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Fibrous metaphyseal defects – determination of their origin and natural history using a radiomorphological study

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Abstract. The radiomorphological appearance of fibrous metaphyseal defects (FMDs) is demonstrated by long-term follow-up studies. A characteristic radiomorphological course rather than a typical single appearance can be established. These findings correlate well with the duration of these tumor-like lesions; therefore, the radiological findings allow conclusions to be made about the age of a fibrous metaphyseal defect. In addition, the characteristic locations of FMDs will be explained in respect of their origins at insertions of tendons and ligaments.

Key words: Fibrous metaphyseal defect in bone – Fibrous cortical defect – Non-ossifying fibroma – Radiology – Radiomorphological course – Characteristic location

Fibrous metaphyseal defects (FMDs) are known to occur during the growth period [2, 4, 9, 13, 14, 19, 20] and are hardly ever observed in adults after the age of 20 years. The average duration of FMDs is 29 months [19]. These findings indicate a changing appearance of FMDs.

Fibrous metaphyseal defects generally occur at the insertion of a tendon or ligament in the perichondrium of the physeal plate. Therefore they always are found at specific sites at the metaphysis of a long bone. With increasing age and bone growth FMDs move into diaphysis, following a straight line, according to skeletal development and growth. This straight line through the long axis of a FMD points to its origin at the insertion of a tendon or ligament (Fig. 1 A–C) [16].

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It is the aim of this study to define the variable radiological appearance of FMDs. In addition, special attention is paid to the location of their origin at the insertion of tendons or ligaments.

Patients and methods

Radiographs of 82 patients (107 FMDs) were analyzed retrospectively. Fifty two patients were male and 30 female, with a ratio between sexes of 2:1. The average age at diagnosis was 14 years (4 to 26 years). Multiple FMDs were observed in 20 patients. Twelve lesions required operation for various reasons and were therefore confirmed histologically.

Seventeen patients (22 FMDs) were followed over a long period with multiple radiological examinations. The average time of observation was 7.3 years (20 months to 16 years). Fifteen patients (18 FMDs) had at least two radiological studies over a mean time of 6.8 years (15 months to 13.4 years). For 50 patients (59 FMDs) only one X-ray was available.

Results

The location and presumed sites of origin for the 107 FMDs are shown in Table 1. All radiographs were analyzed sequentially for location, size, shape, and border definition of the FMD. Special attention was paid to the presence of sclerotic changes. In general, the lesions increased in size, exhibited an increasing marginal sclerosis followed by a progressive ossification that invariably started from the diaphyseal side (Figs. 3 and 6). Finally, this resulted in complete sclerosis of the lesion and the lesion disappeared roentgenographically.

Our results allow definition of four characteristic radiomorphologic stages:

Stage A

The FMD is located eccentrically in the cortex near the epiphyseal endplate. The lesion is small, oval

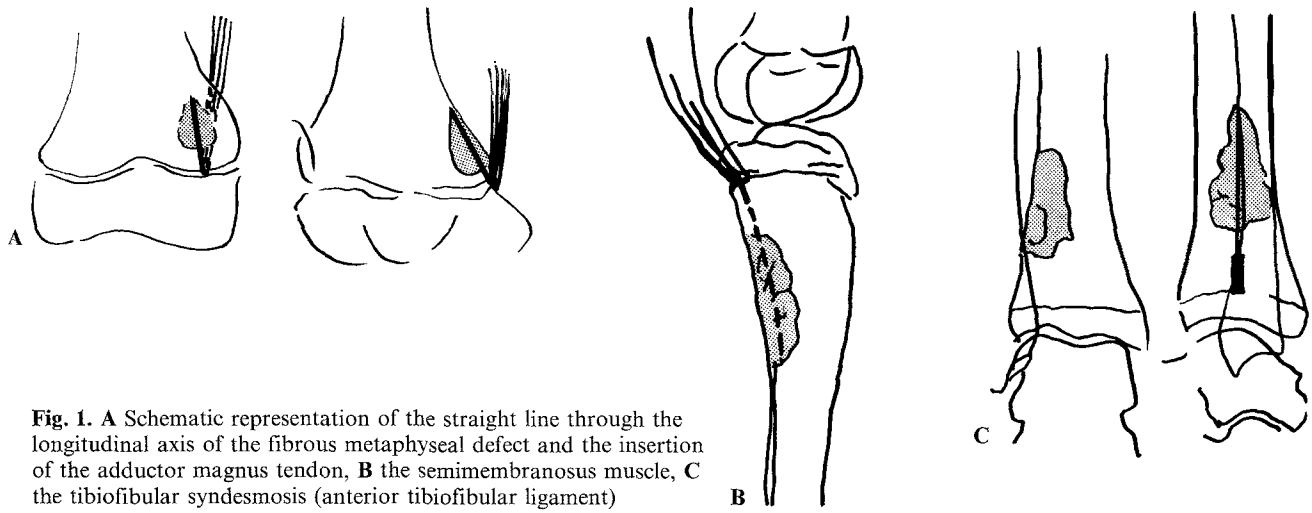
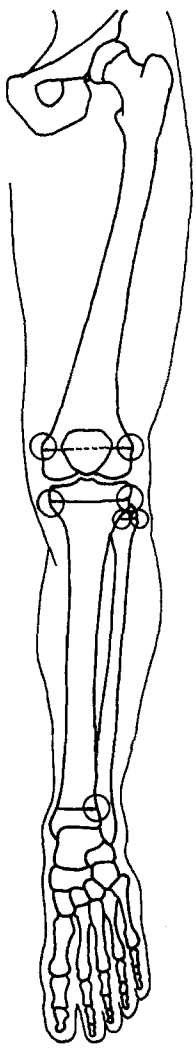


Fig. 1. **A** Schematic representation of the straight line through the longitudinal axis of the fibrous metaphyseal defect and the insertion of the adductor magnus tendon, **B** the semimembranosus muscle, **C** the tibiofibular syndesmosis (anterior tibiofibular ligament)

Table 1. Location and site of origin of fibrous metaphyseal defects



21 Distal femur, dorsolateral
lateral head of gastrocnemius muscle
Proximal point of insertion of iliotibial tract

3 Proximal tibia, dorsolateral
Anterior and posterior ligaments of head of fibula

5 Proximal fibula
Biceps muscle of thigh, collateral ligament lateral knee
Interosseous membrane of the lower leg
Anterior and posterior ligament of head of fibula

19 Distal tibia, lateral
Tibiofibular syndesmosis (anterior and posterior tibiofibular ligament)

36 Distal femur, mediodorsal
Adductor magnus muscle, medial head of gastrocnemius muscle

22 Proximal tibia, mediodorsal
Semimembranous muscle

Distal radius, ulnar
1 Capsular ligaments of distal radioulnar articulation

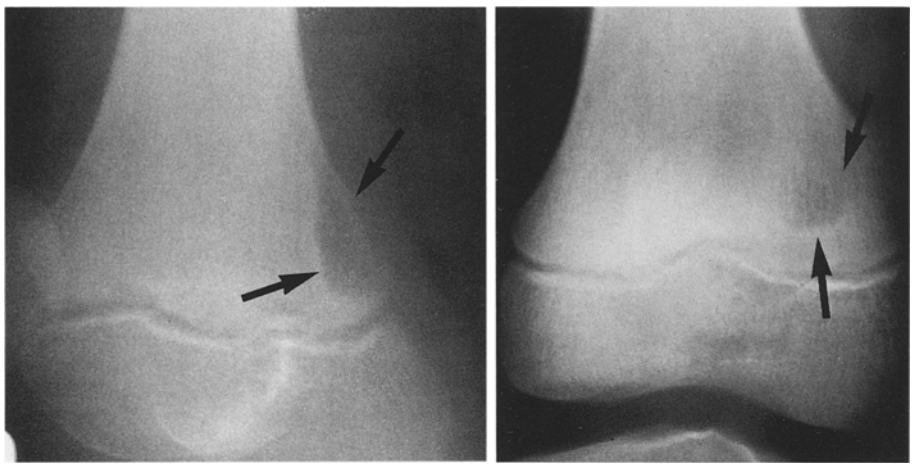


Fig. 2. P.A., 9-year-old male. Radiograph anteroposterior and 45° external rotation of the distal femur exhibits a typical fibrous metaphyseal defect near the epiphyseal cartilage, oval in shape with thin sclerotic border (stage A)

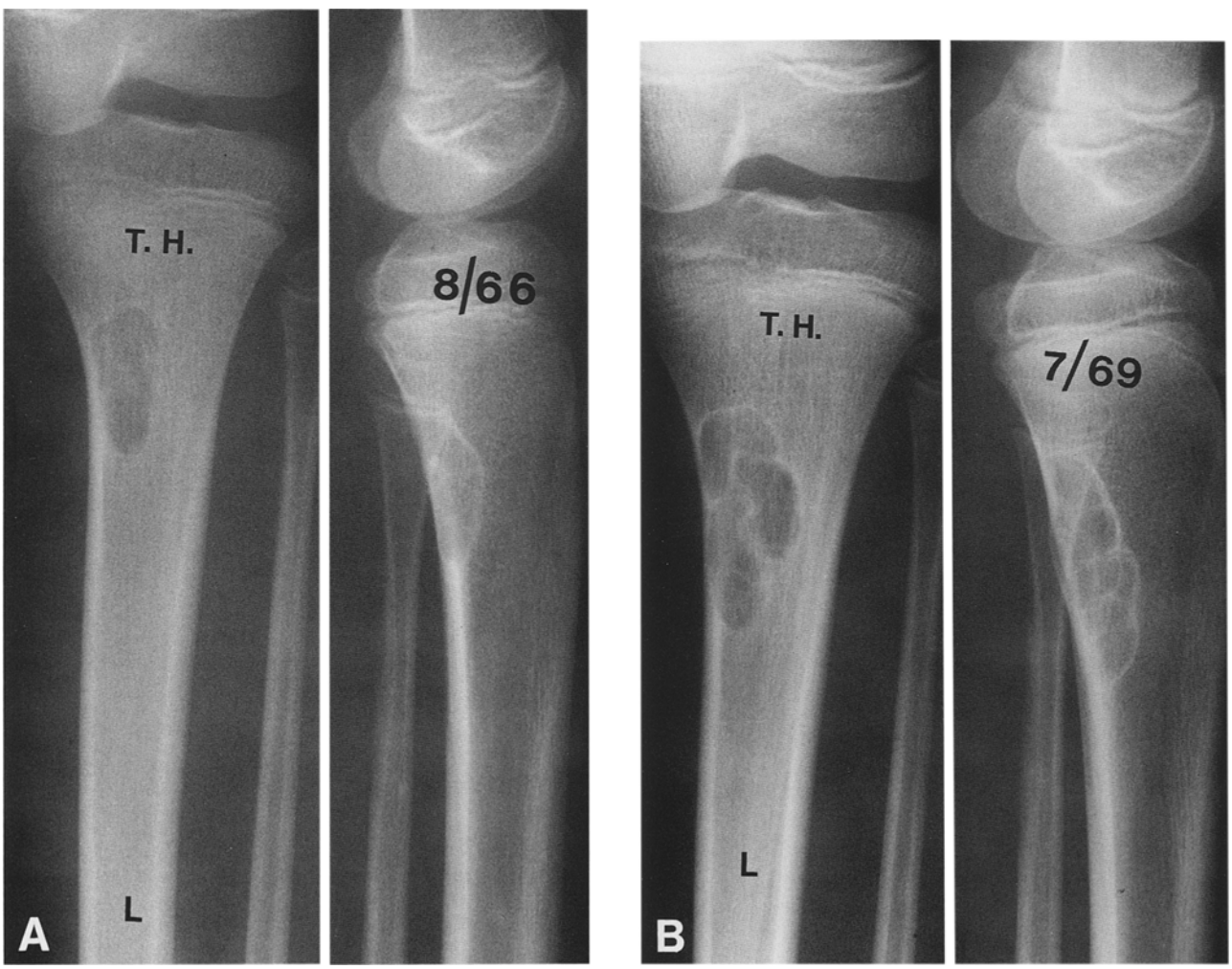


Fig. 3. A T.H., 9-year-old female. Proximal tibia, anteroposterior and lateral radiograph. Fibrous metaphyseal defect of the medial proximal tibia, oval in shape with thin sclerotic border typical for stage A. **B** Radiograph 3 years later. Marked increase in size with polycyclic shape, well-defined sclerotic border, and thin slightly protruding cortex characteristic for stage B. Verified histologically

to slightly polycyclic in shape without a sclerotic border (Figs. 2 and 3A).

Stage B

The FMD is situated at a variable distance from the epiphysis, depending on growth rate. Its shape is polycyclic with thin but clearly sclerotic borders. Although its size is increased and the cortex is thin and occasionally protruding like the shape of an hourglass, no periosteal reaction can be seen (Fig. 3B).

Stage C

The FMD exhibits increasing sclerosis which typically starts from its diaphyseal side. Otherwise findings are similar to stage B (Figs. 4 and 5A).

Stage D

This stage is characterized by complete homogeneous sclerosis of the lesion (Fig. 5B and C).

Table 2 shows the radiomorphological course of 22 FMDs over a mean period of 7.3 years. At the time of first observation nine cases were seen at stage A, eight at stage B, and five at stage C. At the last X-ray examination 11 lesions were no longer detectable. Ten were sclerosed homogeneously but one defect was still at stage C (Table 2).

Table 3 shows the course of 18 fibrous metaphyseal defects over a mean time of 6.8 years of observation with at least two X-rays available. On the first examination, seven FMDs were at stage A, six at stage B, four at stage C, and one already at stage D. On the final examination, four FMDs were at stage B. Two of these with follow-up times of 4.5 and 3.8 years respectively showed no or virtually no progression. Two were at stage C and 12 at stage D with 11 homogeneous sclerotic lesions and one not detectable.

Eight patients listed in Tables 2 and 3 exhibited multilocular FMDs. These additional FMDs were assigned to the 59 cases which we had originally examined by single radiographs. In this group we found 9 at stage A, 17 at stage B, 26 at stage C, and 15 at stage D.

In summary, 30% of the patients in stage B and C exhibited cortical thinning and protrusion. All stage C FMDs showed sclerosis, starting at their diaphyseal side, progressing towards the epiphysis.

Discussion

Due to the fact that a FMD has a limited existence, we wished to analyze its radiological appearance with time. According to our observations, radiological findings can be assigned to four typical stages. This allows one to correlate findings on a single radiograph to a specific lesional age.

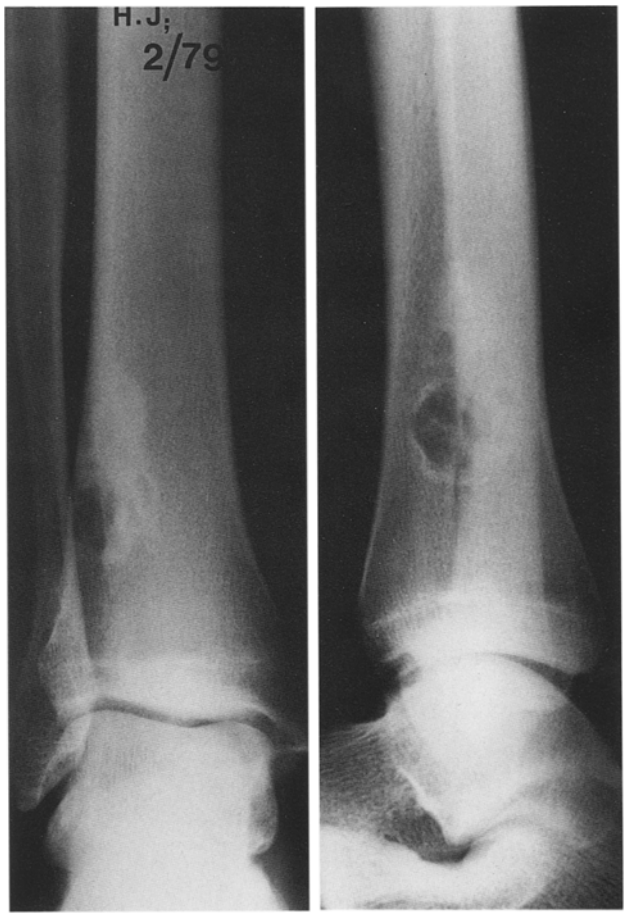


Fig. 4. H.J., 18-year-old, male. Distal tibia, anteroposterior and lateral radiograph. Fibrous metaphyseal defect of the lateral distal tibia with typical ossification beginning at the diaphysis, representing stage C

Initially, the FMD in the vicinity of the epiphyseal cartilage is round, oval, or slightly polycyclic in shape and surrounded by a very thin cortex (stage A) (Figs. 2 and 6). With increasing age, it moves away from the epiphyseal cartilage, maintaining its distinct polycyclic shape, surrounded by a slightly sclerotic border. An increase in size is frequently observed. At this point, the cortex is thin and discrete hourglass-shaped protrusions without periosteal reactions can be seen (stage B) (Fig. 6B). At stage C, sclerosis, starting from the diaphyseal side of a FMD and progressing towards the epiphysis occurs (stage C) (Fig. 6C, D). Healing is observed as an increasing homogeneous sclerosis (stage D) (Fig. 6E). Finally, the FMD is replaced by normal bone structure and disappears completely.

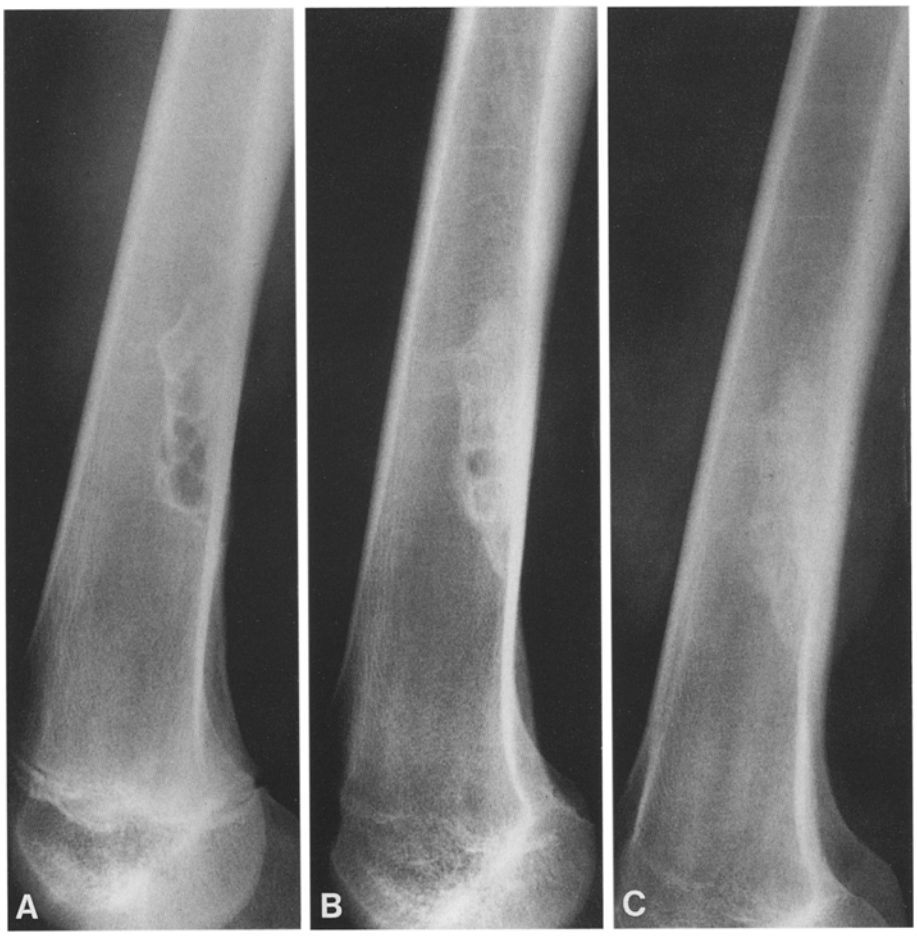


Fig 5 A–C. R.M., 13-year-old, male. Lateral radiograph of the distal femur. **A** 02/72: Stage C of FMD, **B** and **C** 03/76 + 03/78: Development of increasing sclerosis to stage D

Table 2. Long-term observation with multiple radiological controls ($n = 22$)

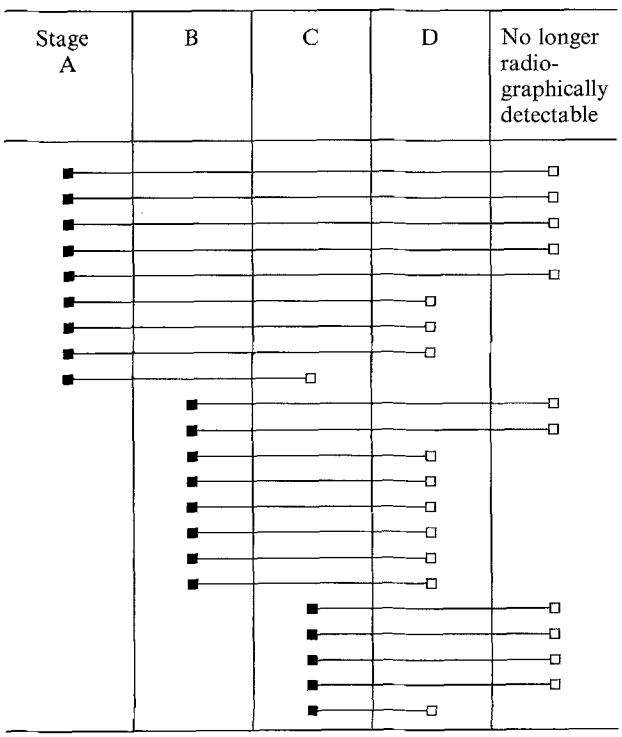
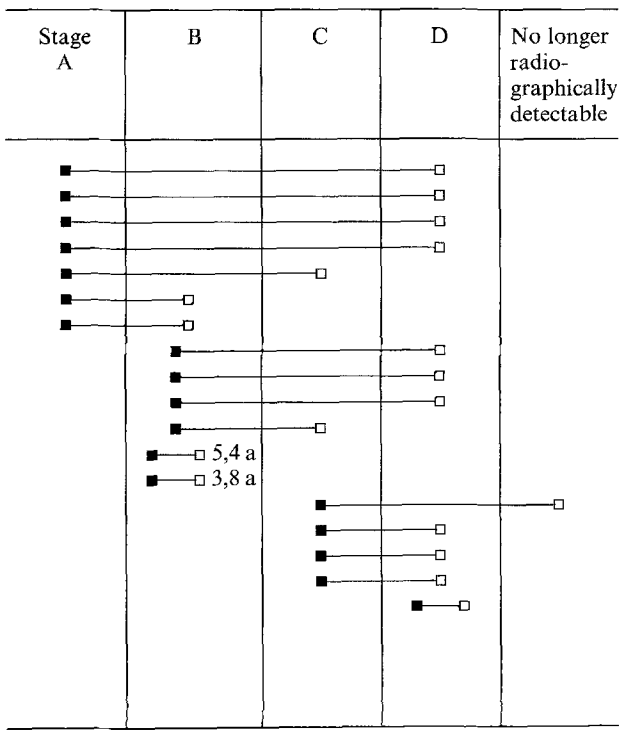


Table 3. Long-term observation with at least two radiological controls ($n = 18$)



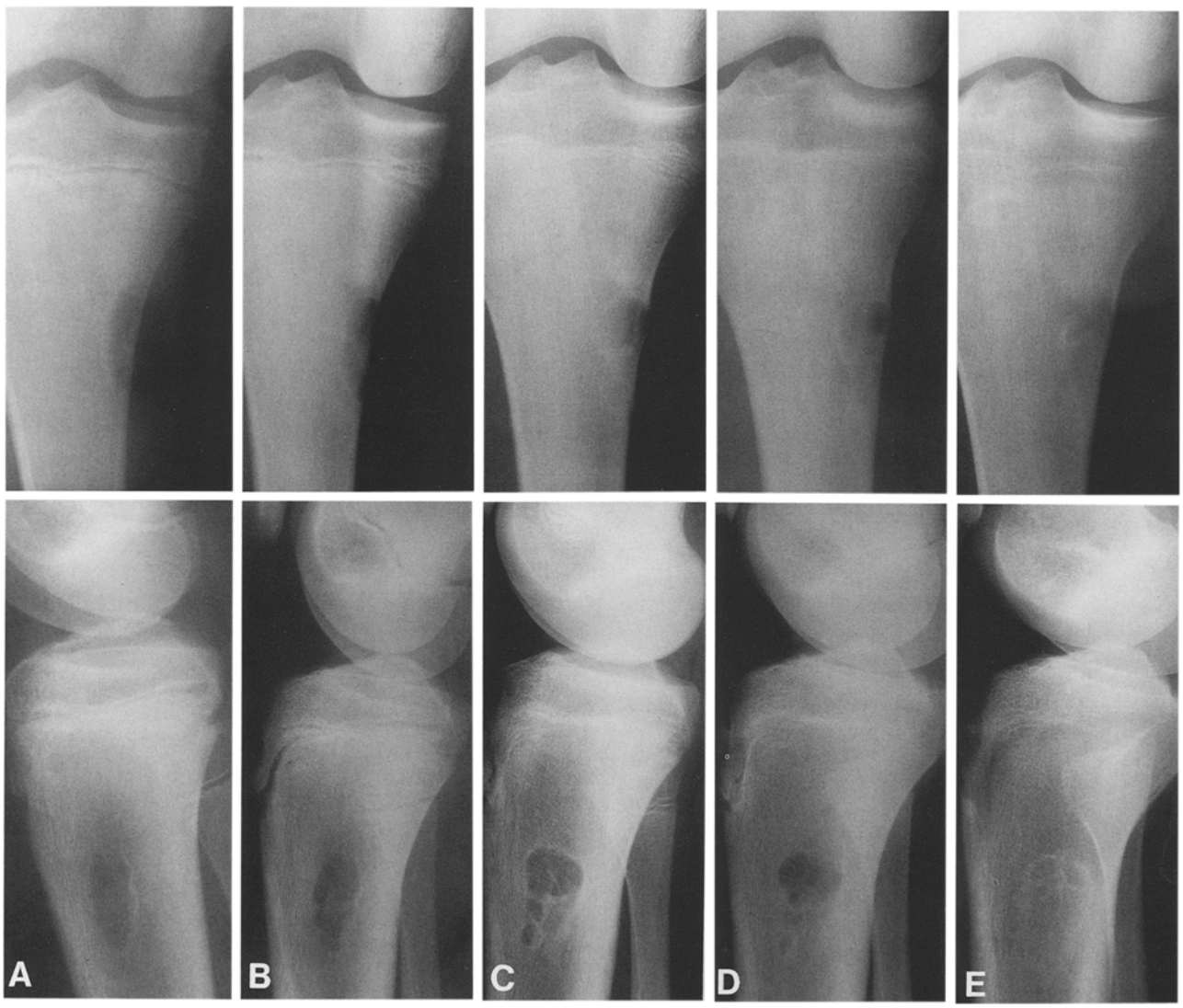


Fig. 6A–E. L.W., 13-year-old female. Long-term follow-up of a FMD. **B** 10/03/77: Polycyclic-shaped fibrous metaphyseal defect at stage B. **C** 13/04/78: Ossification beginning from the diaphysis (stage C). **D** 01/03/79: Progressive healing with extensive ossification (stage C–D). **E** 07/06/84: Homogeneous sclerosis of the fibrous metaphyseal defect (stage D)

The characteristic radiographic appearance which we describe as stage B has been reported by many authors (3–5, 7–11, 14, 17–20]. An increasing homogeneous sclerosis and separation from the epiphyseal endplate is known to occur with increasing age [10]. Enlargement has also been documented in several instances [11, 16, 21]. In general, this increase in size takes place at the transition from stage A to B and at stage B. A polycyclic shape, hourglass-shaped protrusion, and enlargement are the radiomorphological signs of growth. However growth is limited in time and generally not observed in stages C and D. It has not yet been possible to explain the reason for growth of FMDs and why some FMDs become large non-ossifying fibromas.

The development of a FMD can be based on the assumption that it originates at the insertion of a tendon or ligament at the perichondrium of the epiphyseal cartilage (Figs. 7 and 8). As the result of a yet unknown pathogenesis – possibly traumatic – disturbance of metaphyseal bone growth with development of a fibrous cortical defect occurs. After normal bone growth is regained, the FMD moves diaphysially. It follows an imaginary line, parallel to the long axis of the involved bone and through the longitudinal axis of the FMD. This line points to the insertion of the tendon originally involved (Fig. 8). This was observed for all our 107 cases. The locations given in Table 1 can be regarded as the points of origin of FMDs.

Our results and other data [15–20] show that

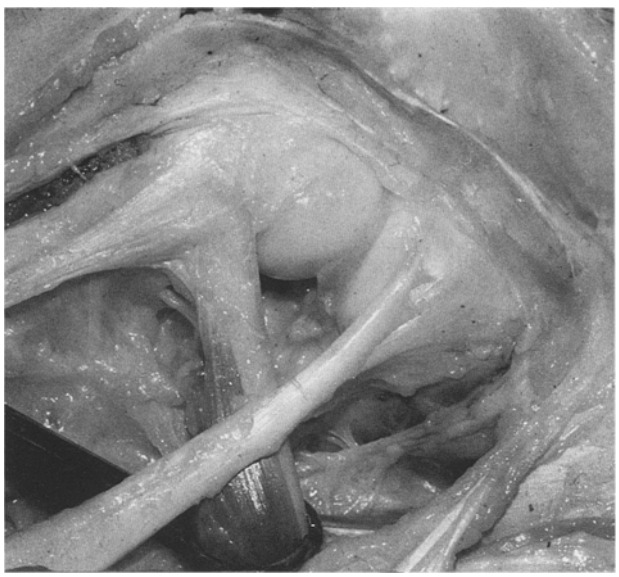


Fig. 7. Post-mortem specimen showing the insertion of the tendons of the semimembranous muscle, medial head of gastrocnemius muscle, adductor magnus muscle in the perichondrium of the epiphyseal cartilage

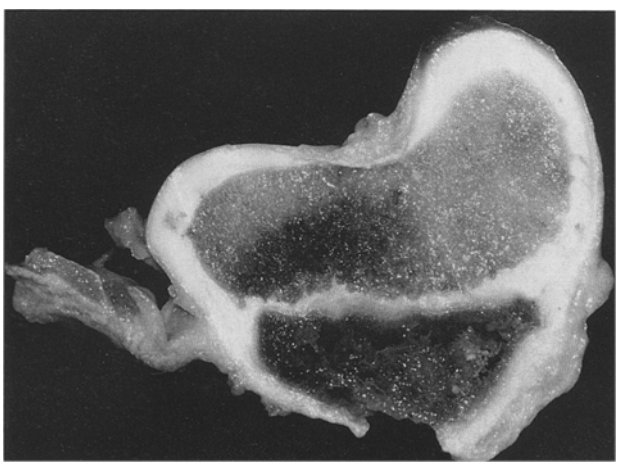


Fig. 8. Frontal section through the distal femur showing the insertion of the adductor magnus tendon in the perichondrium of the epiphyseal cartilage

FMDs cannot be observed at sites where no tendons attach. This supports our theory [16]. Thus for example, no FMDs are observed in the region of the femoral neck, since the epiphyseal cartilage is intra-articular and no tendons insert at the perichondrium of the epiphyseal cartilage in this area [16]. Another example is that FMDs exclusively occur laterally in the distal tibia, on its fibular aspect [16]. This can be explained by the insertion of fibres of the distal fibular syndesmosis in the epiphyseal cartilage of the tibia.

The specific location of FMDs on the circumference of a long bone has considerable differential diagnostic importance. Other lesions with similar appearance do not have such characteristic locations and therefore require further diagnostic evaluation.

Bufkin [1], Dunham et al. [6] and Resnick and Greenway [15] describe a similar lesion mediodistally in the femur called respectively "avulsive cortical irregularity", "developmental defect of the distal femoral metaphysis", or "proliferative cortical irregularity". In the initial phase, these lesions may exhibit cortical spiculae and irregularities also seen in malignant tumors. We agree with Bufkin [1] and Resnick and Greenway [15] that these lesions and FMDs are different entities. Commonly "proliferative cortical irregularities" are located at the medial rim of the linea aspera near the adductor magnus tendon. Their rapid appearance and regression in active young adults indicates a traumatic origin.

In summary our data imply that fibrous metaphyseal defects follow a characteristic course which corresponds with their age. It is therefore possible to determine the life span of a FMD. Its typical location on the circumference of a long bone supports the diagnosis when compared with similar-looking lesions in uncharacteristic locations.

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