

Acute impaction injury patterns range from isolated chondral destruction, isolated subchondral bone bruises and/or fractures, to any combination of these injuries. The degree of bony penetration and trabecular disruption correlates directly with the amount of energy transfer and the area over which the impulse is inflicted across the joint surface. Edema associated with energy dispersal in bone generally assumes a hemispherical geometry with the point of impact being demarcated by the most intense edema. By the time imaging is obtained, there is generally a second pattern of vasogenic edema that surrounds the dilated capsular perforator vessels that supply the injured bone. This pattern is perivascular in distribution and therefore follows a vascular territory that in the femur and tibia is wedge-like in morphology. With subchondral trabecular fracturing, there is a discrete low-signal zone marking the compression of the trabecula and exclusion of marrow fat. **Figures 71-1A to 71-1D** show a typical acute osteochondral impaction fracture involving the anterolateral tibial plateau. **Figure 71-1A** is an axial proton density image with fat saturation in which the arrowheads outline the hemisphere of impaction edema or bruising deep to the zone of trabecular impaction. The arrow shows the perivascular accumulation of vasogenic edema caused by dilatation of an adjacent intraosseous arteriole. **Figure 71-1B** is a coronal T1 image illustrating the usefulness of T1 imaging in displaying the zone of subchondral trabecular impaction. Arrowheads mark the edges of the fracture. The thin cartilage of the anterior tibia is not as well seen as with proton density imaging. **Figure 71-1C** is a sagittal proton density image where the cartilage/subchondral plate interface is much better appreciated. A subtle depression of the subchondral plate is visible (arrowhead) despite no apparent fissuring of the overlying cartilage. In **Figure 71-1C**, there are concentric low-signal linear arcs (arrows) within the trabecular bone far below the zone of obvious trabecular compression that mark the limits of the subchondral fragment illustrated in **Fig. 71-1B**. This may reflect lesser degrees of trabecular injury/fracture and attests to the great amount of energy transmitted into the tibia as the result of its being directly hit by a fellow football player's helmet during a tackle. **Figure 71-1D** is a coronal T2 image with fat saturation that was obtained just posterior to the impaction fracture. The arrow indicates a dilated tibial perforator vessel while the arrowheads show nondilated normal-appearing tibial and femoral perforators. Note the irregular morphology of the deeper perivascular edema on the T2 image. There is, however, lack of trabecular detail on the heavily T2-weighted image.

With uncomplicated healing, this low-signal zone remodels into a normal trabecular pattern. In cases where the subchondral bone goes on to infarction, this subchondral fragment may resorb and get replaced with scar tissue or form subchondral cysts/geodes. In some instances, the fragment may stabilize and act as an in situ graft that results in focal hypertrophy of the subchondral bone. These chronic injuries can be recognized by their rounded incongruent margins that tend to protrude beyond the normal contour of the subchondral plate. They are frequently associated with fibrosis or absence of the overlying cartilage and may also have internal or marginal cystic degeneration. As a result of degeneration/arthritis, chronic lesions may

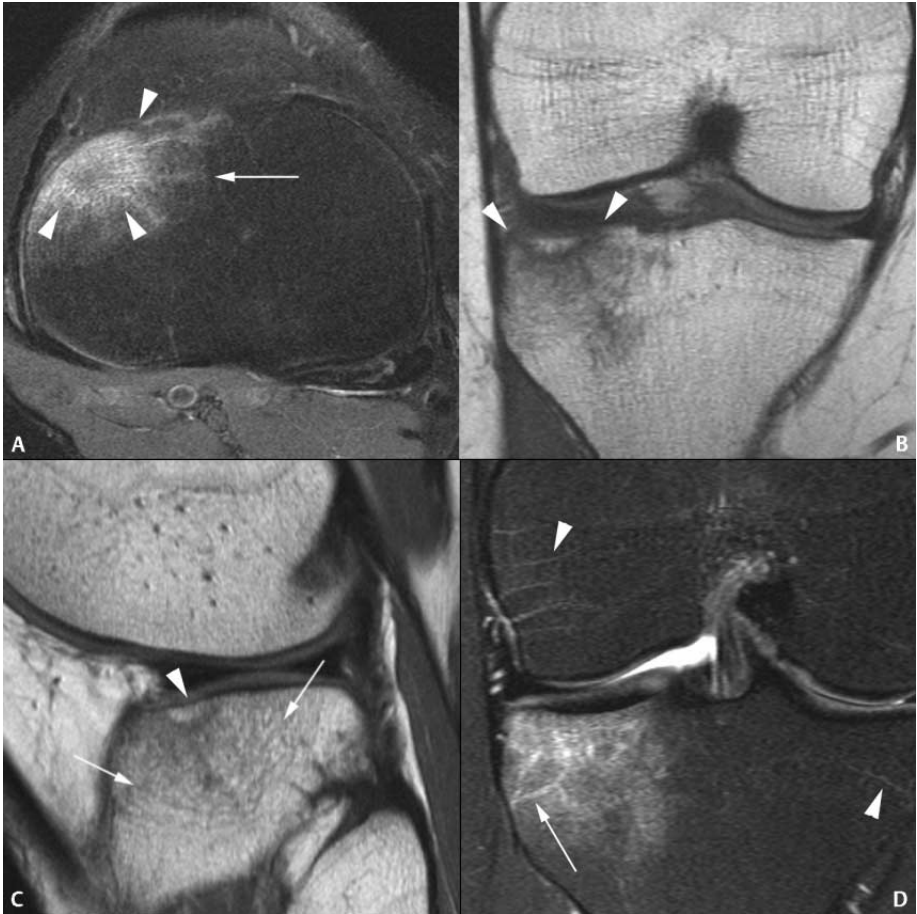


Figure 71-1

remain hyperemic and edematous. **Figure 71-2A** is a sagittal proton density image of a healing osteochondritis dissecans (OCD) lesion in the medial femoral condyle of a skeletally immature male with multiple lesions. Note the rounded and protuberant contour of the subchondral plate along the leading edge (white arrow) and fibrosis in the posterior edge (arrowhead). The black arrow indicates a smaller adjacent tibial lesion. **Figure 71-2B** is a coronal T2 image with fat saturation through the same lesion. The small arrow indicates the zone of demarcation between the donor site and the remodeling trabecular impaction zone. Increased fluid signal in the fragment and within the adjacent bone (large arrow) reflects invading granulation tissue and potential reincorporation of the fragment. Note vasodilatation and edema along the capsular perforator vessels of the femoral condyle (arrowheads). **Figure 71-2C** is a coronal T2 with fat saturation through the lateral femoral condyle of the same knee. The arrow shows sclerosis in this OCD lesion with a less T2-intense halo (arrowhead) deep to this nearly healed smaller and shallower defect. **Figure 71-2D** is a sagittal proton density



Figure 71-2

image in a different patient that shows a completely healed lesion that is particularly hypertrophic and protuberant. The black arrow shows the remnants of the trabecular impaction zone. The white arrow indicates the absence of cartilage that normally separates the surface of the condyle from the adjacent meniscus. Note the change in signal intensity within fibrocartilage/scar tissue deposited between the posterior edge of the lesion and the remaining posterior condylar cartilage (arrowhead). The termination of the posterior condylar cartilage tangential zone is visible at this interface.