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CASE REPORT

Sphenoid Wing Meningioma Mimicking the Clinical Presentation of Stroke

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ABSTRACT

Meningioma is the commonest extra-axial brain tumour in adults presenting with symptoms depending on the sites of its occurrence. We report an uncommon presentation of a meningioma in which a 57-year-old woman presented with a 3-day history of sudden onset of left-sided hemiparesis mimicking a stroke. Radiological imaging revealed an extra-axial lesion in the right sphenoid wing suggestive of a meningioma with perilesional oedema, mass effect, midline shift and herniation. She underwent surgical resection with histopathological examination revealing a meningioma World Health Organization (WHO) Grade I. Six weeks post operatively during follow-up, the left sided weakness had resolved, and patient has had no further symptoms of stroke until now.

KEYWORDS: Sphenoid wing meningioma, stroke, hemiparesis, mass effect

INTRODUCTION

Meningioma is a tumour that arises from the meninges which are the protective membranes that cover the brain and spinal cord. Meningioma is the commonest extra-axial brain tumour found in adults accounting for one-third of all primary intracranial neoplasm [1]. Its incidence increases progressively with age and has a higher preponderance in women, with a female-to-male ratio of 2:1 [1]. Sphenoid wing meningioma (SWM) is the third most common group of meningioma accounting for 11-20% of cases in the general population [2].

Meningioma is graded according to the World Health Organization (WHO) grading system (grades I to III) based on the appearance of the tumour cells [3]. Grade I is the most common type of meningioma accounting for 84% of cases. This non-cancerous type of brain tumour grows slowly and has clear borders. Grade II meningioma (15% of cases) are atypical which is neither malignant nor benign. They tend to recur, grow faster and may become malignant. Grade III meningioma is the most aggressive form and is considered malignant. They account for 1% of cases and is the most aggressive form, invading the surrounding tissues of the brain that are closest to the tumour [3].

The presentation of patients with intracranial meningiomas varies according to the site of the tumour such as seizure, headache, cranial nerve palsy and visual disturbance [2]. In this case report, we describe an uncommon presentation of a meningioma in which the patient presented with a 3-day history of left sided hemiparesis, mimicking a stroke.

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CASE PRESENTATION

A 57-year-old right-handed woman presented to the emergency department (ED) with left-sided body weakness which led to a fall three days prior to presentation while she was trying to get out of bed. Since the fall, she has required assistance in ambulating. She had no prodromal features prior to the body weakness such as vomiting, loss of consciousness, dizziness, palpitations, seizure and no speech or visual disturbances. She did not seek medical attention during her initial presentation, but now presented due to the persistent left-sided weakness for three days. Her past medical history was remarkable for 3-vessel coronary artery disease, type 2 diabetes mellitus, hypertension, dyslipidaemia, and fatty liver. In 2014 she underwent a percutaneous coronary intervention (PCI) after an angiogram showed >85% blockage at the right coronary artery. She drinks alcohol occasionally, never smoked or took any recreational drugs.

On clinical examination, her vital signs were normal. Her Glasgow Coma Scale (GCS) on arrival was 14/15 (E3V5M6). She was orientated to time and place. Neurological examination revealed normal cranial nerve examination and fundoscopy. Her visual field was normal and visual acuity was 6/6 bilaterally with glasses. Motor examination showed normal muscle bulk and tone, but power was 3/5 (Medical Research Council (MRC) scale) in the left upper and lower limbs. Deep tendon reflexes were brisk on the left side with upgoing Babinski sign. Cerebellar examination was normal.

Laboratory blood investigations, chest x-ray and electrocardiogram were normal. A non-contrast enhanced computer tomography (NECT) scan of the head revealed a mass in the right fronto-temporal region (Figure 1) measuring 4.2 x 4.7 x 4.8cm (AP x W x CC) with significant perilesional cerebral oedema, midline shift to the left, hydrocephalus and right uncal and right subfalcine herniation.

In view of the NECT scan findings, she was electively intubated for cerebral protection. The case was discussed with the neurosurgery team from the tertiary centre and intravenous dexamethasone (8mg stat then 6mg 8-hourly) was started. Subsequently, a contrast enhanced magnetic resonance image (MRI) of the brain was done which confirmed the presence of an avidly enhancing supratentorial extra-axial mass with a dural tail in the right fronto-parietal region with perilesional oedema, mass effect and midline shift (Figure 2). This mass was isointense to grey matter on T1 weighted images (T1WI) and slightly hyperintense to grey matter on T2 weighted images (T2WI). There was mass effect to the ipsilateral lateral ventricle, third ventricle and basal cisterns causing hydrocephalus. These findings were highly suggestive of a meningioma. No other significant parenchymal abnormality was detected.

She was then transferred to the care of the neurosurgical team at the tertiary centre and was planned for a right pterional craniotomy and excision of tumour. She subsequently underwent surgical resection of the tumour. Intra-operatively, devascularization of the tumour was performed extradurally by separating the dura from the underlying sphenoid wing and thinning of the hyperostosis sphenoid wing with high-speed drill till near to the cavernous sinus. Upon duratomy, a well-circumscribed dural-based tumour was found along the sphenoid wing with no arachnoid or brain parenchymal infiltration. Complete tumour resection was performed. Post-operatively, the dexamethasone was changed from intravenous to orally on post-operative day 2 when the patient was allowed orally and tapered off within a 1-week period. No surgical complication was reported, and the histological examination revealed a right sphenoid wing meningioma, meningothelial variant, WHO grade I. Post-operatively, on 6 weeks follow-up she regained full strength of her left sided upper and lower limbs to 5/5 (MRC scale) and managed to ambulate with a walking frame on the 8th postoperative day. She underwent a stroke work-up as an out-patient in view of her medical background. Echocardiogram, Holter monitor, and carotid Doppler ultrasound were normal. She has not reported any recurrence of stroke symptoms on outpatient follow-up to date.

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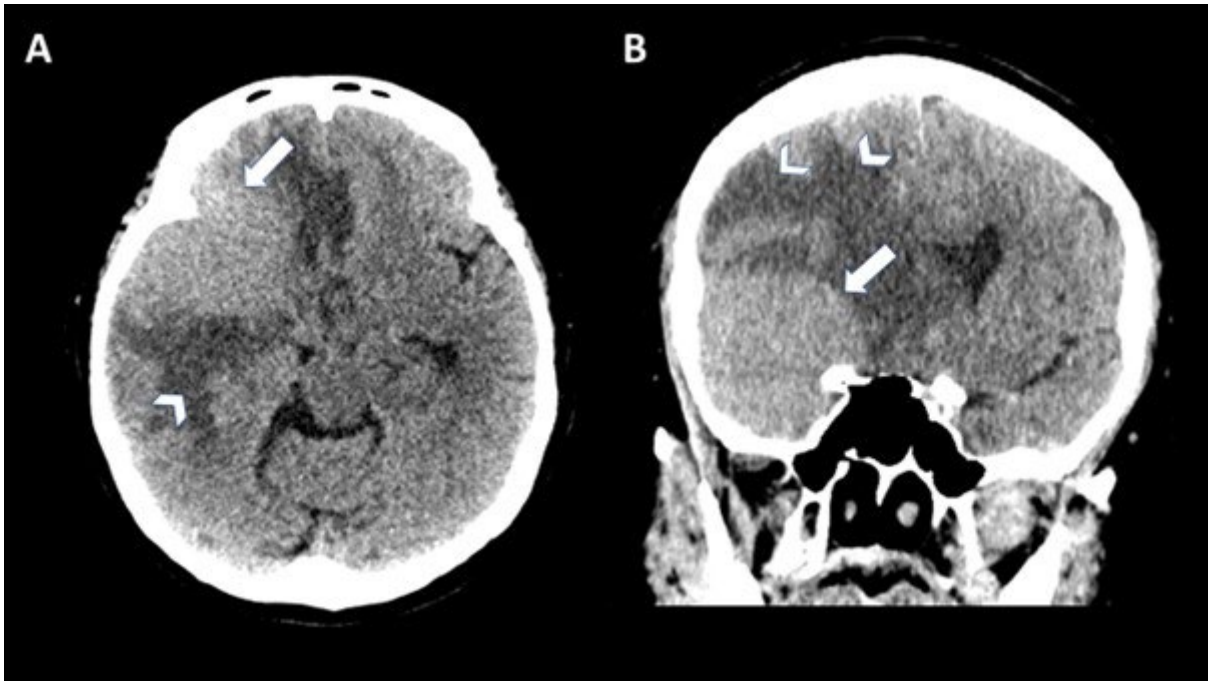

Figure 1 A 57-year-old woman who presented to hospital with an acute onset of left-sided hemiparesis. An NECT brain in axial (Figure 1A) and coronal (Figure 1B) views revealed an isodense mass (white arrows) in the right frontal and temporal regions with significant perilesional oedema (arrow heads) with effacement of the cerebral sulci in the right cerebral hemisphere, significant midline shift, mass effect to the body of the right lateral ventricle, temporal horn of the right lateral ventricle and third ventricle.

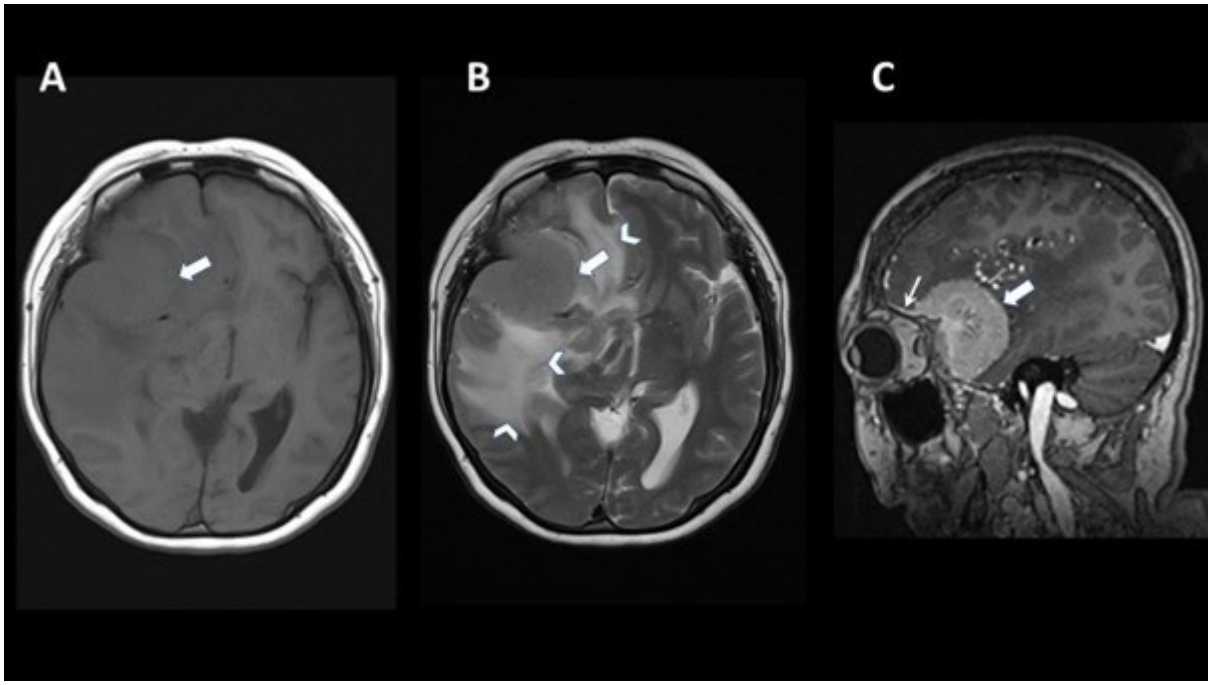

Figure 2 A contrast enhanced MRI brain of the same patient showed an extra-axial mass (thick white arrows) in the right frontoparietal region. It was isointense to the grey matter on axial T1WI (Figure 2A), slightly hyperintense to grey matter on axial T2WI (Figure 2B) and demonstrated avid enhancement post gadolinium on sagittal view (Figure 2C) with a distinct dural tail (thin white arrow). There was mass effect and significant midline shift to the left with perilesional white matter oedema (arrow heads).

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DISCUSSION

Meningioma is a tumour of the meningothelial cells of the arachnoid layer and is classified based on the anatomical location. It often originates in the suprasellar, fronto-basal, temporo-basal, sphenoid wing or petroclival regions. SWM commonly presents with generalized headaches (40.4%), seizure (20.6%) and visual impairment (18.4%) [2]. This is due to the SWM which arises from or extends into the orbital or temporal fossa, thus producing ocular manifestations. Meningioma has also been reported to present as a transient or reversible neurological deficit resembling transient ischaemic attacks (TIA), however, this finding is uncommon and makes up only 0.19% of the presentation of meningioma [4]. Complete strokes in relation to meningioma are extremely rare [4]. There were case reports in which patients presented with pure motor hemiparesis due to malignant glioma (primary central nervous system lymphoma and brain metastasis), but the finding is not common in benign dural-based tumours [5,6].

In this case, the patient presented with left-sided hemiparesis for three days mimicking a stroke. The differential diagnosis initially in ED were of a stroke or space-occupying lesion. However, the CT and MRI brain showed that the radiological findings were consistent with a meningioma and less likely a stroke. A possible explanation of the left-sided hemiparesis in this patient was due to the mass effect of the meningioma and its surrounding oedema on the cerebral arteries to a degree that was sufficient to compromise cerebral blood flow. This leads to reduction in the arterial flow supplying the right side of the brain. Mass effect of the meningioma could also lead to increased focal tissue pressure resulting in neurological defect. It has also been reported that the vascular compression can be associated with coning signifying a preterminal event which was seen in our patient and thus she underwent elective intubation as a means of cerebral protection [7]. Meningioma cells are associated with secretion of vascular endothelial growth factor-A (VEGF-A) to induce angiogenesis and oedemagenesis of both tumour and peritumoral brain tissue with disruption of the blood-brain barrier [8,9]. Dexamethasone was administered in this patient to reduce the tumour-surrounding oedema pre-operatively. The neurological improvement observed with steroids prognosticates a high likelihood of neurological recovery post-resection of the SWM.

Several other postulations that may explain the manifestation of neurological deficit simulating a stroke or a TIA in a meningioma include; i) vascular flow or shunting through the meningioma may result in a "steal" phenomenon in tumours with high vascularity leading to focal brain ischaemia in region of the mass [10], ii) increased regional venous pressure due to obstructed venous outflow [10], iii) tumour-related hypercoaguable state leading to thromboembolic episodes [11], iv) sudden change in the intracranial pressure with haemorrhage into the tumour [12], and v) "spreading depression of Leao phenomenon" as a response of the cerebral cortex to noxious stimuli leading to marked hemiparesis or hemianaesthesia [13].

Reports have shown that SWM does not usually modify the vascular patency even if it has completely compressed the internal carotid artery (ICA) and its bifurcation in view of its slow growth and non-invasive nature [7]. However, Komotar et al. has reported two cases of cerebral infarction secondary to intracranial ICA occlusion by meningioma in which these patients presented as stroke [4]. In this patient, the major vessels were at the surface of the tumour as seen in Figure 2, indicating the high risk of developing an acute infarct following complete resection of the tumour.

In our case, resolution of the patient's hemiparesis with no recurrence following resection of the tumour favours the diagnosis of meningioma as the cause of the hemiparesis. This is supported by the normal stroke work-up of echocardiogram, Holter monitor and carotid Doppler ultrasound findings.

CONCLUSION

In conclusion, this case presents a rare finding of stroke symptom in a patient with sphenoid wing meningioma WHO grade I. It also highlights the importance of a careful clinical history, examination and obtaining early neuro-radiological imaging to recognize these patients who have intracranial masses whom presented with stroke symptoms.

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Conflict of Interest

Authors declare none.

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Authors' contribution

MY Mohd Yusri and SF Badlishah-Sham drafted the manuscript. AB Peter and BS Liew provided the radiology and neurosurgical input for the manuscript respectively and revised it critically for important intellectual content. All authors have read and given approval of the final version of the manuscript. Each author has participated sufficiently in the work to take public responsibility for appropriate portions of the content as described above.

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