

Ruptured Intracranial Dermoid Cyst in Temporal Fossa

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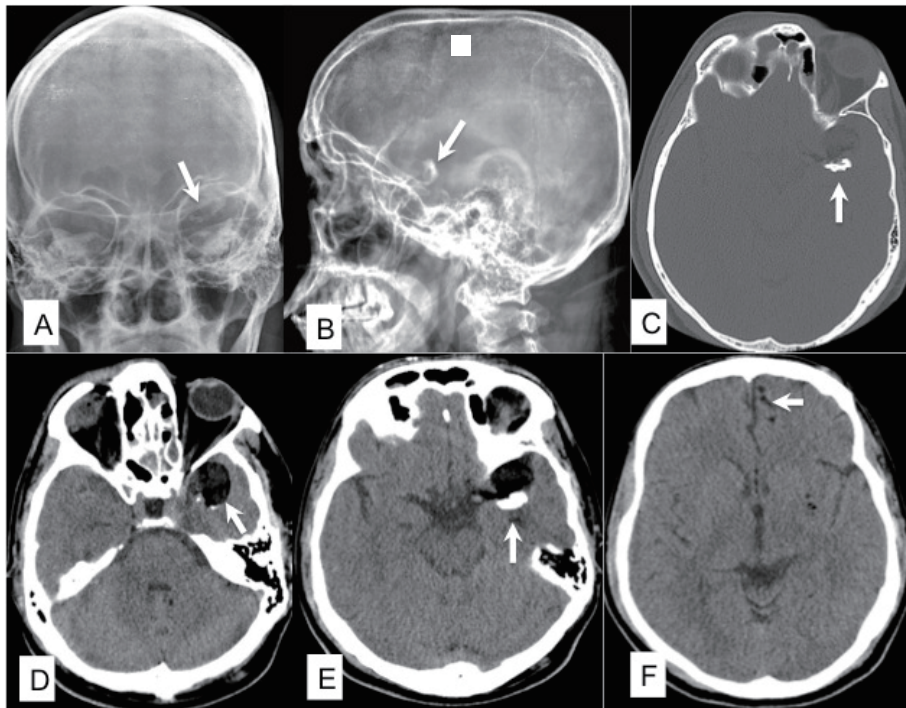


Figure 1. (A)&(B) -Plain X-ray skull anteroposterior (AP) and lateral view. (C) -CT brain (axial) bone window. (D),(E)&(F) -CT brain (axial) soft tissue window.

A 49-year-old male presented with headache for 6 months. There was no vomiting, fever or focal neurological deficits. There was no history of head injury or surgery. Plain radiographs of skull showed irregular calcifications in the left temporal region (Figure 1). Computed tomography (CT) of brain revealed a heterogeneous extra axial lesion in the anterior aspect of left temporal fossa and left middle cerebral artery cistern, showing calcification, soft tissue and fat density. Multiple tiny fat droplets were seen in interhemispheric fissure, left Sylvian cistern, left lateral ventricle and

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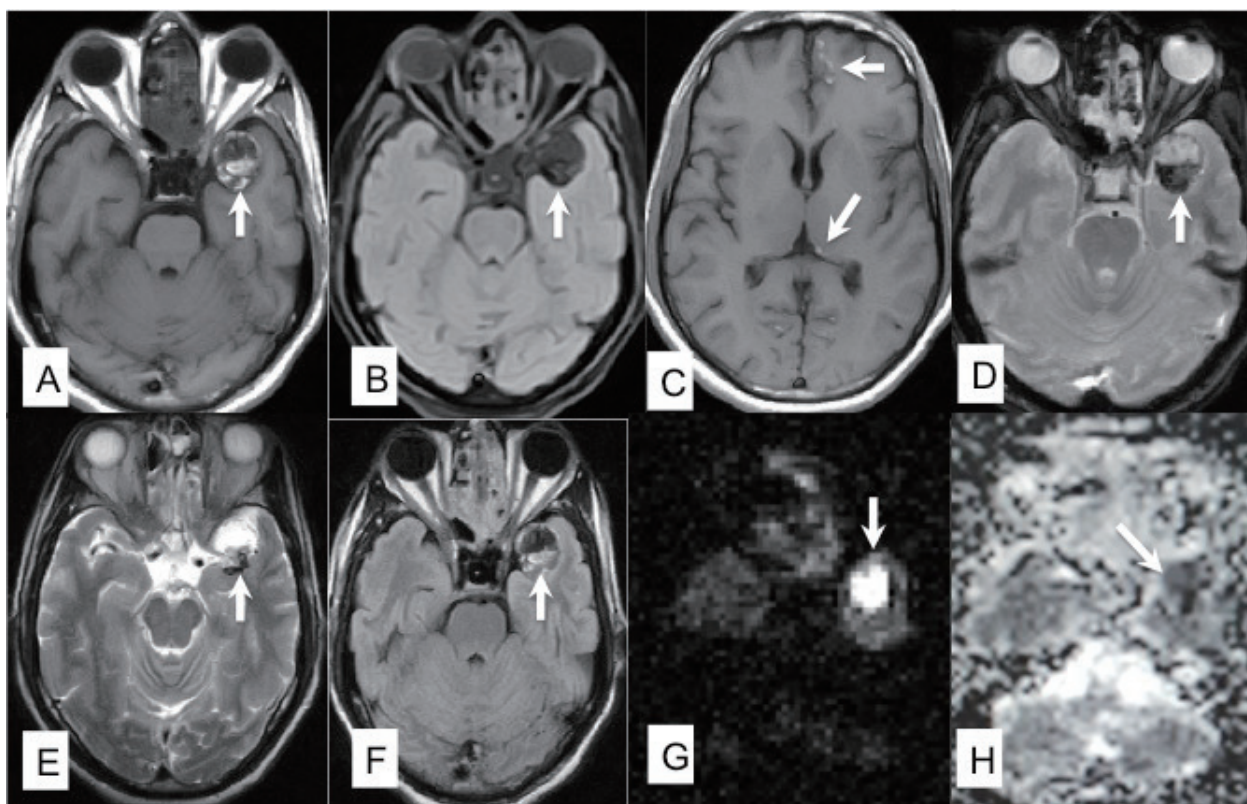


Figure 2. (A)&(C) T1W image. (B) T1 Fat suppression. (D) Gradient recalled echo (GRE). (E) T2W image. (F) T2 FLAIR. (G) Diffusion weighted image (DWI). (H) Attenuated diffusion coefficient (ADC).

bilateral sulcal spaces (Figure 1). Magnetic resonance imaging (MRI) brain with contrast revealed a non-enhancing heterogeneous lesion with restricted diffusion, calcification, soft tissue and fat signal intensity. Multiple tiny T1 hyperintense foci representing fat globules were seen along the bilateral sulcal spaces, left lateral ventricle, interhemispheric fissure and left Sylvian cistern. There was no significant meningeal enhancement (Figure 2). Based on plain radiographs, CT and MRI findings, ruptured intracranial dermoid cyst was considered. Patient was referred to neurosurgery and managed conservatively with anti-epileptics.

Dermoid cysts, non-neoplastic, congenital ectodermal inclusion cysts containing ectoderm derivatives, constitute < 1% of all intracranial masses⁽¹⁾. These slow-growing benign tumors found in sellar, parasellar location and fronto-nasal region, can cause significant morbidity through compression of neurovascular structures or rarely through rupture into the subarachnoid space and may

present as headache and seizures⁽²⁾. These tumors appear heterogeneous on MRI due to calcifications, fat and soft tissue components with diffusion restriction and does not show significant enhancement on contrast admission⁽³⁾. Imaging differential diagnosis include intracranial lipoma (homogeneous fat attenuation/signal intensity, chemical shift artifact, but no restricted diffusion), intracranial epidermoid (contains only squamous epithelium with no macroscopic fat and shows restricted diffusion), arachnoid cyst (follows CSF signal intensity on all sequences), cystic glioma (shows irregular, nodular, peripheral enhancement and perilesional edema) and craniopharyngioma (sellar/parasellar location, vivid contrast enhancement of solid components and absence of macroscopic fat)⁽⁴⁾. As the lesion had large areas of macroscopic fat, calcifications and restricted diffusion, it could be diagnosed as dermoid even without histopathology. Ruptured intracranial dermoids show fat droplets along the subarachnoid space. T1 Fat saturation sequence, T1 post-contrast and Diffusion

weighted images helps in identification of dermoid from other similar intracranial lesions. Rupture of dermoid cystic tumor may be spontaneous, post-traumatic or iatrogenic. Chemical meningitis is relatively rare, seen in ~ 7% of cases of dermoid rupture⁽⁵⁾. Symptom onset typically does not occur during rupture, and may occur about 3 months to 6.5 years after rupture⁽⁶⁾. Dermoid cysts are surgically excised, when symptomatic. Recurrence is uncommon if completely excised⁽⁷⁾.

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