Delayed-onset abducens nerve palsy following parafalcine meningioma complicated by subdural hematoma

Meningiomas are slow-growing, benign brain tumors accounting for approximately 20% of primary intracranial masses.\(^1\) They may remain asymptomatic for long periods of time and can present with headaches, seizures, and other more site-specific symptoms. Although they are highly vascular structures, meningiomas do not typically produce subdural hematoma (SDH).\(^2\) Only 38 cases of meningioma associated with acute SDH have been reported in the English literature.\(^1\) We report a delayed-onset abducens nerve palsy due to an SDH associated with a parafalcine meningioma. To our knowledge, this is the first such reported case in the English-language ophthalmic literature.

A previously healthy 55-year-old Caucasian female presented with 3 days of acute, severe, and worsening headaches. Her medical history was significant for benign essential tremor and migraines. Her medications included armodafinil, butalbital with caffeine and acetaminophen, hydrocodone, multivitamins, pregabalin, and primidone. She did not smoke and reported social alcohol consumption.

Her initial blood pressure was 173/82 mm Hg. Neurologic examination was unremarkable. Non-contrast computed tomography (CT) of the brain revealed a right hemispheric laminar SDH with focal lobulation at the right anterior falx and mass effect (Fig. 1A). An anterior falx hyperdense extra-axial mass lesion was also noted. Computed tomography angiogram of the head was unremarkable. Magnetic resonance imaging of the brain confirmed the right cerebral convexity SDH with an adjacent right parafalcine meningioma and a 3 mm left unruptured superior hypophyseal artery aneurysm. This aneurysm was not thought to be related to the patient’s presentation. She underwent preoperative Onyx (Medtronic, Minneapolis, Minn.) embolization of the meningioma but developed worsening headaches and neurologic decline with an increasing SDH and brainstem compression that required emergency craniectomy. The patient then underwent bilateral middle meningeal artery embolization with an angiographic decrease in the SDH membrane hyperemia seen on postprocedural angiogram. She subsequently underwent gross total resection of a histopathologic World Health Organization grade I meningioma. The patient was discharged to rehabilitation for physical and occupational therapy.

Four weeks after her inpatient stay, the patient presented with 10 days of new sudden-onset painless nonprogressive bilateral horizontal diplopia. There were no other associated symptoms. On neuro-ophthalmic examination, her visual acuity was 20/25 OU. External examination was unremarkable except for a well-healed craniotomy scar on the right. Her pupils were isocoric with no relative afferent pupillary defect. Slit-lamp biomicroscopy, intraocular pressures, and dilated fundus examination were normal. The extraocular motility examination showed \(-1^\circ\) abduction in the left eye with an incongritant esotropia (ET) of 12 prism dipters (PD) in primary position, 16 PD ET in left gaze, and 6 PD ET in right gaze, consistent with a left abducens nerve palsy. Repeat magnetic resonance imaging showed a small subdural collection predominantly overlying the right cerebral convexity measuring up to 5 mm in thickness anteriorly that decreased in size. Mild diffuse pachymeningeal enhancement was also noted, suggesting possible nonlocalizing intracranial hypotension as the potential mechanism of delayed postoperative abducens nerve palsy. At 3-month review, her diplopia had resolved, and extraocular motility examination was normal.

The mechanism of meningioma-related SDH is controversial. Most of these SDH cases (65%) demonstrate a meningioma adjacent to the cerebral convexity. The mean age of these patients was 61 years, with a 57% female predominance.\(^1\) Other risk factors for SDH that have been reported in these meningioma cases include hypertension, trauma, anticoagulation therapy, and pregnancy. None of these factors was present with our patient.

The predominant hypothesis for SDH in meningioma is rupture of an abnormal, thin, fragile vascular network within the tumour. Angioblastic meningiomas commonly contain thin-walled vessels, whereas malignant
meningiomas commonly invade or proliferate, which can congest and necrose the tumour vessels and lead to local hemorrhage within the subdural space. It was for this reason that our patient underwent preoperative Onyx embolization of the meningioma as well as middle meningeal artery embolization for management of the SDH.3

Other proposed mechanisms include the internal release of vasoactive substances within the meningioma (e.g., histamine and substance P). This can lead to dilatation of the meningioma vessels and ensuing hemorrhage. The meningioma also can mechanically stretch and distort bridging veins within the subdural space, increasing the risk of SDH.3

We hypothesize that our patient’s delayed abducens nerve palsy was likely from intracranial hypotension in the setting of her meningioma resection as well as Onyx embolization of the tumour and middle meningeal artery embolization, all of which could have contributed to intracranial hypotension postoperatively. Abducens nerve palsy is a well-known nonlocalizing finding in both intracranial hypertension and hypotension and has been recently reported as a complication of middle meningeal artery embolization performed for SDH (although this was an immediate and not delayed complication).4 In our patient, the magnetic resonance imaging features, as well as her clinical course, are more suggestive of intracranial hypotension as the presumptive mechanism. Intracranial hypotension has been reported in the literature as causing many oculomotor abnormalities—one large review of the literature noted that abducens nerve palsies were the most common manifestation, seen in 83%.5

Of the abducens nerve palsies, 71% were unilateral. Multiple hypotheses for the mechanism of intracranial hypotension causing cranial neuropathy have been postulated in the literature, including compression, traction on the nerve, and vascular congestion of the nerve, and it is possible that asymmetric effects on the nerves may result in unilateral clinical manifestations, similar to in our patient.5

Although intracranial hemorrhage from meningioma is uncommon, SDH is distinctly rare. The SDH in this meningioma case and secondary nonlocalizing abducens nerve palsies are unique attributes of our case. To our knowledge, this is the first such reported case in the English-language ophthalmic literature. Clinicians should be aware that meningiomas may rarely present as SDH because this may alter the initial management and that nonlocalizing abducens nerve palsy may occur from either high or low intracranial pressure.

Sami Younes,* Subahari Raviskanthan,† Peter W. Mortensen,‡ Andrew G. Lee3,5,*,§

*Baylor College of Medicine, Houston, Tex.; †Houston Methodist Hospital, Houston, Tex.; ‡Weill Cornell Medicine, New York, NY; §University of Texas Medical Branch, Galveston, Tex.; §University of Texas MD Anderson Cancer Center, Houston, Tex.; †Texas A&M College of Medicine, Bryan, Tex.; ‡University of Iowa Hospitals and Clinics, Iowa City, Iowa.


Correspondence to Andrew G. Lee, MD; aglee@houstonmethodist.org.

References


Footnotes and Disclosure

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