Bone tumour simulators

Problems and pitfalls in radiology

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A bone tumour simulator is any lesion which is not a bone tumour but which may be mistaken clinically, radiologically or pathologically for a bone tumour. This paper will deal with only those lesions which may be mistaken for bone tumours on plain radiographs.

In any patient presenting with musculoskeletal pain, there is always a possibility that the pain may be due to an underlying tumour, particularly if there is no history of trauma, and the pain is of long duration. When there is a history of significant recent trauma, the likelihood of an underlying tumour contributing to the injury is low, but this possibility should always be considered. This is particularly relevant when patients sustain fractures from relatively trivial injuries. When reviewing radiographs demonstrating a fracture, assessment of the underlying bone is important to ensure that the fracture has not occurred through a lytic bone lesion. This is not a case of a nontumorous lesion simulating a tumour, but rather of a non tumorous lesion (fracture) hiding the presence of a bone tumour. However, caution is advised as the appearance of some fractures may suggest underlying pathology when none exists (Pope et al. 1981). If the underlying bone is abnormal, the presence of the fracture and any attempt at healing of the fracture will complicate the radiographic diagnosis of the underlying lesion (Figure 1).

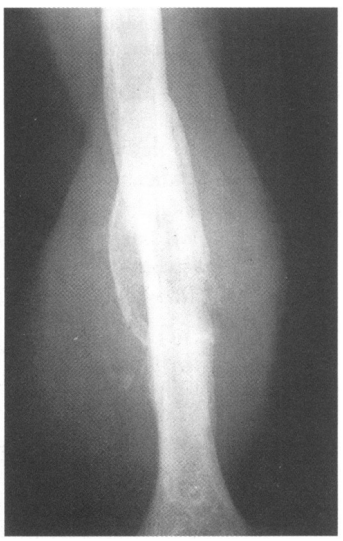

Figure 1. Healing fracture of humerus with mature callus, but with an irregular cortical margin infero-medially suggestive of an aggressive lesion. Subsequent biopsy demonstrated fibrosarcoma. The tumour was not recognized on the original films.

Bone tumour simulators may be classified as normal variants, traumatic lesions, inflammatory and infective lesions, lesions due to metabolic bone disease, vascular lesions, periarticular abnormalities, extraosseous calcific lesions and others. Another possible cause for diagnostic confusion is that benign bone tumours may simulate malignancy and malignant bone tumours may appear benign. (Houghton et al. 1995, Rosenberg et al. 1995, Malloy et al. 1992). For this reason careful clinical, radiological and pathological correlation is always required when assessing any bone lesion.

Normal variants

Normal variants can involve any part of the bony skeleton, and familiarity with their appearances is the only way to prevent misdiagnoses. Some of the more common lesions include the pseudocyst in the humeral head due to the normal arrangement of bony trabeculae, the supracondylar humeral spur (Figure 2) and the supratrochlear foramen in the distal humerus. The appearance of the radial tuberosity and ossification of the radial aspect of the interosseous membrane are also common abnormalities, whilst a Madelung deformity of the distal radius may also be misdiagnosed as a bone tumour (Thomas et al. 1993). In the lower limb, the femoral head fovea, the superimposition of the ischium on the femoral head, and Ward's triangle in the femoral neck may all be mistaken for focal, lytic bone lesions (Keats 1992). Focal bony defects or depressions may represent normal variants, or be the result of repetitive trauma. These include grooves on the under surface of the medial end of the clavicles, grooves for the insertion of the pectoralis major mus-

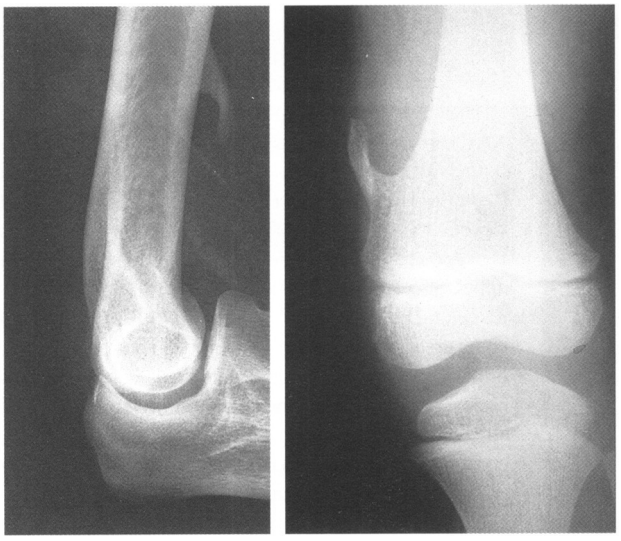

Figure 2. Supracondylar humeral spur (left), looks similar to an osteochondroma (right). Note that osteochondromas always point away from the adjacent joint.

cle on the humerus, Blount' s disease of the proximal tibia, the dorsal defect of the patella and irregularity of the distal femoral cortex (Figure 3). One of the most common sites to raise the suspicion of the radiologist is the pubic rami synchondroses which may look like pathological fractures through lytic expansile lesions (Albertson et al. 1994) (Figure 4). Many of these "normal variants" may represent manifestations of chronic stress injury upon the growing skeleton (Lawson 1993). Lesions included in this category include the carpal boss, accessory navicular, bipartite patella, dorsal defect of the patella, os subfibulare (at the tip of the lateral malleolus, and probably an avulsion fracture) and os supratrochlear dorsal (above the olecranon).

Other unusual normal variants include bony projection such as the iliac horns seen in Fongs disease, and bony bars seen around the scapular margins. In the femoral neck the herniation pit ("Pitt's pit") (Pitt et al. 1982) is extremely common, whilst Schmorl's nodes in the vertebral bodies are unlikely to be confused with tumours unless unusually large or irregular. In the skull lytic lesions due to large venous lakes may be mistaken for neoplasms, whilst the appearance of hyperostosis frontalis interna may occasionally be mistaken for other sclerotic bone lesions or an en plaque meningioma (Figure 5). Experience, and familiarity with the text books on normal variants are the only ways to avoid over calling these lesions.

Traumatic and posttraumatic lesions

There is a large variety of post traumatic lesions which may simulate bone tumours. Virtually all of these lesions are manifestations of the response of bone to the traumatic insult. Since plain film radiology demonstrates the presence or absence of mineralized bone, what we are really assessing is the deposition and resorption of bone in response to the particular insult. The extent and rate of bone resorption and

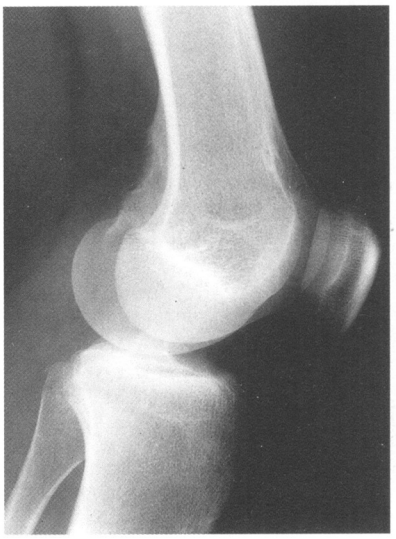

Figure 3. Normal variant—irregular contour of dorsal aspect of distal femur.

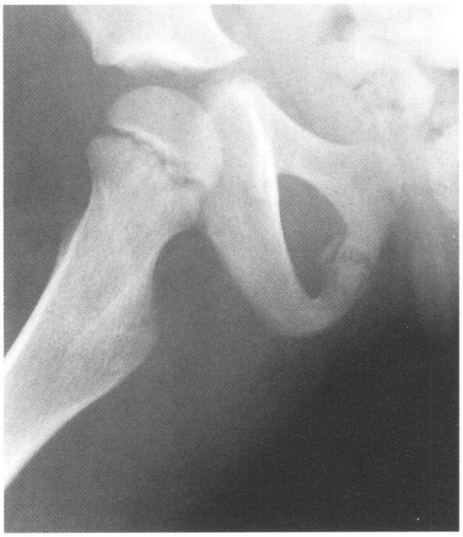

Figure 4. Normal variant—unusual appearance of ischiopubic synchondrosis.

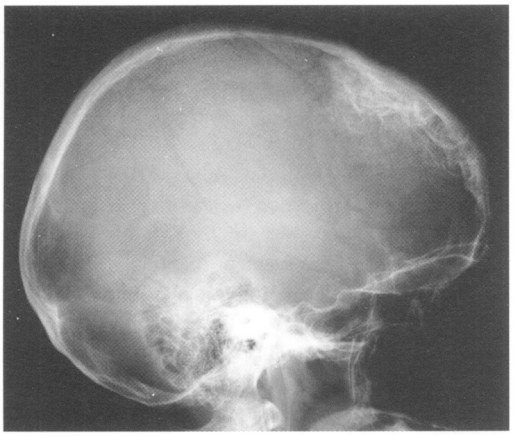

Figure 5. Normal variant—hyperostosis frontalis interna.

new bone formation is dependant on a large variety of factors, including the type of trauma or bone insult, whether it is a single episode or repetitive, the individual bone involved, and the patient's age, biochemical status and presence of underlying disease e.g. osteoporosis, osteomalacia, Paget's disease. Following most bone insults, there is some localized resorption of bone followed by deposition of new bone, and variations and extremes of these processes give rise to a variety of bizarre appearances (Albertsen et al. 1994, Keats 1990).

Healing fractures

The appearance of the progressive maturation of callus at a fracture site is well known to all radiologists. However, when there is no history of a previous fracture, and bony alignment is normal, the appearance of callus may be suggestive of either an aggressive or nonaggressive bone tumour depending on the stage of fracture healing. Some patients are prone to developing abundant callus at fracture sites and the extent of new bone formation may give rise to concerns of an underlying tumour. Abundant callus is seen particularly in patients with head injuries, osteogenesis imperfecta, Cushings disease and in patients with paraplegia. It is important to be aware of the variety of appearances of normal fracture healing, as the histology of callus may be very suggestive of an osteosarcoma. (Figure 6)

Stress reactions and stress fractures

Stress injuries may be classified into stress reactions, and stress fractures. A stress reaction shows no evidence of fracture, but is a bony response to repeated stress e.g. shin splints, tug lesions. Tug lesions may appear quite benign, or may have irregular margins simulating malignancy. Stress fractures can be subdivided into insufficiency fractures (abnormal underlying bone) and fatigue fractures (normal bone with excess stress). These injuries are particularly common in athletes and people performing unusual repetitive exercise, and if an appropriate clinical history is obtained, the diagnosis is usually not difficult. When these lesions are detected radiographically without any relevant history, their diagnosis as stress injuries is not so easy. An extremely useful guide to the varied appearances of these lesions is "Radiology of Musculoskeletal Stress Injury" by Keats (1992). Stress lesions are most common in the pelvis and lower limbs, but also occur in the upper limbs, shoulder girdle, thoracic cage and spine. The radiographic appearance of stress lesions includes simple periosteal reactions, complex and bizarre periosteal reactions, medullary sclerosis with or without associated fractures and localized areas of bone resorption. Stress lesions may also be visualized as entirely normal radiographs with focal or diffuse areas of increased activity on bone scans. The pars interarticularis defect is a well known stress fracture, but in patients with a unilateral pars defect, reactive hypertrophy of the contralateral pedicle and pars may mimic an expansile sclerotic bone lesion. A similar appearance may occur in patients with a congenital deficiency on one side of the neural arch.

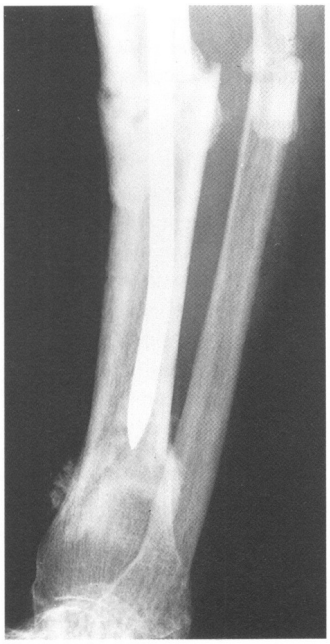

Figure 6. Healing fracture—note unusual periosteal new bone formation.

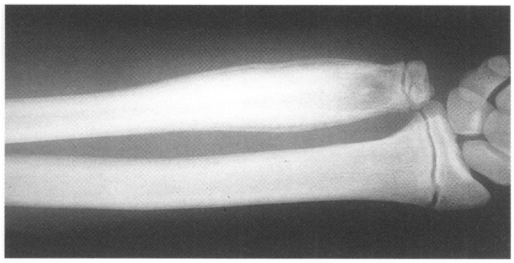

Figure 7. Osteomyelitis of the distal ulna with a small central sequestrum and abundant periosteal reaction. This lesion may be confused with an osteoid osteoma radiologically.

Posttraumatic bone resorption

Bone resorption following trauma may be focal or diffuse, and may take a variety of appearances. These include post traumatic osteolysis as seen in the lateral aspect of the clavicles or the pubic rami. Fractures may also result in severe disuse osteoporosis, and the rapid rate of osteoporosis may result in a rather permeative appearance to the residual bone, raising the possibility of an underlying neoplasm. Mottled osteoporosis is also seen in reflex sympathetic dystrophy, where the overlying soft tissue swelling may be suggestive of an underlying tumour. In the skull a "growing fracture" is an increase in lucency due to herniation of meninges into a fracture defect with CSF pulsations leading to resorption of bone at the margins of the fracture site. In patients who have had limb salvage surgery for a bone tumour, resorption of bone around a prosthesis or allograft due to stress shielding, particle disease or infection, may mimic tumour recurrence (Mund et al. 1994).

Other post traumatic lesions

Avulsion injuries, with or without evidence of attempted healing, may simulate bone tumours. This is particularly the case when there is repetitive injury to a region, and the attempts at healing are superimposed on the effects of repeated trauma. This can result in rather large soft tissue masses, as well as bizarre new bone formation simulating an aggressive tumour. A partially calcified subperiosteal hematoma may be mistaken for a more aggressive lesion and the varying appearances of myositis ossificans can be mistaken for a parosteal osteosarcoma. The zonal nature of calcification in myositis ossificans, with the more mature calcification peripherally often helps with diagnosis. The gross destruction of a Charcot joint, or a hypertrophic non union at a fracture site may also simulate aggressive bone tumours.

Inflammatory and infective lesions

The many appearances of osteomyelitis may simulate almost the entire spectrum of bone tumours, and osteomyelitis should be considered the classic example of a bone tumour simulator (Rosenberg et al. 1995, Malloy et al. 1992, McGrath et al. 1994, Kaweblum et al. 1993, Mulligan, Kransdorf 1993). In its early phases, osteomyelitis may be abnormal on bone scan with no plain film findings. Osteomyelitis may then produce thin periosteal reaction (a large list of differential diagnoses), and then ill defined bone destruction. Progression to a more permeative lesion may simulate infiltrating bone tumours, whilst a well defined focus may mimic an osteoid osteoma (Figure 7). A Brodie abscess mimics more benign bone tumours with a sclerotic margin (Figure 8), whilst chronic sclerosing osteomyelitis may be misdiagnosed as either a bone matrix producing tumour such as an osteosarcoma or as a tumour with abundant reactive new bone formation such as an osteoid osteoma. Tuberculosis, hydatid disease of bone (Figure 9) and syphilis may also produce a variety of appearances simulating bone tumours. Clinical assessment of the patient is often invaluable, as patients with osteomyelitis frequently have malaise and fever with an elevated white cell count. However, if the patient has been given antibiotics, the course of the infection may be more indolent

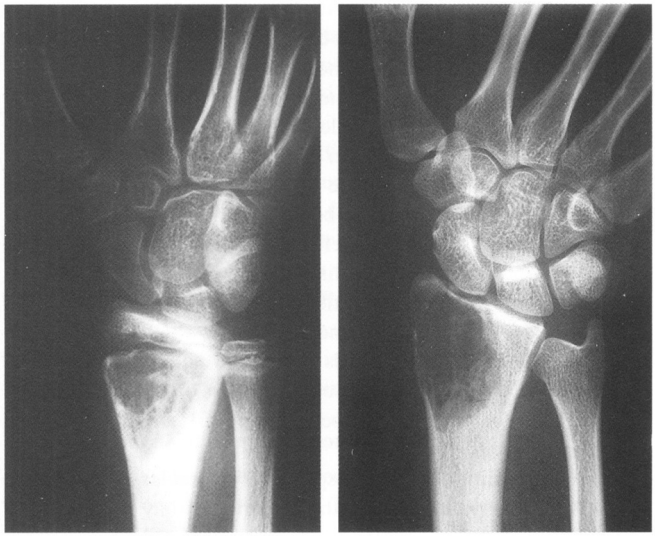

Figure 8. Osteomyelitis of the distal radial metaphysis (left) bears some similarity to a giant cell tumour (right) or aneurysmal bone cyst. Note the sclerosis of the radial shaft and periosteal new bone formation which make osteomyelitis the more likely diagnosis.

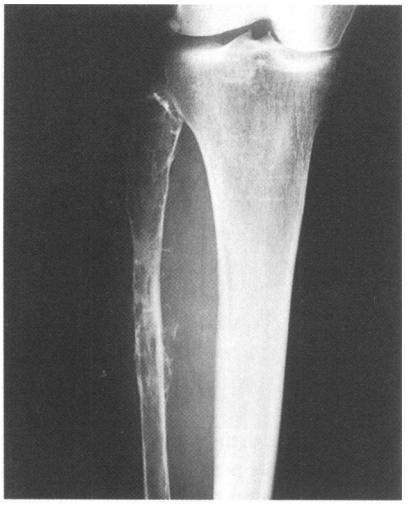

Figure 9. Hydatid disease of fibula demonstrating bone lysis, endosteal scalloping, expansion and irregular periosteal new bone formation.

and less clinically apparent. Other inflammatory disorders which can mimic bone tumours include sternocostoclavicular hyperostosis, which involves the medial ends of the clavicles, sternum and first costochondral junction and may be associated with overlying soft tissue swelling (Fritz et al. 1992). Sarcoidosis of bone has a variety of appearances and the diagnosis may not be considered by the radiologist unless appropriate clinical information is provided (Thomas et al. 1993).

Vascular lesions

The plain film appearances of a calcified medullary bone infarct are quite similar to those of calcified cartilage tumours such as enchondroma and central chondrosarcoma although the calcification of a bone infarct is typically most intense in the periphery. MRI has made differentiation of bone infarct from cartilage tumour more definite, as a bone infarct has a a typical appearance with a well defined geographic irregular shape with a "double line" margin on T2 weighted images, whilst in cartilage tumours MRI will often demonstrate a soft tissue component which is well circumscribed, and extends beyond the calcifications. MRI can also detect avascular necrosis of the femoral head (or other bones) before there are any plain film findings.

This can confirm a clinical suspicion of avascular necrosis, and helps to exclude the presence of any other sinister pathology. Patients who have had local radiotherapy for malignant disease may present with pain suggestive of recurrent malignancy. Radiographic appearances in these patients may be due to recurrent tumour invading bone, postirradiation change in bone with sclerosis and pathological fracture, or radiation induced bone sarcoma (Figure 10). Comparison of sequential films, with multimodality imaging including radiography, CT scanning, nuclear medicine and MRI may be required to make the appropriate diagnosis. The postirradiation changes in bone are essentially due to bone ischemia, and the pathological fractures not infrequently progress to nonunion. Nutrient vessel foraminae can be mistaken for lytic bone lesions, as can venous lakes in the skull. Bone resorption due to subperiosteal hemorrhage in a hemophiliac (hemophilic pseudotumour) is an unusual entity where the diagnosis is extremely difficult to make if one is not aware of the patient's underlying diagnosis (Figure 11). These lesions may be extremely large with a large soft tissue component, but are

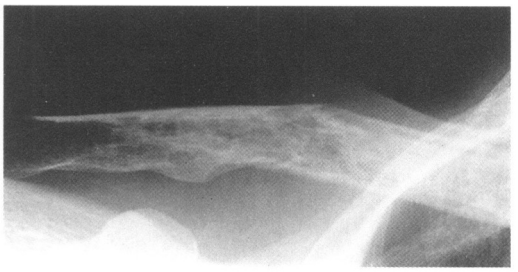

Figure 10. Post irradiation change of the clavicle with a pathological fracture in a patient with breast carcinoma. Note the abnormal trabecular pattern and subtle endosteal scalloping on the superior cortex. Three weeks later, a large soft tissue mass had developed, and biopsy confirmed fibrosarcoma.

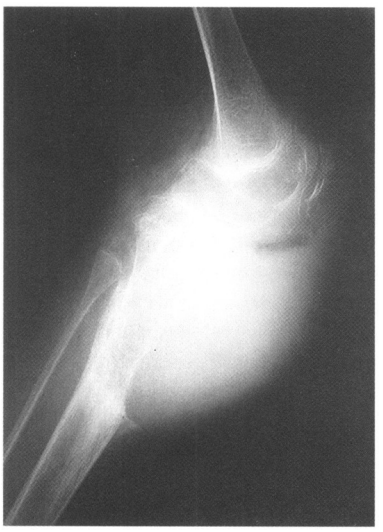

Figure 11. Haemophilic pseudo tumour of proximal tibia associated with typical enlarged epiphyses and changes of haemophilic arthropathy.

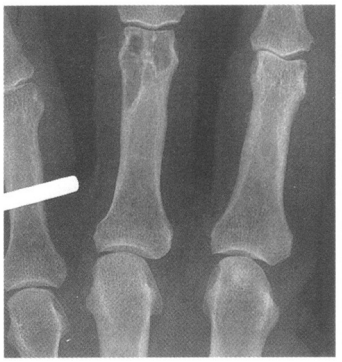

Figure 12. Hyperparathyroidism. Well circumscribed brown tumor in distal end of third proximal phalanx has a benign appearance, whilst irregular lytic lesion in mid shaft of second metacarpal suggests a more aggressive pathology.

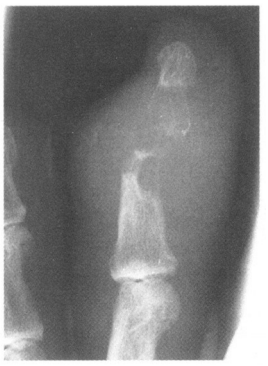

Figure 13. Large gouty tophus complicated by infection and septic arthritis simulating aggressive tumour.

usually well circumscribed with a sclerotic rim and the bony margin indicates a chronic non aggressive lesion. They most frequently occur in the ilium. Aneurysms of the aorta and other vessels may lead to pressure erosion on the surface of adjacent bones, suggestive of a surface tumour.

Metabolic disorders

A wide variety of metabolic disorders have skeletal manifestations which may mimic bone tumours. In hyperparathyroidism, the osteoporosis and subperiosteal bone resorption may mimic an aggressive tumour, whilst brown tumours are focal, lytic bone lesions which are usually well circumscribed although they may have irregular margins. (Figures 12, 14). If multiple brown tumours are found radiographically in a patient not known to have hyperparathyroidism, osteoporosis makes vertebrae appear particularly lucent, and if there is a vertebral compression fracture, it is often difficult to be certain whether there is any underlying bone lesion. Patients with severe, chronic osteoporosis may develop reenforced trabeculae in the femoral neck which simulates cartilage tumour matrix or a bone infarct (Kerr et al. 1986). Loosers zones in osteomalacia have a relatively specific appearance and are not usually mistaken for tumours, although if they occur in an unusual site this may lead to diagnostic difficulties. Both the lytic and sclerotic phases of Paget's disease have been known to be confused for other bone lesions, particularly when the disease is monostotic. Other metabolic disorders may give rise to some confusion if only one segment of the body is imaged. These include thyroid acropathy, hypertrophic pulmonary osteoarthropathy, fluorosis, hypervitaminosis D and osteopetrosis.

Periarticular abnormalities

Periarticular bone destruction typically suggests some form of arthropathy, but when the joint otherwise appears normal, or the lesion is unusual in appearance, a large list of disorders should be considered. Periarticular erosions due to inflammatory joint disease such as rheumatoid arthritis usually are not a diagnostic dilemma, but the erosions due to depositional disorders such as gout, may cause confusion particularly if in an unusual location, or if complicated by infection (Figure 13). Subchondral bone cysts are well defined lytic lesions with sclerotic margins associated with degenerative joint disease. They are extremely common, but when there is a large cyst, and very little evidence otherwise of degenerative joint disease, diagnostic uncertainty arises. Another similar lesion is the intraosseous ganglion. Pigmented villonodular synovitis typically causes multiple well circumscribed large erosions on both sides of a joint, but if there is only a single erosion, solitary bone tumours may be considered as diagnostic possibilities. (Figure 14). Synovial osteochondromatosis has a classical appearance, but when an unusual joint is involved with only a few calcific bodies, other diagnoses such as soft tissue or periosteal cartilage tumours may be considered. Synovial cysts arising from the facet joints of the spine may simulate other spinal tumours, whilst amyloid deposits in patients with myeloma or on dialysis may also be confusing (Reinus et al. 1993). Carpal and tarsal coalitions, the carpal boss, and neuropathic joints in diabetes, syphilis and paraplegia may mimic tumours in or near joints.

Calcific lesions around bone

A large variety of post traumatic calcific lesions can occur around bone. These include normal callus, subperiosteal hematomata, myositis ossificans, calcific tendonitis, and avulsed bone fragments. Calcium pyrophosphate deposition disease (CPPDD), hydroxyapatite deposition disease (HADD) and tumoral calci-

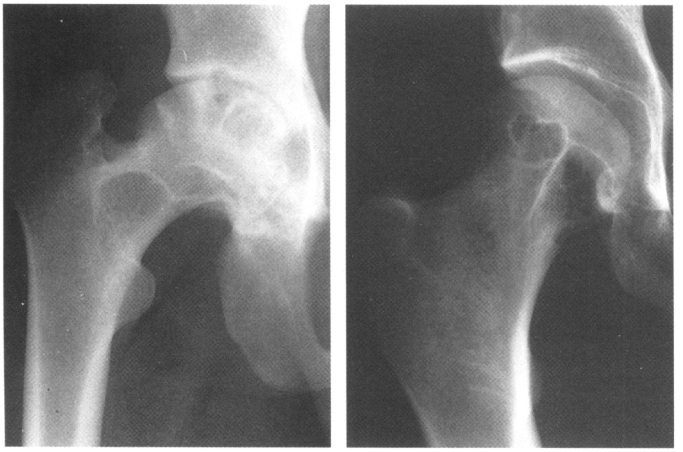

Figure 14. Pigmented villonodular synovitis (left) with large erosion on femoral neck, and numerous small erosions on the femoral head and acetabulum. Note the difficulty in differentiating the surface erosion of the femoral neck in PVNS from the intraosseous lesion due to a brown tumor (right).

nosis all cause periarticular calcification. Whilst they will normally not be mistaken for tumours, when their appearances are extremely florid, they may be confused with a synovial sarcoma or other calcified soft tissue tumour. Florid reactive periostitis, bizarre periosteal osteochondromatous proliferations, melorheostosis and dysplasia epiphysealis hemimelica also cause unusual calcific lesions around bone whilst dermatomyositis, myositis ossificans progressiva, and parasitic infestations cause unusual soft tissue calcifications which may occasionally be mistaken for tumours (Fechner Mills 1993, Breidahl Wylie 1995).

Conclusion

A large variety of unusual bone lesions has been discussed, all of which may mimic bone tumours. Familiarity with the wide range of bone pathologies, and in particular the wide range of normal variants and stress related abnormalities may prevent over investigation of many of these lesions. However, it should be remembered that whilst a normal variant may mimic a bone tumour, a bone tumour may also have a totally normal radiographic appearance. No imaging test can entirely exclude the presence of a bone tumour, as microscopic deposits cannot be detected even with the most advanced imaging modalities. Caution is advised in the diagnosis of all bone lesions, and in particular comparison with any previous films, obtaining follow-up films in cases of dubious abnormalities, and complete staging of lesions prior to biopsy is highly recommended.

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