Soft tissue calcification; a pictorial review

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Learning objectives

1. Be able to confidently construct a list of differential diagnoses of soft tissue calcifications

2. Increase confidence of definitive diagnosis given the radiographic and MRI features of the calcifications

Background

Whilst much emphasis is placed on radiographic interpretation of osseous lesions, the same could be true for assessment of extra-osseous calcification. There are multiple causes of soft tissue calcification and detailed assessment of the distribution, morphology and location provides significant interpretative value to provide a shortened differential or hopefully a definitive diagnosis including recommendation of further investigation. We have used the common approach to developing a differential diagnosis using the 'surgical sieve' including vascular, infections, neoplasms, drugs, connective tissue disorders, metabolic, traumatic and miscellaneous sections. In each section the appropriate plain radiograph, computerised tomography (CT), magnetic resonance imaging (MRI) and nuclear medicine (NM) features are presented for each differential with their typical imaging findings and locations described.

Imaging findings OR Procedure Details

Fig. 1 on page 10 Vascular

Phleboliths

Phleboliths represent calcification within venous structures and are most commonly demonstrated on abdominal radiographs within the pelvic vessels. Phleboliths appear as well-defined calcifications, usually rounded often with radiolucent centres. These appearances are attributed to calcification peripherally in the vessel. These are normally easily demonstrated on plain radiography Fig. 1 on page 10 and CT scan Fig. 2 on page 12, but if there is an associated soft tissue mass then MRI is useful for further characterisation. This allows identification of the vessel anatomy, soft tissue component of the lesion and adjacent anatomy whilst contrast enhanced CT angiography is useful for delineating the vessel anatomy/malformation.
Neoplasm

Soft tissue haemangiomas are common benign vascular tumours occurring most often in children and young adults. Patients present with soft tissue masses of varying size that may cause pain during activity. A soft tissue mass with phleboliths is nearly pathognomonic if present Fig. 3 on page 14. Reactive bony changes and smooth periosteal reaction can also seen. On CT these masses are often intramuscular with reactive overgrowth of fat. On magnetic resonance imaging, haemangiomas contain flow voids, avidly enhance and are often hyperintense to muscle on T1 and T2-weighted images. Compression ultrasonography is easily utilised in superficial soft tissue vascular malformations to assess flow within the lesion and confirm its compressibility. Haemangiomas are seen in association with multiple enchondromas in Maffucci Syndrome Fig. 4 on page 14.

Parosteal lesions

Lesions arising from the surface or periosteal (parosteal) surface of the bone may give the imaging appearance of a calcified soft tissue based lesion. They are therefore included in this differential diagnosis list. The different types of neoplasm both benign and malignant can have their origin from the periosteal surface of bone. They generally follow the classic imaging appearances of the central osseous type tumour.

Periosteal chondrosarcoma is very rare representing about 4% of all chondrosarcomas. It normally occurs in the third to fourth decades with a non-specific painless, slow growing soft tissue mass. Lesions are usually in a metaphyseal or metadiaphyseal region and can reach sizes up to 14 cm in length (1). Thickening or thinning of the underlying cortex is common but complete cortical destruction is not a feature. The typical chondral type matrix mineralisation is present in the majority of cases Fig. 5 on page 16. Imaging reveals a thin peripherally calcified margin in about 50% of cases Fig. 6 on page 16 with MRI revealing a lobular growth pattern with characteristic chondroid type calcification with low to intermediate T1-weighted signal and high T2-weighted signal Fig. 7 on page 17. Surrounding reactive oedema-like changes in the medullary cavity and peri-lesional soft tissue are uncommon findings but can be seen in lesions that exhibit dedifferentiation. It's benign cousin, perisosteal chondroma Fig. 8 on page 18 and Fig. 9 on page 19, has similar imaging appearances with with no major imaging features for differentiation between the lesions barring larger size in chondrosarcoma versus chondroma (2).

Parosteal Osteosarcoma
Parosteal osteosarcoma arises from the cortical surface as a densely mineralised mass which projects into the soft tissues. Parosteal osteosarcoma affects patients in the second and third decades of life and on radiographic examinations there is perpendicular sheets of calcification Fig. 10 on page 20 associated with a juxta-cortical mass lesion. A radiolucent cleavage line represents a plane between the portions of the tumour and cortex of affected bone and is referred to as a 'string' sign. Commonly the tumour stalk is broad based with cortical erosion a prominent feature Fig. 11 on page 21. MRI features include low T1 and T2-weighted signal with high T2 signal a feature of high grade lesions.

They are relatively uncommon compared with central osteosarcomata and generally occur within the lower extremity. Extra-skeletal osteosarcoma can develop as calcified soft tissue masses again with calcified soft tissue masses more common in the lower extremities. Extra skeletal osteosarcomas demonstrate central ossification, which is referred to as reverse zoning (3) Fig. 12 on page 22.

Synovial sarcoma

These lesions typically present in the second to fourth decades with a predominance within the lower limb (60 to 70% of cases). It is a malignant mesenchymal neoplasm which is highly aggressive with 25% of new diagnoses having metastatic disease at presentation (4). There are epithelial and spindle cell components and its the hyalinisation of these that result in punctuate calcification, and ossification in approximately a third of cases. The imaging appearances of these lesions are variable - most appear in a periarticular location but interesting only 5% occur in an intra-articular location (5). Bony involvement varies from superficial erosions, periosteal reaction to invasion. MRI demonstrates poorly defined and infiltrative lesions, some having high T1-weighted signal consistent with haemorrhage. On T2, triple signal intensity is seen due to haemorrhage, fibrous tissue and cystic components often containing fluid-fluid levels Fig. 13 on page 24. Recent literature suggests that stippled fine calcifications in a soft tissue mass should raise suspicions for a synovial sarcoma (6).

Soft tissue metastasis

Although rare, soft tissue metastases from carcinoma should remain a differential diagnosis in any patient presenting with a suspicious soft tissue lump. Whilst the MRI scan appearances can be suggestive of malignancy, they are not diagnostic of metastases (7) Fig. 14 on page 24 and Fig. 15 on page 25. Biopsy could confirm the diagnosis and may be helpful in the detection of the possible origin of the primary. The most common primary sites were lung, kidney and bowel (8).

Parosteal ossifying lipoma
Ossifying lipomata are a rare lipoma variant that usually occur in parosteal locations Fig. 16 on page 26. They are of unknown aetiology but are thought to be a metaplastic process. Lesions are relatively well defined with peripheral calcification and exhibit fat signal on all MRI pulse sequences Fig. 17 on page 28 and Fig. 18 on page 28. Ossification within an intramuscular lipoma is rare (9) Fig. 19 on page 29.

Primary synovial chondromatosis

Primary synovial chondromatosis is a benign intra-articular disorder of unknown origin characterised by multiple intra-articular cartilaginous loose bodies which may or may not be ossified. It is a metaplastic rather than neoplastic process. Patients commonly present in their fourth or fifth decades and the condition occurs more frequently in males. The presenting complaint is normally with pain, swelling and reduced range of motion which progresses over several years. It is a self limiting benign neoplastic process characterised by proliferative chondroid nodules on the synovium. The disease process has three demonstrable phases including the initial phase where there is metaplastic formation of cartilaginous nodules in the synovium followed by the transitional phase with detachment of the nodules leaving free intra-articular bodies and finally the inactive phase where proliferation ceases but the loose bodies may increase in size receiving nourishment from the joint fluid by diffusion Fig. 20 on page 30. The disease process preferentially affects the larger joints with the knee most commonly affected (up to 70% of cases). Occasionally bursae and tendon sheaths may be involved (10). Plain film and CT imaging findings are dependent upon the degree of calcification although approximately 30% of these lesions do not calcify (11). Otherwise, the imaging appearances are rather non-specific ranging from normal to non-specific soft tissue mass surrounding the joint, widening of joint space and erosion of adjacent bones. If there is extensive calcification, then there is easy identification of multiple, uniform sized ossified or chondroid type calcifications throughout the joint. MRI appearances demonstrate signal void consistent with the areas of the mineralisation. Gradient echo sequences make these more conspicuous. Predominantly unmineralised nodules will demonstrate typical chondroid signal characteristics of intermediate to low T1-weighted signal and high T2 weighted-signal with low signal foci representing the calcifications Fig. 21 on page 31. The high T2-weighted signal is secondary to the high water content of the non-mineralised regions. Solitary lesions represent chondromas and can originate from synovium or tendon sheath. Malignant transformation is rare but suggested by multiple recurrences following surgery and marrow permeation and invasion.

Connective Tissue Disorders

Dermatomyositis
There is a recognised female predilection in connective tissue disorders including dermatomyositis. It has a bimodal age of presentation depending on the variant. Juvenile dermatomyositis affects children and tends to be more severe compared with the adult type which typically affects adults around the age of fifty.

Imaging typically show dystrophic calcification in muscles and soft tissues (calcinosis universalis) which is sheet-like although at least four patterns have been described Fig. 22 on page 33. Childhood dermatomyositis classically is seen affecting the thigh regions.

Metabolic

Tumoural calcinosis

Tumoral calcinosis is a rare familial condition characterised by painless, periarticular calcified masses. Hereditary metabolic dysfunction of phosphate regulation with normal calcium levels and is characterised by massive periarticular calcinosis Fig. 23 on page 35. It predominantly affects young black patients with equal male to female preponderance and most often presents in the first or second decades of life. There are large amorphous calcific densities that surround joints, generally the extensor surfaces and may follow the anatomic distribution of bursae. These are separated into lobules by fibrous septae and may demonstrate fluid/calcium levels which represents milk of calcium hydroxyapatite crystals in suspension. The hips, elbows and shoulders are the most common sites of involvement. If there is homogenous calcification this suggests disease inactivity and a reduced likelihood of growth. The cystic appearing type has fluid-fluid levels caused by calcium layering which is referred to as the sedimentation sign (12). Underlying bone erosion or destruction is characteristically absent. MRI demonstrates in-homogenous low T1-weighted signal with low T2-weighted signal in the diffuse type or high T2 signal voids consistent with calcific nodules. Nuclear medicine isotope bone scintigraphy is a sensitive method for diagnosing tumoral calcinosis demonstrating increased tracer uptake Fig. 24 on page 35 (13).

End stage renal disease

End stage renal disease is a common disease due to the number of patients receiving peritoneal or haemodialysis. The systemic manifestations of this disorder are widespread, with musculoskeletal involvement commonly seen. It is secondary to inappropriately high levels of calcium and phosphorus which results in soft tissue calcification. The lesions are mostly asymptomatic but have been reported to cause reduced joint and limb movement, as well as neuropathic symptoms from compression of adjacent nerves. Given their similar aetiology, the imaging appearances closely follow that of tumoral calcinosis. They are generally found in a periarticular distribution and can be commonly found on the
extensor aspects of the extremities. They are typically well-defined, lobulated calcified masses with interwoven fibrous septae that may demonstrate fluid-calcium levels (14) Fig. 25 on page 36.

Tophaceous gout

Tophaceous gout is characterised as deposits of urate crystals secondary to chronic hyperuricaemia. There is a development of a periarticular mass of variable radio-opacity, with or without focal erosions of underlying bone. Urate crystal deposits are typically less radiopaque than normal calcifications Fig. 26 on page 37.

Trauma

Calcific myonecrosis.

This disease process occurs following ischaemic necrosis of muscle and is characteristically seen secondary to compartment syndrome. It is particularly common within the anterior compartment of the lower leg (15). Imaging reveals classic intramuscular lesions with peripheral calcification Fig. 27 on page 39 and Fig. 28 on page 39. The soft tissue lesion is seen to replace the necrotic muscle, and as a result there is little or no mass effect due to this calcific mass lesion (16).

Avulsion injury

This is a common presentation in sports participants especially adolescents. Avulsion injuries can present on imaging at a variety of stages of evolution. In acute injuries, there is a well defined bony fragment which is normally well defined with or without surrounding haematoma or tendon/muscle oedema on MRI fluid-sensitive sequences. If the displaced fragmented is over 2 cm from its donor site fibrous union between the fragments is likely to occur which can cause extended disability. Subacute injuries can demonstrate osteolysis and sclerosis whereas chronic avulsion injuries result from repetitive microtrauma or inactive acute injuries and present with protuberant bony masses which can mimic tumour masses Fig. 29 on page 40 and Fig. 30 on page 40. There are multiple potential avulsion injury sites in and around the pelvis with the commonest site at the hamstring group inserting at the ischial tuberosity. Avulsions can be also be seen in and around the knee, particularly in the case of a Segond fracture which has significant associated intra-articular soft tissue injuries.

Hypertrophic ossification.
Hypertropic ossification is characterised by a mass of maturing bone that can occur at various different sites. It is mostly seen around large joints and is not restricted solely to muscle. There is progressive formation of cortical and trabecular bone structure demonstrated at these extra-articular sites (18) Fig. 31 on page 42. There is abnormal presence of extra-osseous osteoblastic cells that may be secondary to many different insults including fracture, burns, paraplegia or joint replacement.

Myositis ossificans

Myositis ossificans (MO) is characterized by abnormal heterotopic bone formation involving striated muscle, tendons, ligaments, fasciae, and aponeuroses. Myocardium, the diaphragm, tongue, larynx, smooth muscle, and sphincters are all spared. Several subtypes of myositis ossificans exist: post-traumatic myositis ossificans (PTMO), non-traumatic/pseudomalignant myositis ossificans, and myositis ossificans progressiva (MOP) (19).

Although PTMO is a benign self-limiting condition, imaging is an important tool to exclude infection or malignancy. Imaging findings typically change with lesion maturity as documented in the table below:

<table>
<thead>
<tr>
<th>Phase</th>
<th>Early/active</th>
<th>Intermediate/sub-acute</th>
<th>Mature</th>
</tr>
</thead>
<tbody>
<tr>
<td>Time</td>
<td>&lt;2-4 weeks</td>
<td>4 weeks-6 months</td>
<td>&gt;6 months</td>
</tr>
<tr>
<td>Plain film</td>
<td>Soft tissue swelling.</td>
<td>Well-defined peripheral calcification.</td>
<td>Densely calcified lesion. Usually parallel to long axis of adjacent bone.</td>
</tr>
<tr>
<td></td>
<td>Faint peripheral calcification.</td>
<td>Coarser central calcification may be present.</td>
<td>Fig. 32 on page 42</td>
</tr>
<tr>
<td>US</td>
<td>Hypoechoic soft tissue mass with hyperechoic core.</td>
<td>Peripheral lamellar calcification.</td>
<td>Highly reflective, heavily calcified. Rim may be irregular due to lesion shrinkage.</td>
</tr>
<tr>
<td></td>
<td>Fluid levels (hemorrhage) may be present (a nonspecific finding).</td>
<td></td>
<td>Fig. 33 on page 44</td>
</tr>
<tr>
<td>Bone scan</td>
<td>Increased tracer uptake on all phases.</td>
<td>Decreasing tracer uptake.</td>
<td>Normal/ mildly increased uptake.</td>
</tr>
<tr>
<td>-----------</td>
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<td>---------------------------------</td>
</tr>
<tr>
<td>CT</td>
<td>Soft tissue swelling.</td>
<td>Peripheral calcified rim. Central zone isodense to muscle.</td>
<td>Dense calcification of lesion.</td>
</tr>
<tr>
<td></td>
<td>Faint calcification may be present.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>MRI</td>
<td>Intramuscular nodule/swelling which is Isointense on T1 and Hyperintense T2.</td>
<td>Variable central signal. Low signal intensity rim and central foci all sequences.</td>
<td>Generally low signal on all sequences. No perilesional edema.</td>
</tr>
<tr>
<td></td>
<td>Peripheral or general enhancement.</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>May be faint low signal foci or rim</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Miscellaneous**

**Calcitic tendinitis/ bursitis**

Calcitic tendinitis occurs in approximately 3% of adults and in approximately 40% of painful shoulders. There is characteristically depositions of hydroxyapatite within the tendons and tendon sheaths. The aetiology of this disease processes is unclear however there is apparent tissue hypoxia in reparative phase resulting in abnormal fibrocartilaginous metaplasia leading to crystal deposition and ossification. It is typically seen in the shoulder particularly within the supraspinatus tendon and there are several different phases of calcification described. Four stages include pre-calcific, calcific, resorptive and post-calcific stages. Calcific tendinitis can rarely cause invasion of the underlying bone (20) which can mimic tumours and they’re can also be migration of the calcific foci into adjacent bursa Fig. 34 on page 46 and Fig. 35 on page 46 and tendon sheaths.

**Nuchal ligament calcification**
Nuchal ligament calcification causes are quite limited and of unknown aetiology. Idiopathic cause should be considered, after exclusion of an associated soft tissue mass, as well as more commonly occurring entities such as a sesamoid ossicle, round ossified nodules (21), and nuchal fibrocartilaginous pseudotumour. Some reports have suggested that nuchal ligament calcification Fig. 36 on page 47 is associated with underlying cervical spine degenerative changes, although this has only been reported in a small case series (22).

Fibrodysplasia ossificans progressiva

It is a rare autosomal dominant disease characterised by fibrous tissue (including muscle, tendon and ligaments) becoming ossified spontaneously or when damaged (23) Fig. 37 on page 49. In many cases, injuries can cause joints to become permanently ankylosed, and is extremely debilitating and ultimately fatal. Surgical resection of the calcified tissue exacerbates the soft tissue changes.

Images for this section:
**Fig. 1:** Lateral radiograph demonstrating multiple large phleboliths in an extensive arterio-venous malformation of the leg.
Fig. 2: Sagittal T2-weighted MRI image, in the same patient as figure 1, demonstrating the extensive arterio-venous malformation of the leg with large hypointense phleboliths (arrow).

Fig. 3: Lateral radiograph demonstrating a soft tissue mass with phlebolith (arrow) which is virtually pathognononic for haemangioma.
Fig. 4: AP projection of the hand demonstrating multiple enchondromas, note the healing pathological fracture within the proximal phalanx, with soft tissue phlebolith (arrow) consistent with Mafucci’s syndrome.
Fig. 5: Plain radiograph of the pelvis demonstrating typical chondroid type 'rings and arc' soft tissue calcification (arrow) in a recurrent chondrosarcoma following previous right hemi-pelvic resection.
**Fig. 6:** Axial CT scan of the pelvis, in the same patient as figure 5, demonstrating typical chondroid type 'rings and arc' soft tissue calcification (arrow).
**Fig. 7:** Sagittal T2-weighted sequence, in the same patient as figure 5, demonstrating a large soft tissue mass with predominantly high T2-weighted signal with low signal representing the calcifications (arrow).
Fig. 8: AP radiograph demonstrating a well defined peripherally calcified lesion projecting into the soft tissues of the leg (arrow).
**Fig. 9:** Coronal T1-weighted image, in the same patient as figure 8, demonstrating a low T1-weighted signal arising from the cortical surface consistent with a parosteal/surface chondroma (arrow). There is no abnormal underlying bone marrow signal change.
Fig. 10: Axial CT scan demonstrating vertical sheets of ossification related to the posterior cortical femoral surface with associated soft tissue mass extending anteriorly consistent with a parosteal osteosarcoma (arrow).
**Fig. 11:** Axial T2-weighted image, in the same patient as figure 10, demonstrating intermediate to high signal soft tissue mass with ossification demonstrated. There is cortical erosion posteriorly consistent with parosteal osteosarcoma (arrow).
Fig. 12: Sagittal MIP of a contrast enhanced CT demonstrating an enhancing soft tissue mass with central ossification (arrow), which is referred to as reverse zoning, which is typically seen in an extra-skeletal osteosarcoma.

Fig. 13: Axial T2-weighted image demonstrating a soft tissue mass with low, intermediate and high signal components (correlating arrows), known as triple signal intensity, consistent with a synovial sarcoma.
Fig. 14: AP pelvis radiograph demonstrating multiple sclerotic metastases secondary to prostatic carcinoma with focal ossified density projected inferomedially to the ischial tuberosity (arrow).
Fig. 15: Axial post contrast T1-weighted MRI with fat saturation, in same patient as figure 14, demonstrates the ossified lesion separate to the hamstrings group insertion with peripheral contrast enhancement (arrow) suggestive of a soft tissue metastasis.
**Fig. 16:** Lateral radiograph of the shoulder girdle demonstrating a well defined peripherally calcified lesion originating from the anteromedial border of the right scapula (arrow).

**Fig. 17:** Axial fat saturated MRI sequence, in the same patient as figure 16, demonstrates loss of signal centrally in the lesion with peripheral calcification projecting from the anteromedial surface of the scapula (arrow) consistent with an parosteal ossifying lipoma.
**Fig. 18:** Coronal T1-weighted MRI sequence, in the same patient as figure 16, demonstrates fat signal centrally with confirmation of the parosteal location (arrow) with no underlying abnormal bone marrow T1 signal change.
**Fig. 19:** AP radiograph of the pelvis in a patient with a lipoma with an ossified component (white arrow) in the soft tissues overlying the right proximal femoral diaphysis.
**Fig. 20:** Lateral radiograph of the elbow demonstrating multiple intra-articular densities (arrow) in a patient with synovial chondromatosis.
**Fig. 21:** Sagittal T2-weighted image demonstrating extensive high T2 signal representing water in the non-ossified (multiple low T2 foci) components in synovial chondromatosis. There is extensive spread to involve the bursae surrounding the joint (arrows).
**Fig. 22:** AP radiograph of the tibia and fibula demonstrating sheet-like calcification in the adjacent soft tissues in a case of dermatomyositis.

**Fig. 23:** Lateral radiograph of the elbow demonstrating massive periarticular calcinosis (arrow) towards the extensor aspect of the elbow in a case of tumoral calcinosis. There are no erosions or secondary osseous destruction.
**Fig. 24:** Lateral elbow projection of a nuclear medicine study demonstrating increased isotope uptake (arrow) in a periarticular calcification of tumoral calcinosis.
Fig. 25: Axial CT demonstrating periarticular calcinosis in a patient on haemodialysis for end-stage renal failure. There are multiple fluid-fluid levels within the calcinosis (arrow).
**Fig. 26:** Plain radiograph of the great toe in tophaceous gout with long-standing hyperuricaemia demonstrating radiopaque soft tissue surrounding the joint with severe underlying erosive changes (arrow).

**Fig. 27:** Axial CT image of the lower leg demonstrating bilateral, well marginated, peripherally calcified masses in the anterior compartments of the lower legs replacing necrotic muscle (arrows).
**Fig. 28:** MRI imaging, in the same patient as figure 27, confirms the CT findings with high STIR signal lesion with peripheral low signal intensity representing calcification replacing muscle in the anterior compartments of the lower legs (arrows).

**Fig. 29:** AP pelvis radiograph demonstrating a well defined osseous mass projected inferiorly to the left ischial tuberosity (arrow) consistent with a large chronic avulsion of the hamstring group origin.
**Fig. 30:** Sagittal T2-weighted image, in the same patient as figure 29, demonstrating the large avulsed fragment attached to the hamstring tendon (arrow).

**Fig. 31:** Axial CT image demonstrating heterotopic ossification around both hip joints (arrows).
Fig. 32: Lateral radiograph of the femur demonstrates heterogeneous soft tissue mineralization (arrows) with an associated periosteal reaction in acute phase myositis ossificans (arrowhead).
Fig. 33: Anteroposterior radiograph of the thigh showing calcification paralleling the femoral shaft (arrow) in mature myositis ossificans.

Fig. 34: Lateral radiograph of the elbow demonstrating calcification overlying the volar aspect of the proximal radius (arrow) in the region of the biceps tendon attachment.
Fig. 35: Transverse ultrasound image, in the same patient as figure 34, demonstrating anechoic fluid related to the distal biceps tendon with several hyperechoic foci casting acoustic shadows (arrow) consistent with calcific bursitis.
**Fig. 36:** Lateral radiograph of the cervical spine demonstrating non-specific calcification of the nuchal ligament (arrow). MRI, not included, did not reveal an associated soft tissue mass.
Fig. 37: Lateral radiograph of the right elbow demonstrating sheets of soft tissue calcification in fibrodysplasia ossificans progressiva (arrow).
Conclusion

The radiographic and MRI appearances of multiple causes of soft tissue calcifications are presented in a pictorial review to increase confidence in shortening a differential list or providing definitive diagnosis from a broad list of potential causes. Discussion of multimodality imaging in a multidisciplinary setting is advised to try and negate unnecessary biopsy and to optimise ongoing patient management.

References


**Personal Information**