Tibial cortical lesions: a multi-modality pictorial review

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Learning objectives

1. To identify tibial cortical lesions and provide a narrow differential diagnosis.

2. To identify cases which require additional imaging +/- biopsy for further management.

3. To avoid over investigation of straightforward lesions, while expediting imaging, intervention of straightforward lesions, while expediting imaging, intervention and management plans for lesions requiring surgical or medical treatment.
Background

Pain in the lower leg is a relatively common presenting complaint, often affecting young and active patients. A wide range of potential underlying pathologies exist, which can cause difficulty for both the clinician and the radiologist when trying to diagnose and manage patients presenting with shin pain. As well as an accurate clinical history and examination, the arrival of modern techniques/imaging is essential to make a definitive diagnosis or to enable us to identify lesions that require a biopsy or surgical excision. As a general rule, all patients with a suspected tibial cortical lesion should have a radiograph performed and depending on the findings, cross-sectional imaging can then be used for further evaluation if required.

We review the natural history and multimodality imaging findings of the more common causes of cortically-based tibial lesions, as well as the rarer pathologies less frequently encountered in a general radiology department.
1. Non-Neoplastic Tibial Cortical Lesions

We divide lesions into non-neoplastic and neoplastic, and include all imaging modalities, including MRI, CT, plain radiographs, angiography and nuclear medicine studies. Pathologies reviewed include fibrous cortical defect / non-ossifying fibroma, adamantinoma, osteofibrous dysplasia, melorheostosis, venous stasis, osteoid osteoma and stress fractures. We also include medullary lesions which also frequently involve the cortex, such as Brodie's abscess, fibrous dysplasia and chondromyxoid fibroma.

Tibial Stress Injuries

Tibial stress injuries are the commonest cause of exertional leg pain in athletes (1), causing osteoclastic and then osteoblastic activity as well as periosteal and endosteal proliferation resulting in an imbalance that weakens bone and if untreated can be complicated by fracture. They may be classified as due to fatigue (increased stresses on normal bone) or insufficiency (normal stresses on abnormal bone), the latter of which is less common, and is secondary to a variety of conditions including osteoporosis, osteomalacia and rheumatoid arthritis. (2) Medial tibial stress syndrome (shin splints) is characterised by exercise-induced pain along the posteromedial aspect of the distal 2/3 of the tibia. In children and short distance runners, fatigue stress injuries tend to occur more proximally in the tibia.

Plain radiography:

Radiographs are used as the first imaging test for a suspected stress injury. However, they are negative in roughly one third of symptomatic patients and the majority of cortical fractures (up to 94%) go undetected. (2, 3) They demonstrate relatively late findings of stress injury including a benign-appearing periosteal callous reaction, eccentric thickening and increased sclerosis of the cortex and endosteum (fig. 1a) and a lucent fracture line.

Bone scintigraphy

Bone scintigraphy is much more sensitive than radiographs, although MRI is more sensitive and specific. Bone scintigraphy demonstrates the increased bone metabolic activity associated with a stress injury as an area of increased uptake of Technetium-99m methylene diphosphonate. (fig. 2)
Computed Tomography (CT):

CT is not as sensitive as MRI as a single test for stress injury but can demonstrate cortical abnormalities not visible on MRI. CT demonstrates osseous abnormalities in stress injury earlier than plain radiography, including periosteal reaction, cortical thickening (fig. 1b), cortical striations, lucencies and a discrete stress fracture (fig. 4a&4b). The stress fracture is seen earlier on CT than on plain radiographs, and is well visualised in all imaging planes. Tibial stress fractures are most commonly transverse or oblique in orientation and these tend to affect the proximal posterior tibial cortex.

MRI:

MRI is the most sensitive test for diagnosing the early signs of stress injury, particularly those of the periosteum and marrow and it demonstrates adjacent soft tissue abnormalities (fig. 5a,5b&5c).

The most commonly used classification is by Fredericson:

Grade 0: No abnormality.

Grade 1: Periosteal oedema with no marrow signal abnormalities.

Grade 2: Periosteal oedema and marrow oedema visible only on fluid sensitive sequences

(short tau inversion recovery (STIR)/T2-weighted).

Grade 3: Periosteal oedema and marrow oedema visible on both fluid sensitive and T1-weighted sequences.

Grade 4a: Multiple focal areas of intracortical signal abnormality and marrow oedema visible on both T1-weighted and fluid sensitive sequences.

Grade 4b: Linear areas of intracortical signal abnormality (suggestive of a stress fracture) and marrow oedema visible on both T1-weighted and fluid sensitive sequences.

MRI may also demonstrate fascial oedema around the tibial insertion of the soleus muscle.

Fibrous Cortical Defect and Non Ossifying Fibroma
Fibrous cortical defect (FCD) and non ossifying fibroma (NOF) are the mostly commonly encountered benign fibrous bone tumour. They are identical histologically composed of benign fibroblasts, histiocytes and xanthomatic cells. They occur in up to 30% of the asymptomatic population in the 1st and 2nd decade of life. Rather than true neoplasms they are developmental in origin, occur in the metaphysis of the long bones particularly within the tibia and femur in the region of intensive bone growth. It has been suggested that they may be due to subperiosteal haemorrhage secondary to injury at a site of muscle attachment. Distinction between a FCD and a NOF is made on the basis of age and size, with FCD occurring in young children less than 10 years of age and measuring less than 2 cm in size, in contrast with NOF which occurs in adolescents and measures greater than 2 cm in size. (4) They are most commonly an incidental finding although rarely a NOF will present with a pathological fracture. With time they may change in size, become sclerotic or disappear altogether. Multiple NOFs occur in neurofibromatosis.

**Plain Radiography and CT:**

Both FCD and NOF are lucent lesions, have a narrow zone of transition and are typically eccentric in location within the cortical layer of the bone. The lesions have a thin sclerotic border that is often scalloped and slightly expansile. Over time, they heal by gradual intralesional sclerosis. These lesions are usually oval in shape, the long axis of the lesion paralleling the long axis of the bone. Usually the diagnosis can be made on radiographs without the need for further imaging or biopsy.

**MRI:**

Often seen incidentally on MRI performed for other reasons, FCDs and NOFs are demonstrated as a small intracortical lobulated metaphyseal lesion that is hypointense on T1 and more commonly hypointense than hyperintense on T2. The contents of the lesion may be solid and/or cystic with the solid component enhancing following intravenous contrast. (fig. 6b&c).

**Melorheostosis**

Melorheostosis (Leri Disease) is rare, benign disorder resulting in a sclerosing bony dysplasia with adjacent soft tissue masses. It most commonly affects one side of a single tubular bone, usually within the lower limb long bone, but may affect multiple bone. Involvement of the axial skeleton, skull and thoracic cage is rare.

It typically presents with symptoms in late childhood or young adult life and may be detected incidentally at any age and has an equal gender distribution. Patients present with pain, stiffness, restricted motion, contractures, soft tissue masses, limb length discrepancy and deformity. Melorheostosis often has a chronic progressive clinical
course and can lead to significant disability, and surgical management of contractures and limb deformities may be required. Medical therapy with bisphosphonate infusion has been reported to help manage symptoms. (5)

**Plain Radiography and CT:**

Prominent, irregular, longitudinal, cortical and endosteal hyperostosis, with a proximal to distal pattern of extension in long bones, resulting in a classic "dripping candle wax" appearance. (figs.7, 8a&b). (6) Endosteal extension may obliterate the medullary cavity. There may be extra-osseous involvement resulting in fibrous or vascular soft tissue masses, especially in the periarticular regions, which may calcify or ossify over time. (7) Intra-articular extension into joint spaces may also be seen resulting in joint ankylosis and contractures. (8, 9)

**MRI:**

The hyperostotic cortical and subcortical changes are low signal intensity on T1-and T2-weighted imaging in keeping with sclerosis. The adjacent soft tissue masses have variable signal intensity characteristics depending upon their composition and degree of mineralisation. Following administration of intravenous contrast, no enhancement of the cortical hyperostosis is seen, but there is variable enhancement of the soft tissue masses.

**99mTc-Methylene Diphosphonate (MDP) Bone Scan:**

The hyperostotic segments of affected bones often show moderately increased asymmetrical cortical uptake (fig.9) which may cross joints to involve contiguous bones, whilst no abnormal uptake is seen in the adjacent uninvolved medullary cavity.

**Venous Stasis**

Venous stasis characterised by periosteal new bone formation is due to an increase in mean interstitial fluid pressure within the bone (10) and is thought to occur secondary to factors such as tissue hypoxia and venous hypertension. No age predilection exists and the lower extremities are most commonly affected, typically the tibia and fibula. Appearances are usually bilateral, relatively symmetrical but if there is a focal interruption of the blood supply such as with a tibial fracture, the ipsilateral extremity may be affected only.

**Plain Radiography and CT:**
Venous stasis typically results in a circumferential, smooth, wavy, solid periosteal reaction affecting the mid and distal shaft of the tibia and fibula. (10) As the condition progresses, the periosteal new bone becomes incorporated into the adjacent cortex and the appearance changes to diffuse, mature cortical thickening.

Secondary signs of venous stasis are the presence of multiple phleboliths as well as soft tissue swelling due to subcutaneous oedema. (10) Osteopenia may be seen in chronic venous insufficiency, which is often due to disuse.

**MRI:**

MRI is used to exclude alternative causes for periosteal reaction and cortical thickening such as osteomyelitis. Diffuse subcutaneous oedema, periosteal reaction and cortical thickening are findings on MRI. Extension of oedema along fascial planes may occur. Other causes of circumferential periosteal reaction include; infection, bone tumours, healing fractures, chronic stress injury and medical conditions such as thyroid acropachy and hypervitaminosis A.

2. Neoplastic Tibial Cortical Lesions

**Osteoid Osteoma**

Osteoid osteoma (OO) is a benign bone tumour accounting for 12% of benign skeletal neoplasms. The majority of the patients are young males with about half of them in the 2\textsuperscript{nd} decade of their life at presentation. (11)

The lesion consists of a central nidus of woven bone and osteoid rimmed with osteoblasts which tends to be less than 1.5 cm in size. A reactive zone of thickened bone and fibrovascular tissue is surrounding the lesion. OO is commonly cortical with reactive sclerotic cortical thickening or less commonly it is intramedullary and subperiosteal in an intra- or juxta-articular location. The tumour may occur in any bone, but most frequently occurs in the metadiaphyseal regions of the long bones, particularly the proximal femur and tibia, and to a lesser extent the spine, hands, feet and craniofacial bones. (12)

The pain from an osteoid osteoma is usually worse at night, causing sleep disturbance with relief of pain achieved by taking non-steroidal anti-inflammatory medication. Some patients may not respond well to long-term analgesia and until recently local surgical excision was considered the only treatment option. In the last two decades, less invasive procedures such as CT-guided drilling with or without ethanol therapy, laser interstitial
thermal therapy, cryotherapy, and CT guided radiofrequency ablation (RFA) have been introduced. CT guided RFA in particular is very popular, with reported success rates approaching 90%, hospital stay of less than 24 hours and relatively immediate return to normal activity, unlike surgery. (13)

**Plain Radiography:**

The radiographs show a round or oval radiolucent nidus with surrounding sclerosis. (fig. 10a). However, intramedullary and subperiosteal lesions may not have a distinct sclerotic margin or if one is present, it may be distant to the nidus.

**CT:**

CT is the modality of choice for characterisation and localisation of the lesion. It clearly demonstrates the small well-defined radiolucent nidus within the sclerotic region. (fig. 10b&c). The nidus enhances following intravenous contrast. CT can also be used to guide radiofrequency ablation treatment of the lesion.

**MRI:**

On T1-weighted images, the nidus typically reflects a similar signal to that of skeletal muscle. The lesion shows variable signal intensity on T2-weighted images. Surrounding bone marrow oedema on both T1- and T2-weighted/STIR images is present. (fig. 11a&b). Osteoid osteoma can resemble a stress fracture or osteomyelitis on MRI, particularly when the nidus is hidden by extensive surrounding oedema and in this situation, bone scintigraphy or CT can be used for further evaluation. (13)

**Bone Scintigraphy:**

The nidus shows increased activity relative to its surrounding reactive zone. (fig. 12). This is called the double-density sign and is pathognomonic of osteoid osteoma. Bones scans are particularly helpful in a symptomatic patient in which the radiograph is normal. (12)

**Adamantinoma**

Adamantinoma a rare low-grade malignant neoplasm of cortical bone, predominantly affecting long bones, almost exclusively in the tibia and is most often seen in adults in their third or fourth decades of life with a slight male predominance. The most common presenting features are pain and swelling are the, often a preceding history of trauma. On histology, adamantinoma shows a zonal architecture with a fibrous stroma centrally
containing abundant epithelial cells, which decrease in amount towards the periphery of the lesion. (14)

**Plain Radiography and CT:**

Adamantinoma is typically a multifocal, elongated, eccentric, expansile, lytic lesion in the cortex of the anterior tibial diaphysis with remodelling deformity. (Fig. 13). It often exhibits moth-eaten margins, cortical destruction and intramedullary involvement. (fig. 14) and may also show a mixed lytic and sclerotic appearance. There can be satellite lesions and extra-osseous extension with adjacent soft tissue masses that are best depicted on cross sectional imaging.

**MRI:**

Adamantinomas usually have a multilocular appearance with an intermediate to low signal intensity on T1-weighted sequences. (fig 15a) and a non-specific, heterogeneous high signal intensity pattern on T2-weighted imaging (fig. 15b), and foci of low signal intensity on spin-echo sequences are also common due to ossification within the lesion. Marked contrast enhancement may also be observed as some lesions are comprised of vascular components. Classic adamantinoma is often locally aggressive and requires radical surgical resection to reduce the risk of subsequent local recurrence and late distant metastases, which are most commonly seen in the lung.

**Osteofibrous Dysplasia**

Osteofibrous dysplasia (OFD), or ossifying fibroma, is a rare lesion which resembles FD and the stroma of adamantinoma. It is a benign, fibro-osseous lesion with a strong predilection for the cortex of the tibia and/or fibula, and rarely affects the radius and/or ulna. The lesion is most commonly seen in children less than 10 years old, and rarely reported in adults. The lesion may present with pain, progressive bowing deformity, pseudo-arthrosis or fracture. (15, 16) The distinction between FD, adamantinoma and osteofibrous dysplasia can be made histopathologically as OFD demonstrates osteoblastic rimming and bone zonation which are not seen in the other conditions. OFD has a more favourable prognosis than fibrous dysplasia or adamantinoma, as there is a tendency toward regression of the lesion.

**Plain Radiography and CT:**

OFD typically manifests as a long, lytic lesion in the anterior cortex of the tibial diaphysis with a well-defined geographic border and an adjacent sclerotic band. (fig. 17a&b).
Bowing deformity and pathological fracture may also be seen. CT is useful in assessing the extent of the lesion, identifying satellite lesions, and for pre-operative planning.

**MRI (fig. 18,19 &20):**

OFD often exhibits homogeneous intermediate signal intensity on T1-weighted sequences, a variable cystic to solid appearance on T2-weighted imaging and a variable contrast enhancement pattern.

**Osteofibrous Dysplasia-Like Adamantimoma**

Osteofibrous dysplasia-like adamantinoma (OFD-LA), also called differentiated, regressing or juvenile adamantinoma, is another pathological entity with similarities to both OFD and adamantinoma. (15) OFD and OFD-LA are differentiated at histology by the number of epithelial cells present and their staining characteristics. (15)

OFD, OFD-LA and adamantinoma have been shown to share similar cytogenetic abnormalities and are considered to be related conditions along the same pathogenic spectrum with overlapping clinical and imaging features. Longer lesions with moth-eaten margins, cortical destruction and complete intramedullary extension are more frequently seen with classical adamantinoma than with OFD or OFD-LA.

OFD may spontaneously regress with skeletal maturity and has traditionally been treated conservatively with clinical and imaging observation, and bracing for deformity. However, some cases may progress to debilitating bony deformity without surgical intervention. Furthermore, some lesions showing OFD or OFD-LA at needle biopsy have been upgraded to classic adamantinoma following surgical resection, highlighting the possibility of unrepresentative sampling with needle biopsies and the importance of imaging correlation to identify aggressive features. (17) Curettage and localised subperiosteal excision of OFD lesions have been shown to carry a risk of recurrence, whereas no recurrences have been shown with extraperiosteal excision of OFD. Therefore, some specialist bone tumour surgical units suggest more radical surgery for OFD. (18)

**3. Tibial Intra-medullary lesions with typical cortical involvement:**

**Brodie’s Abscess**
A Brodie’s abscess is a chronic infective lesion originating in the medullary cavity, but which typically progresses to involve the cortex and, for this reason, has been included in the review.

Brodie’s abscess is most common in the lower limb metaphysis of young male patients and is characterized by an insidious onset of symptoms and normal laboratory markers. (19,20) The tibia is the most commonly affected bone and Staphylococcus aureus the most common pathogen although cultures are sterile in 20 - 43% of cases. (19-21)

**Plain Radiography:**

On a radiograph a Brodie’s abscess typically appears as a well-defined round or ovoid radiolucent lesion within the metaphysis measuring 0.5 - 9 cm with thick surrounding sclerosis and a periosteal reaction. (fig. 21a). If the lesion becomes tethered to the growth plate, the cavity can elongate during growth and extend into the diaphysis and progression leads to osseous tunnelling and cortical penetration with the formation of a cloaca and soft tissue inflammation, although fistula formation is rare. The presence of a sequestrum, an irregular sclerotic focus of dead bone located eccentrically within the cavity, is highly specific for a Brodie’s abscess. (19,22)

**CT:**

CT demonstrates the radiographic findings with greater sensitivity and is superior to MRI in detecting a sequestrum. (23,24)

CT can also be used to guide percutaneous diagnostic aspiration/biopsy for microbiological and histological analysis to confirm the pathology and guide antimicrobial treatment. (25) Percutaneous CT guided aspiration and irrigation of the cavity can also be performed for therapeutic purposes. (26)

**MRI:**

MRI is the most sensitive and specific imaging technique to evaluate a Brodie’s abscess (figure 22). The central necrotic abscess fluid is hyperintense on T2/STIR, hypointense on T1 and does not enhance following contrast. A thin rim of highly vascular granulation tissue lines the abscess cavity, demonstrating signal hyperintensity on T2 and STIR sequences, which may be more hyperintense than the central abscess fluid. This results in a "double line" sign. (27) On T1 it is also hyperintense and it enhances avidly with contrast. The granulation tissue lines the sclerotic wall of the cavity which is hypointense on T1, T2 and STIR. On T1 images, this results in the "penumbra sign", with the hyperintense granulation tissue appearing as a thin penumbra between the hypointense central abscess fluid and the peripheral hypointense sclerotic wall.(27) This occurs in 27-75% of cases and is said to be more sensitive than the double line sign, which is seen
in only 22% of cases. (27,28) It is also more specific (96-99%) than the double line sign, which is also seen in avascular necrosis. (27,28) Nevertheless, it is not pathognomonic and is also seen in eosinophilic granuloma, chondrosarcoma, intraosseous ganglia and in benign cystic bone lesions following curettage. (29) The adjacent bone marrow is usually oedematous and hyperintense on T2/STIR and hypointense on T1. The intra-medullary abscess may extend through the cortex and form a soft tissue abscess.

**18**FDG PET/CT:

Although not used routinely (due to cost and lack of availability) when the MRI appearances are equivocal and clinical suspicion is high, **18**FDG PET/CT can serve as a useful adjunct. The metabolically active granulation tissue utilizes **18**FDG resulting in increased uptake at this site. Anatomical correlation with the CT component of the study can provide confirmation of the source. (30)

**Chondromyxoid Fibroma**

Chondromyxoid fibroma (CMF) the least common neoplasm derived from cartilage has an incidence of less than 1% of all tumour and tumour-like conditions of the bone. (31-33) It is a rare benign cartilage tumour containing variable amounts of chondroid, fibrous and myxoid tissue. Most of the cases are seen in second and third decades of life with no sex predilection. (32,33) CMF most commonly arises in the metaphysis of long tubular bones, with a predilection for the knee joint, particularly the proximal tibia. Clinically CMFs present with slow onset pain and swelling or also may be asymptomatic and found incidentally on radiography or MRI. Imaging is used to help characterise lesions and differentiate from other bone tumours or alternative cause for symptoms, but definitive diagnosis is made on histopathological analysis. The differential diagnoses for CMF include aneurysmal bone cysts, giant cell tumour of the bone, enchondroma, non-ossifying fibroma, fibrous dysplasia and chondrosarcoma. (34) Treatment options include curettage and packing with allogenic bone, en bloc resection, wide excision and amputation.

**Plain Radiography and CT:**

On conventional plain radiographs, CMF appears as a well-defined, lobulated, expansile, lucent, medullary lesion with a sclerotic rim in the metaphysis of a long tubular bone with an average size of 3 - 10 cm (fig 23). It is normally round or oval and the long axis of the lesion lies parallel to that of the bone. (32) In the long bones, it is normally eccentric, whereas in thin bones such as the ribs, fibula and small tubular bones, it may be more centrally placed. (33) It can extend into the diaphysis but rarely into the epiphysis. Like all chondroid lesions, CMF does demonstrate matrix calcification although less so than
most chondral lesions. Plain radiographs uncommonly demonstrate this calcification. Compared with plain radiographs, CT can better demonstrate the well-defined margins, cortical changes and intra-lesional calcification seen in CMF. However, whilst CT can increase the confidence that a lesion is cartilaginous, it rarely adds much more to the diagnosis.

**MRI (fig 24):**

The main role of MRI in the management of CMF is in pre-operative evaluation to accurately determine the extent of the tumour and aid a complete surgical excision to reduce the chance of recurrence.

MRI features of CMF can be nonspecific. In a series of 19 cases, Kim H-S et al reported that on T1-weighted images, CMF showed hypointense to intermediate signal intensity and hyperintense foci in approximately 40% of cases. On T2-weighted images, all lesions were hyperintense. In 60% of cases there was a peripheral intermediate signal band with central hyperintense signal, while in the remaining 40% there was a diffusely hyperintense heterogeneous pattern. On contrast enhanced T1-weighted images, peripheral nodular enhancement was noted in approximately 70% of the cases with diffuse enhancement in the rest.

Overall, the most helpful MRI findings in CMF are a peripheral intermediate signal band and central hyperintense signal on T2-weighted images, which correspond to the peripheral nodular enhancement and central non-enhancing portion on contrast enhanced images respectively. Periosteal reaction, adjacent abnormal bone marrow or soft-tissue signal and cortical abnormality can also be seen on MRI.

**Bone Scintigraphy and Angiography:**

Tc-99 bone scintigraphy demonstrates markedly increased uptake in the periphery, while there is little uptake on Ga-67-citrate scintigraphy. On Thallium-201 scintigraphy, there will be strong accumulation of the entire lesion in early and late scans and as such, it may be clinically useful to help distinguish CMF from chondrosarcoma.

On angiography, CMF may demonstrate slight neovascularization and it is therefore of limited value in diagnosis.

**Fibrous Dysplasia**

Fibrous dysplasia (FD) is a benign developmental dysplasia of bone with an incidence of 7% in which abnormal differentiation of osteoblasts leads to replacement of normal marrow and cancellous bone by immature bone and fibrous stroma. FD is
a medullary lesion that displays classical radiological features including secondary cortical changes. FD can undergo secondary aneurysmal bone cyst formation due to haemorrhage and can be complicated by pathological fracture and rarely, malignant change which occurs in 0.5% of cases. FD may be monostotic or polyostotic. 75% cases occur before the age of 30 and the male to female ratio is 1:1. Monostotic FD is more common accounting for 70-85% of cases. In polyostotic FD, patients present earlier in life, 67% by the age of 10 years. 30-50 % patients with polyostotic FD have café-au-lait spots and endocrine disorders. McCune-Albright syndrome presents with polyostotic FD, ipsilateral café-au-lait spots and endocrine disturbance, usually precocious puberty in girls. In Mazabraud's syndrome, FD is usually polyostotic and patients present with multiple soft tissue myxomas, typically in large muscle groups.(37,38)

Plain Radiography:

The radiographic appearances are widely variable depending on the degree of fibrous, osseous or cystic change. An FD lesion is usually an intramedullary, expansile and radiolucent with a homogenous, ground glass appearance which is similar to cancellous bone density but without a visible trabecular pattern. It usually has a thin rim of reactive bone that is more sharply defined on the inner border. As the lesion grows, its diameter increases resulting in mild expansion but the thin rim of bone remains although it may be too thin to detect radiographically. Slow endosteal resorption results in endosteal scalloping but there should not be a periosteal reaction unless there is a pathological fracture.

In monostotic FD, the commonest sites are: ribs (28%), proximal femur (23%) and craniofacial bones (20%). In polyostotic FD, two or more bones are involved in more than 75% of the skeleton. The femur, tibia and pelvis are most commonly affected. The Harrison groove (horizontal depressions of the 6th and 7th costal cartilages at the site of attachment of the anterior portion of the diaphragm), coxavara, protrusion acetabuli, the Sheperd's crook deformity of the femur and bowing of the tibia can all be seen as secondary deformities, particularly in larger lesions.(39)

CT:

CT is useful when radiography is equivocal and it is the best test to demonstrate the cortical rim of reactive bone around the lesion which is known as the "rind sign". (4) It also the best way to assess for a ground glass appearance which is typical of FD. (fig. 26)

MRI:

MRI is a useful adjunct to CT and is the best way to assess the content of the lesion. The signal intensity is determined by the predominant tissue type: fibrous - low to intermediate
on all sequences (fig. 27 a&b); cystic degeneration - low on T1 and hyperintense on T2/STIR; subacute haemorrhage - hyperintense on T1 and T2 with fluid-fluid levels. Following intravenous contrast, solid areas show uniform enhancement and cystic areas show rim enhancement.
Images for this section:

**Figure 1a:**
AP and lateral radiographs of the leg, showing cortical thickening of the medial tibial diaphysis (arrow).

**Figure 1b:**
Axial figure CT image showing cortical thickening of the medial tibial diaphysis.

**Fig. 1**

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Figure 2a & 2b:
Early vascular phase and delayed static phase (fig b) TC-99m bone scan, showing increased isotope uptake at the site of the tibial stress fracture.

Fig. 2
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Figure 3:
Lateral radiograph of the leg demonstrating a subtle linear lucency (arrow).

Fig. 3

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Figures 4a and b:
Coronal and axial figure CT images showing the linear hypodensity of a stress fracture (arrow).

Fig. 4

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Figures 5a, b, c:
Axial T1 weighted (fig 5a), T2 weighted (fig 5b), and T1W fat-suppressed post gadolinium (fig 5c) images showing posteromedial cortical and medullary oedema (arrow) secondary to a tibial stress fracture.

Fig. 5

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Figures 6 a, b and c:
Axial T1 weighted (fig 6a), image of a non-ossifying fibroma located in the lateral tibial metaphyseal cortex (arrow). Fat suppressed T1 weighted image pre-gadolinium (fig 6b), and post-gadolinium (fig 6c), showing predominantly peripheral enhancement of the lobulated non-ossifying fibroma.

Fig. 6

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Figure 7: AP radiograph of the lower leg in a patient with melorheostosis involving the anterolateral tibial cortex.

Fig. 7

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Figure 8a:
Lateral radiograph of the knee in a patient with melorheostosis, demonstrating peri-articular and intra-articular involvement with calcified soft tissue masses (arrows).

Figure 8b:
Axial CT image in a patient with tibial melorheostosis, with extension into the medullary cavity (arrow).

Fig. 8

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Figure 9:
$^{99m}$Tc-Methylene Diphosphonate (MDP) Bone Scan, demonstrating increased cortical uptake in a patient with left distal femoral melorheostosis.

Fig. 9

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Figure 10 a, b, c:
Lateral plain radiograph showing sclerosis and thickening of the anterior tibial cortex due to an osteoid osteoma (fig 10a). The nidus is seen as a subtle focus of lucency on the radiograph (arrow). Sagittal CT reformat (fig 10b), and axial CT image (fig 10c) of a tibial osteoma demonstrating a hypodense central nidus (arrows) with a mildly calcified matrix and surrounding cortical sclerosis.

**Fig. 10**

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Figure 11a, b & c:
Coronal T1 weighted image (fig 11a) of the isointense nidus (arrow), within an area of hypointense cortical thickening. Axial T2 SPAIR (fig 11b) image of an osteoid osteoma. The marrow in the tibial medulla returns a high signal intensity (white arrow). Sagittal STIR image (fig 11c) of the osteoid osteoma (red arrow).

**Fig. 11**

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Figure 12:
Early vascular phase Tc-99m isotope bone scan, showing increased isotope uptake in a tibial osteoid osteoma.

**Fig. 12**

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Figure 13:
Plain radiograph showing an expansile mixed lucent and sclerotic tibial cortical adamantinoma (arrow).

Fig. 13
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Figure 14a & b:
Axial CT and CT biopsy images showing a tibial adamantinoma (arrow), with extension into the medullary canal.

Fig. 14

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Figure 15a and b:
Axial T1 weighted (fig 15a), T2 weighted (fig 15b) images showing the heterogeneous tibial adamantinoma (arrows), demonstrating intra-medullary extension.

Fig. 15

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Figure 16:
TC-99m bone scan, showing increased isotope uptake at the site of the tibial adamantinoma.

**Fig. 16**

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Figure 17a and b: Lateral radiograph (fig 17a), and axial CT image (fig 17b), of a well-defined expansile intracortical osteofibrous dysplasia lesion (arrows).

**Fig. 17**

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Figure 18a, b & c:
Axial pd and pd fat-suppressed images (fig 18a and b), and sagittal T2 weighted image (fig 18c) of the same lesion as the plain radiograph and CT, of a well-defined expansile intracortical osteofibrous dysplasia lesion (arrows).

Fig. 18

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Figure 19:
Axial PD image showing an anterior tibial osteofibrous dysplasia lesion.

**Fig. 19**

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Figures 20 a,b,c:
Coronal T1W (fig a), sagittal T2W (fig b) and coronal STIR (fig c) images showing an anterior tibial osteofibrous dysplasia lesion.

Fig. 20

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Fig. 21a: Plain radiograph of a Brodie's abscess involving the proximal tibia (arrow), seen as a metaphyseal lucency with a mildly sclerotic superior margin.

Fig. 21b: Ultrasound of a Brodie's abscess involving the proximal tibia, with cortical break-through into the soft tissues (arrow).

Fig. 21

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Figures 22 a,b&c
Axial T1 weighted (fig 22a), T1 weighted fat- suppressed (fig 22b) and STIR (fig 22c) images through a proximal tibial Brodie’s abscess. Axial pre-gadolinium (fig 22a), and post-gadolinium (fig 22b) T1 weighted images of the tibial diaphysis, showing an enhancing subcortical abscess extending through the anteromedial tibial cortex, into the overlying soft tissues (arrow). Fig 22c showing the hyperintense abscess cavity.

Fig. 22

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Figure 23. Plain AP (a) and Lateral (b) radiographs of a 40 year old man. The lesion is a well-defined, eccentric, lucent lesion in the proximal tibial metaphysis involving the anterior cortex. The lesion is a chondromyxoid fibroma.

**Fig. 23**

© Royal National Orthopaedic Hospital, Stanmore
chondromyxoid fibroma

Figure 24: Axial T1W (a), axial T2W (b) and sagittal STIR (c) images (same lesion as the plain radiographs) show the lesion with intermediate signal intensity on T1W image (a), heterogeneous hyperintense signal on T2W image (b) and uniformly hyperintense on STIR sequence image (c). The lesion is a chondromyxoid fibroma.

Fig. 24

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Figure 25:
AP radiograph of the right lower leg, showing a pathological fracture through a diaphyseal fibrous dysplasia lesion (arrow). Note the ground glass matrix and mild expansion and cortical thinning, best appreciated at the lateral margin of the lesion.

Fig. 25

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Figure 26:
Coronal reformat showing the ground glass matrix (thick arrow), and endosteal scalloping (thin arrow) of fibrous dysplasia.

**Fig. 26**

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Figure 27 a&b:
Coronal T1 weighted (figure 27a), and STIR (figure 27b) of tibial fibrous dysplasia, returning a low signal intensity on T1, and an intermediate signal intensity on the STIR sequence (white arrows). Note the surrounding soft tissue oedema, secondary to the pathological fracture through the cranial aspect of the lesion (black arrow).

Fig. 27

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Conclusion

There are many pathological entities that should be considered in the differential diagnosis of a tibial cortical lesion. We have described pathologies that are most common or important, describing and demonstrating the typical imaging features that aid in making the diagnosis. A CT guided biopsy is typically performed in cases without an unequivocally benign appearance on multi-modality imaging.
References


