Ruptured sylvian fissure dermoid presenting with MCA infarction: a rare case report

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INTRODUCTION

Dermoid cysts are ectodermal inclusion cysts. Intracranial dermoid cysts are rare, accounting for 0.04% to 0.06% of all intracranial tumours. They typically occur in or near midline. Following rupture, dermoid contents disseminate widely throughout subarachnoid space and ventricles. Cyst rupture can result in chemical meningitis, seizure, vasospasm with infarction and even death.

CASE HISTORY

A 23 yr old man was admitted to our department after developing a headache and right sided weakness. There was no history of vision loss, aphasia, fever, nausea, vomiting and seizures. Computed tomography (CT) scan demonstrated a well delineated, uniformly hypodense mass of fat density with capsular calcification (Figure 1). Multiple low density masses are also seen in subarachnoid space and cerebral sulci (Figure 2).

T1 weighted magnetic resonance imaging (MRI) revealed a mixed intensity non-homogenous mass in left sylvian fissure. The mass showed hypointense component along with lipid hyperintense component (Figure 3). There were multiple small T1 hyperintense lesions in subarachnoid space and cerebral sulci suggesting fat deposits (Figure 4). On diffusion weighted MR image left MCA territory infarction was noted (Figure 5).
On the basis of clinical presentation and the radiological images, a diagnosis of ruptured sylvian fissure dermoid cyst with left MCA territory infarct was made.

DISCUSSION

Intracranial dermoids are uncommon and originate from ectodermal inclusions. They are usually found in midline in contrast to epidermoids. Common intracranial locations are parasellar, frontobasal region and the posterior fossa. Patients with dermoid cyst are mostly asymptomatic or present with headache, seizures or signs of compression of neighbouring tissues. Rupture of these cysts can disperse the fatty contents into the subarachnoid space and find itself in any of the cisterns and ventricles. Most often the rupture is spontaneous though in certain instances, trauma has been implicated. Clinical manifestations in ruptured dermoid are very diverse. These include seizures, aseptic meningitis, transient cerebral ischemia as a result of vasospasm and even olfactory delusion. Rarely intraventricular fat leads to rapidly developing hydrocephalus due to granuloma in the aqueduct. CT findings are represented by the fat droplets in the subarachnoid spaces or a fat-fluid level within the ventricles. This is usually associated with an extra-axial or combined extra and intra-axial soft tissue mass representing the dermoid. On MRI, the fat can be seen dispersed as strongly hyperintense signals of T1 weighted image while the other tumour contents appear hypointense. On T2 weighted, the fat component turns slightly hypointense similar to subcutaneous fat. Characteristically, the tumor is often non-homogenous due to its mixed composition. While both CT and MRI are sensitive, MR has some distinct advantages. In addition to allowing multiplanar imaging, the exact extent of the mass and its relation to the skull base can be more easily evaluated because of the lack of bone artefacts. Lastly, the capability of MRI to evaluate the associated vessel displacement and mass effect on adjacent structures makes it the preferred technique.

In conclusion, ruptured intracranial dermoid cyst is a relatively uncommon cause of infarction and is fully diagnosable by radiology imaging. Prognosis of patients diagnosed with intracranial dermoids depends on the spread of the contents and time-period after rupture. Mortality as well as morbidity from complications such as chemical arachnoiditis and infarction can be dealt with and reduced significantly if imaging is done early in these patients.

References
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