Introduction: Gonarthrosis may result from the wear of articular cartilages by the machinery injury of articular cartilages with degenerative changes, which are induced by the loss of the primary function of the meniscus caused by a decline in the quality of the meniscus by degeneration. It is very important to observe the process of meniscal degeneration from the normal condition to functional breakdown in order to clarify the pathogenic mechanism of gonarthrosis. Therefore, in this study, we clarify the process of meniscal degeneration based on histological observations of samples obtained from routine surgeries of knee joints.

Materials and Methods: Unnecessary menisci to be discarded were collected from 20 subjects who underwent knee joint surgery (total knee replacement in most of the cases). Where possible, the interior and exterior menisci were excised as a whole during the total knee replacement surgery. In cases of meniscal injuries, menisci were collected from meniscotomy subjects for whom meniscal suture was not indicated. Subjects were given a full explanation before the surgery that the removed menisci would be discarded during or after the surgery and informed consents were obtained.

For the histological investigation, the immunohistological technique was used. Collected samples were prepared as paraffin sections and stained with HE, Alcian blue and immunohistochemical staining by the LSAB+HRP (DakoCytomation) method using anti-collagen type I, type II and type III antibodies.

Results: Menisci with no grossly observed injury

Collagen types 1 and 2 were distributed on the entire meniscus except for the circulating area.

In addition, they tended to appear intensively on the arcuate longitudinal fibers.

Expression of collagen type 3 was intensively observed on the exterior peripheral border and surface, and along the vessels in the circulating area of the meniscus. Collagen type 3 is expressed avoiding the area with collagen type 2 expression distributed inside the meniscus.

Menisci with grossly observed injury

Tear formation and fibrillation were observed, and reduced expression of collagen type 1 was observed at the corresponding injury site.

At the site with relatively minor injury, up-regulation of collagen type 2 was observed as reparation; however, collagen types 2 and 3 tended to disappear at the site with severe injury.

Degenerative/reparative areas of menisci

Disappearance of collagen types 2 and 3 was noted in the hyalinized area.

Discussion: Basic tissue changes in meniscal injury can be divided into rupture, degeneration and reparation, and it is known that these exist in combination. Rupture is a disruption of the bundle, and degeneration is expressed as a decrease in cell density, cell loss, obscure bundle or necrosis. Reparation includes cell coverage on the tissue surface, granulative change accompanied with neoangiogenesis at the injury site, cell proliferation in the noncirculating area, and cartilaginous metaplasia of proliferated cells. These tissue changes have been observed in histological examinations after HE staining, however, the variations in collagen expression accompanied with each tissue change still remain unclear. We therefore conducted an immunohistological investigation of meniscal tissues obtained during surgery in order to observe these conditions. In these observations, acid mucopolysaccharide and collagen types 1, 2 and 3 existed in a good balance in the meniscus with no grossly observed injury, and the meniscal function was found to be maintained based on this balance.

Even in the meniscus with injury, the injury was repaired by an up-regulation and the meniscal function was maintained if the degree of injury was mild; however, the meniscal tissue tended to become destroyed and disappeared if the degree of injury exceeded the limits of reparation.

When observing the variations in collagen expression accompanied with the tissue changes observed after HE staining, collagen types 2 and 3 disappeared in the hyalinized area, and myxoid degeneration and necrosis were found to be caused by the disappearance of collagen types 1, 2 and 3.


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