Bone scintigraphy appearances of incidentally diagnosed soft tissue disorders in musculoskeletal imaging

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SUMMARY

Bone scintigraphy in clinical practice is primarily used for the evaluation of bony pathology. Incidental uptake in soft tissue is likely to be overlooked because it is generally believed to lack specificity. Although bone scintigraphy is not used as a first line investigation for musculoskeletal injuries, abnormal soft tissue uptake should prompt clinicians to reassess patient symptoms. The onus falls on the radiologist and/or nuclear physician to comment on the findings. Several interesting cases are presented here with findings that have been confirmed by other imaging modalities.

Key words: musculoskeletal system; radionuclide imaging; soft tissue injuries; technetium Tc-99m medronate.

INTRODUCTION

Musculoskeletal injuries affect a wide spectrum of the population. Injuries may be sustained during sports or normal daily activities, which contribute to gradual wear and tear. While the plain radiograph is the first line of investigation, bony lesions are better evaluated by CT or bone scan and soft tissue injuries by ultrasound or MRI.

The cases reviewed here include incidental scintigraphic findings of soft tissue uptake in muscles, tendons or entheses that led to more targeted imaging, typically ultrasound.

PATHOLOGY OF SCINTIGRAPHIC FINDINGS

The practice of radiological–pathological correlation is a dynamic approach to evaluating diagnostic images across the speciality. Specific histological features correspond with radionuclide accumulation seen in bone scintigraphy done with technetium-labelled diphosphonate tracers. The routine triphasic scan includes bone flow, blood pool and delayed phases.

The flow phase of the radionuclide scan is analogous to an angiogram where there is a rapid local accumulation of tracer relative to non-diseased tissue on the basis of differential perfusion. This may occur as a result of an increase in flow rate or an increase in the number of mature blood vessels, such as in vascular malformations or haemangiomata. These abnormalities typically occur in the soft tissue or in the viscera.¹

Uptake seen in the blood pool phase is due to leakage of radionuclide from blood vessels into the soft tissue. This is greatest in granulation tissue and neoplastic tissue where there is active angiogenesis and neovascularization. This process occurs following a stimulus for angiogenesis, such as fibroblast growth factor (in granulation tissue formation). This recruits endothelial cells from a vessel in a non-diseased area that proliferate and organize into capillaries. It is these newly formed capillaries that leak proteins and tracer into the extravascular space.¹

In the delayed phase, tracer uptake is due to the synthesis of new bone by osteoblasts. Following an insult, growth factors stimulate differentiation of precursor cells into osteoblasts which consequently secrete osteoid, the organic matrix of bone. After 5–15 days, mineralization occurs. In this process, calcium and phosphate in the extracellular fluid precipitate within the collagen matrix of the osteoid. The initial precipitate is in a disorganized state of calcium brushite crystals but these
crystals later mature into a highly organized calcium hydroxyapatite crystal. It is the phosphate tag of the radionuclide adhering to the bone crystals during the mineralization phase that gives the appearance seen in the delayed phase of bone scintigraphy.\(^1\)

There are numerous causes of abnormal soft tissue uptake in the muscle, tendons and their entheses. They can be best classified pathologically under two broad categories that correspond to the changes seen in bone scintigraphy: conditions that incite an inflammatory response, and those that lead to abnormal mineralization (Table 1).\(^2\)

We will present the scintigraphic findings of several interesting examples of relatively common musculoskeletal disorders. Table 1 lists the range of soft tissue disorders in musculoskeletal imaging which present with bone scan abnormalities.

**SUPRASPINATUS TEARS**

Most supraspinatus tears are located in the ‘critical zone’ about 1 cm proximal to its insertion on the greater tuberosity.\(^3\) The most common rotator cuff tendon tears involve the supraspinatus, followed by the infraspinatus and subscapularis. Subscapularis is more likely to tear first in anterior dislocation of the glenohumeral joint.

Focal increased isotope activity in bone scintigraphy at the site of the injured enthesis most likely reflects a local inflammatory reaction\(^4\) (Fig. 1). Sonographic findings of tendon tears include focal discontinuity of fibres with interposed fluid. Bursal hemiation into the defect with loss of the tendon’s convex margin may also result. Focal areas of increased echogenicity with an adjacent hypoechoic rim is common in small tendon tears whereas diffuse thinning tends to be seen in longstanding chronic partial thickness tears.\(^5\)

The pathogenesis of rotator cuff tear presenting in individuals with chronic tendinopathy is likely to be a combination of ageing, enthesopathy, previous injury and the inherent properties of the tendon.

**SUPRASPINATUS CALCIFICATION**

Calcific tendinitis is believed to occur secondary to tissue necrosis at the critical zone of the rotator cuff after hypoxic insults. Biochemical analysis of ruptured supraspinatus tendons in patients with degenerative tendinitis demonstrates higher concentrations of calcium crystals (calcium pyrophosphate, hydroxyapatite, tricalcium phosphate and amorphous calcium phosphate) compared to normal tendons. In contrast, patients with radiographically detected calcifying tendinitis have salts with a mineral composition consistent with hydroxyapatite. This suggests that dystrophic calcification in degenerative tendons is a pathologically distinct entity from cell-mediated calcifying tendinitis.\(^6\)

Increased activity at the site of calcium deposition might be explained by the local inflammatory reaction due to tissue necrosis in the blood pool images and active mineralization in the delayed images (Fig. 2).

Tendon calcification in ultrasound can be linear or globular. It is prominently echogenic, exhibiting distal acoustic shadowing but not anisotropy. The globular appearance is seen more typically in hydroxyapatite deposition disease.\(^7,8\) Calcification with little or no distal acoustic shadowing may be due to liquid or milk of calcium.

**INTRAMUSCULAR HAEMATOMA AND MYOSITIS OSSIFICANS**

Trauma, infection, immobility and drug abuse in the form of intramuscular injections are among the most common causes of myositis ossificans. The localized heterotrophic bone or cartilage formation is secondary to an exaggerated healing response of the haematoma. Initially, two distinct zones of the reparative haematoma are identifiable within weeks of the insult: an inner cellular zone and an outer zone of mature bone. Hyperaemia, which occurs after trauma, is responsible for

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**Table 1.** Musculoskeletal disorders with abnormal soft tissue uptake in bone scintigraphy (Adapted from Datz\(^2\))

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<tr>
<th>Inflammatory</th>
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<td>Trauma</td>
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<td>● Intramuscular injections</td>
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<td>● Surgical wound/scar</td>
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<td>● Electrical burns</td>
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<td>● Muscle tears</td>
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<td>● Tendinopathies</td>
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<tr>
<td>● Enthesopathies</td>
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<tr>
<td>● Intramuscular haematoma</td>
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<tr>
<td>● Myositis ossificans</td>
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<tr>
<th>Infection</th>
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<td>● HIV-associated myositis</td>
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<th>Metabolic</th>
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<tbody>
<tr>
<td>● Rhabdomyolysis (e.g. alcoholism)</td>
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<td>● Uraemic myopathy</td>
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<th>Ischaemia</th>
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<td>● Peripheral vascular disease with ischaemia</td>
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<th>Idiopathic</th>
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<th>Iatrogenic</th>
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<td>● Radiopharmaceutical preparations</td>
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<td>● Radiotherapy</td>
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<th>Connective tissue disorders</th>
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<td>● Dermatomyositis</td>
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<td>● Polymyositis</td>
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<td>● Synovitis</td>
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<th>Abnormal mineralization</th>
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<tr>
<td>● Calcific tendinitis</td>
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<td>● Dystrophic calcifications</td>
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<tr>
<th>Tumoral calcinosis</th>
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| Synovial osteochondromatosis |
increased vascularity while the soft tissue isotope activity is due to the expanded interstitial volume producing a relative increase in passive localization of the tracer (Williams S, unpubl. data, 2000). The circumferential pattern of activity adjacent to the haematoma in the delayed images may be due to floccular calcification that can occur in the reparative response of a haematoma.

Later, two patterns of calcification emerge. One is a well-defined lesion adjacent to a long bone and the other has an indistinct veil-like pattern. Calcification can be seen as early as 2 weeks on CT and approximately 6 weeks on plain films. Within 3 months a calcific mass with a central lucency forms and a lucent cleft may separate it from underlying bone. Most lesions start to regress by 4–6 months and some disappear within 1–2 years. Differentials include parosteal sarcomas, osteochondromas, idiopathic fat necrosis, fibromyositis ossificans and dermatomyositis.

When myositis ossificans is symptomatic and surgical removal is considered, a bone scan may be valuable in planning its removal. This is best undertaken when the ossification has stabilized and no further increased osteoblastic activity is seen at the site. When surgical removal occurs at this time, there is a lower recurrence rate of new heterotropic bone formation (Fig. 3).

PLANTAR FASCIITIS
The plantar fascia is a fibrous connective tissue band that maintains the arch of the foot and is a common cause of heel pain. It originates from the medial calcaneal tuberosity (a common site of palpable pain) and inserts into the plantar plates of the metatarsophalangeal joints. It is often referred to as ‘heel-spur syndrome’, a misnomer as the spur is not the initial cause of pain but the result of inflammatory bone growth due to recurrent microtrauma at the point of the fascial attachment. Predisposing factors are obesity, flat feet, high-arched rigid feet, sudden increase in athletic activity, tight Achilles tendons and prolonged weight bearing. The underlying pathophysiology is abnormal pronation whereby the talus will plantar flex and adduct upon standing while the calcaneus everts and stresses the plantar fascia. Although the diagnosis is usually clinical, imaging may have a complementary role. Standard (anteroposterior/lateral) plain films are mainly used to exclude stress fractures and erosions caused by inflammatory bursitis and enthesopathies. Magnetic resonance imaging is rarely indicated but may show plantar fascial thickening and adjacent inflammation. The plantar fascia thickness is normally about 3 mm and may increase to 6–10 mm in plantar fasciitis. Isotope activity in triphasic bone scintigraphy corresponds with the inflammatory changes at the enthesis (Fig. 4).
Fig. 2. (a) A 32-year-old man previously involved in a motorbike accident injuring his left shoulder re-presented after another fall off his bike. Blood pool image demonstrates a focus of increased uptake (black arrow), which corresponds with the site of the left supraspinatus tendon insertion. (b) Slightly increased focal osteoblastic activity is seen in the left humeral head (black arrow) on delayed images. This parallels the region of calcification seen on ultrasound in Fig. 2c. (c) Ultrasound confirms a focus of calcification in the left supraspinatus tendon (white arrow) with associated diffuse hypoechoic change but no demonstrable tears. Combined scintigraphic and sonographic findings are consistent with calcific tendinopathy of the supraspinatus.
Fig. 3. (a) A 34-year-old man who was knocked off his motorbike complained of increasing pain and swelling over his right hip over a 3-week period. Bone scan done to exclude an occult fracture shows increased vascularity and soft tissue uptake overlying the right iliac bone in the initial images. (b) Delayed images demonstrate increased curvilinear osteoblastic activity in the corresponding area (black arrow). No abnormal activity to suggest a recent fracture. (c) Ultrasound shows a large compressible hypoechoic intramuscular haematoma (white arrows) in the right tensor fascia lata muscle. (d) Frontal X-ray of the right hip demonstrates no evidence of calcification.
De Quervain’s tenosynovitis is a chronic stenosing tenosynovitis characterized by inflammation of the tendons of abductor pollicis longus and extensor pollicis brevis. These tendons traverse the wrist joint through the first dorsal (extensor) compartment just lateral to the radial styloid process. This condition is 10 times more common in women, usually in the dominant hand, usually occurring between the ages of 35 and 55 years. These patients typically have a history of chronic overuse injury. This has been described in bricklayers, pianists, golfers and those with an inflammatory arthropathy such as rheumatoid arthritis. If allowed to progress, resultant synovial hypertrophy and fibrosis produces a clinical picture similar to trigger finger with pain typically over the radial styloid and occasionally radiating to the thumb, shoulder or forearm.

The clinical test commonly used to evaluate for De Quervain’s tenosynovitis is the Finkelstein’s test, which involves the patient grasping the thumb of the affected hand with the other...
fingers and actively pulling it towards the little finger. Sharp pain is elicited over the radial styloid in sufferers of De Quervain’s tenosynovitis.

Scintigraphic findings demonstrate increased activity in the region around the radial styloid in the early images, probably due to the increased vascularity from the inflammatory response as granulation tissue is being laid down. Ultrasound findings include a hypoechoic halo surrounding both tendons best seen in transverse scans. A comparison with the contralateral side helps make the diagnosis. Sometimes the pathology is only seen at the musculotendinous junction in the more proximal variant of De Quervain’s tenosynovitis. T2-weighted MR imaging typically shows fluid around the tendons in the first extensor compartment (Fig. 5).

CONCLUSION

While abnormal soft tissue uptake of isotope is an unexpected finding in bone scintigraphy, the recognition of specific disorders with characteristic or suggestive appearances greatly enhances the diagnostic value of the examination. When combined with clinical findings, the range of soft tissue disorders can be diagnosed more confidently. Moreover, the great advantage of bone scintigraphy is the simplicity of the technique with a high degree of lesional contrast.

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REFERENCES