

Cystic Meningioma Masquerading as a Metastatic Tumor: A Case Report

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Abstract

Cystic meningioma is a rare form of intracranial meningioma. Meningiomas are typically solid tumors but may rarely have cystic components. The diagnosis of cystic meningioma is clinically challenging as the finding of multiple intra-axial tumors, including metastatic tumors, is relatively common. We report a case of cystic meningioma initially diagnosed as a metastatic tumor from a recurrence of acute lymphoid leukemia. However, postoperative histopathological examination demonstrated an atypical meningioma.

Keywords: cystic meningioma, acute lymphoid leukemia, metastases

Introduction

Meningiomas are the commonest type of primary non-gliar intracranial tumor accounting for 14%–19% of primary intracranial neoplasms. The incidence of meningioma increases with age with a female to male ratio of 1.8:1. Cystic meningiomas are rare with an incidence in adults of 2%–4% and more common in males (1,2). The diagnosis of a cystic meningioma is often clinically challenging as they mimic many intracranial cystic masses, such as haemangioblastoma, astrocytoma, neuroblastoma, and metastatic tumors (3). Herein we present a case of cystic temporoparietal meningioma masquerading as a metastatic tumor due to a relapse of acute lymphoid leukemia (ALL).

Case Report

We report the case of a 37-year-old gentleman diagnosed with ALL at the age of 3. At that time, he presented with abdominal bloating and spontaneous bruising over the limbs and trunk. He was diagnosed with ALL and received multiple blood transfusions over a 5-year period. Metastasis to the right testis was detected after the completion of two cycles of radiotherapy. He underwent right orchidectomy followed by 20 cycles of radiotherapy to the right testis and

chemotherapy via parenteral and enteral routes. The patient was finally discharged with complete resolution of ALL at the age of 20.

On this occasion, the patient presented with the sudden onset of unresponsiveness and aphasia. He had a history of persistent headache and vomiting over a 2-day period. Physical examination revealed a Glasgow Coma Score of 5/15 (E1, V1, M3) and unequal pupils with a fixed, 3 mm dilated right pupil. Neurological assessment revealed hypertonicity of all limbs with grade 3 muscle power. CT imaging revealed a mixed solid and cystic lesion in the right temporoparietal region with surrounding edema causing significant midline shift and hydrocephalus (Figure 1). A preoperative diagnosis of cerebral metastases secondary to relapsed ALL was made. Intraoperatively, a well-encapsulated tumor with mixed solid and cystic components was observed with a distinct tumor-brain interface. The cystic component was yellowish in colour. The tumor was completely excised along with dural attachments. Histopathological examination demonstrated an atypical meningioma (Figure 2).

Postoperatively, the patient made a good recovery achieving normal function and was able to resume activities of daily living at two months postoperatively. Follow-up MRI found no evidence of residual tumor. The patient was subsequently referred to oncology for radiotherapy.

Discussion

Meningiomas are the commonest type of extra-axial neoplasm accounting for 14%–19% of primary intracranial neoplasms. The incidence of meningioma peaks at 45 years of age with a female preponderance. Meningiomas are typically observed as isointense on T1WI and hypodense on T2WI imaging (2). In contrast to typical meningiomas, cystic meningiomas are rare with an incidence of 2%–4% in adults. Cystic meningiomas have a slight male preponderance (6) and a higher incidence in children of approximately 10%–18% (4).

The first report of cystic meningioma was by Penfield in 1932. This was followed by a report of 13 cases of cystic meningioma out of 313 intracranial meningiomas by Cushing and Eisenhardt in 1938 (4). Mehmet T et al. described a number of classification systems for cystic meningioma. Rengachary classified cystic meningiomas into two groups, namely intratumoral cysts and peritumoral cysts, whereas Nauta classified cystic meningiomas into four

different subtypes according to the location of the cyst cavity with respect to the brain: 1) centrally located intratumoral cysts; 2) peripherally located intratumoral cysts; 3) peritumoral cysts in adjacent parenchyma; and 4) peritumoral cysts between the tumor and adjacent parenchyma. This case most likely represented Type 2 Nauta classification.

No single theory fully explains the pathogenesis of cystic meningioma. However, there are many possible pathophysiological mechanisms underlying the development of cystic meningioma. At present, the formation of peritumoral cysts is thought to involve a number of processes including reactive gliosis, loculated widened of subarachnoid spaces, peritumoral demyelination or hemorrhage, fibroblastic proliferation, and mechanical trapping of cerebrospinal fluid (3,5). The development of intratumoral cysts is thought to be due to a

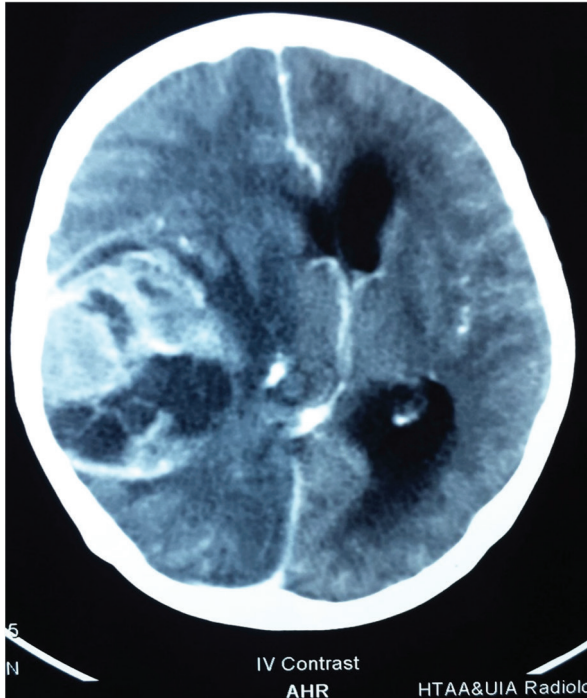


Figure 1: A contrasted CT scan showing a mixed solid and cystic lesion in the right temporo-parietal region with significant peri-lesional odema and midline shift.

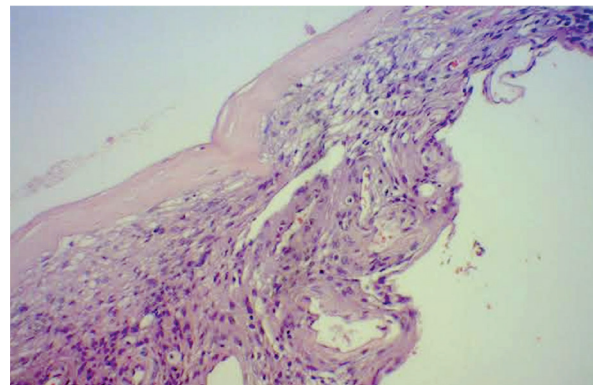


Figure 2: Haematoxylin-eosin staining showed tumour tissue arranged in solid sheets exhibiting whirling pattern especially surrounding the blood vessels. Tumour cells were mild to moderately pleomorphic, having round to mainly oval shaped nuclei, fine chromatin, some show inconspicuous nucleoli and moderate to abundant fibrillary cytoplasm. The cystic was composed of cyst wall made up of similar tumour cells having thin outer fibrous capsule and displayed large size vascular channels with surrounding stromal edema and foamy macrophages. Focal areas of necrosis were seen. Mitotic figure was 4 to 5 per 10hpf. These features were consistent with atypical meningioma.

combination of microcytic degeneration, ischemic necrosis, and intratumoral hemorrhage (2,3,4). The differentiation between cystic meningioma and other brain tumors, such as metastatic tumors, is clinically challenging preoperatively. The diagnosis of cystic meningioma is commonly based on histopathological examination and advanced imaging modalities such as MRI and cerebral angiography. The characteristic finding of cystic meningioma on MRI is an extra-axial lesion with the enhancement of a solid component (6). Coronal MRI allows visualisation of enhancing mural nodules and attachments to the falx or dura (1). Contrast enhanced MRI may have utility in distinguishing between cyst walls infiltrated by tumor cells and cyst walls consisting of gliotic tissues (1,4). Cerebral angiography allows delineation of the meningeal supply to the tumor from the external carotid artery in cases of meningioma (4). In solid meningiomas, the sensitivity and specificity of CT brain imaging is almost 100% and 90%, respectively. However, CT brain imaging allows the diagnosis of cystic meningioma in less than 30% of cases (2,5). Metastatic tumors from underlying leukemia also appear as hypodense foci in white matter with dilatation of cerebrospinal fluid spaces on CT brain imaging (7,8,9). In this case, we were unable to perform MRI because the patient demonstrated signs of herniation requiring immediate surgical intervention.

It is widely believed that atypical and malignant meningiomas, such as cystic meningiomas, exhibit more aggressive behaviour with a higher risk of recurrence. Cystic meningiomas have the potential to spread through the cystic fluid to cyst walls. Complete resection is currently the standard of care in preventing the recurrence of meningioma (1,2).

Conclusion

The preoperative diagnosis of a cystic meningioma may be clinically challenging due to the rarity of this tumor type. As illustrated in this case, possible disease recurrence in cases with a strong history of underlying leukemia may lead to negligible suspicion of cystic meningioma. Complete surgical excision and confirmation by histopathology examination are the current standard of care in cases of cystic meningioma.

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