Illustrated Medicine
Post-traumatic osteoarthritis (part 1)
Injury to synovial joints
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Relevance to PI litigation
At Artery Studios we deal with hundreds of personal injury cases a year. Many of these involve injury to joints, leading to osteoarthritis (OA). This first paper in a two part series examines the anatomy and physiology of joints and explains why what may appear to be only a minor injury to cartilage can lead to long-term complications in the form of post-traumatic OA. Personal injury lawyers should be aware of the slow/weak healing response of cartilage – especially invisible damage to it – to fully appreciate and communicate the long-term future consequences of a client’s injury in arguing for appropriate damages.

Anatomy of joints
Synovial joints such as the knee, hip or elbow are formed by four key anatomical structures: articular cartilage (“chondro”) that caps the ends of the bones, a surrounding tough joint capsule, the synovial membrane lining the inside of the capsule, and the lubricating synovial fluid that is produced by this membrane.

Figure 1: Schematic depiction of a synovial joint.

Anatomy of cartilage
Articular cartilage, along with its lubricating synovial fluid, facilitates the smooth gliding motion of a joint and absorbs weight-bearing stresses. It is a unique type of tissue in that it has neither nerve nor blood supply (avascular). Also known as hyaline cartilage, it is formed by chondrocytes – cells that encase themselves with a strong gel-like cartilage matrix that is secreted to form a supporting scaffold. The matrix contains collagen fibrils, which can withstand significant tensile forces. It is composed of 65-80% tissue fluid (water) which allows for the diffusion of nutrients from the synovial fluid to the sealed off chondrocytes.

Figure 2: Microscopic view of articular cartilage. Collagen fibrils in the superficial zone arch over one another to form a smooth surface. The deepest layer of cartilage is interlocked with the supporting subchondral bone.
Injury to cartilage

For healthy articular cartilage to be disrupted, an impact with significant force is required, as the cartilage and underlying bone are adept at absorbing and distributing loads from normal activity. Buckwalter (2004) notes three classifications of cartilage injury:

Type 1 (visibly intact cartilage): Microscopic damage to the cells, matrix or subchondral bone with no visible injury to the joint.

Type 2 (chondral fractures): Cartilage fissures, tears or other defects that may be evident on CT or MRI.

Type 3 (osteochondral fractures): Disruption of the articular surface and underlying bone, often readily demonstrated by radiological imaging.

Osteochondral fracture response

Although they produce matrix throughout their lifespan, chondrocytes respond poorly to injury to the cartilage in which they’re embedded. When only the cartilage is injured, without damage to the underlying subchondral bone (Type 1 or 2), the chondrocytes react with a limited response with only a brief repair phase. Further, some may die from direct injury. The matrix may then become stiffer with distortion or disruption of the remaining collagen fibrils, resulting in increased mechanical stresses on them. If the exposed activated chondrocytes cannot adequately respond to the cartilage defect in time, a permanent defect may remain, altering joint mechanics and leading to further degeneration. With Type 3 fractures involving the subchondral bone, a different repair response is triggered when cells from the bone marrow migrate into the cartilage cleft to form new connective tissue. However, the cartilage and underlying bone are rarely restored to their original healthy configuration. The replacement cartilage does not have the functional properties of the original hyaline cartilage and is prone to early degeneration, particularly if the trauma causes any differences in the elevation of cartilage surfaces such that joint incongruity increases stresses on these surfaces. This is particularly relevant to PI cases where radiological imaging demonstrates step deformities, post-operative fracture clefts or non-united fractures, prognosticating future OA outcomes.

Next issue

The details of osteoarthritic degeneration of injured cartilage will be explored in Part 2.


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