A CASE OF FIBROUS DYSPLASIA OF BONE

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Clinical history and examination

A TWENTY-FOUR year old soldier was seen on 4 March 1969 with a two month history of cramp-like pains in the right thigh and front of right leg. Pain was intermittent, aggravated by standing for more than a half hour, or walking more than a half mile.

Five years ago he had received a direct blow to the right tibia in a traffic accident and since then he had noticed two lumps on the right shin bone. On examination two bony lumps were felt along the subcutaneous border of the right tibia. They were not tender. There was no evidence of vascular insufficiency to the limb, and no evidence, on exercise, of the anterior tibial syndrome. Neurological examination was normal.

Investigations

An X-ray of the right leg was taken (Fig. 1). There were cystic areas in the shafts

![Image](http://jramc.bmj.com/)

of the right tibia and fibula both within the cortex and the medulla of the bones. In places the cysts had expanded the cortex to egg-shell thinness. There was some posterior bowing of the right fibula in its lower third. Skeletal survey revealed no cysts in any other bone.

The results of other findings were—Serum Calcium 10.2, 10.3 and 11.6 mg/100 ml.

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"Inorganic" Phosphate 3.7 and 4.3 mg/100 ml: Alkaline Phosphatase 10.5 K.A. units/100 ml: 24 hour urine Calcium excretion of 243 mg: w.b.c. 9,200 cu mm: e.s.r. 3 mm/hour: Urine—no protein.

A diagnosis of fibrous dysplasia of bone was made, and a bone biopsy of the upper tibial lesion was taken on 20 March 1969.

Pathological findings

The original cortical bone was eroded by several expanding lesions to the medullary cavity of the shaft of tibia and fibula. The curetted specimen was gritty and firm in consistency and whitish grey in colour. There was no evidence of cartilaginous or cystic elements in this tissue.

Microscopically this lesion was a blend of osseous and fibrous tissue, their relative proportions varying in various parts of the section. The fibrous element was composed of spindle cells forming interlacing bundles interspersed with newly formed bone trabeculae of irregular contour, which did not show surface osteoblasts or orientation along the lines of stress. Cement lines, cartilage, cysts or foam cells were not observed in these sections.

Discussion

There are two types of fibrous dysplasia: the polyostotic or mono-ostotic variety in which there is only a skeletal abnormality present and Albright's syndrome, in which additional hormonal defects may be found like precocious puberty in females, premature skeletal maturation and hyperthyroidism. The former is more common. These malformations are not true neoplasms and they seldom undergo malignant change.

It has been claimed by Harris, Dudley and Barry (1962) and Reed (1963) that fibrous dysplasia is a maturation defect, in that the collagenous fibres never become transformed into lamellar bone as shown by silver stains. Appositional osteoblasts do not appear on the surface of the bony spicules and there is no tendency to recurrence; this differentiates it from ossifying fibroma.

The only occasional abnormal blood finding in fibrous dysplasia is a raised alkaline phosphatase activity. Unlike hyperparathyroidism, calcium is never raised in the serum and calcium and potassium balance studies are within normal range (Lechtenstein and Jaffe 1958).

On clinical grounds enchondromatosis of bones of one limb may raise diagnostic problems. Punched out appearances on X-ray as opposed to the ground glass picture of fibrous dysplasia and the finding of cartilage occupying the interior of the bone differentiates enchondromatosis from fibrous dysplasia.

The bone cyst shadow in an X-ray of enchondromatosis is more uniformly translucent and the pathological specimen is haemorrhagic containing fibrin clots, delicate bone trabeculae, foam cells and giant cells as opposed to the more fibro-osseous lesion of fibrous dysplasia.

In lipid granulomata the skeleton is more widely affected and foam cells are present in great abundance.
Conclusion

Fibrous dysplasia is a disease of growing bone and most often presents between the ages of ten and fifteen years. Clinically the presenting symptom is pain, swelling or pathological fracture.

The course of the disease is relevant to the problem of treatment. It is generally believed that the disease becomes inactive after puberty. According to figures from Bristol Bone Tumour Registry (Henry 1969) in a series of 50 cases, 24 presented after puberty, but only 6 after the age of thirty.

Surgical treatment is directed towards the prevention of deformity, to fix a fracture internally or to remove the pathological tissue associated with bone pain. In this case there was no major deformity, pathological fracture was unlikely and bone pain not severe.

The patient is to be kept under observation at six monthly intervals with a view to curettage and bone grafting should the lesions increase in size.

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