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Department of Orthopaedic Surgery, University Hospital, Pellenberg, Belgium.

GANGLION CYSTS OF BONE

Report of Two Cases and Review of the Literature

D. WILLEMS, J. C. MULIER, M. MARTENS & M. VERHELST

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The occurrence of subchondral bone cysts was first noted by Hicks in 1956. Since then 51 cases have been reported. The purpose of this paper is to describe an additional two cases having similar clinical and histological features, with a review of the literature.

CASE HISTORIES

Case 1: A 47-year-old man presented with a 4-year history of pain at the lateral aspect of his left knee, with limping and increasing disability. There was no locking or giving away of the joint. Clinically, there was tenderness and mild swelling over the left fibula. X-rays revealed a cystic lesion of the proximal fibula with a thin sclerotic border (Figure 1). Laboratory analysis was normal. At operation, after fenestration of the fibula, there was an outpouring of thick mucoid fluid. The lesion was curetted. Histologically the lining of the cyst resembled a ganglion cyst. The curettage resulted in a relief of the complaints.

Case 2: A 45-year-old man had noted intermittent aching discomfort in the left ankle for two years. His symptoms were accentuated by activity. X-rays revealed a cystic lesion located at the medial malleolus (Figure 2). At operation the cyst had a whitish lining and was filled with myxomatous material, typical of a ganglion cyst. The lesion was curetted with good results. Histologically the wall of the cyst exactly simulated that of ganglion cyst of soft tissue (Figures 3 and 4).

DISCUSSION

Typically this lesion is encountered during middle age. The cases reported range from an 18-year-old (Nigrisoli 1971) to an 86-year-old patient (Sim 1971).

Sex difference is apparently not great enough to be meaningful. There is no distinct sex difference.

Figure 1. AP radiograph of the left proximal fibula showing a cystic lesion with discrete borders of surrounding sclerosis.

The most frequent localisation is at the ankle (Figure 5). The onset of the condition is insidious and the complaints are present for several months, sometimes several years, before medical attention is sought. The patients usually present with aching discomfort related to activity.

Laboratory analysis is usually normal. X-rays show a well-defined cystic lesion, extending to the subchondral bone, with discrete borders of surrounding sclerosis. Most of the lesions are unilocular; less commonly they are multilocular (Nigrisoli 1971, Sim 1971). Histologically the border of the lesion and the mucoid gelatinous content are identical to that of soft tissue ganglion cysts.

Treatment by curettage and bone grafting appears to be effective; in two cases treated in this manner (Crabbe 1966, Sim 1971) there has been a recurrence. One case recurred six times over a 16-year period (Sim 1971). There is still discussion about the etiology and patho-

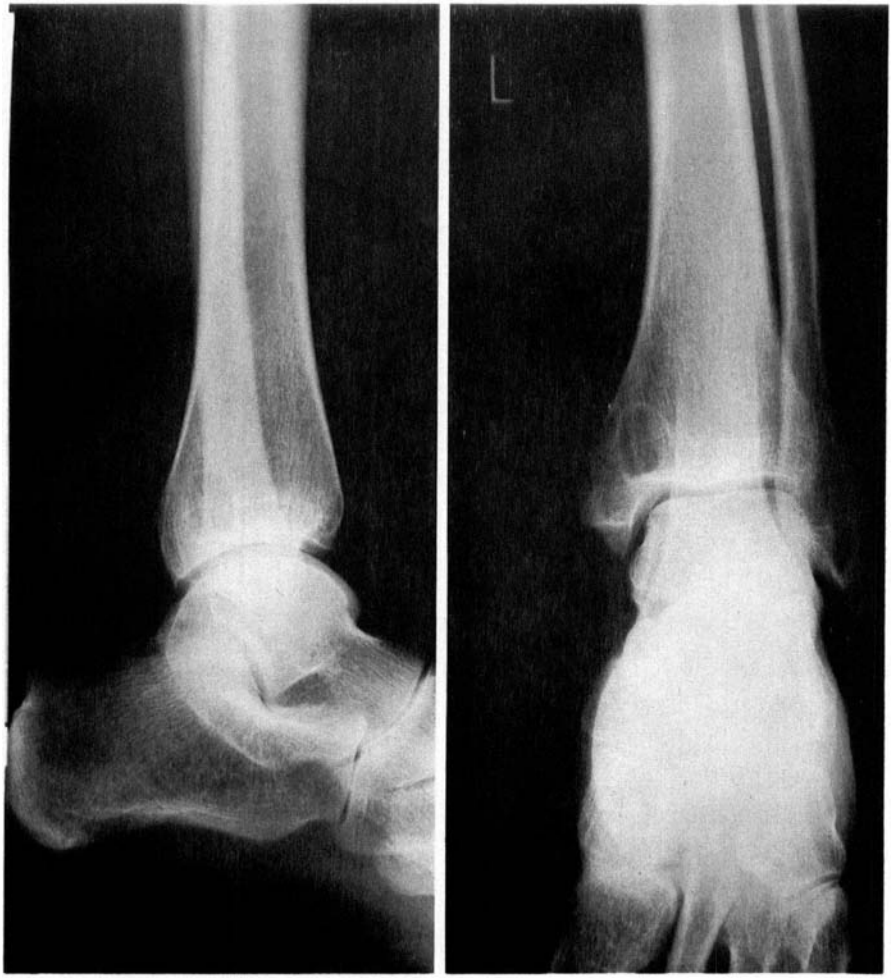

Figure 2. AP and lateral radiograph of the left ankle showing a cystic lesion occupying the medial malleolus.

genesis of this lesion and its more common soft-tissue counterpart. Hicks (1956) applied the term "synovial cyst" in his paper; he described a distinct layer of synovial-like cells. His suggestion about pathogenesis was that skeletal connective tissue undergoes synovial differentiation. Crane & Scarano (1967) also used the term "synovial cyst" to refer to cysts occurring in relation to joints and tendons. They postulated a defect in the articular surface with either simple extrusion of synovial fluid or proliferation of the synovial membrane through the

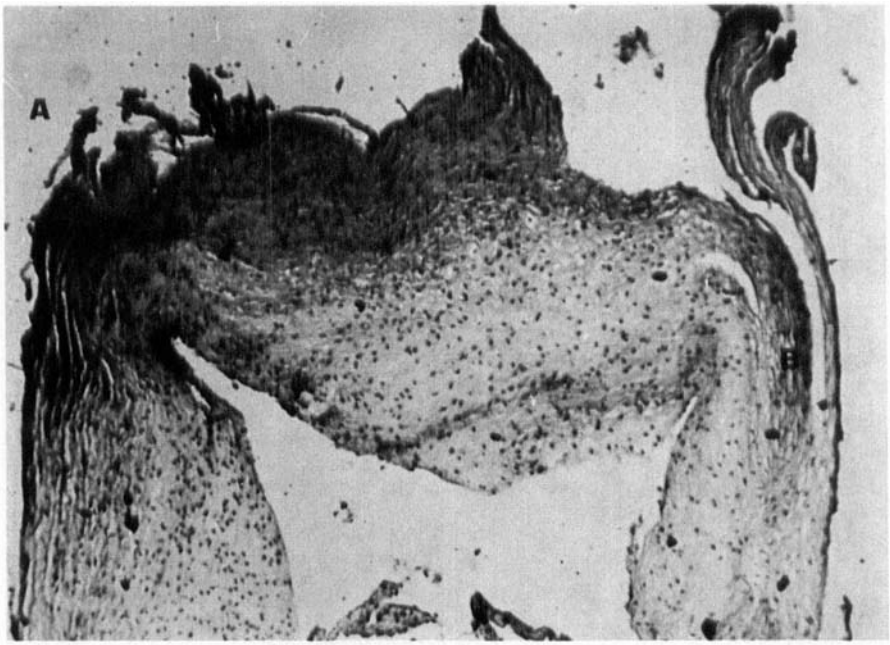

Figure 3. This is a low power photomicrograph of the lining of the cystic lesion (× 27).

defect. They demonstrated in their cases strips of tissue arrangement which fairly closely resembled synovial membrane.

Most authors did not describe a distinct layer of flattened synovial-like cells. At operation, a communication between the cystic bone lesion and the articular cavity was only observed in four cases (Scaglietti 1960, Crane 1967, Nigrisoli 1971). This does not rule out that there has been or is a small connection in the other cases.

Histologically the appearance of these lesions may be similar to that of the cysts found in osteoarthritis. In their study of osteoarthritis, Harrison et al. (1953) concluded that the cysts are the results of small foci of bone necrosis, the result of infarction occurring at pressure areas, and that communication with the articular cavity and the pressure of the joint fluid prevented healing.

To Ferguson (1964) the histological findings suggested trabecular fractures, repair callus was formed and clefts appeared within the repair tissue. In the older clefts, the wall thickened and a process very akin to ganglion formation takes place. Smaller cysts coalesce to form larger ones.

Figure 4. Detail A shows a dense fibrous wall of the cyst with a more myxomatous component to the centre of the cyst (× 70).

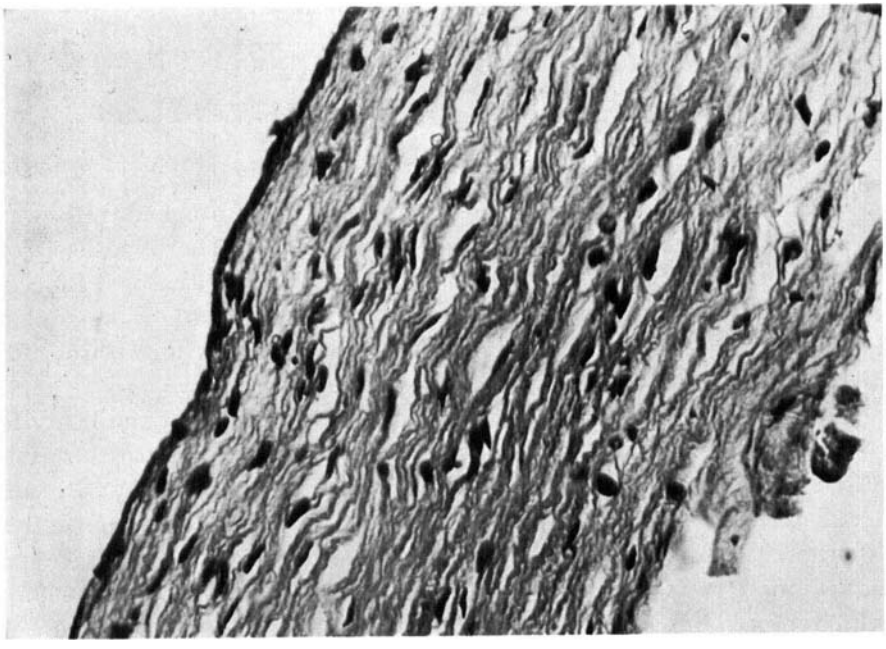

Figure 4. Detail B: thick collagenous fibrous tissue (× 170).

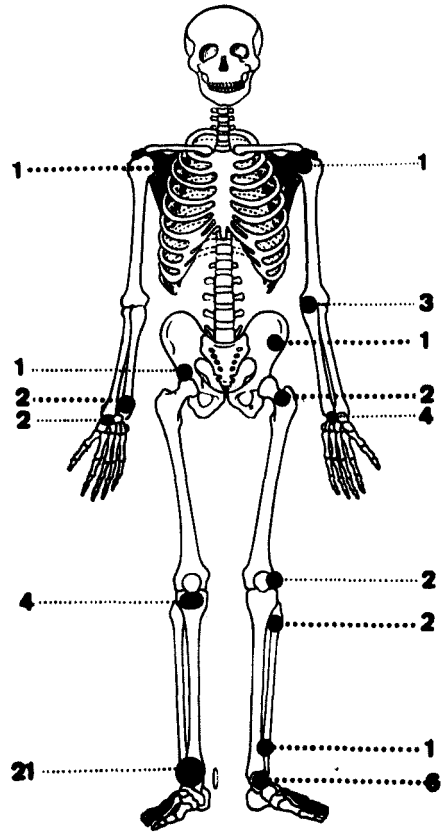

Figure 5.

Neither in our cases nor in those reported by the other authors was there any evidence of a degenerative joint disease. The hypothesis of vascular disturbance by Woods (1961) must also be discarded. Indeed, a careful study of all the cases reported by different authors has failed to reveal any evidence of bone necrosis, nor was there any fibrosis of marrow on the outside of the cyst.

In view of the very close macroscopical and histological similarity to simple soft-tissue ganglia, it is deemed reasonable to regard this condition as an exactly comparable condition occurring in bone. Ritschl (1895), Thorn (1896) and Ledderhouse (1925) agreed that connective tissue may undergo degeneration as a result of chronic damage and liquefaction. A study in depth has been performed by Soren (1966), who reported on 200 patients with soft-tissue ganglia. This author

described the various stages leading to formation of a lesion which is grossly and histologically similar to the subchondral bone lesion. In the initial phase, collagen fibers of loose connective tissue are swollen, bifurcated and fragmented. Small cavities develop with myxoid degeneration: some of them coalesce by liquefaction of interposed septa to form a larger cavity which contains mucoid material. In the final stage the degenerative process of the inner wall of the lesion stabilizes. The rim of the lesion becomes broader by proliferation of collagen bundles. Sometimes, fibrocytes lying along the inner wall of the cavity form an incomplete lining of flattened synovial-like cells.

The etiology of this lesion has not yet been elucidated. The majority of all the reported cases have no history of trauma. Soren (1966) suggested that intensive use of limbs or joints may cause excessive stress in connective tissue, which may undergo degenerative changes. Besides this, there must be a constitutional weakness of connective tissue; some patients display multiple soft-tissue ganglia. Further biochemical and immunological investigations of connective tissue in patients with soft-tissue or bone lesions will probably elucidate the etiology of these lesions.

SUMMARY

The clinical, radiological and pathological features in two cases of subchondral bone cyst are described. Various hypotheses on etiology and pathogenesis of the lesion are discussed. It seems reasonable to regard the soft-tissue lesion as an exactly comparable condition to the one occurring in bone. The etiology has not yet been elucidated. Excessive stress and a constitutional factor seem to be important in the etiology. A cystic degeneration of loose connective tissue might be mentioned.

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Correspondence to:

D. Willems, M.D.
Department of Orthopaedic Surgery
Academic Hospital
Weligerveld, 1
B 3041 Pellenberg, Belgium