THE PATTERN OF BONE AND CARTILAGE DAMAGE IN THE RHEUMATOID KNEE

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Synovectomy has become a common operation in the treatment of rheumatoid arthritis of the knee. The surgeon thus has a unique opportunity to inspect the macroscopic features at various stages of the disease process. In particular the early morphological changes can be studied in fresh material, even before radiological signs have appeared.

Fifty consecutive cases of synovectomy of the knee were studied at operation in order to determine whether there is any constant pattern in the changes seen at synovectomy and, if so, to assess how the pattern is related to 1) the duration of symptoms in the knee; 2) the macroscopic appearance and volume of the synovium removed; and 3) the presence and type of fibrinous bodies.

MATERIAL

Fifty synovectomies performed at the Rheumatism Foundation Hospital were studied. All patients had rheumatoid arthritis fulfilling the criteria of the American Rheumatism Association, and all had had a previous course of medical treatment including, in forty-four cases, an intra-articular injection of osmic acid, without clinical improvement.

The method of synovectomy was standard in all cases. It was done through two parapatellar incisions. The volume of synovium removed was measured immediately before any shrinkage by drying. A record of the bone and cartilage lesions seen at operation was made in "map" form. This information was added to the clinical findings and later transferred to a punch-card for detailed analysis.

There were eleven male and thirty-nine female patients. The average age at operation was forty-five years, with a range from seven to fifty-nine years.

OBSERVATIONS

PATTERNS OF BONE AND CARTILAGE DESTRUCTION

Early in the series it became clear that there were two distinct types of cartilage destruction. There were some small areas where the cartilage had been completely destroyed. These defects often extended down to and sometimes into the underlying bone and were usually filled with a soft granulation tissue. They will be called "erosions". In other situations the cartilage had been roughened, softened and "fibrillated" but not removed completely.

Amongst the fifty cases a complete spectrum of cartilage damage was seen. Only three knees showed no evidence of cartilage damage. In two cases damage was confined to the patella. Each of these five patients had had symptoms for less than a year, and in only one of them did the synovium appear active. All the remaining cases showed damage to the femoral condyles with or without evidence of patellar or tibial cartilage damage.

The pattern of destruction of the femoral condyles—The earliest sign of cartilage destruction was an area of softening running from the lateral margins, across the femoral condyles, to the intercondylar notch. The area lay in front of that part of cartilage which articulates with the tibial condyles and menisci when the knee is straight. This early finding was seen in only two knees, but in both cases the disease was minimal: in one of them symptoms had been present in the knee for only three months. The synovium was smooth and inactive.
The amount of synovial fluid was minimal and the volume of synovium removed was only 40 millilitres. This area was easy to pick out after the injection of osmic acid because damaged cartilage then becomes lightly stained (Fig. 3).

Peripheral erosions were the most common finding. Their geographical distribution was remarkably constant. Figure 1 is a map of the femoral condyle showing the sites of erosions in twenty-one knees with early peripheral erosions only. This shows that the areas a–b–c–d are most commonly invaded. Figure 4 is from such a case.

Erosions in these sites seemed to be much more extensive than elsewhere around the periphery. Further extension of this pattern was seen in an additional seven cases in which the areas a and b extended towards, and met, the areas c and d respectively. Thus a “line” was formed running horizontally across from the intercondylar notch to the lateral border of the articular cartilage (Fig. 5).

Extension of the erosive pattern then appeared to progress indiscriminately except for a distinct tendency to spare the central weight-bearing cartilage. Figure 2, illustrating this point,

![Figure 1](image1.png)  ![Figure 2](image2.png)

Figure 1—Outline of the visible cartilage on the femoral condyles showing the commonest sites for early erosions in twenty-one knees. Figure 2—Map of the visible area of the femoral condyle showing the sites of erosion in all the fifty cases.

was compiled from the erosive pattern in all the cases. Two cases were found in which there was evidence of repair along this line: an irregular thin cartilage layer had been produced along the ridge.

Mild osteophytosis was seen in eighteen knees, all of which showed extensive cartilage erosions. Frank eburnation was seen in only five cases.

**Patella**—The cartilage of the patella showed unique features. It was possible to grade the changes found in the patella into the five groups shown in Table I.

The earliest change was softening of the central patellar cartilage which later became thickened and then showed fibrillation (Fig. 6). Such thickening did not occur elsewhere in the knee. Thickening tended, however, to affect only the central part of the patellar cartilage.

Erosions only occurred as a late change and then they occurred predominantly from the lateral edges of the patella rather than from the proximal border. Bone was invaded initially at the same site as cartilage, but later the cartilage destruction advanced ahead of the bone destruction.

**Tibial condyles**—Here cartilage erosions were much less common. They occurred around the periphery of the condyles and were present in only fifteen cases. Ten other cases showed a peripheral “groove” under the tibial plateau due to bone erosion by granulation tissue but without macroscopical destruction of the overlying cartilage. Here the erosions were sometimes
Figure 3—Early cartilage softening on the femoral condyle. Figure 4—There is a large erosion on the lateral femoral condyle and softening of the opposite condyle was found along the “line”. (The black staining of the synovium is from the intra-articular injection of osmic acid before operation.) Figure 5—A large erosion is seen running across the medial femoral condyle with adherent pannus. (The black staining is from selective uptake of osmic acid by the synovium.) Figure 6—Fibrillation of the patellar cartilage. Figure 7—Erosions beneath the tibial condylar cartilage.
quite deep into the bony trabeculae, but the overlying cartilage was surprisingly resistant to destruction. An early case is shown in Figure 7.

In one case there was a large area of collapse of the cartilage into a large rheumatoid cyst in the cancellous bone of the medial tibial condyle, but the cartilage itself had not been eroded. In two cases there was no residual cartilage on the tibial plateau, the surface consisting entirely of eburnated bone.

**Menisci**—In twenty-five cases the menisci had either disappeared completely or were less than half the expected size; all had had swelling in the knee for six months or more. In all the synovium was either very active or the destructive changes were already severe. The menisci

### TABLE I

**Macroscopical Appearances of the Patella in the Fifty Cases**

<table>
<thead>
<tr>
<th>Appearance</th>
<th>Number of cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>Not macroscopically involved</td>
<td>11</td>
</tr>
<tr>
<td>Softening of the cartilage</td>
<td>15</td>
</tr>
<tr>
<td>Chondromalacia—softening of the cartilage with fibrillation or fissuring of the surface</td>
<td>12</td>
</tr>
<tr>
<td>Erosions</td>
<td>10</td>
</tr>
<tr>
<td>Osteoarthritic changes (eburnation or osteophytes)</td>
<td>7</td>
</tr>
</tbody>
</table>

* Five cases showed both erosions and chondromalacia

### TABLE II

**Length of Symptoms Related to Findings at Synovectomy**

<table>
<thead>
<tr>
<th>Length of symptoms</th>
<th>Number of cases</th>
<th>No cartilage changes</th>
<th>Cartilage changes</th>
<th>Average synovial volume (millilitres)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Six months or less</td>
<td>9</td>
<td>5</td>
<td>3</td>
<td>0</td>
</tr>
<tr>
<td>Six months to one year</td>
<td>15</td>
<td>5</td>
<td>8</td>
<td>2</td>
</tr>
<tr>
<td>One to two years</td>
<td>14</td>
<td>6</td>
<td>8</td>
<td>0</td>
</tr>
<tr>
<td>Over two years</td>
<td>12</td>
<td>1</td>
<td>5</td>
<td>6</td>
</tr>
</tbody>
</table>

were destroyed from within outwards, the peripheral rim remaining until the last. Menisci were usually quite mobile and easy to remove, showing that the peripheral attachments had been weakened.

**The Pattern of Degeneration Related to the Duration of Symptoms**

The cases fell naturally into four groups based on the length of symptoms (Table II). As expected, cartilage erosions became more pronounced with increasing duration of the disease process, but an important observation was the speed at which the changes occurred. Even within six months, one-third of the cases had progressed to the stage of having a "line" across the femoral cartilage together with other peripheral erosions.

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The incidence of osteophytosis and eburnation of the bone increased rapidly after the
first year of the disease. It was noted that radiologically osteoarthritis was not seen so often
as it was macroscopically.

MACROSCOPICAL APPEARANCE AND VOLUME OF THE SYNOVIAL REMOVED

Table II also shows that the volume of synovium removed was markedly greater in those
cases having symptoms for six months or less. As the length of symptoms increased, so the
volume of synovium removed decreased, only to rise again in the group with symptoms for
two years or more. However, cartilage destruction progressed relentlessly.

Neither the appearance nor the volume of the synovium removed bore a constant
relationship to the damage occurring on the cartilage surfaces. The largest amount of synovium
removed from any case was 450 millilitres and the knee showed only moderate erosions.
The patient was only twenty-two years old and it exemplified a feature in the series that the
younger the patient the more resistant was the cartilage to rheumatoid erosions.

FIBRINOUS BODIES AND FEATURES OF THE SYNOVIAL FLUID

Massive fibrinous bodies—Four cases had massive fibrinous bodies which were either discrete
or matted together as a large shaggy mass. Three had had symptoms for more than three
years and the fourth for two years. Only one showed severe cartilage changes and the other
three showed very mild changes only. In this variation of the disease cartilage destruction is
mild and occurs late.

Small fibrinous bodies—Fourteen other cases had many smaller but discrete fibrinous bodies.
The duration of symptoms had been up to two years and the pattern of destruction was more
extensive and rapid than in the four cases with massive fibrinous bodies.

DISCUSSION

This study was undertaken in response to a plea by Taillard and Lagier (1969) for more
detailed observations on the macroscopical appearances of the interior of the knee joint at
synovectomy. By this means it may be possible to gain insight into the pathological process
causing joint damage.

It is important to distinguish between cartilage lesions and bone lesions. Moberg (1969)
maintains that, in the metacarpo-phalangeal joint the pattern of bone erosion is relatively
constant and easily visible radiologically. According to Moberg, bone destructions “are
always found along the nutrient foramina of the bone, which became enlarged. Granulations
will invade along these vessels, enlarge them further and destructions are initiated”. This
seems to be the mechanism involved in the tibial plateaux in the knees in this series, where
the damage is subchondral. Moberg continued “...lesions in the cartilage are quite different
from case to case...lesions in the cartilage are very similar to the corrosion on the surface
of a metal plate”. The pattern of lesions in the femoral condyle seen here suggests that the
cartilage lesions are in fact relatively constant. There may be a direct anatomical reason
for their appearance; for example, it may be that those areas of cartilage in contact with
synovium only are liable to be destroyed.

It remains in doubt how cartilage is destroyed in rheumatoid arthritis. Damage could
theoretically be produced by 1) a diffuse enzymatic process; 2) local destruction due to some
inherent property of the invading pannus; or 3) a primary disease process in the chondrocytes
themselves. Support for 1) comes from Kulka (1959) who reported that in destructive cartilage
lesions, pannus begins at the articular margins and advances centripetally. In active lesions
the chondrocytes are necrotic well in advance of pannus so that the synovial granulation tissue
is mainly replacing devitalised hyaline matrix. The evidence of this series supports this concept
of cartilage destruction on the femoral condyles. The area of softening seen on the condyles before erosions appear suggests that the cartilage is damaged before pannus erodes it.

The unusual feature of the cartilage over this linear area is that it is always in contact with the synovium covering the alar pad of fat. The cartilage here also never comes into contact with another area of articular cartilage, either on the patella or on the tibial condyles. This raises two possibilities: either the cartilage is damaged by direct contact with synovium, or the absence of pressure from another area of articular cartilage deprives the cartilage of nutrients so that it becomes especially sensitive to enzymatic activity or pannus.

Salter, McNeil and Carbin (1966), in their paper on the experimental destruction of normal cartilage, found that cartilage disappeared where it was constantly in contact with synovium in a deformed joint. The cartilage was apparently neither eroded nor actively destroyed by synovium in these non-rheumatoid joints. Earlier, Salter and McNeil (1965) had reported that in human joints with persistent deformity, those areas of articular cartilage which were persistently in contact with synovial membrane underwent progressive degenerative changes. In the absence of movement, the synovial membrane became adherent to the adjacent articular cartilage and this prevents nourishment from taking place.

In the cases in this series the "linear" erosion was usually, but not always, covered by pannus, but in no case was the synovium from the front of the joint adherent to the area.

Ball (1969) points out that cartilage erosion is focal and must involve a highly controlled release of degrading enzymes. Enzymes released into the synovial fluid may produce a general loss of metachromasia but do not produce general dissolution of cartilage. In this series two features suggest cartilage destruction by generalised enzymatic activity: 1) the high incidence of softening and fibrillation of patellar cartilage—seen in over 50 per cent of cases without contact with synovial membrane; 2) the menisci are commonly absent or at least markedly reduced in size.

There was no evidence of destruction of the menisci by pannus in early cases where the menisci were partially intact. This is in direct contradiction to the findings of Stevens and Whitefield (1966), who, in a series of 102 knees, found that the menisci were often absent, but when present were usually covered by pannus suggesting local destruction by pannus. Wegelius, Klockars and Vainio (1970) found that meniscal disintegration started at the anterior and posterior horns. The middle part might still be fairly normal. Enzyme activity was higher in the soft degenerating parts of the menisci than in the still healthy parts.

From all the above it can be postulated that in the rheumatoid knee joint cartilage damage is produced mainly by enzyme activity which is most pronounced where the synovial membrane comes into contact with a plane surface of articular cartilage. These changes can be surprisingly rapid in the diseased joint but are unrelated to the macroscopical appearance or volume of the synovial membrane. Cartilage to cartilage contact protects articular cartilage from damage by synovium. Massive fibrinous bodies also prevent synovial membrane from coming into face to face contact with cartilage. However, primary disease of the chondrocyte cannot be entirely excluded. The longer survival of those parts of cartilage under direct pressure could possibly be caused by better nutrition and stimulation of the chondrocytes by pressure.

**SUMMARY**

1. Fifty knees affected by rheumatoid arthritis were studied in detail at synovectomy.
2. The destructive lesions found were relatively constant and are described in detail.
3. Cartilage lesions were much more common than was expected radiologically.
4. The pattern described suggests that articular cartilage is destroyed by contact with diseased synovial membrane but protected by contact with another cartilaginous surface.

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REFERENCES


