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SOFTENING OF THE CARTILAGE AND ARTHRITIS OF THE RHEUMATOID TYPE

Discussion in Relation to Classical Aspects of Chondromalacia

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For some years synovectomy has often been used in the treatment of chronic inflammatory rheumatism; a reason is the hope that it will interrupt the course of deterioration of the cartilage secondary to synovitis. It is therefore logical to make use of the information it provides to study minor or incipient forms of deterioration which herald the advent of the classical lesions in their developed form. Such a study is possible in conditions not afforded by post-mortem examination, and morphological correspondence of cases under clinical study can be detected in fresh material; but such evidence must be interpreted within the limits of normal changes in subjects of the same age.

The observations reported here were made on patients with arthritis of rheumatoid type, the diagnosis of which had been clearly established clinically, on joints with a synovitis that had been histologically verified, and on hyaline cartilaginous surfaces that exhibited neither patches of ulceration nor pannus (apart from a few slight marginal expansions of connective tissue).

MATERIAL AND METHODS

The clinical material consisted of 5 men and 4 women, aged 22 to 58, 7 of whom, including 3 who were seronegative, had adult rheumatoid arthritis; one had ankylosing spondylitis of the Scandinavian type; and the 9th juvenile rheumatoid arthritis operated on in adulthood. All together 12 synovectomies were performed. Most of these operations were performed on the knee and 2 of them were bilateral; but in 2, a group of metacarpophalangeal or interphalangeal joints were affected. All the joints operated on had chronic synovitis that histologically presented the classic rheumatoid picture (except for the case of sero negative rheumatoid arthritis presented below). The following 4 cases deserve special attention, since they are examples of different forms of chronic inflammatory rheumatism.

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Case G. S. (CO 66) (pathological report T 6062/66).

A 22-year-old male, who for more than one year had had ankylosing spondylitis of the Scandinavian type, with swelling of the knees, ankles, and metatarsophalangeal joints. X-ray of the knees showed nothing remarkable. E.S.R. 17/40 mm (at 1 and 2 hours). Latex test negative for the blood and the synovial fluid of the knee operated on. The synovial fluid contained 1200 leucocytes per mm^3 and 31 g/l protein; normal mucin test.

The right knee was operated on because of recurrent hydrarthrosis. Macroscopically, and microscopically, the synovial membrane showed the characteristics of rheumatoid synovitis (Figure 1A), but it was worthy of note that the hyperplasia was less marked than it usually is in rheumatoid arthritis.

Case N.L.R. (CO 66) (pathological report T 10970/66).

A male, aged 49, who had had seropositive rheumatoid arthritis since the age of 20. X-ray of the vertebral column and sacro-iliac joints excluded ankylosing spondylitis. Both knees were the site of recurrent fluid effusions, but no lesion was radiologically demonstrable. E.S.R. 40/73 mm (at 1 and 2 hours). Latex test positive for the blood and the synovial fluid of the knee operated on. The synovial fluid contained 13,200 leucocytes per mm^3 and 49 g/l protein; mucin test abnormal.

The right knee was operated on. The synovial membrane exhibited the macroscopic and microscopic characteristics of rheumatoid synovitis.

Case C.M. (CO 66) (pathological report T 11.175/66).

A female, aged 54, had had seronegative rheumatoid arthritis of the wrists and knees since the age of 40. There had been recurrent effusions into the knees since the onset of the disease, but no change in the joint spaces was radiologically demonstrable. E.S.R. 17/40 mm (at 1 and 2 hours). Latex test negative in the blood and the synovial fluid of the knee operated on. The synovial fluid contained 900 leucocytes per mm^3 and 37 g/l protein; mucin test normal.

The right knee was operated on and the synovial membrane was found to be only slightly thickened, mostly smooth, but with fine villosities in some parts. The difference from classical rheumatoid synovitis was even more obvious under the microscope (Figure 1C and D).

Case F.S. (CO 66–67) (pathological reports T 12.935/66 and T 3080/67).

A male, aged 24, who had since the age of 14 months had juvenile rheumatoid arthritis of the wrists, hands, ankles and knees; no roentgenologic change of the knees. E.S.R. 56/88 mm (at 1 and 2 hours). Latex and haemagglutination tests of the blood negative.

The first synovectomy was performed on the right knee. The synovial fluid contained 38,400 leucocytes per mm^3 and 45 g/l protein; the latex test was negative and the mucin test normal. Macroscopically and microscopically, the synovial membrane exhibited the characteristics of fibrinous rheumatoid synovitis.

Synovectomy of the left knee was carried out 4 months later. The synovial fluid contained 41,600 leucocytes per mm^3 and 55,5 g/l protein; the latex test was negative and the mucin test was slightly abnormal. At the same operation, synovectomy of the metacarpophalangeal joints of the two thumbs was performed. The synovial membranes of these joints exhibited, macroscopically and microscopically,

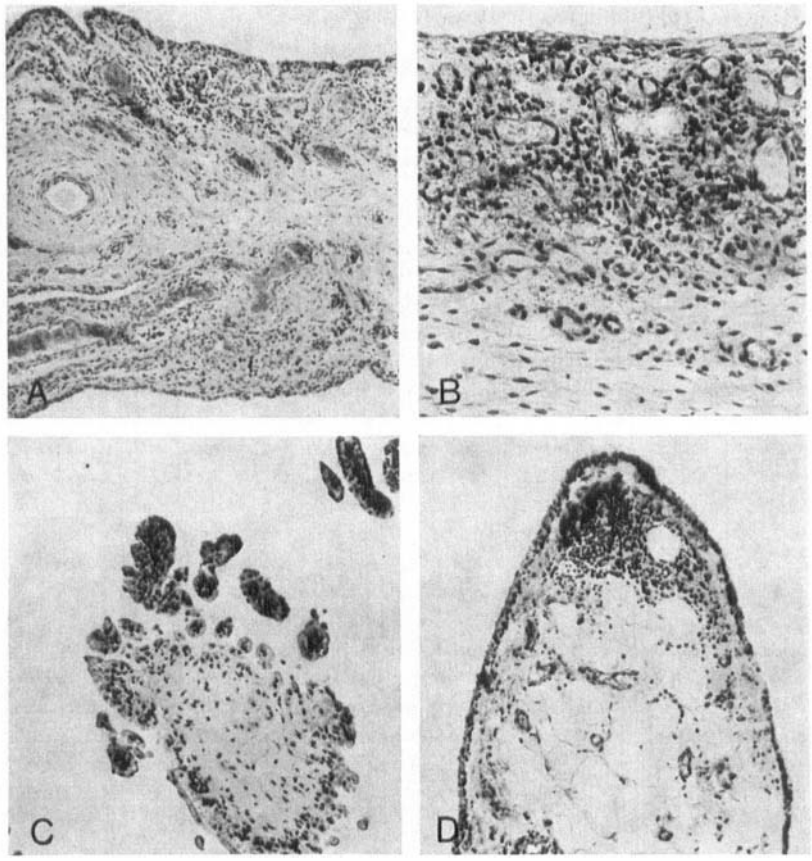

Figure 1. Different appearances of chronic synovitis observed in the cases studied in this paper.

(A) T 6062/66 (haematoxylin-eosin × 52). Ankylosing spondylitis of the Scandinavian type. Synovial fringe with synoviocytes showing very little hyperplasia. Fairly scanty lympho-plasmocytic infiltration. A blood vessel with thickened fibrous wall is noticeable.

(B) T 3080/67 (haematoxylin-eosin × 131). Juvenile rheumatoid arthritis of the right knee. Wall of synovial membrane with no hyperplasia of the marginal layer of synoviocytes, but with new vessels and infiltration of lympho-plasmocytes (mixed with some polymorphonuclear leucocytes).

(C) and (D) T 11175/66 (haematoxylin-eosin × 71, × 86). (C) Small synovial fringe with moderate papillary hyperplasia of synoviocytes; this hyperplasia is dissociated from a lympho-plasmocytic infiltration not visible in this microscopic field. (D) Extremity of fatty fringe with slight nodular lympho-plasmocytic infiltration noticeable. Very little hyperplasia of the synoviocytes.

For the cases denoted by (A) and (B), more classic pictures of hyperplastic rheumatoid synovitis are to be found, with fibrin in case T 3080/67 (ref. Figure 1 of study no 40 of the list of references for case T 6062/66). Figures (A) and (B) therefore clearly show that slighter degrees of synovitis existed in those cases.

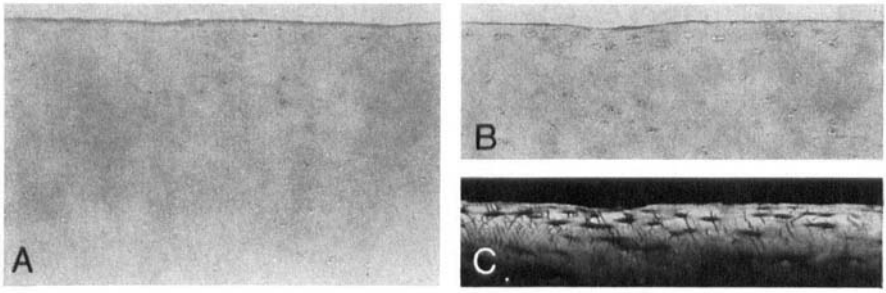

Figure 2. T 3080/67 (toluidine blue × 42). Juvenile rheumatoid arthritis. Specimen of softened patellar cartilage from the left knee.

(A) Structure normal but loss of polysaccharide substance, as shown by disappearance of metachromasia.

(B) and (C) Section adjacent to and analogous to the previous one, photographed by (B) normal light and (C) polarized light. Examination with polarized light shows fine superficial fissures not visible with normal light, corresponding to the separation of the arciform collagen fibres. Even if they are artefacts due to the histological technique employed, they represent the lines of least resistance. Similar appearances may sometimes be seen in cartilage considered normal, even in young subjects.

the characteristics of rheumatoid synovitis (Figure 1B), that of the knee being very fibrinous.

The specimens of softened cartilage in these 4 cases were taken from the central part of the patella, from the hyaline cartilage and at a certain distance from the edges so as to exclude any possible slight extension of the synovial membrane. From this study, therefore, we eliminated marginal specimens that had been taken for the study of slight pannus that had appeared without infiltration of inflammatory cells. Moreover, in these marginal specimens the structure of the proper cartilaginous matrix showed no fundamental change in relation to the rest of the joint cartilage.

The specimens were fixed in 4 per cent formol and embedded in paraffin. We do not think that such fixation impairs the histological demonstration of the acid polysaccharides (13). The sections were stained by different methods: haematoxylin-eosin, van Gieson, toluidine blue (1 per cent aqueous solution at pH 4 during 6 hours), Alcyan blue, and Gomori's silver impregnation. The study was completed by examining the sections in polarized light.

Also specimens of softened patellar cartilage from 7 subjects in whom the diagnosis of rheumatoid arthritis had been formally excluded were studied under corresponding conditions.

OBSERVATIONS

In all the cases but one, palpation of the accessible cartilaginous surfaces during the course of the operation revealed more or less marked

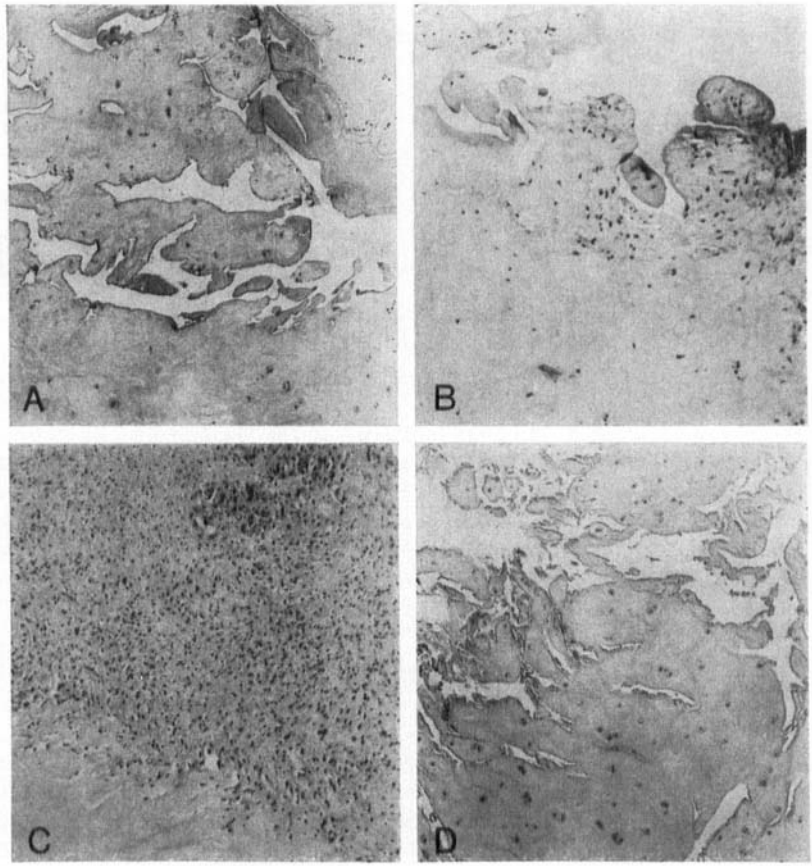

Figure 3. Different aspects of structural deterioration of cartilage observed in the cases studied in this paper.

(A) T 6062/66 (haematoxylin-eosin × 30). Ankylosing spondylitis of Scandinavian type. Frayed patellar cartilage with loss of basophilia, finely furrowed structure, and small islets of chondrocyte proliferation.

(B) T 6062/66 (haematoxylin-eosin × 53). Frayed patellar cartilage with loss of basophilia and replacement of hyaline by fibroblastic or fibrocytic structure. A few rare fibrin deposits on the surface.

(C) T 6062/66 (haematoxylin-eosin × 58). Replacement of hyaline structure (below and to left) by a fibroblastic structure with new vessels (detail of Figure 2 in study no 40 in the list of references).

(D) T 11175/66 (haematoxylin-eosin × 31). Seronegative rheumatoid arthritis. Frayed cartilage with loss of basophilia, some fine furrowing, and some areas of chondrocyte proliferation.

areas of softening of the cartilage. The exception was in the right knee of G.S., where the pearly-white cartilage covering appeared to be of normal consistency. Naturally, no specimen was taken from it.

The softened cartilage was white or ivory in colour and its surface was smooth or slightly velvety. In two cases (one of them being N.L.R.), we noticed slight ulceration in the centre. Although the softening predominated in the centre of the patella, it did not appear to follow any systematic pattern in the joint; this suggested that it was not the result of mechanical factors only, but of more general changes in the joint cavity. It affected the superficial layers, the removal of specimens of softened cartilage leaving behind a base that seemed to be of normal consistency.

One of the most interesting changes was found in the right knee of case G.S.; the left knee had not shown any such changes 4 months previously. The patellar cartilage was ivory white and had remained perfectly smooth, but it exhibited a considerable softening superficial in the centre and on the external aspect, giving on palpation the sensation of pitting oedema. Histological examination of the 4 flakes removed showed that the structure had been preserved, and this was confirmed by silver impregnation and examination under polarized light (Figure 2C). On the other hand, the acid polysaccharide level had fallen; as was shown by the decrease of basophilia when stained with haematoxylin-eosin, by a drop in tinctorial activity with Alcyan blue, and by a loss of metachromasia with toluidine blue (Figures 2 A and B). We consider these changes to be significant on comparison with control sections processed in the same conditions (Figure 4 A).

The fall in the acid polysaccharide level was found in the other specimens of softened cartilage, but it was accompanied by structural change with the beginning of fissuring, the disappearance of the hyaline structure and its replacement by a fibrous structure, and chondrocyte proliferation (Figures 3 A, B and D). Such changes could lead to fraying, and the disappearance of the hyaline cartilaginous structure could even result in the appearance of fibroblastic tissue, sometimes vascularized (Figure 3 C).

Comparison of these different cases suggested that the simplest manifestation of the condition is the fall in the acid polysaccharides without any structural change. Next comes proliferation of chondrocytes, developing towards fibroblastosis or fibrocytosis accompanied by fissuring or break-up of the hyaline structure, which evolves in the direction of a fibrous tissue. The sections from F.S. showed all the

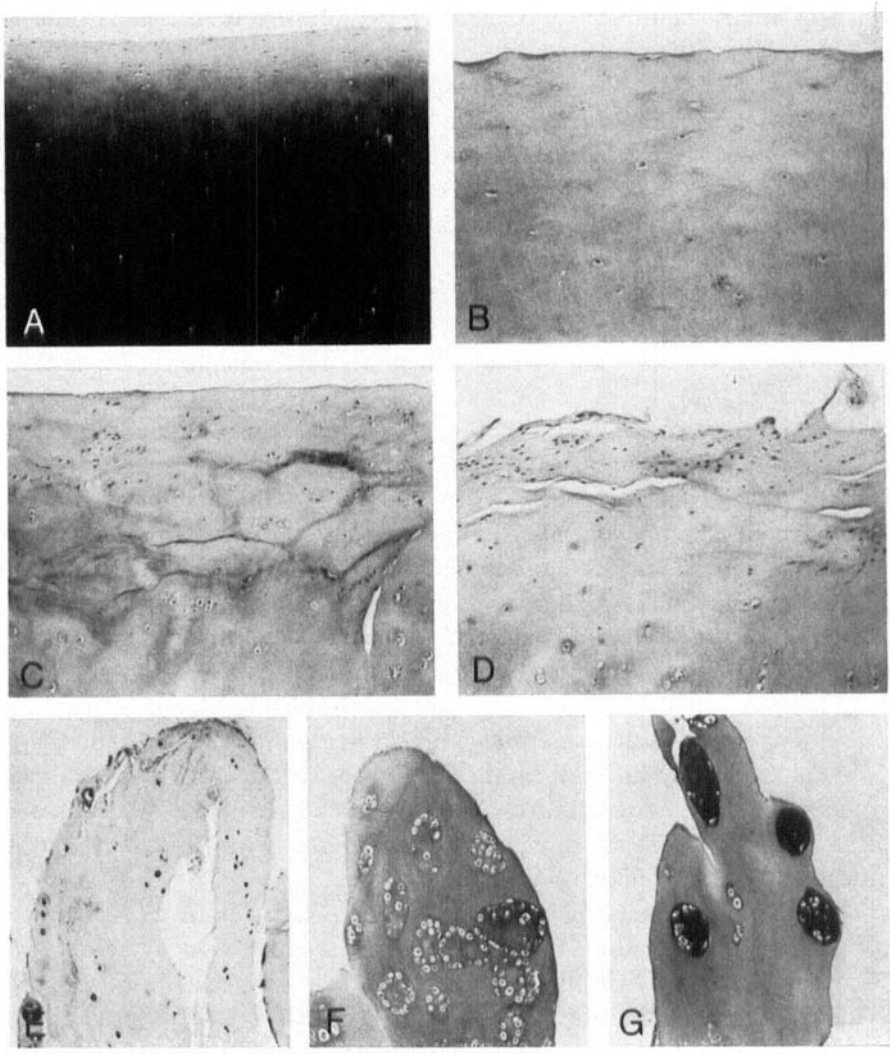

Figure 4. Various appearances of cartilage observed in knees showing the clinical characteristics of chondromalacia patellae after trauma but without rheumatoid disease.

(A) Twenty-two-year-old woman who had twisted her left knee several months earlier. The operation revealed patellar chondromalacia with chronic synovitis and effusion.

(B) to (G) Thirty-five-year-old man who had twisted his left knee on several occasions. The operation showed chondromalacic lesions on the lateral and medial aspects of the cartilaginous surface of the patella and the anterior aspect of the medial femur condyle.

(A) T 6443/67 (toluidine blue × 44). Specimen of patellar cartilage. Normal

stages of this development, some of the sections containing several areas of cartilage with the structure fully preserved and with or without the normal complement of polysaccharides.

DISCUSSION

The number of cases observed was only 12, in 4 of which a systematic histological study was made of the chondromalacia found. They are therefore of interest only as a guide in an analysis of a larger amount of material to be obtained in the future. From this point of view they deserve discussion in the light of three considerations:

- the normal constitution of the hyaline cartilage of the joint,
- the concept of chondromalacia in general,
- the influence of rheumatoid synovitis on cartilaginous tissue.

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structure and staining in this section, with metachromasia of the perpendicular zone. (The metachromasia is reduced in parts of specimens taken from adjacent areas).

(B) T 4686/67 (toluidine blue × 60). Specimen of cartilage from the lateral aspect of the patella. Normal structure. Loss of polysaccharide substance as shown by disappearance of metachromasia.

(C) T 4686/67 (toluidine blue × 60). Specimen from anterior aspect of medial femur condyle. Metachromasia lost but surface normal. Fine furrowing in the depth and sporadic replacement of normal by fibrocytic structure.

(D) T 4686/67 (toluidine blue × 69). Section adjacent to that of (C). The changes previously observed here affect the surface area.

(E) T 4686/67 (toluidine blue × 69). Specimen from the lateral aspect of the patellar cartilage. This piece of cartilage has lost its metachromasia and it frayed easily when subjected to laboratory techniques. Several small areas of chondrocyte proliferation, in places emphasized by a very basophil metachromatic halo.

(F) T 4686/67 (toluidine blue × 69). Specimen from the lateral aspect of the patellar cartilage. Very reduced metachromasia and many areas of chondrocyte proliferation.

(G) T 4686/67 (toluidine blue × 60). Specimen from the lateral aspect of the patellar cartilage. Much reduced metachromasia. Isogenic chondrocyte groups emphasized by very basophil metachromatic haloes.

N. B. As in Figure 2, evidence of the significance of the loss of metachromasia is provided by the successful staining of areas of normal cartilage subjected to the same laboratory techniques and conditions. (Some are to be seen on the same sections, as those in the figures).

1. *The Normal Hyaline Cartilage of the Joint*

The framework of normal cartilage consists of collagen fibres, the arciform arrangement of which has been described in a classical study by *Benninghof*. The fibres are embedded in ground substance containing combinations of proteins and neutral and acid polysaccharides (PP). The latter are the best known, partly because of studies of costal cartilage; they are chondroitin-sulphuric acid, the level of which falls with age, and kerato-sulphuric acid, the level of which rises until about the age of 25 years and then remains fairly constant (26). These acids are produced by the chondrocytes and condition the hydration of the cartilage by bound water (26).

The acid polysaccharides are responsible for cartilage metachromasia, which is normally clearly visible in the perpendicular zone, and not in the tangential and transitional zones (Figure 4 A) (19); the same applies to stainability with Alcyan blue. The whole complex exhibits a consistency whose elasticity can be measured by elastometry (19). The consistency varies according to the site and seems to be related to the mechanical stress.

Hirsch has defined normal articular cartilage as follows: "Thus, when I say healthy cartilage, I mean cartilage in which chondroitin-sulphuric acid is retained in the perpendicular zone, *i.e.* in most of the cartilage, and where its content only varies in the tangential and transitional zones. Cartilage of this kind has been shown to have a typical elasticity curve."

2. *The Concept of Chondromalacia*

(a) *Definition.* First recognized by *Büdinger* in 1906, softening of the articular cartilage has been mostly studied in the patella, where it occurs most commonly and earliest. Various terms have been proposed for it in the German literature: "traumatische Knorpelrisse" (*Büdinger*) (7), "fissurale Knorpeldegeneration" (*Läwen*) (25), "Chondropathie der Patella" (*Frund*) (14). But it is better known under the etymologically descriptive name of "chondromalacia", usually applied to the patella.

The term "chondromalacia", which had already been used by *Aleman* since 1917, appeared for the first time in the literature in 1924 in a paper by *König* (cit. in 20). The definitions of these authors agree with that given by *Hirsch* in 1944: "By chondromalacia of the patella I mean all changes in the patellar cartilage revealed in softening, fissuring or tuft formation or a combination thereof, whether or not any deformity

in the joint or subchondral reaction is present". *Hirsch* thus compared malacic cartilage with normal cartilage on the basis of histological staining supplemented by chemical titration and elastometric measurements defining more accurately the impressions furnished by palpation. "By malacic cartilage I mean herein cartilage which has a poorer pressure elasticity than normal cartilage and in which the reduction of chondroitin-sulphuric acid extends down into the perpendicular zone." (19).

(b) *Macroscopic appearance.* In fact, the concept of chondromalacia covers two appearances clearly distinguished by several authors (14, 19, 32, 37). In the first, the cartilage is simply softened. In the second, it presents surface fraying or fissures [hence the expression "Knorpelrisse" (7) or "fissurale Knorpeldegeneration" (25)]. The fissures may be superficial (19), leaving a macroscopically healthy base after surgical excision of specimens; but they may also be deeper, extending down to the subjacent bone (7, 45).

The latter form is usually explained as a development of the former (7, 14, 19, 37). *Büdinger* thus described the cartilage on the periphery of a fissured zone: "eigentümlich verändert, weich, hydropisch, geschwollen und hat den Glanz verloren"(7). *Hirsch* thought that the fissures appeared at the junction of areas of cartilage of different consistency and summed up his views as follows: "It seems probable, therefore, that in my series cartilaginous fissures were not the primary cause of the patellar chondromalacia. They seemed to occur secondarily in already altered cartilage" (19).

(c) *Microscopic appearance.* The earliest manifestation of chondromalacia, as seen in the microscope, is a change in the staining characteristics of the perpendicular zone of the cartilage: the basophilia diminished (32, 45), and Alcyan blue stains less well, corresponding to a loss of metachromasia with toluidine blue (19) (Figure 4, B). These changes reflect a fall in the level of chondroitin-sulphuric acid.

The general structure is preserved, as can be confirmed by examination with polarized light. The cells may be normal in their arrangement (45) or sometimes already show a certain amount of irregular proliferation.

The next manifestation is uncovering of the fibrils, with fissure formation or even the appearance of small cavities (19, 25, 32, 45) (Figure 4, C and D). Examination with polarized light clearly reveals these changes as well as an important characteristic noticeable even in the early forms: the disappearance of the hyaline structure of the cartilage begins with dissociation of the fibrils and leads to its replacement by a more or less fibrous conjunctival structure (25). This process can also be observed by silver impregnation.

The cells take part in this transformation. Small nests of chondrocytes proliferate, mainly on the margin of the fissures (Figure 4, E and F); or the chondrocytes are replaced by fibroblasts or fibrocytes (25). These cellular changes correspond to the structural dedifferentiation referred to above.

The changes with fissure formation are also accompanied by loss of metachromasia histologically reflecting the fall of chondroitin-sulphuric acid observed with chemical methods (5, 29). In some places, however, nests of proliferating chondrocytes show hyperbasophilia with strong metachromasia corresponding to overproduction of sulphated acid polysaccharides (Figure 4, G). This was also observed in cartilage in rheumatoid diseases (41). *Collins* showed this distinctly by an increased 35 S fixation; he noticed such appearances in fissured cartilage and considered them secondary to changes of the surrounding matrix (10).

Research by *inter alia Öwre* has shown that the proportion of microscopic lesions is higher than the proportion of macroscopic lesions (32).

(d) *Consequences.* Because of this loss of elasticity with the fall in the level of chondroitin-sulphuric acid and often fissure formation, the cartilage no longer fulfils its function as an elastic buffer (19); the pumping mechanism that ensures that it receives its nutrition from the synovial fluid is uncertain. Fragmentation and even disappearance of the cartilaginous covering may then occur, creating the morphological conditions for a "modelé arthrosique" (23). In its completed form, this osteo-arthrosis is characterized by remodelling of the underlying bone, osteophyte formation, visible radiographically, and often associated synovitis (14, 32, 45).

Several authors have stressed the possibility of chondromalacia of the patella being a pre-arthrotic condition (14, 37, 45). This possibility is reflected in the time interval observed in anatomical studies of large series of knees between the maximum age for osteo-arthrosis and that for chondromalacia (37).

(e) *Origin and nosological status.* Systematic study of large series of joints in subjects of different ages, even with no obvious clinical manifestations, has shown how common chondromalacia is, especially in the knee and primarily in the patella. The chief manifestations in the patella are on the medial aspect, which is particularly subject to mechanical stress (31, 43). This suggests that mechanical stresses may play a part in the pathogenesis of the lesions, just as topographic studies of chondromalacia suggest in other joints.

The changes are early; they have been shown in many post-mortem statistics to appear as soon as the second decade (3, 18, 21, 32, 37). *Öwre,* who distinguishes between "oedematous" and "fissural" forms, has seen none of the latter below the age of 20 years; but he has seen the former in 5 out of 18 subjects of a group aged less than 20 years, all below the age of 14 years.

The daily experience of surgeons also testifies to the frequency of chondromalacia, particularly in young patients; in 220 arthrotomies performed mostly on patients between 20 and 30 years of age for various indications, *Aleman* noticed chondromalacia in 33 per cent of the cases.

Surgeons have also observed that certain young patients, suffering from pain after local trauma, presented at operation more or less marked chondromalacic lesions of the patella. Their observations explain some of the various terms that have been used: "traumatische Knorpelrisse" (7), "traumatische Chondropathie der Patella" (14), "Chondromalacia post-traumatica patellae" (1). This evidence, while not excluding the possibility of traumatic fissuring in healthy cartilage (19), provides a justification for the view that the condition is due to the association of trauma with a previous predisposing change (7, 25, 45). *Wiles et al.* sum up this view as follows: "... trauma is of importance more often as an aggravating than as a primary factor, and (that) it acts by disrupting already degenerate cartilage" (45).

The condition forms the nosological entity known under the name of "chondromalacia patellae". Its clinical importance is dependent on the extent of the changes in the cartilage and especially on the subsequent synovial reaction (1, 25, 30, 45). Early chondrectomy has been advocated for its treatment and to prevent it, to a certain extent, from developing into an osteo-arthrosis (1, 6, 25, 30, 44, 45).

3. *Influence of Rheumatoid Synovitis on Cartilage*

The influence of the rheumatoid synovial membrane on cartilage has been suspected for a long time (39), and it has been the subject of more exact study in the last few years, especially since lysosomal enzymes have been revealed in it and in the corresponding joint fluid (2, 16, 27, 33, 36, 42).

Thinning and erosion may indicate the action of rheumatoid synovitis on collagen substance, and in fact a collagenase has been detected in cultures of rheumatoid synovial membrane (11). But this is not the type of change that we are considering here.

Of more interest is the action on the gound substance or, to be precise, on the protein-polysaccharide complexes (PP). *Ziff, Gribetz* and *Lospalluto* have shown that extract of rheumatoid synovial membrane (as also of leucocytes) degrades a cartilage mucoprotein; this action is destroyed by heat (46). *In vitro*, the degradation of cartilage protein-polysaccharide complexes has been achieved by lysosomal extracts (12) or purified fractions (38) and by proteolytic enzymes under certain experimental conditions. The *in vivo* or *in vitro* action of plasmin, a blood protease (22), is analogous to that of non-activated papain (17); it affects the protein moiety and liberates chondroitin-sulphuric acid. Nevertheless, a degradation of chondroitin-sulphuric acid due to enzymes is considered as possible (4, 5 and 8).

It is also possible that cartilage deterioration may also be due besides to changes induced in the synovial fluid by rheumatoid synovitis, manifested in particular by a fall in viscosity (9, 36). This could act by changing the lubrication of friction surfaces and the nutrition by imbibition of the cartilage (17, 36).

These changes are thus capable of destroying the equilibrium of the collagen fibre arcades, which depends on their being embedded in ground substance. The result, perhaps comparable to the one observed in cultured cartilage (34), is fibril formation and cellular changes. Thus a process takes place analogous to that postulated for other chondromalacias observed in joints without rheumatoid synovitis.

CONCLUSION

The concept of a non-specific chondromalacia induced by rheumatoid conditions is suggested by our observations in synovectomized joints and supported by *in vivo* or *in vitro* biological data. Its demonstration would be important in providing an explanation, at least in part, for the origin of the classical developed lesions (9, 15, 35), particularly those of the connective layer known as pannus. The existence of such a process could also explain the appearance of a "modelé arthrosique" in joints with rheumatoid arthritis on which synovectomy had previously been performed at an early stage with apparent clinical success.

Because it would act on complexes with a much more rapid turnover than the adjacent collagen (28, 36), the existence of such a process might make possible an early therapeutic attack. One should recall however that the origin of this process is perhaps not only in synovitis, but also in chondrocytic changes.

Nevertheless, before such a conclusion could be drawn, the concept of rheumatoid chondromalacia would require much more study. Research is needed to establish whether it has characteristics of its own (particularly in relation to site and extent) and to define more closely the range of chondromalacias of other types (particularly in relation to frequency).

It is important then that surgeons, when they carry out arthrotomies on patients with or without chronic inflammatory rheumatism, should make an effort to produce the detailed information required.

SUMMARY

Twelve operations of synovectomy were performed on nine patients with various rheumatoid conditions (adult rheumatoid arthritis, ankylosing spondylitis, juvenile rheumatoid arthritis). They concerned the knees, except in two cases, where groups of finger joints were operated on.

Softened areas were frequently observed on cartilaginous surfaces, with the same macroscopic and microscopic characteristics as in other forms of chondromalacia.

These observations are to be compared to experimental data concerning the role of enzymatic factors in the degradation of protein-polysaccharides complexes of cartilage. The existence of such a process secondary to rheumatoid synovitis (perhaps yet to chondrocytic changes) could explain more advanced articular lesions. Nevertheless, before this concept is accepted, further observations are needed to establish the comparative characteristics of chondromalacia with or without rheumatoid conditions.

RESUME

Douze synovectomies ont été pratiquées chez neuf malades souffrant de différents états rhumatoïdes (arthrite rhumatoïde de l' adulte, spondylarthrite ankylosante, arthrite rhumatoïde juvénile). Il s'agissait du genou, sauf dans deux cas où l'intervention a porté sur des groupes d'articulations digitales.

Des plages de ramollissement ont fréquemment été observées sur les surfaces cartilagineuses, avec les mêmes caractéristiques macroscopiques et microscopiques que dans d'autres formes de chondromalacie.

Ces observations sont à rapprocher de certaines données experimentales concernant le rôle des facteurs enzymatiques dans la dégradation

des complexes protéine-polysaccharide du cartilage. L'existence d'un tel processus secondaire à la synovite rhumatoïde (peut-être aussi à des modifications chondrocytaires) pourrait expliquer des lésions articulaires plus avancées. Néanmoins, avant d'admettre cette conception, de plus amples observations sont nécessaires pour établir les caractéristiques comparatives de la chondromalacie avec ou sans synovite rhumatoïde.

ZUSAMMENFASSUNG

Zwölf operative Synovektomien wurden bei neun Patienten durchgeführt, die an verschiedenen rheumatoiden Leiden erkrankt waren (pcP des Erwachsenen, Spondylarthritis ankylopoietica, pcP des Jugendlichen). Abgesehen von zwei Fällen, wo man die Synovektomie an einzelnen Fingergelenken ausführte, betraf sie in den übrigen Fällen das Kniegelenk.

An Gelenkoberflächen fielen beim Knorpel häufig erweichte Herde auf, die die gleichen makro- und mikroskopischen Kennzeichen aufwiesen, wie jene anderer Chondromalazieformen.

Diese Beobachtungen dürften mit gewissen experimentellen Daten in Einklang gebracht werden, insbesondere was den durch gewisse enzymatische Faktoren bedingten Abbau von Protein-Polysaccharidkomplexen des Knorpels anbelangt. Das Vorhandensein eines solchen Vorganges, sekundär nach einer rheumatoiden Synovitis, vielleicht auch nach Knorpelzelländerungen, könnte fortgeschrittenere artikuläre Schäden erklären. Bevor man aber eine solche Auffassung zulässt sind weitere vergleichende Beobachtungen notwendig, um die Charakteristiken von Chondromalazien mit und ohne rheumatoiden Synovitiden feststellen zu können.

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