Hard scalp lump with headache

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Answer

Intraosseous calvarial meningioma.

Discussion

Computed tomography (CT) of the head revealed localized osteosclerotic thickening in the left temporo-parietal bone, with spiculated margins of the inner and outer tables, involving the coronal suture and extending to the frontal bone, with no evident bony erosion. Magnetic resonance imaging (MRI) showed an expansile hypointense lesion on the T1- and T2-weighted images in the left temporo-parietal bone with thickening of the extracranial soft tissues and no associated intracranial lesions. Wide surgical resection of the lesion was carried out with cranioplasty. Histopathological examination showed clusters of meningothelial cells, forming small whorls and infiltrating the trabecular bone spaces, which gave the diagnosis of intraosseous calvarial meningioma (ICMs).

Intraosseous calvarial meningiomas are defined as meningiomas confined to the skull with no dural involvement [1, 2]. Meningiomas arising outside the dural compartment have

been called ectopic, extracranial, extradural, calvarial, extraneuraxial, or intraosseous meningiomas. To avoid the confusion in nomenclature, Lang et al. has proposed a single term, primary extradural meningiomas (EDMs), for such lesions [3].

Primary EDMs have an incidence of less than 2 % of all meningiomas [3–7] and have been reported to arise from the scalp tissue, orbit, paranasal sinuses, nasopharynx, salivary glands, neck, skin, lung, mediastinum, adrenal and paraspinal regions, and even the fingers [3–5]. Primary EDMs have a slight female preponderance (1:1.4) with a bimodal age distribution, the first peak being in the 2nd decade and the next in the 5th to 7th decades [1, 3–5].

Intraosseous meningiomas are exceedingly rare, forming 14 % of all cases of primary EDMs [6, 7]. The calvarium is involved in 68 % of intraosseous meningiomas, with the fronto-parietal and orbital regions being the commonest locations [1, 3, 5, 6].

Several explanations are proposed in the literature for the probable origin of ICMs. They are thought to arise from ectopic meningocytes or arachnoid cap cells trapped in the cranial sutures during molding of the head at birth. A second explanation is post-traumatic misplacement or entrapment of meningothelial cells into suture or fracture lines. A third explanation is that meningiomas may develop from multipotential mesenchymal cell precursors, thus explaining the occurrence of meningiomas in uncommon locations [1, 5, 6]. An association of ICM with neurofibromatosis type II has been reported in a case by Poptodorov et al., in which ICM and spinal meningioma were the initial manifestations of this disease [8].

The commonest presenting feature of ICM is a gradually expanding painless mass [1, 3–5, 7], usually benign and slow-growing. However, it is reported that ICMs are more prone to developing malignant changes

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than intracranial meningiomas, with a reported incidence of 11 % and 2 % respectively [3, 5].

Osteoblastic lesion is the most common radiographic feature of ICMs, seen in 59 % of cases. Osteolysis is reported in 35 % and a mixed osteoblastic–lytic lesion in 6 % of cases [1, 5, 7]. An osteolytic lesion with an extracranial soft-tissue mass suggests an aggressive nature and a suspicion of malignancy [5]. CT with a bone window is necessary for the detection of the osteoblastic lesion and exclusion of any cortical destruction. However, MRI provides better anatomical delineation and evaluation of the soft tissue component. The most common MRI appearance is diffuse hypointense signal in the expansile lesion on T1 sequences and variable heterogeneous signal on T2 sequences [4, 5].

Intraosseous calvarial meningiomas can be differentiated from the hyperostotic en plaque meningiomas, by the latter showing a sheet-like dural lesion along the inner table of the skull associated with disproportionate large bone hyperostosis [9]. Other differential diagnoses of osteoblastic ICM include fibrous dysplasia, osteoma, and osteoblastic metastasis. Rarer conditions include Paget's disease, osteosarcoma, and ossifying fibromas [1, 2, 6, 9, 10].

Total wide surgical tumor resection and cranioplasty is the treatment of choice. If only subtotal resection is possible because of the involvement of critical structures within the orbit, paranasal sinuses, or skull base, then the residual tumor should be followed radiologically. Adjuvant radiation therapy is recommended in patients whose residual lesions are symptomatic and/or show evidence of malignancy [1, 2, 4, 10].

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