Olfactory Groove Meningiomas: Acute Presentation and Potential

Pitfalls in Management and Functional Restoration

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Introduction

Meningiomas are typically benign, slow-growing lesions that present after an insidious onset of symptoms related to mass effect. The acute presentation of a patient who has suffered a transtentorial herniation event due to a meningioma is rare. There are only few publications describing such a presentation in the absence of hemorrhage [1]. In this case report, a patient with an olfactory groove meningioma presenting with signs and symptoms of transtentorial herniation in the absence of tumor-associated hemorrhage is discussed. This is a unique presentation of such a lesion. The patient developed Anton's syndrome—binocular visual loss with blindness denial. Management considerations for patients with meningiomas that present with acute deterioration are discussed.

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Patient History and Presentation

Preoperative Course

The patient is a 53-year-old female who was neurologically intact until 2 weeks prior to her acute decline. At this time she noticed deterioration of her visual acuity and obscured visual fields. Concurrently she developed dull headaches exacerbated by recumbency and valsalva. These symptoms brought her to an emergency room at another hospital, where imaging revealed a midline anterior cranial fossa mass with bifrontal edema. The patient was discharged home on Decadron (dexamethasone; Merck) with the intention for surgical intervention within the following 2 weeks. Four days after the initial diagnosis, she was found unresponsive in her house and was emergently transferred to our hospital.

On admission to our neurosciences critical care unit the patient was intubated for airway protection, her Glasgow Coma Scale score was 3 T. On examination, the patient was not able to open her eyes, had bilaterally dilated and fixed pupils, intact corneal reflexes, intact cough/gag reflex, and extensor posturing to stimuli. An admission head computed tomography (CT) scan demonstrated a 4 × 4 cm midline anterior cranial fossa mass with bifrontal edema, effaced lateral ventricles, and basilar cisterns. Medical interventions to lower intracranial pressure and cerebral edema were initiated, including hyperventilation, dexamethasone (20 mg q 4 h) and hyperosmolar therapy (23.4 % boluses and 3 % hypertonic saline infusion). Shortly after the initiation of medical management, including seizure medication, the patient's clinical examination improved. She now had spontaneous eye opening, dilated but minimally reactive pupils, and spontaneous, purposeful movements with the upper and lower extremities.

Once the patient was stable, gadolinium-enhanced magnetic resonance imaging (MRI) of the brain was obtained. The MRI demonstrated a homogenously enhancing mass

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most consistent with an olfactory groove meningioma (Fig. 1b). In addition, although bilateral occipital lobe infarcts were evident (Fig. 1c), MRI did not reveal evidence of brain stem injury. The presence of bilateral posterior cerebral artery infarcts was attributed to the patient's transtentorial herniation.

Operative Course

Subsequently she was taken to the operating room for an extended bifrontal craniotomy and surgical tumor resection. A gross total resection of the lesion was achieved (Simpson Grade II). Tumor-associated hemorrhage was not observed. Pathology was consistent with a meningioma.

Postoperative Course

Postoperative imaging confirmed a gross total resection with resolving bilateral frontal edema (Fig. 1d). Final pathology was a WHO grade I meningioma. At follow-up at 2 weeks and 3 months, the patient was awake and alert, cognitively intact, and with full strength in all extremities. Despite having no light perception bilaterally, she continued to deny visual loss.

Discussion

Meningiomas are typically slow-growing, extra-axial tumors that present with a gradual onset of symptoms related to mass effect. The acute presentation of a patient harboring a meningioma is rare and is almost always associated with hemorrhage [2–13] and sometimes with new onset of seizures. The hemorrhage may be intratumoral, intracerebral, subdural, or subarachnoid [2–13]. It has been proposed that extensive tumor infarction may precipitate tumor-associated hemorrhage and the acute clinical decline of such patients [2]. In this case report, we describe a patient with an olfactory groove meningioma who presented with the signs and symptoms of a herniation syndrome in the absence of tumor-associated hemorrhage. This is a unique presentation for an anterior skull base meningioma

A possible mechanism of deterioration in this patient is the acute development of central tumor necrosis. This could have precipitated an abrupt increase in cerebral edema and intracranial pressure leading to the herniation syndrome observed. On preoperative CT and MR imaging, the presence of significant bifrontal vasogenic edema was evident. Central tumor necrosis has been considered the cause of an acute decline in a previously reported patient with a meningioma who did not, though, progress to herniation [1].

Another plausible mechanism of deterioration includes a seizure event. The degree of mass effect related to cerebral edema could have placed the patient in a critical threshold, where a single seizure (with consequent hypoventilation/hypercarbia) could precipitate catastrophic herniation. This hypothesis raises the importance of prophylactic antiepileptic medications in patients with meningiomas associated with surrounding edema.

A management nuance highlighted by this case is the timing of surgery once a patient harboring a meningioma acutely deteriorates. In this case, the patient was immediately placed on hyperosmolar therapy and steroids to mitigate the effects of increased intracranial pressure. Fortunately, she had a clinical response to this treatment and the decision was made to continue medical therapy for an additional 36 h prior to surgery. In situations where a clinical response to aggressive medical intervention does not occur and concerns for elevated intracranial pressure persist, urgent surgical decompression should be considered. For patients with meningiomas and extensive parenchymal edema, the paradigm for surgical intervention would be similar to a protocol for intractable intracranial pressure, where urgent intervention is considered when a poor clinical examination is refractory to medical therapy.

Interestingly, although the patient had no light perception on follow-up examination and had MRI evidence of bilateral occipital infarcts, she denied any visual impairment. Anton-Babinski syndrome or Anton's blindness, where the patient is blind but refuses to acknowledge the condition, is generally the result of trauma or stroke where damage has occurred to the bilateral occipital lobes, resulting in cortical blindness [14]. Although a previous report has associated an anterior skull base meningioma with the syndrome, our case is unique in regard to the acute presentation of a herniation syndrome [15].

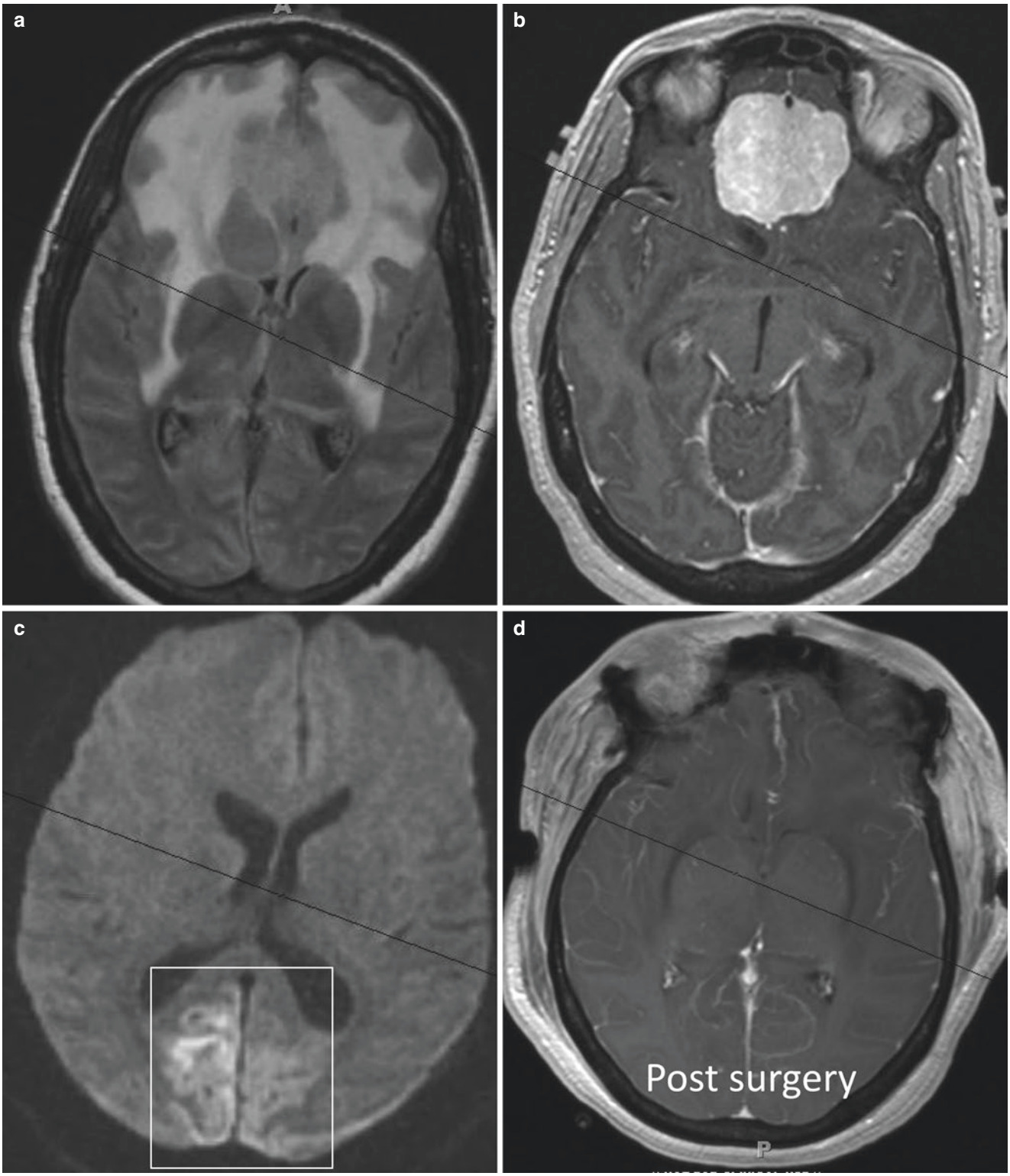

Fig. 1 (**a**) Preoperative T2-weighted magnetic resonance imaging (MRI) scan, demonstrating significant bilateral frontal lobe edema tracking posteriorly along the white fiber tracts. (**b**) Preoperative T1-weighted MRI scan with contrast, demonstrating olfactory groove meningioma. (**c**) Preoperative diffusion weighted imaging, demonstrating bilateral occipital lobe infarcts (highlighted by *box*). (**d**) Postoperative imaging demonstrating complete resection

Conclusions

To our knowledge this is the first report of a skull base meningioma presenting as an acute herniation syndrome in the absence of intratumoral hemorrhage. Acute medical and surgical management are reviewed. Central tumor necrosis exacerbating peritumoral edema is our leading hypothesis to explain herniation. Resuscitation with anti-edema therapy followed by surgical excision is the treatment of choice.

Conflict of Interest Statement We declare that we have no conflict of interest.

References

1. McDowell D, Harper CG (1991) Central tumour necrosis: a cause for the acute presentation of benign meningiomas. Aust N Z J Surg 61(7):553–556
2. Barua KK et al (2000) Spontaneous peritumoral haemorrhage associated with sinus confluence meningioma: case report. Surg Neurol 54(3):254–259
3. Hayashi K et al (2003) A case of atypical meningioma associated with acute deterioration and cerebral herniation. No Shinkei Geka 31(12):1309–1313
4. Helle TL, Conley FK (1980) Haemorrhage associated with meningioma: a case report and review of the literature. J Neurol Neurosurg Psychiatry 43(8):725–729
5. Kim DG et al (2000) Meningioma manifesting intracerebral haemorrhage: a possible mechanism of haemorrhage. Acta Neurochir (Wien) 142(2):165–168
6. Kohli CM, Crouch RL (1984) Meningioma with intracerebral hematoma. Neurosurgery 15(2):237–240
7. Lefranc F et al (2001) Intracranial meningiomas revealed by non-traumatic subdural haematomas: a series of four cases. Acta Neurochir (Wien) 143(10):977–982; discussion 982–983
8. Martinez-Lage JF et al (1991) Meningiomas with haemorrhagic onset. Acta Neurochir (Wien) 110(3–4):129–132
9. Modesti LM, Binet EF, Collins GH (1976) Meningiomas causing spontaneous intracranial hematomas. J Neurosurg 45(4):437–441
10. Niiro M et al (2003) Clinico-pathological study of meningiomas with haemorrhagic onset. Acta Neurochir (Wien) 145(9):767–772
11. Renowden SA, Hourihan MD (1992) Case report: acute subdural haematoma--an unusual presentation of a meningioma. Clin Radiol 45(5):351–352
12. Sakowitz OW et al (2005) Acute haemorrhage into a microcystic meningioma leading to cerebral herniation. Br J Neurosurg 19(3):260–264
13. Wakai S et al (1982) Spontaneous intracranial hemorrhage caused by brain tumor: its incidence and clinical significance. Neurosurgery 10(4):437–444
14. Misra M, Rath S, Mohanty AB (1989) Anton syndrome and cortical blindness due to bilateral occipital infarction. Indian J Ophthalmol 37(4):196
15. Wessling H et al (2006) Anton's syndrome due to a giant anterior fossa meningioma. The problem of routine use of advanced diagnostic imaging in psychiatric care. Acta Neurochir (Wien) 148(6):673–675; discussion 675