# Increased bone mineral turnover without increased glucose utilization in sclerotic and hyperplastic change in fibrous dysplasia

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Fibrous dysplasia is a benign bone disorder. It is diagnosed by distinctive X-ray radiography, CT, and MRI findings. Although bone scintigraphy helps to identify the tumor origin according to accelerated bone turnover, the glucose metabolism in fibrous dysplasia has not yet been investigated. We reported a case of fibrous dysplasia in craniofacial bone which showed signs of the acceleration of bone mineral turnover without elevated glucose utilization by Technetium-99m-HMDP SPECT and Fluorine-18-FDG PET. We concluded that the growth of fibrous dysplasia needed the acceleration of bone mineral turnover without an increase in glucose metabolism.

**Key words:** fluorine-18-fluorodeoxyglucose, positron emission tomography, fibrous dysplasia, Tc-99m-hydroxymethylene diphosphonate

## **INTRODUCTION**

FIBROUS DYSPLASIA has been defined as a developmental anomaly of the bone in which the medullary cavity is replaced by fibrous connective tissue. Recently, magnetic resonance imaging (MRI), 1-3 as well as X-ray radiography and X-ray computed tomography (CT), 4 has been used as a tool for diagnosing fibrous dysplasia which need to be differentiated from other types of tumors. Although bone scintigraphy with Technetium-99m-phosphate complexes have shown increased bone mineral turnover in fibrous dysplasia, 5.6 glucose metabolism in this tumor on positron emission tomography (PET) with Fluorine-18-Fluorodeoxyglucose (FDG) has not been demonstrated. We measured the glucose metabolism of fibrous dysplasia where bone mineral turnover was incresed.

Received December 11, 1997, revision accepted March 4, 1998.

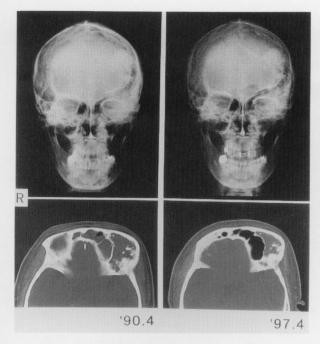
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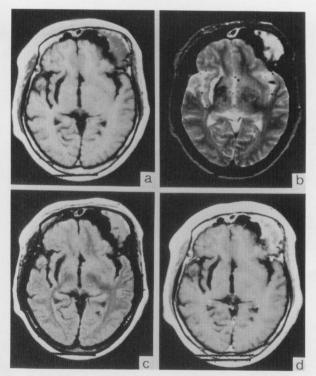
# **CASE REPORT**

Seven years previously, a 48-yr-old woman presented with a deformity of the left facial bone without tenderness. X-ray radiography and CT (Toshiba, X vigor) of the skull showed sclerotic and hyperplastic change in the left maxillary and frontal bone without periosteal reaction. Seven year follow-up studies with CT and X-ray radiography strongly indicated fibrous dysplasia (Fig. 1). The signal of the tumor core in the left craniofacial bone showed low to intermediate on the T1-weighted image (TR/TE: 570/15) (Fig. 2-a), high on the T2-weighted image (2500/90) (Fig. 2-b), and intermediate intensity, which was the same intensity as for the cerebral gray matter, on the proton mobile-weighted image (2500/15) (Fig. 2-c) with MRI (Siemens 1.0T, Magnetom Impact). The tumor in the marginal area was considerably enhanced but not that in the core area on the T1-weighted image after an intravenous Gd-DTPA injection (Fig. 2-d), and this finding suggested abundant cell component in the tumor. An old focus was also identified in a right putaminal hemorrhage.

We investigated bone mineral turnover and glucose metabolism of fibrous dysplasia in the left craniofacial bone. Three hours after an intravenous injection of 740 MBq Tc-99m-hydroxymethylene diphosphonate (HMDP), whole-body image and head images by means of single



**Fig. 1** X-ray radiography and X-ray CT scans reveal sclerosis and hyperplasia in the left facial bone without significant change over 7 years except for surgery of maxillary sinus cyst.



**Fig. 2** The signal intensity of the tumor in left craniofacial bone is low to intermediate on T1-weighted image (a), high on T2-weighted image (b) and intermediate intensity on proton mobile-weighted image (c). The intensity in the marginal area of the tumor is considerably enhanced after Gd-DTPA injection on T1-weighted image (d).

photon emission computed tomography (SPECT) were obtained with a dual head gamma camera [ADAC, Vertex; transaxial resolution of 8.5 mm full width at half maximum (FWHM)]. After 5 hours fasting, FDG study was performed 40 minutes after an intravenous injection of 185 MBq FDG and 7 contiguous slices of the head were obtained with ring shaped PET camera (Shimadzu, Headtome; transaxial resolution of 6.0 mm FWHM). For the semi-quantitative analysis of FDG accumulation, the standardized uptake value (SUV; the tissue activity of FDG per body weight)<sup>7</sup> was calculated over the tumor. HMDP intensively accumulated in the left craniofacial bone extending to the opposite side in transaxial SPECT images and the whole body image was normal except for abnormal accumulation in the craniofacial bone. No uptake of FDG was demonstrated in the left craniofacial bone in FDG-PET images (Fig. 3) with SUV being 0.58 and 0.60, in the involved and contralateral normal sides, respectively. We concluded that bone mineral turnover was accelerated without increased glucose utilization in sclerotic and hyperplastic change with fibrous dysplasia.

## DISCUSSION

Fibrous dysplasia is a benign disorder of unkown cause in which the normal bone structure is replaced by fibrous connective tissue. Once the craniofacial bone is involved, patients have noticeable facial deformity. Radiography and X-ray CT4 can reveal the characteristic sclerotic and hyperplastic change in bone in fibrous dysplasia. Furthermore, MRI findings can help to assess the amount of cellular components involved that can indicate malignancy. 1-3 On the other hand, the activity of phosphate complexes visualized by bone scintigraphy might reflect the bone mineral turnover including calcium. Furthermore, FDG-PET could measure increased glucose utilization which might indicate malignancy in brain tumors8.9 and musculoskeletal10,11 regions. Although abnormal accumulation of phosphate complexes in the sclerotic and hyperplastic areas of fibrous dysplasia due to increased bone mineral turnover have been reported, 5.6 it has not yet been investigated whether the glucose utilization increases in fibrous dysplasia. We therefore examined FDG-PET for the assessment of glucose metabolism in the present case of fibrous dysplasia.

The fibrous dysplasia described here did not change for 7 years according to X-ray radiography and CT, and MRI revealed abundant cell components. HMDP accumulated in the marginal area of fibrous dysplasia as previously described, <sup>5,6</sup> but FDG did not in the same region. We could therefore conclude that fibrous dysplasia needed an acceleration of bone mineral turnover without an increase in glucose metabolism, suggesting a better understanding of histochemical behavior in the growth of fibrous dysplasia.

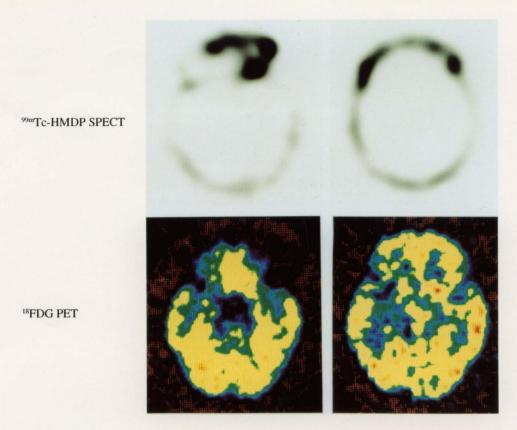


Fig. 3 No FDG accumulated in the left craniofacial bone where HMDP was accumulated intensively.

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Vol. 12, No. 3, 1998 Case Report 155