Neuroimaging Highlight

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Subdural Hematoma: A Rare Presentation of a Convexity Meningioma

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CASE PRESENTATION

A 69-year-old male presented to a peripheral emergency department with a several day history of increasing confusion and headache. On admission, his Glasgow Coma Scale (GCS) score was 10 (E3 M6 V1). He was mute but would obey commands intermittently. Cranial nerve exam was normal and there was no evidence of weakness. A computed tomogram (CT) head (Figure 1) showed a chronic subdural hematoma with midline shift, as well as a mass within the left frontal region that appeared consistent with a convexity meningioma seen on magnetic resonance imaging (MRI) two years prior (Figure 2). The patient was not on any blood thinners and had no history of falls. Past medical history also included hypertension and prostate cancer.

MANAGEMENT AND OUTCOME

Over the course of the night, the patient’s GCS deteriorated to 8 and he required intubation. He was brought to the operating room and underwent left craniotomy. Intra-operatively we found a subacute clot attached to the tumor (Figure 3) and final pathology confirmed WHO grade 1. Patient was transferred to local hospital for ongoing rehabilitation after significant improvement post-surgery.

DISCUSSION

Although meningiomas are highly vascularized tumors, bleeding is a rare complication, with an overall frequency of 1.8% obtained by aggregating the three largest studies

Figure 1: A-C: Pre-op CT head.
available.1-3 The most common type of bleeding is subarachnoid hemorrhage, followed by intra-cerebral hemorrhage and intratumoral hemorrhage, with subdural hemorrhages—both acute and chronic—being especially rare.4 A report in 2006 of two cases of meningiomas presenting as chronic subdural hematoma identified 18 prior cases.5 Convexity meningiomas account for 75% of subdural bleeding in that series but in general account for 20% of meningiomas in most surgical series.6 A number of theories have been postulated as to the mechanism by which hemorrhagic meningiomas occur. These include rupture of weak, thin-walled, or eroded tumor vessels, rupture of enlarged tortuous feeding arteries due to high flow, growth of the tumor causing stretching and rupture of subdural bridging veins, and intratumoral necrosis due to venous thrombosis or rapid tumor growth.7

REFERENCES