Mediopatellar Plica as a Risk Factor for Knee Osteoarthritis?

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Introduction

Knee osteoarthritis (KOA) has many risk factors, such as overweight, old age, and female gender,[1,2] and patients with KOA are generally asked to lose weight as well as have moderate exercise.[3-5] However, up to now, it is still unclear what causes the pathological conversion from nondisease to disease. And there still remains many KOA features that without proper explanation. For example, why KOA is more common in black and Chinese people than in white people?[6] Except for the racial and ethnic differences, the former seems to eat less and do more manual labor than the latter. Furthermore, since mechanical malalignment is believed to be the most potent factor in KOA progression,[7] why some pathological changes (such as worn articular cartilage, osteophytes, bony sclerosis and cysts) happened at the edge of the bone, which are supposed to be seldom influenced by the load? And patellofemoral joint compartments, most commonly affected by KOA is an unload-bearing joint.[6,8] There may exist some factor that act on the cartilage surface and cause these changes.

We take mediopatellar plica as a KOA risk factor is mainly based on our two direct clinical observations: One is that many old patients with KOA underwent total knee arthroplasty were found to have mediopatellar plica, these patients were all diagnosed with the patellofemoral osteoarthritis, the most common subtype of KOA. Considering its intimate anatomic relationship with patellofemoral joint, we believe that it stands a chance to be associated with the occurrence of patellofemoral osteoarthritis. However, the correlation of how many KOA patients have mediopatellar plica is still unknown for KOA patients are mainly in an old age, and they are incapable of the arthroscopic technique for the narrow joint space to do such survey.

The other one is that some patients with severe synovial plica syndrome underwent arthroscopic treatment were noticed that the mediopatellar plica as a band or strap to insert into the articular cartilage surface and causes grooves for repeatedly friction. We believe this microscopic observation is not that rare for many other arthroscopy doctors. Though arthroscopic surgery of the knee is found to provide no additional benefit to KOA,[9-11] but the surgery is indeed valuable for synovial plica syndrome at least to reduce the articular cartilage degeneration. Christoforakis reported in 1000 consecutive knee arthroscopies, that an increased incidence of articular lesions was found in patients with synovial shelves, in comparison with patients without shelves (94.7% vs. 81% respectively).[12] Moreover, a histomorphological study from Lyu, reported that patients with severe medial compartment osteoarthritis had 100% prevalence of the mediopatellar plica,[13] which implies a close relationship between this structure and KOA.

Someone may wonder that since the plica usually keep silent and don't cause clinical symptoms in normal condition, so why should we take caution of it? Here what we emphasize is that such plica may really be able to lead to the occurrence of KOA while the knee meets with trauma or chronic irritation. In that way, we cannot ignore its possible effect. Once the plica becomes the "plug", it may cause similar KOA clinical symptoms, such as anterior knee pain, snapping, giving way, and flexion and extension restriction. And the disturbance to the intraarticular environment may involves both biomechanical and biochemical effect, which could cause the occurrence and progression of KOA.

Early changes of mediopatellar plica are usually asymptomatic, but this does not mean lesions on the articular cartilage have not yet initiated. Besides, differential diagnosis of plica syndrome is sometimes hard with other intra-articular disorders. Not all characters suffering an uncomfortable knee joint would demand for medical treatment timely, and not all doctors would take account of plica disease if the symptoms are relatively light as well. In general, the patients may firstly turn to the pain relief drugs, whereas this cannot completely terminate the damage to the joint. Moreover, the plica might be worn out by repeated abrasion and decrease in size with

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the patient's age increases, and when the patient advances into the end stage of KOA and needs a total knee arthroplasty, it may disappear and cannot be observed at all.

To test this hypothesis, a controlled trial is requested to observe whether plica excision could prevent the KOA occurrence or progression in patients with synovial plica syndrome. This study should last for many years and is not so easy to test for us at present, for we believe this is a long pathological process, and patients in the placebo group receiving only physical therapy may not agree with this study in our country. In addition, some *in vitro* studies such as biomechanical experiments for simulation of such joint movement, and biochemical analysis of the plica are both useful to confirm the definite role of this tissue in KOA pathogenesis, if our viewpoint attracts some scientists' interest in the future.

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