# Illustrated Medicine: Post-traumatic osteoarthritis (part 2)

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***Due to the prevalence of poor outcomes from injury to joints of the body, personal injury lawyers can benefit from an awareness of the compromised ability of cartilage to heal itself and the science of osteoarthritic deterioration manifested in this type of injury. This is an important consideration when discussing future needs and cost of care in a PI file. This paper continues the discussion begun in Part 1, examining the development and implications of post-traumatic osteoarthritis development.***

## Types of joint injuries

Joint injuries involving **impact loading**, associated **soft tissue injuries** (to ligaments, menisci, etc.), **dislocations** and notably **intra-articular fractures** can lead to post-traumatic osteoarthritis (PTOA) – a significant medical-legal and societal concern. Research in 2006 indicated that 5.6 million people in the US were affected by PTOA severe enough to cause them to consult with a joint-replacement surgeon, and that PTOA patients rack up medical costs of $USD 3.06 billion annually (Brown, et al, 2006). People may manifest different symptoms and radiological findings, but common amongst all sufferers is pain, structural damage to the joint cartilage, and movement limitations (Aigner, Schmitz, 2013).

## Features of degenerated joints

As PTOA advances: cartilage that is normally capable of sustaining repetitive loading breaks down; subchondral bone becomes thickened and denser; bone marrow lesions and cysts may form; osteophytes grow; and the synovium (cells lining the joint capsule) and joint capsule are altered. The collagen network then becomes stiffer, making it more rigid and less able to undergo normal physiological deformation, and becomes prone to micro fracture. The subchondral bone plate may become visible or "eburnated" (smooth ivory-like surface). At an advanced stage of degeneration, cartilage nodules or "tufts" may be found on the joint surface or within the bone marrow.

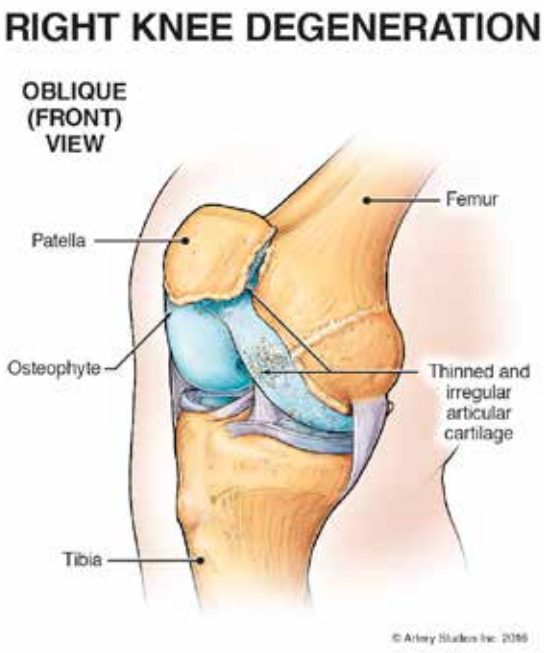


***Figure 5: Illustration of a knee with PTOA degeneration, including in the patellofemoral joint – the articulation of the kneecap with the femur.***

## Breakdown of cartilage

The articular cartilage in an injured joint is exposed to continuous mechanical wear and tear including stresses from shearing, stretching and hydrostatic pressure. Initial degeneration characteristics include softened, swollen or roughened cartilage as the collagen matrix absorbs water and becomes unstable. Microscopically, fissures may be seen in the matrix, followed by visible tears.

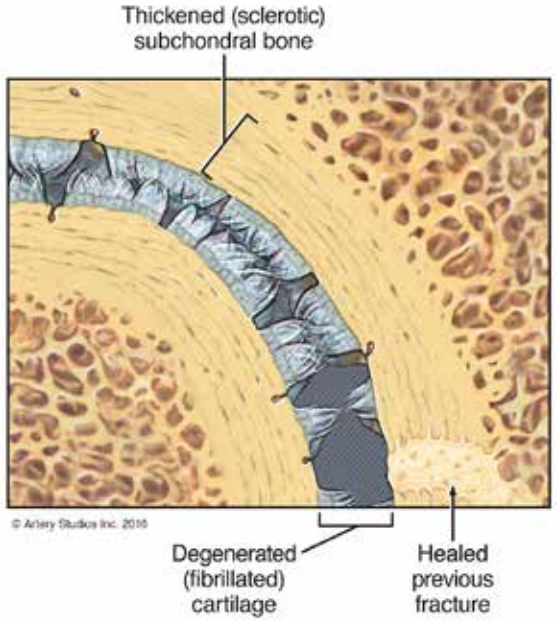


***Figure 6: Detailed view of deteriorating cartilage adjacent to a previous intra-articular fracture.***

## Subchondral bone

The subchondral bone may become a source of joint pain in PTOA. As a result of mechanical and physiological changes, it remodels and undergoes thickening and becomes denser (sclerosis) as does the adjacent bony trabeculae. With advanced cartilage destruction, some radiographs demonstrate patches of bone necrosis. Since this occurs adjacent to areas of bare bone, it is believed that synovial fluid gains access to the bone marrow through these areas to form cysts.

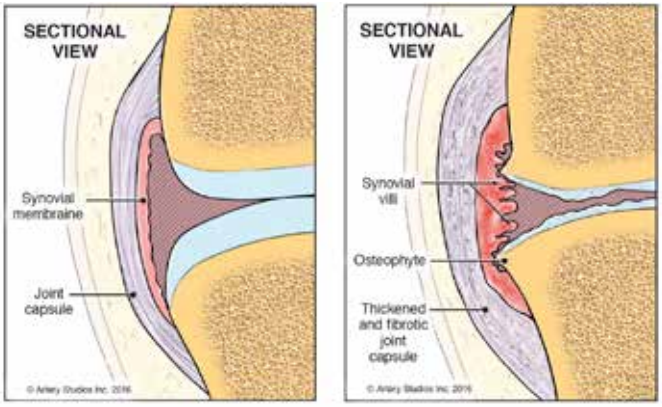


***Figure 7: Characteristic features of PTOA include inflammation and associated thickening of the synovium as well as fibrosis of the joint capsule.***

## Synovial membrane/capsule

The synovial capsule and surrounding ligaments provide stability for a joint and facilitate flexibility and motion. Cells lining the synovial membrane (synoviocytes) are normally only a single layer thick, but proliferate with advancing OA. The synovium becomes inflamed in reaction to the release of cartilage debris from the damaged joint surface, and may be an additional source of pain. In the later stages of PTOA, the entire synovial surface can become significantly thickened, forming infoldings (synovial villi) due to the redundant tissue. Capsular fibrosis – shortening and thickening of the joint capsule – may also develop, resulting in further joint stiffness.

## Osteophytes

Frequently osteo-cartilaginous outgrowths appear at the margins of the damaged joint to form ossified plate-like structures. These were originally believed to be a repair response to the altered biomechanical loading due to adjacent cartilage deterioration, thereby providing additional weight-bearing support, however many are found in non-weight-bearing areas. It is now believed that they are produced in response to the release of growth factors in the deteriorating joint environment.

## Treatment

Obesity contributes to the development of OA in the knee but less so for other joints. This is believed to be due to increased loading of the injured joint, but also has to do with metabolic factors associated with obesity that act on the chondrocytes in the cartilage matrix. Hence, weight loss is often recommended.

Research continues in the area of surgical treatments including joint cartilage replacement (e.g., mosaicplasty) and drilling into the subchondral bone to initiate fracture repair response (despite the fact that the cartilage that reforms is of inferior quality). Joint replacement with prosthetic components still remains the gold standard for treating advanced PTOA.

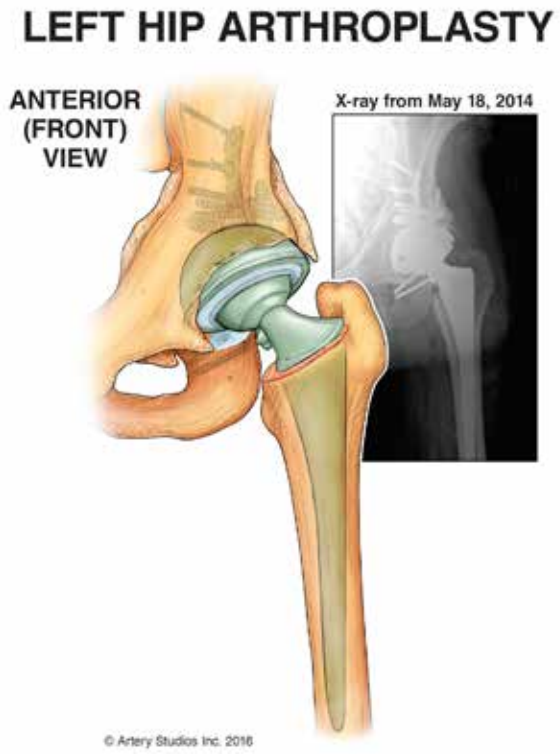


***Figure 8: Total joint replacement ("arthrodesis") is a frequent surgical outcome from degeneration of intra-articular fractures of the hip joint.***

## Conclusion

Issues of deterioration of injured joints with post-traumatic osteoarthritis are an important consideration in presenting medical-legal cases. A seemingly small injury to cartilage may become a significant functional limitation over time. Healing in cartilage is limited and deterioration and the development of PTOA is highly likely. It is therefore imperative that consideration of these long-term sequelae be presented when arguing damages in personal injury cases to ensure the full magnitude of the injury is appreciated.

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## References

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**Thomas Aigner and Nicole Schmitz, May 28, 2013, *Pathogenesis and pathology of osteoarthritis; SECTION 13 OSTEOARTHRITIS AND RELATED DISORDERS.* https://www.med.unc.edu/tarc/events/event-files/Hichberg%20text.%20OA%20path..pdf (accessed Apr. 14, 2016).**