Editorial

Response to "Degenerative changes" by van Meer *et al.*

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Comment on: van Meer BL, Oei EH, Meuffels DE, *et al.* Degenerative Changes in the Knee 2 Years After Anterior Cruciate Ligament Rupture and Related Risk Factors: A Prospective Observational Follow-up Study. Am J Sports Med 2016;44:1524-33.

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"*I don't deserve this award, but I have arthritis and I don't deserve that either.*"—Jack Benny

Anterior cruciate ligament (ACL) injuries are common, and some (but not all) individuals develop degenerative joint disease (DJD) after tearing their ACL, with or without reconstructive surgery. It would be beneficial to identify those at highest risk of post-ACL DJD, so we could study them more intensively and possibly intervene to delay or prevent the onset of DJD. In "Degenerative changes in the knee 2 years after anterior cruciate ligament rupture and related risk factors: a prospective observational follow-up study" published in the March 2016 *American Journal of Sports Medicine*, van Meer *et al.* sought to identify early degenerative changes as assessed on magnetic resonance imaging (MRI) after 2-year follow-up in patients with a recent ACL rupture and to evaluate which factors were related to those changes (1). In the authors' words, "*To change the course of OA in patients after an ACL rupture, we need to identify those patients at risk for OA development and subsequently either develop a treatment strategy or intervene in the progress of degenerative changes in the early stage.*" They followed a cohort of 143 patients, age 18 to 45, who were treated by Dutch guidelines on ACL injury, with 50 non-operative patients and the remainder receiving surgical intervention. They focused on the appearance and/or progression of MRI changes, including chondral defects, bone marrow lesions (BML), meniscal tears, joint effusions, and osteophytes between the injury, 1, and 2-year follow-up.

The 2-year MRI data showed progression of osteophytes and/or cartilage defects in 57 of the 143 total patients, or 39.9%. A multivariate analysis demonstrated that in the medial tibiofemoral compartment, the presence of a cartilage defect at baseline, presence of BML 1 year after injury, and presence of medial meniscal tear were related to the emergence or progression of cartilage defects/osteophytes. In the lateral tibiofemoral joint, male sex and the presence of lateral meniscal tear were related to the same osteoarthritic markers. An effusion 1 year after ACL rupture was also related to cartilage and osteophyte changes.

The use of MRI to find and follow the progression of macroscopic cartilage degenerative defects as little as 2 years after ACL rupture/reconstruction has been employed previously (2). Much like the study of this discussion, a correlate was found between cartilage changes and meniscal/BML lesions. In terms of concomitant meniscal injuries associated with ACL tears, an association with osteoarthritic changes at 10+ years of follow-up has been found a number of times in the last 5 years (3-5). The fact that van Meer *et al.* did not find an osteoarthritic relationship at 2 years between partial meniscectomy with ACL reconstruction is probably due to the short time interval (average 21 months) between the meniscal injury and final MRI. Other studies have shown a relationship, as previously mentioned (6).

Meniscal loss leads to 50–200% increases in medial contact pressure in meniscectomized versus normal knees (7,8). Previous clinical studies have reported that the amount of meniscus removed during surgery is directly related to postoperative radiographic progression of osteoarthritis (9-11). Investigators have also found that patients treated in a non-delayed fashion with ACL reconstruction do better than those who are either reconstructed in a delayed fashion (6–12 months depending on study) or treated non-operatively in terms of secondary meniscal tears or

progression of the original meniscal tear (12). Finally, other literature supports the repair of potentially repairable meniscal tears over meniscectomy, despite the higher reoperation rate, especially in younger patients (13,14).

The second factor discussed were the effects of BML in the emergence or progression of chondral defects and/or osteophytes. Roughly 1/3 of patients with BMLs resolved the initial lesion yet developed a new lesion, an occurrence that also increased the risk of osteoarthritis. Since the vast majority of these lesions occur in the lateral compartment, it is understandable that this compartment runs a higher risk of arthritic progression after an ACL injury. A study from our group has found that most lesions associated with traumatic ACL rupture occur in the lateral femoral condyle and posterolateral tibial plateau and are associated with lateral meniscal tears (15). We also found that post-ACL BMLs were more common in men. The majority of these traumatic lesions abate between 6 months and 2 years (16); however, 10 of the 27 original patients with BMLs in van Meer *et al.* developed new lesions at 2 years. This coincided with the data of Frobell, who noted that 1/3 of patients with acute BMLs developed new BMLs after 2 years (17).

While van Meer *et al.* note they only saw significant osteoarthritic changes in the medial tibiofemoral compartment, it has been previously reported that the presence of BMLs 1 year after injury was a strong predictor for new onset of progression of osteoarthritic changes (18). Although there was not a correlation between patients with meniscal tears and BMLs, one would expect that these patients would have an even higher incidence of chondral damage and osteophyte formation.

The presence of a persistent effusion 1 year after ACL rupture had a significant impact on the progression of osteophytes in the tibiofemoral compartment. Similar degenerative changes were noted in previous studies (19) and the changes can be attributed to the persistent inflammatory milieu present in the injured knees. Male sex was associated with progression of degenerative defects only in the lateral compartment, although other data suggest women are more prone to osteoarthritis after ACL injury (20).

Surprisingly, topics previously attributed to be causes of osteoarthritic changes, especially at later time points, such as ACL reconstruction *vs.* non-operative treatment (21), age, and BMI (22), were not found to correlate in the van Meer article. This is probably due to the short follow-up period, since factors like age and BMI may take 10+ years to erode the cartilage and produce osteophytes.

Van Meer noted lateral compartment changes more often than medial and attributed this variation to the mechanism of ACL rupture. It is well documented that lateral injuries predominate in the acute period (15) and it would be understandable that these would predispose this compartment to osteoarthritic changes at 2 years. It is also well documented that knee kinematics change after ACL injury and can cause increased pressure and variation in wear characteristics across the lateral compartment (23).

The 2-year follow-up period means we will have to wait to see if the patients with MRI changes actually develop symptomatic DJD, and we would encourage continued follow-up of this group. The challenge will be to separate the effect of other intervening factors in such a small set of subjects, such as new injuries, differences in activity levels (which may have a small effect over 2 years but a large effect over the decades that it may take to develop significant DJD), and changes in BMI that occur over time, just to mention a few. In the meantime, van Meer *et al.* have provided us with risk factors that can help identify patients that may benefit from more intense scrutiny and/or early intervention in the first 2 years following an ACL injury.

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Footnote

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