ELSEVIER

Seminars in
ROENTGENOLOGY

Radiography of the Hip: Lines, Signs, and Patterns of Disease

Scot E. Campbell, MD

The complex anatomy of the pelvis and the often subtle but significant radiographic findings can be challenging to the radiologist. A sound understanding of the standard radiographic techniques, normal anatomy, and patterns of disease affecting the pelvis can be helpful in accurate diagnosis. This article will review the common radiographic projections in conventional radiography of the pelvis and hip and will discuss radiographic anatomy, including the various lines used to evaluate the pelvis and hip joint. Specific signs and patterns of disease will be addressed, with the goal of providing a fundamental approach to interpreting hip and pelvis radiographs.

Radiographic Technique

Commonly used radiographic projections of the pelvis and proximal femur include the anteroposterior (AP) view of the pelvis, anterior and posterior oblique (Judet) views of the pelvis, AP view of the hip, and frog-leg lateral (Dan Miller) view of the hip.[1-3] The AP radiograph of the pelvis (Fig. 1) or hip is taken with the patient supine, and both feet in approximately 15° of internal rotation. This reduces the normal 25 to 30° femoral anteversion, allowing better visualization of the femoral neck.[2] Judet views are performed with the patient in a 45° oblique position.[2,3] When the affected hip is in a posterior oblique position, the posterior column and anterior acetabular rim are well seen (Fig. 2). Conversely, with the affected hip in the anterior oblique position, the anterior column and posterior acetabular rim are well seen. The frog-leg lateral view (Fig. 3) is performed with the patient supine, feet together, and thighs maximally abducted and externally rotated.[2] The radiographic tube is angled 10 to 15° cephalad, directed just above the pubic symphysis.[2] The anterior and posterior aspects of the femoral neck, as well as the lateral aspect of the femoral head, are seen with this projection.

Additional views that may be helpful include the pelvic outlet (Ferguson) view, the pelvic inlet view, and the groin-lateral (Dan Miller) view of the hip. The Ferguson view is performed in the same position as the AP view, with the radiographic tube angled 30 to 35° cephalad, and the central beam directed at the center of the pelvis.[2] This projection allows excellent visualization of the sacroiliac joints, the pubic rami, and the posterior acetabular rim (Fig. 4). The pelvic inlet view is performed in the same position as the AP view, with 30 to 35° of caudal angulation of the radiographic tube.[2] This view allows visualization of the sacral promontory, the iliopectineal line (anterior column), the ischial spine, and the pubic symphysis (Fig. 5). The groin-lateral view of the hip is performed with the patient supine, the nonaffected leg elevated and abducted, and the affected leg extended.[2] The radiographic tube is directed horizontally toward the medial aspect of the affected hip, with 20° of cephalad angulation (Fig. 6).

Anatomy

The pelvis is composed of three bones, the ilium, ischium, and pubis, all of which contribute to the structure of the acetabulum.[2-5] The ilium is composed of a body and a large flat portion called the iliac wing.[5] The body forms, with the bodies of the ischium and pubis, the roof of the acetabulum. The arcuate line is a bony ridge projecting from the sacroiliac joint to the pubis, dividing the iliac body from the iliac wing. The superior border of the iliac wing is the iliac crest. Anteriorly, there are two projections from the ilium, the anterior superior and inferior iliac spines. Posteriorly, there are two projections from the ilium, the posterior superior and inferior iliac spines. Posteromedially, the ilium articulates with the sacrum via the sacroiliac joint. The distal one-third of the sacroiliac joint is a synovial joint, whereas the proximal two-third forms a syndesmosis.[5]

The pubis is composed of a body and two rami.[5] The pubic body fuses with the iliac and ischial bodies to form the anterior border of the acetabulum. The superior pubic rami project anteroinferiorly from the acetabuli. A linear bony

Department of Radiology and Imaging, Hospital for Special Surgery, New York, NY.

The opinions and assertions contained herein are those of the authors and should not be construed as official or as representing the opinions of the Department of the Army or the Department of Defense.

Address reprint requests to Scot E. Campbell, MD, Department of Radiology and Imaging, Hospital for Special Surgery, 535 E. 70th Street, New York, NY 10021. E-mail: scammbell@msn.com

0037-198X/05/$-see front matter © 2005 Elsevier Inc. All rights reserved.
doi:10.1053/j.ro.2005.01.016

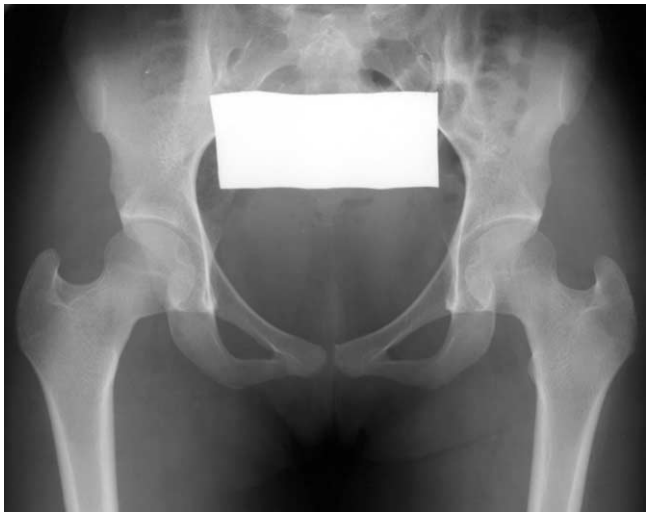


Figure 1 Anteroposterior radiograph of the pelvis.

ridge along the superomedial border of the superior pubic ramus is present, called the pecten pubis, or pectineal line. This is continuous with the arcuate line of the ilium, forming the iliopectineal line (Fig. 7), the anterior border of the "anterior column."[2,3] The iliopectineal line is an important osseous landmark to visualize on every radiograph of the hip or pelvis, as traumatic, metabolic, or neoplastic conditions affecting the anterior column of the pelvis will cause discontinuity, thickening, or an abnormal course of this line.[3,4] The inferior pubic rami project inferiorly from the medial border of the superior rami and have a symphyseal surface, which articulates with the pubic symphysis. The pubic symphysis may be widened in traumatic symphyseal diastasis or in bladder extrophy (Fig. 8).

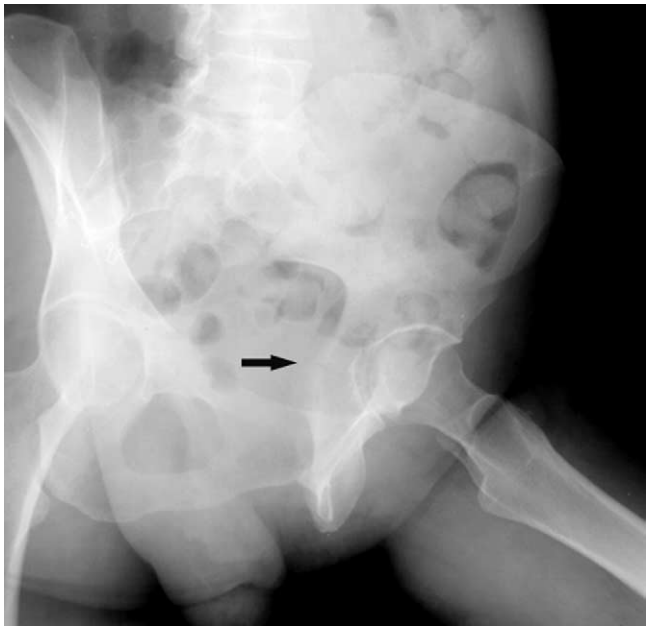


Figure 2 Forty-five-degree posterior oblique view of the left acetabulum reveals a non-displaced fracture through the posterior column.

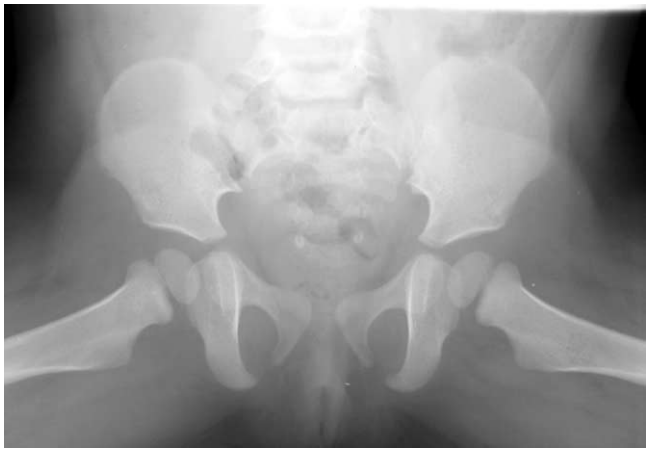


Figure 3 Frog-leg lateral radiograph of the pelvis.

The ischium is also composed of a body and two rami.[5] The body forms the posterior border of the acetabulum. At birth, the three bones contributing to formation of the acetabulum are not fused and are separated by the triradiate cartilage.[6] A posterior projection from the body of the ischium is called the ischial spine. The curved notch between the posterior inferior iliac spine and the ischial spine is the greater sciatic notch, and the notch between the ischial spine and the ischial tuberosity is the lesser sciatic notch. The superior ramus of the ischium extends inferiorly from the body to the ischial tuberosity. On the AP radiograph, a line can be drawn from the ilium to the ischial tuberosity and is called the ilioischial line.[3,4] This line is part of the "posterior column" (Fig. 7) and is also an important landmark to be visualized on every radiograph of the pelvis.[3,4] The inferior ischial ramus projects anteriorly to fuse with the inferior pubic ramus, forming the obturator foramen.

The proximal femur can be divided into the femoral head, femoral neck, trochanters, and femoral shaft.[5] The fovea is seen at the medial aspect of the femoral head. The femoral

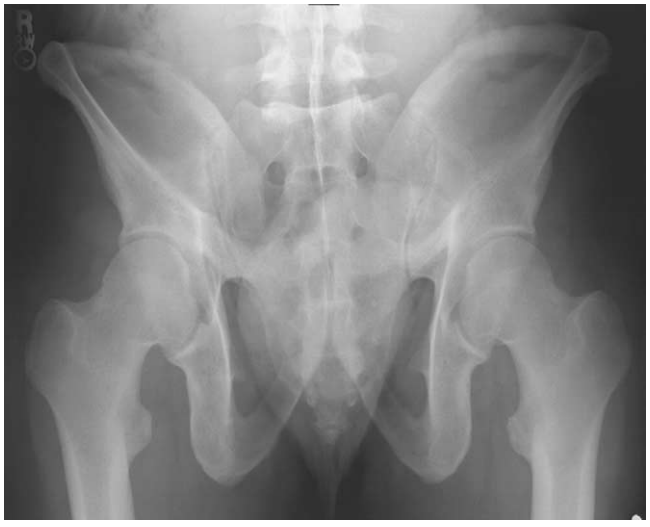


Figure 4 Pelvic outlet view allows good visualization of the sacroiliac joints and pubic rami.

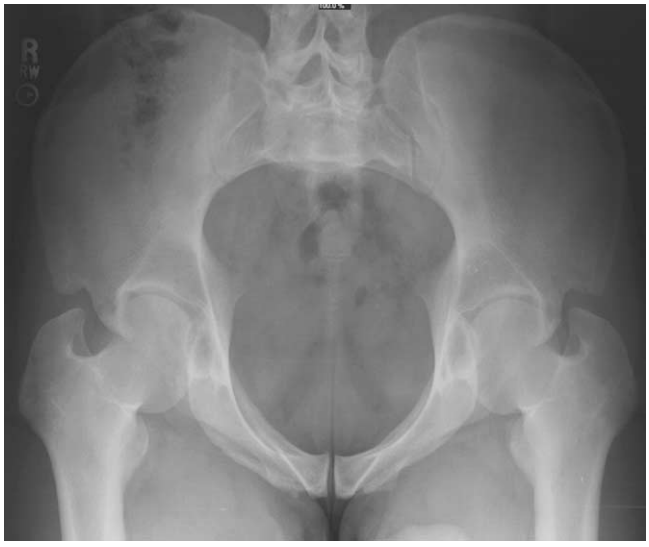


Figure 5 Pelvic inlet view reveals the sacral promontory and iliopectineal line to good advantage.

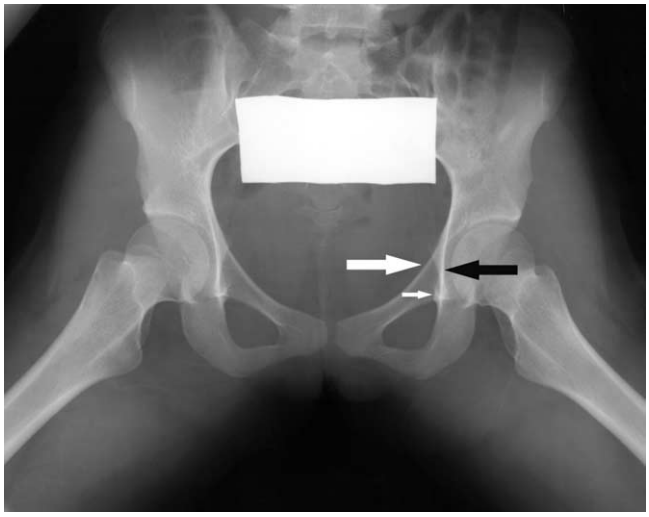


Figure 7 The iliopectineal line is part of the anterior column (large white arrow); ilioischial line is part of the posterior column (black arrow), and teardrop appearance (small white arrow).

neck can be divided into subcapital, transcervical, basicervical, intertrochanteric, and subtrochanteric portions.[2] The latter three of these are extracapsular.[2,5] The femoral head is normally angulated approximately 125 to 135° with respect to the long axis of the femoral shaft, and anteverted approximately 25 to 30°.[2,5] The major trabeculae of the proximal femur are well demonstrated on the AP radiograph.[2] Long, arc-shaped trabeculae extending from the femoral head to the intertrochanteric ridge are the principal tensile trabeculae, while the principal compressive trabeculae are more vertically oriented, coursing along the medial aspect of the femoral neck (Fig. 9).

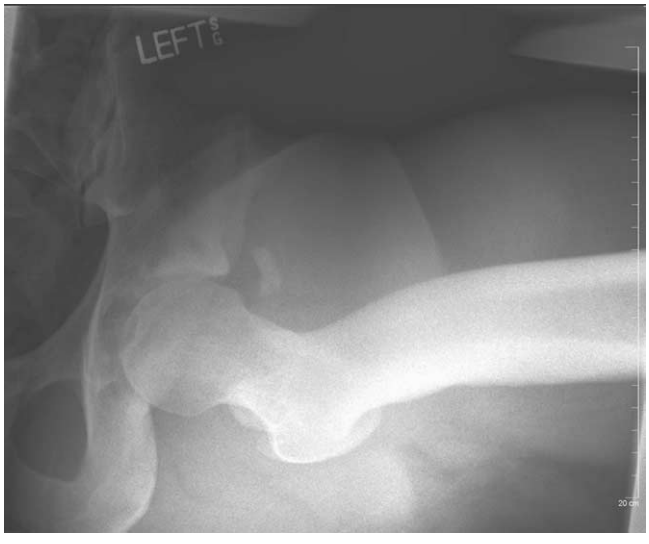


Figure 6 Long-leg lateral view of the left hip shows prominent femoral head–neck junction, narrowing of the anterosuperior joint space, and sclerosis of the anterosuperior acetabulum. Heterotopic bone formation is seen from previous hip arthroscopy for anterior labral debridement in a patient with femoroacetabular impingement.

Lines

On the standard AP view of the pelvis, the iliopectineal line (also called the iliopubic line) extends from the medial border of the iliac wing, along the superior border of the superior pubic ramus[2-4] to end at the pubic symphysis (Fig. 1). This line is seen as the inner margin of the pelvic ring and defines the anterior column of the pelvis (Fig. 7). As mentioned above, the anterior column is well demonstrated by a 45-degree anterior oblique radiograph.[3] Fractures extending through the anterior column disrupt the contour of this line (Fig. 10). In addition, this line may be thickened in patients with Paget disease[7] or in patients with familial idiopathic hyperphosphatasia.[8]

The ilioischial line also begins at the medial border of the iliac wing and extends along the medial border of the ischium[2-4] to end at the ischial tuberosity (Fig. 4). This defines the posterior column of the pelvis (Fig. 7). As mentioned above, the posterior column is well demonstrated by a 45-

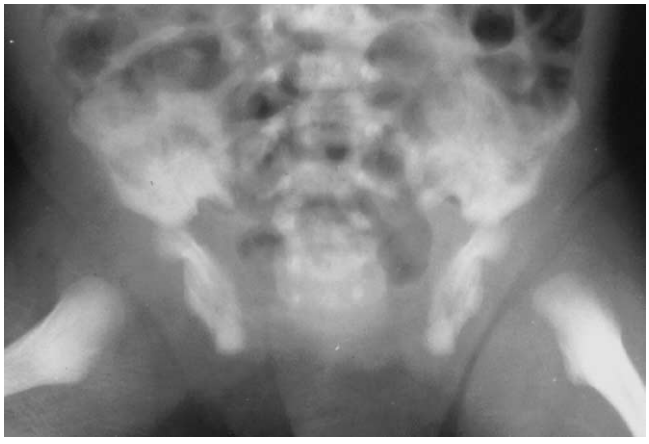


Figure 8 Marked widening of the pubic symphysis is seen in this patient with bladder exstrophy.

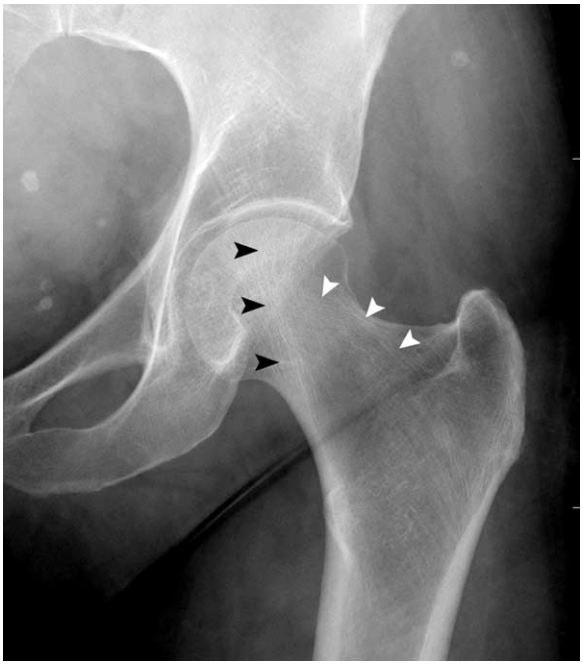


Figure 9 Anteroposterior view of the hip shows the principal compressive (black arrowheads) and principal tensile (white arrowheads) trabeculae.

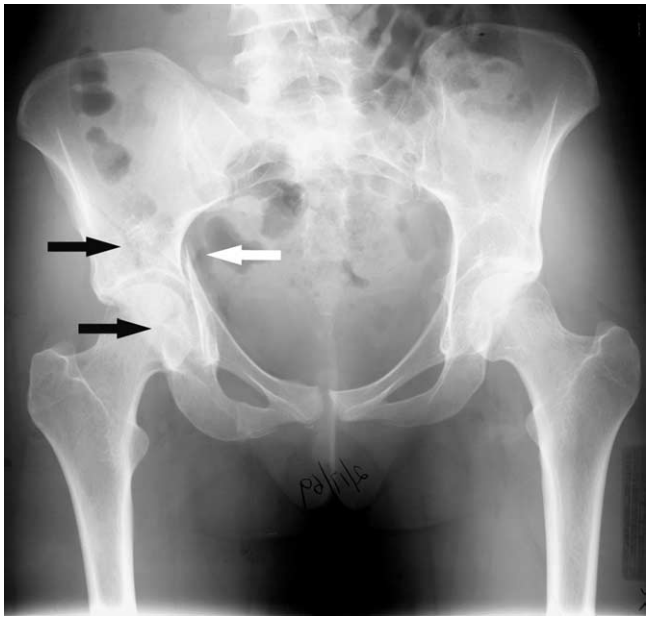


Figure 10 The iliopectineal line is disrupted (white arrow) indicating anterior column fracture. There is also a comminuted fracture through the posterior column and posterior acetabular wall (black arrow).

degree posterior oblique radiograph.[3] Fractures extending through the posterior column of the pelvis disrupt the contour of the ilioischial line (Fig. 2).

The anterior rim of the acetabulum is seen as the more medial of two obliquely oriented arc-shaped lines on the AP view[2-4] (Fig. 11). The anterior acetabular rim is seen well in profile on the 45-degree posterior oblique view[2] (Fig. 2). The posterior rim of the acetabulum is the more lateral arc-shaped line on the AP radiograph and is seen well in profile on the 45-degree anterior oblique view.[2] The teardrop represents a summation of shadows of the medial acetabular wall[9] (Fig. 7). Teardrop distance is measured from the lateral edge of the teardrop and the femoral head. Side-to-side comparison of the teardrop distance can be useful to evaluate for hip joint effusion or for hip dysplasia.[9]

Line of Kline is a line drawn along the long axis of the superior aspect of the femoral neck, which normally will intersect the epiphysis. The Shenton arc is a smooth curvilinear line connecting the medial aspect of the femoral neck with the undersurface of the superior pubic ramus. A horizontal line connecting the triradiate cartilages (Hilgenreiner line) and a perpendicular to this line through the lateral edge of the acetabulum (Perkins line) define four quadrants in which, in normal hips, the femoral head should be in the lower inner quadrant.

The sacroiliac joints are seen at an angle on the AP radiograph, resulting in some overlap of structures. The normal sacroiliac joint is a syndesmotic joint in its upper two-third and a synovial joint in its lower (anterior) one-third.[5] The lower sacroiliac joint is well seen on a pelvic outlet view.[2] A normal sacroiliac joint will have a thin white line without erosions or sclerosis (Fig. 4).[1,2] Early sacroiliitis may demonstrate erosions and apparent widening of the sacroiliac joint space.[10] Subchondral sclerosis develops due to reactive changes in the bone.[2,10]

The sacral foramina are symmetric foramina with thin, well-defined rims. Disruption or irregularity of the sacral foramina may be a subtle clue to traumatic or insufficiency fractures of the sacrum. Inability to visualize the thin rims of the sacral foramina may be a clue to the presence of a lytic mass or erosive process in the sacrum (Fig. 12).

Several osseous projections of the pelvis and hip serve as tendon attachments and can be avulsed with tendon injuries.[5,11-13] The external abdominal oblique muscles insert on

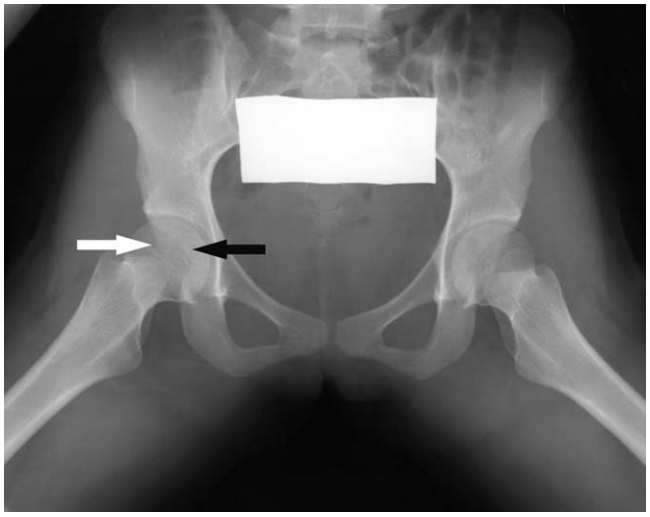


Figure 11 The anterior (black arrow) and posterior (white arrow) walls of the acetabulum.

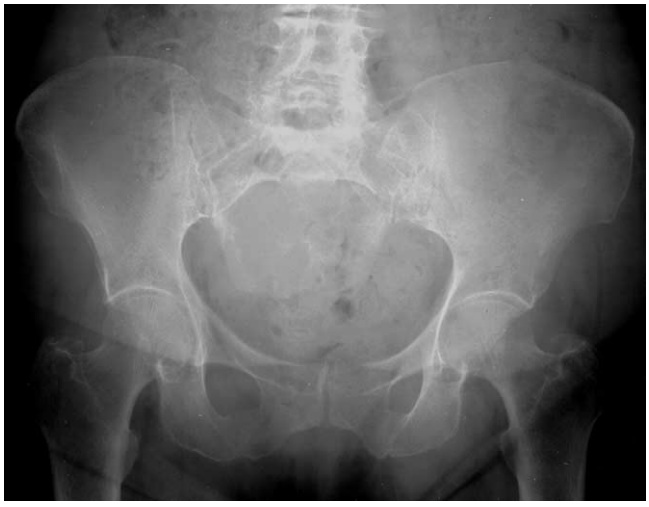


Figure 12 The sacrum is destroyed by a lytic mass (chordoma). Note the absence of the sacral foraminal lines.

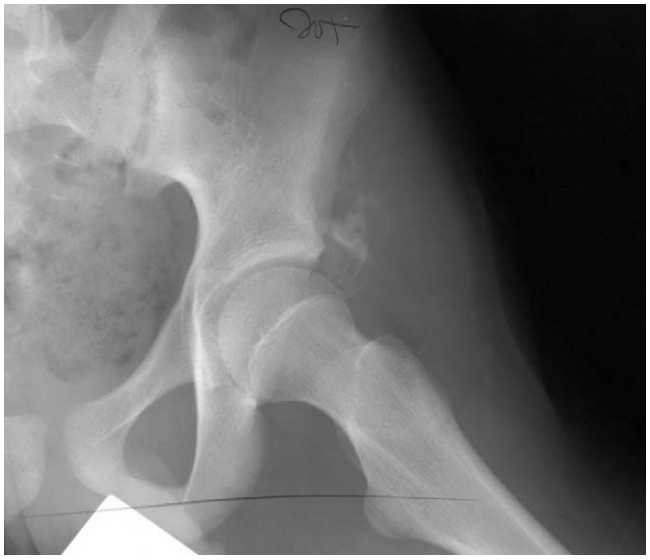


Figure 13 Multiple osseous fragments at the anterior inferior iliac spine are seen from a rectus femoris avulsion.

the iliac crest. The anterior superior iliac spine serves as site of origin of the sartorius muscle. The rectus femoris originates at the anterior inferior iliac spine (Fig. 13). The hamstrings originate from the ischial tuberosity and inferior pubic ramus (Fig. 14). The adductor muscles originate from the inferior pubic ramus near the pubic symphysis (Fig. 15). The gluteus medius and minimus insert on the greater trochanter of the femur. The iliopsoas tendon inserts on the lesser trochanter.

Fat Stripes

Several fat planes can also be seen on the AP radiograph.[5,14] The gluteal fat stripe is seen as a straight line paralleling the superior aspect of the femoral neck on a true AP radiograph and represents normal fat between the gluteus minimus tendon and the ischiofemoral ligament (Fig. 16). This line bulges superiorly in the presence of a hip joint effusion (Fig. 17).[14] The iliopsoas fat stripe is seen as a lucent line immediately inferior to the iliopsoas tendon (Fig. 16). The obturator fat stripe parallels the iliopectineal line and is formed by normal pelvic fat adjacent to the obturator internus muscle (Fig. 16), which may be displaced by fracture, hematoma, or mass.

Table 1 summarizes the important anatomic landmarks to be evaluated on every radiograph of the pelvis and hip joint.

Patterns of Disease

Trauma

The various radiographic projections mentioned above are used to determine which anatomic structures are disrupted in traumatic injuries to the pelvis. Injuries to the pelvis or hip may be fractures, dislocations, stress or insufficiency fractures, or avulsion injuries. In the setting of pelvic trauma, consideration should be given to the status of the nerves, arteries and veins of the pelvis, and proximal thigh, since their proximity to the osseous pelvis places these structures at risk.[15]

Pelvic Fractures

The Young and Burgess classification system divides pelvic fractures into types by mechanism of injury.[16] Anteroposterior compression, lateral compression, vertical shear (Fig. 18), and combined mechanical injuries are commonly associated with high-energy trauma, such as motor vehicle accidents. Associated injuries are common and can be life-threatening, including pelvic hemorrhage, especially with lateral compression and anteroposterior compression injuries.[15] Stabilization with pelvic compression, though useful in some pelvic fractures, is contraindicated in lateral compression injuries as it may compound the degree of collapse.[17]

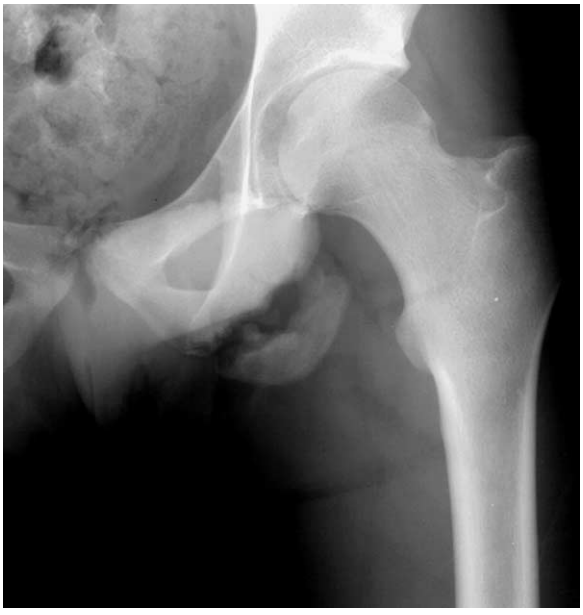


Figure 14 Complete avulsion of the ischial tuberosity, hamstring muscle origin.

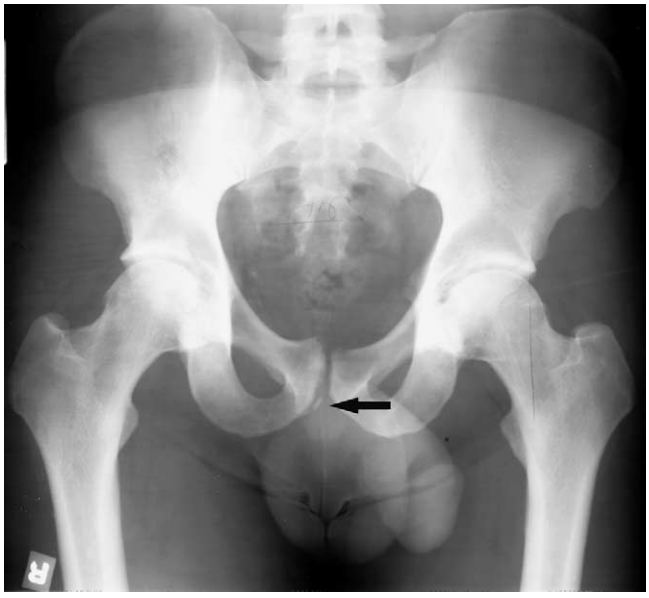


Figure 15 Small bone avulsion at the inferior aspect of the right pubic symphysis (arrow).

Avulsions

Avulsion injuries (Figs. 13-15) are most commonly sports-related acute traumatic injuries.[11,12] They can occur from chronic overuse, and in this setting, differentiation from osteomyelitis or neoplasm can be difficult radiographically.[11,12] Special note is made of avulsion injuries in adults with no history of trauma, as this should raise the possibility of an underlying neoplasm.[13]

Acetabular Fractures

The Judet and Letournel classification system divides acetabular fractures into anterior column, anterior acetabular rim, posterior column, posterior acetabular rim, transverse, T-type, posterior column and wall (Fig. 19), transverse and posterior wall, anterior column and posterior hemitransverse

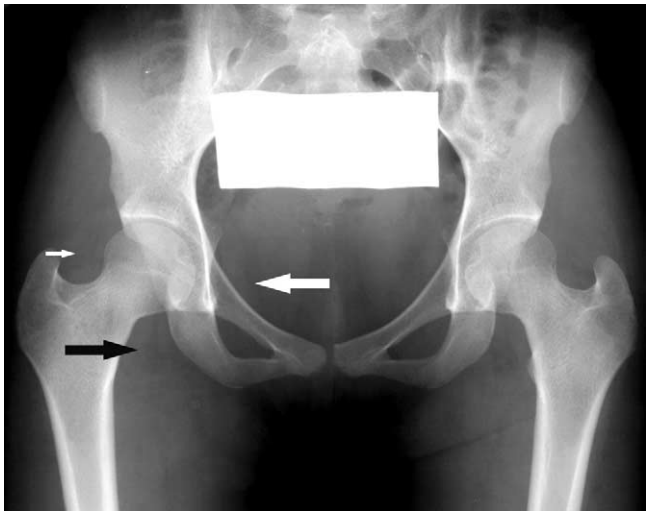
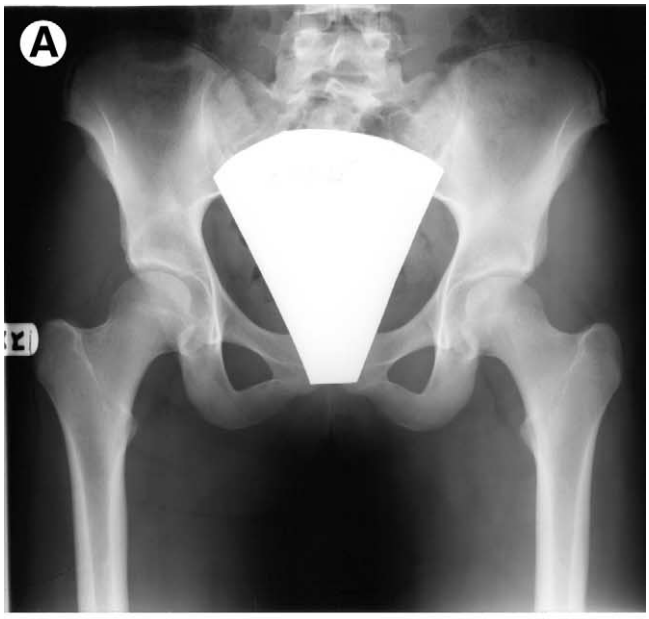


Figure 16 The gluteus minimus fat stripe (small white arrow), obturator internus fat stripe (large white arrow), and iliopsoas fat stripe (black arrow).



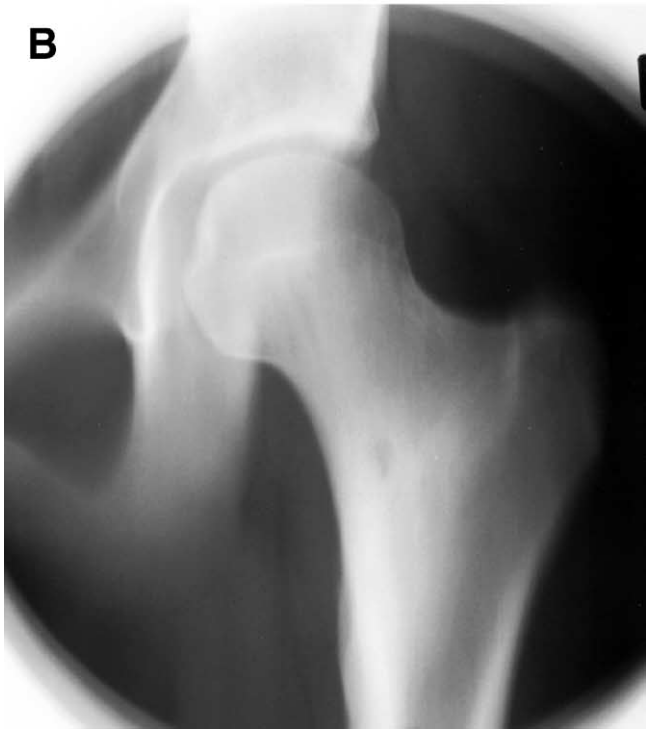


Figure 17 (A) The left hip joint is distended, as seen by elevation of the gluteus minimus fat stripe. Subtle sclerosis in the medial base of the left femoral neck is almost imperceptible. (B) Tomographic view demonstrates the nidus of an osteoid osteoma.

fractures, or both column associated fractures.[18] Recently, Harris and coworkers described a new CT-based classification of acetabular fractures.[19]

Femoral Head Fractures

Femoral head fractures are uncommon injuries, occurring typically in the setting of posterior hip dislocation.[20] Risk of avascular necrosis from this injury is high. Interposed fracture fragments may prohibit closed reduction.[20] The Pipkin

Table 1 Radiographic Evaluation of the Pelvis and Hip Joint

Symphysis pubis <5 mm in width
Sacroiliac joint 2-4 mm in width
Pelvic ring should have no disruption
Obturator ring should have no disruption
Sacral foraminal lines should be visible
Check transverse processes of lower lumbar vertebrae for fracture
Check the fat stripes: gluteal, iliopsoas, obturator internus
Iliopectineal or arcuate line disruption = fracture of anterior column
Ilioischial line disruption = fracture of posterior column
Radiographic U or teardrop
Acetabular roof
Anterior lip of the acetabulum
Posterior lip of the acetabulum
Line of Klein drawn along superior edge of femoral neck should intersect epiphysis
Shenton line, drawn between medial border of femoral neck and superior border of obturator foramen, should be smooth, continuous arc

classification divides these injuries into those with fracture fragment located below the fovea (type I, seen in 35% of cases), fracture fragment above the fovea (type II, 40% of cases), a combined fracture of the femoral head and neck (type III, 10% of cases), and combined fracture of the femoral head and posterior acetabular wall (type IV, 15% of cases) (Fig. 20).[20]

Femoral Neck Fractures

Several different classification systems have been suggested for classification of femoral neck fractures (Fig. 21). Probably the most widely used is the Garden system, which divides subcapital fractures of the femoral neck into grades I-IV.[21] Grade I is technically an incomplete fracture, though it has become customary to include impacted non-displaced fractures as Grade I, since the treatment is the same.[21] Grade II is a complete fracture, non-displaced. Grade III is a complete fracture with partial displacement and rotation. Due to the medial rotation of the proximal fragment with respect to the distal fragment, the trabecular lines of the femoral head do not align with those of the acetabulum.[22] Grade IV is completely displaced, so that the femoral head is no longer located in the acetabulum, and the trabeculae are aligned with those of the acetabulum.[22] The Garden system has consistently demonstrated low interobserver reliability in the literature,[21,22] although there is a consistently higher rate of complications, such as avascular necrosis with the higher grades (Fig. 22). One report found that the method of treatment was nearly always the same for grade III fractures as grade IV, while another report found that there was insufficient difference in fracture healing between grades III and IV to justify the distinction.[21,23] Thus, several authors have suggested a simplification of the Garden system into non-displaced fractures and displaced fractures.[21,22] Among other classifications, the Pauwels system has been reported to have poor ability to predict fracture healing and risk of complications, and the AO system has demonstrated poor interobserver and intraobserver agreement.[21,23]

Basicervical fractures of the femoral neck are generally considered separately, since they have much less risk of nonunion or avascular necrosis.[23] Intertrochanteric fractures may occur in a slightly older age group than femoral neck fractures, and perhaps as a result of this, appear to have a slightly higher rate of morbidity and mortality.[24]

Stress Fractures

Stress fractures may be defined as either "fatigue" fractures, occurring from abnormal stress on normally mineralized bone, or "insufficiency" fractures, occurring from normal stress on osteoporotic or poorly mineralized bone.[25] The earliest radiographic appearance of stress fracture is graying of the cortex due to bone resorption. Subsequently, a subtle area of sclerosis, usually linear and oriented perpendicular to the trabeculae, is seen.[25] The appearance progresses from smooth periosteal elevation to increasing amounts of periosteal new bone formation, as attempted healing takes place (Fig. 23).[25] Complete healing of the stress fracture is manifested by thick periosteal reaction, with disappearance of the

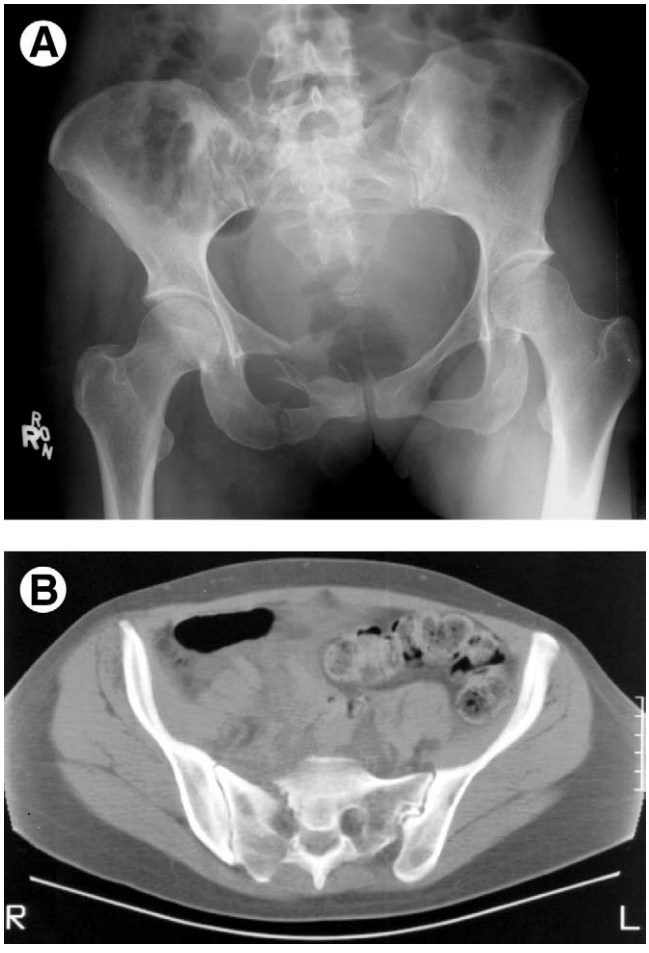
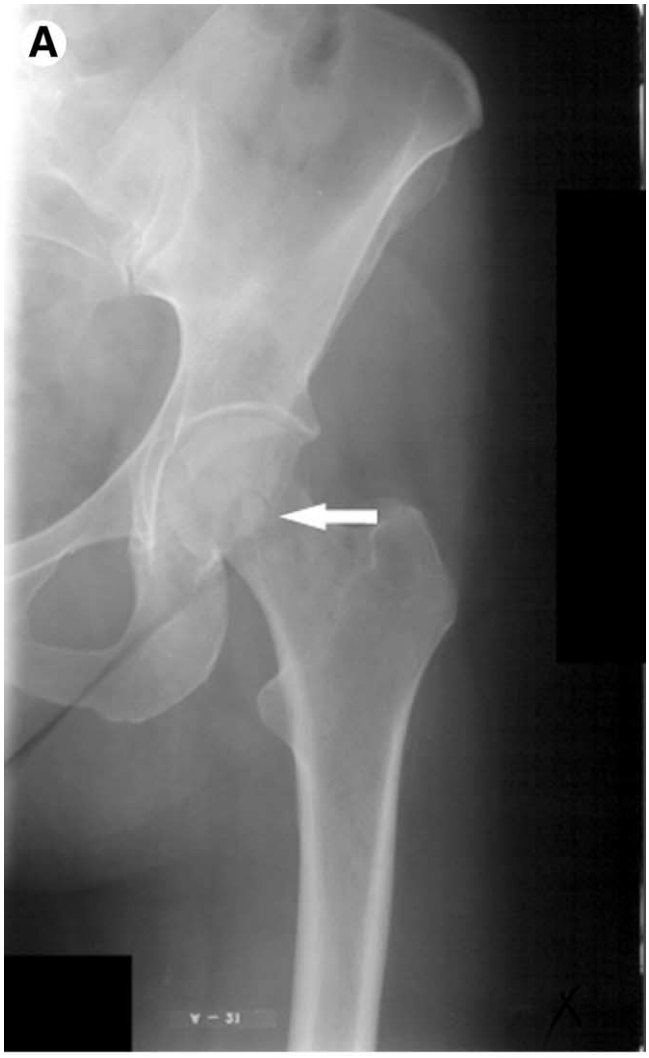


Figure 18 (A) The right superior and inferior pubic rami and right sacral ala are disrupted, and the right femoral head and acetabulum lie cephalad compared with the left. (B) The sacral fracture is better seen on CT. Vertical shear injury.



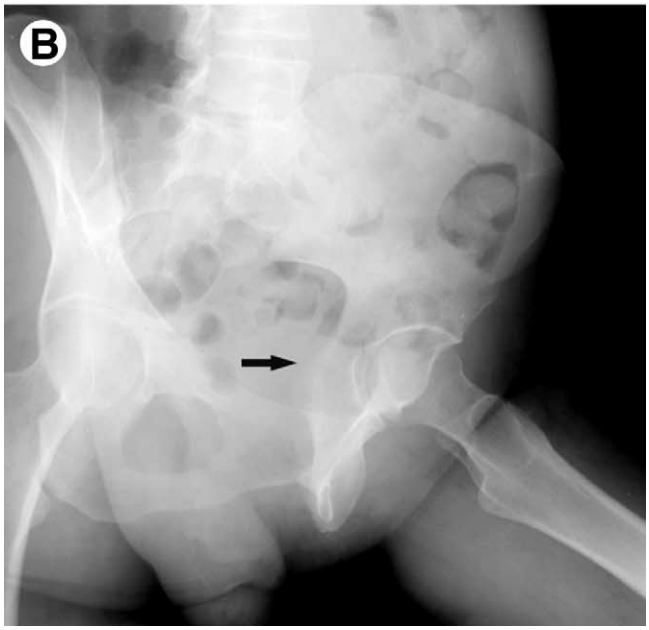


Figure 19 (A) Fracture line through the posterior acetabular wall is seen. Anterior column is intact. (B) The fracture through the posterior column is better seen on the 45° posterior oblique view.

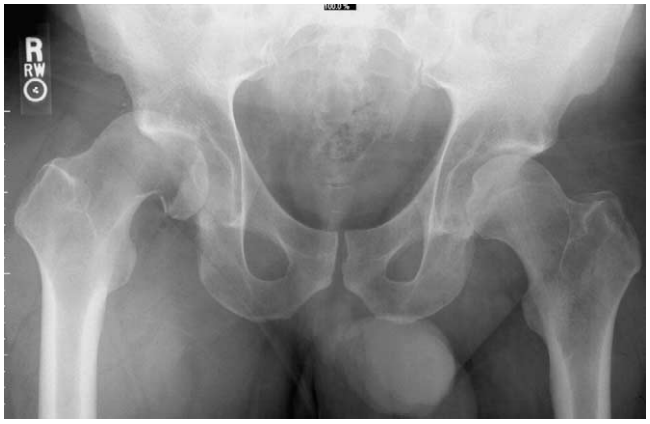


Figure 20 Posterior dislocation of the right hip is accompanied by femoral head fracture and posterior acetabular wall fracture, making this a Pipkin type IV fracture.

fracture line.[25] If continued stress occurs, however, the fracture may progress, rather than healing (Fig. 24). Tensile side stress fracture requires longer time to heal than compressive side stress fracture and may need surgical intervention. Occasionally, osteoid osteoma (Fig. 17) or infection could appear radiographically similar to a stress fracture. CT or MR imaging can be helpful in this differential diagnosis by demonstrating the fracture line.[25] Radionuclide bone scan and MR imaging are more sensitive than radiography for early detection of stress fractures.

Slipped Capital Femoral Epiphysis

Evaluation for slipped capital femoral epiphysis (SCFE) can be assessed using the anteroposterior and frog-leg lateral views of the hip.[2] The condition occurs most commonly in adolescents, around the time of puberty.[26] Boys are affected

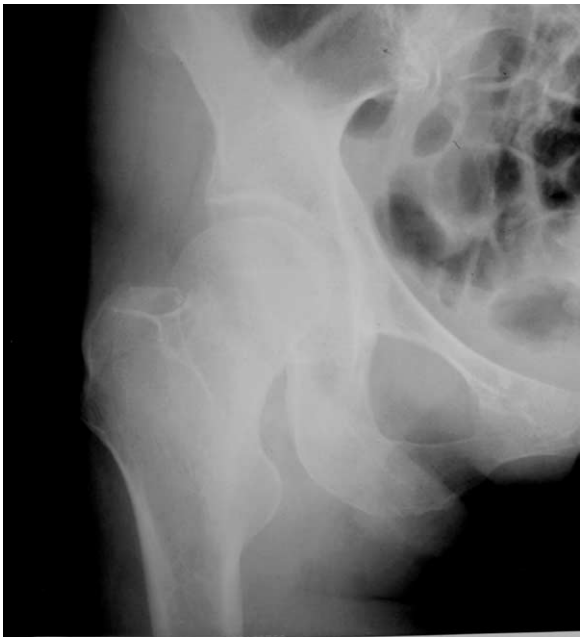


Figure 21 Impacted fracture of the right femoral neck with valgus angulation.

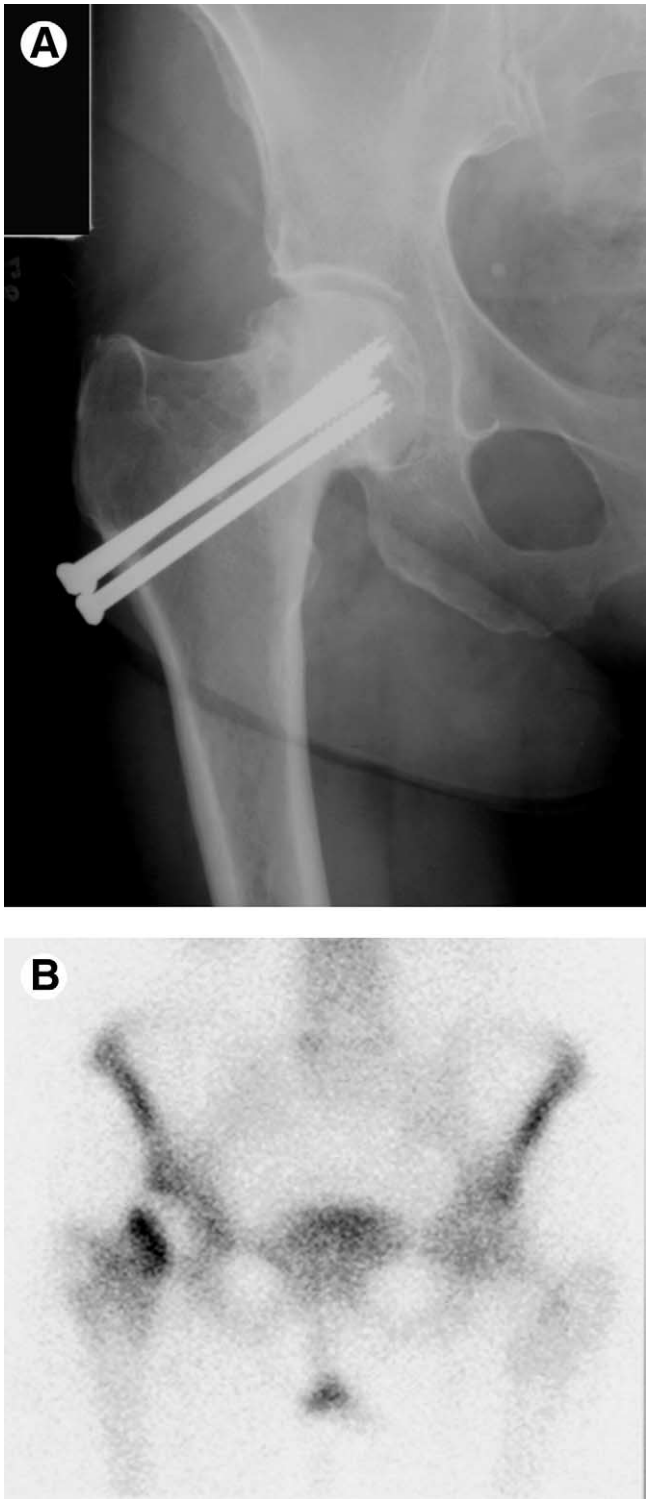


Figure 22 (A) Fixation of subcapital femoral neck fracture, with subsequent development of avascular necrosis. (B) Bone scan from the same patient showing photopenic defect within the right femoral head consistent with avascular necrosis.

more commonly than girls, with patients tending to be overweight. SCFE represents a Salter type I fracture, through the physis, resulting in the femoral head "slipping" inferomedially with respect to the femoral neck.[26] Radiographically, there may be widening or blurring of the physis or an apparent loss of epiphyseal height on the anteroposterior view.[27] A line drawn along the long axis of the superior aspect of the femoral neck, line of Kline, normally will intersect the epiphysis, but may not do so in the case of SCFE (Fig. 25).[2,26] Complications include avascular necrosis and chondrolysis.[26] In the chronic setting, a Herndon hump and secondary osteoarthritis may be seen.[2]

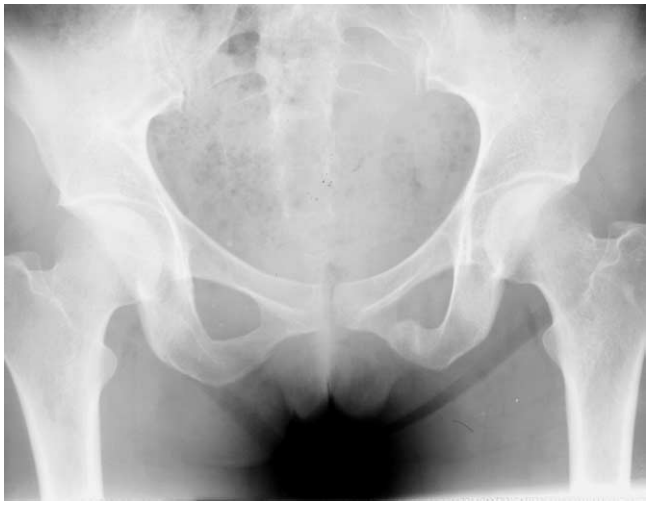


Figure 23 Healing stress fracture of the left inferior pubic ramus.

Infection

Septic Joint

Septic joint occurs most commonly from pyogenic infection[28] and may result from hematogeneous dissemination, contiguous spread of infection from local tissues, direct inoculation, or contamination at surgery. If an effusion is present, it may manifest radiographically with increased teardrop distance[29] or elevation of the gluteus minimus fat stripe, but these findings can be unreliable.[29] Subacute or chronic

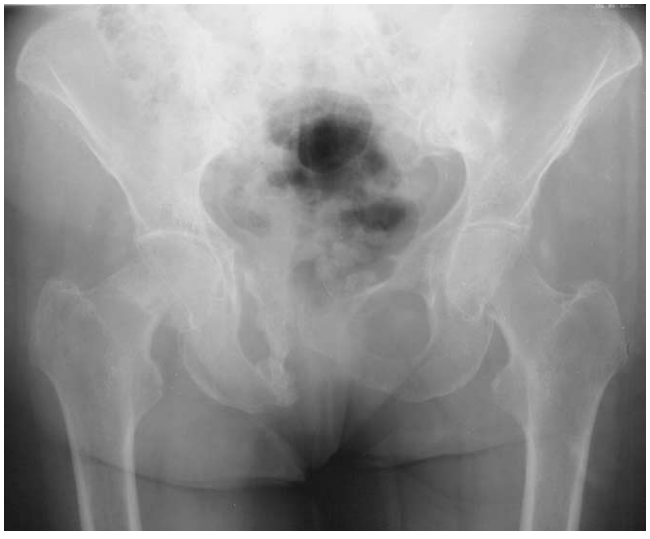


Figure 24 Stress fractures of the right femoral neck and superior and inferior pubic rami. There is varus angulation of the femoral neck fracture.

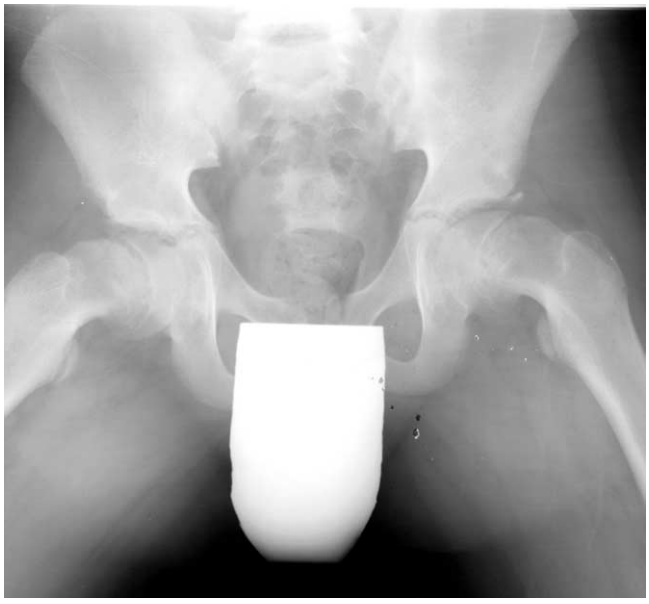


Figure 25 Bilateral slipped capital femoral epiphysis.

infections demonstrate bone erosions, loss of joint space (Fig. 26), and areas of avascular necrosis.[30] A joint aspiration is typically necessary to confirm the diagnosis.

Septic Sacroiliitis

Septic sacroiliitis is an uncommon infection seen most commonly in pediatric patients and young adults, and occasionally in the peripartum period.[31] Due to vague presenting symptoms and difficulty in localization at physical examination, diagnosis is often delayed.[32] The sacroiliac joints may be involved with pyogenic or tuberculous infection, arising most commonly from blood-borne pathogens.[31] Erosions of the sacroiliac joints may be seen (Fig. 27) and may be associated with osteomyelitis or soft-tissue abscess.[31] Differentiation between infectious and inflammatory sacroiliitis can at times be challenging (Table 2). Joint aspiration is often necessary for diagnosis.[31]

Tuberculosis

Osseous or articular involvement by tuberculous infection occurs in approximately 1 to 3% of all tuberculous infections (Fig. 28).[2,33] Slowly progressive monoarticular arthritis involving large joints such as the hip or knee is the most common presentation of tuberculous arthritis, though polyarticular involvement can occur and can be mistaken for inflammatory arthritis.[33,34] A low index of suspicion is indicated, as tuberculous infection is frequently not considered until irreversible damage to the joint has already taken place. Synovial biopsy improves diagnostic yield over aspiration of synovial fluid alone.[33,34]

Osteomyelitis

Osteomyelitis of the pelvis is much rarer than osteomyelitis of tubular bones, occurring most commonly in the ilium from hematogenous dissemination.[35] Symptoms are often nonspecific, and diagnosis is frequently delayed.[35] Radiographically, lytic lesions, sclerosis, smooth periosteal reaction, and/or a soft-tissue mass may be seen (Fig. 29).[2,35]

Arthritis

Osteoarthritis

The radiographic hallmark of osteoarthritis is joint space narrowing.[36] Most commonly, this narrowing is associated with subchondral sclerosis, marginal osteophytes, cyst formation, and superolateral subluxation of the femoral head (Fig. 30). An atrophic form of osteoarthritis has been described with joint space narrowing and superolateral subluxation of the femoral head, but with minimal osteophyte formation.[37] The atrophic type is most common in elderly women and is more frequently associated with hip dysplasia than the hypertrophic type of osteoarthritis.[37] Altered weight bearing due to

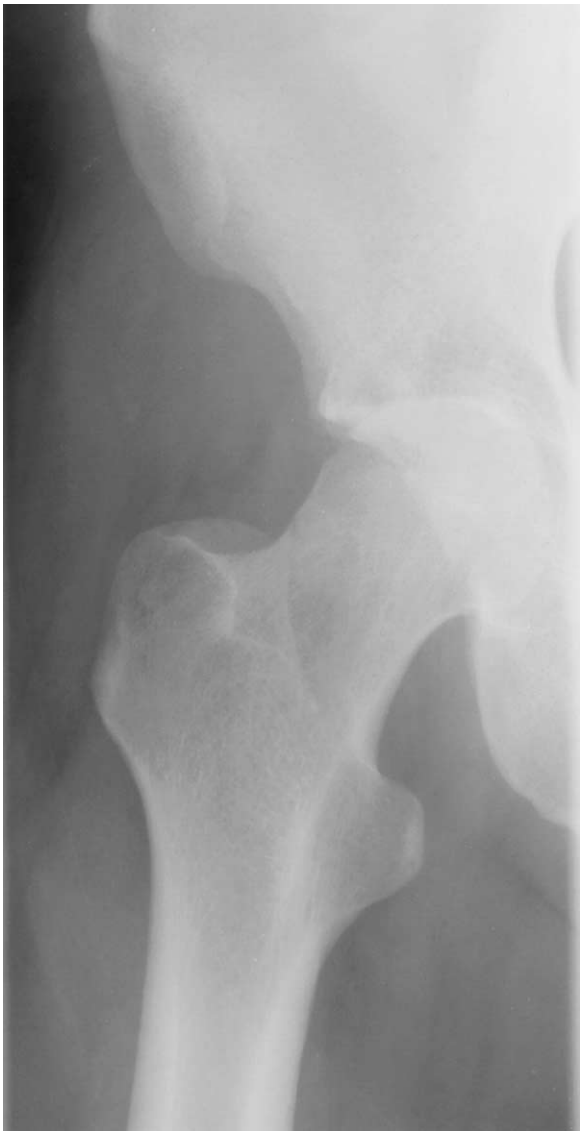


Figure 26 There is complete loss of joint space in the right hip, without appreciable osteophyte formation. Erosions of the superior acetabulum are present. Joint aspiration yielded pus. Arthrogram demonstrates filling defects within the joint (not shown).

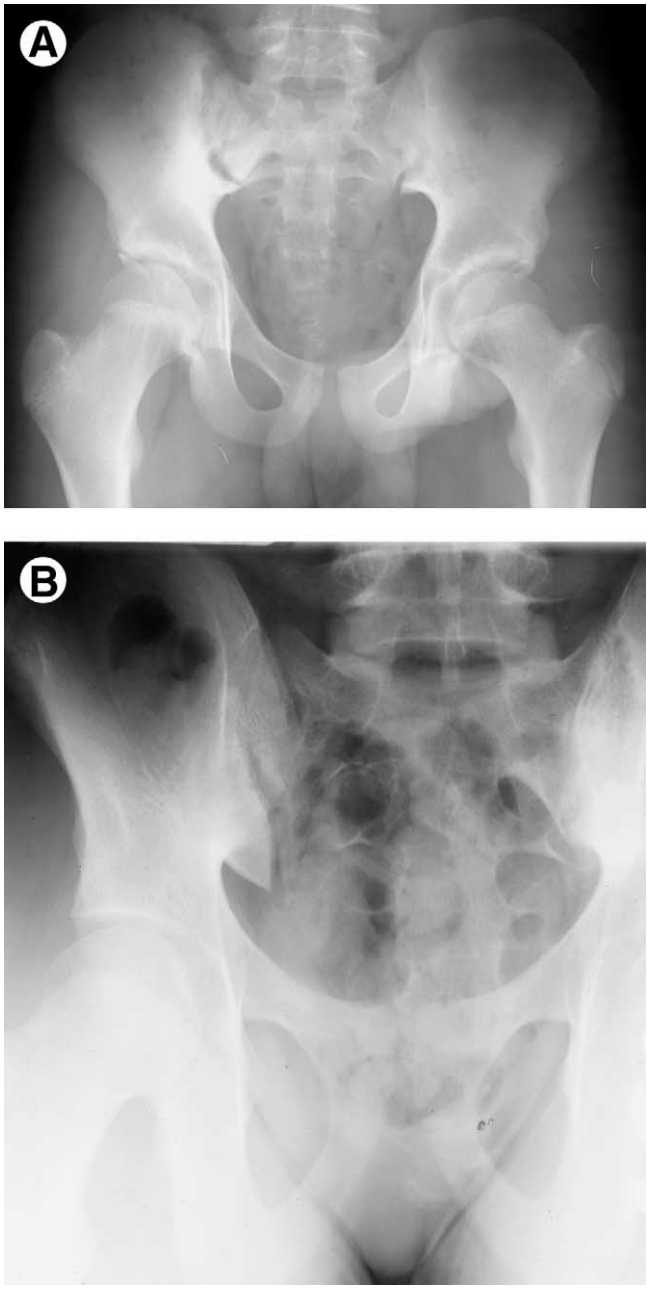


Figure 27 (A) and (B) Erosions and sclerosis of the right sacroiliac joint are seen. Aspiration yielded pyogenic infection.

traumatic injury or congenital anomalies may predispose to early development of osteoarthritis.

Neuropathic Arthropathy

Severe, occasionally rapidly progressive degeneration of the hip joint has been described in the setting of spinal cord injuries or sensory abnormalities (Fig. 31).[38] The cause is hypothesized to be an absence of the normal protective mechanisms of the neuromuscular structures about the joint.[39]

Rheumatoid Arthritis

Rheumatoid arthritis is an autoimmune disease affecting approximately 1% of the population, characterized by chronic, repeated episodes of synovial inflammation with eventual destruction and deformity of affected joints.[40] Bilateral, symmetric involvement of the hands and wrists is most common, but any joint may be involved. In the hip, distention of the joint and/or bursae, joint space narrowing, and protrusio acetabuli may be seen (Fig. 32).[2,40]

Table 2 Distinguishing Features between Septic Sacroiliitis and Spondyloarthropathy

Radiologic Features	Septic Sacroiliitis	Spondylo-arthropathy
Fluid within the sacroiliac joint	+	+
Joint space widening	+	+
Subchondral erosions	+/−	+
Subchondral bone marrow edema	+	+
Subchondral sclerosis	−	+
Transarticular bone bridges	−	+
Shiny corner sign or "Romanus" lesion	−	+
Bilateral involvement	−	+
Muscle infiltration	+	−
Abscess	+	−
Sequestration	+	−
Subperiosteal infiltration or "lava cleft phenomenon"	+	−

Seronegative Spondyloarthropathies

The seronegative spondyloarthropathies include ankylosing spondylitis, enteropathic arthropathy, Reiter's disease, and pso-

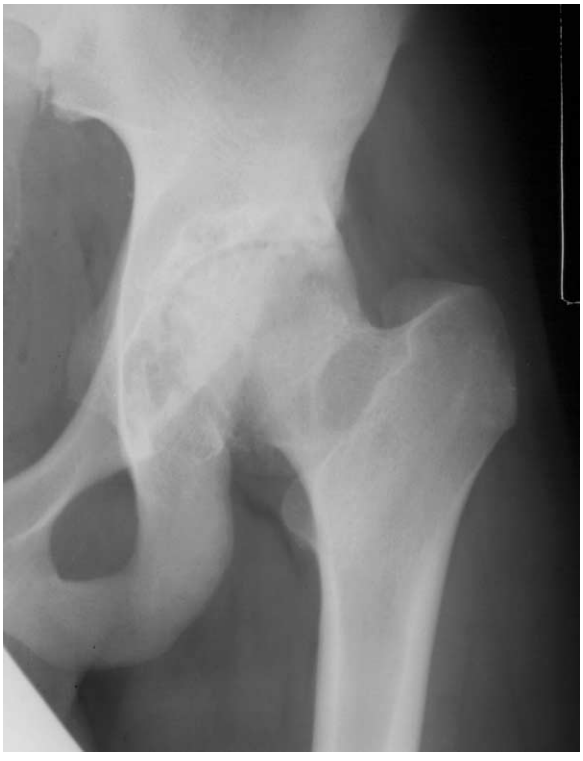


Figure 28 Erosions of the femoral neck, femoral head, and acetabulum, with severe loss of joint space are due to tuberculous arthritis.

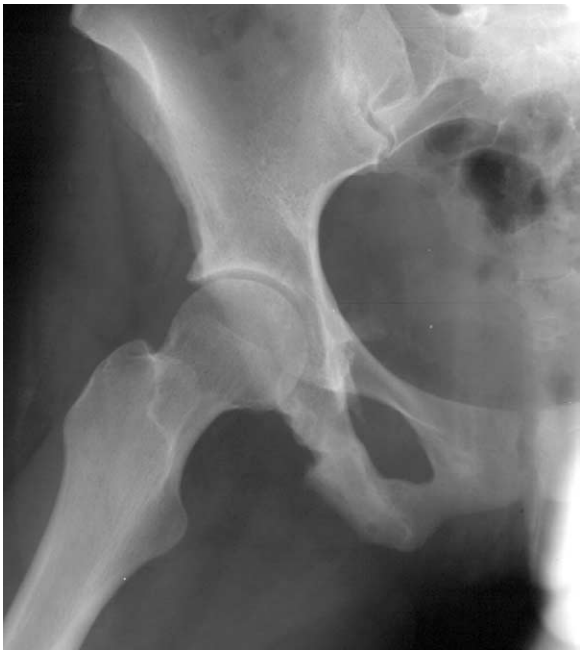


Figure 29 Sclerosis and irregularity of the right ischium are demonstrated, due to chronic osteomyelitis.

riasis. Radiographs of the pelvis in these disorders may demonstrate "whiskering" of the iliac crests and ischial tuberosity, sacroiliitis, protrusio acetabuli,[41] and/or lower lumbar spine fusions or osteophytes. Involvement of the sacroiliac joints occurs in the synovial portion of the joint, which is the caudal (and anterior) one-third of the joint.[42] Bilateral symmetric sacroiliitis is classically present in ankylosing spondylitis or enteropathic arthropathy.[43] Psoriatic arthritis and Reiter's disease classically cause asymmetric sacroiliitis, although it can be bilateral, and can at times appear symmetric.[43] Early inflammatory sacroiliitis may demonstrate erosions, particularly on the iliac side of the joint (Fig. 33).[43] Later there is sclerosis on both sides of the joint. In the chronic setting, fusion of the sacroiliac joints may occur (Fig. 34).[43]

Connective Tissue Diseases

Systemic lupus erythematosus patients are particularly at risk for osteonecrosis due to chronic corticosteroid intake and probably due to the auto-inflammatory process itself as well.[43] Evaluation for osteonecrosis can be assessed on standard views of the hip, but a more sensitive evaluation is obtained with MR imaging or radionuclide bone scan. Osteoporosis is also a sequela of this disease and may present clinically as an insufficiency fracture of the sacrum, pubic ramus, acetabulum, or femoral neck.

Dermatomyositis patients often develop soft-tissue calcifications, most commonly in the proximal upper and lower extremities (Fig. 35), and more often in children than in adults.[44] There is evidence that in juvenile dermatomyositis, up-regulation of genetic markers involved in producing interferon-alpha and -beta occurs, possibly as a result of exposure to a viral antigen.[45]

Scleroderma patients also may develop soft-tissue calcifications but typically have a myriad of other manifestations of the disease involving the hands and other organs, such as esophagus, small bowel, colon, and lungs.[43]

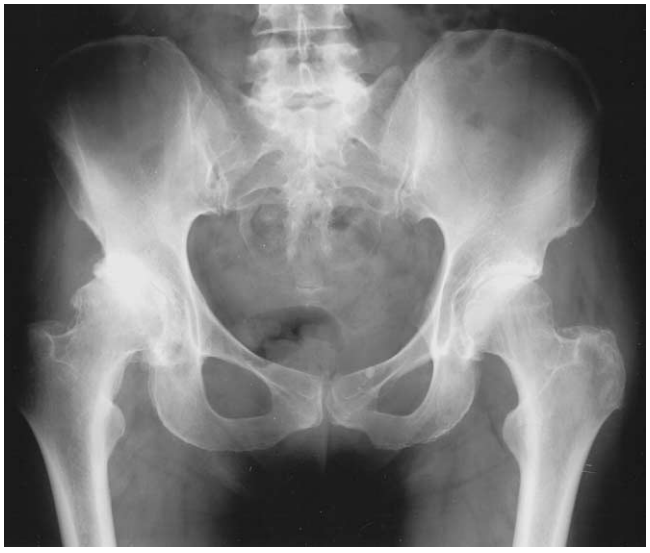


Figure 30 Characteristic findings of osteoarthritis: superior joint space narrowing, subchondral sclerosis, osteophyte formation, and mild superolateral subluxation of the right femoral head.

Metabolic, Synovial, and Crystal Deposition Diseases

Osteonecrosis

Osteonecrosis represents death of bone and may be caused by a wide variety of disease processes including trauma, alcoholism, Gaucher's disease, other infiltrative processes,

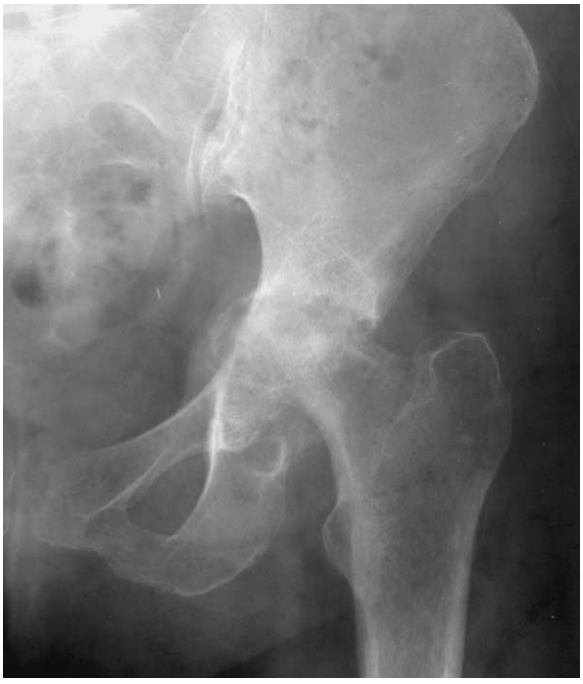


Figure 31 Severe joint space narrowing, destruction, debris within the left hip joint, and protrusio acetabuli are due to neuropathic joint.

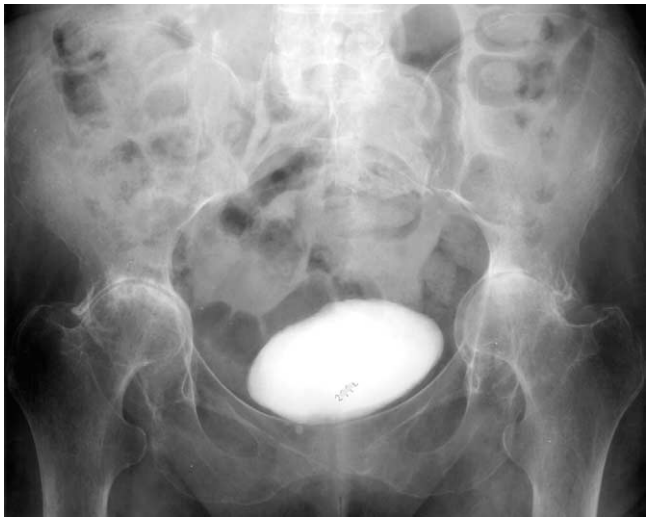


Figure 32 Symmetric joint space loss, erosions of the femoral heads, and acetabular protrusio are due to rheumatoid arthritis.

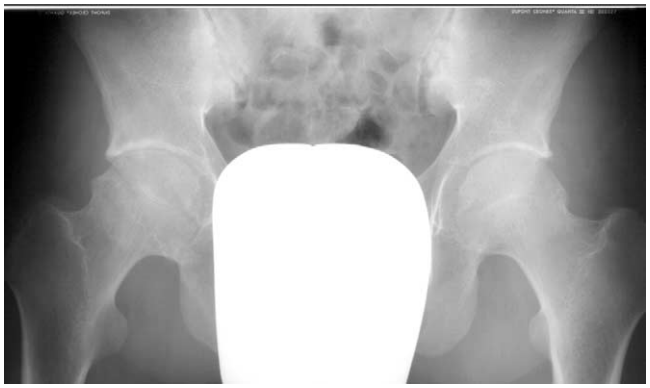


Figure 33 Erosions and sclerosis of the sacroiliac joint bilaterally are seen symmetrically. Ankylosing spondylitis.

sickle cell disease (Fig. 36), radiation, and many others.[46] The end result of these processes is decreased blood supply to the bone. In some cases, such as Legg–Calve–Perthes disease, no cause is evident. According to the Ficat staging,[47] the hip may have a normal appearance (stage I), vague increased density in the femoral head (stage II), subchondral collapse of the femoral head often producing a "crescent" appearance (stage III) (Fig. 37), or secondary osteoarthritis in the chronic setting (stage IV). Magnetic resonance imaging and radionuclide bone scan are much more sensitive than plain film radiography for the detection of early osteonecrosis (Fig. 37).[43]

Legg–Calve–Perthes Disease

Legg–Calve–Perthes disease occurs more commonly in males, between the ages of 4 and 8 years.[48] The condition is bilateral in about 10% of cases.[48] The etiology is unknown, leading it to be termed "idiopathic osteonecrosis." Radiographically, Perthes disease presents similarly to other types of osteonecrosis, as described above (Fig. 38). Overgrowth of the articular cartilage as a result of the osteonecrosis results in the eventual development of a large, deformed femoral head, called coxa magna. Associated lateral subluxation of the femoral head and secondary osteoarthritis may be seen. Treatment of Perthes disease varies from conservative to various types of acetabular or femoral osteotomies to maintain the femoral head in the acetabulum.[48]

Osteoporosis

Osteoporosis represents normally mineralized bone but with decreased overall amount of bone mass.[2] This can occur from a wide variety of congenital or acquired causes and manifests radiographically as increased lucency in the bone and cortical thinning. The risk of fracture increases with severity of osteoporosis. Singh and coworkers attempted a radiographic classification based on the theory that there is progressive loss of trabeculae (Fig. 9) with increasing severity of osteoporosis.[49] The Singh index divides this process into six grades of trabecular loss. In grade 6, all major trabecular groups are still present. In grade 5, some trabeculae in the secondary compressive, secondary tensile, and greater trochanter groups are lost, while the remaining trabeculae in the principal compressive and tensile groups are more prominent. In grade 4, the principle compressive and tensile groups are slightly reduced in number, while the secondary groups are nearly completely absent. In grade 3, there is a break in continuity of the principal tensile trabecular group. In grade 2, only the principal compressive group of trabeculae remains, which are reduced in number. In grade 1, even the principal compressive trabecular group is markedly reduced in number. Reports have varied in the literature concerning the reliability of the Singh index for measuring severity of osteoporosis.[50] Dual energy x-ray absorptiometry (DEXA) is more sensitive and accurate in quantifying osteoporosis.[50]

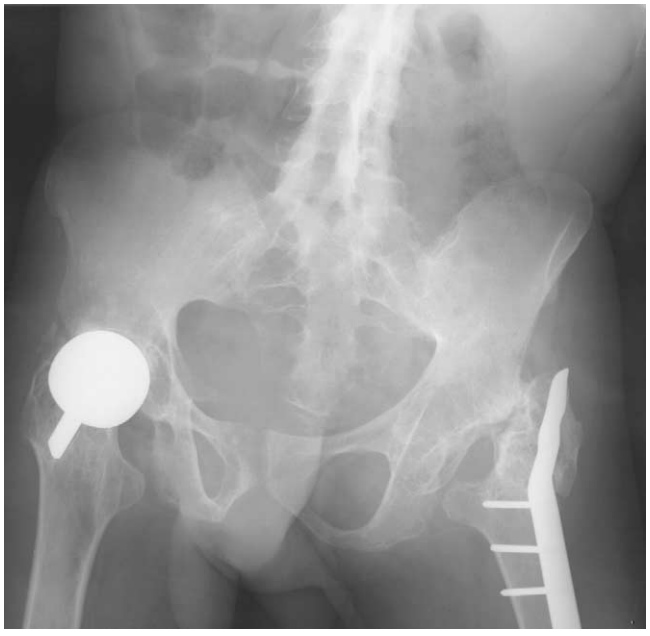


Figure 34 Complete fusion of the left and near complete fusion of the right sacroiliac joints are seen. There is also fusion of the facet joints and spinous processes in the lumbar spine. Deformity and dislocation of the left hip is also noted. Chronic ankylosing spondylitis.

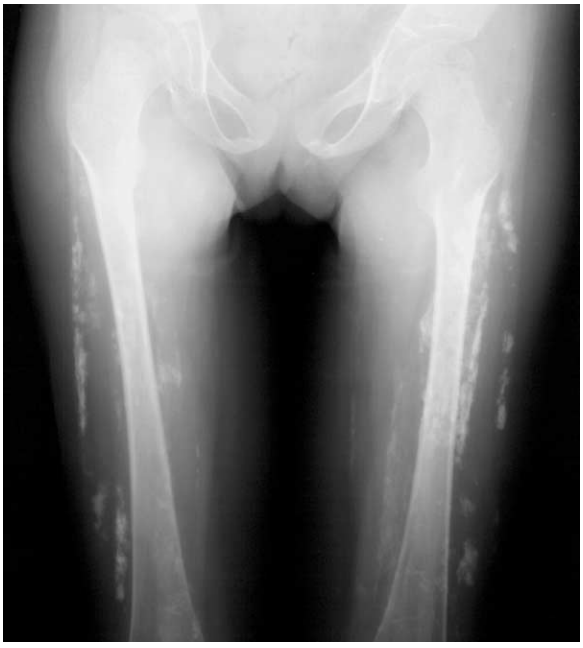


Figure 35 Extensive soft-tissue calcifications are noted bilaterally in the lower extremities of this patient with dermatomyositis.

Hyperparathyroidism

Primary or secondary forms of hyperparathyroidism may occur.[51] Primary hyperparathyroidism results from excess production of parathyroid hormone by the parathyroid gland. Secondary hyperparathyroidism occurs as a result of phosphate retention by poorly functioning renal tubules, leading to excess excretion of serum calcium, and subsequent increase in release of parathyroid hormone.[51] Subperiosteal bone resorption and cortical thinning may be seen, similar to the appearance seen in the phalanges.[51] Brown tumors may occur (Fig. 39). A "rugger-jersey"appearance to the vertebral bodies may be noted.[51]

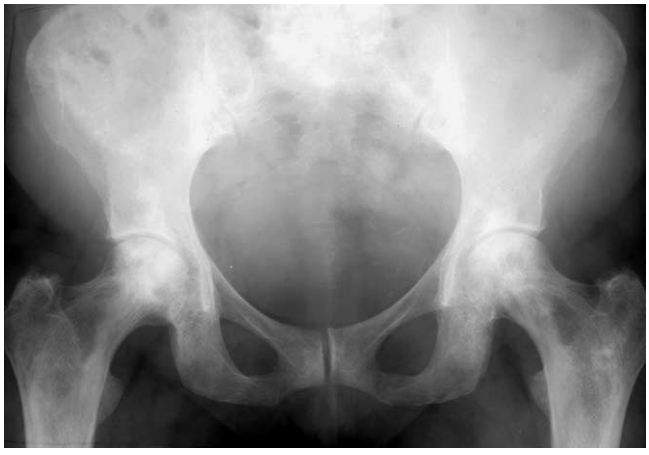


Figure 36 Abnormal appearance of the bones diffusely, with marked, patchy sclerosis in bilateral femoral heads, superior acetabuli, and pubic symphysis, resulting from repeated episodes of avascular necrosis in a patient with sickle cell disease.

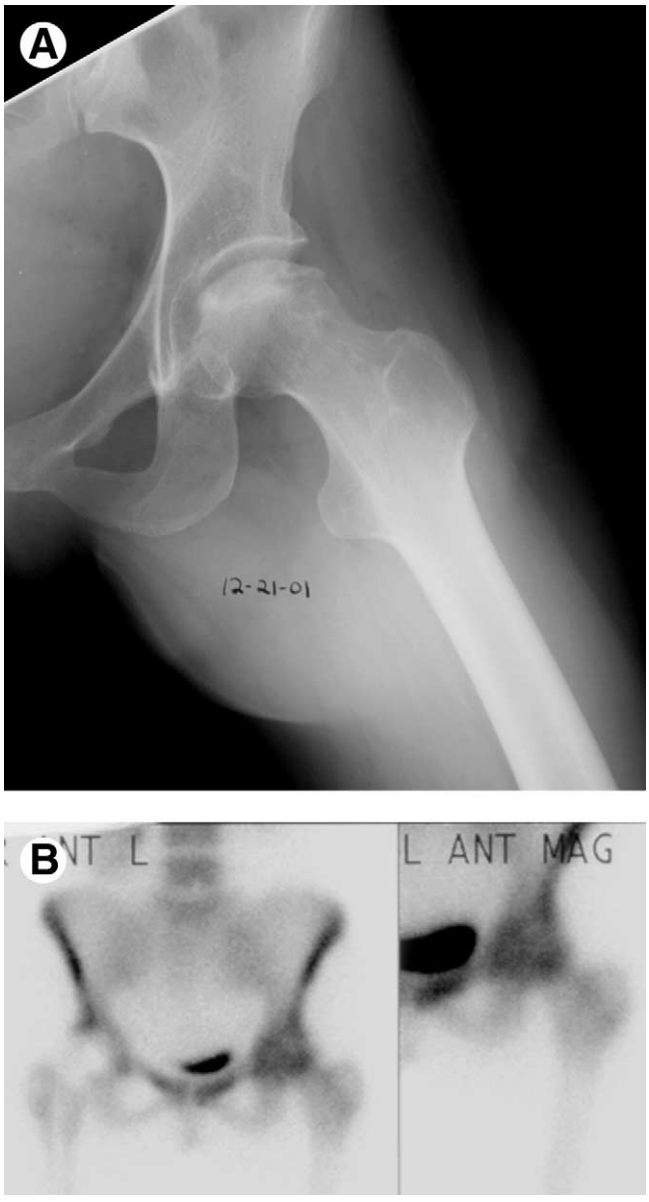


Figure 37 (A) In this frog-leg lateral view of the left hip, flattening of the femoral head contour and subchondral lucency denote collapse from avascular necrosis. (B) Bone scan in the same patient with photopenic defect in the superolateral femoral head, where bone is necrotic. The remainder of the femoral head shows increased uptake, due to the reparative response of surrounding bone.

Osteomalacia

Osteomalacia represents abnormally mineralized bone and may be caused by inadequate intake of vitamin D, inadequate absorption of vitamin D due to malabsorptive states or surgical bypass, or inadequate formation of the biologically active form of vitamin D due to renal or hepatic disease.[52] Osteomalacia has also been associated with other underlying disorders including Wilson's disease, neurofibromatosis, and some neoplasms.[2] Looser's zones, which are essentially poorly healed stress fractures due to inadequate callus formation, may be seen[52] (Fig. 40). These appear as linear lucencies, perpendicular to the cortex of the bone, occurring typ-

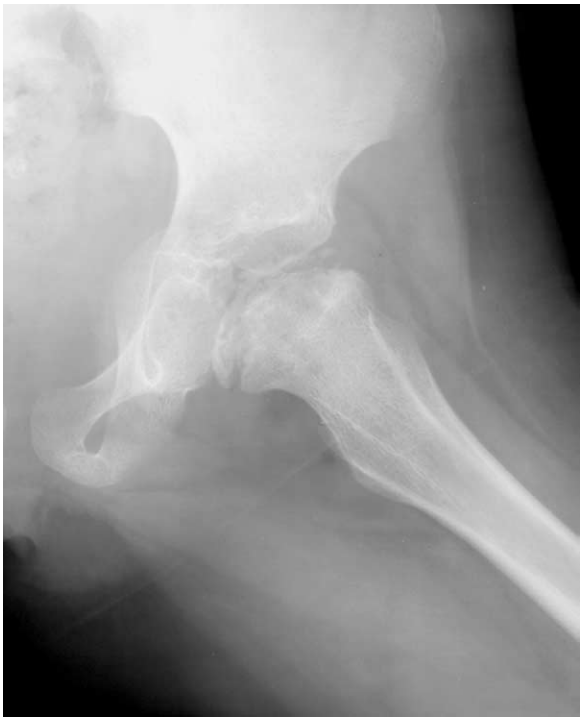


Figure 38 Severe collapse of the left femoral head in a patient with Legg–Calve–Perthes disease.

ically in the medial cortex of the femoral neck, as well as the pubic rami, ischial rami, ribs, and scapulae. When osteomalacia is due to renal dysfunction, the radiographic manifestations of secondary hyperparathyroidism usually predominate, including subperiosteal bone resorption and cortical thinning.[2]

Rickets

Rickets is the pediatric correlate of osteomalacia, resulting from abnormal mineralization of bone due to inadequate vitamin D intake, absorption, or hydroxylation. Different types of rickets have been described, with infantile rickets typically found in patients between the ages of 6 months and 2 years[53] (Fig. 41). The various types of vitamin D resistant rickets are typically found in patients over 3 years of age and are characterized by shortening and bowing of long bones, occasionally with sclerosis and ectopic ossifications[54] (Fig. 42). It may be seen with glycosuria or with defective renal tubular absorption of amino acids, glucose, and phosphate (Fanconi syndrome).[2]

Paget Disease

Paget disease has a characteristic appearance on radiographs, bone scan, MR, or CT with coarsened trabeculae and thickening of the cortex[55] (Fig. 43). Paget disease typically occurs in patients over the age of 50 years and progresses in three phases—predominately lytic, mixed lytic and sclerotic, and finally, sclerotic.[2,55] Increased osteoclastic activity leads to abnormal bone remodeling. The etiology of Paget disease is unknown, although a viral etiology is hypothesized.[55] Similar to osteomalacia, Looser's zones may form, representing inadequately healed stress fractures. A small percentage of patients with Paget disease will develop a secondary sarcoma.[56] New development of a lytic lesion or soft-tissue mass associated with Pagetoid bone is evidence of sarcoma formation. These tend to be high-grade lesions with a poor prognosis.[2,56]

A similar radiographic appearance may be seen in familial idiopathic hyperphosphatasia, with abnormalities seen diffusely[53] (Fig. 44).

Pigmented Villonodular Synovitis

Pigmented villonodular synovitis (PVNS) is a disorder of synovial proliferation characterized by recurrent effusions, with bleeding into the joint, bursa, or tendon sheath affected.[57] The knee is most commonly affected, but the disease can affect any synovial joint. In the hip, erosions may be seen in the femoral head, femoral neck, or acetabulum[57] (Fig. 45). MR is the imaging modality of choice, demonstrating synovial proliferation, effusion, and the paramagnetic effect of hemosiderin,[57] which may also be seen in patients with hemophilia or rheumatoid arthritis. Recurrence after synovectomy occurs in as many as 50% of cases.[2]

Synovial Osteochondromatosis

Synovial osteochondromatosis is a metaplasia of the synovium, most commonly involving the knee, although any joint may be involved.[58] Radiographs may demonstrate multiple ossified bodies in the joint space, with joint effusion, erosions on one or both sides of the joint, and scalloping of the femoral neck[59] (Fig. 46). Joint narrowing and osteophyte formation may be absent until late in the course of the disease. The chondroid bodies in the joint may or may not be ossified, but are evident at MR imaging or arthrography.[59] The amount of ossification increases over time. About 5% develop malignant degeneration.[58]

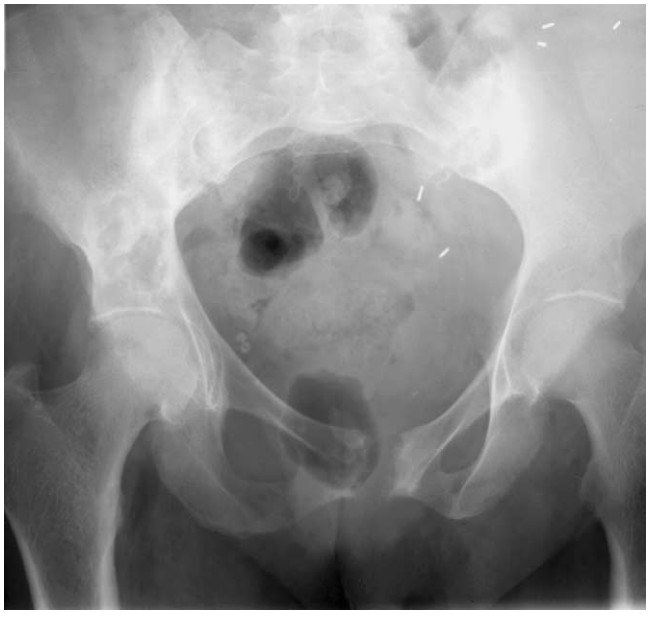


Figure 39 Subperiosteal bone resorption is noted in the pubic symphysis and sacroiliac joint bilaterally, and a lucent lesion is noted in the right superior acetabulum (Brown tumor). Hyperparathyroidism.

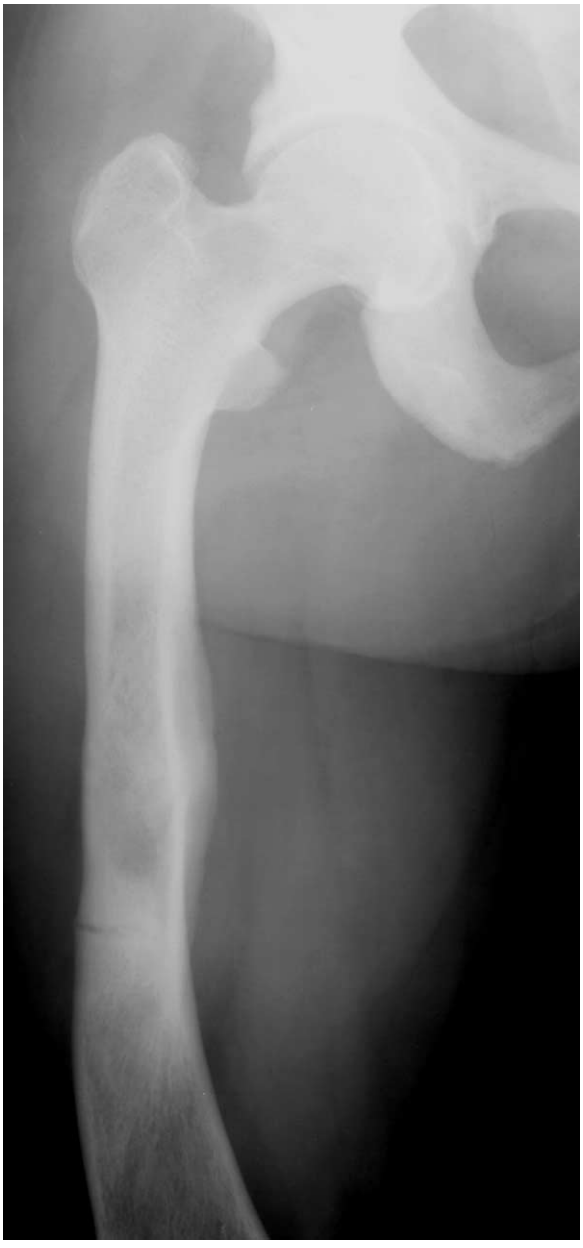


Figure 40 There is bowing of the femur, a lucent line perpendicular to the shaft (Looser's zone), and thickening of the medial femoral cortex in this patient with osteomalacia.

Calcium Pyrophosphate Arthropathy

The arthropathy associated with calcium pyrophosphate crystal deposition occurs in males and females with nearly equal incidence.[2] Deposition of calcium pyrophosphate crystals produces structural damage to the cartilage and resulting in joint space narrowing, subchondral sclerosis, and osteophytes. Chondrocalcinosis of the symphysis pubis, tendons, ligaments, articular cartilage, or joint capsule may be seen[60] (Fig. 47). The hip is less commonly involved than the knee, wrist, or shoulder.[60]

Hydroxyapatite Deposition Disease

Hydroxyapatite deposition disease is characterized by deposition of calcium hydroxyapatite crystals in and around tendons, bursae, or joint capsules, with resultant local swelling and pain.[61] Calcific tendonitis occurs less commonly in the hip than the shoulder.[61] "Toothpaste-like" calcifications of the gluteus medius and minimus tendons may be seen[61] (Fig. 48).

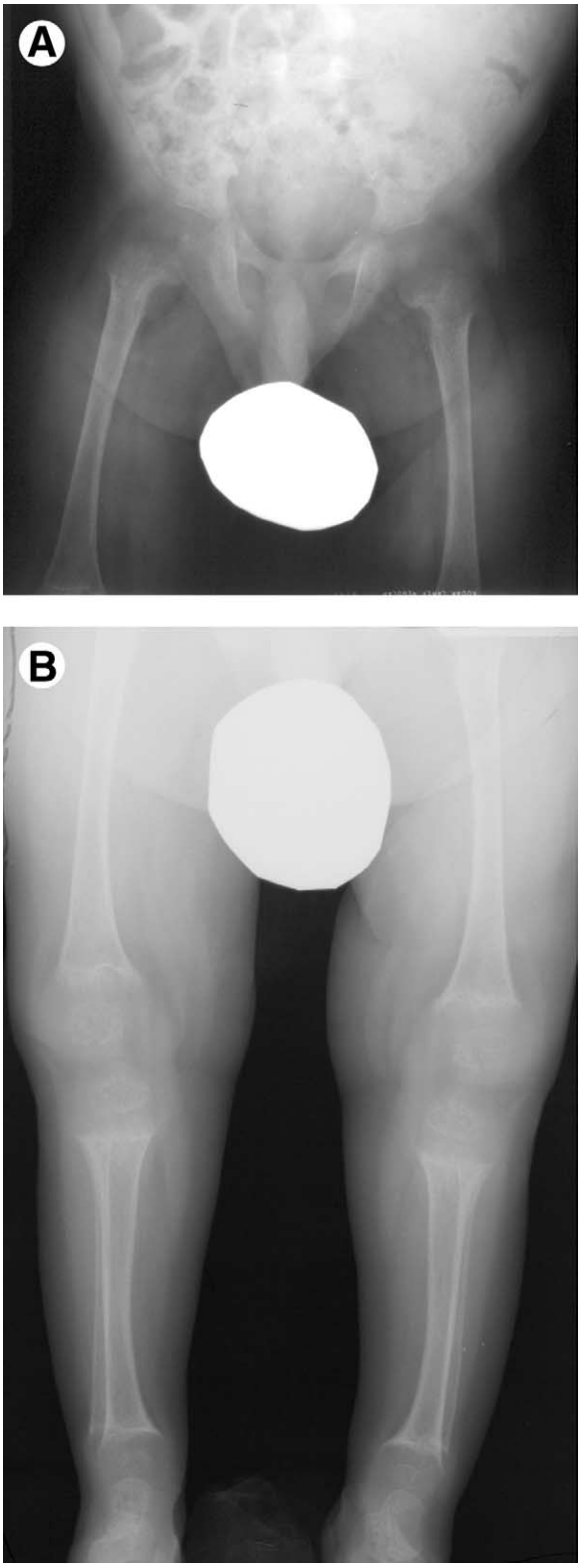


Figure 41 (A) and (B) Widened, frayed physis, broad metaphysis, and osteopenia are characteristic of infantile rickets.

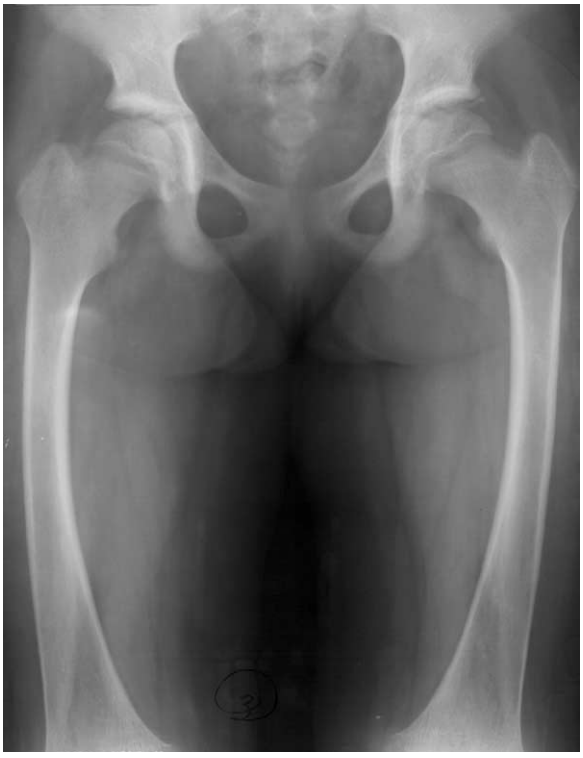


Figure 42 Bowing of the femurs, broad metaphyses, and widened proximal femoral physes are seen in vitamin D resistant rickets.

Ochronosis

Ocronosis occurs in males and females with equal incidence, as an autosomal recessive inherited condition.[62] Lack of the enzyme homogentisic acid oxidase, involved in the breakdown of amino acids phenylalanine and tyrosine, results in a build-up of homogentisic acid.[62] This leads to dystrophic calcifications in the intervertebral disks, articular cartilage, tendons, and ligaments, and formation of osteoarthritis[62] (Fig. 49).

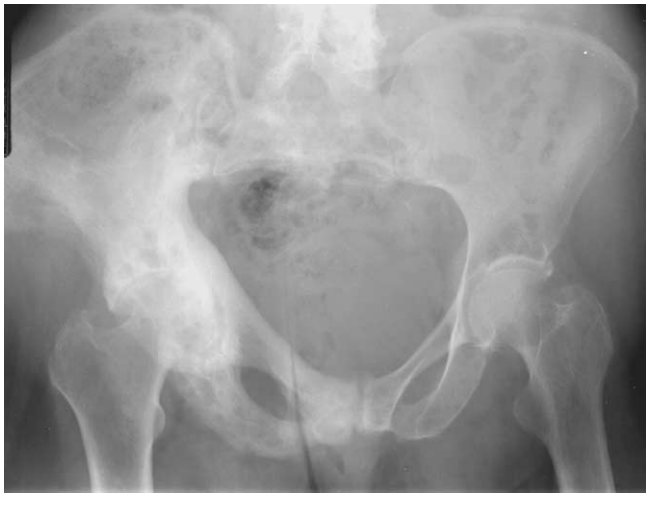


Figure 43 The right pelvis is enlarged, with trabecular and cortical thickening. The right hip demonstrates secondary osteoarthritis. Paget disease.

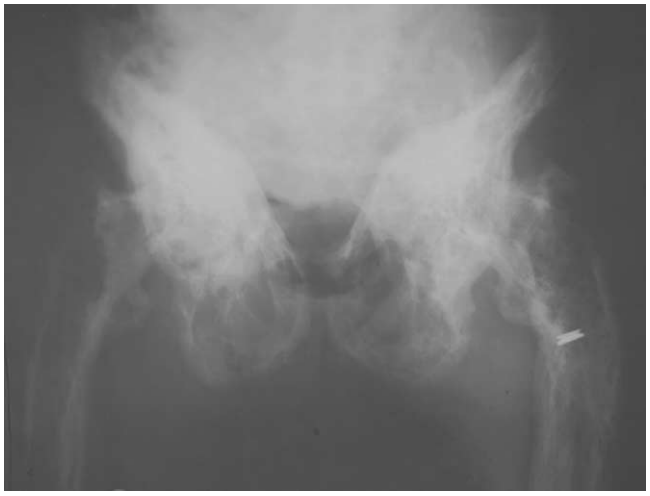


Figure 44 Enlarged bones with thickened trabeculae and cortex and heterogeneous mineralization diffusely are seen in this patient with idiopathic hyperphosphatasia.

Dysplasias and Congenital Anomalies

Sclerosing Bone Dysplasias

The various types of sclerosing bone dysplasias occur either from excess bone production due to abnormal osteoblastic activity or from failure of bone resorption and remodeling due to defective osteoclastic activity.[63] The excess bone accumulation affects endochondral bone formation in osteopetrosis, pyknodysostosis, or osteopathia striata.[64] Intramembranous bone formation is primarily affected in progressive diaphyseal dysplasia and a few rare endosteal hyperostosis.[64] Both endochondral and intramembranous bone formation are affected in melorheostosis and metaphyseal dysplasia.[64]

There are three types of *osteopetrosis*, the infantile-malignant type, which is autosomal recessive, and the most severe form, an intermediate type, also autosomal recessive presenting typically in the first decade of life, and an autosomal-dominant-type with full life-expectancy.[65] In the infantile type, pancytopenia, cranial nerve dysfunction, and mental retardation occur.[65] Radiographs of the hip may demonstrate curvilinear bands of sclerosis in the ilium, with a "bone-in-bone" appearance.[2,65] The vertebrae may show similar bands of sclerosis along the vertebral endplates, with a "sandwich vertebrae" appearance. In the long bones, undertubulation, broadened metaphyses, and pathologic fractures are seen[65] (Fig. 50).

Pyknodysostosis is a rare autosomal-recessive disorder with radiographic characteristics of both osteopetrosis and cleidocranial dysplasia.[2,66] Delayed closure of fontanelles, short stature, undertubulation of long bones, and diffuse sclerosis are seen. Bones are brittle and prone to fracture (Fig. 51). Genetic research has demonstrated a mutation causing inactivation of the gene encoding cathepsin K, which is involved in osteoclastic function.[63]

In *melorheostosis*, both endochondral and intramembranous bone formation are abnormal.[64] It is characterized by hyperostosis, typically of one side of the cortex, with a lobulated, wavy appearance resembling dripping candle wax.[67]

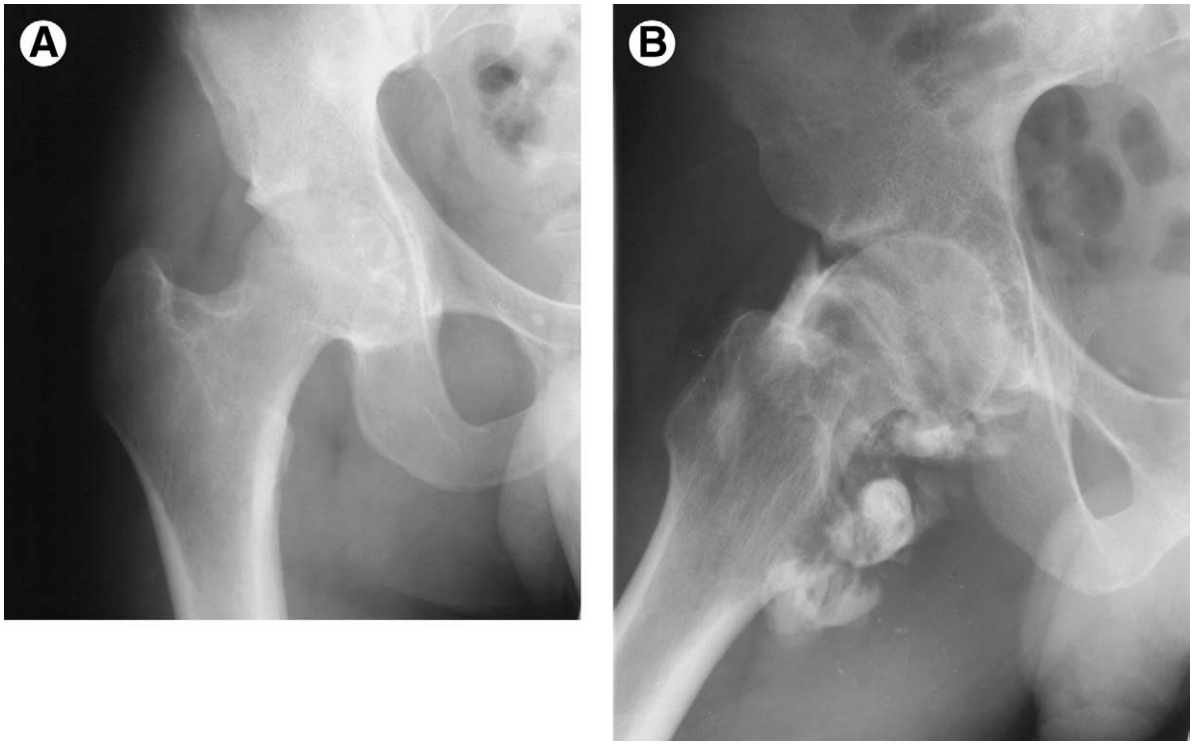


Figure 45 (A) Scalloping of the femoral neck and erosions of both femoral head and acetabulum are seen in this patient with PVNS. (B) Arthrogram demonstrates nodular-appearing filling defects.

Ossifications and fibrosis in periarticular soft tissues are also common.[67] The abnormalities may follow a dermatomal distribution[67] (Fig. 52). Treatment includes soft-tissue releases and excisions, and if necessary, osteotomies.[67] It commonly recurs.

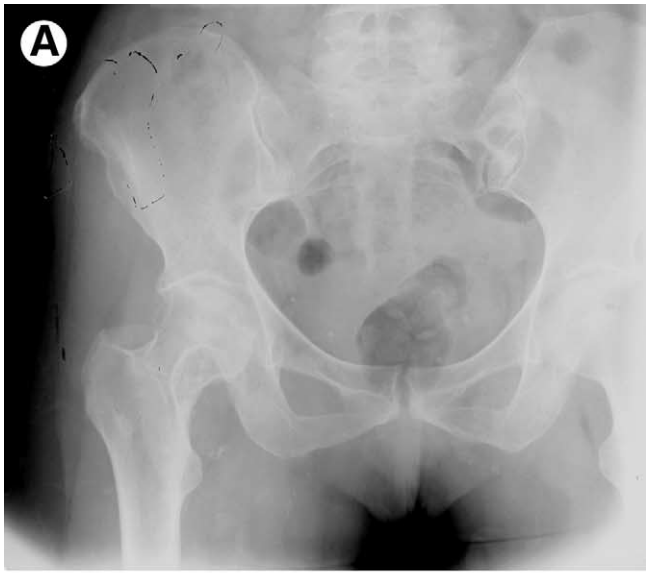
In *osteopoikilosis* and *osteopathia striata*, there are localized foci of cortical bone in which resorption and remodeling fail, while in the remainder of bone, the process of endochondral ossification proceeds normally.[68] The result is numerous foci of sclerotic bone (enostoses or "bone-islands") throughout the skeleton in osteopoikilosis (Fig. 53), or linear striations of sclerotic bone in osteopathia striata.[68] Differentiation from sclerotic metastases may at times be difficult by plain radiographs. Although radionuclide bone scan has been thought to be critical to differentiate osteopoikilosis from osteoblastic metastases, there are reports of increased radiopharmaceutical uptake in osteopoikilosis, particularly in young patients.[69] Osteopoikilosis, osteopathia striata, and/or melorheostosis can coexist in the same patient (Fig. 52) and probably represent a range of manifestations of the same disease process.[2]

Osteogenesis Imperfecta

Osteogenesis imperfecta (OI) is a hereditary disorder characterized by abnormal type I collagen, resulting in weakened, fragile bones, ligament laxity, abnormal dentition, blue sclerae, and hearing impairment[70] (Fig. 54). Most subtypes of OI are inherited as autosomal-dominant mutations in the COL1A1 and COL1A2 genes that encode for the pro alpha 1 and pro alpha 2 chains in type I collagen.[70] Types I-IV, described by Sillence and coworkers, are as follows: type I, autosomal-dominant and relatively mild, with relatively normal stature, blue sclerae, and hearing impairment; type II, with subtypes described as autosomal-dominant or autosomal-recessive, the most severe form, lethal in the fetal or newborn period, with severe deformity and intrauterine growth retardation; type III, also with both autosomal-dominant and autosomal-recessive cases described, severe and progressive but with longer survival than type II; type IV, rare, autosomal dominant and mild with normal sclera and normal hearing.[71] More recently, additional types V-VII have been described, which are not associated with defects in the genes encoding type I collagen.[72] Treatment with biphosphonates improves bone mass in all types, but long-term outcomes from biphosphonate therapy are not known.[70]

Developmental Dysplasia of the Hip

The etiology of developmental dysplasia of the hip (DDH) involves both genetic and environmental factors.[73] Risk factors include oligohydramnios, breech delivery, positive family history, and certain ethnic backgrounds including Native Americans.[73] Diagnosis can be made at birth in the vast majority of cases.[73] If diagnosed at birth, the likelihood of successful nonoperative treatment such as a Pavlik harness, and the overall prognosis, is much better than with delayed diagnosis.[73,74] Ultrasound is more sensitive than radiography for diagnosis.[73] Radiographically, dislocation or subluxation of the hip can be demonstrated by discontinuity of the Shenton arc, a curvilinear line connecting the medial femoral neck with the undersurface of the superior pubic ramus[73] (Fig. 55). With hip dislocation, the femoral head moves into the upper outer quadrant.[2] If the dislocated hip is in contact with the ilium, a pseudoacetabulum will form[73] (Fig. 55). Essen-



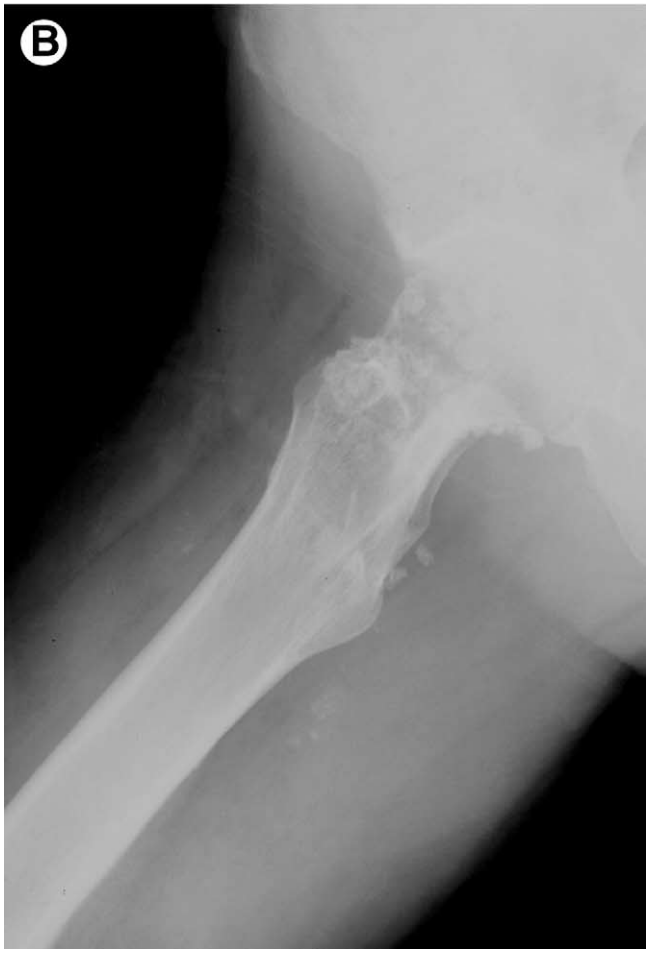


Figure 46 (A) Scalloping and erosions of the femoral neck and head are seen, with ossific densities in the joint. Synovial osteochondromatosis. (B) The same patient 4 years later demonstrates enlargement of the ossified intraarticular bodies.

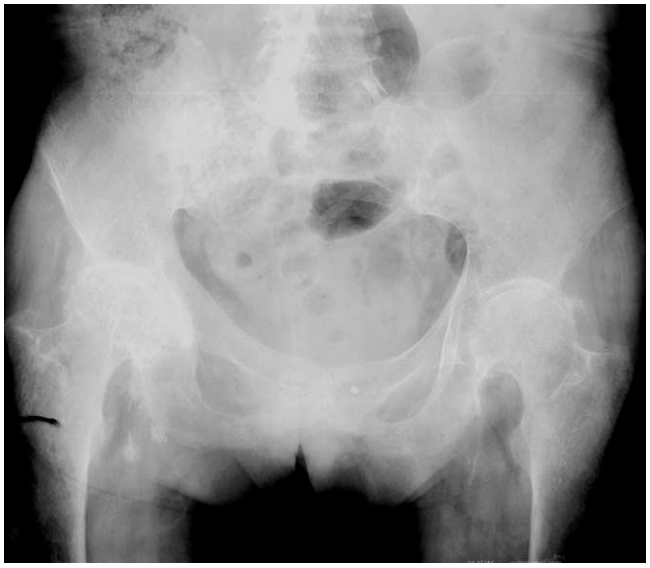


Figure 47 Chrondrocalcinosis of the pubic symphysis and both hips, with severe diffuse degenerative changes. Calcium pyrophosphate arthropathy.

tially all patients with hip subluxation or dislocation will develop osteoarthritis, usually in the 3rd or 4th decade of life.[73]

Acetabular Dysplasia in Adults

Dysplasia of the acetabulum may occur without hip dislocation, and mild dysplasia may go undiagnosed until adulthood.[73] Acetabular dysplasia occurs in females more often

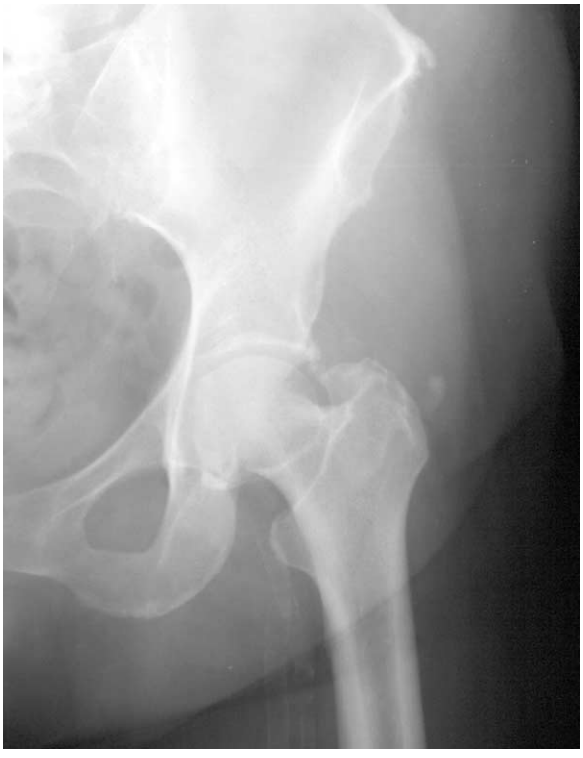


Figure 48 "Toothpaste"-like calcifications at the hip abductor insertion.

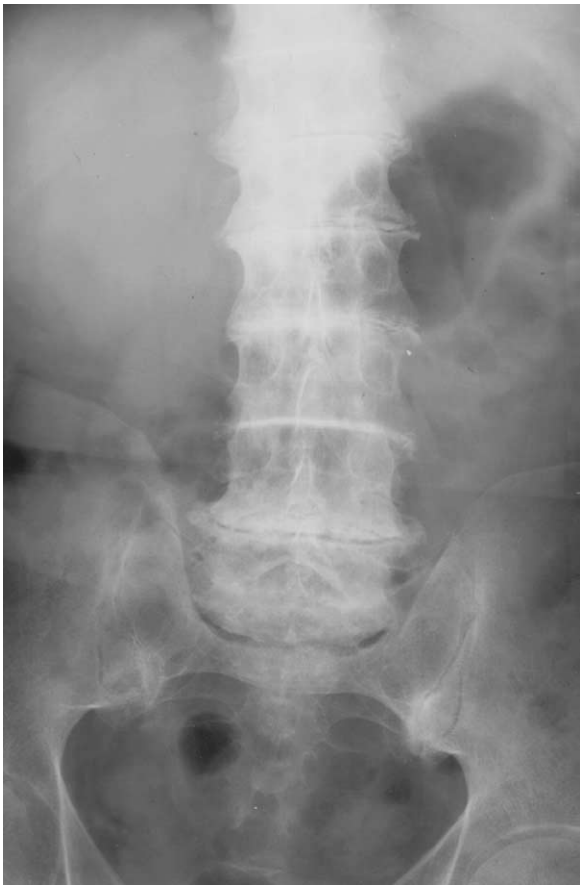


Figure 49 Diffusely calcified intervertebral disks. Ochronosis.

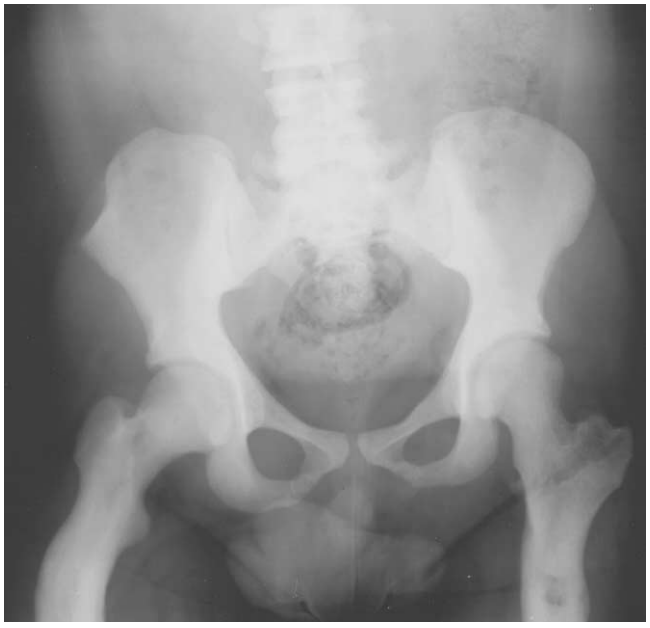


Figure 50 All visualized bones are sclerotic. Previous fractures of the right femoral shaft and left femoral neck with residual deformity. Osteopetrosis.

than males and has been demonstrated to lead to development of hip joint osteoarthritis[73,75] (Fig. 56). Evaluation for acetabular dysplasia can be performed using the center-edge angle of Wiberg, performed by measuring the angle between a line drawn vertically from the center of the femoral head and a line from the center of the femoral head through the edge of the acetabulum.[75] Angle measures less than 20° are dysplastic; 20 to 25° are classified as borderline dysplasia, and greater than 25° are normal.[75]

Femoroacetabular Impingement

The theory behind femoroacetabular impingement is that certain anatomic variations lead to impingement between the proximal femur and acetabular rim with flexion and internal rotation.[76,77] This leads to shearing and impaction of the anterior articular cartilage of the femoral head, as well as anterior labral tears.[76,77] There are two types of femoroacetabular impingement. The first is the "cam" type, thought to be caused by an enlarged femoral head or an abnormal contour of the femoral head/neck junction, which causes impingement anteriorly against a normal acetabulum (Fig. 6). The second, or "pincer" type, is thought to be due to "over-coverage" of the femoral head anteriorly from either coxa profunda or a retroverted acetabulum.[76] Radiographs may demonstrate reduced offset of the femoral head–neck junction, acetabular abnormalities such as retroversion, coxa valga, coxa profunda, or protrusio acetabuli, and the eventual development of osteoarthritis. MR imaging is more sensitive to the early findings of labral tear and cartilage injury.[76,77]

Achondroplasia

Achondroplasia is a congenital disorder of endochondral bone formation affecting fetuses in utero, transmitted as an

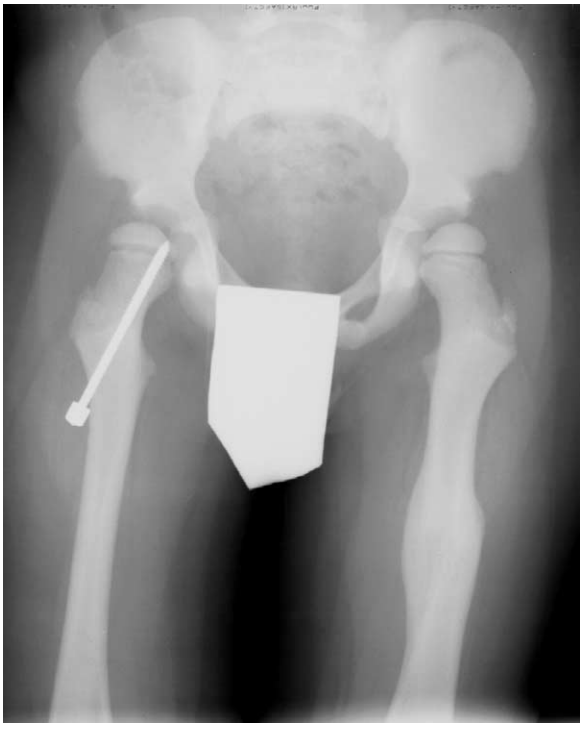


Figure 51 The diffusely sclerotic bones, undertubulation, and fractures resemble osteopetrosis. Pyknodysostosis.

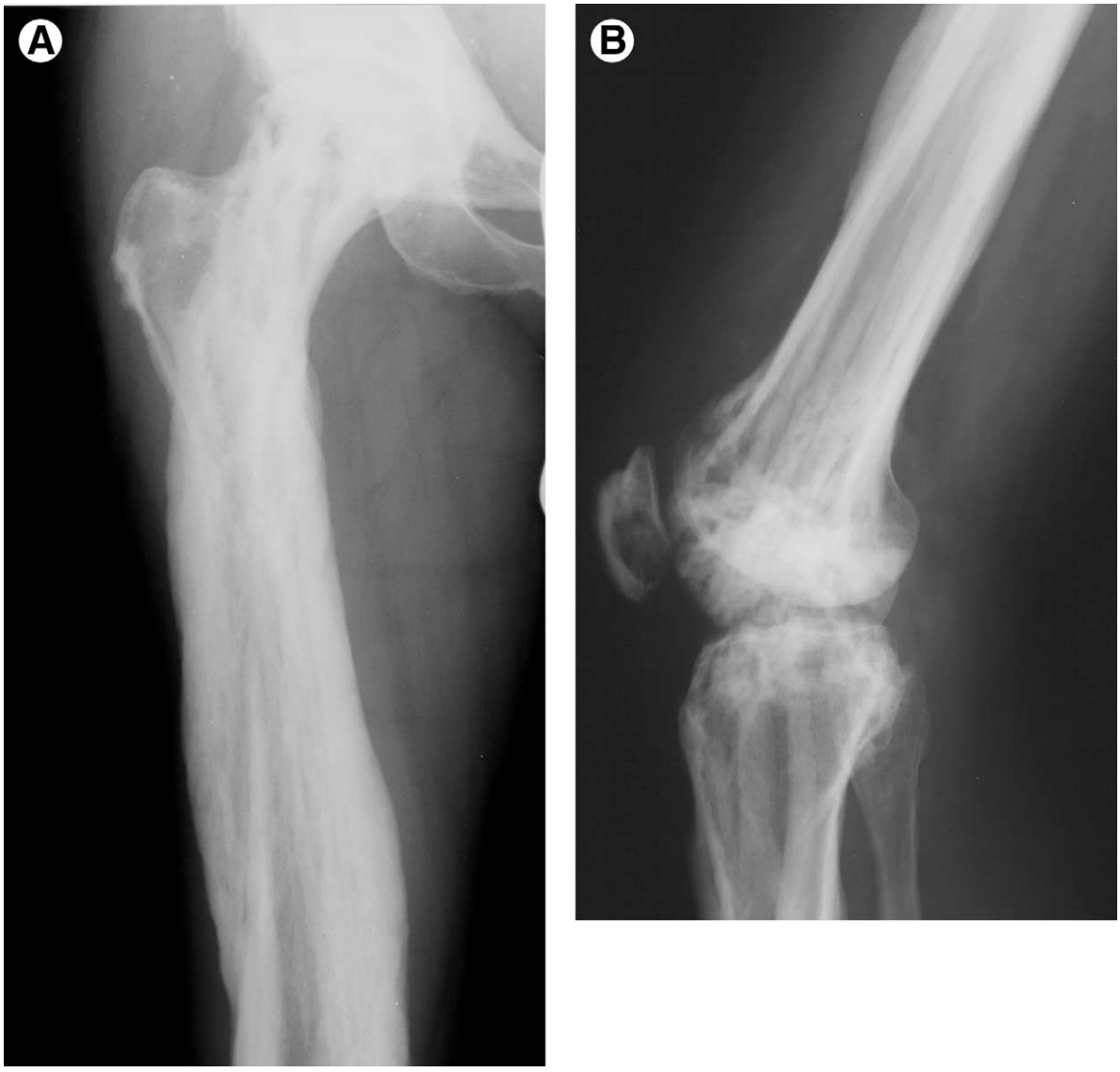


Figure 52 (A) Dense sclerotic, wavy cortical thickening in the femoral shaft and superolateral acetabulum are noted. (B) Wavy cortical thickening in the distal femoral shaft is seen indicative of melorheostosis. Linear striations in the medullary bone are areas of osteopathia striata.

autosomal-dominant trait.[78] The genetic defect involves an allele encoding fibroblast growth factor receptor 3, on chromosome 4p, which is the same allele implicated in both hypochondroplasia and thanatophoric dwarfism.[78] Patients have short stature, with limb shortening affecting more severely the proximal extremities. Short pedicles can predispose to spinal stenosis. Narrowing of the interpedicular width in the lower lumbar spine is seen, along with horizontally oriented acetabular roofs, small sciatic notches, and rounded, "ping-pong-paddle"-shaped iliac bones[78] (Fig. 57). Cervicomedullary compression has been shown to be associated with sudden death in infants with achondroplasia.[79]

Multiple Epiphyseal Dysplasia

In multiple epiphyseal dysplasia, the abnormal growth of the femoral head epiphysis typically leads to a varus alignment of the femoral neck. This occurs due to overgrowth of the trochanteric ossification center and infundibulum, a cartilaginous connection between the femoral head and trochanteric ossification centers in the infant[73] (Fig. 58). Secondary osteoarthritis eventually develops.

Proximal Focal Femoral Deficiency

Proximal focal femoral deficiency (PFFD) represents a congenital disorder characterized by varying severity of shortening and dysplasia of the femur and acetabulum, and varus angulation of the proximal femur[80] (Fig. 59). A common classification system divides the disorder into types, A-D, in increasing order of severity.[80] In type A, the femur is shortened compared with the normal size, but the femoral head is present and located within the acetabulum. In type B, the femur is short with a varus angulation, and there is a gap between the femoral head, which is located within the acetabulum, and the femoral neck. In type C, the femoral head is rudimentary or absent. The femur is markedly short, and the

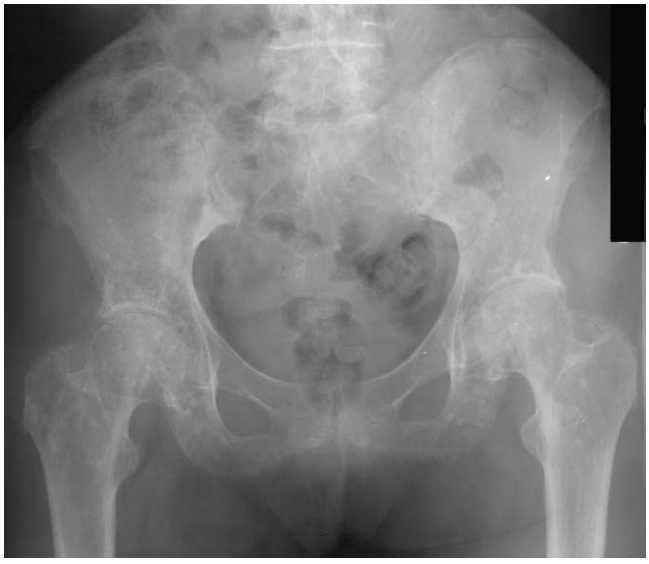


Figure 53 Punctate sclerotic lesions (bone islands) scattered diffusely bilaterally. Osteopoikilosis.

acetabulum is dysplastic. In type D, the entire femur is rudimentary, with absent femoral head and acetabulum.

Various treatments have been used for patients with this disorder. In one recent report, patients reported similar mobility and improved satisfaction with nonoperative treatment using extension prosthesis, compared with surgical ankle disarticulation with fitting of an above-knee prosthesis.[81]

Mucopolysaccharidoses

This represents a heterogeneous group of disorders characterized by accumulation of various mucopolysaccharides as a result of congenital lack of certain enzymes.[82] Many if not all of these exhibit similar radiographic findings in the pelvis, including flared and dysplastic femoral heads, narrowed and distorted pelves, and flared iliac wings[82] (Fig. 60).

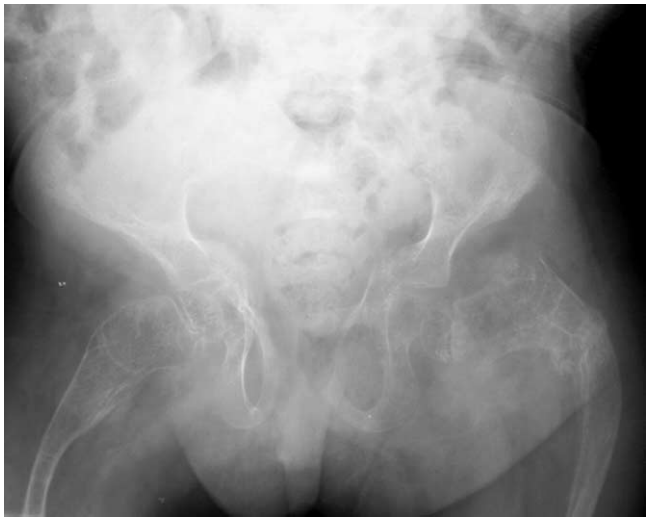


Figure 54 Severe deformity and osteopenia, with multiple fractures in different stages of healing. The pelvis is deformed and narrow. Osteogenesis imperfecta.

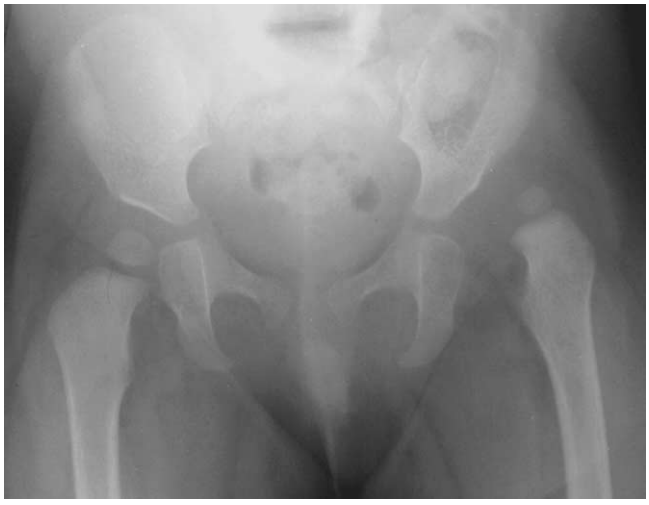


Figure 55 Superior dislocation of the left hip, with pseudoacetabulum formation. Mild subluxation also of the right hip.

Fibrodysplasia Ossificans Progressiva

Fibrodysplasia ossificans progressiva (FOP) represents a rare congenital disorder characterized by progressive heterotopic ossification of tendons, ligaments, muscles, and other soft tissues (Fig. 61) with deformity of the great toe.[83] No known treatment or preventive measure exists.[83] The typical course of the disease is progressive restriction of movement, frequent falls, and eventual respiratory difficulty from involvement of the chest wall. Most patients die of pulmonary complications in their 40s or 50s.[83] Recent advances include mapping of the gene for FOP to chromosome 4q, and identification of a key protein found in lesion cells and lymphocytes.[83] These findings may prove beneficial in treating this condition in the future.

Tumors

A variety of benign and malignant tumors may affect the pelvis and proximal femur. Close inspection for the presence

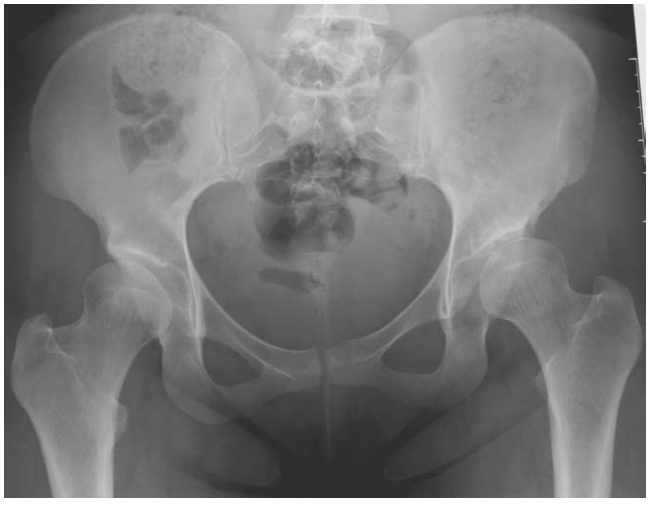


Figure 56 Bilateral acetabuli are dysplastic, with only partial covering of the femoral head. On the left, early osteoarthritis has begun to develop.

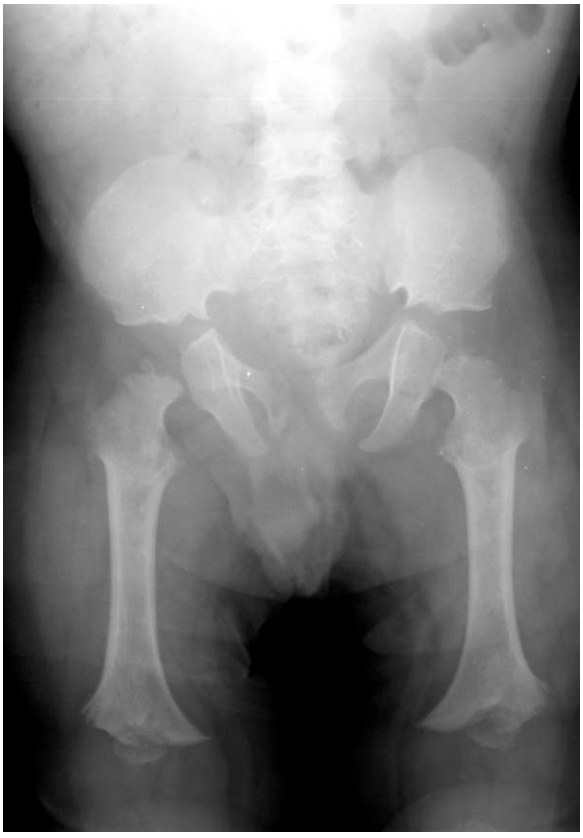


Figure 57 Characteristic findings of achondroplasia—narrowing of the interpedicular distance in the lower lumbar spine, flat acetabular roofs, small greater sciatic notches, and short femurs with widened femoral metaphyses.

of disruption or displacement of the anterior column, posterior column, cortex, sacral ala, or trabecular lines may reveal a subtle lesion. Although an in-depth discussion of all tumors that can affect the pelvis and proximal femur is beyond the scope of this article, a few common or characteristic lesions will be discussed.

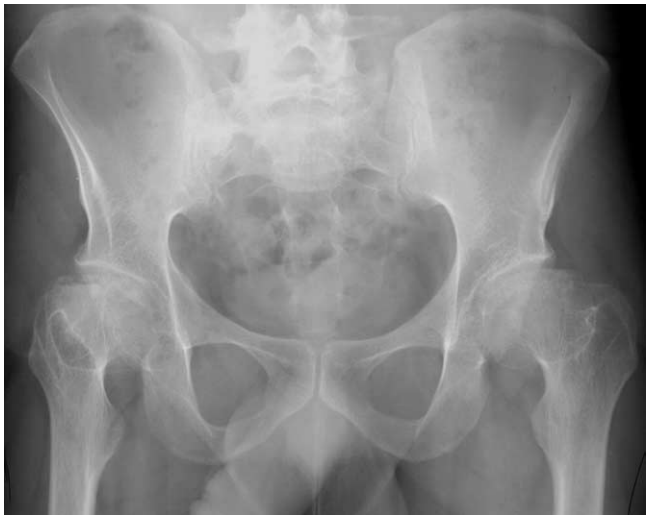


Figure 58 Broad, dysplastic femoral heads, with varus angulation of the femoral necks bilaterally. Multiple epiphyseal dysplasia.

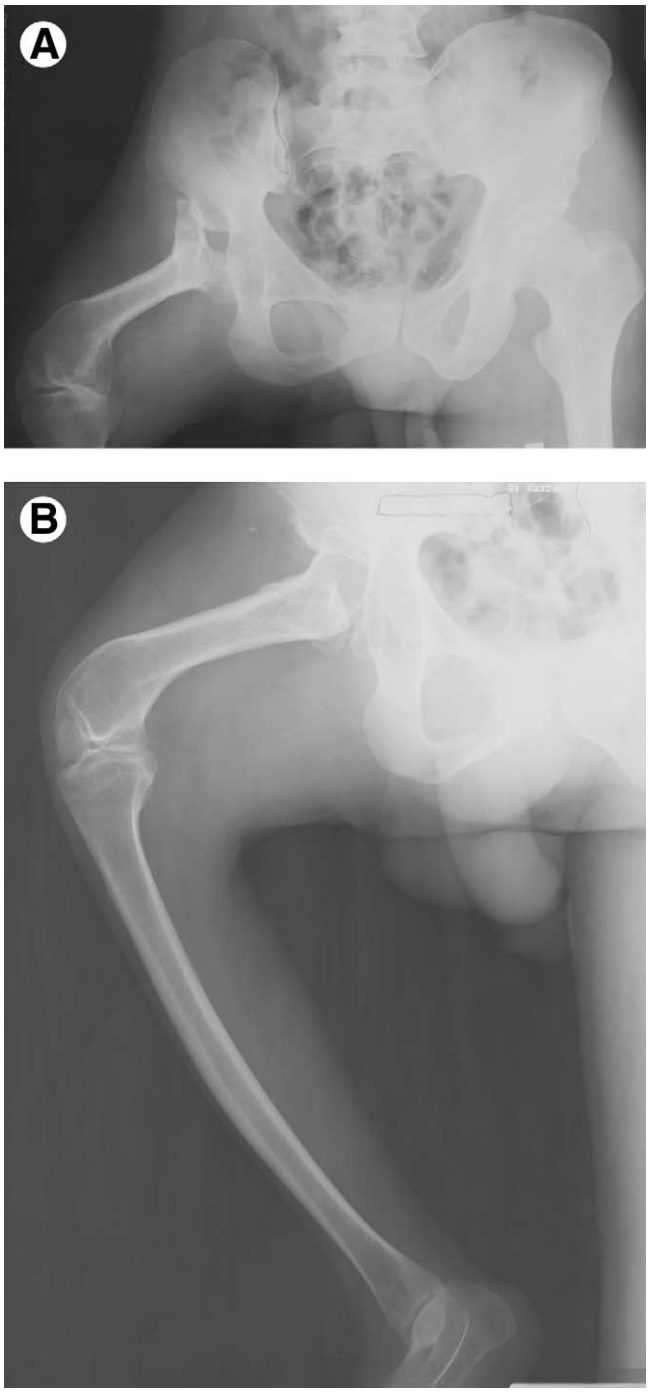


Figure 59 (A) and (B) The right femur is short and the femoral head is rudimentary. The right acetabulum is dysplastic. The fibula is absent. PFFD type C.

Myeloma

Myeloma is the most common primary bone tumor and is a malignancy of the bone marrow.[2] Most commonly affecting males over the age of 50 years, myeloma may be seen as a solitary plasmacytoma or multiple lesions in multiple myeloma.[2,84] The axial skeleton is most commonly affected, and the lesions are typically lytic[84] (Fig. 62). A small minority of

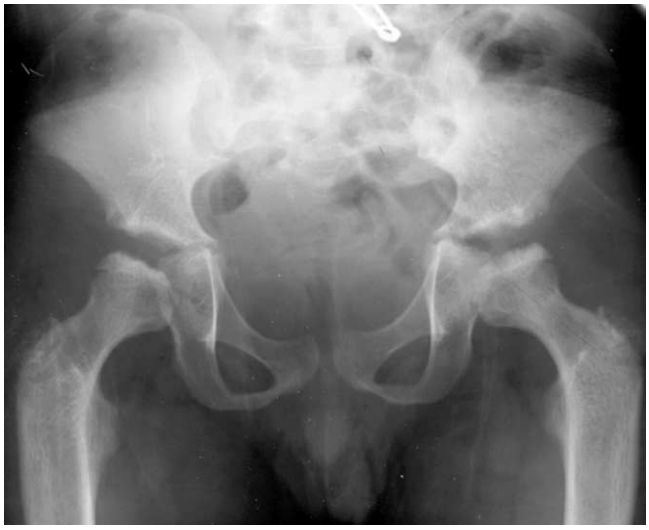


Figure 60 The iliac wings are broad, the pelvis narrow, and the femoral heads dysplastic in this patient with Hurler's syndrome.

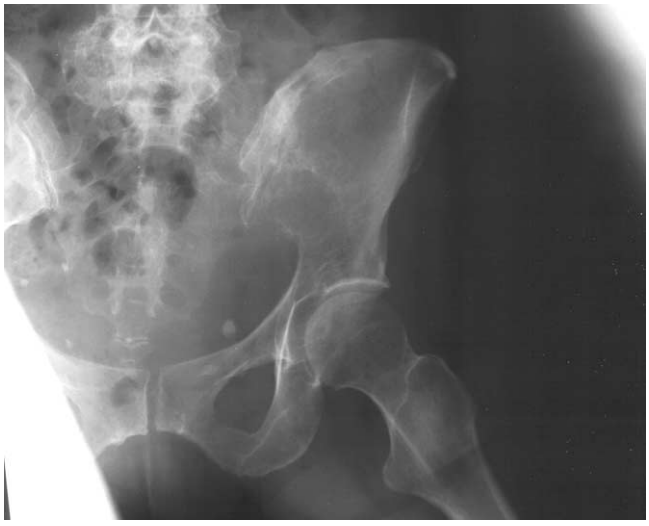


Figure 62 Destructive lesion without definable borders involving the sacrum and left ilium. Myeloma.

cases (less than 1%) may be sclerotic (Fig. 63), with nearly half of these developing peripheral neuropathies.[2,85]

Chondrosarcoma

The pelvis is a common site of involvement of chondrosarcoma, a malignant cartilage-forming tumor.[86] It most commonly affects patients between ages 30 and 60 years.[86] Chondrosarcoma may be primary (of which there are several types), or may arise secondarily in the setting of preexisting enchondromatosis, Paget disease, osteochondroma, or synovial chondromatosis.[2,86] Conventional chondrosarcoma appears radiographically as an expansile, lytic lesion with ring-, arc-, or popcorn-shaped internal calcifications (Fig. 64). There may be thickening or scalloping of the cortex, and there may be a soft-tissue mass. Metastases are uncommon.

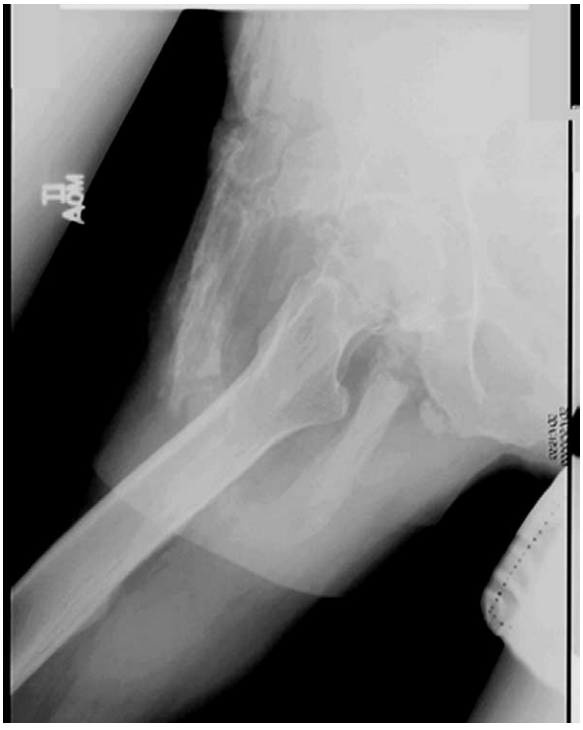


Figure 61 Marked heterotopic ossification bridging the hip joint medially and laterally. Fibrodysplasia ossificans progressiva.

Chordoma

In the differential diagnosis for a destructive lesion of the sacrum is a chordoma, a tumor arising from notochord remnants[86] (Fig. 12). Typically affecting patients over 40 years of age, the tumor is seen slightly more commonly in men. It is seen most commonly in the clivus, the sacrum, and the C2 vertebra, typically as a lytic lesion, with occasional calcifications in the matrix.[86]

Fibrous Dysplasia

Fibrous dysplasia is a benign tumor characterized by replacement of normal cancellous bone by fibroblasts and fibrous matrix, with interspersed trabeculae of immature woven bone.[87] Typically affecting patients under 30 years

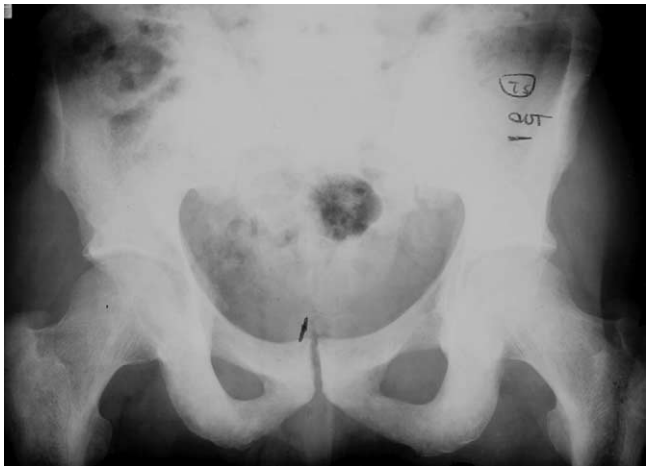


Figure 63 Diffuse sclerosis, an unusual manifestation of myeloma.

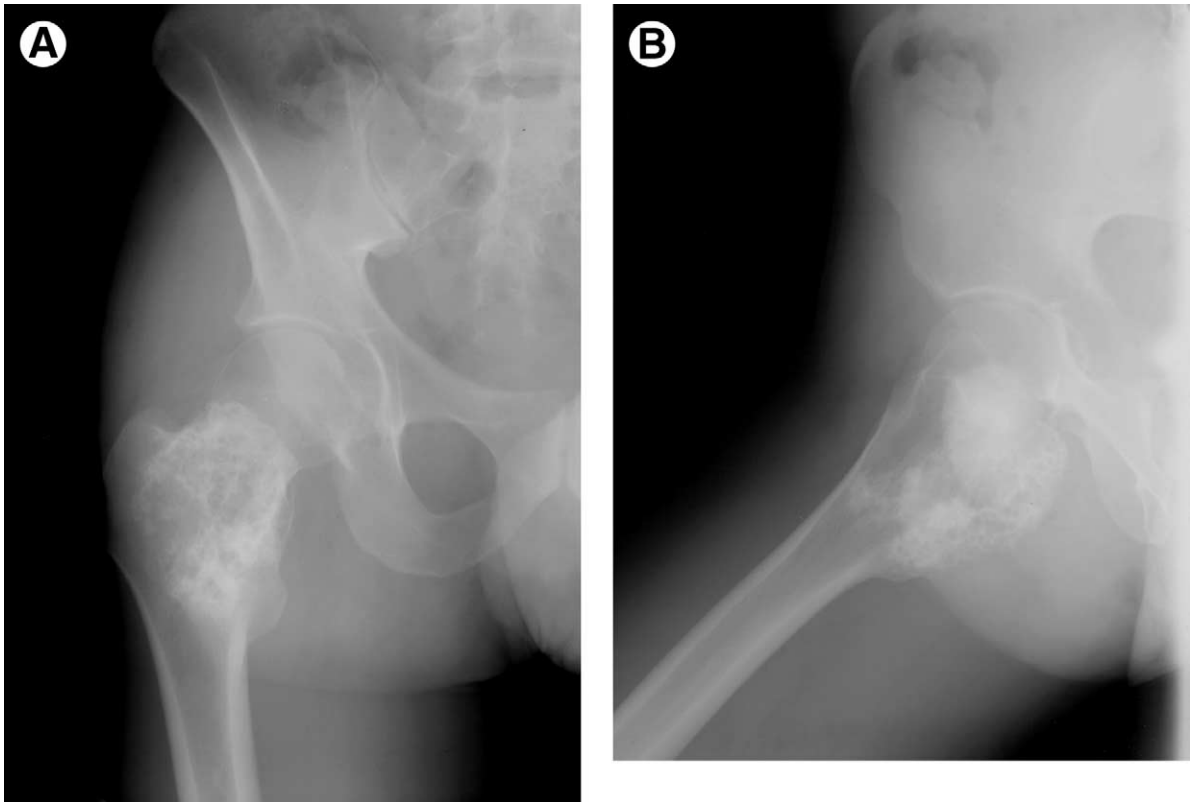


Figure 64 (A) and (B) Lucent and sclerotic lesion in the femoral neck, with narrow zone of transition, but with scalloping and probable disruption of the medial cortex. Punctate and curvilinear calcifications are seen internally. Chondrosarcoma.

of age, the lesion is centrally located within the bone, expansile, with a narrow zone of transition and internal hazy "ground-glass" appearance[87] (Fig. 65). Prominent trabeculae and a sclerotic margin may or may not be seen. Fibrous dysplasia most commonly affects the proximal femur, tibia, or humerus, but is also seen in the pelvis, ribs, and craniofacial bones.[87] Cystic or cartilaginous portions of the lesions may be seen.[87,88] A minority of affected

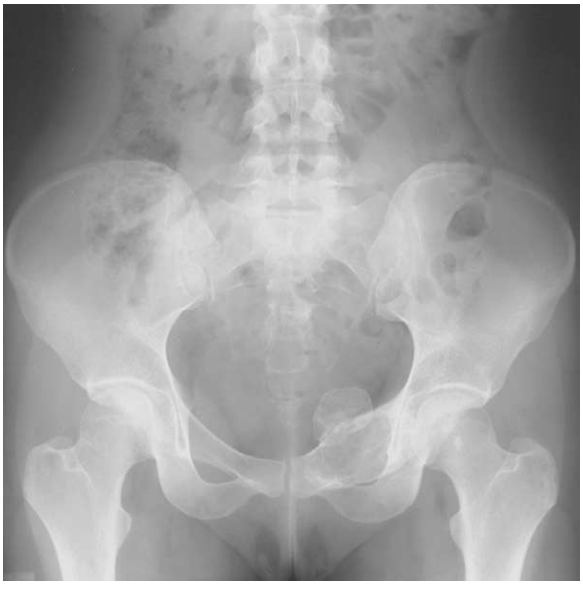


Figure 65 Geographic, expansile lucent lesion in the left superior pubic ramus is noted with internal ground-glass density and septations. The exophytic portion of the tumor is an unusual appearance of fibrous dysplasia. This patient had polyostotic fibrous dysplasia with endocrinopathy (Albright's syndrome).

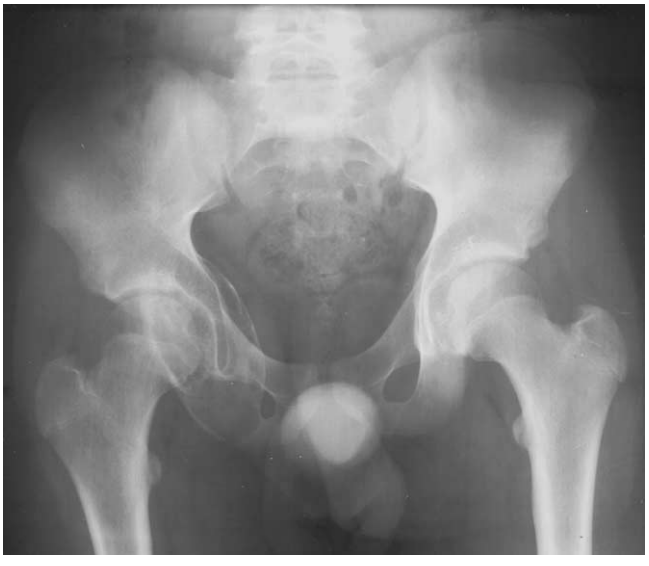


Figure 66 An aneurysmal bone cyst in the ischium displaces the contour of the posterior column. The lesion is expansile and lucent, with thinning of the cortex.

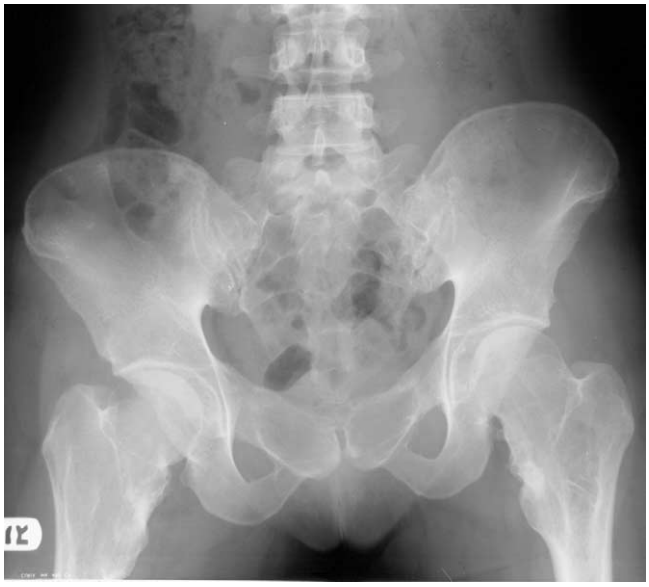


Figure 67 Deformity of the proximal femur and superior pubic ramus bilaterally due to osteochondromatosis, a characteristic appearance for hereditary multiple osteochondromatosis.

patients have polyostotic involvement, which may be accompanied by endocrine disturbances in McCune–Albright syndrome.[87]

Aneurysmal Bone Cyst

Aneurysmal bone cyst is a benign, expansile, lucent bone lesion, typically affecting patients between the ages of 10 and 30 years.[86] Believed to result from venous obstruction or vascular malformation in the bone, the internal architecture is composed of blood-filled cavities with intervening septae, although a solid variant has been described.[89] They occur most commonly about the knee, with the pelvis involved in 10 to 15% of cases.[86] They may arise from a preexisting benign or malignant lesion. Radiographically, aneurysmal bone cysts appear as lucent lesions with well-defined sclerotic borders, and often "ballooning" of the cortex (Fig. 66). On MR, fluid–fluid levels can be seen. After curettage, they frequently recur.

Osteochondroma

The most common benign bone lesion, osteochondroma, is an abnormal projection of bone with continuity of the cortex and medullary cavity with the underlying bone.[2,86] These lesions are typically located in the metaphysis, often demonstrating continuity with the growth plate. A cartilage cap is present, and through endochondral ossification, these lesions continue to enlarge until skeletal maturity.[86] Multiple lesions, often with deformity of involved bones, are seen in hereditary multiple exostoses (HME) (Fig. 67). Malignant transformation occurs in about 1% of solitary lesions, but 5 to 15% of cases with HME.[86]

Enchondromatosis

The pelvis and femur may be involved with enchondromatosis.[86] Deformity of involved bones and expansile, irregularly shaped lesions with internal dystrophic, ring- or arc-shaped calcifications are seen[86] (Fig. 68).

Neurofibromatosis

Neurofibromatosis is an autosomal-dominant, inherited dysplasia characterized by development of skin lesions, nerve tumors, and sarcomas.[90] Osseous involvement of the pelvis may demonstrate deformity of the pelvis or acetabulum, scoliosis, and chronic hip dislocations[90] (Fig. 69). In long bones,

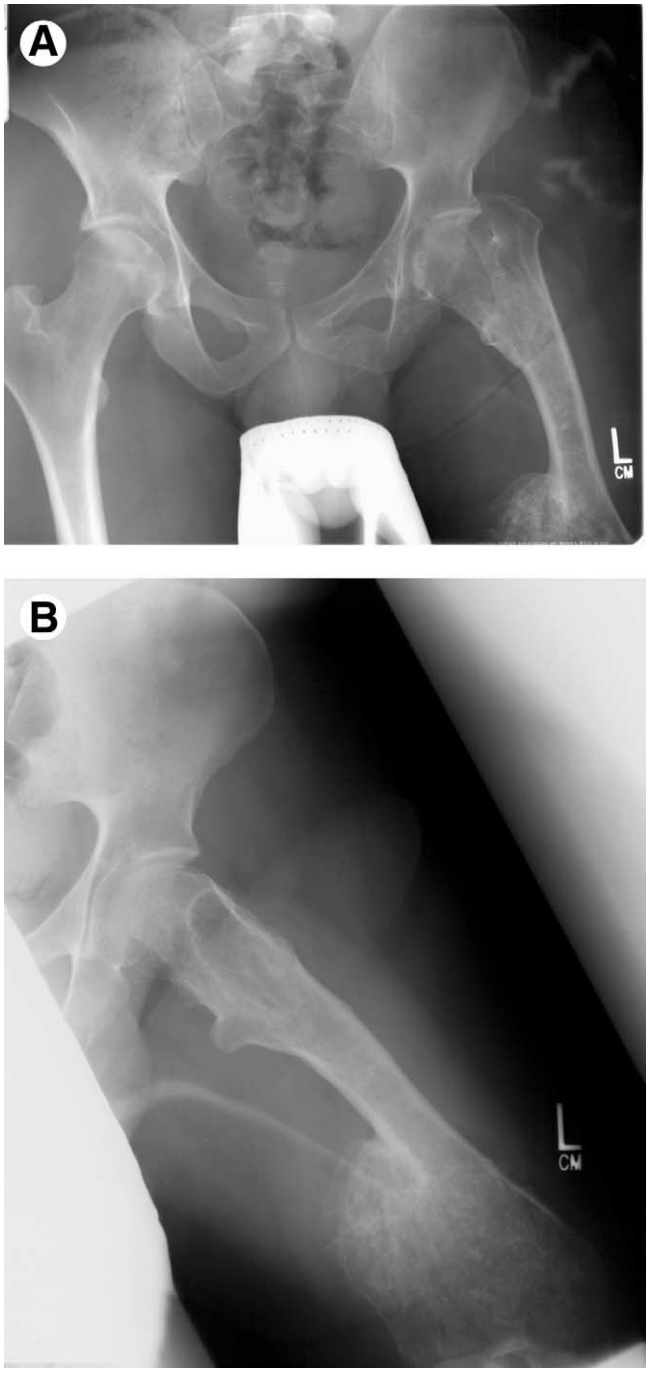


Figure 68 (A) and (B) The proximal and distal femur are markedly deformed and to a lesser extent the left ischium and inferior pubic ramus. Punctate and curvilinear calcifications may be seen in the exophytic lesion in the left distal femur. Ollier's disease.

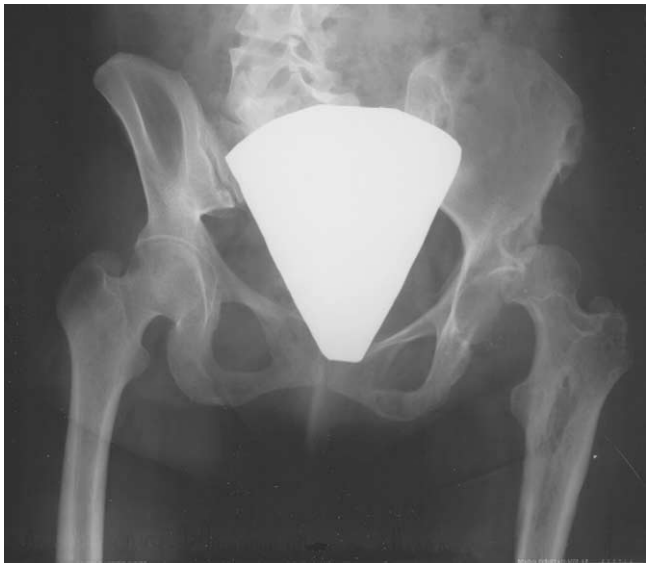


Figure 69 The left hip is chronically dislocated, with pseudoacetabulum formation. Scalloping of the femoral neck is also noted. The lower lumbar spine is scoliotic and deformed in this patient with neurofibromatosis type I.

such as the tibia, pseudarthroses, cortical erosions, nonossifying fibromas, and deformity may be seen.[2]

Simple Bone Cyst

Simple bone cyst is thought to be a localized disturbance of bone growth, and not a true neoplasm.[2] Most commonly affecting patients under 30 years of age, the etiology of simple bone cysts is unknown.[90] It affects the proximal femur and humerus most commonly, with less common sites including the ilium and calcaneus.[90] The lesion appears as a centrally located lucent lesion with sclerotic margins (Fig. 70). Osseous septae may be seen in the cyst, and a pathologic fracture may yield a "fallen fragment" sign of a bone fragment lying dependently in the cyst.[90]

Langerhans Cell Histiocytosis

A nonneoplastic proliferation of mononuclear cells, Langerhans cell histiocytosis most commonly affects children under 15 years of age.[85] Any bone can be affected, with the axial skeleton and long bones most commonly involved. A variety of radiographic appearances may be seen, varying from a sharply marginated lytic lesion to a lesion with a wide zone of transition and periosteal elevation (Fig. 71). In the spine, vertebral involvement can lead to collapse of the vertebra, yielding a vertebra plana appearance.[85]

Metastasis

Metastases are the most common malignant tumors of bone.[2,85] The axial skeleton is most commonly affected, as a result of hematogenous spread of malignancy. Many different radiographic appearances may be seen with metastatic disease, including sclerotic, lytic, or mixed sclerotic and lytic lesions (Fig. 72). A high index of suspicion should be maintained when evaluating a lesion in the pelvis. MR imaging, CT, or radionuclide bone scan may be helpful to characterize

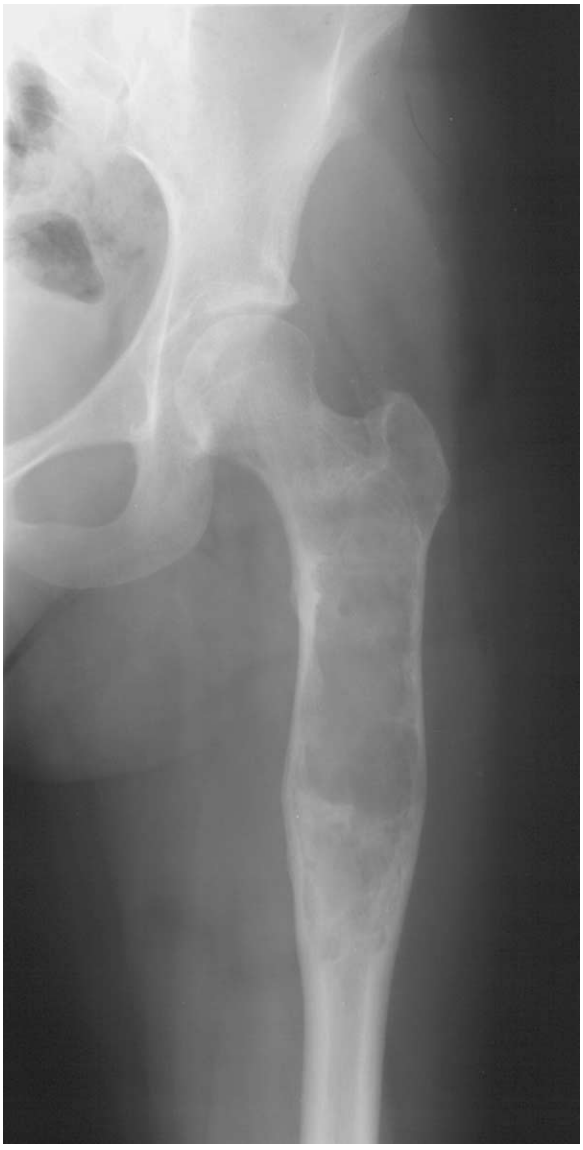


Figure 70 Mildly expansile lucent lesion of the proximal femoral shaft. Simple bone cyst.

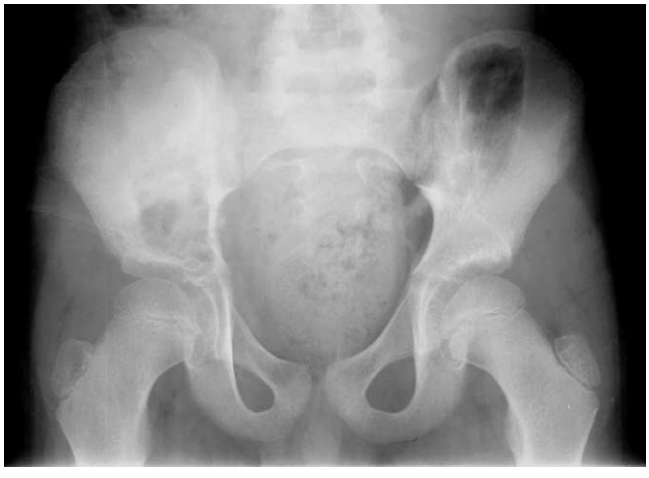


Figure 71 Lucent lesion in the right superior acetabulum is associated with periosteal elevation medially. Langerhans cell histiocytosis.

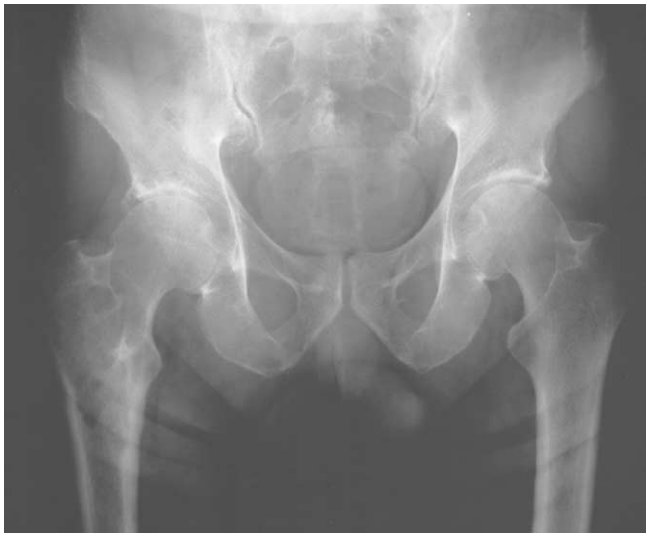


Figure 72 Lucent lesion in the femoral neck and proximal shaft without sclerotic rim, nonspecific appearance. Lung cancer metastasis.

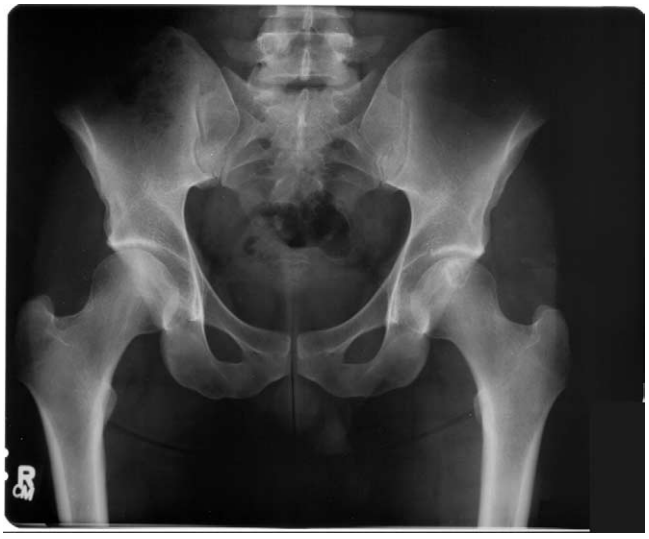


Figure 74 Small lucent lesion with sclerotic rim in the left femoral head. Chondroblastoma.

a lesion, look for a primary tumor, or evaluate for other metastatic lesions.

Giant Cell Tumor

Giant cell tumor is an abnormal proliferation of osteoclasts and stromal cells, typically located eccentrically in the ends of long bones (Fig. 73). It occurs most commonly in patients 20 to 40 years of age.[91] Typically an aggressive lesion, a sclerotic border is usually absent. About 5 to 10% of cases develop metastases, generally to the lungs, though no radiographic feature reliably distinguishes benign from malignant giant cell tumors.[91]

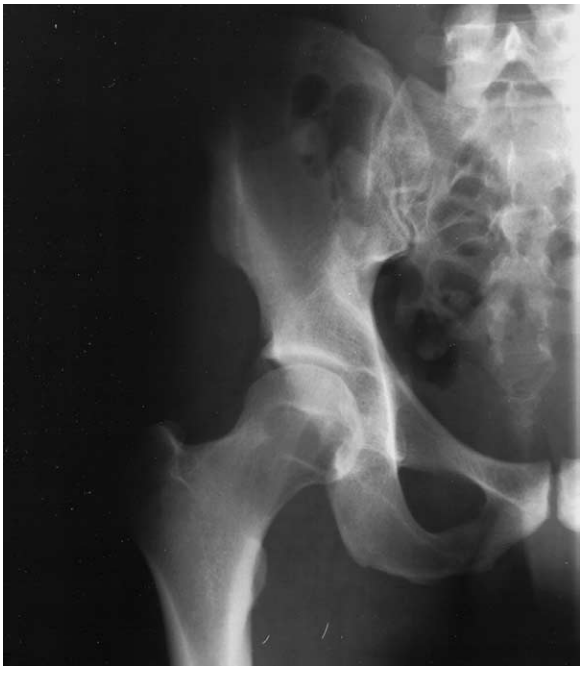


Figure 73 Eccentric, geographic lucent lesion in the proximal metaphysis and epiphysis of the right femur. The lesion appears to extend nearly to the inferomedial articular surface of the femoral head. Giant cell tumor.

Chondroblastoma

Chondroblastoma is a rare lesion but is in the differential diagnosis of an epiphyseal lesion in a skeletally immature patient.[92] Occurring most commonly in patients between 5 and 25 years of age, these lesions are typically found in the distal femur and proximal tibia, less commonly in the proximal femur. Radiographically, an eccentric lucent lesion with sclerotic border, at times extending through the physis into the metaphysis, may be seen[92] (Fig. 74).

Conclusion

Conventional radiography of the hip and pelvis are useful to demonstrate a broad spectrum of inherited and acquired disease. Accurate detection and classification of these abnormalities assist the clinician in treating and counseling the patient. Understanding of the radiographic anatomy and disease patterns seen in the pelvis and hip improves the radiologist's ability to make the correct diagnosis.

Acknowledgement

The author expresses appreciation to the Department of Radiology and Imaging at the Hospital for Special Surgery, for the use of images from their teaching file in preparing the manuscript.

References

1. Bergquist TH, Coventry MB: The pelvis and hips, in Berquist TH (ed): Imaging of Orthopedic Trauma and Surgery. Philadelphia, PA, WB Saunders, 1986, p. 181
2. Greenspan A: Lower limb I: pelvic girdle and proximal femur, in Greenspan A (ed): Orthopedic Radiology: A Practical Approach (ed 3). Philadelphia, PA, Lippincott, Williams, and Wilkins, 2000
3. Armbuster TG, Guerra J Jr, Resnick D, et al: The adult hip: an anatomic study. Part I: the bony landmarks. Radiology 128:1-10, 1978

4. Saks BJ: Normal acetabular anatomy for acetabular fracture assessment: CT and plain film correlation. Radiology 159:139-145, 1986
5. Johnson D, Williams A (eds): Gray's Anatomy (ed 39). London, UK, Churchill-Livingstone, 2004
6. Ponseti IV: Growth and development of the acetabulum in the normal child. Anatomical, histological, and roentgenographic studies. J Bone Joint Surg Am 60:575-585, 1978
7. Whitehouse RW: Paget's disease of bone. Semin Musculoskelet Radiol 6:313-322, 2002
8. Cundy T: Idiopathic hyperphosphatasia. Semin Musculoskelet Radiol 6:307-312, 2002
9. Bowerman JW, Sena JM, Chang R: The teardrop shadow of the pelvis; anatomy and clinical significance. Radiology 143:659-662, 1982
10. Deesomchok U, Tumrasvin T: Clinical comparison of patients with ankylosing spondylitis, Reiter's syndrome and psoriatic arthritis. J Med Assoc Thai 76:61-70, 1993
11. Stevens MA, El-Khoury GY, Kathol MH, et al: Imaging features of avulsion injuries. Radiographics 19:655-672, 1999
12. Sundar M, Carty H: Avulsion fractures of the pelvis in children: a report of 32 fractures and their outcome. Skeletal Radiol 23:85-90, 1994
13. Bui-Mansfield LT, Chew FS, Lenchik L, et al: Nontraumatic avulsions of the pelvis. AJR Am J Roentgenol 178:423-427, 2002
14. Dihlmann W, Tillmann B: Pericoxal fat stripes and the capsule of the hip joint. The anatomical-radiological correlations. Rofo Fortschr Geb Rontgenstr Neuen Bildgeb Verfahr 156:411-414, 1992
15. Eastridge BJ, Burgess AR: Pedestrian pelvic fractures: 5-year experience of a major urban trauma center. J Trauma 42:695-700, 1997
16. Burgess AR, Eastridge BJ, Young JW, et al: Pelvic ring disruptions: effective classification system and treatment protocols. J Trauma 30: 848-856, 1990
17. Young JW, Burgess AR, Brumback RJ, et al: Lateral compression fractures of the pelvis: the importance of plain radiographs in the diagnosis and surgical management. Skeletal Radiol 15:103-109, 1986
18. Judet R, Judet J, Letournel E: Fractures of the acetabulum: classification and surgical approaches for open reduction. Preliminary report. J Bone Joint Surg Am 46:1615-1646, 1964
19. Harris JH Jr, Coupe KJ, Lee JS, et al: Acetabular fractures revisited: part 2, a new CT-based classification. AJR Am J Roentgenol 182:1367-1375, 2004
20. Hougaard K, Thomsen PB: Traumatic posterior fracture-dislocation of the hip with fracture of the femoral head or neck, or both. J Bone Joint Surg Br 70:233-239, 1988
21. Oakes DA, Jackson KR, Davies MR, et al: The impact of the Garden classification on proposed operative treatment. Clin Orthop 409:232-240, 2003
22. Caviglia HA, Osorio PQ, Comando D: Classification and diagnosis of intracapsular fractures of the proximal femur. Clin Orthop 399:17-27, 2002
23. Blundell CM, Parker MJ, Pryor GA, et al: Assessment of the AO classification of intracapsular fractures of the proximal femur. J Bone Joint Surg Br 80:679-683, 1998
24. Fox KM, Magaziner J, Hebel JR et al: Intertrochanteric versus femoral neck hip fractures: differential characteristics, treatment, and sequelae. J Gerontol A Biol Sci Med Sci 54:M635-640, 1999
25. Daffner RH, Pavlov H: Stress fractures: current concepts. AJR Am J Roentgenol 159:245-252, 1992
26. Boles CA, el-Khoury GY: Slipped capital femoral epiphysis. Radiographics 17:809-823, 1997
27. Bloomberg TJ, Nuttall J, Stoker DJ: Radiology in early slipped femoral capital epiphysis. Clin Radiol 29:657-667, 1978
28. Stutz G, Kuster MS, Kleinstuck F, et al: Arthroscopic management of septic arthritis: stages of infection and results. Knee Surg Sports Traumatol Arthrosc 8:270-274, 2000
29. Volberg FM, Sumner TE, Abramson JS, et al: Unreliability of radiographic diagnosis of septic hip in children. Pediatrics 74:118-120, 1984
30. Milgram JW, Rana NA: Resection arthroplasty for septic arthritis of the hip in ambulatory and nonambulatory adult patients. Clin Orthop 272:181-191, 1991
31. Osman AA, Govender S: Septic sacroiliitis. Clin Orthop 313:214-219, 1995
32. Ford LS, Ellis AM, Allen HW, et al: Osteomyelitis and pyogenic sacroiliitis: a difficult diagnosis. J Paediatr Child Health 40:317-319, 2004
33. Malaviya AN, Kotwal PP: Arthritis associated with tuberculosis. Best Pract Res Clin Rheumatol 17:319-343, 2003
34. Ellis ME, el-Ramahi KM, al-Dalaan AN: Tuberculosis of peripheral joints: a dilemma in diagnosis. Tuber Lung Dis 74:399-404, 1993
35. Zvulunov A, Gal N, Segev Z: Acute hematogenous osteomyelitis of the pelvis in childhood: diagnostic clues and pitfalls. Pediatr Emerg Care 19:29-31, 2003
36. Conrozier T, Tron AM, Mathieu P, et al: Quantitative assessment of radiographic normal and osteoarthritic hip joint space. Osteoarthritis Cartilage 3:81-87, 1995 (suppl A)
37. Conrozier T, Merle-Vincent F, Mathieu P, et al: Epidemiological, clinical, biological and radiological differences between atrophic and hypertrophic patterns of hip osteoarthritis: a case-control study. Clin Exp Rheumatol 22:403-408, 2004
38. Avimadje AM, Pellieux S, Goupille P, et al: Destructive hip disease complicating traumatic paraplegia. Joint Bone Spine 67:334-336, 2000
39. O'Connor BL, Palmoski MJ, Brandt KD: Neurogenic acceleration of degenerative joint lesions. J Bone Joint Surg Am 67:562-572, 1985
40. Scutellari PN, Orzincolo C: Rheumatoid arthritis: sequences. Eur J Radiol 27:S31-38, 1998 (suppl 1)
41. Gusis SE, Riopedre AM, Penise O, et al: Protrusio acetabuli in seronegative spondyloarthropathy. Semin Arthritis Rheum 23:155-160, 1993
42. Puhakka KB, Melsen F, Jurik AG, et al: MR imaging of the normal sacroiliac joint with correlation to histology. Skeletal Radiol 33:15-28, 2004
43. Brower AC, Flemming DJ: Arthritis in Black and White (ed 2). Philadelphia, PA, Saunders, 1997
44. Pachman LM: Juvenile dermatomyositis. Pediatr Clin North Am 33: 1097-1117, 1986
45. Pachman LM: Juvenile dermatomyositis: immunogenetics, pathophysiology, and disease expression. Rheum Dis Clin North Am 28:579-602, 2002
46. Aldridge JM 3rd, Urbaniak JR: Avascular necrosis of the femoral head: etiology, pathophysiology, classification, and current treatment guidelines. Am J Orthop 33:327-332, 2004
47. Ficat RP: Idiopathic bone necrosis of the femoral head. Early diagnosis and treatment. J Bone Joint Surg Br 67:3-9, 1985
48. Thompson GH, Salter RB: Legg-Calve-Perthes disease. Current concepts and controversies. Orthop Clin North Am 18:617-635, 1987
49. Singh M, Nagrath AR, Maini PS: Changes in trabecular pattern of the upper end of the femur as an index of osteoporosis. J Bone Joint Surg Am 52:457-467, 1970
50. Koot VC, Kesselaer SM, Clevers GJ, et al: Evaluation of the Singh index for measuring osteoporosis. J Bone Joint Surg Br 78:831–814, 1996
51. Jevtic V: Imaging of renal osteodystrophy. Eur J Radiol 46:85-95, 2003
52. Reginato AJ, Coquia JA: Musculoskeletal manifestations of osteomalacia and rickets. Best Pract Res Clin Rheumatol 17:1063-1080, 2003
53. States LJ: Imaging of metabolic bone disease and marrow disorders in children. Radiol Clin North Am 39:749-772, 2001
54. Hardy DC, Murphy WA, Siegel BA, et al: X-linked hypophosphatemia in adults: prevalence of skeletal radiographic and scintigraphic features. Radiology 171:403-414, 1989
55. Whitehouse RW: Paget's disease of bone. Semin Musculoskelet Radiol 6:313-322, 2002
56. Rousiere M, Michou L, Cornelis F, et al: Paget's disease of bone. Best Pract Res Clin Rheumatol 17:1019-1041, 2003
57. Bhimani MA, Wenz JF, Frassica FJ. Pigmented villonodular synovitis: keys to early diagnosis. Clin Orthop 386:197-202, 2001
58. Crotty JM, Monu JU, Pope TL Jr: Synovial osteochondromatosis. Radiol Clin North Am 34:327-342, 1996
59. Kim SH, Hong SJ, Park JS, et al: Idiopathic synovial osteochondromatosis of the hip: radiographic and MR appearances in 15 patients. Korean J Radiol 3:254-259, 2002
60. Ea HK, Liote F: Calcium pyrophosphate dihydrate and basic calcium

phosphate crystal-induced arthropathies: update on pathogenesis, clinical features, and therapy. Curr Rheumatol Rep 6:221-227, 2004
61. Garcia GM, McCord GC, Kumar R: Hydroxyapatite crystal deposition disease. Semin Musculoskelet Radiol 7:187-193, 2003
62. Phornphutkul C, Introne WJ, Perry MB, et al: Natural history of alkaptonuria. N Engl J Med 347:2111-2121, 2002
63. de Vernejoul MC, Benichou O: Human osteopetrosis and other sclerosing disorders: recent genetic developments. Calcif Tissue Int 69:1-6, 2001
64. Vanhoenacker FM, De Beuckeleer LH, Van Hul W, et al: Sclerosing bone dysplasias: genetic and radioclinical features. Eur Radiol 10:1423-1433, 2000
65. Shapiro F: Osteopetrosis. Current clinical considerations. Clin Orthop 294:34-44, 1993
66. Karkabi S, Reis ND, Linn S, et al: Pyknodysostosis: imaging and laboratory observations. Calcif Tissue Int 53:170-173, 1993
67. Rozencwaig R, Wilson MR, McFarland GB Jr: Melorheostosis. Am J Orthop 26:83-89, 1997
68. Lagier R, Mbakop A, Bigler A: Osteopoikilosis: a radiological and pathological study. Skeletal Radiol 11:161-168, 1984
69. Mungovan JA, Tung GA, Lambiase RE, et al: Tc-99m MDP uptake in osteopoikilosis. Clin Nucl Med 19:6-8, 1994
70. Rauch F, Glorieux FH. Osteogenesis imperfecta. Lancet 363:1377-1385, 2004
71. Sillence DO, Senn A, Danks DM: Genetic heterogeneity in osteogenesis imperfecta. J Med Genet 16:101-116, 1979
72. Roughley PJ, Rauch F, Glorieux FH: Osteogenesis imperfecta—clinical and molecular diversity. Eur Cell Mater 5:41-47, 2003
73. Weinstein SL, Mubarak SJ, Wenger DR: Developmental hip dysplasia and dislocation: Part I. AAOS Instruc Course Lecture 53:523-530, 2004
74. Weinstein SL, Mubarak SJ, Wenger DR: Developmental hip dysplasia and dislocation: Part II. AAOS Instruc Course Lecture 53:531-542, 2004
75. Cooperman DR, Wallensten R, Stulberg SD: Acetabular dysplasia in the adult. Clin Orthop 175:79-85, 1983
76. Lavigne M, Parvizi J, Beck M, et al: Anterior femoroacetabular impingement: part I. Techniques of joint preserving surgery. Clin Orthop 418: 61-66, 2004
77. Beck M, Leunig M, Parvizi J, et al: Anterior femoroacetabular impingement: part II. Midterm results of surgical treatment. Clin Orthop 418:67-73, 2004
78. Lemyre E, Azouz EM, Teebi AS, et al: Bone dysplasia series. Achondroplasia, hypochondroplasia and thanatophoric dysplasia: review and update. Can Assoc Radiol J 50:185-197, 1999
79. Keiper GL Jr, Koch B, Crone KR: Achondroplasia and cervicomedullary compression: prospective evaluation and surgical treatment. Pediatr Neurosurg 31:78-83, 1999
80. Anton CG, Applegate KE, Kuivila TE, et al: Proximal Femoral Focal Deficiency (PFFD): more than an abnormal hip. Semin Musculoskelet Radiol 3:215-226, 1999
81. Kant P, Koh SH, Neumann V, et al: Treatment of longitudinal deficiency affecting the femur: comparing patient mobility and satisfaction outcomes of Syme amputation against extension prosthesis. J Pediatr Orthop 23:236-242, 2003
82. Chen SJ, Li YW, Wang TR, et al: Bony changes in common mucopolysaccharidoses. Zhonghua Min Guo Xiao Er Ke Yi Xue Hui Za Zhi 37:178-184, 1996
83. Mahboubi S, Glaser DL, Shore EM, et al: Fibrodysplasia ossificans progressiva. Pediatr Radiol 31:307-314, 2001
84. Chang MY, Shih LY, Dunn P, et al: Solitary plasmacytoma of bone. J Formos Med Assoc 93:397-402, 1994
85. Michel JL, Gaucher-Hugel AS, Reynier C, et al: POEMS syndrome: imaging of skeletal manifestations, a study of 8 cases. J Radiol 84:393-397, 2003
86. Unni KK: Dahlin's Bone Tumors: General Aspects and Data on 11,087 Cases (ed 5). Philadelphia, PA, Lippincott-Raven Publishers, 1996, pp 291-390
87. Fitzpatrick KA, Taljanovic MS, Speer DP, et al: Imaging findings of fibrous dysplasia with histopathologic and intraoperative correlation. AJR Am J Roentgenol 182:1389-1398, 2004
88. Hermann G, Klein M, Abdelwahab IF, et al: Fibrocartilaginous dysplasia. Skeletal Radiol 25:509-511, 1996
89. Haga N, Nakamura S, Taniguchi K, et al: Pathologic dislocation of the hip in von Recklinghausen's disease: a report of two cases. J Pediatr Orthop 14:674-676, 1994
90. Ahn JI, Park JS: Pathological fractures secondary to unicameral bone cysts. Int Orthop 18:20-22, 1994
91. Tunn PU, Schlag PM: Giant cell tumor of bone. An evaluation of 87 patients. Z Orthop Ihre Grenzgeb 141:690-698, 2003
92. Ramappa AJ, Lee FY, Tang P, et al: Chondroblastoma of bone. J Bone Joint Surg Am 82-A:1140-1145, 2000