Osteoarthritis

10

Alfred Cividino and John O'Neill

Overview

Osteoarthritis (OA), also termed degenerative joint disease, is the most common arthritis. There are two main forms, primary and secondary. *Primary OA* is an age-related disease with a genetic susceptibility in some individuals to earlier onset and more rapid progression. It occurs in joints without local predisposing factors. Primary OA may be localized or generalized. OA is a complex interaction of advancing age, genetic predisposition, mechanical stress, obesity, as well as metabolic and biochemical factors all of which may affect the degree, extent, and progression of disease. Obesity is the only modifiable risk factor. Primary OA increases significantly in prevalence after the age of 50 with the majority of individuals demonstrating some form of OA after the age of 70 years.

Secondary OA on the other hand occurs in abnormal joints. Predisposing conditions include but are not limited to traumatic joint injuries, inflammatory arthropathies, CPPD, prior septic arthritis, congenitally abnormal joints such as congenital hip dysplasia, metabolic- and endocrine-related arthropathies, and avascular necrosis.

Inflammatory markers are usually normal, and there is no increased prevalence of rheumatoid factor positivity above the general population. *Erosive OA* is a subset of primary OA and is also termed inflammatory OA. Middle-aged and elderly women are predominantly affected with a male:female ratio of 1:12. It usually presents with abrupt onset with pain and swelling of the hands and is discriminated by the development of central erosions. The pathological changes of OA include degradation and loss of articular cartilage, sclerosis and remodeling of subchondral bone, development of osteophytes, and synovial inflammation

Presentation

Clinical features include joint pain worse with use, mild stiffness often worse with immobility, pain on movement, restricted range of motion, periarticular tenderness, bony enlargement, soft tissue swelling, and joint crepitus. The joints

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most frequently affected include the knees, hips, feet, hands, and cervical and lumbar spine. The diagnosis is usually clinical, and further investigations are usually not required. Radiographs can confirm the diagnosis and assess the degree and extent of the disease. The characteristic features of hand OA include the development of deformity with bony enlargement of the DIP and PIP joints with so-called Heberden's and Bouchard's nodes, respectively, and squaring of the first carpometacarpal phalangeal joint. Though uncommon the second and third MCP joint can be affected. Hemochromatosis may be suspected in these cases.

Erosive inflammatory OA often presents with joint swelling in a typical OA distribution however clinically resembles RA or PsA. Knee osteoarthritis is insidious and associated with pain on weight bearing. Joint swelling, flexion contracture, and varus or valgus deformities can develop. Hip osteoarthritis results in groin pain radiating down the anterior thigh to the knee, frequently insidious, and associated with weight bearing. Referred back pain may mimic these findings. OA of the foot typically affects the first metatarsophalangeal joint with accompanying hallux valgus and bony enlargement. Spinal disease is reviewed in detail in Chap. 15.

Imaging Features

Key radiographic features of osteoarthritis include joint space narrowing, osteophytosis, altered bone contour, bone sclerosis and cysts, periarticular calcification, and soft tissue swelling (Fig. 10.1). Additional imaging descriptions have been provided in Chap. 3.

The subchondral region bone plate is located deep to the articular surface, separated only by a thin layer of calcified articular cartilage (Fig. 10.2). When there is loss of the overlying cartilage, as in osteoarthritis, the subchondral bone is directly exposed to the stresses across the joint with subsequent trabecular collapse, flattening and *eburnation* (Fig. 10.3). Cartilage loss is discussed in greater detail in Chap. 3 and the Outerbridge classification of cartilage loss is outlined (see Table 3.5). The subchondral regions in areas of lower stress become increasingly vascularized. This stimulates endochondral ossification with new bone formation, *osteophytes* (Fig. 10.4). They are usually marginally growing as an extension of the joint margin. New bone formation may also occur centrally in areas of full cartilage loss and appear as new bone formation at the cortical surface, often with an irregular margin.

Subchondral cysts develop between the deformed trabeculae in areas of eburnation (Fig. 10.5). They are of variable size. The overlying cortex may be intact or may have a focal defect allowing communication of the cyst with the joint space. Prominent subchondral cyst formation raises possibility of underlying crystal disease. Joint space loss has long been attributed to cartilage thickness; however, in the knee, meniscal degenerative changes can also contribute to narrowing. Marked joint space narrowing is seen in advanced disease (Fig. 10.6).

On *radiographs* eburnation is noted as subchondral sclerosis and may demonstrate flattening and collapse of its surface. Osteophytes are noted as new bone forming at the periphery of the joint space. Subchondral cysts are variable in size, have a thin sclerotic margin, and may demonstrate communication with the joint space, e.g., intra-articular gas may extend into the cyst. The overlying cortex is usually intact or demonstrates a focal defect. Occasionally it may be difficult to differentiate a subchondral cyst from an erosion if there is collapse of the cyst overlying cortical margin. Subchondral cysts are however associated with joint space loss, subchondral sclerosis, and osteophytosis. Additional studies other than radiographs are rarely required, and the above changes however can be detailed on other imaging modalities when performed for alternative indications.

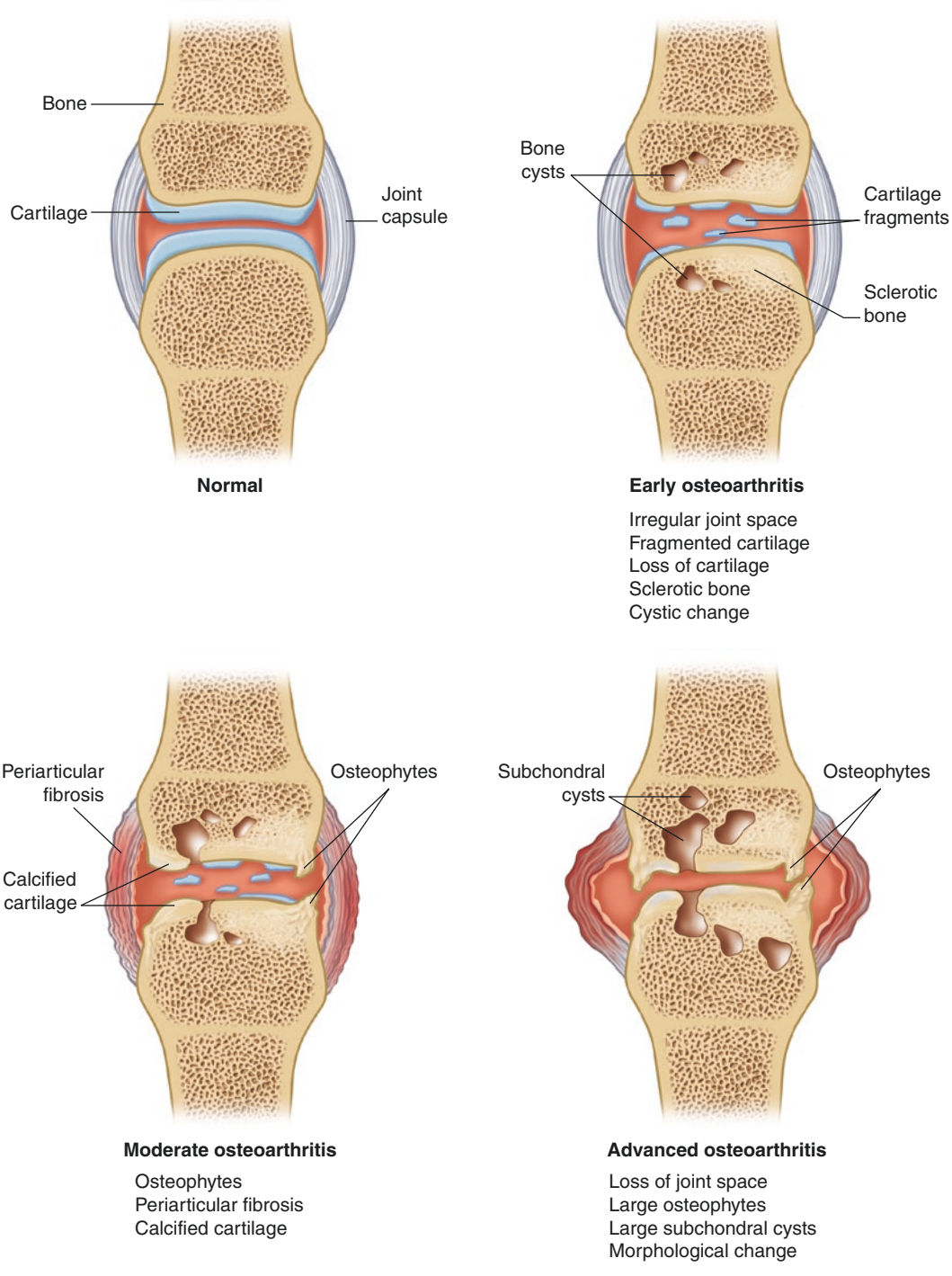

Fig. 10.1 Development and progression of osteoarthritis

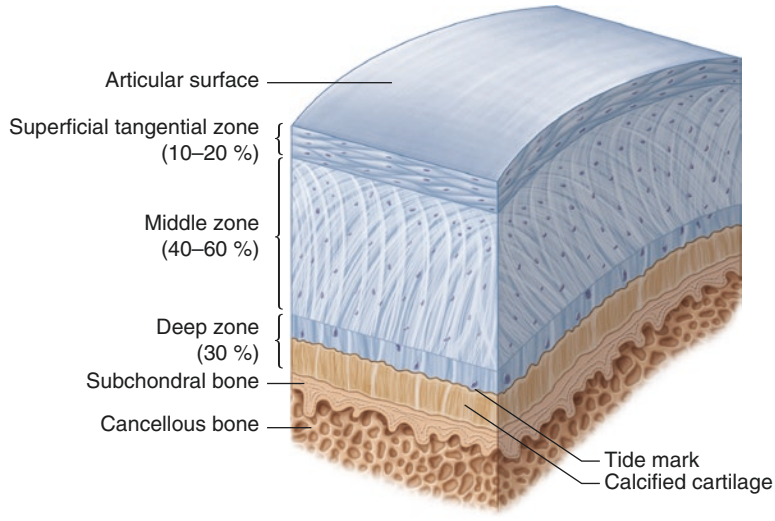

Fig. 10.2 Cartilage layers and subchondral bone

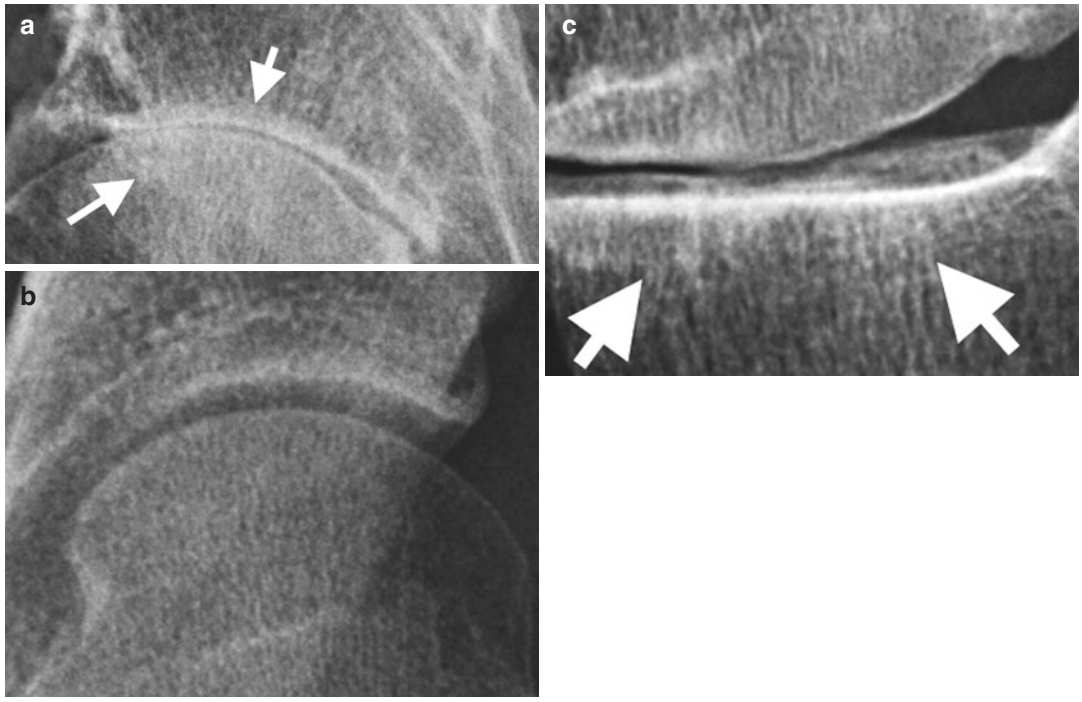

Fig. 10.3 Eburnation (*arrows*) on radiograph, (**a**) magnified AP subchondral region hip joint demonstrating increased subchondral sclerosis on both sides joint, (**b**) normal example for reference, (**c**) similar sclerosis secondary to degenerative disease medial tibiofemoral joint space

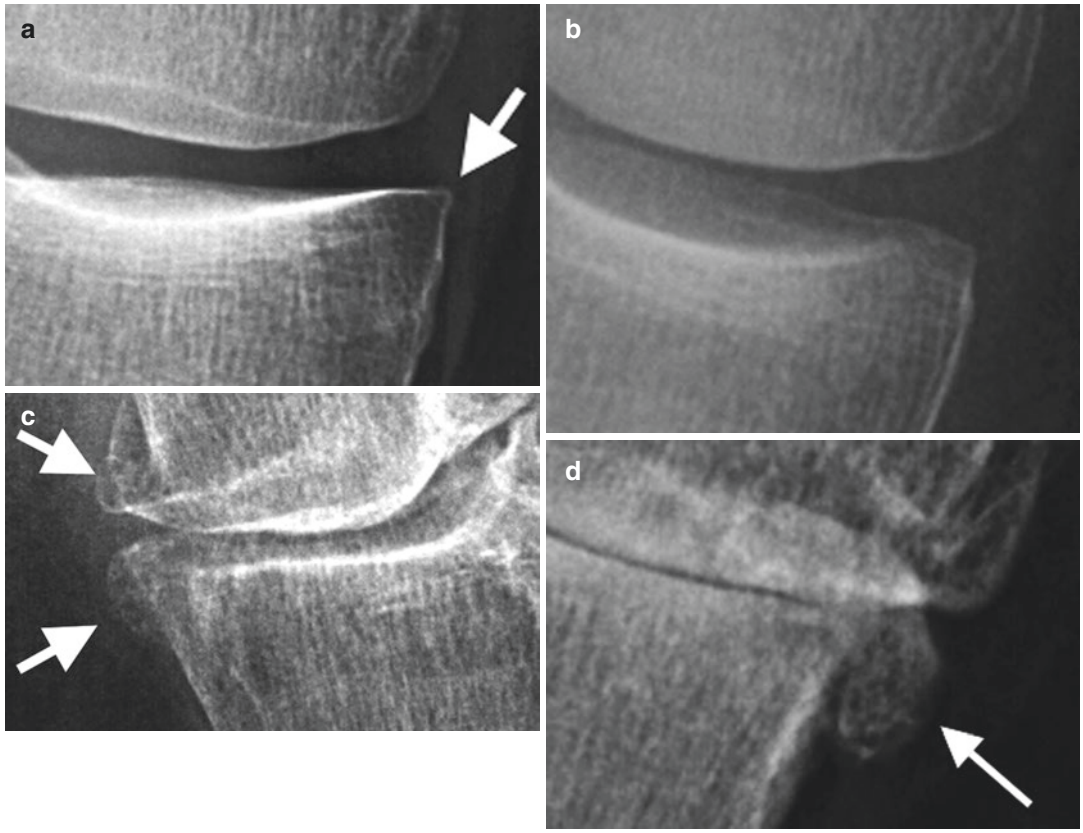

Fig. 10.4 Osteophyte on radiograph, (**a**) early osteophyte formation medial margin of the medial tibial plateau, (**b**) normal example for reference, (**c**) moderate and (**d**) large osteophytes (*arrows*)

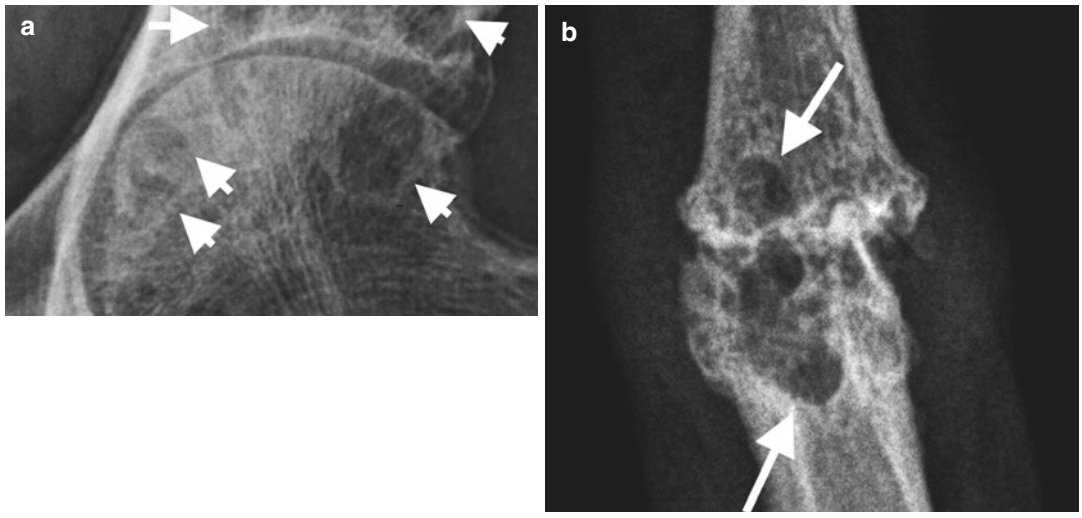

Fig. 10.5 Subchondral cysts (*arrows*) on radiographs, (**a**) well-defined subchondral lucencies with well-defined sclerotic margins in a degenerative hip joint (normal example for reference, see Fig. 10.3b), (**b**) large subchondral cysts in a degenerative PIPJ hand

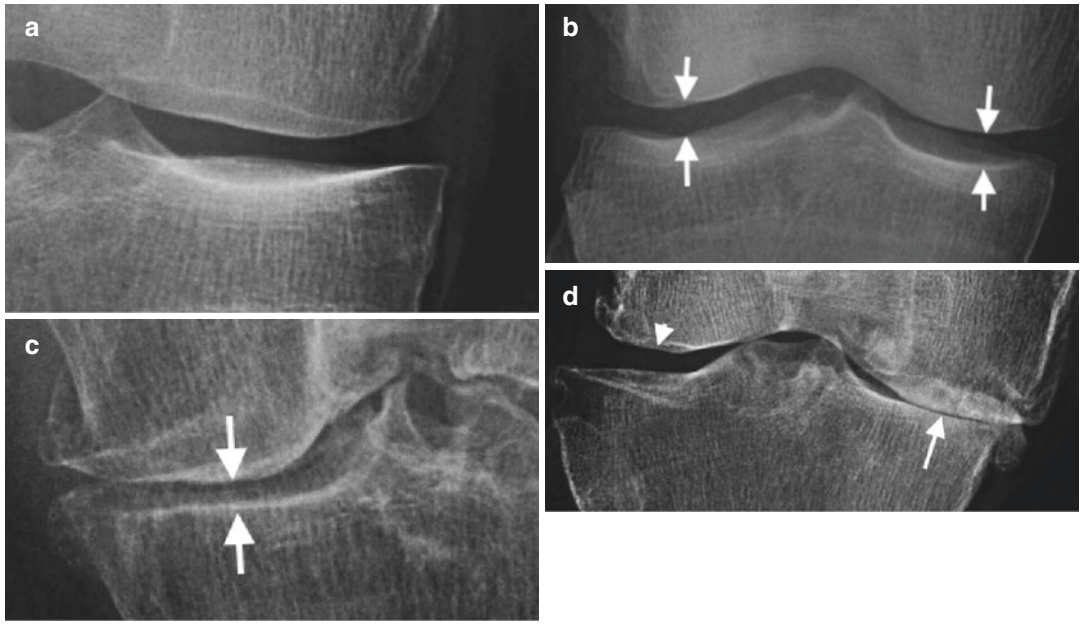

Fig. 10.6 Joint space (*arrows*) loss on radiographs. (**a**) Mild medial tibiofemoral joint space loss, (**b**) normal example for reference, (**c**) moderate to severe, and (**d**) severe, almost complete, joint space loss medially; note maintained lateral tibiofemoral joint space (*arrowhead*). True joint space loss is best assessed on standing for weight-bearing joints

CT demonstrates exquisite detail of subchondral changes as described in the above radiographic findings; however, it is rarely required in the assessment of OA. CT is occasionally used for assessment of intra-articular loose bodies and residual bone mass, e.g., glenoid, prior to joint replacement (Fig. 10.7).

Subchondral sclerosis on *MRI* is of low SI on both T1- and T2-weighted sequences. Subchondral cysts may contain proteinaceous material or joint fluid if they communicate with the joint and are of high SI on T2 and usually low SI on TI. MRI is the gold imaging standard in assessing cartilage. Cartilage abnormalities include changes in signal intensity, fibrillation cartilage surface, fissuring eventually extending full-depth cartilage, and areas of full-thickness cartilage loss (Fig. 10.8). These are often the sites of associated subchondral cyst formation and bone marrow edema. MRI of joints also allows visualization of the soft tissues including ligaments, capsule, synovium, and bone marrow changes. It should be stressed however that although MRI is useful in detecting lesions that are not possible with radiographs, they may not add to the clinical picture when plain radiographs demonstrate osteoarthritic changes. Further imaging beyond radiographs in clinical practice should be considered when the results will affect the management of the patient.

There is an array of studies using MRI as a semi- and quantitative imaging tools in the assessment of osteoarthritis. These studies are focused predominantly on the knee, hand, and hip. The required high-resolution imaging required for detailed cartilage review is mainly confined to research studies at this time due both to the extended time required to acquire and interpret

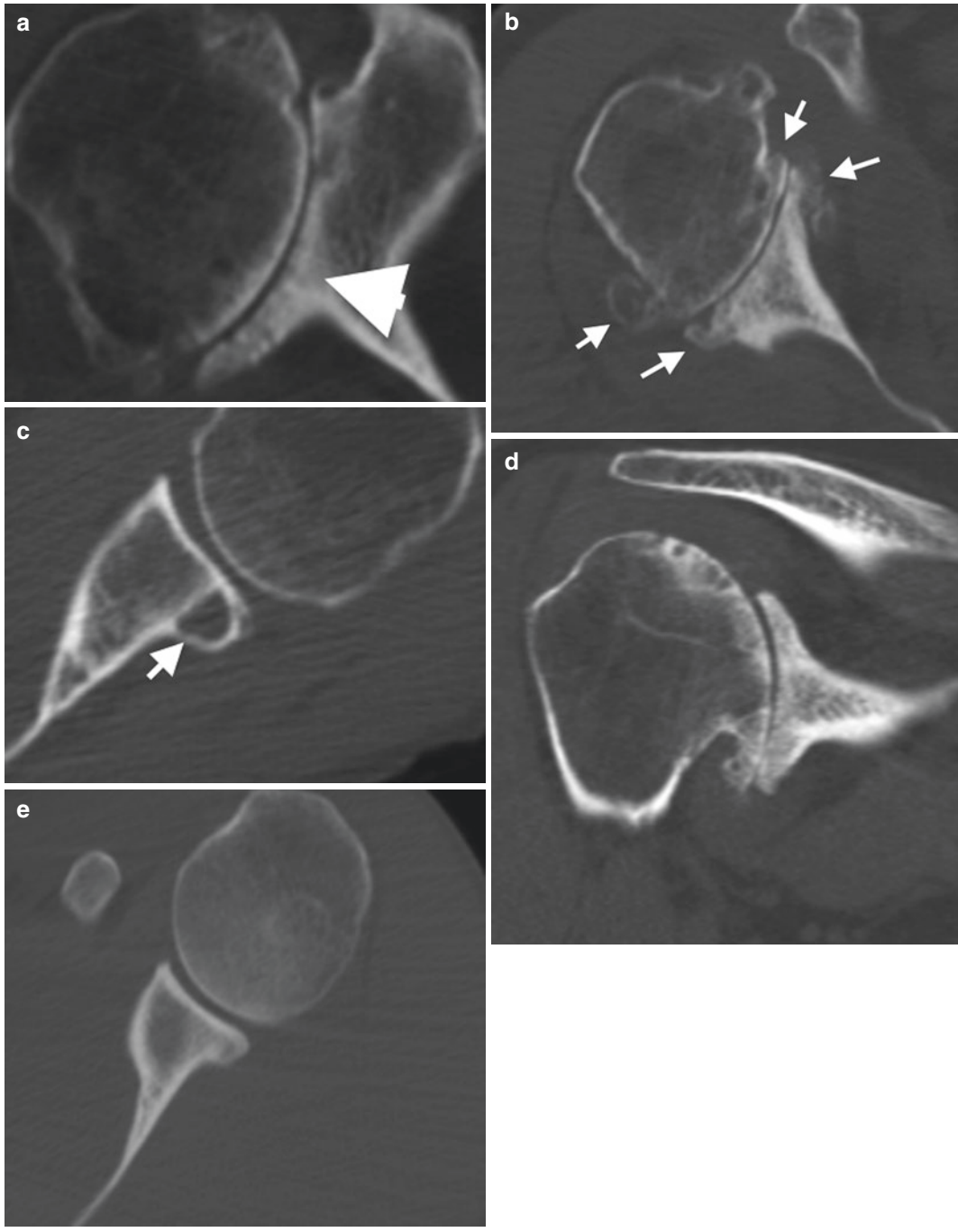

Fig. 10.7 CT secondary degenerative joint disease on the right shoulder. (**a**) Subchondral sclerosis/eburnation (*arrowhead*); (**b**) moderate osteophytosis (*arrows*); (**c**) subchondral cyst glenoid with well-defined sclerotic margins (*arrow*); (**d**) reformatted coronal image with joint space loss, subchondral sclerosis, osteophytes, and subchondral cysts; (**e**) normal axial CT for reference

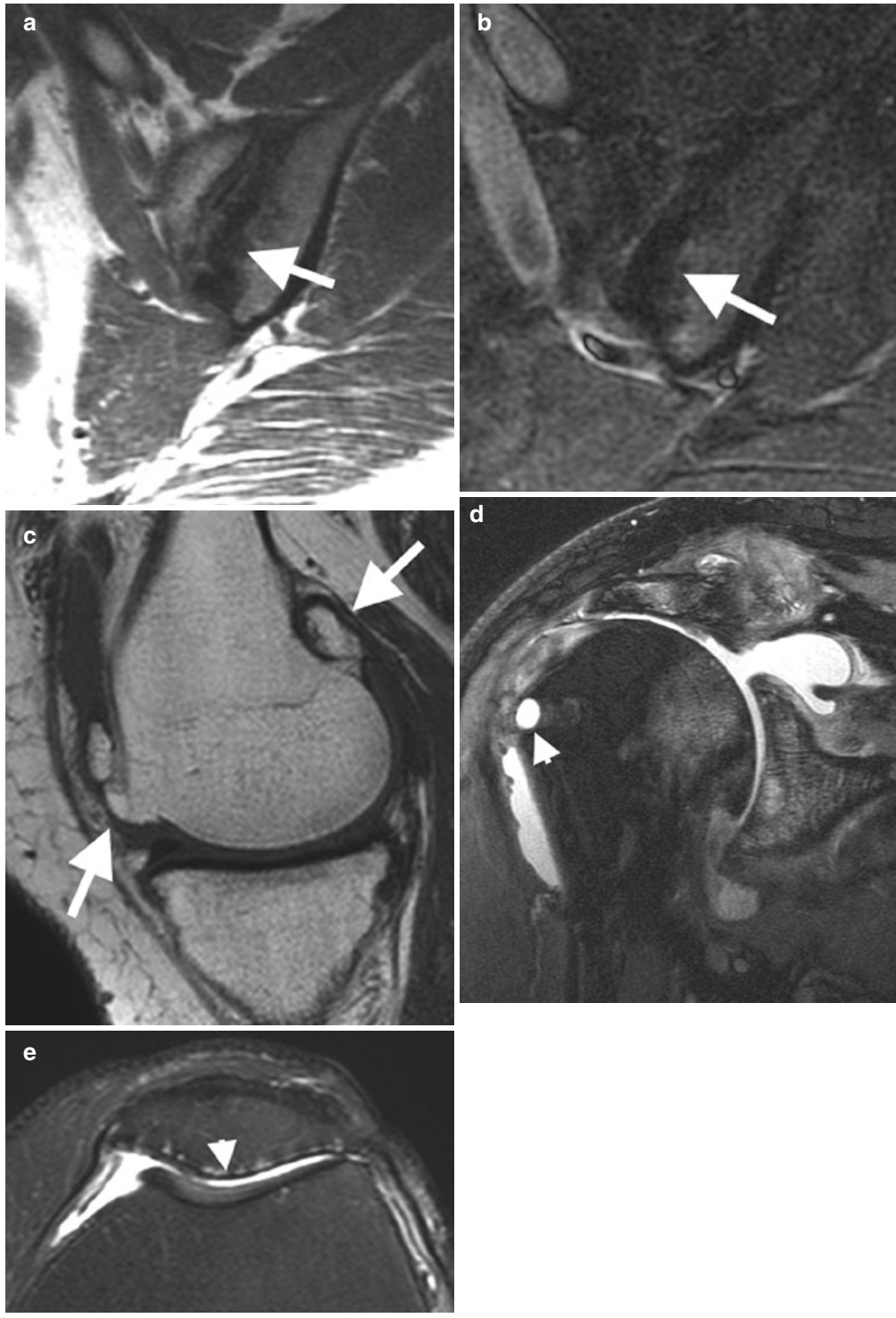

Fig. 10.8 MRI degenerative disease. (**a**) Subchondral sclerosis (low SI on all imaging sequences) left sacroiliac joint in patient with osteitis condensans ilii (OCI) with subchondral low signal (*arrows*) on Cor T1 and (**b**) Cor T2FS. (**c**) Moderate osteophytosis in medial femoral condyle on sagittal T1 (*arrowheads*), (**d**) rotator cuff arthropathy on the right shoulder with joint space loss, complete cartilage loss, subchondral bone marrow edema, and early subcortical cyst formation on Cor T2FS (*arrowhead*) low SI sclerotic rim on the greater tuberosity, (**e**) cartilage loss, full-thickness patellar aspect patellofemoral joint (*arrowhead*) with mild subchondral edema

these sequences and whether these sequences affect clinical management beyond currently performed sequences. *Ultrasound* is used predominantly for assessment of joint fluid and synovitis. Ultrasound can also demonstrate associated changes such as joint space loss, cartilage thinning and loss in the periphery of superficial joints, cortical irregularity, and osteophytosis. Nuclear medicine is generally not indicated.

The description of osteoarthritis will be subdivided into primary OA of large and small joints with typical examples provided.

Osteoarthritis: Small Joints

Osteoarthritis of the *hand* will be used to demonstrate the typical changes within small joints. The proximal and distal interphalangeal joints and the first carpometacarpal joint are the most commonly affected small joints. The PIP and DIP joints may demonstrate a symmetrical appearance in joints involved and the degree of osteoarthritis (Fig. 10.9). Localized bony outgrowths from the DIP and PIP joints are termed Heberden's and Bouchard's nodes, respectively. The joint

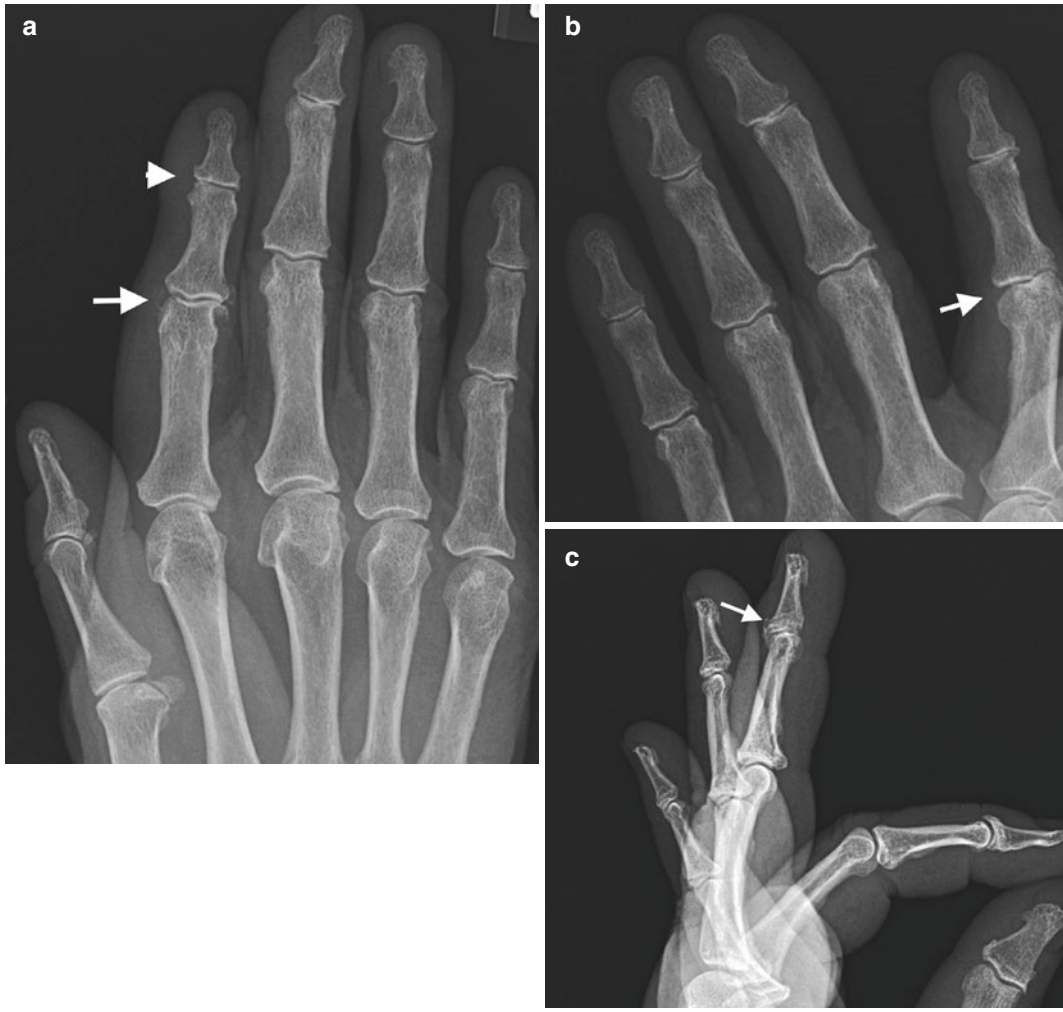

Fig. 10.9 Osteoarthritis PIP and DIPJs. (**a**) AP radiograph (magnified) demonstrating bony outgrowths at the second PIPJ (*arrow*) and DIPJ (*arrowhead*) in keeping with early Bouchard's and Heberden's nodes, respectively, (**b**) oblique (magnified) radiograph allows for better appreciation of joint space loss (*arrow*) as does the (**c**) lateral radiograph for dorsal osteophytes (*arrow*)

spaces are narrowed with subchondral sclerosis. There may be mild radial or ulnar subluxation. Metacarpophalangeal joint involvement occurs usually in the presence of more advanced disease at the PIP and DIP joints. If disease is predominantly at the MCP joints, then one should consider alternative underlying pathology such as CPPD arthropathy and hemochromatosis.

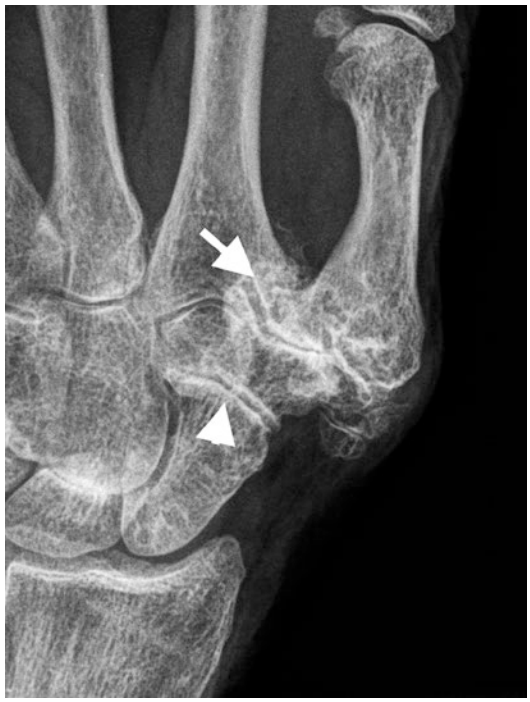

Fig. 10.10 AP radiograph (magnified) demonstrates advanced degenerative disease at the 1st CMC joint (*arrow*) and mild to moderate degeneration at the scaphotrapezial joint (*arrowhead*)

The first carpometacarpal joint is predisposed to degeneration given its multidirectional capabilities and the various articulations involved (Fig. 10.10). Initially the joint space may be widened due to ligamentous laxity. Joint space loss, subchondral cysts, intra-articular (ossified and chondral) bodies, and subluxation may occur. Degeneration may progress to involve the scaphotrapezial joint. The first CMC is also commonly involved in CPPD arthropathy, and review for supporting changes for OA (PIPJ and DIPJ) or CPPD (chondrocalcinosis, radiocarpal degeneration) should be actively sought (Fig. 10.11).

Erosive Osteoarthritis

Erosive or inflammatory OA is a subset of primary OA and predominantly involves the hands. The PIP and DIP joints are more commonly involved although any of the above joints in primary OA may be involved. There are bony outgrowths at the joint margins, joint space loss, and central erosions. The latter produces the "sea gull" appearance (Fig. 10.12). Disease may progress with eventual ankylosis. Lack of marginal

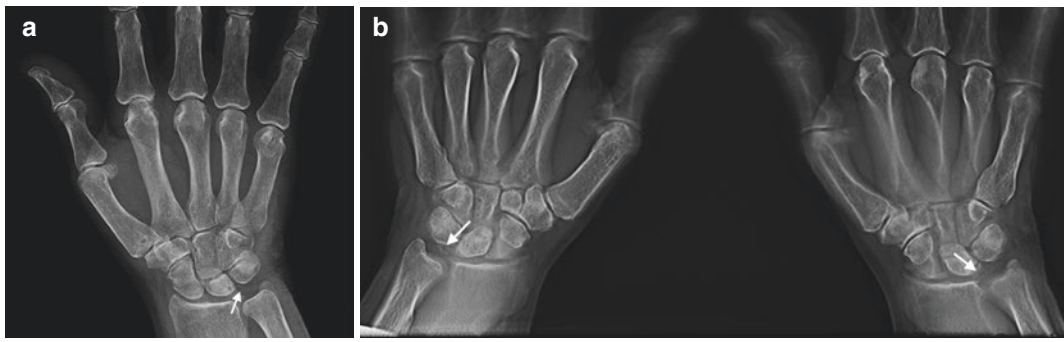

Fig. 10.11 CPPD arthropathy. (**a**) AP radiograph (magnified) of the right hand with joint space loss at the MCPJs, mild subchondral sclerosis, and early osteophytic lipping. MCPJ osteoarthritis is commonly secondary. Note subtle calcification (*arrows*) wrist, lunotriquetral ligament better appreciated on (**b**) single image from tomosynthesis series, also present contralateral hand. Incidental sclerosis and subchondral cyst lunate related to a prior injury

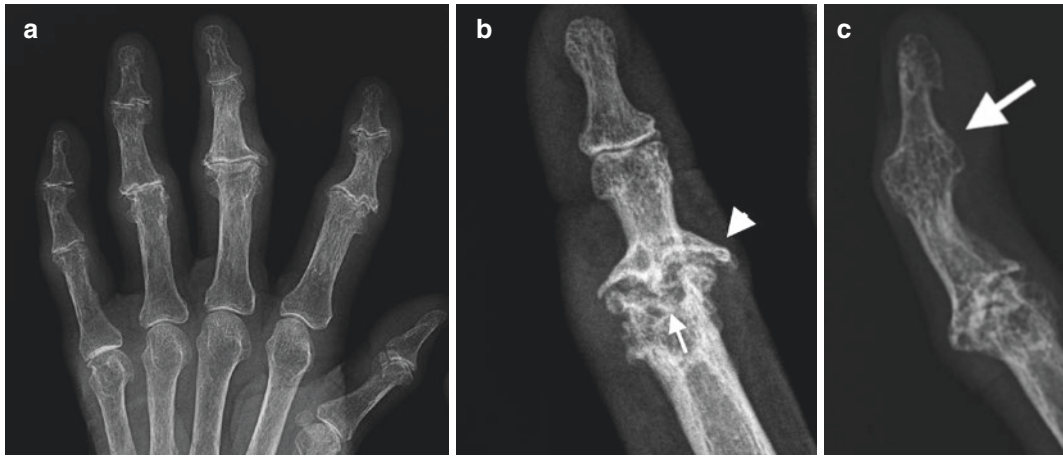

Fig. 10.12 Erosive osteoarthritis on radiographs, (**a**) magnified AP left hand central erosions, (**b**) soft tissue swelling and moderate degenerative changes at the PIPJs and DIPJs with joint space loss, osteophytosis, (**b**) magnified AP second digit different patient with central erosion (*arrow*) with gull wing appearance and moderate osteophytes (*arrowhead*), and (**c**) osseous fusion across fifth DIPJ secondary to erosive osteoarthritis (*arrow*)

erosions, periosteal reaction, and new bone formation, other than osteophytes, helps to separate the disease from psoriatic arthropathy.

Osteoarthritis: Large Joints

Knee

The knee joint is commonly involved with osteoarthritis, often with nonuniform involvement of the patellofemoral and medial and lateral tibiofemoral articulations. Standing radiographs allow for better assessment of joint space loss versus supine studies. Thus when comparing progression of disease, consistent patient positioning is important for an accurate assessment. The medial tibiofemoral joint is more commonly involved than the lateral space and may progress to cause a varum deformity (Fig. 10.13). Joint space in the tibiofemoral space is related both to articular cartilage and the menisci. Occasionally the dominate etiology is meniscal, prior meniscectomy or severely torn and displaced meniscal tissue, and there will be joint space loss without the characteristic additional changes of osteoarthritis.

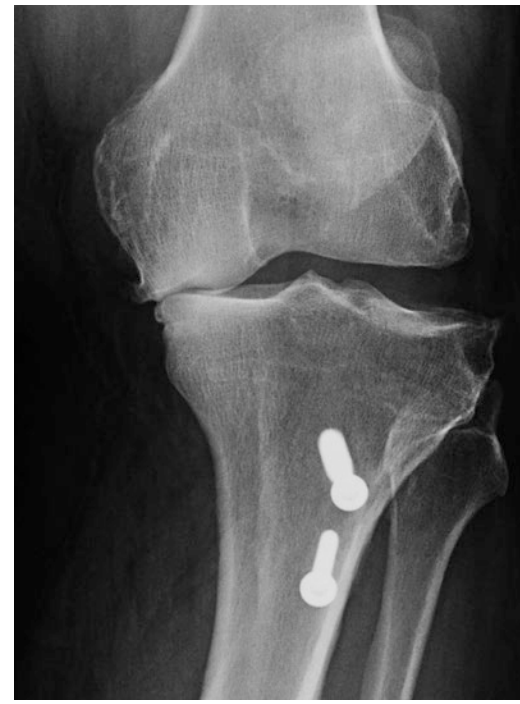

Fig. 10.13 AP radiograph of the left knee with severe medial tibiofemoral joint space loss, osteophytosis, subchondral sclerosis, and secondary genu varum. Orthopedic screws related to prior surgery and repositioning tibial tuberosity

There are several radiographic scoring systems available with the Kellgren and Lawrence classification being most commonly cited (Table 10.1, Fig. 10.14). This system grades conventional x-ray based on a scale of 0–4 by assessing the presence and severity of osteophytes, joint space narrowing, subchondral bony sclerosis, and deformity of the bony contour.

Table 10.1 Kellgren and Lawrence radiographic scoring of knee OA

Grade 0: Normal
Grade 1: Doubtful narrowing of joint space and possible osteophytic lipping
Grade 2: Definite osteophytes, possible narrowing of joint space
Grade 3: Moderate multiple osteophytes, definite narrowing of joint space, some sclerosis, and possible deformity of bone contour
Grade 4: Large osteophytes, marked narrowing of joint space, severe sclerosis, and definite deformity of bone

Knee joint effusion is often present with distension of the suprapatellar recess. This is best appreciated on a lateral radiograph with increased soft tissue attenuation between the post quadriceps and pre-femoral fat pads (Fig. 10.15). Intra-articular loose bodies, osseous and chondral, are also common. Occasionally soft tissue prominence may be identified in the popliteal fossa and represent a Baker's cyst, which often communicates with the joint (Fig. 10.15). Osseous bodies can be identified within the Baker's cyst when a communication exists. Rupture of a Baker's cyst can be confirmed on ultrasound. When dominant involvement of the patellofemoral joint is present, one should consider CPPD arthropathy, which may be present in the absence of radiographic chondrocalcinosis in the same joint.

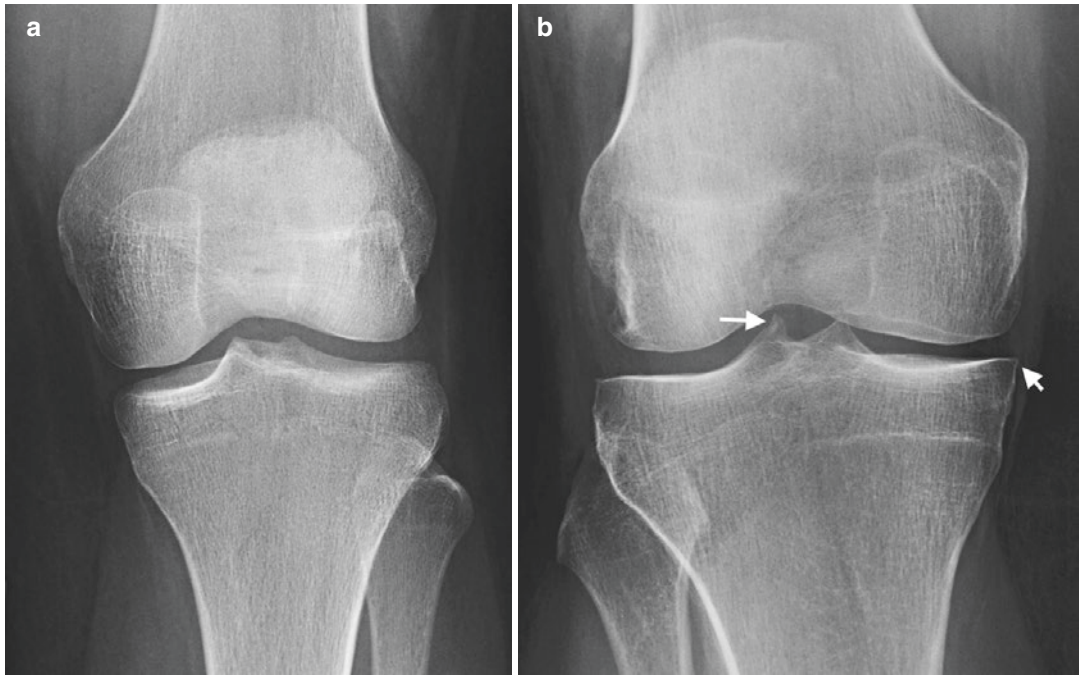

Fig. 10.14 Kellgren and Lawrence radiographic scoring of knee OA. (**a**) Normal AP radiograph of the left knee, grade 0. (**b**) Grade 1: doubtful narrowing of joint space and early osteophytic lipping (*arrow*). (**c**) Grade 2: definite osteophytes, possible narrowing of joint space. (**d**) Grade 3: moderate multiple osteophytes, definite narrowing of joint space, some sclerosis, and possible deformity of bone contour. (**e**) Grade 4: large osteophytes, marked narrowing of joint space, severe sclerosis, and definite deformity of bone

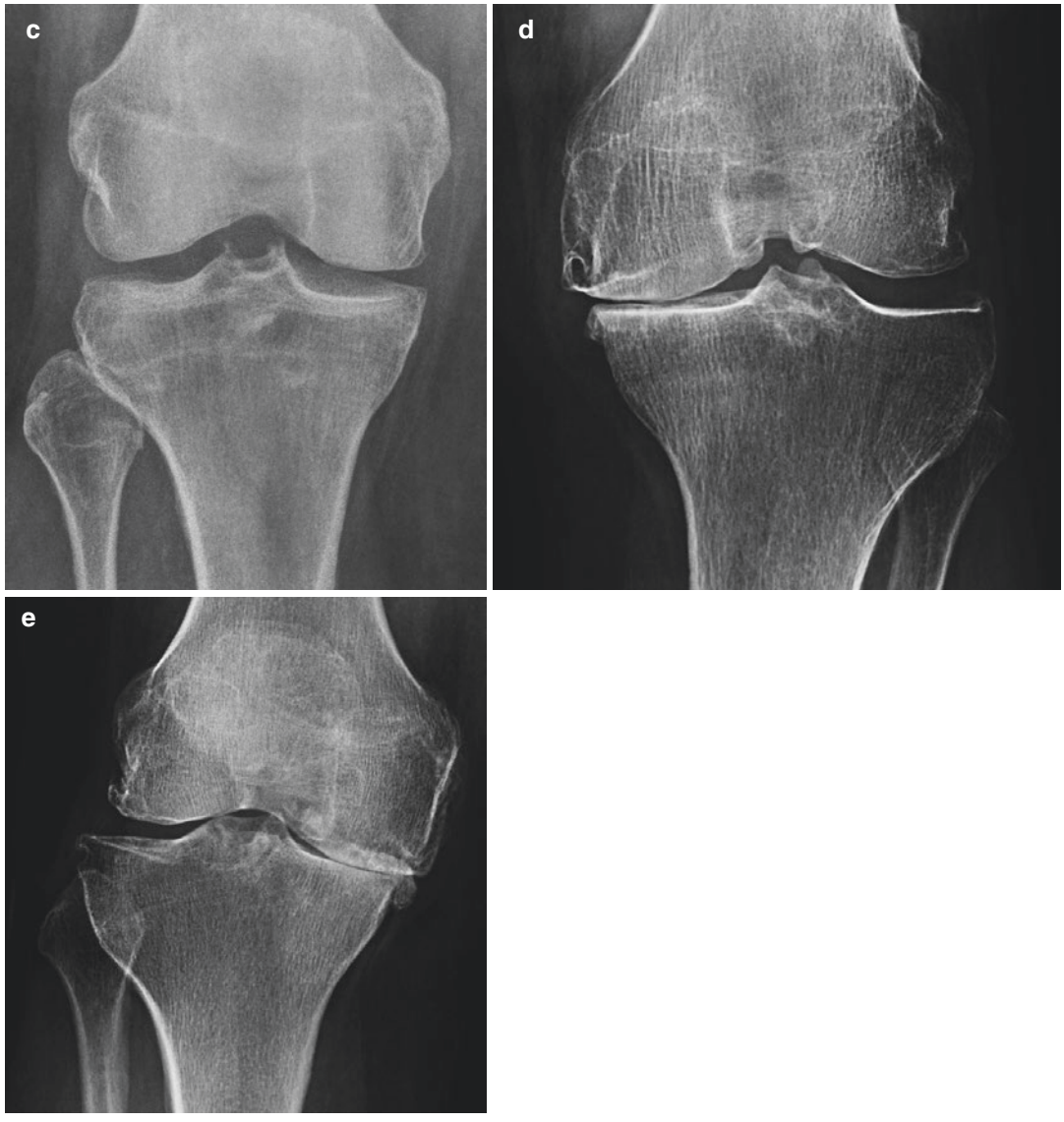

Fig. 10.14 (continued)

Hip

Joint space loss and subsequent migration of the femoral head may occur superolaterally, medially, or axially. Superolateral is the most common with joint space loss most pronounced on the upper and outer aspect of the hip joint, with subsequent migration of the femoral head (Fig. 10.16). The altered dynamics exaggerate secondary OA changes of subchondral sclerosis, cysts, and osteophyte formation. Medial migration may also occur and is more common in women and may relate to acetabular over coverage and subsequent pincer type femoroacetabular impingement. Axial migration occurs in line with the femoral neck and suggests secondary OA, usually related to inflam-

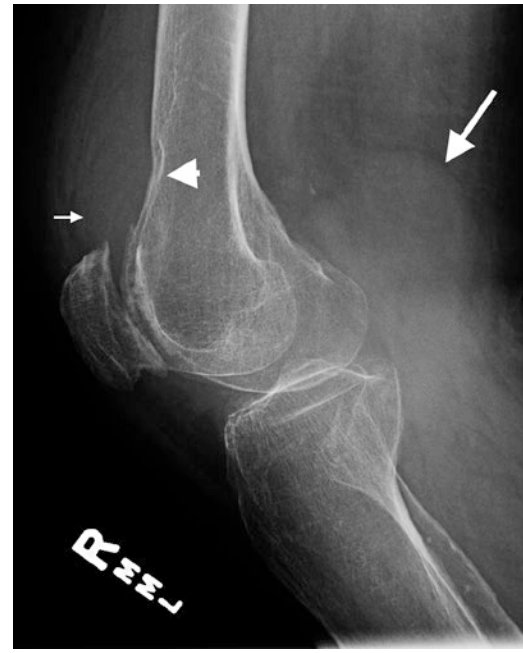

Fig. 10.15 A 58-year-old female lateral radiograph knee demonstrating patellofemoral advanced degeneration, with scalloping of the anterior margin distal femur (*arrowhead*). More pronounced degeneration at the patellofemoral joint should always raise the possibility of CPPD as in this case. Note suprapatellar recess moderate effusion (*small arrow*), soft tissue swelling within the popliteal fossa (*long arrow*) in keeping with a distended Baker's cyst (confirmed with ultrasound)

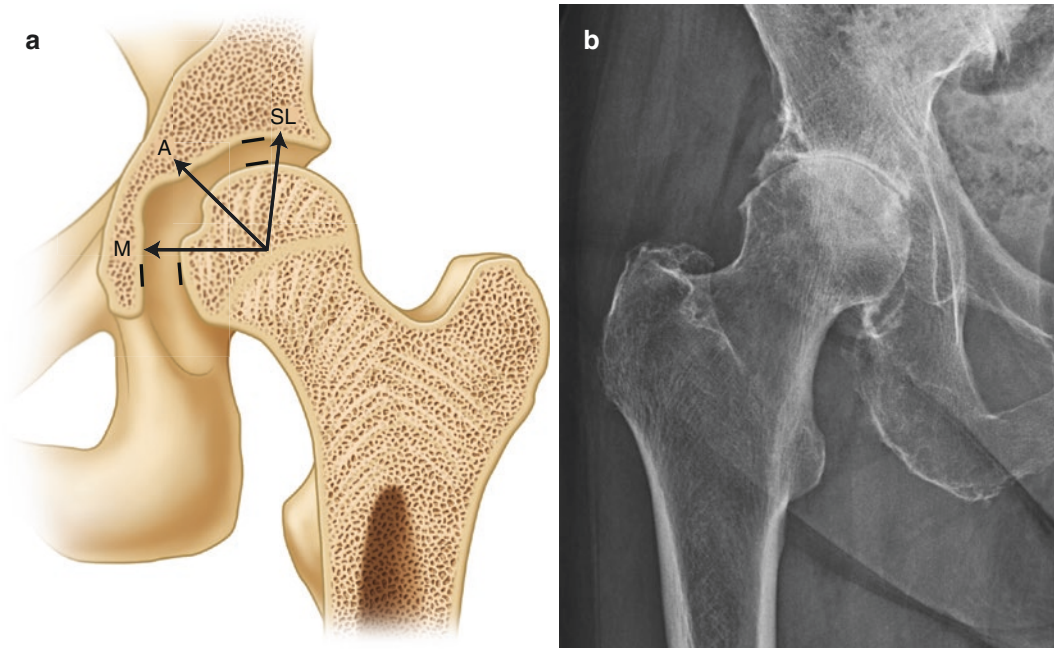

Fig. 10.16 (**a**) Femoral head migration patterns, *SL* superolateral, *A* axial, *M* medial. (**b**) AP radiograph hip with superolateral migration femoral head with joint space loss most pronounced on the upper and outer aspect of the hip joint. (**c**) Axial migration occurs in line with the femoral neck and suggests secondary OA, in this case secondary to CPPD arthritis which is suggested by the prominent subchondral cysts. (**d**) Axial migration with acetabular overcoverage. (**e**) Normal radiograph for comparison

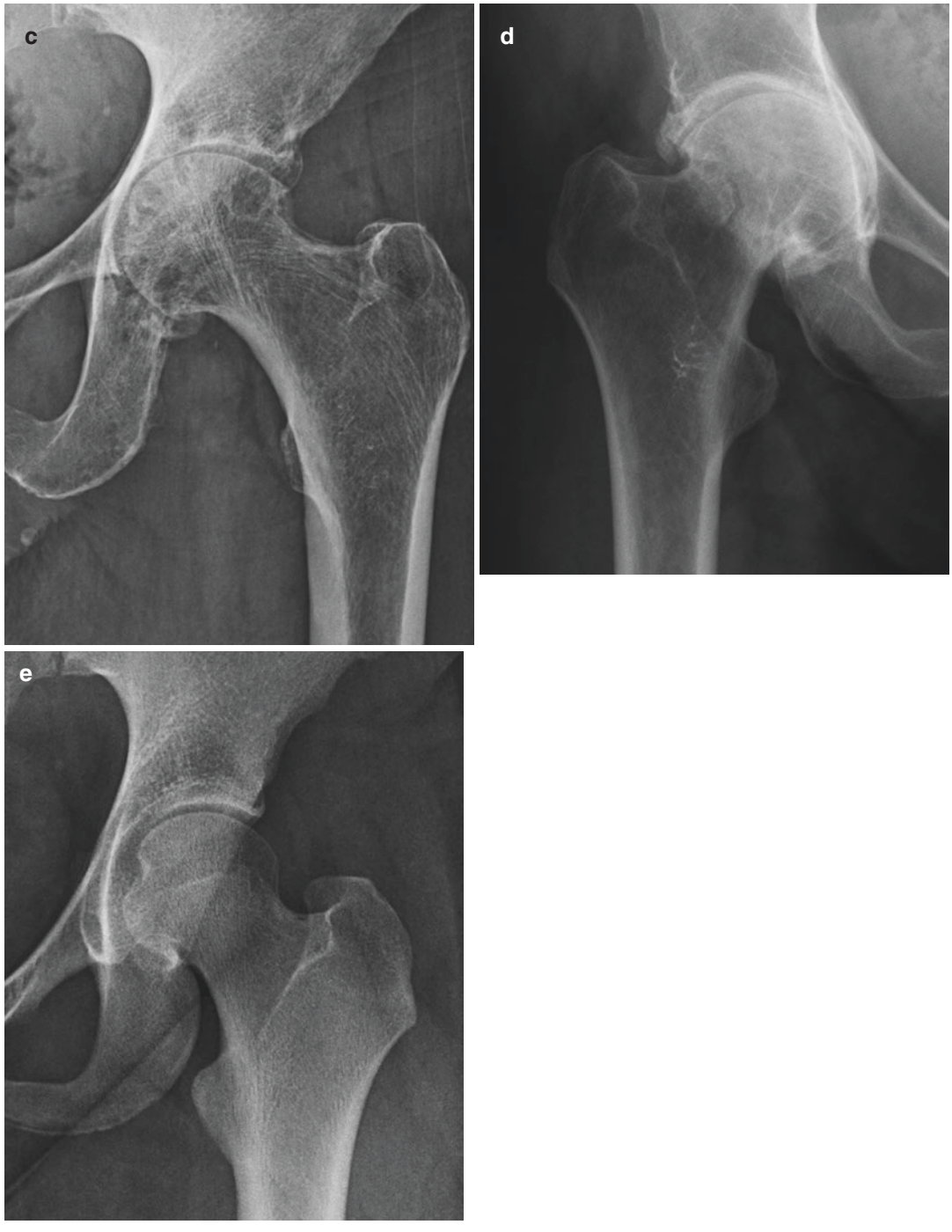

Fig. 10.16 (continued)

matory arthropathy such as rheumatoid arthritis. Buttressing, thickening of the medial cortex femoral neck with endosteal and periosteal new bone formation, is a stress response related to altered dynamics upon the hip joint due to OA.

Femoroacetabular impingement is an increasingly recognized cause of premature osteoarthritis. There are two main types, which often coexist to varying degrees in up to 80 % of patients, Cam and Pincer. Cam type, described after a cam mechanism, occurs in the presence of a femoral head neck dysplasia, i.e., abnormal bone formation at the femoral head neck junction, usually anterior or anterolaterally. This bony "bump" impacts upon the acetabulum on flexion and internal rotation of the hip with subsequent injury to the acetabular labrum and adjacent cartilage. It is more common in males. Pincer type is more common in middle-aged women and is related to acetabular overcoverage (Figs. 10.17 and 10.18). Note that these radiographic abnormalities may be present in joints that are asymptomatic and have no evidence of osteoarthritis.

Postel's coxarthropathy is an uncommon rapidly progressive destruction of the hip joint. It is thought to be secondary to insufficiency fracture with collapse and osteolysis of the femoral head. Degenerative changes are present; however, there is little osteophyte formation. The condition usually occurs unilaterally in elderly women (Fig. 10.19). Differential includes crystal arthropathy.

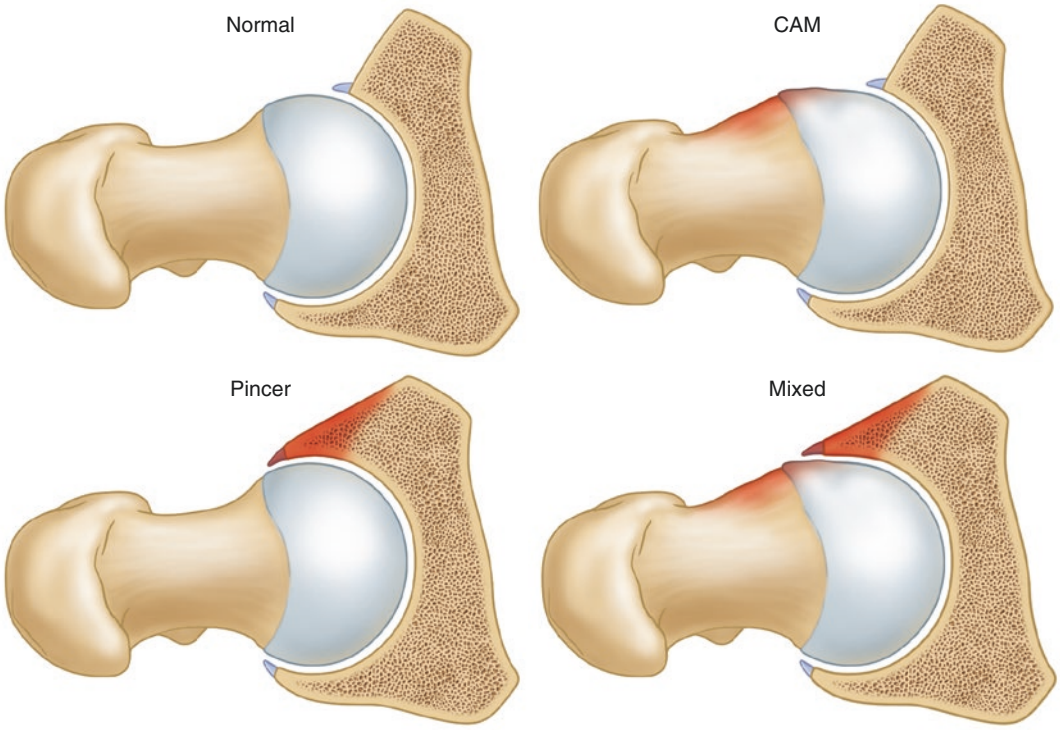

Fig. 10.17 Different types of femoroacetabular impingement

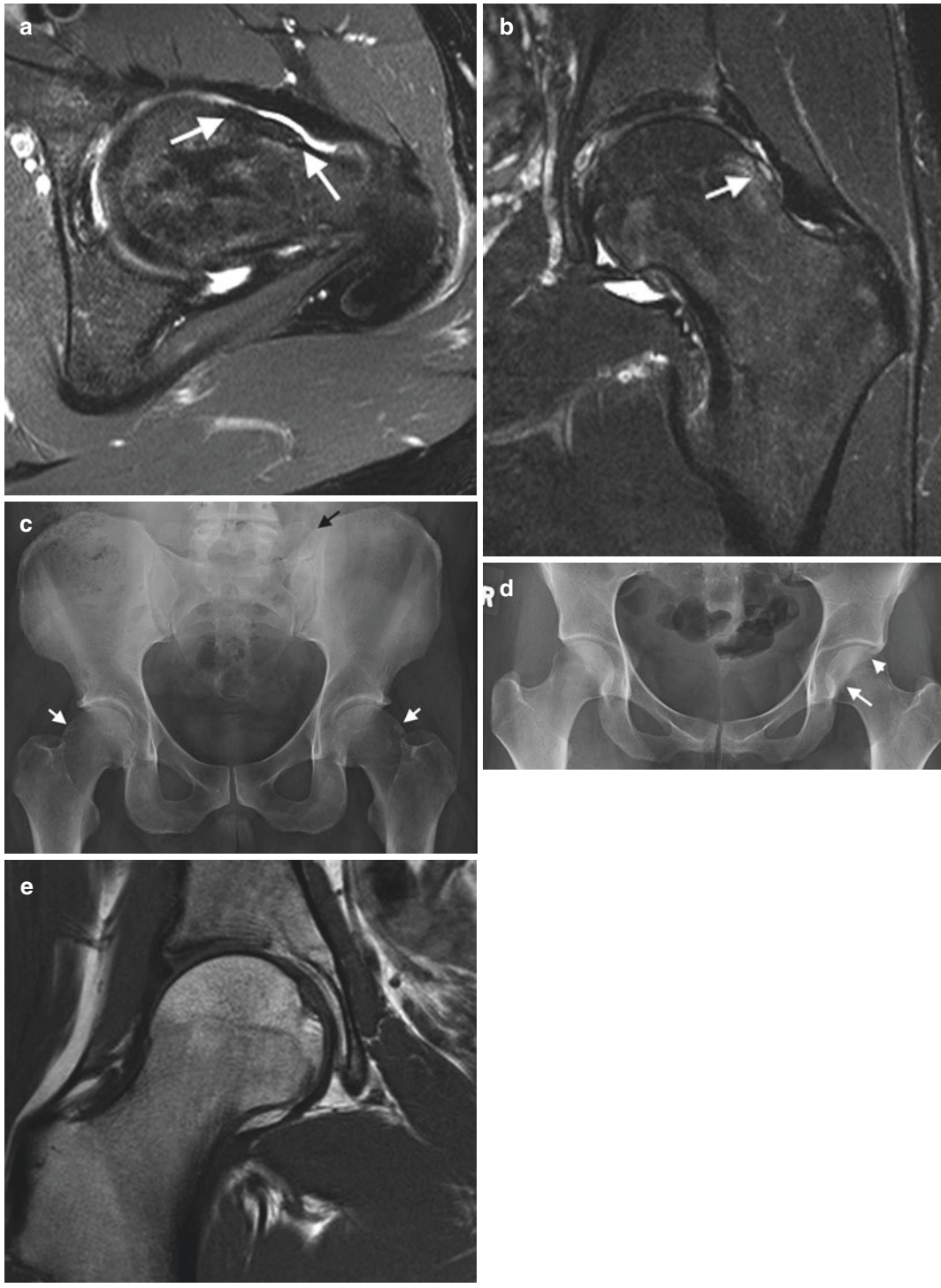

Fig. 10.18 (**a**) Axial oblique and (**b**) Coronal T2FS demonstrating femoral head-neck dysplasia (*arrows*) with associated mild bone marrow oedema; (**c**) AP pelvic radiograph demonstrating femoral head–neck dysplasia (*arrows*), incidental left transitional vertebra; (**d**) acetabular retroversion and (**e**) normal Cor T1 MRI right hip for reference

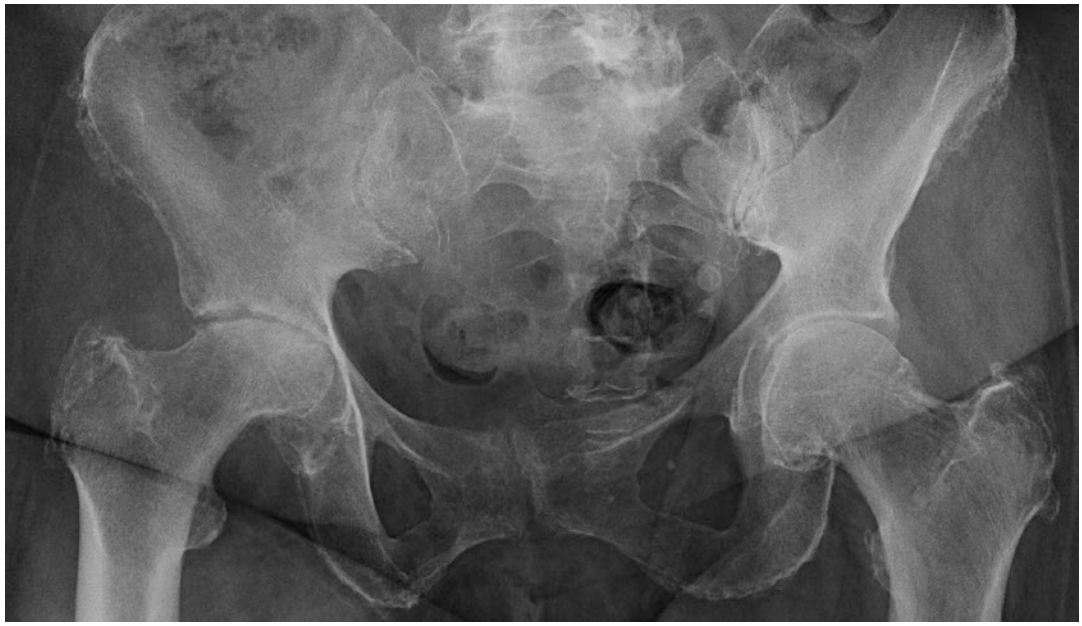

Fig. 10.19 AP radiograph of bilateral hips. Partial collapse right femoral head, likely related to previous undiagnosed insufficiency fracture on a background of degenerative disease. Note degenerative changes left hip and prominent enthesophyte formation; the later is related to known DISH in this patient

Glenohumeral OA

Glenohumeral osteoarthritis is almost always secondary in nature. Predisposing factors include prior trauma, instability, crystal disease, rotator cuff disease, and inflammatory, metabolic, and endocrine arthropathies. Rotator cuff arthropathy, common in the elderly patient, occurs in the presence of a complete tear of the supraspinatus tendon and often tears of the remaining rotator cuff and long head biceps (Fig. 10.20). There is secondary superior translation of the humeral head due to the unopposed upward pull of the deltoid muscle. The humeral head erodes the undersurface of the acromion with cortical loss, irregularity, and cyst formation, particularly within the superior humeral head and greater tuberosity. The inferior humeral head subsequently articulates abnormally with the glenoid with cartilage and joint space loss and inferior glenohumeral osteophytosis. This progresses to diffuse glenohumeral degeneration. In the absence of rotator cuff disease, trauma history, or evidence of an inflammatory arthritis, crystal disease should be considered and is reviewed in greater detail in Chap. 8.

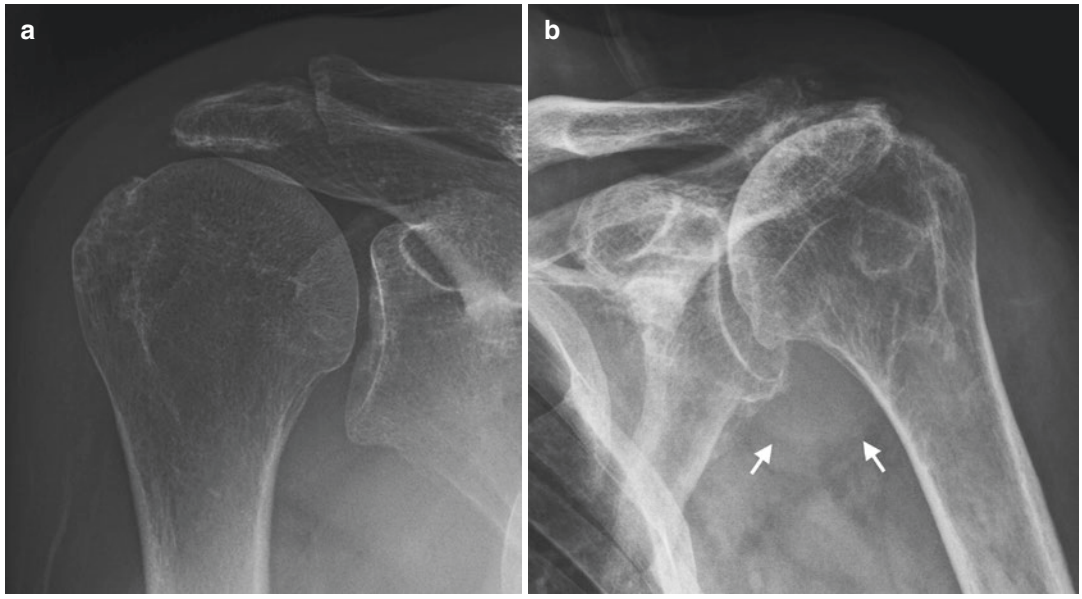

Fig. 10.20 Rotator cuff arthropathy in (**a**) 67-year-old female with massive rotator cuff tear, secondary superior translation humeral head and early cortical changes acromial undersurface to impaction from the humeral head, note cortical irregularity greater tuberosity secondary to rotator cuff disease (**b**) more advanced rotator cuff arthropathy in a 78-year-old male with osteophytosis inferior glenohumeral joint and extensive erosion acromion. Note joint effusion with soft tissue joint distension inferiorly (*arrows*)

Further Reading

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