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ANGULAR VARUS DEFORMITY IN THE DISTAL END OF THE TIBIA

By

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In Vaasa provincial hospital 2 children were seen, both of whom had an angular varus deformity at the distal end of the tibia, clinically completely similar in appearance. In one of the cases it was caused by severe rickets, in the other the etiology was obscure. In medical literature we have not found descriptions of similar cases.

Tibia vara, a nonrachitic deformity appearing in the upper end of the bone was defined as a disease in itself by *Blount* (1937). He also gave it the name osteochondrosis deformans tibiae. Many cases had earlier been published under different names. The etiology of tibia vara is unknown. It has been considered an asptic bone necrosis or something comparable with it (*Nilsson* 1929, *Blount* 1941), but, on the other hand, microscopic studies have shown changes in the cartilage only and not in the bone (*Langenskiöld* 1952). Obesity has often appeared in connection with the infantile type of tibia vara (*Langenskiöld*). *Barber* (1939) has presented a case in which there was varus deformity in both ends of the tibia, but not in the diaphysis. However, the X-ray changes of the distal epiphysis were slight and the case in question is not of the same type as our case No. 1. There is still a rare congenital varus deformity, where bending occurs in the distal third of the tibial diaphysis. The narrowing of the diaphysis of the tibia and the higher density at the bent point, without epiphyseal changes, can be seen in X-ray pictures (*Blount* (2), *Langenskiöld*).

Multiple ossification centres (*Christensen* 1955) and irregularity of ossification (*Fairbank* 1947, *Leeds* 1960) are seen in dysplasia epiphysealis multiplex. This disease is generally hereditary (*Waugh* 1952, *Christensen*, *Maudsley* 1955, *Shephard* 1956, *Barrie* 1958 and *Leeds*) and according to *Maudsley*, does not appear in early childhood. The

patients discussed in books dealing with the subject have been older, between 4–50 years (*Christensen, Freiburger 1958, Leeds*). These patients are usually of short stature or of stunted growth and have short and clumsy fingers (*Fairbank, Maudsley, Barrie*). In our opinion, our own case No. 1 does not belong to the category discussed, in which, it is true, different forms of the disease appear.

Rachitic bow-legs are nowadays rare. Sometimes rachitic deformity appears although the child has had the customary vitamin D prophylaxis. Our second case belongs to this category. Renal, intestinal, hormonal and enzymate factors of many kinds and also the so-called vitamin D resistant rickets (*Pedersen & McCarroll 1951, Hepp 1961*), among which, it is true, renal rickets is also often included, should be taken into consideration as etiological factors. In our case the cause was intestinal. Vitamin D in fat-soluble form was not sufficiently absorbed. The deformity may clinically be the same in appearance both in rachitic and nonrachitic cases.

OUR OWN CASES

Case No. 1. Girl, 13 months old. The general development was good. Started walking at the age of 8.5 months. When the child had been walking for some time, the parents found that the legs of the child were bowing into varus. No other symptoms indicated rickets. Calcium, phosphorus and alkaline phosphatase in the blood were normal.

Symptomata localis: Severe varus deformity in the lower part of each leg. Nothing special in other joints.

X-ray status: see Figs. 1 a, b and c.

Treatment: Osteotomia sphaerica tibiae et correction et osteotomia fibulae 1.dx. Osteotomy was performed in the metaphysis near the epiphyseal line. Varus deformity was corrected easily after osteotomy. Plaster was applied.

After 6 days the same operation was carried out on the left side.

Decursus morbi: change of plaster after 1 month. Weight-bearing was not allowed. See Figs. 2 a and b, taken 1 month after the operation.

The plaster cast was removed after 4 months, weight-bearing was started. See Fig. 3, which was taken later on and in which complete recovery has been achieved.

Case No. 2. A baby girl, 18 months old. She was given codliver oil from the age of 3 months, not always regularly; started standing at the age of 9 months. After that her legs began to bow into varus. She started walking at the age of 16 months.

Cranium: the anterior fontanel was about 1×1 cm., sutures closed, no craniotabes. Thorax normal, Harrison's groove of hardly noticeable size. Calcium in the blood 9.3 mg%, phosphorus in the blood 5.1 mg%. Alkaline phosphatase in the serum over 10 B-L-units.

Symptomata localia: Severe angular varus deformity in the distal parts of each leg. Nothing special in other joints. X-ray status: see Fig. 4.

Treatment: Osteotomia sphaerica tibiae, cum correctio et osteotomia fibulae 1.sin.



Figs. 1 a, b and c.

Case 1. Distal epiphysis of each tibia is irregular and displaced backwards and medially. The medial part of the epiphysis of femur is deformed.



Figs. 2 a and b.

Case 1. Pictures were taken about 1 month after operation. Varus deformity is disappearing, epiphysis and epiphysial lines are indistinctly visible.



Fig. 3.

Case 1. Picture was taken 2 years after operation, at which time the child was 3 years old. Distal epiphysis and epiphysial line of tibia are normal in appearance. Function of limbs is normal.

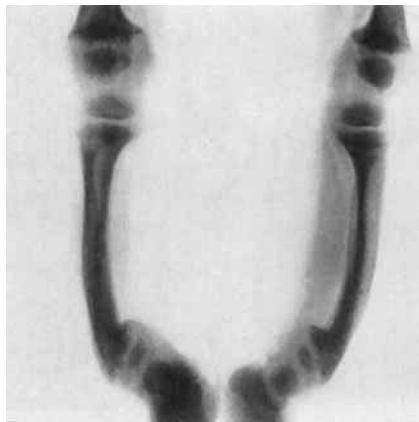
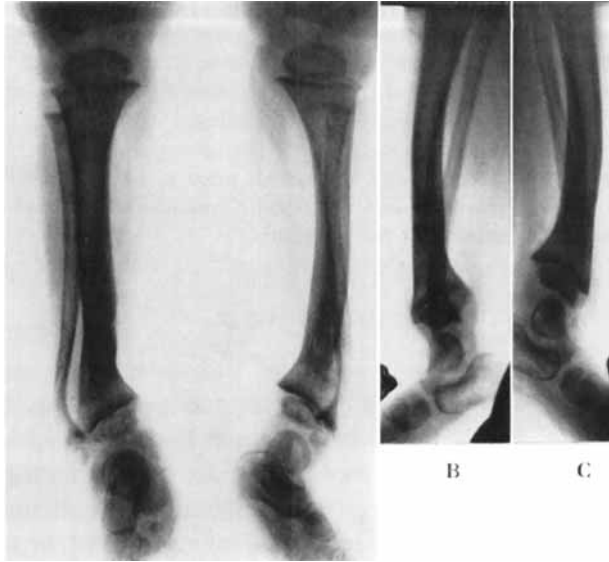


Fig. 4.

Case 2. Epiphysial lines of both femur and tibia are ragged and have become broader. The distal epiphysial line of the tibia is deformed and spread out fanwise. Severe varus deformity. Diagnosis: rickets.



Figs. 5 a, b and c.

Case 2. Picture were taken about 2 months after operation. Epiphysial lines have become comparatively regular. Still some varus deformity and antecurvatum in the diaphysis of tibia.

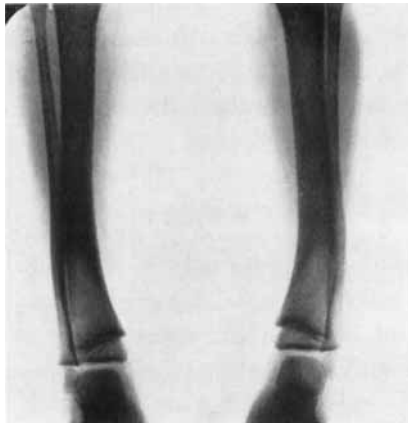


Fig. 6.

Case 2. Varus deformity has disappeared, but still antecurvatum in the diaphysis of tibia. Epiphysis and epiphysial lines seem normal.

Osteotomy was performed on the cranial side of the epiphysial line. Varus deformity was corrected and a plaster cast was applied. In addition oral antirachitic treatment with water-soluble vitamin D and with calcium was given.

An operation of the same kind was performed after 6 days on the right side.

Decursus morbi: Change of plaster after 2 months.

See Figs. 5 a, b and c, taken 2 months after the operation.

The limbs were in plaster for over 4 months, after which the child was allowed to start walking. The patient received antirachitic treatment at the time. Fig. 6 was taken 2 years and 2 months after the operation.

DISCUSSION

Gordon (1961) presented a series of radiographs of the legs of a child who at the age of nearly 1 year had marked bowlegs. In addition to that, at the age of 1½ years, some changes had appeared in the distal epiphysis of the tibia and in the distal part of the metaphysis. The changes described by *Gordon* resemble in some respects our own case No. 1. *Gordon* considers the deformity congenital, but in our opinion the radiographical appearance is suggestive of rickets.

Our own case No. 1 differs from the case referred to above in that the diaphysis of the tibia is straight and the ossification in the epiphyseal region is irregular. In these respects the case differs from rickets, as does Blount's disease. We do not know the etiology in case No. 1 and we cannot exclude the possibility that the patient had had an early rickets, as a result of which the distal epiphysis of the tibia remained weak.

By comparing cases No. 1 and No. 2 with each other, we can state that the formed recovered more rapidly. In case No. 2 recovery was slower in spite of medication, and some antecurvatum deformity remained in the diaphysis of the tibia. However, the result of treatment in each case is to be considered good.

SUMMARY

Two cases of severe varus deformity in the distal epiphysial area of the tibia in children are discussed. The etiology in one case was rickets caused by intestinal malabsorption. The etiology in the other case was obscure. We cannot with certainty exclude the possibility that the patient had had an early rickets, as a result of which the distal epiphysis of the tibia remained weak. On the other hand this case suggests the possibility that a non-rachitic deformity similar to tibia vara (Blount's disease) may occur in the lower end of the tibia. Both cases were cured by osteotomy.

RESUME

Deux cas de déformité varus grave dans la région épiphysaire distale du tibia chez des enfants sont discutés. L'étiologie dans un cas était un rachitisme causé par une mauvaise absorption intestinale. L'étiologie était obscure dans l'autre cas. On ne peut exclure avec certitude que le malade n'avait pas souffert de rachitisme précoce d'où il était résulté que l'épiphyse distale du tibia était restée faible. D'un autre côté, ce cas laisse entrevoir la possibilité qu'une déformité non rachitique similaire au tibia varus (maladie de Blount) peut se produire dans l'extrémité inférieure du tibia. Les deux cas ont été guéris par ostéotomie.

ZUSAMMENFASSUNG

Zwei Fälle von schwerer Varusdeformität in der distalen Epiphysenregion bei Kindern werden besprochen. Die Ätiologie war in einem Falle Rachitis, die infolge intestinaler Malabsorption entstand. Die Ätiologie in dem anderen Falle war unklar. Wir können nicht mit Sicherheit die Möglichkeit ausschliessen, dass der Patient eine frühzeitige Rachitis gehabt hat mit der Folge einer zurückbleibenden Schwäche der distalen Tibia epiphyse. Andererseits jedoch deutet dieser Fall auf die Möglichkeit hin, dass eine nicht-rachitische Deformität ähnlich wie die Tibia vara (Blounts Erkrankung) auch am unteren Ende der Tibia vorkommen kann. Beide Fälle wurden mittels Osteotomie geheilt.

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