Coexistence of Malignant Meningioma and Anaplastic Ganglioglioma

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ABSTRACT
The case of a 45-year-old Chinese man with coexisting intracranial tumours is reported. Following presentation with left hemiparesis, computed tomography and magnetic resonance imaging of the patient’s brain identified a large, soft tissue mass, with massive oedema in the adjacent brain parenchyma. Coarse calcifications were noted at the periphery of the mass. Surgical resection of the mass was performed and microscopic examination of the specimen revealed the presence of a malignant meningioma, and an anaplastic ganglioglioma.

Key Words: Computed tomography, Ganglioglioma, Magnetic resonance imaging, Meningioma

CASE REPORT
A 45-year-old Chinese man was admitted to Queen Elizabeth Hospital with acute onset of left-sided weakness. The patient had a history of epilepsy since childhood, which was well controlled with anti-convulsant medication, prescribed by a neurologist. Physical examination revealed a left hemiparesis, with motor power in the left upper and lower limbs both graded as 4/5. Neurological examination was otherwise unremarkable. Computed tomography (CT) brain scanning on admission showed a large, ill-defined, hyperdense mass in the right posterior parasagittal region, bowing the falx to the left. Marked oedema was present in the adjacent subcortical white matter, causing midline structural shift. Irregular coarse calcifications were noted at the periphery of the mass (Figure 1). Magnetic resonance imaging (MRI) confirmed the mass as extra-axial in location, and heterogeneously isointense with gray matter on both T1- and T2-weighted images. The lesion had an irregular contour, and was heterogeneously enhanced after the administration of intravenous gadolinium-DTPA (Figure 2).

A review of the patient’s previous medical records identified a CT brain scan performed 5 years earlier, which showed coarse calcifications at the right posterior parasagittal region (Figure 3). Comparing the current and previous imaging results, it appeared that a calcified mass in the brain had been present for at

Figure 1. Non-contrast axial computed tomography at the time of admission shows a large, hyperdense mass in the right posterior parasagittal region. Marked oedema was present in the adjacent subcortical white matter. Note the coarse calcifications at the periphery of the mass.
least 5 years. The most recent images revealed a newly developed extra-axial mass, forcing the calcifications away from the original site. The associated vasogenic oedema noted in the white matter of the adjacent brain was absent in the earlier CT scan.

The preoperative diagnosis was a parasagittal meningioma. Intraoperatively, the mass was seen to arise from the falx. Gross total resection was achieved. Pathological examination showed multiple fragments of friable, tan coloured tissue. The aggregated fragments measured 6.5 cm x 2.5 cm x 2 cm. The cut surfaces of the tissue fragments were variegated, with haemorrhagic and yellowish areas.

Microscopic examination showed that the tumour had two different components. One portion of the tumour comprised diffuse sheets of polygonal cells, with
hyperchromatic nuclei and a large amount of eccentric eosinophilic cytoplasm, imparting a rhabdoid appearance to the tumour cells. Focal necrosis and frequent mitosis were seen (Figure 4a). Although the typical features of meningioma, such as whorls and psammoma bodies were not found, the tumour cells were immunoreactive for epithelial membrane antigen, suggesting the presence of meningeal differentiation. Staining for glial fibrillary acid protein (GFAP), and synaptophysin were negative. The overall morphology and immunohistochemistry of the tumour was compatible with that of a malignant meningioma with rhabdoid features.

Another tumour with a different microscopic appearance was seen to coexist with the malignant meningioma (Figure 4b). This tissue was cellular and composed of tumour cells with round to oval nuclei, amongst a fibrillary background (Figure 4c). Synaptophysin and GFAP positive cells were identified. This tumour, therefore, was seen to exhibit both glial and neuronal differentiation, compatible with a histological diagnosis of anaplastic ganglioglioma. Of interest, a low grade component of the ganglioglioma consisted of an admixture of glial and ganglion cells. The cellularity in this area was found to be low, and calcification was readily identified (Figure 4d).

Postoperatively, the patient gradually regained full motor power of the limbs and subsequently underwent a course of whole brain radiotherapy. He remained well until two months later, when he complained of bone pain. A technetium-99m labeled methylene diphosphonate bone scan showed multiple foci of increased tracer uptake, compatible with the presence of bony metastases. The patient’s condition deteriorated, and he died as a result of disseminated disease, four months after surgery.

**DISCUSSION**

The case report presented is, to the authors’ knowledge, the first reported case of a malignant meningioma...
Coexistence of Malignant Meningioma and Anaplastic Ganglioglioma

coeexisting with an anaplastic ganglioglioma. Meningioma is the most common, primary non-glial intracranial neoplasm, representing 15-20% of all primary brain tumours. According to the World Health Organisation classification, 88 to 94% of meningiomas are benign or typical, 5 to 7% are atypical, and only 1 to 2% are anaplastic or malignant. Peripheral oedema is seen on approximately 59% of CT studies and 66% of MRI studies; it may be extensive, involving the white matter tracts of an entire hemisphere. Oedema associated with a meningioma may be due to compressive ischaemia, venous stasis, or parasitisation of pial vessels. Venous sinus occlusion or venous thrombosis can also cause intraparenchymal oedema. Nearly all meningiomas enhance rapidly and intensely following contrast administration. There is no reliable correlation between the degree of enhancement or surrounding oedema and tumour size or histologic subtype. Meningiomas rarely metastasise — in only 0.1 to 0.2% of cases. Location and histopathology do not correlate with metastasis. Some metastatic tumours are histologically benign, while some anaplastic meningiomas do not metastasise. Malignant meningioma can spread via the cerebrospinal fluid, and occasionally may travel to extraneural sites, including the lungs, liver, bone and lymph nodes.

Gangliogliomas, although relatively rare, are the most frequently occurring mixed glioneuronal tumour of the central nervous system, with an incidence rate ranging from 0.4 to 1.3% of all brain tumours. These lesions occur predominantly in children and young adults. Peak incidence is in the second decade of life. Gangliogliomas can be found in all brain sites, but have a predilection for the temporal lobe, followed by the frontal and parietal lobes. The tumours are generally well circumscribed and slow growing. Calcifications are seen on CT brain scans in 28 to 83% of gangliogliomas. Malignant transformation of a ganglioglioma is extremely rare, with malignant changes occurring most often in the glial component of the tumour. Although gangliogliomas do not metastasise, very rare cases of leptomeningeal and subarachnoid dissemination have been reported. There has also been a reported case of extracranial spread of an anaplastic ganglioglioma through a ventriculo-peritoneal shunt.

The case presented is unusual, with the patient having two coexisting tumours of different lineage — one a malignant meningioma and the other an anaplastic ganglioglioma. In this case, the anaplastic ganglioglioma probably resulted from malignant transformation of a pre-existing low grade ganglioglioma. The calcified mass noted in the first CT brain scan most probably represented this low grade ganglioglioma. Serial CT scans of the patient showed the pre-existing tumour calcification being displaced by the newly developed, dural based meningioma. The extensive oedema in the adjacent brain parenchyma appeared to be due to the dural based meningioma. The possibility of anaplastic change of the pre-existing ganglioglioma could not be determined by imaging studies alone, however, requiring histological assessment. Bony metastases developed in this patient two months after presentation. As a postmortem was not completed, it was not possible to determine which tumour was the source of metastatic disease in this case.

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REFERENCES