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Infection of the appendicular skeleton

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Abstract In relation to the clinical course, infection in bone can be divided into acute, subacute and chronic osteomyelitis. The diagnosis of acute osteomyelitis is often challenging but can best be made by correlating radiography, bone scintigraphy and MRI with clinical information. Radiography should routinely be supplemented by sonography in the newborns and infants, if applicable. Brodie's abscess, which is clinically a subacute form of osteomyelitis, is best diagnosed by the combination of radiography and MRI. Chronic osteomyelitis is divided into primary haematogenous forms and exogenous, mostly post-traumatic, osteomyelitis. In the majority of patients, post-traumatic osteomyelitis is a clinical diagnosis; however, in a number of patients only the correlation of clinical findings, blood tests and imaging reveals the correct diagnosis. Often, MRI and scintigraphic methods, such as scanning with labeled leucocytes, together establish the diagnosis. Chronic recurrent multifocal osteomyelitis may mimic bacterial osteomyelitis but is a distinct disease probably associated with the SAPHO syndrome.

Keywords Bone · Infection · Radiography · Radionuclide imaging · Magnetic resonance

Introduction

Osseous infection can be caused by haematogenous spread of organisms to bone (haematogenous osteomyelitis) or by direct local invasion by bacteria. Haematogenous spread of organisms follows bacteremia due to, for example, urogenital infections, enteritis, cholangitis or endocarditis; often the infective focus is not identified. Direct spread to bone can occur from bacterial invasion, e.g. through penetrating wounds or postoperative infection. This route is most likely after contaminated soft tissue trauma, as well as in diabetic patients with plantar ulcers or in bedridden patients with decubitus ulcers [1].

Microorganisms implicated in osseous infections are:

1. *Staphylococcus aureus* in 80–90% of cases
2. *Streptococcus B* (in the newborn)
3. *Pseudomonas*, which is more frequent in drug addicts than in the general population
4. *Salmonella*, in patients with diabetes mellitus or sickle cell anaemia
5. *Mycobacterium tuberculosis*
6. *Spirochaetes*
7. Fungi (*Candida*, *Actinomyces*)
8. Viruses
9. Helminths (e.g. *Echinococcus*)

Infection develops as the organisms spread into the perivascular interstitial tissue, leading to a leucocytic infiltration that permeates the bone marrow. The dissemination progresses along Volkmann's canals and through the Haversian system. The infiltration leads to vascular compression and compromised nutrition of the bone marrow. Combined with the effect of the bacterial toxins, this ultimately causes osteonecrosis. The necrotic tissue may be resorbed and replaced with new bone. If this process fails, purulent cavities (abscesses) and/or sequestra are formed [2].

Based on the clinical course, osteomyelitis can be divided into:

1. Acute osteomyelitis with a septic clinical course
2. Subacute osteomyelitis. This term is mostly used in the English-language literature [3, 4, 5], reflecting the low-grade clinical course of a hypovirulent infection. Brodie's abscess in its different forms is a classical example. This entity also highlights the fact that a clear division between subacute and chronic osteomyelitis does not exist; however, chronic osteomyelitis often progresses from an uncontrolled acute septic infection, whereas Brodie's abscess appears to be subacute ab initio. In addition, if the septic phase of acute osteomyelitis passes without symptoms, subacute or chronic osteomyelitis can become apparent. Failure to eliminate an acute infection completely may be due to therapeutic measures that have been inadequate or inappropriate, or to organisms that are especially resistant to antibiotics.
3. Chronic osteomyelitis. Primary chronic haematogenous osteomyelitis, as well as hypovirulent forms of Brodie's abscess, must be distinguished from secondary exogenous, chronic osteomyelitis. The secondary form tends to be characterized by an acute onset and recurrent exacerbations with fever, pain, tenderness and warmth, whereas the primary chronic form tends towards vague symptoms and clinical signs with evidence of a longstanding process on imaging evaluation.

Increasingly, the bacterial aetiology of the majority of the so-called "primarily chronic" forms of osteomyelitis is being challenged. With the exception of Brodie's abscess, pathogenic organisms are rarely cultured from specimens and bacteraemia is never demonstrated. This finding has led to the concept of the so-called reactive, osteomyelitis [6]. A heterogeneous group of entities, such as "plasma cell osteomyelitis" or "chronic sclerosing osteomyelitis", may be encompassed by this term.

It is almost certainly also the case that "chronic recurrent multifocal osteomyelitis" is another manifestation of non-bacterial osteomyelitis (see below).

Acute haematogenous osteomyelitis

Acute haematogenous osteomyelitis occurs predominantly in the paediatric age group but shows increasing incidence again after the age of 50 years. Depending on the age of manifestation, a distinction is made between newborn, juvenile osteomyelitis, and adult haematogenous osteomyelitis [1].

The challenge for imaging is to detect and diagnose the condition in its early stages and enable early onset of appropriate treatment and thereby improve the prognosis. Acute osteomyelitis is treated with high doses of antibiotics and, if necessary, surgery. Imaging must reveal the presence of abscesses or sequestra, since they may not be reached by antibiotics and may require early surgical intervention.

Osteomyelitis in the newborn

Osteomyelitis is rare in the newborn infant. It is usually an acute disease which has a predilection for the proximal and distal femoral metaphyses. Precipitating conditions are infections of the umbilicus, ear, nose and throat, often by streptococci. If untreated, the infection spreads rapidly through the Haversian canals into the subperiosteal space and/or across the still-patent meta-epiphyseal vessels to the epiphysis. The result may be septic arthritis with capsular empyema, enzymal destruction of the epiphyseal cartilage and epiphyseal separation, ultimately causing severe joint deformity [7].

Juvenile osteomyelitis

From the second year of life, the metaphyseal vessels no longer penetrate the growth plate and form dilated loops in the metaphysis through which the flow of blood is sluggish, providing favourable conditions for extravasation of bacterial organisms to the bone marrow; therefore, osteomyelitis in children usually starts as a metaphyseal inflammatory focus. The absence of vascular penetration produces a physeal barrier which prevents spread to the epiphysis in most cases, so that the infection can spread into the joint only if the metaphysis is within the joint capsule, as in the hip and knee. In the peripheral bones the suppurative process rapidly penetrates the thin cortex and spreads along the subperiosteal space with elevation of the periosteum [8].

The organisms found in juvenile osteomyelitis are predominantly staphylococci, especially *Staphylococcus aureus*.

Adult osteomyelitis

Acute haematogenous osteomyelitis in the adult is relatively rare and primarily affects flat bones, vertebral bodies and the diaphyses of the tubular bones. The suppurative process can involve the entire medullary space (medullary phlegmon).

In adults, the growth plates are closed and the vascular connection between the metaphysis and epiphysis has been restored with subchondral extension of the vessels. Spreading of metaphyseal infection into the joint capsule therefore is more frequently seen. Since the metaphyseal cortex is thick and the overlying periosteum is firmly at-

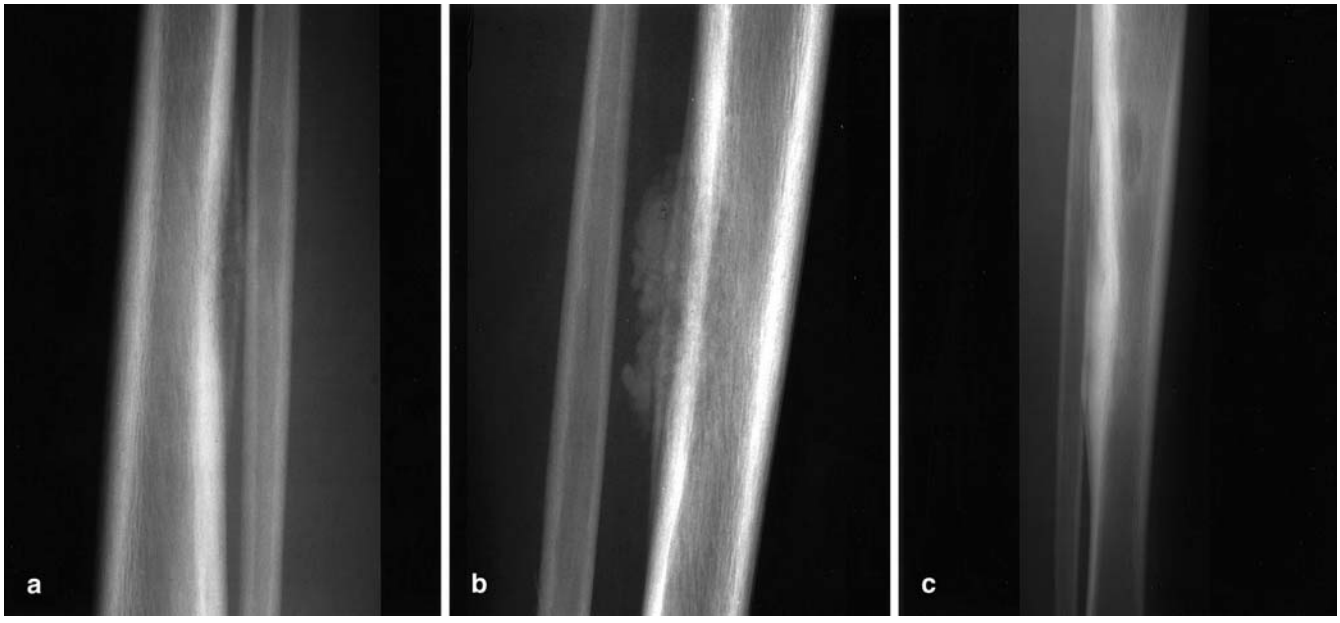

Fig. 1 Acute osteomyelitis of the tibia at different stages of disease. **a** Initial radiograph shows thinning of the cortex with stippled lucencies. A lamellar periosteal reaction is present. **b** Ten days later, after starting antibiotic therapy, an additional cloud-like, dense periosteal reaction has developed. **c** Six months later, a follow-up study shows a lucent defect with periosteal and endosteal thickening of the cortex. The patient was symptom free and had returned to normal activity

tached by thick Sharpey's fibres, subperiosteal abscesses rarely develop [1].

Imaging features of acute haematogenous osteomyelitis (all ages)

Radiography

It takes from 10 to 21 days for an osseous lesion to become visible on conventional radiography, because a 30–50% reduction of bone density must occur before radiographic change is apparent [9]; however, radiography is always valuable as a primary imaging technique as it can exclude other diagnoses. It is also valuable to review the effect of therapy. Several radiographic findings have to be considered [8]:

1. Localized osteopaenia and trabecular destruction are early signs of a suppurative acute process in the bone.
2. The type and extent of cortical destruction is variable (Figs. 1, 2, 3). A wide spectrum is encountered, ranging from a solitary radiolucency to irregular, multiple radiolucencies ("mottling") to a permeative pattern. The individual lesions are generally indistinct and irregular in outline.
3. Lamellated periosteal reactions are invariably present (Figs. 1, 2, 3).
4. The reparative phase during therapy is characterized by endosteal and periosteal new bone formation, development of surrounding sclerosis and sometimes large osteosclerotic areas (Fig. 1).
5. Soft tissue changes, such as swelling and obliteration of tissue planes, are rarely of diagnostic value in adults.
6. In newborns and infants, however, loss of normal fat planes within days of the onset of symptoms may be an early sign of soft tissue swelling. In this age group lamellated periosteal changes are generally discernible before any bone destruction. A late manifestation is the ballooned metaphysis, sometimes with involvement of the epiphysis.

Nuclear medicine

The basic scintigraphic examination in haematogeneous osteomyelitis is the sensitive but non-specific bone scan (Tc-99m phosphonates). More specific information can be revealed by a white blood cell scan (e.g. ^{111}Indium-labeled leucocytes). F-18-FDG-PET might play a role in the future but its value is not yet defined (J. Sciuk, pers. commun.).

Three-phase bone scintigraphy with Tc-99m diphosphonate

In osteomyelitis the bone scan should always be acquired as a three-phase study. Acute osteomyelitis produces strong focal activity in all three phases. Increased blood

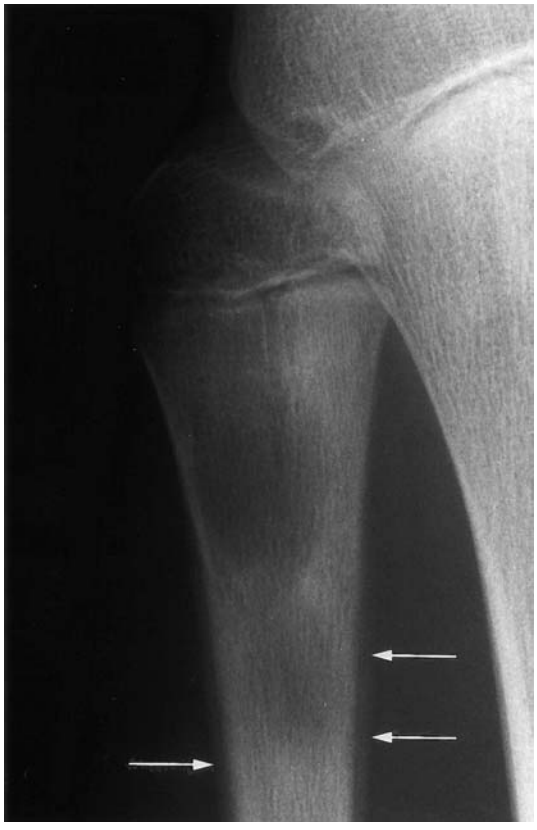

Fig. 2 Acute osteomyelitis of the proximal fibula in a 14-year-old boy. The radiograph shows rounded lucencies and lamellar periosteal reaction (*arrow*)

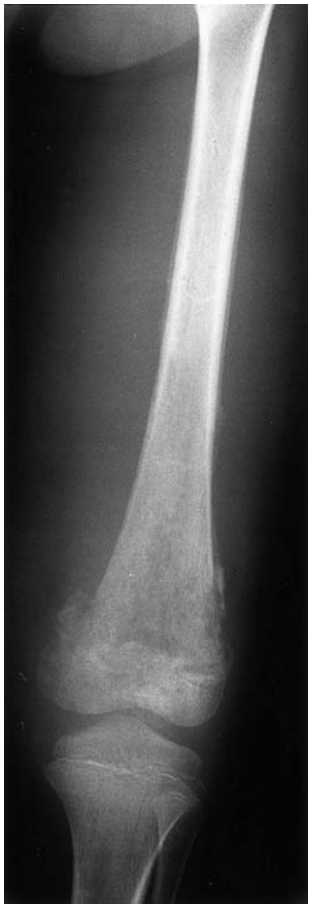

Fig. 3 Acute osteomyelitis of the femur. The radiograph shows a moth-eaten appearance of the distal metaphysis and extensive meta-diaphyseal periosteal reaction

pool activity without osseous uptake in the bone phase suggests that the inflammation is confined to the soft tissues. The reported sensitivity of bone scintigraphy in the detection of osteomyelitis varies from 32 to 100%. Technical improvements, such as high-resolution cameras and single photon emission computed tomography (SPECT), have improved diagnostic sensitivity. Reported specifity is lower and dependent on age, clinical setting and location of the infective process [10].

As a cautionary note, in the newborn, acute osteomyelitis can lead to a decreased uptake, resulting in a false-negative bone scan or a "photon-deficient" lesion. This can be attributed to increased intramedullary pressure. Another false-negative finding can be introduced by the inability to discern an increased uptake against the physiologically accentuated uptake in the growth plates [11].

White blood cell scintigraphy

Many different techniques for white blood cell (WBC) studies are available. The gold standard in osseous infections is the labelling of separated leucocytes in vitro with ^{111}Indium [12, 13]. Alternatively in vivo labelling of neutron-like granulocytes by a monoclonal antibody is easier to perform and achieves similar accuracy [12].

Any tracer accumulation outside the expected physiological pattern suggests osteomyelitis. Used alone or combined with three-phase bone scan, the WBC scan significantly increases the specifity [13]. Since non-specific WBC accumulation can occur in both, tumours and fractures, correlation with current conventional radiographs is necessary.

Sonography

Ultrasound cannot directly access bone marrow abnormalities present in osteomyelitis but can document osteomyelitis indirectly by identifying periosseous soft tissue abnormalities [14].

The acoustic properties of neonatal anatomy are especially suitable for sonography. The very first sonographic sign, seen even before any periosteal reaction, is edematous swelling of the deep soft tissues. This is followed by the sonographic detection of a thin hypoechoic fluid layer, which elevates the periosteum. This finding can progress to a space-occupying abscess. [15, 16]. The

value of sonography, however, should not be over-rated since the sonographic findings described, depending on the site examined, are not always detectable. Moreover, subperiosteal fluid collections are non-specific and may be encountered in acute bone infarction and sickle cell patients [17].

Sonography becomes less important with increasing age of the patient, and becomes an examination that aids in the evaluation of soft tissue changes induced by acute osteomyelitis. Abscesses, cysts and haematomas are excellently demonstrated as hypoechoic or anechoic lesions and as such are suitable for sonographically guided aspiration [14]. Any associated cortical destruction may be delineated if the sonographic conditions are optimal. Any diffuse soft tissue swelling is characterized by echogenic thickening of the subcutaneous tissue. Diffuse soft tissue infection and abscess formation can confidently be differentiated by sonography.

Furthermore, sonography can crucially detect a joint effusion, which might not only explain the pain and restriction of motion, but frequently also prompts a diagnostic joint aspiration.

MRI

Examination technique

The most suitable sequences for screening are the short tau inversion recovery (STIR) or the T2-weighted fat-suppressed fast spin-echo (TSE) sequence because of excellent visualization of inflammatory oedema [7]. These sequences should be supplemented by T1-weighted spin-echo (SE) images, which provide excellent anatomic detail. Unless contraindicated, intravenous gadolinium-based contrast medium should be administered, since this technique has been found helpful to distinguish abscesses or necrotic bone (sequestrum) from inflammatory oedema [18]. Furthermore, it may help to differentiate osteomyelitis from malignancy [19]. T2-weighted fast ("turbo") SE sequences without fat suppression should be avoided since they tend to mask intramedullary oedema, unless the image contrast can be augmented by frequency-selective fat suppression.

Morphology and signal pattern

The affected areas are of high signal intensity on STIR images, with a corresponding low-signal intensity on T1-weighted images. The inflammatory areas are usually large and irregularly outlined. As a general rule, periosseous oedema is consistently present and a delicate high signal rim should be sought on axial sections. This rule, however, applies only to STIR or T2-weighted TSE images with fat suppression. As long as high-resolution coils are used, STIR and T2-weighted images invariably reveal intracortical areas of increased signal intensity, also seen on post-contrast images [8].

Intraosseous abscesses generally are sharply demarcated and display a rim of low-intensity signal on STIR and T2-weighted SE images. Strong peripheral contrast enhancement without central contrast enhancement confirms the presence of an abscess. Necrotic sequestra also fail to show contrast enhancement and on STIR or T2-weighted images show regions of signal void, in contrast to the high signal intensity of an abscess.

As with other imaging methods, differentiating osteomyelitis from septic arthritis can be difficult. Septic arthritis can induce oedematous changes in the epiphysis and metaphysis, without evidence of underlying osteomyelitis, but the reverse also applies. No imaging modality has thus far solved this dilemma, and image-guided joint aspiration remains unsurpassed in the diagnostic evaluation [20]. In contrast, MRI is generally quite successful in differentiating between osteomyelitis and primary soft tissue inflammation; the latter is not (except late in the disease) associated with bone marrow signal alterations [21].

It is noted that the bone marrow findings of acute osteomyelitis on MR imaging, although highly sensitive [22, 23], are nonspecific. Noninfectious inflammatory and metabolic conditions of osseous tissue, bone contusions, stress fractures, healing fractures, osteonecrosis and tumours can all produce signal alterations in different sequences similar to those seen in osteomyelitis. This holds true for the long bones of the appendicular skeleton but is especially true for the bones of the foot. It is the experience of this author as well as others [18] that, when faced with the diagnostic uncertainty in the evaluation of the abnormal marrow findings, the most useful additional information is usually gathered from a review of the patient's history and from findings at physical examination.

Role of imaging in diagnosing acute osteomyelitis

If acute osteomyelitis is suspected clinically, conventional radiography remains the method of choice for initial evaluation. Although radiographs often appear normal during the first 1–2 weeks, they are valuable for the exclusion of other disorders such as tumours or foreign bodies and as a background for the evaluation of therapy. Radiography should routinely be supplemented by sonography in the newborn and in infants unless the clinically affected area cannot be approached sonographically. If clinically indicated, evaluation should continue with MRI or scintigraphy. These competing modalities should be used alternatively and not additively. Due to its higher sensitivity and specifity, MRI is to be preferred, except when multiple lesions are suspected. Scin-

tigraphy can show the whole skeleton in one examination and should also be favoured if there is no definite clinical localization of the lesion. The MRI should always be performed if an abscess is suspected.

Computed tomography no longer plays a role in the early detection of acute osteomyelitis but remains indicated in the search for sequestra or foreign bodies [8].

Subacute osteomyelitis

As discussed previously, no clear distinctions between acute, subacute and chronic osteomyelitis are possible. An incompletely healed acute infection may proceed to subacute or even, after months and years, chronic osteomyelitis. This happens more often in adults than in children.

Subacute osteomyelitis may present as an intramedullary abscess—Brodie's abscess [4]. It is usually a round or ovoid abscess cavity, measuring approximately 1–4 cm in diameter and often progressing to intraosseous tunnelling but rarely forming a fistulous tract. It primarily involves the metaphyses of the leg. *Staphylococcus aureus* is the most common pathogen. In approximately 50% of the cases the culture is positive [24]. The clinical findings can be minimal and patients may not be seen by a physician, even for years.

Radiography

Primary subacute osteomyelitis is characterized by a mixture of osseous destruction and reactive sclerosis, and more or less well-defined osseous contours. Brodie's abscess usually presents as a well-defined round or ovoid radiolucency with thick surrounding sclerosis (Fig. 4).

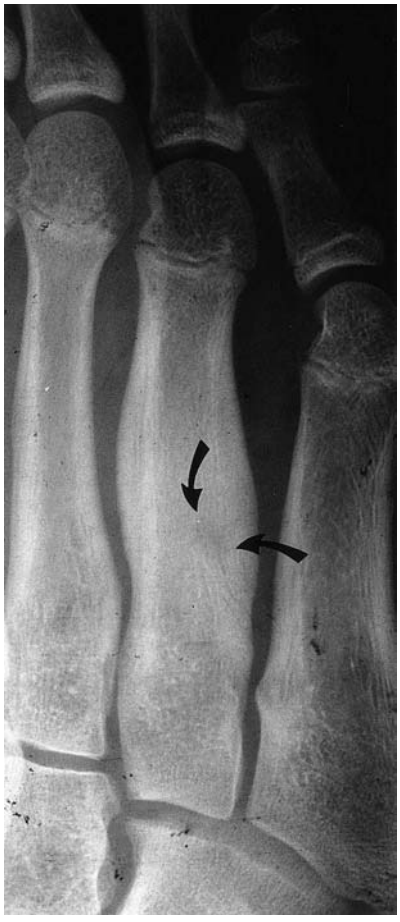

Fig. 4 Brodies's abscess of the 4th metatarsal bone in a 12-year-old boy. The radiograph shows a small lucency (*arrow*) and extensive solid periosteal reaction

MRI

Brodie's abscess generally appears as a defect without central contrast enhancement; however, the defect can enhance if the abscess cavity is filled with granulation tissue rather than pus. The lesion has a high signal intensity on STIR and T2-weighted images. The sclerotic rim is of low signal intensity but enhances faintly [21]. Lesions penetrating the cortex are characterized by signs of intramedullary inflammation as well as some degree of concomitant soft tissue inflammation.

Chronic osteomyelitis

As discussed previously, primary chronic haematogenous osteomyelitis, without evidence of preceding clinically overt acute or subacute infection, must be an extremely rare event. Most of these cases have to be regarded as non-bacterial inflammatory bone lesions; however, inadequate antibiotic treatment and decreased body immunity (such as in AIDS or as a result of chemotherapy) may change an acute or subacute bony infection to chronic osteomyelitis [21].

The dominating radiographic feature is usually an irregular osseous sclerosis with loss of the normal trabecular bone structure. Hyperostosis of the bone is common, mainly due to a solid periosteal reaction which has become incorporated into the cortex of the involved bone. During active infection, the sclerotic region often contains lytic areas, representing intraosseous abscesses or collections of granulation tissue, and sequestration with the formation of fistulae may occur.

The role of imaging is to establish the diagnosis of primary chronic osteomyelitis and to detect and delineate areas of residual active infection in patients with known chronic osteomyelitis. Reliable signs of active infection are the demonstration of bone marrow abscess, sequestra and sinus formation. Both MRI and scintigraphy can be

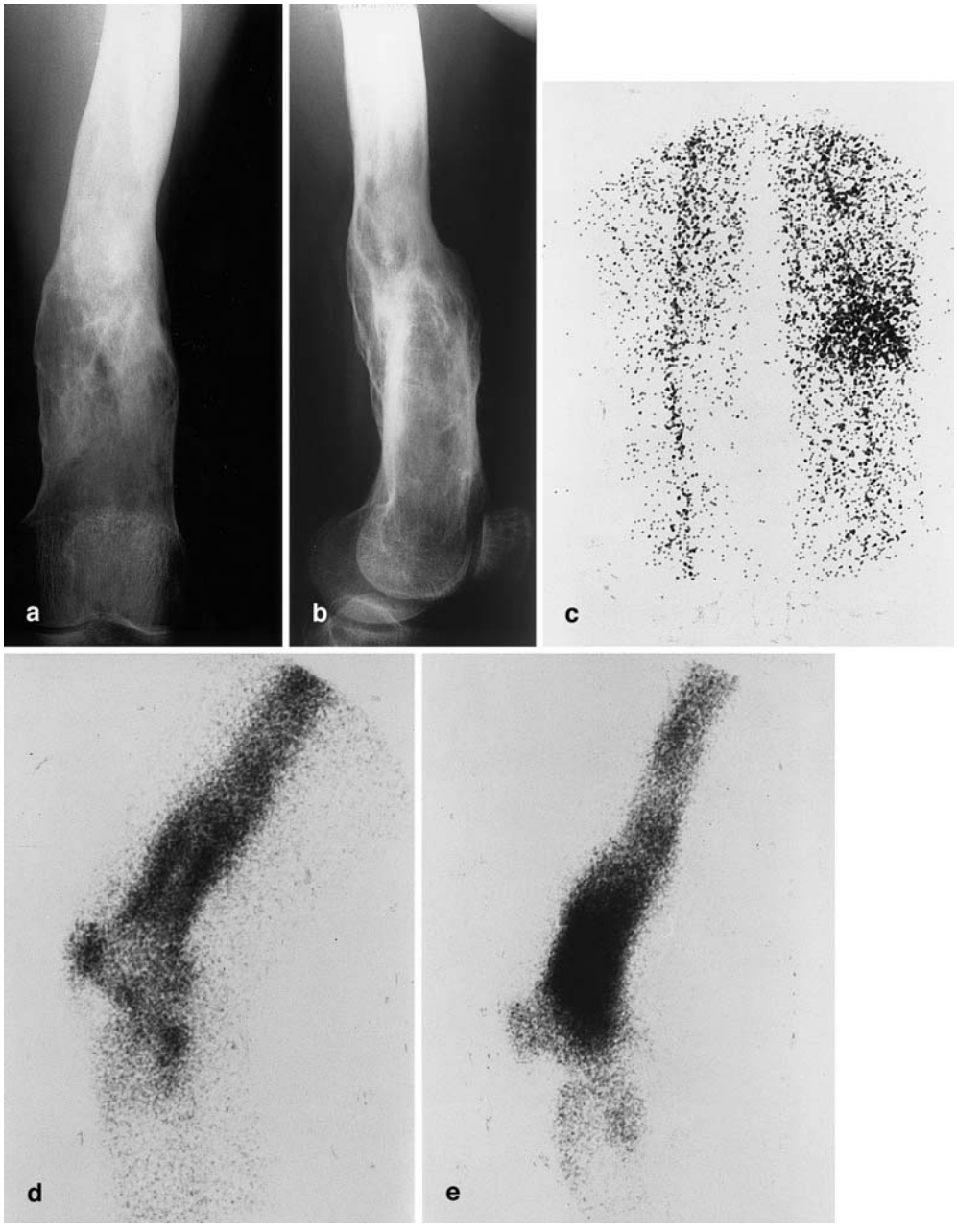

Fig. 5 Chronic posttraumatic osteomyelitis of the distal femur. Note osseous modeling deformity, cortical thickening and irregularity, sclerosis and radiolucencies. A definite diagnosis of an acute exacerbation of chronic osteomyelitis cannot be made on the basis of the radiographs (**a**, **b**). Three-phase bone scintigraphy shows uptake in the flow study (**c**), in the blood pool phase (**d**) and the static phase (**e**)

used to detect and exclude new infectious foci. The MRI provides the best diagnostic sensitivity and specificity, and superior contrast, as well as anatomical resolution in both marrow and soft tissue. This improves preoperative planning by providing reliable assessment of the intramedullary and extracompartmental extent of the infection. Active foci appear as low-intensity areas on T1-weighted images with increased intensity on STIR and T2-weighted images. Granulation tissue surrounding the infection enhances on contrast-enhanced images. The MRI should also exclude any suggestion of sequestration which might require a CT examination.

Exogenous (post-traumatic) osteomyelitis

Osteomyelitis due to spread of microorganisms from contiguous sources of infection, direct implantation of bacteria or postoperative contamination may be acute, subacute or chronic depending on the resistance of the host and the virulence of the bacteria [25].

Exogenous infection is usually a local infection of the bone (osteitis). The spread of the infection depends not only on the number and virulence of the organisms and resistance of the patient, but also on the local conditions, such as the extent of soft tissue damage, the status of the

nutrient blood supply, the stage of bone healing and presence of any introduced foreign material. Despite speedier trauma care and improved surgical techniques, the infection rate of open fractures still remains between 2 and 16% [26, 27]. Impaired vascularization and altered stability of the traumatized soft tissues and bones are conditions favourable for the insidious development of chronic osteomyelitis.

For many patients, chronic post-traumatic osteomyelitis remains a life-long disease. Chronic osteomyelitis can persist without overt clinical signs or symptoms, mimicking a state of cure; however, the danger of recurrence remains present throughout the rest of the patient's life [28].

Radiography

The radiographic findings of post-surgical infection after trauma or surgical procedures depend on the level of activity of the infection. In acute infection, comparison with the immediate post-surgical radiographs is critical for early diagnosis. The osseous structures appear rarefied at a time when signs of callus formation and osseous bridging should be present, and any inserted hardware presents a radiolucent halo. Furthermore, infection can elicit linear periosteal reaction. Careful analysis of the inserted screws, pins and plates reveals an indistinct osseous interface to the surrounding halo, in contrast to the sclerotic border of the halo caused by loosening [19].

The generally protracted course of the infection induces endosteal and periosteal reactions. In the true sense of its meaning, a "variegated picture" develops, whereby later in the process new bone formation largely predominates over the destructive changes (Fig. 5). Demonstration of a sequestrum confirms the diagnosis [29] and has therapeutic implications, since it must be surgically resected. Instillation of contrast medium into the fistulous tract often documents the communication between the tract and a sequestrum or a foreign body. Tumeh et al. reported a disappointing sensitivity and specificity of only 14 and 70%, respectively, for plain-film radiography in the diagnosis of post-traumatic osteomyelitis, even when changes on serial plain films were considered [29].

Computed tomography

Computed tomography provides excellent multiplanar reconstructions of the axial images allowing delineation of even subtle osseous changes. The CT is superior to MRI in the detection of sequestra and identifies even small devitalized bony fragments. Because resection of necrotic bone with thorough debridement of infected bone and excision of soft tissue fistulae are two major aims of surgical treatment, CT has considerable importance in determining operative therapy [25].

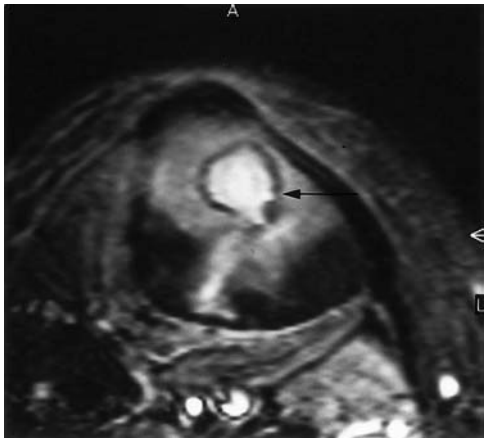

Fig. 6 Chronic postraumatic osteomyelitis with intraosseous abscess of the proximal tibia. In MRI, the axial STIR sequence shows increased signal intensity, surrouded by a rim of low signal intensity (*arrow*). Perifocal edema is also present

Nuclear medicine

Secondary chronic osteomyelitis generally shows persistent increased uptake as a manifestation of increased bone turnover (Fig. 5). Though the intensity of the vascular and blood pool activities of the three-phase bone scan can be used as an indication of a reactivation, the WBC scan with labeled autologous leucocytes is superior in determining the activity of the inflammatory changes [12].

MRI

The findings in exogenous chronic osteomyelitis are similar to those observed in haematogenous osteomyelitis, although the lesions are more irregular in outline due to post-traumatic alterations in endosteal and periosteal new bone formation. Inflammatory oedema induces an increased signal intensity on both STIR and T2-weighted images with fat suppression (Fig. 6). Either sequence invariably shows the associated soft tissue oedema. Identifying acute activity within chronic osteomyelitis is always difficult in the first 6 months after trauma or surgery and in some cases can persist for up to 12 months [20, 30]. In this relatively early phase, oedema, granulation tissue and fluid accumulation of high signal intensity can be present, without yielding a positive bacteriological culture. These areas enhance after intravenous gadolinium contrast media, due to hyperaemia and increased endothelial permeability, but this does not lead to improved specificity [31]. Cystic accumulations of sterile fluid can be observed even years after a fracture and/or surgical intervention and even without inflammatory changes, revealed by high-signal structures with the appropriate sequences. These cystic regions, which are found in both the medulla and the soft tissues, are

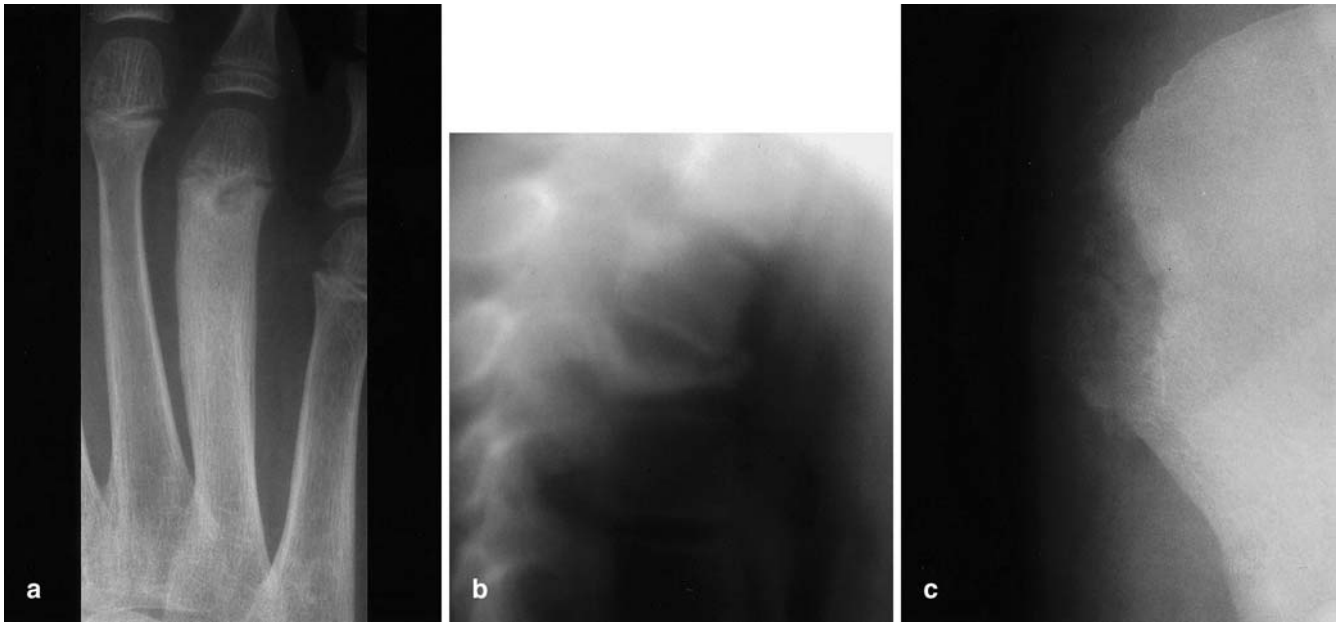

Fig. 7 Chronic recurrent multifocal osteomyelitis (CRMO). This 10-year-old girl presented with pain in thoracic spine and the forefoot. The radiograph of the 4th metatarsal (**a**) bone reveals classical signs of subacute or chronic osteomyelitis, comparable to Brodies's abscess. Conventional tomography of the upper thoracic spine (**b**) demonstrated a vertebra plana with flattening and sclerosis of the remaining parts of the vertebral body. In the iliac bone (**c**), additionally a radiolucency with a broad sclerotic rim is observed. Ten years later, at the age of 20, the patient developed another sclerotic focus in contralateral iliac bone (not shown)

sharply demarcated, and without surrounding oedema, the absence of which distinguishes them from reinfection. In such cases, therefore, the use of enhancement has been helpful in differentiating acute inflammatory changes from scar tissue with cystic inclusions [32].

Role of imaging in diagnosing exogenous osteomyelitis

In the majority of cases, post-traumatic osteomyelitis is a clinical diagnosis, only confirmed by the isolation of microorganisms from the biopsy specimen and by histopathological examination. In the remaining patients only the combination of clinical symptoms, blood tests, imaging and laboratory examinations can provide an accurate diagnosis.

Conventional radiography provides basic information about the osseous condition, and above all shows the post-traumatic and postoperative changes that must be considered in the general assessment. Diagnostic evaluation must generally be supplemented by determination of the extent of osseous involvement, as its preoperative assessment forms the basis for adequate surgical therapy, hence improving the final outcome. The MRI offers the best information on the extent of infection and frequently reveals unexpected findings. Scintigraphy, especially the WBC scan with labeled autologous WBC, is superior to MRI in establishing the status of osteomyelitic reactivation, especially during the first 12 months after trauma and/or surgery [12]. The CT is indicated if a sequestrum cannot be excluded or confirmed with certainty by conventional radiography or MRI. The value of sonography, which can be performed at the bedside, should not be underestimated since it can expeditiously assess the soft tissues and exclude or confirm an abscess.

Chronic recurrent multifocal osteomyelitis

The term chronic recurrent multifocal osteomyelitis (CRMO) summarizes a variety of subacute and chronic osteomyelitis of unknown course that primarily occurs in childhood and that frequently reveals multiple and symmetric alterations [1]. The association between SAPHO syndrome (synovitis, acne, pustulosis, hyperostosis, osteitis) and CRMO has become progressively more evident in recent years [6, 33, 34, 35].

The diagnosis of CRMO is one of exclusion based on the following criteria: lack of causative organism and no formation of abscess; fistula; or sequestrum. The atypical location of the lesions compared with infectious osteomyelitis favours the diagnosis of CRMO, with frequent involvement of the clavicle and usually multifocal lesions. The radiographic features suggest subacute or chronic osteomyelitis, although in the early phase of the disease osteolytic areas may mimic acute osteomyelitis [36] (Fig. 7). The histopathologic and laboratory findings are non-specific [33]. The course is benign, but prolonged over several years and fluctuating, with recurrent

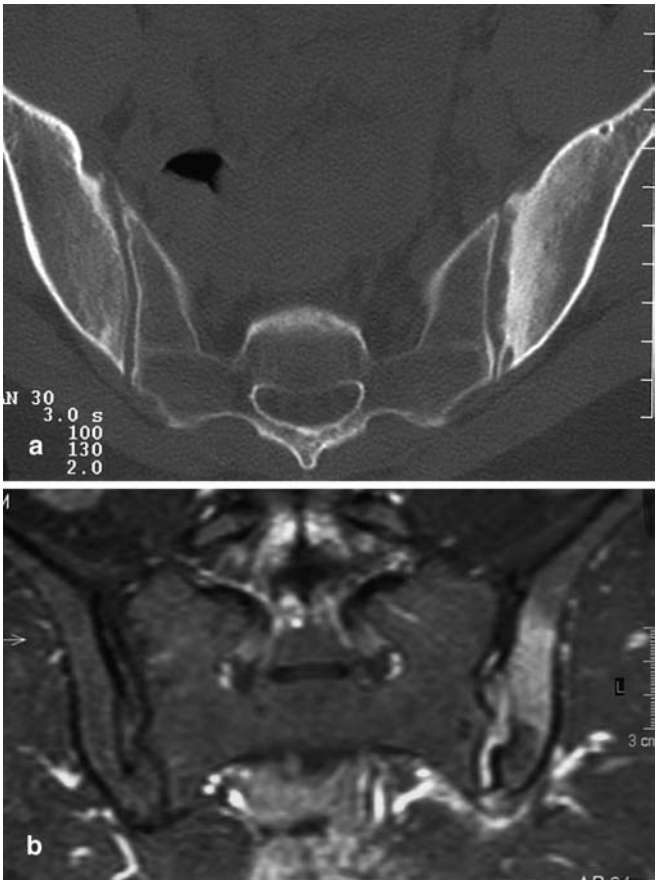

Fig. 8 Chronic sclerosing osteomyelitis in a 37-year-old woman. No skin changes were present at the time of diagnosis. No bacteremia and no organism could be cultured from the aspirate (CT-guided puncture). The disease was classified as a "SAPHO-like", "reactive" osteomyelitis. CT (**a**) revealed a sclerotic iliac focus with irregularities of the iliiac margin of the SI joint. The sacral element of the SI joint is normal. MRI (**b**) (fat-suppressed T1-weighted spin-echo sequence after i.v. administration of gadolinium) additionally emphasized the involvement of the joint without any change in the sacrum

episodes of pain and swelling, usually without concomitant systemic manifestations.

The lesions of CRMO, based on all patients reported, are predominantly located in the appendicular tubular bones, followed by the clavicle and the spine. Involvement of pelvic bones and the sternum also occurs rarely. Changes in involved tubular bones are usually characteristc on radiography. Initially, lytic metaphyseal destruction appears, usually with an identifiable sclerotic margin demarcating it from normal bone [35, 36]. The findings on MRI consist of decreased marrow signal intensity on T1-weighted images and, during active phases, dispersed regions of high signal intensity on T2-weighted images adjacent to the metaphyseal lesions, without discernible abscess formation (Fig. 8). The extent of marrow involvement is always more pronounced than indicated by conventional radiography [33].

Clavicular lesions are characterized in the early active stages by lytic medullary destruction in the medial half of the clavicle and laminated periosteal new bone formation. During periods of remission, the destruction tends to heal with the formation of sclerosis. The periosteal new bone becomes organized, scleroses and fuses with the clavicle, resulting in hyperostosis [36].

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References

1. Resnick D (2002) Diagnosis of bone and joint disorders, vol III, 4th edn. Saunders, Philadelphia, pp 2377–2509
2. Oudjhane K, Azouz EM (2001) Imaging of osteomyelitis in children. Radiol Clin North Am 39:251–266
3. Jones NS, Andersson DJ, Stiles PJ (1987) Osteomyelitis in a general hospital: a five-year study showing the increase in subacute osteomyelitis. J Bone J Surg 69:779–783
4. Hayes CS, Heinrich SD, Craver R, MacEwen GD (1990) Subacute osteomyelitis. Orthopedics 13:363–366
5. Gladhill RB (1973) Subacute osteomyelitis in children. Clin Orthop 96:57–69
6. Schilling F (2002) Die Osteitis und primär chronische Osteomyelitis der Clavicula: Exposition, historische Übersicht und Nosologie. Osteologie 11:96–102
7. Boutin RD, Brossmann J, Sartoris DJ, Reilly D, Resnick D (1998) Update on orthopedic infections. Orthop Clin North Am 29:41–66
8. Bohndorf K, Jurik A (2001) Peripheral osteomyelitis and its differential diagnosis. In: Schulthess GK von, Zöllikofer CL (eds) Syllabus muskuloskeletal diseases. Springer, Berlin Heidelberg New York, pp 117–126
9. Bonakdarpour A, Gaines VD (1983) The radiology of osteomyelitis. Orthop Clin North Am 14:21–33

10. Wegener WA, Alavi A (1991) Diagnostic imaging of musculoskeletal infection: roentgenography, gallium, indium-labelled white blood cell, gammaglobulin, bone scintigraphy, and MRI. Orthop Clin North Am 22:401–418
11. Pennington WT, Mott MP, Thometz JG, Sty Jl, Metz D (1999) Photopenic bone scan osteomyelitis: a clinical perspective. J Pediatr Orthop 19:695–698
12. Sciuk J, Erlemann R, Schober O, Peter PE (1992) Bildgebende Diagnostik der Osteomyelitis. Dt Ärzteblatt 89:2462–2473
13. Schauwecker DS (1989) Osteomyelitis: diagnosis with^{111}In-labeled leukocytes. Radiology 171:141–146
14. Cardinal E, Bureau NJ, Aubin B, Chhem RK (2001) Role of ultrasound in muskuloskeletal infections. Radiol Clin North Am 39:191–201
15. Kaiser S, Rosenborg M (1994) Early detection of subperiosteal abscesses by ultrasonography: a means for further successful treatment in pediatric osteomyelitis. Pediatr Radiol 24:336–339
16. Riebel TW, Nasir R, Nazarenko O (1996) The value of sonography in the detection of osteomyelitis. Pediatr Radiol 26:291–297
17. Larcos G, Antico VF, Cormick W, Gruenewald SM, Farlow DC (1994) How useful is ultrasonography in suspected acute osteomyelitis? J Ultrasound Med 13:707–709
18. Mosher TJ (2002) Diagnostic effectiveness of gadolinium-enhanced MR imaging in evaluation of abnormal bone marrow signal. Radiology 224:320–322
19. Bohndorf K, Imhof H, Pope TL Jr (eds) (2001) Muskuloskeletal imaging. Thieme, Stuttgart, pp 136–151
20. Bohndorf K (1996) Bildgebende Diagnostik der akuten und chronischen Osteomyelitis. Radiologe 36:786–794
21. Tehranzadeh J, Wong E, Wang F, Sadighpour M (2001) Imaging of osteomyelitis in the mature skeleton. Radiol Clin North Am 39:223–250
22. Mazur JM, Ross G, Cummings J, Hahn GA, McCluskey WP (1995) Usefulness of magnetic resonance imaging for the diagnosis of acute musculoskeletal infections in children. J Pediatr Orthop15:144–147
23. Sammak B, Abd El Bagi M, Al Shahed M, Hamilton D, Al Nabulsi J, Youssef B, Al Thagafi (1999) Osteomyelitis: a review of currently used imaging techniques. Eur Radiol 9:894–900
24. Stephens MM, MacAuley P (1988) Brodie's abscess: a long-term review. Clin Orthop 234:211–216
25. Gross T, Kaim AH, Regazzoni P, Widmer AF (2002) Current concepts in post-traumatic osteomyelitis: a diagnostic challenge with new imaging options. J Trauma 52:1210–1219
26. Gustilo RB (1989) Management of chronic osteomyelitis. In: Gustilo RB, Grunninger RP, Tsukyama DT (eds) Orthopedic Infection. Saunders, Philadelphia, pp 155–165
27. Ehara S (1997) Complications of skeletal trauma. Radiol Clin North Am 35:767–781
28. Roesgen M, Hierholzer G, Hax PM (1989) Post-traumatic osteomyelitis. Arch Orthop Trauma Surg 108:1–9
29. Tumeh S, Aliabadi P, Weissman B, McNeil B (1987) Disease activity in osteomyelitis: role of radiography. Radiology 165:781–784
30. Kaim A, Ledermann HP, Bongartz G, Messner P, Müller-Brand J, Steinbrich W (2000) Chronic post-traumatic osteomyelitis of the lower extremity: comparison of magnetic resonance imaging and combined bone scintigraphy/immunoscintigraphy with radiolabelled monoclonal antigranulocyte antibodies. Skeletal Radiol 29:378–386
31. Ledermann HP, Kaim A, Bongartz G, Steinbrich W (2000) Pitfalls and limitations of magnetic resonance in chronic post-traumatic osteomyelitis. Eur Radiol 10:1815–1823
32. Hopkins K, Li K, Bergman G (1995) Gadolinium-DTPA-enhanced magnetic resonance imaging of the muskuloskeletal infectious processes. Skeletal Radiol 24:325–330
33. Jurik AG, Egund N (1997) MRI in chronic recurrent multifocal osteomyelitis. Skeletal Radiol 26:230–238
34. Boutin RD, Resnick D (1998) The SAPHO syndrome: an evolving concept for unifying several idiopathic disorders of bone and skin. AJR 170:585–591
35. Sunderam M, McDonald D, Engel E, Rothman M, Siegfried, EC (1996) Chronic recurrent multifocal osteomyelitis: an evolving clinical and radiological spectrum. Skeletal Radiol 25:333–336
36. Demharter J, Bohndorf K, Michl W, Vogt H (1997) Chronic recurrent multifocal osteomyelitis: a radiological and clinical investigation of five cases. Skeletal Radiol 26:579–588