year-old male presented to the emergency department with headaches of unknown cause. These headaches had persisted for approximately 3 months and were associated with swelling over the right forehead. His presenting neurological exam was intact with full strength, normal sensation, and normal reflexes throughout. MRI of the brain was performed and demonstrated a large dural-based transosseous mass that was most likely consistent with a meningioma (►Fig. 4A–C). A formal preoperative cerebral angiogram was performed and identified occlusion of the SSS. He ultimately underwent bifrontoparietal craniectomy for gross total resection. The involved dura and SSS were resected as well. Pathology revealed a WHO grade II atypical meningioma. The patient had a cranioplasty procedure at 6 weeks after surgery. Approximately 4 months postoperatively, he developed hydrocephalus with a large subgaleal component (►Fig. 4D–F). He had no new neurological symptoms from this hydrocephalus, although he had developed fluid collection that was both large and sufficiently superficial to show cutaneous signs. He then underwent a subgaleal to peritoneal shunt (using a programmable Medtronic Strata valve, set to 1.5), which led to a significant improvement in his hydrocephalus (►Fig. 4G–H). However, several months after this shunting procedure, the patient returned with re-accumulation of the pseudomeningocele and hydrocephalus. As a result, he underwent a shunt revision and conversion of his shunt to a ventriculoperitoneal shunt, which ultimately improved his hydrocephalus. The patient declined to have any adjunctive treatment for grade II meningioma. No tumor recurrence was observed at 3-year follow-up.
Discussion

Since Cushing’s groundbreaking paper in 1938, much more information regarding meningiomas has come to light. However, there have only been limited case reports regarding the subset of large, vertex-based, meningiomas. In the present work, we add to this limited literature by presenting a series of four cases of large, vertex-based, meningiomas. To the best of our knowledge, this may be the first discussion of the development of external hydrocephalus following surgical resection of these large lesions.

In this case series, we describe a variety of patient and tumor demographics. Key differences among the patients that give this series some generalizability include the wide age range (24–70 years), embolization preoperatively versus no embolization (half the tumors were embolized prior to resection), and both WHO grade I and grade II meningiomas. Additionally, the shunt type placed to treat the hydrocephalus varied among the patients. Each of these factors helps support the argument that postoperative hydrocephalus has the potential to affect any large, vertex-based, meningioma. It should be noted that all cases involved in this series were male, thus somewhat limiting our ability to extrapolate this information, and that there were only four cases.

External hydrocephalus has been primarily described in the pediatric population with few reports arguing its significance in adults. These reports suggest that external hydrocephalus in adults presents with symptoms similar to either acute or normal pressure hydrocephalus, but do so in a situation where the ventricles are resistant to dilatation. Additionally, they suggest that external hydrocephalus, while appearing similar to subdural hygromas, typically causes brain compression, and resolves with CSF shunting (whereas benign extra-axial fluid collections will worsen with shunting). Given this (albeit limited) criteria, we argue that our patients also fit in this classification of adult external hydrocephalus, due to the observations of brain compression, resolution with shunting, and postoperative symptomology of high pressure.

In each of the four cases we describe, there were no apparent obstructive sources for a hydrocephalus. Furthermore, these lesions were not involved nor close to the ventricular system. As stated previously, we are aware of no prior reports of postoperative hydrocephalus development in giant-sized vertex transosseous meningioma resections. Thus, determining the circumstances behind hydrocephalus development in this patient population requires extrapolating to other clinical scenarios. Skull base meningioma resections, hemispherectomies, decompressive craniectomies in the setting of traumatic brain injury or stroke, and SSS thrombosis, have similarities to our patient population. Thus, studying the pathophysiology of hydrocephalus development in these scenarios may help elucidate the cause in the present case series.

Postoperative hydrocephalus following the resection of skull base meningiomas has been previously described. Excluding obvious compressive-obstructive sources, there are a few hypotheses on why hydrocephalus occurs following skull base meningioma surgery. It is known that the vast majority of CSF absorption occurs at the level of the arachnoid villi. The microscopic nature and pressure dependence of these spaces potentially place these arachnoid villi at risk for scarring or “clogging” following an infection, or from diffuse subarachnoid debris. Most of the literature concerning the development of hydrocephalus following skull base meningioma resection argues that the spillage of blood products into CSF cisterns, scarring of cisterns (from both surgery and postoperative radiation), and postoperative CSF infections, all critically affect the arachnoid villi and decrease their ability to re-absorb CSF. This, in turn, causes absorptive hydrocephalus. Fortunately, none of our patients had postoperative CSF infections. The meningiomas presented in the present case series were extremely vascular, with two out of four requiring preoperative embolization. As a result, it seems plausible that high blood spillage and subsequent “clogging” of arachnoid villi from these large, vascular vertex meningiomas could have contributed to the postoperative development of hydrocephalus. This is likely, at least in part, for the circumstances surrounding the large vertex transosseous meningiomas described herein.

Outside of skull base-related pathologies, hemispherectomy surgery is another area that could be correlated with large, vertex-based, meningioma resections. This comparison can be made since there are similarities in the tissue volume removed and the surgical techniques and approaches. Postoperative hydrocephalus in the setting of hemispherectomies has been previously well-described. Once again, the concept of a large amount of blood leakage into the cisterns is one theory for hydrocephalus development. However, in hemispherectomies, much more tissue is resected than in typical skull base meningiomas. As a result, Lew et al argue that there may be a component of hydrocephalus development related to the sheer volume of tissue resected. Specifically, they suggest that resecting a large section of parenchyma may lead to an effect on brain elasticity and compliance which may ultimately affect the brain’s CSF absorptive capacity. Obviously in meningioma resection, the goal is not to resect brain parenchyma itself; however, removing such large, space-occupying lesions could easily affect parenchyma compliance and thus its absorptive capacity.

Postoperative hydrocephalus following decompressive craniectomy for either traumatic brain injury or stroke is a well-described phenomenon. In each of the cases we presented, the vast amount of transosseous involvement required a large amount of the skull to be permanently removed. This creates a scenario very similar to decompressive craniectomies. While no definitive answer as to why hydrocephalus develops following decompressive craniectomies exists, many hypotheses offer suggestions as to its cause. First, the previously discussed phenomenon of arachnoid villi scarring or “clogging” from blood products in surgery is frequently mentioned in this literature. Another argument from decompressive craniectomy hydrocephalus development suggests that large cranial defects affect the CSF and blood pressure pulsation transmission throughout the cranial, and that this alteration (specifically the dampened effect of the pressure waves given the open vault) disturbs the pressure-dependent absorption into the arachnoid villi and can, therefore, lead to
decreased CSF outflow. This is supported by the observation that larger decompression craniectomies (and therefore more significant changes in CSF and blood pressure pulsation transmission) correlate with a higher rate of postoperative hydrocephalus. If this is true, then cranioplasty placement should help improve CSF dynamics, and therefore decrease hydrocephalus. This too has been demonstrated. However, it is unclear why cranioplasty did not improve the hydrocephalus in our patient series. It is possible that the multiple other variables we discussed, including the occlusion of the SSS, and spillage of blood products into the subarachnoid space, had effects that were too large for a simple cranioplasty to fix, and thus ultimately demanded CSF shunting.

Three of the four patients presented here developed hydrocephalus after cranioplasty, which suggests that there is likely another component to CSF accumulation following the resection of giant-sized vertex transosseous meningiomas. Each of the cases presented here also required the resection of the already occluded SSS. As previously discussed, arachnoid villi reabsorption occurs primarily along the SSS. Additionally, there are reports of communicating hydrocephalus development in acute SSS thrombosis. The patients presented in this manuscript did not develop hydrocephalus preoperatively despite occlusion of the SSS. We would argue that this is secondary to the chronic nature of this occlusion, such that the cranium had time to adapt to the slow changes in CSF dynamics from this occlusion caused by the tumor. However, given the chronic occlusion, there is likely increased potential for the development of hydrocephalus given the decreased absorptive capacity of the SSS. We could thus argue that the resection of segments of the SSS likely changed the CSF reabsorption capacity just enough that this ultimately leads to hydrocephalus. Furthermore, it has been previously suggested that large tumor and tumor capsule veins may actually provide a source of flow between the proximal and distal sinus. We therefore argue that resection of these tumor veins could similarly disrupt venous flow and further potentiate CSF accumulation. Ultimately, we suspect that the development of hydrocephalus postoperatively could be in part related to the acute resection of the SSS.

In each of the described cases, a shunt was placed to divert CSF and improve the external hydrocephalus. Two of the shunts (the ventriculoperitoneal and one of the subgaleal-peritoneal shunts) continued to work in the patients without any complications. The other two shunts (both subgaleal-peritoneal) required re-operation and removal. However, only one of these was truly due to a shunt failure. The shunt in case #4 did ultimately fail and require removal, followed by ventriculoperitoneal shunt placement. The shunt in case #3 did not technically fail. The patient in this case had extremely fragile skin and the wound near the valve broke down, exposing the hardware underneath. This resulted in wound and shunt infection, which prompted shunt removal. The shunt was still functional at the time of removal, as there was no evidence of external hydrocephalus or pseudomeningocele at that time. Given these outcomes, we feel that it is reasonable to continue considering placement of subgaleal-peritoneal shunts. This allows for direct diversion of the accumulated fluid from the affected compartment and does not have the risks associated with intraventricular placement. However, a ventriculoperitoneal shunt could still be considered in these situations as this would still divert CSF and result in improvement in the external hydrocephalus.

**Conclusion**

In the case series presented here, we observed that postoperative hydrocephalus developed following the resection of giant-sized vertex transosseous meningiomas. Each patient developed the hydrocephalus without preceding infection or radiation. Based on our observations, experience, and literature review, we propose that the brain’s CSF absorptive capacity was altered, and this altered absorptive capacity leads to hydrocephalus formation via a combination of SSS occlusion/resection, spillage of blood into CSF cisterns, and the subsequent scarring of arachnoid villi, and changes in brain elasticity/compliance due to large craniectomy defects. More research needs to be performed concerning this phenomenon and how it affects the resection of large vertex-based meningiomas. Until that time, we can only extrapolate from observations and studies based on other procedures. Regardless, neurosurgeons need to be aware that this postoperative course can quickly develop in this tumor population, and that correction of hydrocephalus with shunting can readily be achieved.

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**Conflict of Interest**

None declared.

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**References**

20 Chakor RT, Jakhare S, Gavai BY, Santhosh NS. Communicating hydrocephalus due to cerebral venous sinus thrombosis treated with ventriculoperitoneal shunt. Ann Indian Acad Neurol 2012;15(04):326–328