BONY ANKYLOSIS IN GOUT

BY

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The acute joint manifestations of gout are now recognized as being due to "crystal deposition disease" (McCarty, 1966) or "crystal synovitis" (Mason, 1966). The pathological changes of chronic gout have not been so fully studied and may be more complex. They result from the deposition of urate crystals in and about the articular structures, the subsequent erosion and destruction of the cartilage with attendant giant cell invasion, sub-chondral bone resorption, and eventually fibrous ankylosis. These changes are further complicated in severe cases by the development of secondary osteoarthritis (Lichtenstein, Scott, and Levin, 1956). Bony ankylosis has, so far as we can determine, been described only twice—once by Ludwig, Bennett, and Bauer (1938), and again by Dixon (1967) in an unpublished paper read at the Royal Society of Medicine. The frequency and distribution of bony ankylosis is not known, but Ludwig, Bennett, and Bauer pointed out that the mid-tarsal joints are characteristically affected.

A patient with chronic tophaceous gout of sufficient severity to justify amputation, and in whom bony ankylosis was previously suspected radiologically, has provided the opportunity to study the advanced gouty changes in the joints of the feet.

Case Report

A 42-year-old male developed occasional joint pains at the age of 7, and had his first classical attack of podagra at the age of 21 years. This was followed by recurrent polyarticular gouty attacks with progressive deformity of the hands, feet, and elbows. From the age of 30, tophi developed, becoming massive and producing mechanical disability. Within 7 years he was bed-bound, with a plasma uric acid level ranging from 9.3 to 13.5 mg./100 ml. At the time of his first admission to hospital at the age of 37, his urinary uric acid excretion was 500 mg./24 hours (baseline estimation).

Radiographs of the feet at this time showed the presence of numerous large tophi, some being radio-opaque (Fig. 1).

Treatment with uricosuric drugs (Ethebenecid 4 g./day and Sulphinpyrazone 600 mg./day) lowered the plasma

Fig. 1.—Clinical radiograph of foot.

uric acid levels from 12 to 8 mg./per cent., and it was not until allopurinol became available, when the patient was 38 years old, that more satisfactory metabolic control was achieved. Acute gouty attacks gradually ceased and the tophi became smaller though continuing to produce severe disability. He was unable to walk because of mechanical disability and haemorrhages from ulcerated tophi. At the age of 40, therefore, a left below-knee

amputation and a right Symes amputation were performed.

Using prostheses he is now able to walk 200-300 yards with the aid of sticks. This is the first time he has walked for 3 years.

Pathology

The specimens were preserved in alcohol (to avoid dissolution of the water-soluble uric acid) and were subsequently sliced in the sagittal plane. Both feet were severely ulcerated and the skin pigmented with melanin. The large deformities present were due to tophi (Fig. 2).

Fig. 2.—Sagittal section of amputation specimen from left foot through 5th metatarsal.

Fig. 3.—Thin slice (2-3 mm.) radiographs of right foot showing metatarsal fusion and radio-opaque tophus.

There were tophi around the joints, in bone, and in the loose connective tissues of the feet. The mid-tarsal joints were the most severely affected. The joint spaces had almost completely disappeared and were represented by thin white lines. Material from these tophi was examined by polarized light microscopy and was shown to consist largely of needle-shaped negatively birefringent crystals having the optical properties of urate. Analysis, by x-ray crystallography, found them to be mainly sodium urate monohydrate with a minor constituent of carbonate apatite, the latter probably accounting for the radio-opacity.

Thin (2-3 mm.) slices of the sagittal sections were used to make radiographs (Fig. 3, opposite). These supported the previous clinical and radiographic impression that bony fusion might have occurred in the mid-tarsal joints.

Low-power microscopy of the mid-tarsal joints showed cancellous bone crossing and partly obliterating the joint space, producing bony fusion between adjacent tarsal bones (Fig. 4). This occurred at many sites throughout the length of individual joints, though especially marked at the periphery. Histological examination showed extensive gouty degeneration of the joints, especially those of the mid-tarsal region. Large urate tophi were present, with erosion of the cartilage of the joints, osteoclastic bone destruction with sub-chondral bone

resorption, and areas of new bone formation. Further examination of unstained 5μ sections under crossed polars confirmed the extensive deposition of uric acid crystals within cartilage (Fig. 5) and extending into the sub-chondral bone (Fig. 6, overleaf).

Fig. 5.—Urate crystals in joint cartilage shown by polarized light microscopy. 5μ unstained section. $\times 8$.

Discussion

The joint destruction in this patient therefore results from a most severe form of tophaceous gout, the disease having started in the prepubertal period and having been difficult to bring under metabolic control.

The pathological specimens obtained from this patient have been submitted to a variety of techniques, including macroscopic examination of thin, whole slices, "thin slice radiography", routine and polarized light microscopy, and x-ray crystallography.

These techniques have conclusively shown the

Fig. 4.—Extensive bony ankylosis of a mid-tarsal joint both along the joint line and at the periphery (close-up photograph).

Fig. 6.—Erosion of cartilage and sub-chondral deposition of urate examined under polarized light microscopy. 5μ section. Light methylene blue counterstain. $\times 10$.

presence of bony ankylosis, not only at the periphery of the joint, although this was the site where it was most marked, but along the whole length of the articular surfaces. This would appear to have followed the destruction of cartilage and sub-chondral bone, following the deposition of urate crystals. The true incidence of bony ankylosis in severe gout is unknown and it may be that, if detailed examination were made of every possible case of gout coming to surgery or autopsy, a greater incidence of this complication might be discovered.

Summary

Clinical and pathological findings are reported in a patient with gross tophaceous gout with bony ankylosis in the mid-tarsal joints.

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Ankylose osseuse dans la goutte

RÉSUMÉ

On rapporte les résultats cliniques et pathologiques d'un cas de goutte multito-phacée avec ankylose osseuse des articulations médio-tarsiennes.

Anquilosis ósea en la gota

SUMARIO

Se relatan los resultados clínicos y patológicos de un caso de gota multito-fácea con anquilosis ósea de las articulaciones tarsales medias.