THE PATHOGENESIS AND SIGNIFICANCE OF MENISCAL CYSTS

H. J. BARRIE

From the Orthopaedic and Arthritic Hospital, Toronto

From a total of 1571 surgically excised menisci, 112 (7.1 per cent) were found by gross and microscopic examination to contain one or more cysts. All of these cysts were associated with tears, either primarily horizontal or with a horizontal component. Tracks were often demonstrable leading from the tear to the cysts, and in some cases of osteoarthritis, detritus of bone could be found in their periphery. It is concluded that the cysts are fuelled by synovial fluid. The relationship of cysts to "myxoid" change of the meniscus is discussed.

The first description of a meniscal cyst was made by Ebner in 1904 and, since then, there have been innumerable papers on the subject. Theories regarding their pathogenesis may be found in reviews by Wijnbladh (1938), Hertz (1955) and Smillie (1962). There would be no point in adding to the literature were it not that the commonly accepted explanation is that they resemble ganglia elsewhere, and are due to primary degeneration of mesenchymal tissue. In the case of common ganglia, this theory owes more to the failure of attempts to find a communication with a synovial cavity than to anything else.

This paper points out that such is not the case with meniscal cysts. It reports a study of 112 surgically excised specimens of cystic menisci, all of which showed horizontal tears, or bucket-handle tears which had a peripheral horizontal component, and, in the majority, tracks were present linking these tears with the cystic areas. The natural conclusion was that these cysts were fuelled by synovial fluid, a possibility which was suggested by Gallo and Bryan (1968). The situation was a little more complex than this as so-called "myxoid degeneration" of the inner zone of the meniscus could be found in unruptured menisci (Topler 1933; Smillie 1962). Furthermore, Liese (1937), Krauss (1939) and Schallock (1939) all claimed that this well-known appearance had nothing to do with a degeneration but was a consequence of traumatic loosening of the collagen fibres with increase of ground substance. Liese (1937) suggested that the synovial fluid was the source of this increased ground substance. To avoid the prejudice inherent in the name "myxoid degeneration", this paper will use the term "foamy change". It will show that a series of changes is recognisable, ranging from simple foamy change to tears with microcyst formation and then to an extension of the cyst formation towards the periphery.

MATERIAL AND METHODS

This is a retrospective study of 112 surgical specimens indexed as meniscal cysts out of a total of 1571 excised menisci, an incidence of 7.1 per cent. The number of sections that had been cut from each meniscus ranged from one to twelve, and they had been stained with haematoxylin and eosin. Many of the blocks were recut and restained using the common range of laboratory stains, but relying mainly on phosphotungstic acid haematoxylin, haematoxylin and eosin, and alcian blue. Some of the slides were incubated with commercial hyaluronidase and some with bacterial hyaluronidase before staining with alcian blue. All sections were examined under polarised light to establish the fibre patterns in the three zones: Zone 1, the inner avascular portion of the meniscus; Zone 2, the outer vascular portion; and Zone 3, the paramenisceal connective tissue. A study was made of the tears present, the presence and degree of foamy change, the cellular reactions around the cysts, the changes in the large vessels, the capillary hyperplasia, the penetration of capillaries into the avascular zone, the content of the cysts, and any fibrin deposits and haemorrhages. Clinical records were consulted to establish the age, sex, history of trauma and findings at operation.

RESULTS

Anatomy. The structure of the meniscus was found to correspond in the main to the description given by Schallock (1939). Its bulk is formed of closely packed tendinous fibres lying in the long axis and bound together by oblique fibrils. The smooth surfaces are formed by a thin mat of closely packed collagen fibrils running radially. Additional isolated radial fibres accompany the blood vessels in the vascular zone and continue centrally where a progressive decline in the

H. J. Barrie, F.R.C.Path., Pathologist, Toronto East General and Orthopaedic Hospital, Inc., 825 Coxwell Avenue, Toronto, Ontario, Canada M4C 3E7

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number of longitudinal bundles results in the inner portion being composed solely of radial fibres. In about 16 per cent of the menisci reviewed the radial fibres formed a central raphe which could be recognised macroscopically (Fig. 1). A few of the radial fibres are curved and end in the surface mat. In the vertical plane a few fibres cross the meniscus irregularly from the inferior to the superior articulating surface. The meniscus has a structure akin to that of a tendon but, unlike a tendon, has radial fibres and a metachromatic ground substance. It is of importance that the collagen fibres of the meniscus are not interwoven but constitute a felting, and the transition from Zone 2 to parameniscal tissue can be established easily by polarised light as in the latter the collagen fibres are woven. A thick-walled artery runs longitudinally in the parameniscal tissues and gives off uniform, evenly-spaced parallel branches running centrally and radially in Zone 2 as far as a line joining the points of reflection of the synovium.

Size and distribution of cysts. One case differed from the others. The cyst was unilocular, measuring $3.5 \times 2.5 \times 2.5$ centimetres and was paramenisceal without any involvement of the meniscus proper. It ran under the patella and had replaced most of the fat pad. It was considered to be an adventitious bursa. In the other specimens, cysts were multilocular. Measurement of their size was only a partial indication of the amount of fluid in the tissues, as much distension was often present in Zones 2 and 3 without actual cyst formation, and the cysts in Zone 3 had often ruptured during meniscectomy, thereby leading to difficulty in measurement. As far as possible, an arbitrary but practical division into four groups was made of the cysts according to their size: Group 1—cysts 0.3 millimetres in diameter or less; Group 2—cysts 0.4 to 0.9 millimetres; Group 3—cysts 1 to 9 millimetres; and Group 4—cysts 1 centimetre or over.
In Zone 1 there was no hard line to be drawn between the microcysts and severe foamy change, for it was simply a matter of the fluid present. The seventy microcysts of Group 1 were found in all zones. These small cysts lay beside a laceration. In half the specimens, cysts were present in two or more zones, giving a total of 215 cysts, and the distribution of the various sizes of cysts in the three zones is shown in Figure 2. Material staining positive with alcian blue was found in all cysts (unless they had lost all their content). Many of the collagen fibres bordering the cysts also took up this stain but there was no colouring of the contents of the cyst nor of the fibres in slides which had been incubated in commercial hyaluronidase. Bacterial hyaluronidase abolished the staining of the contents of the cyst but not that of the collagen fibres. It is probable therefore that the cysts contained hyaluronic acid and that the glycans in the collagen fibres were sulphated.

**Foamy change.** The foamy change (the so-called mucoid or myxoid degeneration) is commonly studied in transverse sections of the meniscus but a better understanding is achieved by sections cut in the longitudinal plane (Figs. 3 and 4). The early phase of the foamy change was found to be due partly to an increase in the size of the collagen bundles caused by separation of their fibrils, and partly to an accumulation of alcian blue-positive material running parallel to the bundles, and thus forming fissures. This change was also found in menisci without local tears, and in them the change was always central. There was no change in the size and staining reactions of the cells which were merely separated more widely from each other. A significant increase in the amount of fluid leading to microcyst formation was nearly always associated with a local tear. Pools of fluid were often elongated in the direction of the collagen bundles (Fig. 4).

**Meniscal tears.** Tears were present in all of the cystic menisci. Microcysts limited to Zone 1 occurred with a variety of tears, but cysts in Zones 2 and 3 were always associated with horizontal tears. Usually these started at or near to the inner margin of the meniscus (Fig. 5). Some were very short and careful inspection was needed if they were to be recognised. Others were of the bucket-handle type but showed a horizontal component directed towards or involving Zone 2 (Fig. 6). Cysts were never associated with pure vertical tears either longitudinal or transverse. Thus, the horizontal tear was of overriding importance to cyst formation. Discoid or semidiscoid menisci with long horizontal tears and the parrot-beak deformity of Smillie (1962) were present in nine specimens. Tears had often developed an eosinophilic lining giving positive fibrin reactions with phosphotungstic acid haematoxylin and Weigert’s fibrin stains, and had occasionally developed an endothelial lining. Tears often followed the course of radial striae.

**Tracks.** In sixty of the menisci, discontinuous tracks were visible between the end of the tear and the site of the peripheral cysts. The majority could be seen to run beside the radial striae, and in Zone 2 this meant a close relationship to the radial vessels. These tracks sometimes contained glycans but in most instances there was just a faint blue staining of the fibrils of connective tissue. Tracks often diverged to follow two or more radial striae, each leading to a separate collection of cysts (Fig. 7). In Zone 3, tracks were not discernible but longitudinal sections showed that the spread of fluid had been longitudinal, often forming a spiral or a varicosity with deep septa between the loculi.

**Trauma.** Clinical records, which were available in all but four cases, revealed a history of injury in 51 per cent of the lateral menisci with cysts, and in 54 per cent of the medial menisci.
Reactive changes. Table 1 shows the reactive changes present and their relative incidence. They correspond to what has already been described in the literature and need little elaboration. Vascular stenosis was a common finding in the paramenisceal region. The complete vascular occlusions were associated with large cysts, around which considerable reactive fibrosis and vascular hyperplasia were noted. Capillary hyperplasia was present in Zones 2 and 3 at the stage in which fluid was first gaining access to the tissues. It was at this early stage that the fluid infiltrating interwoven collagen fibres in Zone 3 gave rise to the “myxoid” appearance. When cysts were well developed they had a lining similar to that of ganglia. An “endothelial” lining was present in parts of some cysts in 27 per cent of the specimens but synovial differentiation was not a feature of the early stages. In two specimens, haemorrhage had occurred sufficiently often to have resulted in localised aggregates of spindle cells and multinucleate cells of the type found in villonodular synovitis. One of these aggregates formed a well-demarcated nodule in Zone 2. Vascular penetration from Zone 2 to Zone 1 had usually occurred along an acquired track or fissure.

**Table 1. Reactive changes (per cent)**

<table>
<thead>
<tr>
<th>Vascular</th>
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<tr>
<td>Occlusion</td>
<td>5.2</td>
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<tr>
<td>Stenosis</td>
<td>3.6</td>
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<tr>
<td>Hyperplasia</td>
<td>35.1</td>
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<tr>
<td>Penetration</td>
<td>23.9</td>
</tr>
<tr>
<td>Haemorrhage</td>
<td>5.2</td>
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<tr>
<td>“Endothelial” lining</td>
<td>27.0</td>
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Associated changes. Two of the menisci contained deposits of calcium pyrophosphates but these deposits bore no anatomical relationship to the cysts. Many showed the yellow pigment of lipid deposition but again this bore no constant relationship to cysts, tracks or foamy change. There was a negative correlation with rheumatoid disease. In spite of, or perhaps because of, the severe fragmentation of menisci which is found in many rheumatoid joints, we have never seen associated cysts. Twenty-eight of the patients had had moderate to severe osteoarthritis. This did not seem to have any special significance except for the occurrence in five of
them of recognisable bone detritus in and around the cysts, even in Zone 3, a phenomenon that was regarded as being extremely important as an indicator of the direction of fluid flow. Material indistinguishable from hyaline cartilage was also observed in some of the cysts and was at first thought to have migrated from the cavity of the joint. Further studies suggested that it could have developed by metaplasia in the connective tissue of attenuated septa which were being stretched and isolated between expanding cysts.

DISCUSSION

There seem to be three stages of glycan accumulation in menisci. In the first, the amount is minor, the disposition is central and there may be no visible breach of the surface even though the central fibres are disrupted. In the second, there is a laceration alongside which there is a major accumulation of glycans resulting in the formation of microcysts. In the third, there are pools forming cysts in all zones, the largest accumulations in Zones 2 and 3 frequently being connected to tears by fissured tracks. The nature of the glycans, the cause of disruption of the central tissue, the cause of horizontal tearing and the sequence of changes leading to the formation of a cyst peripherally all merit consideration and discussion.

Origin of glycans. This material could either be secreted by the cells of the meniscus or be derived from synovial fluid. Cellular secretion is implicit in the theory of "myxoid degeneration", and King (1940) pointed to signs of activity of the Golgi apparatus in support of this theory. His material was derived from peripheral cysts where the activity of the cells could equally as well have been a sign of absorption of synovial fluid. The synovial fluid of the knee must normally diffuse into Zone 1, as it does into cartilage, as both cartilage and the meniscus can live as loose bodies in the joint. It is probably the source of the metachromatic ground substance in the normal meniscus. Liese (1937) and Schallock (1939) both criticised the term myxoid degeneration, stating that such an appearance in the centre of the meniscus was due merely to a disruption of collagen fibres and fibrils together with an increase of intercellular material almost certainly derived from the synovial fluid. Liese (1937) reproduced the appearance of this foamy change by exposing crushed portions of calf meniscus to synovial fluid.

Disruption of the tissue. The central disposition of this change may well be explained by the fact that during rotation under load, the upper and lower surfaces of the meniscus are under forces acting in opposite directions, thus producing a central shearing stress. As disruption occurs this could be followed by an accumulation of fluid, as happens in other tissues of the body. Where contact with the condyles is over a wide area, such as in a discoid meniscus, the greater is the tendency towards central disruption. Extension of this process could lead to horizontal tearing which would provide free access of synovial fluid and the formation of a bellows which, during movements, might propel the fluid peripherally. The close association of peripheral cysts with horizontal tears was noted by Lindblom (1948). The longstanding controversy (Raszeja 1938) as to whether the foamy change precedes laceration or vice versa, may be resolved by saying that either can occur, the laceration merely producing greater degrees of foamy change together with microcysts.

Sequence of changes. Zone 1 is composed of compact tissue under intermittent pressure and hence it is understandable how large cysts cannot form there. Zones 2 and 3, however, are not under pressure and are rich in lymphatics (Rostock 1940). Thus only a small breach into Zone 2 would allow free access of fluid and
the extent to which this zone can be expanded is shown in Figures 8 and 9. The semi-rigid nature of Zone 2 allows tracks or tears to be recognisable in transverse section. Fluid gaining access to Zone 3, either directly through a track, a fissure or through the lymphatics, is no longer in a semi-rigid environment and is free to accumulate and spread, which it does in a longitudinal fashion. The loose peripheral attachment of the lateral meniscus allows full freedom of expansion. The attachment of the joint capsule to the medial meniscus has a limiting influence but if the fluid penetrates this capsule it may track down below the periosteum creating the ganglion migrans described by Wijnbladh (1938). In Zones 2 and 3 the blood vessels are susceptible to rupture and it may well be that injury could sometimes bring the cysts to clinical attention by producing bleeding into them. A direct blow on the side of the knee would also cause tearing of the tissues, including the lymphatics, just as Martin (1920) postulated in the case of ganglia. In the knee, however, it would not be pure lymph that escaped but synovial fluid in the process of being absorbed. For every meniscus which shows an increased uptake of synovial fluid leading to cyst formation in Zones 2 and 3, there may be many in which a balance is struck between uptake and resorption.

CONCLUSION

The established facts regarding menisceal cysts are as follows. They contain fluid histochemically resembling synovial fluid. They are always associated with horizontal tears which could provide both access to synovial fluid and a pumping mechanism. The numerical distribution of cysts indicates progression from the inner part to the periphery. Tracks are often demonstrable and, in osteoarthritis, detritus may be found in the periphery. Recurrence of cysts is common unless the menisci are removed (Hertz 1955). These features may well be considered of sufficient importance to abandon the accepted concept of a primary "myxoid degeneration" whose nature has never been defined.

The author is grateful to the orthopaedic surgeons of the Orthopaedic and Arthritic Hospital and the Toronto East General Hospital in the provision of the surgical specimens and clinical records that have made this study possible, and to Dr Robert B. Salter for help in preparation of the manuscript.

REFERENCES