INTRAOSSEOUSLIPOMA OF THE FRONTAL BONE : CT – MRI DISCORDANCE

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ABSTRACT

Intraosseouslipoma is a rare, benign mesenchymaltumor that is frequently found in the appendecular skeleton, involving the calcaneus[1] and the metaphyses of long bones. Intraosseouslipoma of the skull is even rarer with only a handful of cases described in the literature. In course, they usually undergo varying degrees of involution with necrosis, cyst formation, and calcification. Here we report a case of a 8 year old girl, who presented with a hard bony swelling of the calvarium on the left side for the past 3 months. A preliminary non contrast Computed Tomography (CT) scan was suggestive of fibrous dysplasia. However a Magnetic resonance imaging (MRI) scan revealed an expansile lesion of the frontal bone on the left side which was homogeneously hyperintense on T1 and T2 weighted images with uniform suppression on fat saturated images, which was suggestive of Milgram stage 1 intraosseouslipoma. The above mentioned CT-MRI discordancelead to subsequent biopsy of from the lesion, which confirmed our diagnosis of an intraosseouslipoma.

Key words: Intraosseouslipoma, frontal bone, Magnetic resonance imaging (MRI), Computed Tomography (CT)

Case Report

A 8 year old girl presented to us with a hard swelling of the left side of her calvarium for the past 3 months. She had no other significant complaints. A preliminary non-contrast Computed Tomography (CT) scan of her brain, done outside, revealed an expansile lesion in the left side of her frontal bone with homogeneous ground glass opacity (Hounsfield unit of 180 to 200) and without any obvious foci of fat attenuation, the appearance suggestiveof fibrous dysplasia (Fig. 1). She subsequently underwent an MRI scan of her brain which revealed the same large expansile in tradiploeic lesion in the left side of the frontal bone. The lesion was uniformly hyperintense in both T1 and T2 weighted images (Fig. 2 & 3) with complete suppression on Coronal T1 fat suppressed sequence (Figure 4). No diffusion restriction was seen in the lesion. The above findings were consistent with Milgram stage1 intraosseouslipoma. No obvious signal intensity changes in the underlying brain parenchyma was noted. The CT- MRI discrepancy prompted us for a biopsy, which revealed that the lesion was composed of mature lobular adipocytes with no evidence of cellular atypia,mitoses, calcification or necrosis and without any admixed bony trabeculae, suggestive of benign intraosseouslipoma.

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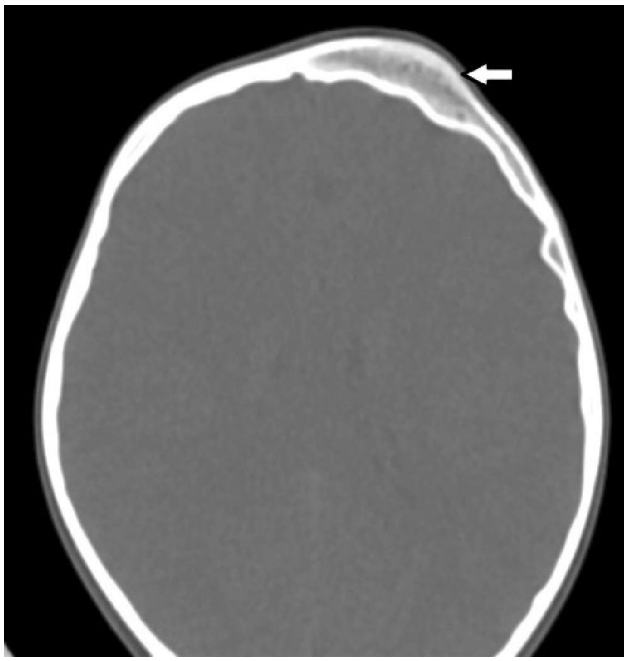

Figure 1: Axial CT scan of the Brain (bone window) shows an expansile lesion (arrow) with homogeneous ground glass opacity in the left side frontal bone.

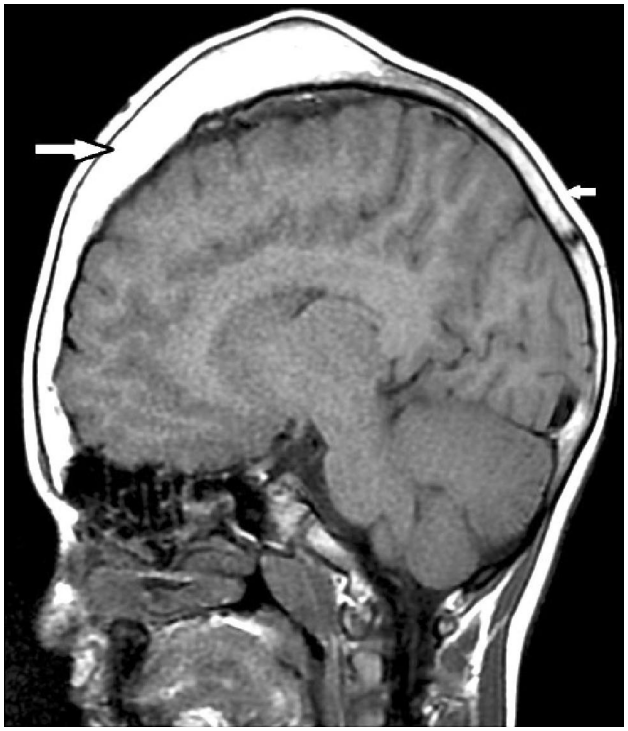

Figure 2: Sagittal T1 (TR/TE, 200/15 ms) image shows an expansile, homogeneously hyperintense intradiploeic lesion (large arrow) involving the frontal bone, having similar intensity to subcutaneous tissue (small arrow).

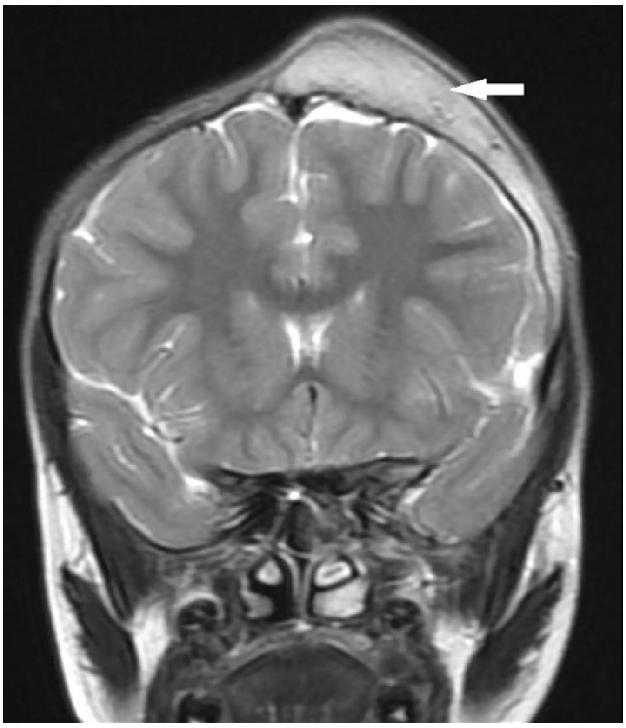

Figure 3: Coronal T2 (TR/TE, 3200, 80 ms) image shows the expansile lesion involving the left side of frontal bone (arrow), which is homogeneously hyperintense.

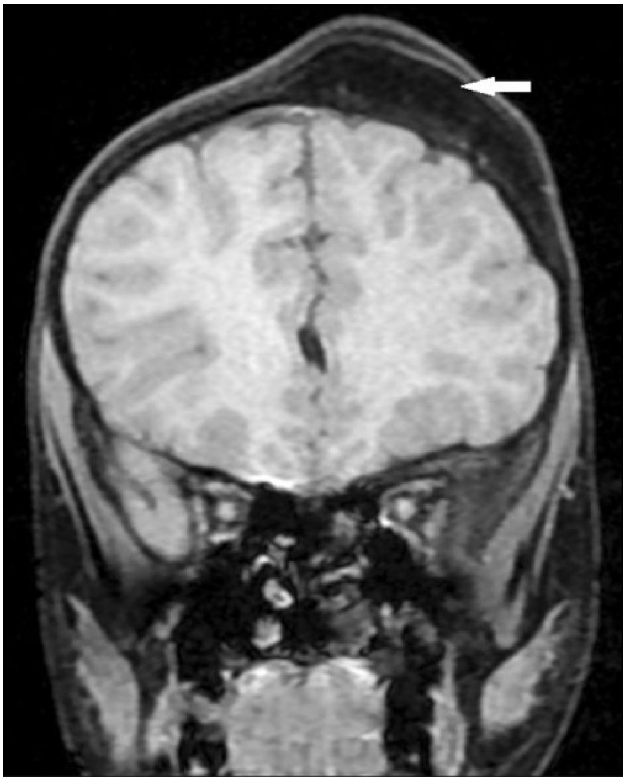

Figure 4: Coronal T1 FS (TR/TE, 3200, 80 ms) image shows that the lesion uniformly suppresses on fat saturated sequence, suggestive of its fat containing nature.

Discussion

Intraosseouslipoma, first reported in 1880[1] is a rare benign tumor, accounting for approximately 0.1% of bone tumors. The lesions are usually located in the lower limb (71%), involving the calcaneus in about 30% of cases. Other frequent locations include subtrochanteric and intertrochanteric regions of the femur, pelvis and flat bones. Rare involvement of the cranial bones is seen in only 4% of cases.[1,2] Till date only a handful of cases involving the frontal bone, have been reported.[3]

The intraosseouslipomas are typically caused by a proliferation of mature lipocytes within the marrowof normal trabecular bone.[4] They are usually encountered in the 4th or 5th decade, although it can present in an age range of 5 to 85 years. Males are affected slightly more than females.[5] These tumors have low potential for malignant transformation. Three sources for intraosseouslipomas have been described - fat in the medullary cavity of long bones(medullary lipoma), periosteum (parosteallipoma) and surrounding soft tissue with subsequent bone infiltration (soft tissue lipoma).[6]

Milgramdivided intraosseouslipomas into 3 stages based on their respective histology, reflecting the degree of involution of the lesion.[5] The radiographic features of intraosseouslipomas often parallel those of the histologic stage.[7] The stage 1 lesions contain viable non-necrotic fat with resorption of bony trabeculae and show expansion or remodeling in half of the cases.[5] Stage 2 lesions contain viable lipocytes which contain foci of calcification (secondary to fat necrosis). Stage 3 lesions reflect resorption of normal bone with thick sclerotic borders, containing diffuse areas of infarction, cyst formation and dystrophic calcification.[4,7]

On CT, stage 1 intraosseouslipomas exhibit resorption of bone trabeculae with bone expansion, areas of fat attenuation (HU of -60 to-100) and mild marginal sclerosis. Stage 2 lesions have areas of fat with variable amount of central and peripheral areas of calcification or ossification. Stage 3 intraosseous lipomas show thick marginal ossification with variable amounts of central calcification on radiographs. With progressive ischemia and involution, the fat undergoes cystic degeneration. Thus a heterogeneous expansile lesion with central calcification surrounded by a rim of peripheral fat (bull'e eye pattern) is diagnostic.[5,7] On MRI, stage 1 intraosseouslipomas contain fat, which is homogeneously hyperintense on both T1 and T2-weighted sequences (identical to subcutaneous fat) with complete suppression of fat suppressed sequences. A thin circumferentialrim of low signal intensity on T1 and T2 weighted sequences is typically present demarcating the margin of the lesion, consistent with reactive sclerosis. In stage 2 lesions, one can again identify fat and the circumferential rim of marginal sclerosis. Low-signal- intensity areas within the central portion ofthe lesion on T1- and T2-weighted images are suggestive of calcification. Stage 3 lesions show a thin peripheral rim of fat, with central calcification and a thick rim ofsurrounding sclerosis, which have low signalintensity on T1- and T2-weighted sequences.Areas of infarction with necrosis and cystic degeneration have a variable signal onT1-weighted and increased signal on T2-weighted images.[4,7] No contrast enhancement is seen in post contrast study.

The differential diagnosis of calvarial intraosseous lipoma that need consideration include fibrous dysplasia, intraosseous meningioma, hemangiomas, angiolipoma and chondroidtumors.[8] Bone infarcts can closely resemble Milgram stage 3 intraosseouslipoma. A non expansile lesion with peripheral calcifications and thin serpentine rim of sclerosis surrounding central fat and without trabecular resorption is suggestive of bone infarct rather than intraosseouslipoma.[7] Rarely, a fatty marrow island within an enlarged normal cancelousbone may also mimic an intraosseouslipoma.[1]

Conclusion

Intraosseouslipoma of the frontal bone is indeed very rare. However modern imaging techniques like CT and MRI have revolutionized the diagnosis of the very rare tumors, even without the need for histopathological correlation. However the CT-MRI discrepancy in our case prompted us for a biopsy which indeed proved to be a stage 1 intraosseouslipoma. To our knowledge , this CT – MRI discordance of an intraosseouslipoma has not been previously described in literature which prompted us to report this very rare entity.

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