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Osteolytic Lesions of Skull in Pediatric Age Group

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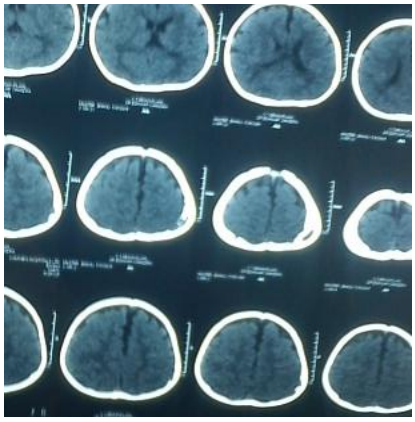
Abstract: *Lytic lesions of the calvarium in pediatric age group have a wide range of different etiologies, ranging from normal variants to congenital, traumatic, inflammatory and neoplastic lesions,They manifest as palpable masses, with or without associated pain. Imaging approach frequently begins with CT and MR being complementary and the method of choice for the assessment of bone and associated soft tissue masses. We are hereby reporting two such cases with review of literature.*
Keywords: *lytic lesion of skull, calvaria, eosinophilic granuloma, fibrous dysplasia.*

I. Introduction

Primary skull lesions of skull are estimated to amount for 0.8% of all bone tumors.Dermoidandepidermoid account for 20-60% of all reported tumors with other lesion making up theremainder.tumors of skull in children present as painless bumps , with benign lesions having long history while malignant lesions are multiple and are more aggressive. Local pain and tenderness are less commonly seen [8]Imaging is advisable in most of the lesions, CT scan is the imaging modality of choice for bony lesions , it allows better evaluation of bony erosion or invasion and involvement of both inner and outer tables MRIis recommended when intracranial extension is suspected.definitive diagnosis.we are here discussing two such lesions in pediatric age group.

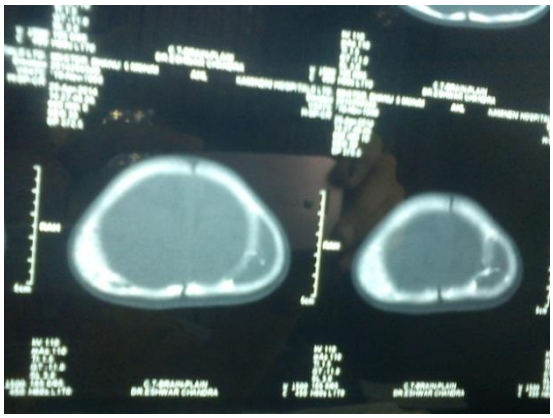
Case 1

Eight month old male child presented with progressive swelling of parietal region of head since two months as noticed by the parents.not associated with pain or irritability .no history of constitutional symptoms on examination child was active , appropriate milestones for his age, local examination showed diffuse bony swellings of parietal bones, non tender. Routine blood investigation was normal



Legend 1

CT scan of brain with bony windows was done which revealed bilaterallparietal bone had osteolytic lesion with ground glass appearance with no involvement of brain parenchyma

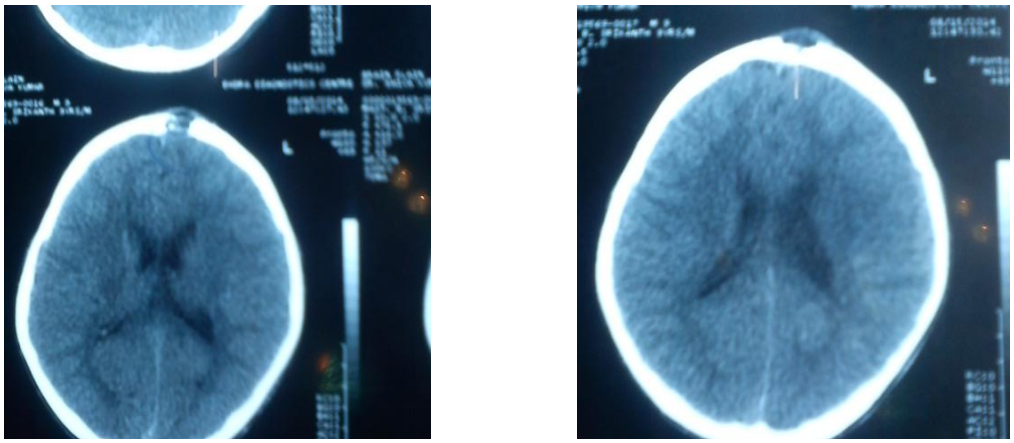


Legend 2

CT scan of the calvaria showing lytic lesion in left parietal bone and ground glass appreancein Right parietal boneChild was subjected for excisional biopsy , interoperatively there was widening of diploeccavity with pale granulation tissue in the spaceHistopathology was suggestive of possibility of fibrous dysplasia
Child is under follow up and is doing fine

Case 2

A 10 year old boy presented our department with a painful swelling over midline of the forehead for 30 days. There was no history of trauma, headache or vomiting. Systemic examination was unremarkable. Local examination revealed a circumscribedswelling of 4*4cm in diameter in midline in the frontal region which was hard, not mobile and tender. underlying bone was eroded The overlying skin appeared normal. Routine hematological and biochemical investigations were within normal limits.



Legend 3

CT scan of brain with bony windows revealed circumscribed osteolytic lesion in the midline of frontal bone with erosion of both outer and inner table with irregular bony edgesper operative the lesion was seen below the scalp , reddish lesion in diploe cavity of the bone with eroded outer and inner table extending upto the dura but not infiltrating it. Curettage of lesion was done HPE was suggestive ofeosinophilic granuloma

II. Discussion

Osteolytic skull lesions may have many different causes, anatomical variations beingresponsible for up to 60% of casesIn general population, seven diagnosis include 85% of all causes - by decreasingorder,

dermo/epidermic cysts,
hemangioma
,metastasis,
multiple myeloma
,Langheranshistiocytosis,
Paget disease of bone
fibrous dysplasia

In adults, tumoral causes are predominant - metastasis and myeloma [8] - whereas inchildren, besides congenital defects, dermoid cysts and eosinophilic granuloma are themost frequent diagnosis.[7 benign lesions tend to have well-defined borders with sclerotic margins,a quite predictable location, mostly near the midline, and are usually solitary. On theother hand, those with a permeative appearance, multiple and randomly distributed, areprobably aggressive.In adults and elderly patients, metastases are by far responsible for the majority of lytic skulllesions, whereas children and young adults present more frequently one of the congenital, inflammatory, traumatic or benign neoplastic conditions mentioned below

Normal Variants
Transcalvarial venous lakes and venous channels
Arachnoid granulations
Enlarged parieatal foramina

Congenital
Meningocele CSF lined by meninges
Gliocele CSF lined by glial tissue
Meningoencephalocele CSF and brain
Meningoencephalocystocele CSF, brain and ventricules
AtreticcephaloceleSinus percranii

Traumatic
Skull defects
growing skull fracture orlepto meningeal cyst
inflammatory
eosinophilic granuloma
sarcoidosis
Infective
Osteomyletis
Tuberculosis
Benign lesions
fibrousdyspasia
Dermoid
epidermoid cysts
hemangioma
Osteoma
Ossifyingfibroma
Malignant
neuroblastoma
osteoblastoma
ewings sarcoma
fibrosarcoma
angiosarcoma
lymphoma
metastasis [8]

fibrous dysplasia is a benign congenital disorder in which there is osteoblastic dysfunction causing replacement of normal bone with woven bone,lesions have ground glass appearance in CT scan less commonly cystic or sclerotic is also seen this condition is seen in children and young adults.[9]

Eosinophilicgranuloma is a rare entity in frontal bone , hence discussed with review of literature Eosinophilic granuloma (EG) is a rare bone tumor, representing less than 1% of all bone tumors The term "eosinophilc granuloma" was first introduced by Lichtenstein and Jaffe in1940. Eosinophilic granuloma is the benign form of three clinical variants of LangerhansCellHistiocytosis; the other two variants are Lettere-Siwe disease and HandSchullerChristian disease[1]Langerhans Cell Histiocytosis (LCH) is a rare disease with unknown etiology. The incidence of LCH is estimated to be 0.2-0.5 cases per 100.000 per year. Bone is the most frequent site of this disease. It is usually considered to be a disease of childhood. Many patients are 1-15 years old, however the diagnosis frequently is made in adults and many cases of childhood onset progress into adult life.[2]The etiology

of LCH is unknown and there is continuing debate whether this condition is neoplastic or non neoplastic Inflammation, autoimmunity and loss of controlled proliferation of Langerhans cells are assumed etiologies

Table I. The staging system proposed by Greenberger et al.1981

stageI	a) Single monostotic bone lesion
	b) Multiple lesions in one or multiple bone
stageII	>24 months of age at diagnosis and having one of the following systems involved: diabetes insipidus, teeth, gingivae, lymph nodes, skin, mild lung involvement (i.e., infiltrates seen on chest radiograph without pulmonary symptoms or gross consolidation), focally positive bone marrow
Stage III	a) Age <24 months at diagnosis with any of the systems involved in stage II
	b) Age >24 moths with involvement of liver and/or spleen, massive nodal involvement (nodes > 5 x 5 cm in several sites above or below diaphragm), honeycomb lung (major involvement in all areas with apparent fibrosis), bone marrow packed
Stage IV	Spleen > 6 cm (palpable below costal margin) and fever >1 month with or without any or all of the above systems involved
Stage v	"Special" monocytosis in peripheral blood > 20% of differential cell count, in addition to stage III or IV

The staging system of Langerhans cell histiocytosis proposed by Greenberger et al has classified lesions primary limited to the bone as stage Ia or Ib (Table I).About 80% of patients show bone lesions at diagnosis and about 40% of them havethe disease limited to the bone.

Table II. Anatomic distribution and frequency of bone lesions in LCH

parietal	17%
Frontal	9.5%
Occipital	9.5%
Temporal	7.5%
Sphenoid	2.8%
Maxilla	0.9%
Mandible	13.3%

Solitary eosinophilic granuloma of skull is a rare condition,the natural history of whichhas not been defined completely .Characteristically the patient notices an enlarging tender skull mass for weeks to months; most commonly seen in parietal bone of the skull.Our patient had involvement of frontal bone with a gradual increase in size of swelling.Eosinophillic granuloma of any skull bone can be within diploe may or may not compress brain parenchyma without dural infiltration or it can be projecting into the cranial cavity compressing the brain parenchyma with dural infiltration [3]EG can be asymptomatic or can present as localized pain, tender swelling and fever. Rarely does it present with epidural hematomas, suppression of bone marrow and pathological fracture. Headache, neurological symptoms, chronic mastoiditis and exophthalmos may be seen in cases of skull lesion[4]langerhan cells of eosinophillic granuloma express HLA-DR, S-100 and CD1a.

Unlike normal resident dendretic cells, it is the co-expression of CCR6 and CCR7 which allows the langerhan cells to migrate into the tissues that express the relevant chemokine-CCL20 in skin and bone (ligand for CCR6)and CCL19and 21 in lymphoid organs (ligands for CCR7).Langerhan cells can cause osteolysis by elaboration of interlukin-1 and prostaglandin-E2. Histopathological examination of the lesion provides definitive diagnosis of the condition. The lesions cellular components includepathologiclangerhan cells, chronic inflammatory cells and eosinophils.Radiograph and computed tomography shows extensive frontal bone destruction[5]generally accepted treatment of choice for solitary bone lesions, especially for these Affectingcalvaria, is surgical excision when the lesion is readily accessible. Data of many authors indicate that surgical curettage is a very successful treatment. If necessary, excision is combined with concurrent bone grafting.However some authors report the higher risk of local recurrence after surgery alone.

Persistence symptoms of disease, or expansion of the lesion aftersurgical intervention, may respond to the subsequent radiotherapy [1],[6]Eosinophillic granuloma of bone has been reported to resolve after low dose irradiation ,intralesional corticosteroids, simple biopsy, subtotalcurettage with or without postoperative low dose radiotherapy or chemotherapy and craniectomy withcranioplasty for lesions >4cms[3]. Systemic evaluation and long term follow-up afterany kind of treatment modality is mandatory in every case.

III. Conclusions

Calvarialtumors in child hood age group runs from benign to malignant disease, they have to be carefully evaluated with imaging and surgical excision is usually mainstay of treatment in most of lesions.

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