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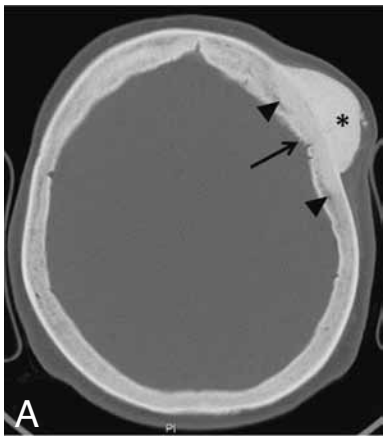
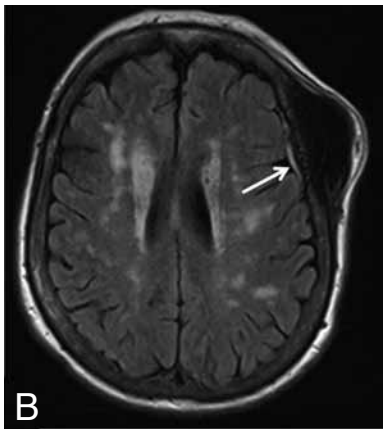
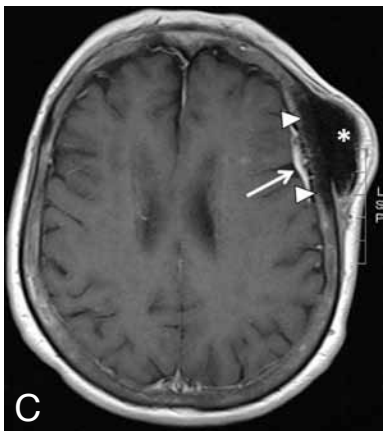
IMAGES IN CLINICAL RADIOLOGY







Hyperostotic meningioma mimicking skull osteoma

M. Eyselbergs[1,2], F.M. Vanhoenacker[1,2], D. Kools[3]

A 79-year-old female patient was referred to the radiology department because of a slowly enlarging bony mass on her left forehead. Neurological and systemic examinations were otherwise unremarkable.

Computed tomography (CT) with bone window settings showed a broad based hyperostotic lesion on the tabula externa of the left frontal bone (asterisk) with associated sclerosis of the diploë (arrowheads) and an irregular delineation of the tabula interna (arrow) (Fig. A). Magnetic resonance imaging (MRI) also showed flat thickening of the dura that was slightly hyperintense to brain parenchyma on FLAIR (Fig. B, arrow). T1-weighted imaging (WI) after gadolinium contrast administration clearly revealed enhancement of the thickened dura adjacent to the skull lesion (Fig. C, arrow) and confirmed the overlying sclerosis (arrowhead) and hyperostosis (asterisk) depicted on CT. The imaging features were compatible with findings of meningioma en plaque (MEP).

Resection of the tumor was performed with subsequent duraplasty and cranioplasty. Histological examination confirmed the MEP arising from arachnoid meningoendothelial cells.

Comment

Meningioma is the most frequently observed intracranial non glial tumor in the adult population (20%) with a female predominance. The tumor arises more frequently in African-Americans. Approximately 10% of the meningiomas are clinically silent.

Typically, this tumor is slowly growing, sharply demarcated and surrounded by a capsule. Two main morphological configurations can be encountered: a spherical lobulated dural based one and a more sheetlike 'en plaque' configuration with dural and sometimes overlying bony infiltration. Although hyperostosis is a well known imaging characteristic of most meningiomas, this feature predominates in MEP.

Both CT and MRI are useful imaging modalities for diagnosis of MEP. CT with bone window settings often demonstrates adjacent bony involvement such as erosions, sclerosis and hyperostosis. The degree of hyperostosis is usually disproportionate to the underlying size of the lesion. Sometimes a subdural plaque of ossification can be seen that is separated from the sclerotic or hyperostotic bone by a linear translucency corresponding to dura mater. On MRI the lesion is iso- to hypo-intense on T1-WI and has a variable appearance on T2-WI correlating to pathological features. A cerebrospinal fluid cleft can often be visualized on T2-WI which confirms its primary extra-axial localization. After contrast administration more than 95% of the lesions enhance vividly both on CT as on MRI.

Multiple mechanisms are proposed for the hyperostosis associated with meningiomas but tumoral invasion of the overlying bony structures seems to be the most accepted theory.

The differential diagnosis of focal or regional skull hyperostosis is extensive and includes benign and malignant lesions. In the benign group, an osteoma must be considered since these neoplasms are the most common primary benign bone tumors in the craniofacial skeleton. However, an osteoma lacks contrast enhancement after contrast administration and normally does not involve the diploë. Other benign lesions consist primarily of fibrous dysplasia, Paget's disease, calcified cephalo- or subdural hematoma and hyperostosis interna. Malignant causes of focal skull hyperostosis comprise osteoblastic metastasis and lymphoma.

Treatment of choice of symptomatic lesions consists of resection of the meningioma with the associated hyperostotic bone since definite proof of tumoral invasion can only be obtained after careful histological examination. An initial "wait and see policy" may be considered for asymptomatic lesions.

Reference

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1. Department of Radiology, AZ Sint-Maarten Duffel-Mechelen, Duffel, 2. Department of Radiology, Antwerp University Hospital, Edegem, 3. Department of Neurosurgery, AZ Sint-Maarten Duffel-Mechelen, Duffel, Belgium.