85. Avascular Necrosis of the Hip

Pelvic and hip MRI is typically obtained utilizing a large surface coil, enabling simultaneous bilateral imaging. Unilateral imaging with a smaller surface coil, however may allow for increased SNR and spatial resolution. The detection and treatment of early avascular necrosis (AVN) of the femoral head—the most common location for this condition in the body—is the primary indication for hip MRI. SI characteristics of femoral head AVN correspond well with underlying pathology: the commonly described double line sign within the femoral head refers to the appearance of a hyperintense line immediately adjacent to linear hypointense signal on T2 or PDWI. The former line correlates pathologically with granulation tissue and the latter with reactive sclerotic bone and fibrosis. Together the double line represents the interface between normal and necrotic marrow. The Mitchell system classifies the MRI progression of femoral head ischemia. Early, lesions (Class A) demonstrate fat-like SI as shown in the left femoral head of Figure 85.1A,B. Here, (A) T1WI demonstrates isointensity of the lesion to normal marrow superior to a hypointense line correlating with a nidus of fibrovascular proliferation and demarcating pathologic from normal bone. A full double-line sign is not seen, as is the case 20% of the time. On (B) FS T2WI, the linear border demonstrates high SI, although the SI of the superomedial area of necrosis remains low (i.e. that of attenuated fat). The appearance of early AVN may, in fact, be indistinguishable from other causes of bone marrow edema, and CE T1WI may be necessary to differentiate ischemia from completely devitalized bone. In advanced osteonecrosis, CE examinations differentiate viable (although possibly ischemic), enhancing bone from necrotic, nonenhancing bone. Class B lesions are
rarer and demonstrate SI changes compatible with late subacute hemorrhage (high SI on T1 and T2WI), while a Class C lesion consists of edema-like SI within the ischemic femoral head. A Class D lesion is illustrated in the right femoral head of Figure 85.1A,B. Superomedial to the line demarcating necrotic and normal bone, hypointensity is seen on both (A) T1WI and (B) FS T2WI, correlating with chronic fibrosis. Important ancillary findings in this patient include the presence of small bilateral effusions within the hip joints (right greater than left) demonstrating typical edema-like SI. As illustrated here, AVN occurs bilaterally in the majority of cases. PDWI of Figure 85.2A,B demonstrate two additional cases of AVN. In both, hypointensity within the superior aspect of the femoral head denotes either a Class C or D lesion. In the (B) second image, marrow intensity is similar, but partial femoral head collapse is noted—a condition predisposing to chondral fracture, chondromalacia, and chronic osteoarthritis. Bone remodelling and collapse may be better assessed and classified on CT. Transient osteoporosis of the hip may mimic early femoral head AVN, but is distinguishable clinically by its rapid onset and a lack of AVN risk factors and on MRI by the presence of diffuse edema throughout the femoral head without a clearly differentiable area of subchondral osteonecrosis. Some believe that transient osteoporosis (transient marrow edema syndrome) may represent salvaged AVN. In the pediatric patient (typically between 4 and 9 years old), MRI is utilized for evaluation of idiopathic AVN of the femoral head epiphysis, known as Legg-Calves-Perthes disease. The earliest MRI finding of this condition may simply consist of a joint effusion—the presence of which in an appropriately-aged child with high clinical risk for the condition warrants MRI followup. T1WI typically reveals diminished SI of the epiphysis, whereas SI on T2WI is variable. Non-enhancing regions of the femoral head often correlate with necrotic, nonviable tissue. Group 1 lesions involve only the anterior epiphysis, while later, progressive changes include physeal bridging or compression with subchondral fissure
formation (Group 2), diffuse metaphyseal involvement (Group 3), and eventually epiphyseal collapse (Group 4). Obese children are predisposed to a Salter Harris Type 1 fracture of the femoral head known as a slipped femoral capital epiphysis and demonstrated on the coronal PDWI of Figure 85.3. Here, as is typical, physeal widening with inferomedial epiphyseal displacement is present. Hyperintense signal compatible with edema was seen within the proximal metaphysis on STIR images (not shown). Sequelae of slipped capital femoral epiphysis include femoral head AVN, premature physeal fusion, and osteoarthritis.

![Fig. 85.3](image-url)