Atypical lytic lesions of skull: Clinical and radiological correlation

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Abstract

Imaging alone cannot differentiate various isolated atypical lytic lesions involving the skull. Clinical and radiological correlation is mandatory in reaching to a diagnosis. Histopathology remains the gold standard. We describe few atypical cases presenting as isolated lytic lesions of skull with characteristic imaging findings and a brief clinical approach to reach towards the diagnosis.

Key Words

Lytic lesion, skull, soft tissue

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Introduction

Skull or the calvarium encases the brain parenchyma. It consists of an inner table, outer table and the space between the two layers is known as diploe. The thicknesses of outer and inner table are 1.5 mm and 0.5 mm, respectively. Focal lesions in skull may arise from bony structures, or they may be secondary to invasion of the skin — or brain-based lesions into bony structures. Benign lesions have well-defined borders and sclerotic margins whereas malignant lesions lead to destruction. Plain radiography is the first step in radiological evaluation followed by computed tomography (CT) and magnetic resonance imaging (MRI) if required. Imaging alone cannot differentiate various isolated atypical lytic lesions involving the skull. Clinical and radiological correlation is mandatory in reaching to a diagnosis. Histopathology remains the gold standard. We describe few atypical cases presenting as isolated lytic lesions of skull with characteristic imaging findings and a brief clinical approach to reach towards the diagnosis.

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Discussion

Isolated involvement of skull by a lymphoma usually occurs in immunocompromised patients. On imaging it is seen as a hyperdense mass on non-contrast images showing marked enhancement after contrast administration with bony destruction [Figure 1]. Histology shows lymphoid cells scattered in diffuse pattern. Immunohistochemistry is essential for further typing and characterization. Differentials include osteomyelitis and metastases. In osteomyelitis, fever and leukocytosis or other signs of inflammation are usually associated which help to clinch the diagnosis [Figure 2]. Histological examination in osteomyelitis reveals dead bone, inflammatory cells, granulation tissue and small vessel thrombosis. CT examination of chest and abdomen should be done in all the cases of lytic skull lesions to rule out a primary malignancy in the lungs and in the intra-abdominal organs [Figure 3].[1]

Hemangiopericytoma needs to be differentiated from meningiomas. On imaging, hemangiopericytoma appears as lobulated hyperdense mass with bone erosion without any evidence of calcification and hyperostosis [Figure 4]. Histological examination of hemangiopericytoma reveals uniformly cellular, variably ecstatic or compressed thin-walled branching vessels with gaping sinusoidal spaces (staghorn configuration). Tumor cells are spindled to round with small amounts of pale or eosinophilic cytoplasm, indistinct margins; bland vesicular nuclei. Meningiomas typically show a broad-based dural attachment on CT and MR studies whereas hemangiopericytoma has a narrow attachment with heterogeneous enhancement on post-

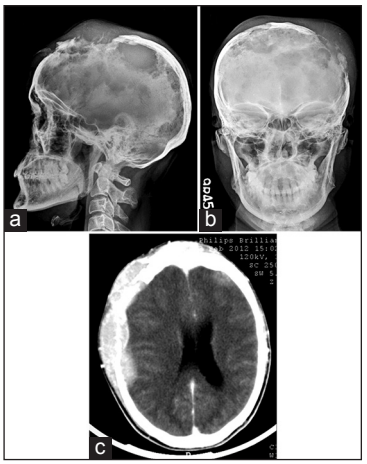

Figure 1: A 40-year-old HIV-positive male presenting with headache and swelling in scalp caused by lymphoma (a) Lateral and (b) AP skull radiograph showing destruction of calvaria with a soft tissue component (c) Contrast-enhanced CT scan head shows erosion of calvaria in the right frontal region with homogenously enhancing lobulated dural-based soft tissue component causing mild midline shift

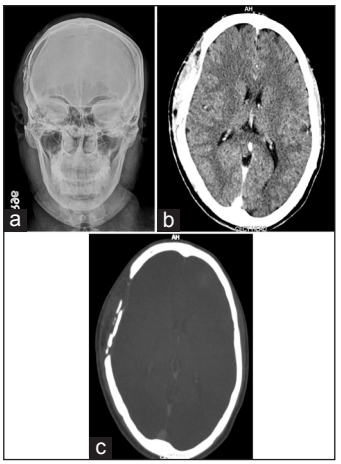

Figure 2: A 17-year-old male with history of fever and tenderness in scalp caused by osteomyelitis (a) Skull radiograph AP view showing irregularity and erosion of right sided calvaria with soft tissue swelling (b) Contrast-enhanced CT head shows mild heterogenous enhancement of soft tissues in right frontal region (c) CT bone window showing irregular erosion of outer and inner calvaria

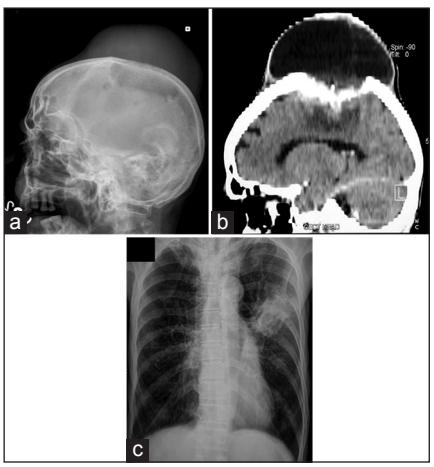

Figure 3: A 50-year-old male with scalp swelling and chest pain caused by metastasis from lung primary (a) Skull radiograph lateral view shows lytic lesion with large soft tissue component (b) Contrast-enhanced CT sagittal reconstructed image shows destruction of calvaria with large soft tissue component causing underlying oedema in the brain (c) Chest radiograph shows malignant mass in the left lung

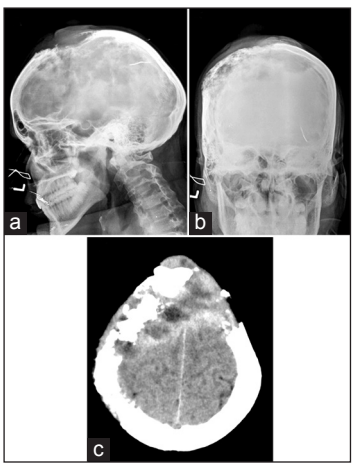

Figure 4: A 39-year-old female with scalp swelling, malaise and headache caused by hemangiopericytoma (a) Lateral and (b) AP skull radiograph shows irregular lytic lesion of skull with soft tissue (c) Contrast-enhanced CT scan shows heterogenously enhancing lobulated soft tissue with underlying bony destruction and no evidence of calcification or hyperostosis

contrast images. Histopathology of meningiomas depends on the grade. Depending on the grade, there may be presence of mitotic figures, sheeting, prominent nucleoli, hypercellularity, and the formation of small cells. Prominent internal flow voids may be seen on T2- weighted images in hemangiopericytoma.[2]

Squamous cell carcinoma of scalp is seen in fifth to seventh decade of life and is a common cancer among Caucasians. Involvement of skull although rare, it is seen in organ transplant recipients with alopecia, HIV-infected patients, and patients with scleroderma or burn scars of the scalp.[3] Heterogenous soft tissue component predominantly towards the outer surface of calvaria and early involvement of superior sagittal sinuses are more commonly seen in appendageal squamous cell carcinoma of scalp [Figure 5].[3,4] Histology reveals prominent dyskeratosis, aberrant mitoses in epidermis along with parakeratosis.

Langerhans cell histiocytosis is more common in the pediatric age group. Involvement of skull in Langerhans cell histiocytosis is seen classically as solitary or multiple punched out lytic lesions with or without sclerotic rim. Edges are bevelled due to greater involvement of the inner than the outer table and lytic lesion with button sequestrum representing residual bone or geographic skull may be seen [Figure 6].[5] Histopathological examination reveals presence of histiocytes "LCH cells", macrophages, lymphocytes, eosinophils, giant cells and less commonly plasma cells and neutrophils. LCH cells may be abundant or scarce.

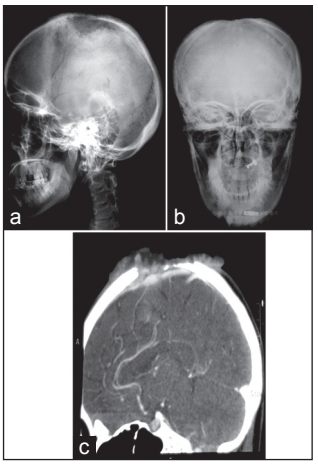

Figure 5: A 57-year-old male with scalp growth caused by skin appendageal carcinoma of scalp. (a) Lateral and (b) AP skull radiograph shows irregular massive lytic lesion of skull in the midline (c) Contrast-enhanced CT scan head with sagittal reconstruction image showing irregular soft tissue component more towards the outer surface with superior sagittal sinus involvement and bony destruction

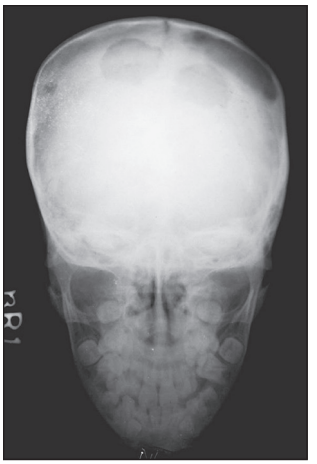

Figure 6: A 12-year-old male presenting with pancytopenia in a case of histiocytosis. Skull AP radiograph shows multiple lytic lesions with irregular geographic margins and no soft tissue component

Teaching Point

Plain X-ray, CT and MRI are complementary tools for evaluating skull lesions. Patient age, history of trauma or a primary disease, enhancement pattern, presence of destruction or expansion, and the multiplicity of lesion should be taken into consideration. Biopsy and histopathological confirmation is the gold standard for definite diagnosis.

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