Atlas of Nuclear Medicine in Musculoskeletal System

Case Oriented Approach

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To the patients of all cases presented in this atlas textbook

Preface

Many nuclear medicine textbooks and case studies in forms of atlas have been published so far, but there seems to be no in-depth nuclear medicine imaging atlas focused on diseases of the musculoskeletal system. Since an independent volume focusing on nuclear imaging of musculoskeletal disease has been rare, publication of this book has its significance. In the attempt to cover a specified area of musculoskeletal nuclear medicine, this book includes a large number of correlative clinical images in musculoskeletal disorders. Therefore, the authors wish to write about common cases as well as rare musculoskeletal disorders in which various imaging techniques of nuclear medicine (bone scan, SPECT, SPECT/CT, PET/CT, etc.) are useful based on the author's clinical experience in many different hospitals.

Nuclear medicine imaging in the musculoskeletal system with its ability to assess disease activities has contributed to accurate diagnosis and improved medical and surgical treatment. This book is intended to share the reading experiences of the authors with nuclear medicine and radiology residents and board specialists, and to help other clinicians who manage musculoskeletal disorders such as orthopedic and rheumatology, through various cases of musculoskeletal disorders to support their patient care. We aim to publish an easy-to-read clinical atlas by organizing the proper roles and features of various nuclear medicine imaging technics in musculoskeletal disorders by case-oriented approach.

Please consider that the format of each chapter varies according to the characteristics of each chapter title, and it is challenging to achieve complete integrity by respecting the opinions of the authors of each chapter. The editors wish to thank all contributors who spent much time and efforts in the preparation of their chapters. All the authors who participated in this issue are experts in their field. We are indebted to them for their time and effort.

It is our expectation that the original purpose of publishing cases of musculoskeletal disease including various nuclear medicine images experienced in hospitals in Republic of Korea as a case-oriented textbook has been fulfilled to a certain extent and becomes a helpful book to readers.

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Part I

Inflammatory and Infectious Disorders

Musculoskeletal Infections

1

Jung Mi Park, Jae Pil Hwang, Joon Ho Choi, Jang Gyu Cha, and Yu Sung Yoon

Abstract

Pedal ulcer occurs in approximately 25% of the diabetics. Three-phase bone scan plays a role in the assessment of vascular supply including small arteries and capillary vessels in diabetic foot ulcer. Peri-prosthetic joint infection occurs in 1%–2% of primary and in 4% of revision arthroplasties. Serum CRP may be less specific after post-operative infection and antibiotics therapy; however, combined WBC scan with three-phase bone scan can detect peri-prosthetic infection accurately. Charcot neuropathic osteoarthropathy is a non-inflammatory and progressive destruction of the bone and joint. Bone single-photon emission computed tomography/computed tomography (SPECT/CT) provides an additional anatomical information to distinguish from bone and soft tissue inflammation or infection in evaluating Charcot foot. Typical pyogenic spondylitis affects two adjacent vertebrae and the intervening disc. Differential diagnosis for tuberculous spondylitis could be performed with clinical symptom and imaging findings.

Keywords

Diabetic foot infection · Chronic prosthetic joint infection · Charcot foot · Pyogenic spondylitis

1.1 Diabetic Foot Infection

1.1.1 Clinical Course, Assessment, and Treatment

Development of pedal ulcer can be estimated to occur in 25% of the diabetics. Diabetic foot disorder is the most common cause of lower extremity amputations [1]. Hyperglycemia can cause direct damage to the nerves and blood vessels. Diabetic vascular disease has three components: arteritis and small vessel thrombosis, neuropathy, and large vessel atherosclerosis. Once tissue damage has occurred in ulcer or gangrene, the two main threats are infection and ischemia. Various foot ulcer classifications have been proposed to organize the appropriate treatment plan: the University of Texas diabetic foot ulcer classification is based on ulcer depth and is graded according to the presence or absence of infection and ischemia. Many ulcers where critical ischemia exists fail to heal and lead to irreparable tissue damage and amputation [2].

The 5-year mortality in patients with diabetes and critical limb ischemia is 30%, and the 5-year mortality in patients with diabetic foot infections

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who have foot amputations is about 50% [3]. While the neuropathic foot is characterized by warm, dry, bounding pulses as a result of peripheral vasodilation, callosities, painless penetrating ulcers at pressure points, painless necrosis of toes, spreading infection along plantar spaces, and loss of pain and thermal sensation, the ischemic foot is characterized by cold, absent pulses, trophic changes, painful ulcers around heels and toes, and claudication and rest pain. Although these factors may co-exist, it is important to early detect ischemia in the diabetic foot ulcer. Diabetes itself shows a 25% increased risk for peripheral arterial disease [4]. Bone scan can provide a useful assessment of vascular supply including small arteries and capillary vessels [5]. Proper vascular assessment in small vessel disease with associated gangrenous toes can be help to provide a successful treatment with debridement and minor amputation instead of wide amputation. In chronic and progressive diabetes, a conservative surgical approach such as revascularization can be considered. But primary amputation is better than revascularization or percutaneous transluminal angioplasty in the case of bedridden patients and patients with life-threatening sepsis and extensive muscle necrosis.

Case 1.1

A 58-year-old man was referred from an outside hospital; he was treated for necrosis of the left first toe for 3 months. He had a history of having his entire right toes amputated 3 years ago for atherosclerosis obliterans. His left first to third toes were discolored black, and gangrene was progressing. *Enterobacter cloacae* was cultured from his wound; he was treated with antibiotics. His angiography showed multifocal stenosis in both superficial femoral arteries. His plain radiography could not depict any significant bone abnormality on his left toes. However his three-phase bone scan showed perfusion defect in the left first to third toes with complete absence of bone uptake (Fig. 1.1). WBC SPECT/CT demonstrated the strong WBC uptake in the left fourth and fifth toe soft tissue, as well as a cold defect in the left first to third toe gangrene (Fig. 1.2).

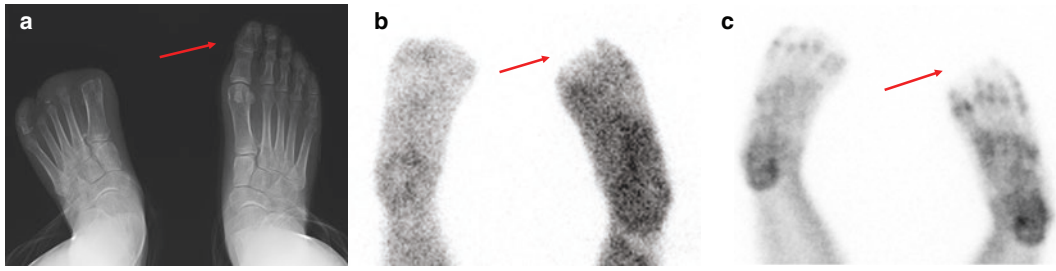

Fig. 1.1 There is no significant abnormal bone lesion in the left foot plain radiography (**a**). Perfusion defects and loss of bone uptake in the left 1st–3rd toes are clearly observed on the blood pool image and bone phase image of the three-phase bone scan (**b**, **c**)

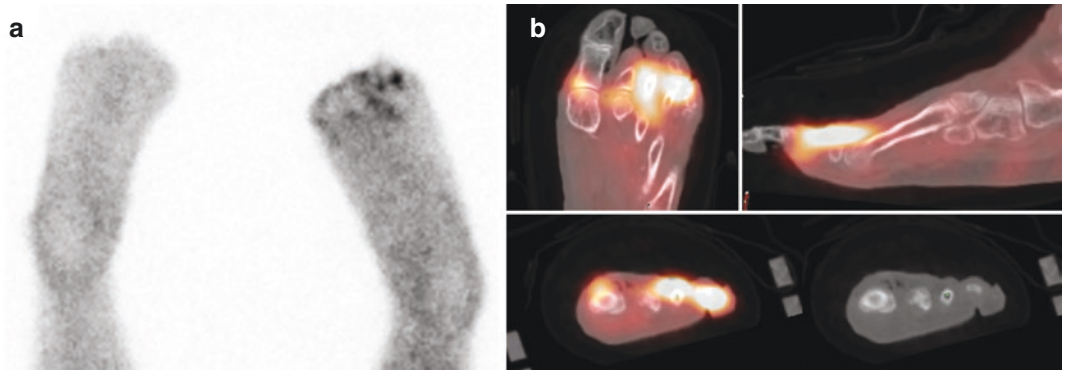

Fig. 1.2 WBC scan and SPECT/CT show cold defects in all of the left toes as well as increased uptake in the soft tissue overlying the 3rd–5th toes (**a**, **b**)

1.2 Chronic Prosthetic Joint Infection

1.2.1 Clinical Course, Assessment, and Treatment

Peri-prosthetic joint infection (PJI) occurs in 1%–2% of primary and in 4% of revision arthroplasties. Management of PJI requires multiple surgical revisions and long-term antimicrobial treatment. About two thirds of PJI results from intra-operative inoculation of microorganisms [6]. However, all prosthetic joints have hematogenous seeding from a distant primary focus, where highly vascular peri-prosthetic tissue is exposed to the highest risk of hematogenous infection in the first years after implantation. The most common primary foci are skin and soft tissue infection, respiratory tract infections, gastrointestinal infections, or urinary tract infections. According to the definition criteria for PJI by the European Bone and Joint Infection Society 2018 in Helsinki, if there is one more criteria, PJI including chronic infection can be diagnosed. It shows better sensitivity for diagnosing PJI [7].

The sensitivity of synovial fluid culture is 45% to 75% with a specificity of 95% [7]. The sensitivity of intra-operative swab is low, and the swab should be avoided. This is because a swab from the wound or sinus tract can mislead by detecting colonizing microorganisms. Generally three to five intra-operative tissue samples should be obtained for the culture. Histopathology of peri-prosthetic tissue should be considered a standard procedure in the diagnosis of PJI.

In PJI caused by low-virulence pathogens, blood tests such as WBC, ESR, and CRP are often normal [8]. CRP can be increased after surgery, due to post-operative inflammation. Serial measurements of CRP are more important for accurate interpretation. In acute post-operative infections (<4 weeks) or acute hematogenous infections, debridement, antibiotics, and implant retention are the best treatment. Local antibiotics can be additionally used during revision surgery. Antibiotic-loaded polymethylmethacrylate (PMMA) beads can become colonized by bacteria due to rapid decrease of local antibiotic concentration, resulting in new biofilm formation [6]. In patients with numerous previous revisions, or when local conditions require time, two-stage exchange with 4- to 6-week antibiotics treatment can be applied. Longer intervals (>8 weeks) of persistent sign of infection, debridement, and antibiotic-loaded spacer cement are used for dead space management with two-stage revision surgery for treatment of golden standard.

Case 1.2

A 78-year-old woman with diabetes visited the hospital for discharge from the right pre-tibial area, which had undergone knee arthroplasty at an outside hospital 7 months ago. She underwent prostheis removal due to septic arthritis and antibiotic bead insertion, and baseline three-phase bone scan showed increased perfusion and joint uptake in the right knee joint and pre-tibial space (arrow) suggesting septic arthritis (Fig. 1.3). Her serum CRP level decreased from 5.2 mg/L to 0.13 mg/L after 2 months of antibiotic IV therapy and antibiotic bead insertion. She underwent a baseline three-phase bone scan (Fig. 1.3) and 2 months later follow up scan (Fig. 1.4). It was to determine an optimal timing of revision of arthroplasty; contrary to expectations, the scan showed severely increased perfusion and bone uptake in the right tibia shaft (Fig. 1.4). WBC scan also showed diffuse WBC uptake along the previously inserted antibiotic beads in the tibia (Fig. 1.4). Rapid decrease of local antibiotic concentration in the inserted antibiotic-coated beads resulted in new biofilm formation. She underwent surgery to remove infectious granulation tissues meticulously in the bone marrow with massive irrigation and new cement insertion with VPMMA for removal of dead space in the tibia. A few months later, she underwent joint replacement surgery. Three-phase bone scan and WBC scan may be useful for accurate diagnosis of infection even when serum CRP returns to normal range after antibiotic treatment.

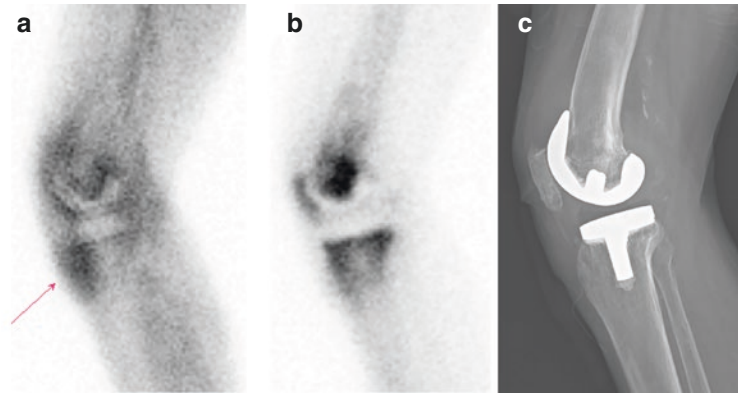

Fig. 1.3 Pretibial soft tissue hyperperfusion (arrow) and diffuse hyperperfusion surrounding the right knee joint are seen on the three-phase bone scan (**a**, **b**). Soft tissue swelling and fluid collection are seen in the patellofemoral compartment of the knee on the plain radiography (**c**)

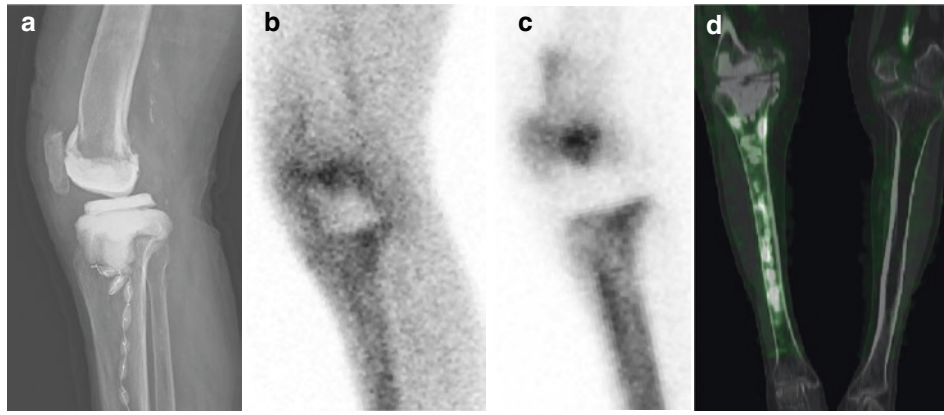

Fig. 1.4 Prosthetic removal and antibiotic bead insertion in the right tibia are seen on the plain radiography (**a**). After 2 months, serum CRP has returned to the normal range. Followed up three-phase bone scan finds the infection widely extends to the tibial shaft (**b**, **c**). WBC SPECT/CT shows strong WBC accumulation in the tibia, consistent with bone marrow infection (**d**)

1.3 Charcot Foot

1.3.1 Etiology and Clinical Significance

Charcot neuropathic osteoarthropathy is a disease spectrum of the bone, joint, and soft tissue and is non-inflammatory and progressive destruction of the bone and joints. Charcot theory is not yet clear for its pathogenesis or mechanism; there is consensus that the cause is multifactorial including polyneuropathy (loss of sensation and proprioception), neurotraumatic, and neurovascular conditions with combined osteoarthropathy [9].

From a clinical perspective, its early phase is characterized by a hot or warm, red, and swelling of foot, often without pain due to polyneuropathy, and by osteopenia with fractures. The disease will progress without proper treatment, and may result in Lisfranc's joint destruction and callopse of the longitudinal arch of the foot. The typical end-stage appearance of a Charcot foot is the rocker bottom deformity. Calcaneal insufficiency fracture is an uncommon, which can be associated with neuroarthropathy or severe osteoporosis, and may be caused by spontaneous condition or repeated microtrauma of the pull of the calcaneal tendon.

1.3.2 Radiographic Imaging

The Charcot foot can be classified using various systems according to anatomical landmarks and clinical symptoms. The most common one is the Sanders and Frykberg classification; this classification identified five zones of disease distribution according to the anatomical location. The most

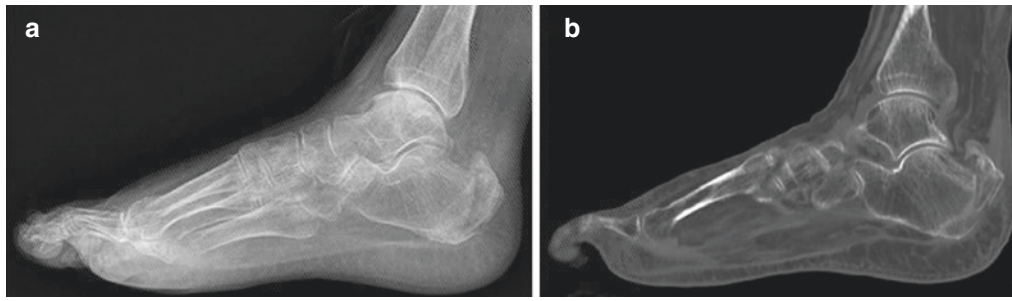

Fig. 1.5 Plain radiography and sagittal image of 3D foot CT show fracture in the calcaneal tuberosity and fluid collections at the posterior aspect of the calcaneus and tibiotalar joints (**a**, **b**)

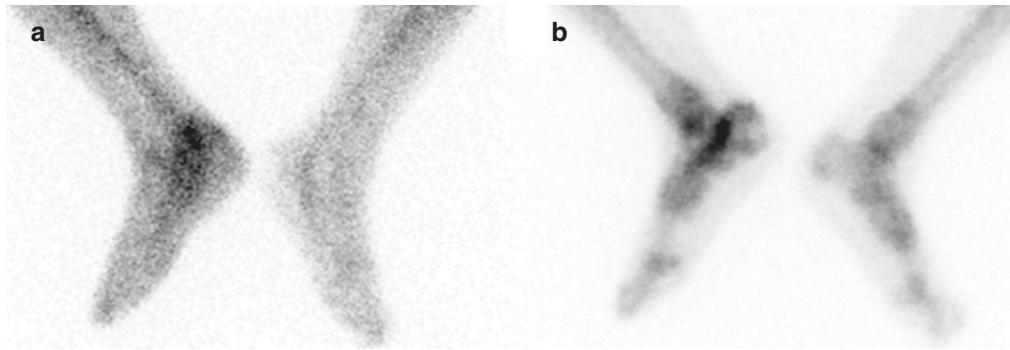

Fig. 1.6 Three-phase bone scan shows decreased radioactivity in the calcaneal fracture site and diffusely and mildly increased perfusion and bone uptake in the talocalcaneal joints (**a**, **b**)

commonly involved areas are about 45% in zone II in about 35% in zone III of cases.

Conventional radiographs of the Charcot foot are traditionally the standard imaging technique to establish the diagnosis, to stage, and to monitor the disease. MRI is a well-known imaging modality to diagnose a suspected early active Charcot disease. Early signs of a Charcot foot in MRI are bone marrow edema, soft tissue edema, joint effusion, and subchondral microfractures. MRI of late-stage Charcot foot shows joint destruction, cortical fractures, joint dislocations, bone marrow edema, superior and lateral dislocation of Lisfranc's joint, prominent well-marginated subchondral cysts, bone proliferation, sclerosis, debris, intraarticular bodies, and dislocation of talus and navicular bones [10].

Three-phase bone scintigraphy is generally used to exclude osteomyelitis in diabetic patients. Increased perfusion and bone uptake are not specific to diagnose osteomyelitis, because they may also occur in chronic soft tissue infections, fractures, and neuropathic joints. Bone SPECT/CT provides an additional anatomical information to distinguish bone and soft tissue inflammation or infection. Bone scintigraphy with radiolabeled leukocytes is more specific for osteomyelitis [11].

Case 1.3

An 82-year-old woman presented to the outpatient clinic due to left heel pain developed 2 days ago. She sprained her foot from walking 3 days ago and had a long time of diabetes history for 25 years. Plain radiography and foot 3D CT revealed right calcaneal tuberosity fracture and fluid collections at posterior aspect of calcaneal fracture site and anterior aspect of tibiotalar joints (Fig. 1.5). ^{99m}Tc-DPD three-phase bone scan and bone SPECT/CT also showed diffusely increased perfusion and bone uptake along the anterior and posterior talocalcaneal joints (Figs. 1.6 and 1.7). These additionally showed decreased perfusion and bone uptake from bony fragmentation at calcaneal avulsion fracture site. Based on the clinical exam including a long his-

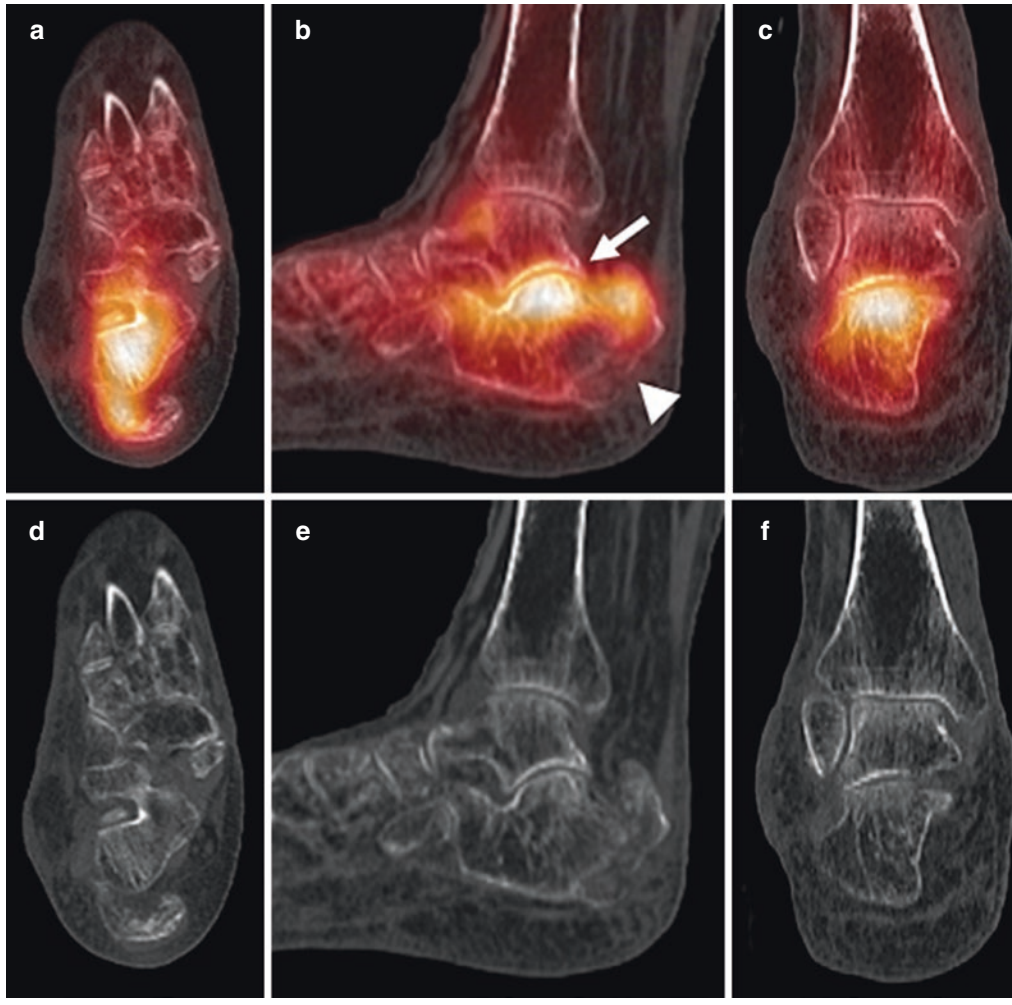

Fig. 1.7 Bone SPECT/CT shows diffuse osteopenia and small bony fragment in the talocalcaneal joint area as well as the avulsion fracture in the calcaneal tuberosity (arrowhead in **b**) on its fusion axial, sagittal, and coronal images (**a**–**c**), axial, sagittal, and coronal CT images (**d**–**f**)

tory of diabetes mellitus, no evidence of tenderness, and imaging work-ups including avulsion fracture and joint activity of common location, early Charcot arthropathy was suggested.

1.4 Malleolar Bursitis

1.4.1 Etiology and Clinical Significance

The bursa is fluid-containing, extra-articular closed sacs that provide cushioning and assist in decreasing friction between skeletal and soft tissue structures, including bone-tendon, bone-skin, and tendon-ligament interfaces. The bursa can be divided into anatomical and adventitious bursae; lateral malleolar bursa is adventitious type. Lateral malleolar bursitis is a rare cause of ankle pain and swelling characterized by bursa wall thickening and excess bursal fluid accumulation. This disease is caused by the inflammation or infection, repetitive irritation, constant pressure, swelling, complication from arthritis, and repeated stress or injury of the lateral malleolar area of the ankle. Treatment includes a lifestyle modification, combination of

oral or parenteral antibiotics, and needle aspiration or incisional drainage, and surgical intervention may be necessary in some cases.

1.4.2 Radiographic Imaging

Typical ultrasonographic finding is a fluid-filled anechoic structure with a thickened hyperechoic wall. On MRI, the bursa is seen as a high T2 fluid-filled structure, and CT shows the inflamed bursa as hypodense with an enhancing wall [12]. Three-phase bone scan and perfusion SPECT/CT show hyperemia and focal increased bone uptake. In addition, SPECT/CT can provide higher diagnostic accuracy and anatomical information distinguished bone and soft tissue inflammation or infection due to additional CT imaging technique [11].

Case 1.4

A 52-year-old man presented to the outpatient clinic due to right ankle pain with ulceration (Fig. 1.8). He has had diabetes mellitus for a long time. Radiographs showed no bony abnormality except for soft tissue shadow corresponding the lesion in the right lateral malleolar area (Fig. 1.9). ^{99m}Tc-DPD three-phase bone scan showed increased soft tissue uptake in the right lateral malleolar area on blood pool phase image and mildly increased uptake in the distal fibular area suggesting reactive change on delay bone phase image (Fig. 1.10). Blood pool phase SPECT/CT showed localized increased uptake with diffuse soft tissue swelling centering around the lateral malleolar bursa of right ankle. Otherwise, delay bone phase SPECT/CT showed increased bone uptake suggesting reactive change in the right distal fibula (Fig. 1.11).

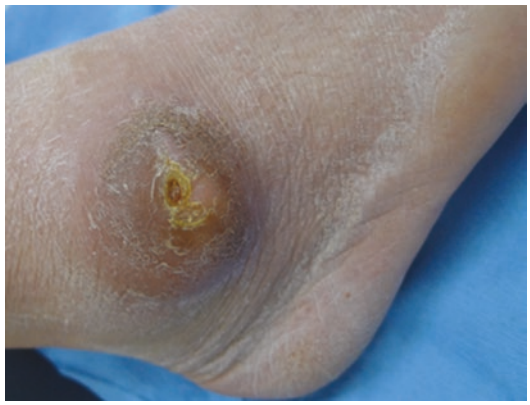

Fig. 1.8 Photograph of lateral malleolar lesion of right ankle at the time of admission

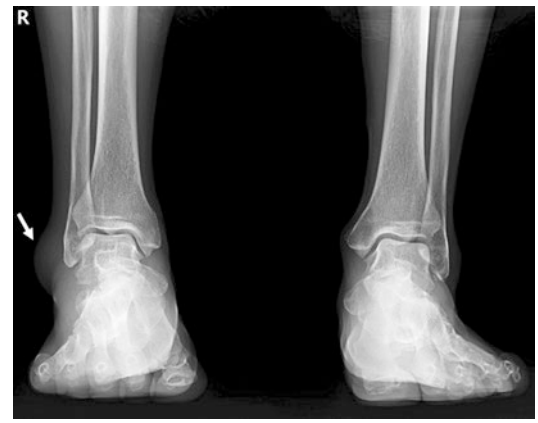

Fig. 1.9 Plain radiography of both feet and ankles

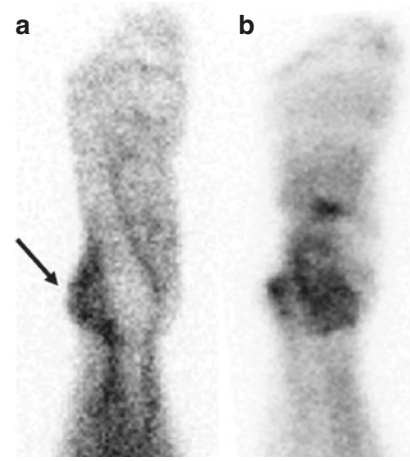

Fig. 1.10 Three-phase bone scan shows increased perfusion in the swelling area of the right lateral malleolus and mildly increased bone uptake in the lateral malleolus (**a**, **b**)

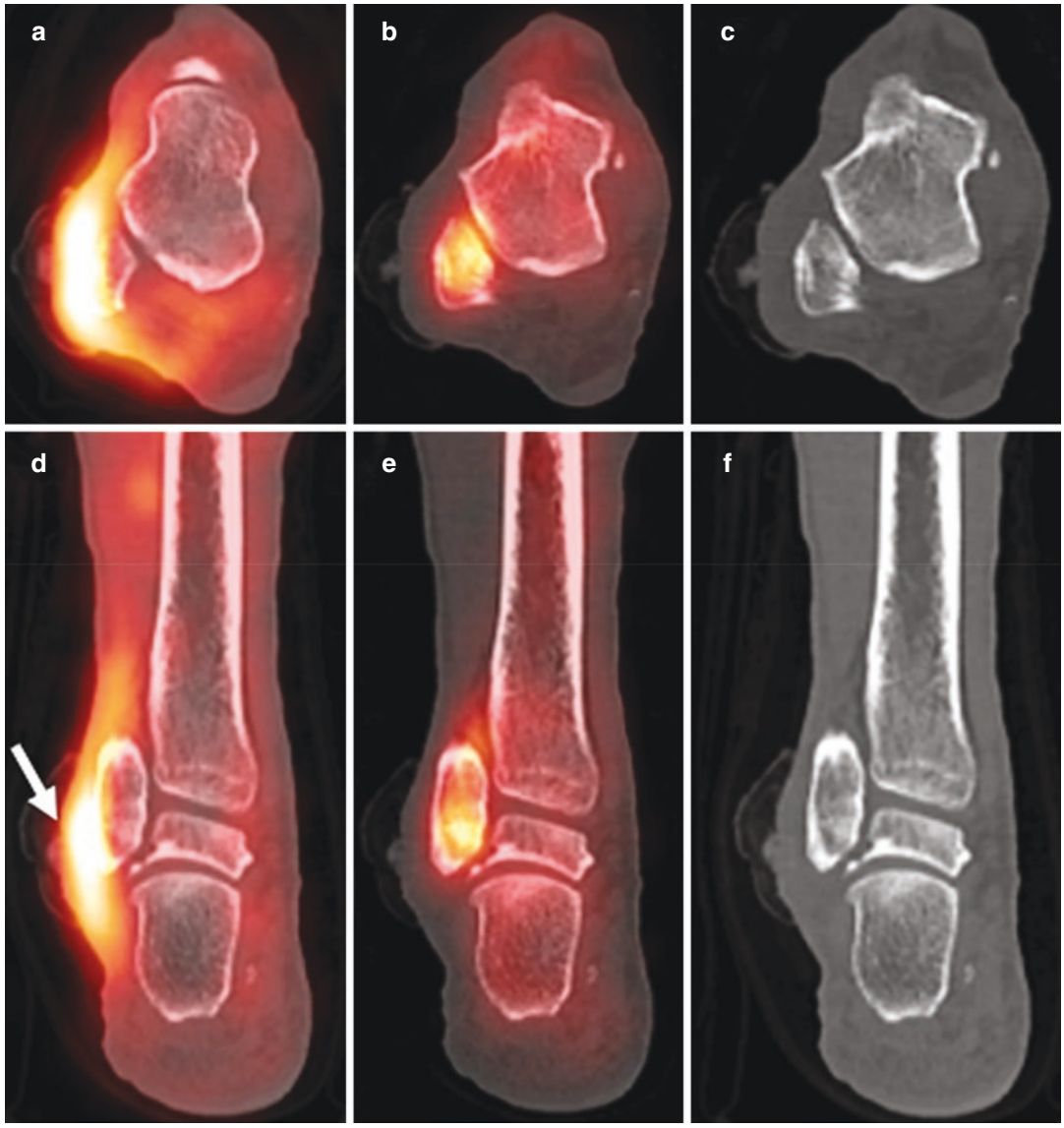

Fig. 1.11 Blood pool and bone SPECT/CT; blood pool phase axial, bone phase axial, bone axial CT images (**a**–**c**), blood pool phase coronal, bone phase coronal, and bone coronal CT (**d**–**f**). SPECT/CT can provide accurate anatomical differentiation between soft tissue and bone, while in a planar three-phase bone scan increased uptake area can be obscure for an exact localization

1.5 Pyogenic Spondylitis

1.5.1 Etiology and Clinical Significance

The term "pyogenic spondylitis" is a broad term which includes pyogenic spondylodiscitis, vertebral osteomyelitis, septic discitis, and epidural abscess. Pyogenic spondylitis usually develops from bacterial origin. The arterial route is more widespread than the venous route, usually from the skin, oral cavity, respiratory tract, and genitourinary tract [13]. The vertebral segmental artery provides an intervening disc as well as the upper and

lower portion of vertebrae. Thus, typical pyogenic spondylitis affects two adjacent vertebrae and the intervening disc. The spines infections could involve all levels of the spines. The result shows the lumbar spine (45–50%) is the most common site, and the rest of the level is the thoracic (35%), cervical (3–20%), and sacral regions [14].

1.5.2 Radiographic Imaging

Radionuclide studies showed more sensitive results than radiograph images in early stages. Bone scans reveal little anatomical details and can be positive in osteoporotic fractures and neoplastic disease. Magnetic resonance imaging (MRI) is known as the gold standard for detecting pyogenic spondylitis. The infection commonly begins at the anterolateral vertebral body near the endplate [15]. Associated edema is declared and includes much of the vertebral body and intervertebral disc. MRI is also a dependable method for evaluating and assessing the spinal canal, especially the epidural space and spinal cord. Epidural abscess with neurological deficit is a surgical emergency [13].

1.5.3 Differential Diagnosis with Pyogenic Spondylitis Versus Tuberculous Spondylitis

Infective spondylitis may result from hematogenous spread, direct external inoculation, or contiguous tissues. The hematogenous arterial route is predominant in pyogenic spondylitis, starting infection from various sites to the vertebral column. Contrary to pyogenic infections, tuberculous infection usually spread from the venous system such as Batson's venous plexus. In the case of tuberculous spondylitis, there are few clinical symptoms such as fever, pain, and swelling due to infection, and the disease progresses gradually. However, infective spondylitis is highly likely to be accompanied by severe pain and high fever [16].

The most characteristic features of tuberculosis spondylitis are (a) predominantly pattern of bone destruction, (b) relatively preserved disc due to a lack of proteolytic enzymes in mycobacteria [17], (c) enhanced focal and heterogeneous contrast of vertebral bodies, (d) well-defined perivertebral regions of abnormal signal intensity, and (e) rim enhancement of vertebral intraosseous lesion in the sagittal plane. On the other hand, the common findings of pyogenic spondylitis are (a) mainly the appearance of intervertebral disc disease, (b) mild to moderately peridiscal bone involvement, (c) relatively diffuse and homogeneous enhancement of the vertebral body, (d) ill-defined abnormal signal intensity paraspinal region, and (e) intervertebral rim enhancement findings. If three or more of the five criteria are found, it is strongly suggestive of tuberculosis or pyogenic spondylitis [17].

Case 1.5
An 87-year-old woman visited an outpatient clinic with her back pain for 4 months, and tenderness was elicited at left lower back area. The laboratory findings showed elevated ESR and CRP. MR showed enhancement of bone marrow and disc in L4–L5 with bilateral paravertebral abscess and phlegmon (Fig. 1.12a). Three-phase bone scan with bone SPECT/CT showed diffuse increased perfusion and bone uptake in the L4 and L5 vertebare (Fig. 1.12c–f). Pyogenic spondylitis was diagnosed by bone biopsy.

Case 1.6
A 64-year-old man visited an outpatient clinic for back pain and left leg numbness sensation with fever. Laboratory results showed positive for the blood TB-specific antigen. MR revealed well-defined paravertebral soft tissue abscess formation (Fig. 1.13), combined with edematous bone change and heterogenous cortical loss but relatively preserved disc. Three-phase bone scan and following SPECT/CT showed increased perfusion and bone uptake in the L3–L4 bodies with increased perfusion in the paravertebral soft tissues at the L3–L5 level (Fig. 1.13c).

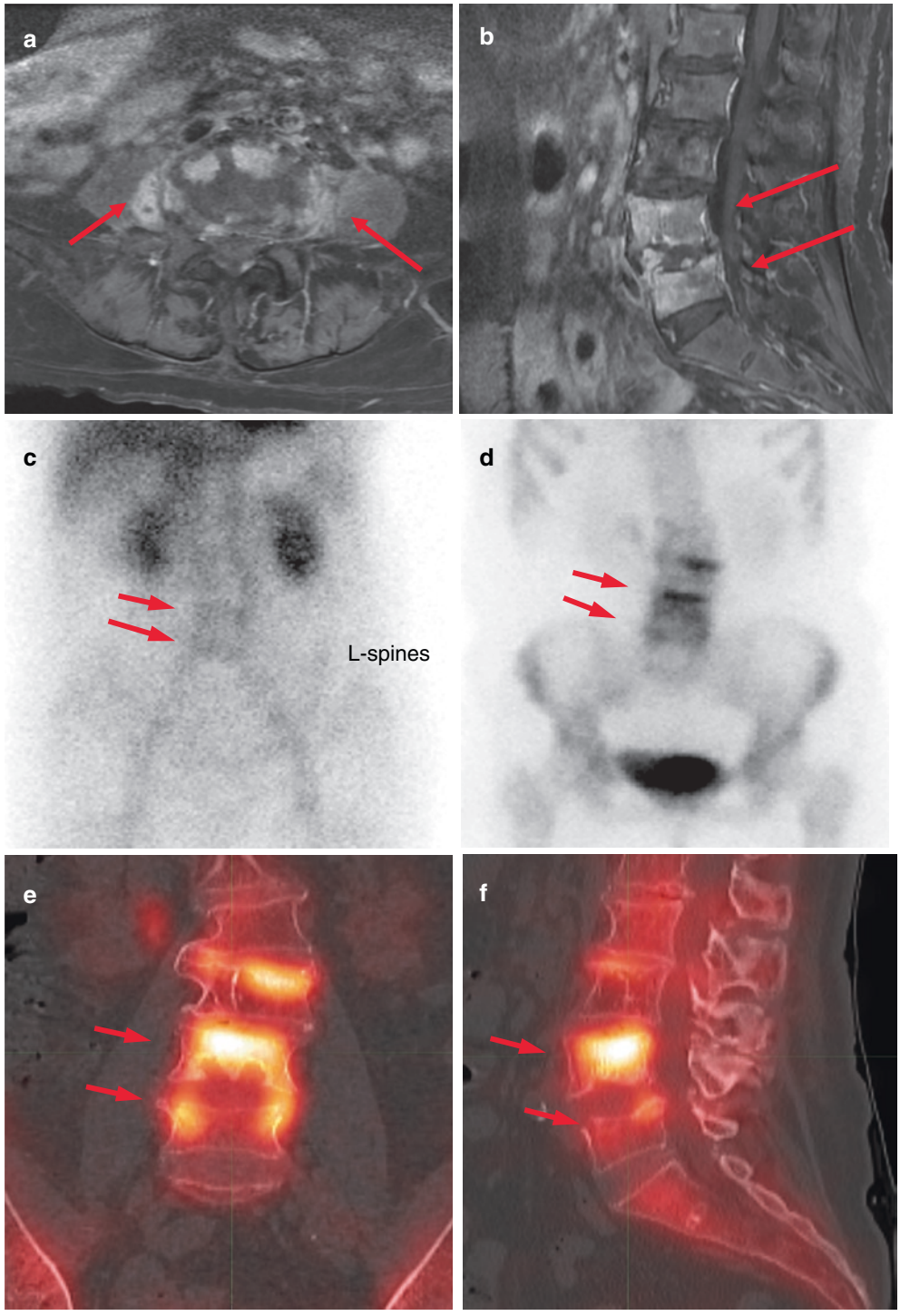

Fig. 1.12 Contrast-enhanced MR images of L-spines show enhancement of the bilateral paravertebral abscesses (arrows in **a**) and two adjacent vertebrae with the intervening disc (arrows in **b**). Three-phase bone scan (**c**, **d**) and bone SPECT/CT (**e**, **f**) show increased perfusion and bone uptake in the L4 and L5 spines

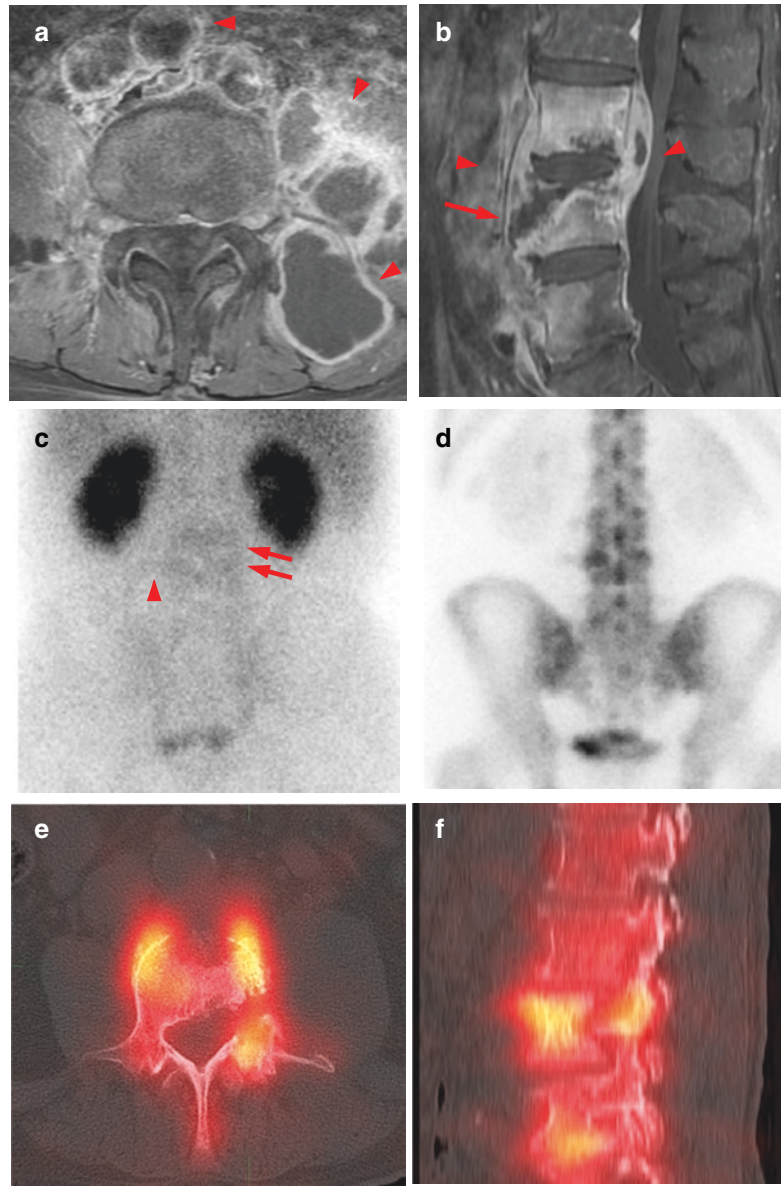

Fig. 1.13 Contrast-enhanced MR images of L-spines show multiple paravertebral abscesses (arrowheads in **a**, **b**) and bone loss in the L3 (arrow in **b**). Posterior view of three phase bone scan shows subtle increased soft tissue perfusion in the left paravertebral area (arrowhead in **c**), mildly increased perfusion in the L3 and L4 (arrows in **c**), and relatively subtle increased bone uptake in the L3 and L4 (**c**, **d**). Bone SPECT/CT shows bone defect with mildly increased bone uptake in the L4 body (**e**, **f**)

Case 1.7

A 58-year-old man visited an outpatient clinic due to back pain after receiving interbody fusion 4 months ago. He was treated with antibiotics intensively, however his clinical symtpoms were not improved. MR showed hyperenhancement on L2 and L3 bodies and paravertebral soft tissue (Fig. 1.14a, b). Prosthetic loosening and associated edema were also revealed. Three-phase bone scan showed increased perfusion and bone uptake in the left side of screw of L3 body (Fig. 1.14c, d). White blood cell scan with SPECT/CT demonstrated cold defect in L2 and L3 spines (Fig. 1.14e, f) suggesting osteomyelitis. In chronic sponydlitis, the presence of necrotic bones can reduce the effectiveness of antibiotic treatment by preventing the antibiotics from entering the tissue, which in turn may produce a new inflammation or thrombosis and result in more severe necrosis.

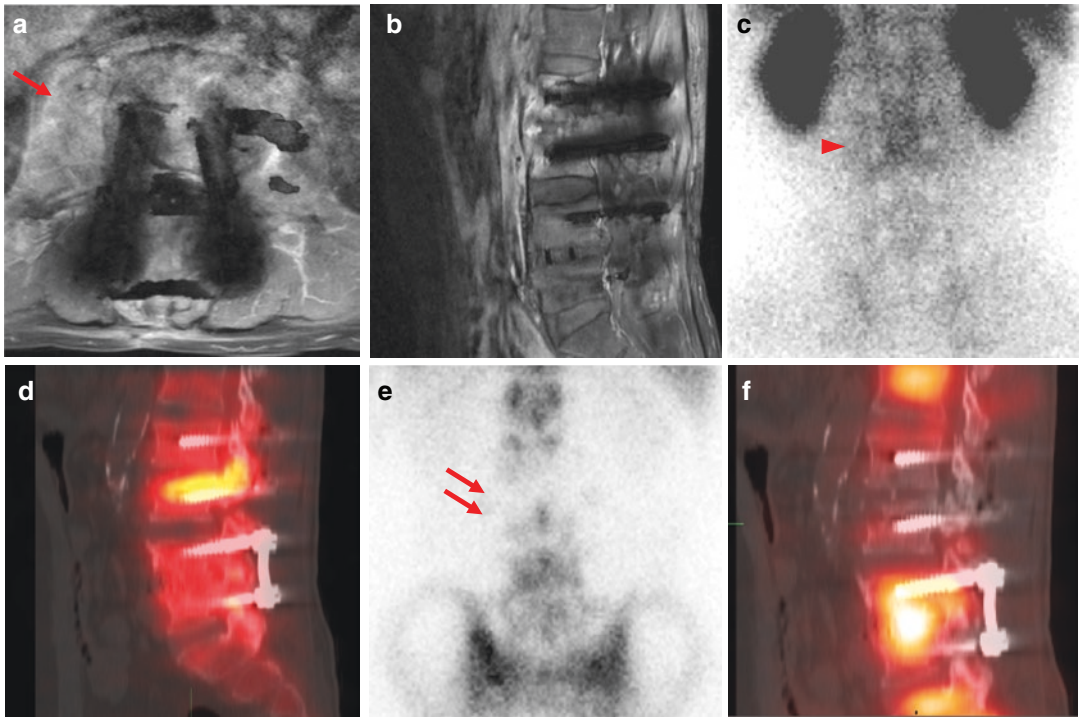

Fig. 1.14 Contrast-enhanced MR images of L-spines show enhancement of paravertebral soft tissue at the level of L2–L3 (**a**, **b**). Bone scan and SPECT/CT show increased perfusion and bone uptake in the level of L2-L3 (**c**, **d**). WBC scan and SPECT/CT show cold defects in the L2 and L3 bodies in comparison to the uptake in other lumbar spines suggesting osteomyelitis (**e**, **f**)

Case 1.8

A 78-year-old man presented to the outpatient clinic with neck pain and fever. Laboratory study showed elevation of ESR and CRP. MR demonstrated decreased intervertebral space and paravertebral abscess at C6–C7 level with anterior epidural abscess formation (Fig. 1.15a). Three-phase bone scan revealed mildly increased perfusion and bone uptake in the lower C spines at anterior and oblique views (Fig. 1.15c–e). Empirical antibiotic treatment was applied for pyogenic spondylitis. Bone scan for cervical spondylitis should be carefully reviewed, because it has a relatively low incidence and bone uptake of C spine lesion is easily obscured in comparison to the thoraco-lumbar spines.

Teaching Points

- Bone scan can provide a useful assessment of vascular supply including small arteries and capillary vessels in diabetic foot.
- Even CRP can be less specific after post-operative infection and antibiotics therapy, combining WBC scan with three-phase bone scan can be useful for detecting peri-prosthetic infection accurately.
- Early detection and proper treatment of Charcot foot are important for prevention of disease progression and prediction of disease prognosis.

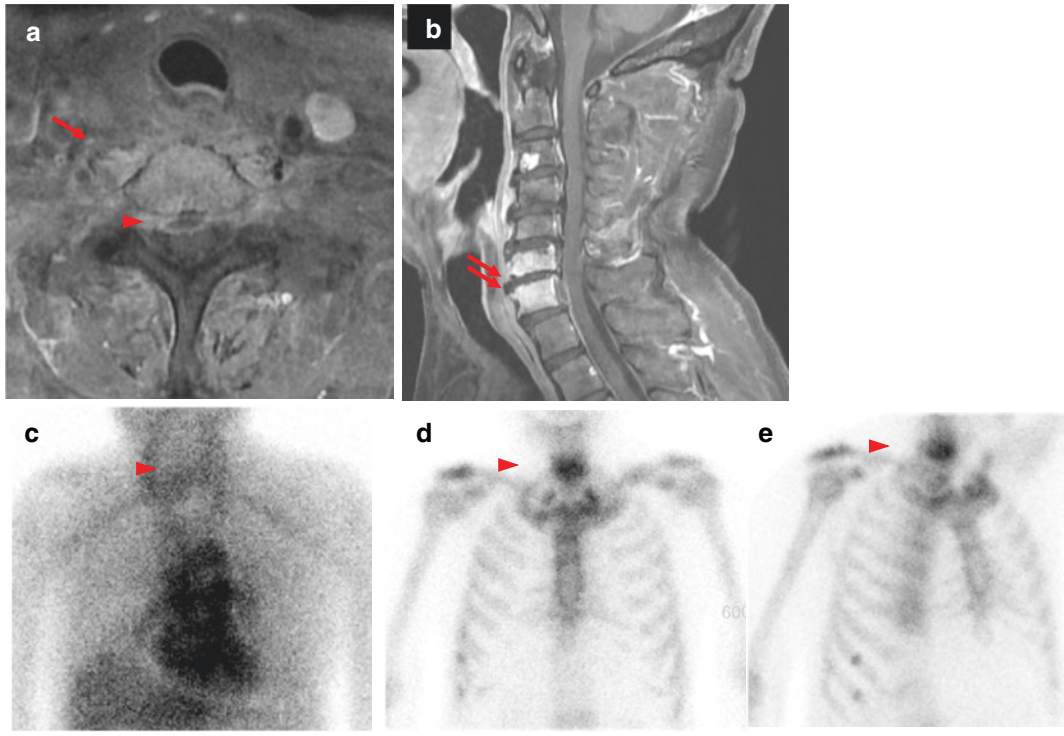

Fig. 1.15 Contrast-enhanced MR images of C-spines show paravertebral abscess at the level of C4–C7 (arrow, double arrows in **a, b**) and anterior epidural abscess at the C6–C7 level (arrowheads in **a**). Suspicious mildly increased perfusion in the lower C spine area and increased bone uptake in the lower C spines on the three-phase bone scan (arrowheads in **c**–**e**)

- SPECT/CT allows accurate anatomical correlation with CT to functional information with SPECT; it can improve the diagnostic accuracy for inflammation or infection, traumatic injury, and degenerative change of the foot and ankle.
- WBC SPECT/CT can help increase diagnostic accuracy for infections in violated bone lesions compared to relatively low sensitivity of three-phase bone scan.
- Malleolar bursitis is a rare inflammatory disease involving the soft tissue of the ankle and foot.
- Blood pool and delay bone phase SPECT/CT can help discriminate the involvement of bony inflammation or infection.
- Pyogenic spondylitis involves various clinical entities such as pyogenic spondylodiscitis, septic discitis, vertebral osteomyelitis, and epidural abscess.
- Pyogenic spondylitis affects two adjacent vertebrae and intervening disc and infectious spondylitis can show cold defect in WBC scan.
- Differential diagnosis between pyogenic spondylitis and tuberculous spondylitis can be made by clinical symptom and imaging findings.

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Septic Arthritis

2

You Mie Han

Abstract

Septic arthritis is a painful infection in a joint induced by an infectious agent. Release of proteolytic enzyme from bacteria and inflammatory cells may cause articular cartilage damage within hours. So, prompt diagnosis and treatment are essential to prevent significant morbidity and mortality. Although arthrocentesis is commonly used to make an accurate diagnosis of septic arthritis, imaging modalities are helpful to evaluate the disease. Whole-body or three-phase bone scintigraphy has been widely used in diagnosis of septic arthritis. Although its findings are nonspecific, it is a sensitive study to diagnose septic arthritis and detect associated bone erosion or osteomyelitis under suspicion of infectious condition. A total of six cases of septic arthritis are presented in this chapter: four bacteria-confirmed and one bacteria-suspected infections and one tuberculous infection. Each case contains a closely correlated combination of images of three-phase bone scintigraphy, simple radiography, MRI, and/or PET/CT.

Keywords

Septic · Infectious · Arthritis · Tuberculous Bone · Scintigraphy

2.1 Etiology and Pathophysiology

Septic arthritis is also known as infectious arthritis or pyogenic arthritis, a painful infection in a joint induced by an infectious agent. It can be caused by bacterial, viral, mycobacteria, or fungal infections. The most common causative organism is *S. aureus* (*Staphylococcus aureus*). An organism can enter the joint by the blood stream from another infected body focus, by contiguous spread from infected periarticular tissue, or by direct inoculation via penetrating injury, surgery, or injection. Knees and hips are commonly affected joints, but septic arthritis can affect other joints including both large and small joints. Symptoms and signs of septic arthritis are acute pain, swelling, redness, and heating sensation on the affected joint with discomfort and limited range of motion. Active bacterial proliferation resulted from invasion of the highly vascular synovium. Release of proteolytic enzyme from bacteria and inflammatory cells may cause articular cartilage damage within hours. Furthermore, increased intra-articular pressure from accumulation of the purulent fluid results in

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