THE AETIOLOGY OF MULTIPLE LOOSE BODIES

SNOW STORM KNEE

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We report four patients who showed hundreds of brilliant white loose bodies at arthroscopy of the knee after a short history of pain and crepitus. Histological, historical and clinical evidence is presented which indicates that the aetiology of this condition is the culture of chondrocytes in synovial fluid. It is suggested that reversal of the usually accepted order of events in synovial osteochondromatosis could provide a better and unified explanation for both that condition and multiple loose bodies. The term 'snow storm knee' is proposed to describe the dramatic picture seen at arthroscopy.

It is unusual to discover hundreds of brilliant white loose bodies at arthroscopy of the knee in a young patient with a short history of pain and crepitation. Multiple loose bodies are usually attributed to synovial osteochondromatosis. We present four such cases and discuss the possible diagnosis, giving histological, historical and clinical evidence that this may be a new pathological and clinical entity with an aetiology not recognised in the literature.

MATERIALS AND METHODS

Four patients of from 16 to 33 years of age presented with this condition. One had been referred for a second opinion after an arthroscopic meniscectomy; the other three had a short history of pain, crepitus and swelling. Radiographs and blood tests were normal in all cases.

At arthroscopy hundreds of brilliant white loose bodies were found, giving the appearance of a snow storm. Thorough examination of the knee showed no areas of abnormal synovium to account for them. The loose bodies were washed out of the knee and together with synovial biopsies were examined histologically. Loose bodies were also examined histochemically.

CASE REPORTS

Case 1. A 33-year-old man presented with a locked knee after a fall and was initially treated by manipulation under anaesthesia. Arthroscopy at eight weeks showed a bucket-handle tear of the medial meniscus which was removed arthroscopically. No loose bodies or synovial abnormalities were seen.

After this the patient developed severe patellofemoral crepitus and was referred for a second opinion. At eight months from injury a repeat arthroscopy revealed multiple brilliant white loose bodies and normal synovium. There was some fibrillation of the articular cartilage on the femoral condyles. The loose bodies were washed out.

Case 2. A 32-year-old man gave a three-month history of pain, crepitus and giving way of his left knee, with no history of previous trauma. At arthroscopy loose bodies were washed out. The synovium and articular cartilage appeared normal.

Case 3. A 16-year-old schoolboy presented with a two-month history of pain and swelling. He had an effusion with some retropatellar crepitus. At arthroscopy the characteristic loose bodies were seen and washed out. The synovium was reddened but otherwise normal; the articular cartilage was normal.

Case 4. A 27-year-old man gave a two-month history of pain and crepitus in his right knee after a minor football injury, and had an effusion. At arthroscopy multiple loose bodies were seen and washed out. The synovium
appeared normal but there was some fibrillation of the articular cartilage over the femoral condyles. There were over 1,500 loose bodies, with a total volume estimated to be greater than that of all the articular cartilage in a normal knee.

HISTOLOGY

All the loose bodies from all four cases looked alike, ranging in size from minute particles to 5 mm lobulated grains. They were strikingly homogeneous in each case and between cases. The bodies felt rubbery and lacked any colour or calcification (Fig. 1).

On histological examination the loose bodies had a chondroid matrix with various degrees of metachromasia with toluidine blue. They reacted as chondroitin sulphate with the critical electrolyte concentration technique, and also showed reactivity with fluorescein-labelled polyclonal antibodies against human type II collagen.

Cells within the chondroid nodules showed the cytoplasmic eosinophilia characteristic of condrocytes. They were aggregated in groups of various size in the matrix and had large open nuclei with visible nucleoli (Fig. 2). Mitoses were rarely seen. The morphology of the cells on the surface of the loose bodies was distinctive and different from those deeper within the tissue, having similar nuclear appearances and cytoplasmic staining but, instead of being round, the surface cells had a characteristic dendritic appearance with cytoplasmic processes up to 80 μm long and frequently branched (Figs 3 and 4). The matrix adjacent to these cells was less metachromatic and the critical electrolyte concentration reactivity was more in keeping with dextran sulphate than chondroitin sulphate.

The synovium showed only a minor degree of villous hypertrophy and synoviocyte recruitment. The subintima was oedematous and most notably there was perivascular oedema. Nowhere was there evidence of chondroid metaplasia or of inflammation.

The loose bodies were somewhat different from those seen in cases of synovial osteochondromatosis. Though similarly composed of type II collagen, they were relatively hypercellular and had some mitotic figures not found elsewhere in osteochondromatosis. The matrix appeared to have a focally higher content of dextran sulphate rather than chondroitin sulphate. The surface layer of dendritic cells has not been described in synovial osteochondromatosis. There were no vascular remnants associated with the bodies nor any calcification or bone formation within them. There was no layering or evidence of remodelling of the bodies to suggest that they had been present for any length of time.

These findings are suggestive of active chondrocyte replication with synthesis of a somewhat abnormal matrix. The synovial fluid would seem to be acting as a ‘culture medium’ for chondrocyte growth. There was no histological evidence to suggest a synovial origin for the loose bodies.

DISCUSSION

The literature on intra-articular loose bodies is extensive and as early as 1900 apologies were being made for adding to it (Schmieden 1900). Many theories have been proposed to account for the formation of loose bodies from the articular surface. Trauma, disturbed nutrition, aseptic necrosis, interrupted blood supply, infection, embolism of various types, defects in bone metabolism, inflammatory arthritis and osteoarthritis were being
considered in the early part of this century (Phemister 1924). At the same time attention was being drawn to a rare pathological process in joints, bursae and tendon sheaths termed osteochondromatosis (Jones 1924). The synovium 'for some undiscovered reason' was noted to form loose bodies of cartilage and bone. The process was felt to be peculiar as the bodies seemed to be formed from the synovium and not the articular surface. These bodies were composed of organised tissue as opposed to the 'corpora oryzoidea' (rice bodies) of conditions such as tuberculosis and rheumatoid arthritis. Four aetiological theories were put forward: infection, trauma, embryological origins and neoplasia.

Fall into Phase III, but the very short histories make transition through three phases of the disease unlikely. Milgram, in his original papers, comments upon the relatively short history of the Phase III patients when compared with those in Phases I and II. Of the 13 knees in Milgram's series, Phase II patients had histories less than one-third as long as the shortest histories from Phases I and II. Statistical analysis suggest that either his Phase III patients are from an entirely different disease group, or that patients develop Phase III disease before Phases I and II (p = 0.05, by Wald-Wolfowitz runs test comparing Phase III with Phase I and II)

The concept of the dynamic nature of the chondrocyte is not new, and the idea that the synovial fluid provides good tissue culture conditions and allows cartilage bodies to grow and change compositional and shape is a recurring theme (Jones 1924; Phemister 1924; Sokoloff 1974; Milgram 1977a,b; Barrie 1978). The fact that loose bodies from different causes can assume similar histological appearances given time (Phemister 1924) can make their origin very uncertain. The loose bodies described here lack any of the changes associated with time, so indicating their recent formation.

Cases with synovial involvement reported in the literature over the last 100 years have, on average, histories of about six years duration (Jones 1924; Milgram 1977a,b). The routine use and availability of arthroscopy means that intra-articular examinations are being performed much earlier in the course of joint disease. One effect of this should be that synovial osteochondromatosis is diagnosed in the earlier phases of the disease; this does not seem to be the case.

We suggest that 'snow storm knee' is a result of chondrocyte culture within the synovial fluid of the joint. Minor trauma is probably the initiating factor, damaging articular cartilage and allowing the subsequent release of cartilage and chondrocytes into the synovial fluid.

The loose bodies we describe can only fit within the pathological framework of synovial osteochondromatosis if Milgram's theory is turned on its head. Milgram Phase III patients then represent the first step in the disease process; loose bodies are formed by chondrocytic cloning within the culture medium of the synovial fluid. These loose bodies are eventually taken up by the synovium, in an attempt to clear them from the knee joint, producing the appearances of Phases I and II.

The ability of the synovium to remove bodies from various sources is well recognised both clinically and experimentally (Jones 1924). The macroscopic and histological appearance of cartilaginous bodies in various states of attachment to the synovium is well known, but there is no evidence to indicate whether a body attached by a synovial stalk represents one about to drop off or one just becoming attached. Because of continuing movement, loose bodies probably become attached and detached with some regularity until they are fixed firmly within the synovium.

Fig. 3
Dentritic cells on the surface of a loose body. Nomarski phase optics (a, ×400; b, ×500).

Fig. 4

The synovial origin of loose bodies has been supported by describing mechanisms of re-attachment, vascularisation and resorption to account for older-looking bodies in the synovium and the spontaneous radiographic disappearance of calcific bodies. The implications are that bodies may become detached then attached again. The concept that loose bodies develop primarily within the synovial fluid and are gradually taken up by the synovium is far simpler and fits well with the evidence.

In all our cases the synovium was basically normal. Previous reports on cartilaginous bodies attached to synovium note that blood vessels are associated with them and that some contain true bone. True bone formation, as opposed to calcification, can only take place in the presence of a blood supply. This would suggest that the loose bodies become attached, vascularised and then form new bone. Had they developed in the synovium and dropped off one would expect to find both vascular remnants and bone.

The accepted view of synovial osteochondromatosis is that it is a benign neoplasia or a form of metaplasia. Neither of these pathologies can explain either spontaneous resolution, or resolution after incomplete synovectomy or simple removal of loose bodies (Jeffreys 1967). McIvor and King (1962) describe the removal of loose bodies coupled with the fortuitous cessation of synovial activity. Resolution is more easily understood if the loose bodies result from chondrocytic culture rather than synovial neoplasia.

Conclusions. We suggest that our cases challenge the accepted aetiology of multiple loose body formation. They either represent a new and discrete clinical and pathological entity, or are the earliest stage in a revised theory of synovial osteochondromatosis. The term snow storm knee is suggested to describe the dramatic arthroscopic appearance which follows a short history.

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REFERENCES