PICTORIAL ESSAY | Musculoskeletal Imaging

Tenosynovial Giant Cell Tumours: MRI Imaging Pearls - A Pictorial Review

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ABSTRACT

Tenosynovial giant cell tumours (TSGCTs) are uncommon synovial neoplasms arising from the synovium of joints, bursae, and tendon sheaths. Although histologically benign, these lesions may exhibit locally aggressive behaviour, particularly in diffuse forms, leading to progressive joint destruction, functional impairment, and frequent recurrence after surgical treatment. Early recognition and accurate characterization are therefore essential for appropriate management.

Magnetic resonance imaging (MRI) is the imaging modality of choice for the evaluation of TSGCT due to its excellent soft tissue contrast and its ability to demonstrate characteristic features related to the tumour's histopathological composition. Typical MRI findings include low-to-intermediate signal intensity on T1-weighted images, relatively low or heterogeneous signal intensity on T2-weighted sequences, and susceptibility-related blooming artefacts caused by intralesional hemosiderin deposition. Additional features such as synovial proliferation, joint effusion, and bone erosions may further support the diagnosis, particularly in diffuse disease.

KEY WORDS: Tenosynovial giant cell tumour, Pigmented villonodular synovitis, Synovial thickening, Magnetic resonance imaging, Soft tissue tumours

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This pictorial essay provides a comprehensive overview of the imaging spectrum of TSGCT with emphasis on MRI findings and their correlation with underlying pathology. The different subtypes according to the current World Health Organization classification are illustrated. In addition, the complementary role of other imaging modalities such as ultrasound, radiography, and computed tomography is discussed.

Introduction

Tenosynovial giant cell tumours (TSGCTs) represent a group of rare proliferative disorders arising from synovial tissue. They originate from the synovial lining of joints, tendon sheaths, and bursae and are characterised by synovial proliferation, inflammatory cell infiltration, and hemosiderin deposition. Although these tumours are histologically benign and do not metastasize, they can demonstrate locally aggressive behaviour, particularly in diffuse forms, resulting in cartilage damage, bone erosion, and progressive joint dysfunction [1–3].

Historically, different terms were used to describe these lesions. The intra-articular form was commonly referred to as pigmented villonodular synovitis (PVNS), while extra-articular lesions arising from tendon sheaths were known as giant cell tumours of the tendon sheath. These terms reflected differences in anatomical location rather than distinct pathological entities. As knowledge of their molecular and histopathological characteristics evolved, it became clear that these conditions represent variations within the same disease spectrum [2–3].

To resolve inconsistencies in terminology, the World Health Organization (WHO) classification of soft tissue tumours unified these lesions under the single entity of tenosynovial giant cell tumour [1]. According to the current WHO classification, TSGCTs are subdivided based on two parameters: growth pattern and anatomical location [1].

The growth pattern distinguishes localized from diffuse disease. Localized lesions typically present as well-circumscribed nodular masses, whereas diffuse lesions demonstrate infiltrative synovial proliferation involving large portions of the joint or surrounding tissues.

The anatomical classification distinguishes intra-articular lesions arising within the joint capsule from extra-articular lesions involving tendon sheaths or periarticular soft tissues [1].

Combining these two parameters results in four recognised subtypes [Fig 1.]:

- Localized intra-articular TSGCT
- Localized extra-articular TSGCT
- Diffuse intra-articular TSGCT
- Diffuse extra-articular TSGCT

This classification is clinically important because the biological behaviour, treatment strategy, and recurrence rates differ significantly between these subtypes [1].

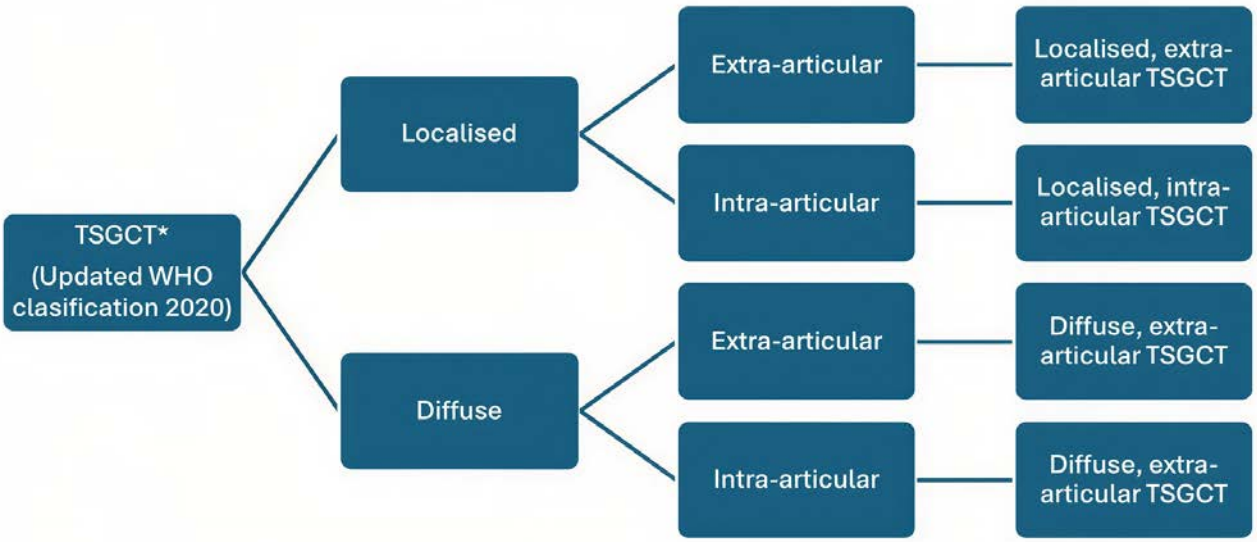

Fig. 1: *Classification of tenosynovial giant cell tumours. *TSGCT, tenosynovial giant cell tumours.*

TSGCTs most commonly affect young to middle-aged adults, with a peak incidence between 30 and 50 years of age. A slight female predominance has been reported in several studies [1, 5-6]. The disease is typically mono-articular, with the knee being the most frequently affected joint, followed by the ankle and hip. Localized lesions most commonly occur in the fingers and hand, arising from the flexor tendon sheaths [5–6].

Clinical presentation varies depending on subtype and anatomical location. Localized extra-articular lesions often present as slowly growing painless masses, particularly in the hand. In contrast, diffuse intra-articular disease typically presents with joint-related symptoms such as pain, swelling, stiffness, and recurrent effusions. Because these symptoms are non-specific and may develop gradually, diagnosis is often delayed [1].

At the molecular level, TSGCTs are driven by overexpression of colony-stimulating factor-1 (CSF1), usually caused by chromosomal translocations involving the CSF1 gene. Although only a small fraction of tumour cells harbours this genetic alteration, CSF1 overexpression leads to recruitment of large numbers of inflammatory cells expressing the CSF1 receptor. These recruited cells form the majority of the tumour mass and contribute to the characteristic histological appearance of the lesion [4]. Histologically, TSGCTs consist of a mixture of mononuclear histiocyte-like cells, osteoclast-like multinucleated giant cells, foam cells, fibroblasts, and varying degrees of fibrosis. A hallmark feature is the presence of hemosiderin deposition caused by repeated intralesional haemorrhage [1, 4]. These histopathological characteristics explain the typical imaging appearance of TSGCT, particularly the presence of low signal intensity and susceptibility artefacts on MRI.

Imaging plays a crucial role in the evaluation of suspected TSGCT. Conventional radiography and ultrasound may provide initial clues, but MRI remains the most important modality for diagnosis and staging. MRI allows accurate evaluation of lesion morphology, anatomical location, intra-articular extension, and bone involvement, all of which are essential for treatment planning [5]. The aim of this pictorial essay is to review the imaging spectrum of TSGCT with emphasis on MRI findings and correlation with underlying pathology. The different subtypes according to the WHO classification are illustrated using representative imaging examples, and the role of other imaging modalities is discussed.

MRI Characteristics of Tenosynovial Giant Cell Tumours

A. General findings

Magnetic resonance imaging is the most sensitive imaging modality for evaluating TSGCT and plays a central role in diagnosis, preoperative planning, and follow-up.

Across all subtypes, TSGCTs typically demonstrate low to intermediate signal intensity on T1-weighted images and variable but often relatively low signal intensity on T2-weighted sequences. This signal pattern reflects the combined effects of hemosiderin deposition, fibrous tissue, and cellular tumour components [5-7] [Fig. 2].

One of the most characteristic MRI findings in TSGCT is the presence of susceptibility-related blooming artefacts on gradient-echo or susceptibility-weighted sequences [Fig. 3]. These artefacts are caused by magnetic susceptibility effects of hemosiderin deposits within the lesion. Gradient-echo sequences are particularly sensitive to susceptibility effects and may demonstrate more extensive signal loss than conventional spin-echo sequences.

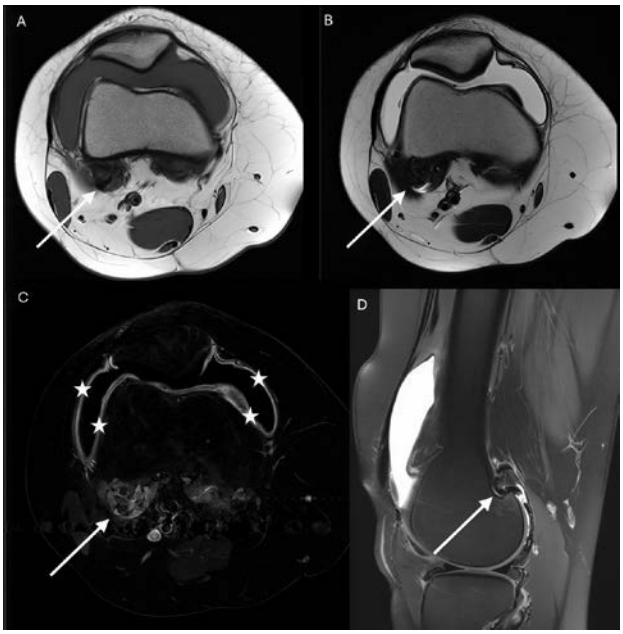

Fig. 2: *Axial and sagittal MR images of the knee demonstrate a diffuse intra-articular tenosynovial giant cell tumour with a solid nodular component posterior to the medial femoral condyle (arrows). The lesion shows low to heterogeneous signal intensity on T1- and T2-WI as on fat saturated imaging respectively (arrows, A, B and D). The solid nodular component shows heterogeneous contrast enhancement on T1-fs with gadolinium (arrow, C). There is also diffuse synovial thickening and enhancement (stars, C).*

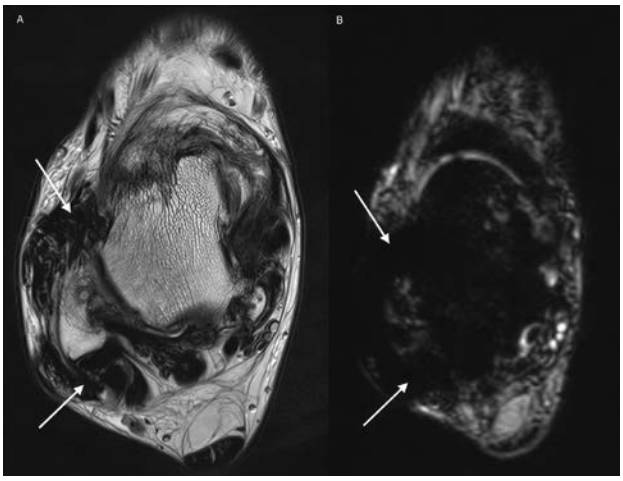

Fig. 3: *Axial MR images of the ankle demonstrate a diffuse intra-articular tenosynovial giant cell tumour with extensive synovial involvement. On T2-weighted turbo spin-echo image (arrows, A), the lesion is of heterogeneous low-to-intermediate signal. The gradient-echo sequence (arrows, B) reveals more extensive signal loss due to pronounced blooming artefacts. This illustrates the superior sensitivity of GRE imaging for detecting hemosiderin deposition in diffuse intra-articular TSGCT.*

In some cases, blooming artefacts may be visible even when intralesional low signal intensity is not obvious on standard T1- or T2-weighted images [5-7].

After administration of gadolinium-based contrast agents, TSGCTs typically demonstrate heterogeneous and often intense enhancement, reflecting the vascularised cellular stroma of the tumour [Fig. 4].

Additional MRI findings may include:

- Synovial thickening
- Joint effusion
- Bone erosions
- Cartilage damage in advanced disease

The presence and extent of these findings often correlate with the growth pattern of the tumour and are particularly prominent in diffuse disease.

B. Localized Intra-articular TSGCT.

Localized intra-articular TSGCT typically presents as a well-circumscribed nodular lesion arising from the synovium within a joint cavity. The knee is the most commonly affected joint, particularly the infrapatellar (Hoffa's) fat pad and the intercondylar region [Fig. 5].

On MRI, these lesions usually appear as nodular masses with intermediate signal intensity on T1-weighted images and low-to-intermediate signal intensity on T2-weighted sequences. Foci of low signal intensity corresponding to hemosiderin deposition are frequently present [5-7].

Blooming artefacts may be visible on susceptibility-sensitive sequences but are usually less extensive than in diffuse disease. Contrast-enhanced images typically demonstrate homogeneous or mildly heterogeneous enhancement. Joint effusion may occasionally be present but is usually limited. Bone erosions are uncommon in localized intra-articular lesions. Recognition of this imaging pattern is important because these lesions

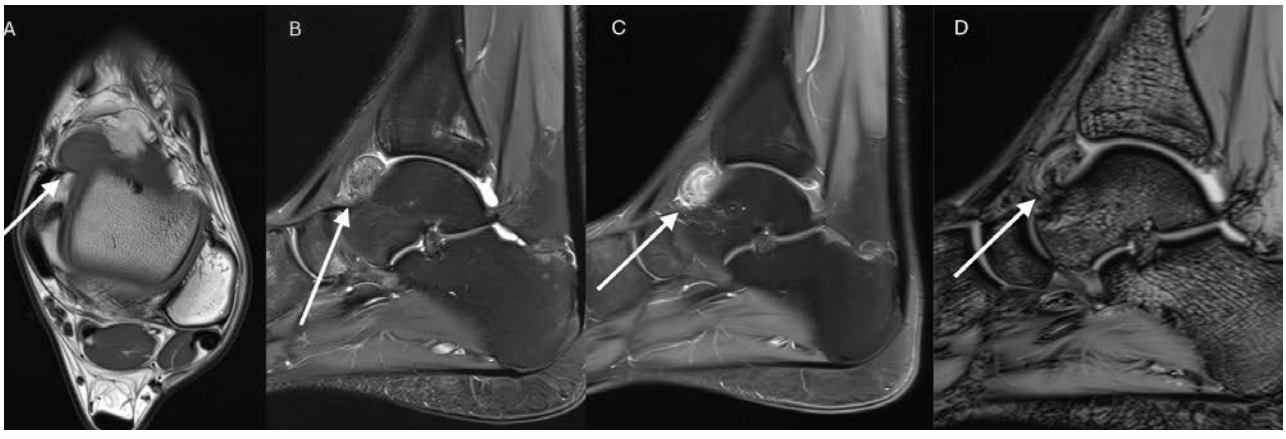

Fig. 4: *MRI of the ankle demonstrates a localized intra-articular TSGCT, presenting as a well-circumscribed nodular lesion along the anterior tibiotalar joint recess (arrows). The lesion shows low-to-intermediate signal intensity on non-contrast T1-weighted and intermediate-weighted images (arrows, A and B). On post-contrast T1-weighted imaging, it demonstrates marked, heterogeneous enhancement (arrow, C), representing the cellular stroma. These findings are typical for localized intra-articular TSGCT and aid in differentiation from cystic or non-enhancing synovial lesions. Gradient-echo imaging demonstrates clear blooming artefact (arrow, D), reflecting hemosiderin deposition and supporting the diagnosis of localized intra-articular TSGCT.*

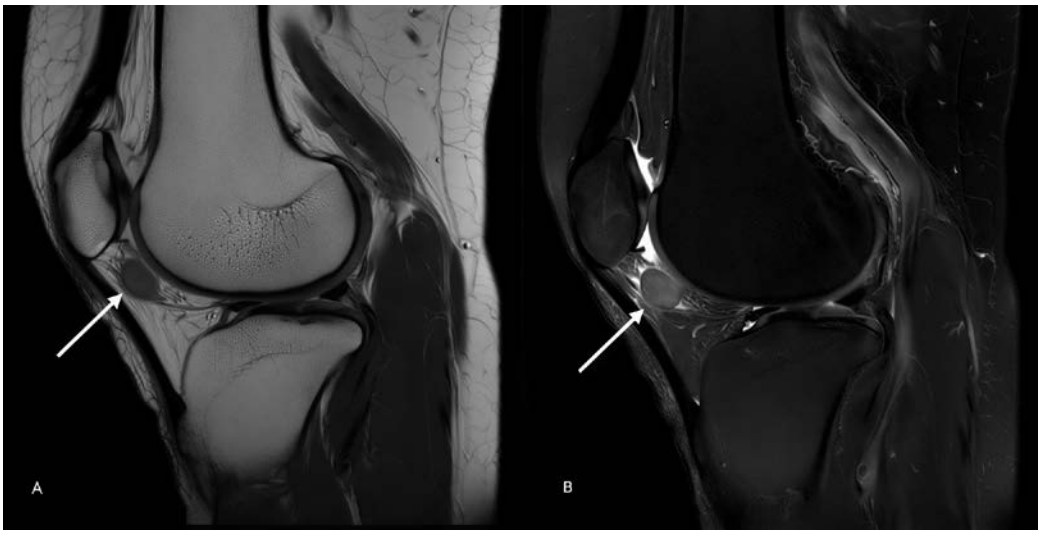

Fig. 5: *Sagittal MRI of the knee demonstrates a localized intra-articular TSGCT, appearing as a well-defined nodular lesion within the anterior joint compartment (arrows, A and B). The lesion shows low signal intensity on proton density (PD) WI (arrow, A) and low-to-intermediate signal on fluid-sensitive fat saturated sequences (arrow, B), in keeping with a solid synovial mass containing hemosiderin.*

are usually amenable to complete surgical excision and have relatively low recurrence rates [5, 7-8].

C. Localized Extra-articular TSGCT.

Localized extra-articular TSGCT corresponds to the classic giant cell tumour of the tendon sheath. These lesions most frequently occur in the fingers and hand, particularly along the flexor tendon sheaths [Fig. 6-7]. MRI typically demonstrates a well-defined soft-tissue mass adjacent to a tendon sheath. Signal characteristics resemble those of localized intra-articular lesions, with intermediate T1 signal intensity and low-to-intermediate T2 signal intensity [5-7].

Intralesional low-signal foci due to hemosiderin deposition are common and may produce prominent blooming artefacts on gradient-echo sequences.

Contrast enhancement is usually moderate to strong and may be homogeneous or heterogeneous.

Although these lesions are usually well circumscribed, chronic lesions may cause pressure erosions of adjacent bone [1,5, 7].

D. Diffuse Intra-articular TSGCT

Diffuse intra-articular TSGCT represents the most aggressive form of the disease and corresponds to what was historically known as pigmented villonodular synovitis. This subtype is characterised by extensive synovial proliferation affecting large portions of the joint cavity. The knee is by far the most frequently affected joint [Fig. 8], followed by the ankle and hip [Fig. 9]. MRI typically demonstrates diffuse synovial thickening with villous, nodular, or frond-like morphology. Signal intensity is often low on both T1- and T2-weighted se-

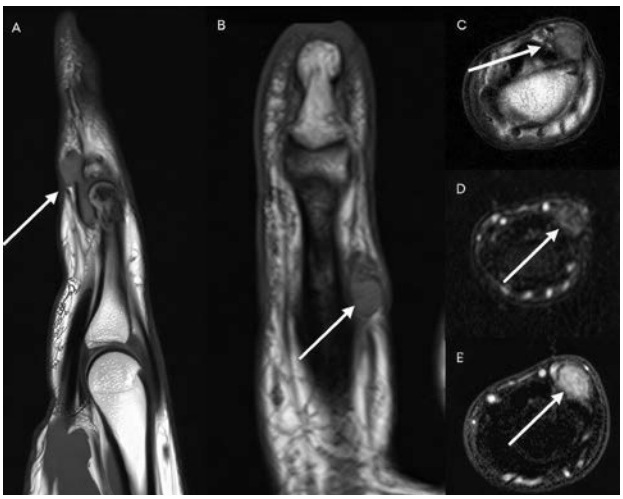

Fig. 6: *MRI of the finger demonstrates a localized extra-articular TSGCT, arising along the flexor tendon sheath (arrows, A-E). The lesion appears as a well-circumscribed soft-tissue mass with low-to-intermediate signal intensity on T1-WI (arrows, A-C) and on T2-WI fs (arrow, D). The lesion shows heterogeneous enhancement on T1-WI after contrast administration (arrow, E). These findings are typical for localized extra-articular TSGCT and reflect hemosiderin deposition and contrast enhancing cellular stroma within a solid tendon sheath lesion.*

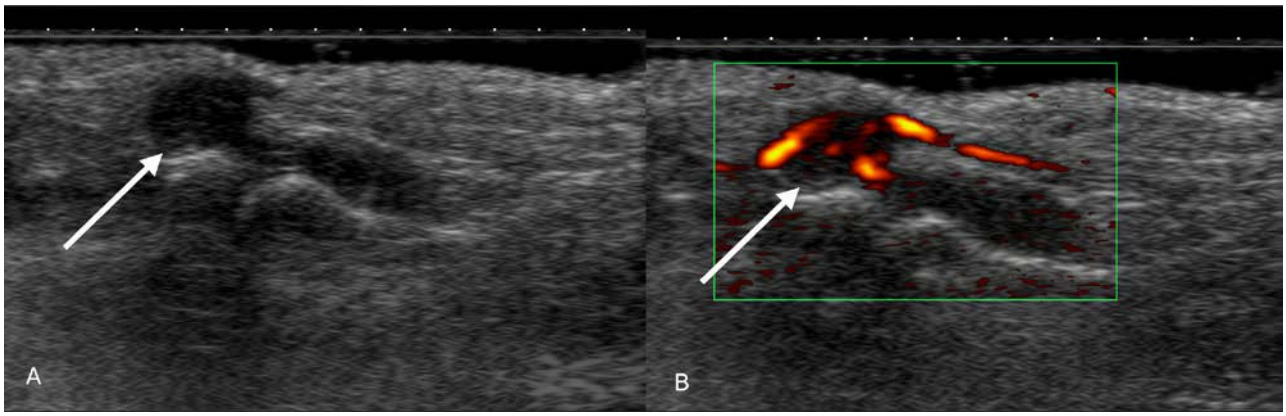

Fig. 7: *Ultrasound of the same lesion in the left index finger demonstrates localized extra-articular TSGCT adjacent to the flexor digitorum tendon sheath at the level of the proximal interphalangeal joint (arrows, A and B). The lesion appears as a solid hypoechoic nodular mass (arrow, A) with marked intralesional vascularity on colour Doppler imaging (arrow, B). These sonographic features are suspicious for localized TSGCT and explain the recommendation for complementary MRI for further characterization and assessment of intra-articular extension.*

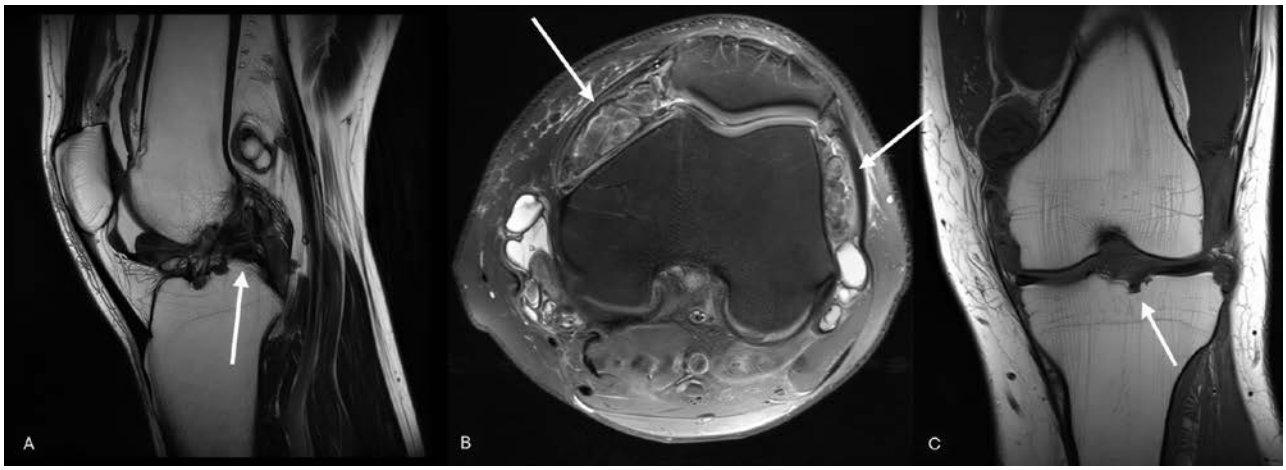

Fig. 8: *MRI of the knee demonstrates diffuse intra-articular TSGCT with extensive synovial proliferation (arrows, A-B). The lesion shows diffuse, ill-defined synovial thickening on PD-fs (A) and solid components of low-to-intermediate signal intensity on PD (B) non–contrast-weighted sequences, associated with marked synovial thickening (arrows, A-B) and adjacent bone erosions on T1-WI (arrow, C).*

quences due to abundant hemosiderin and fibrous tissue [5-7].

Blooming artefacts on susceptibility-sensitive sequences are typically extensive and represent one of the most characteristic imaging findings.

After contrast administration, diffuse intra-articular TSGCT demonstrates marked heterogeneous enhancement of the proliferative synovium.

Joint effusion is common and may be substantial. In advanced disease, bone erosions and cartilage damage may occur due to chronic pressure and inflammatory activity [5-8].

E. Diffuse Extra-articular TSGCT

Diffuse extra-articular TSGCT involves infiltrative proliferation along tendon sheaths or periarticular soft tissues and may extend beyond the joint capsule [Fig. 10].

MRI findings are similar to those of diffuse intra-articular disease. Lesions typically demonstrate low signal intensity on both T1- and T2-weighted images with prominent blooming artefacts [5-7].

The lesions often appear multilobulated and infiltrative, with heterogeneous enhancement and involvement of adjacent soft tissues.

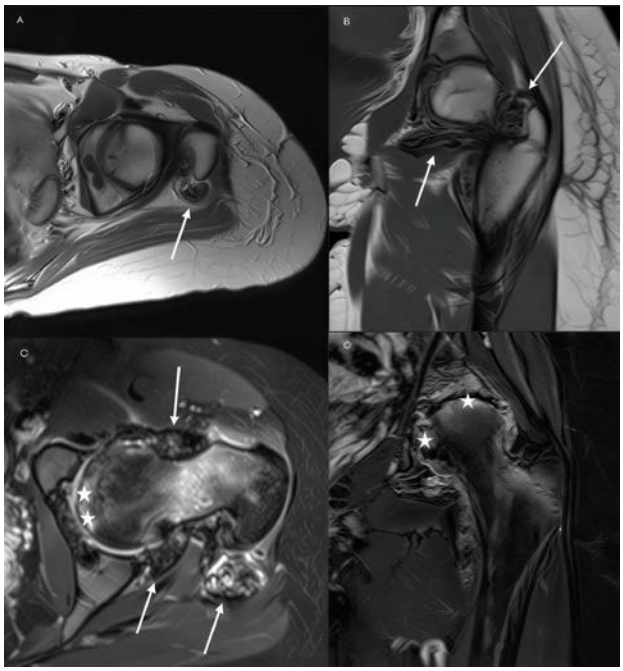

Fig. 9: *MRI of the hip demonstrates diffuse intra-articular TSGCT with extensive synovial proliferation and solid components (arrows, A-C). The lesion shows diffuse synovial thickening with low-to-intermediate signal intensity on T1-WI (A, B) and heterogeneous enhancement of the solid components and the synovium on T1-WI after gadolinium administration (arrows, C and D). Adjacent osseous erosions and surrounding bone marrow (stars, C-D), reflecting the locally aggressive and infiltrative behaviour of diffuse intra-articular TSGCT.*

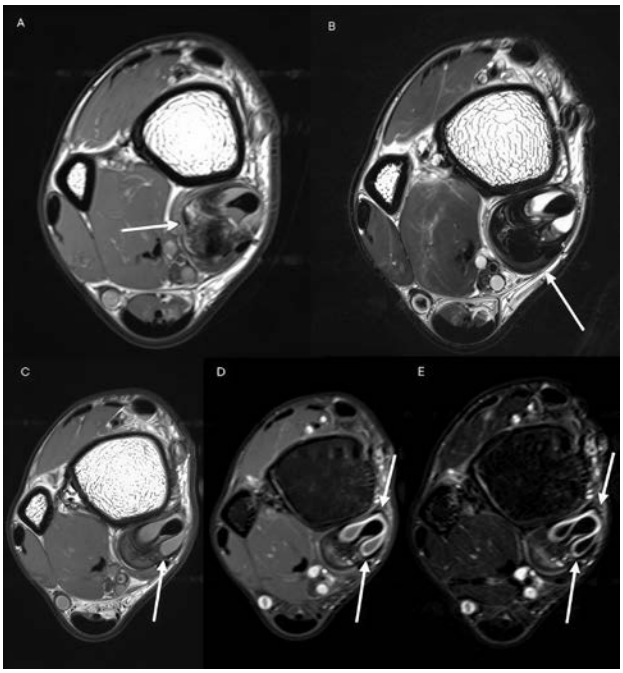

Fig. 10: *MRI of the ankle demonstrates a diffuse extra-articular TSGCT involving the tendon sheath of the tibialis posterior tendon. The lesion shows irregular delineated soft-tissue proliferation of low-to-intermediate signal on T1- and T2-WI (arrows, A and B). There is also heterogeneous enhancement of the tendon sheath and surrounding fatty tissue (arrows) on T1-WI (C) with gadolinium contrast (D) and subtraction images (E).*

Because of their infiltrative appearance, diffuse extra-articular lesions may mimic malignant soft-tissue tumours. Recognition of hemosiderin-related susceptibility artefacts is therefore crucial for suggesting the correct diagnosis [5-8].

Other Imaging Modalities

A. Ultrasound

Ultrasound is frequently used as the initial imaging modality for evaluating superficial soft-tissue masses, particularly in the hand and wrist [Fig. 11].

On ultrasound, TSGCT typically appears as a solid hypoechoic nodular mass adjacent to a tendon sheath. The lesion may have a lobulated contour and is usually well defined in localized disease.

Colour Doppler imaging commonly demonstrates internal vascularity, reflecting the hypervascular synovial proliferation characteristic of these tumours [9].

Diffuse disease may appear as synovial thickening with irregular margins, sometimes associated with joint effusion [Fig. 12-13].

Ultrasound is particularly useful for:

- assessing the relationship between the lesion and adjacent tendons
- detecting small superficial lesions
- guiding percutaneous biopsy

However, ultrasound has limitations in evaluating deeper intra-articular lesions and in assessing the full extent of diffuse disease [6, 9].

B. Conventional Radiography

Radiography is often the first imaging modality obtained in patients with joint-related symptoms. Although radiographic findings in TSGCT are frequently non-specific, they may provide important diagnostic clues.

The most common radiographic finding is periarticular soft-tissue swelling. In diffuse intra-articular dis-

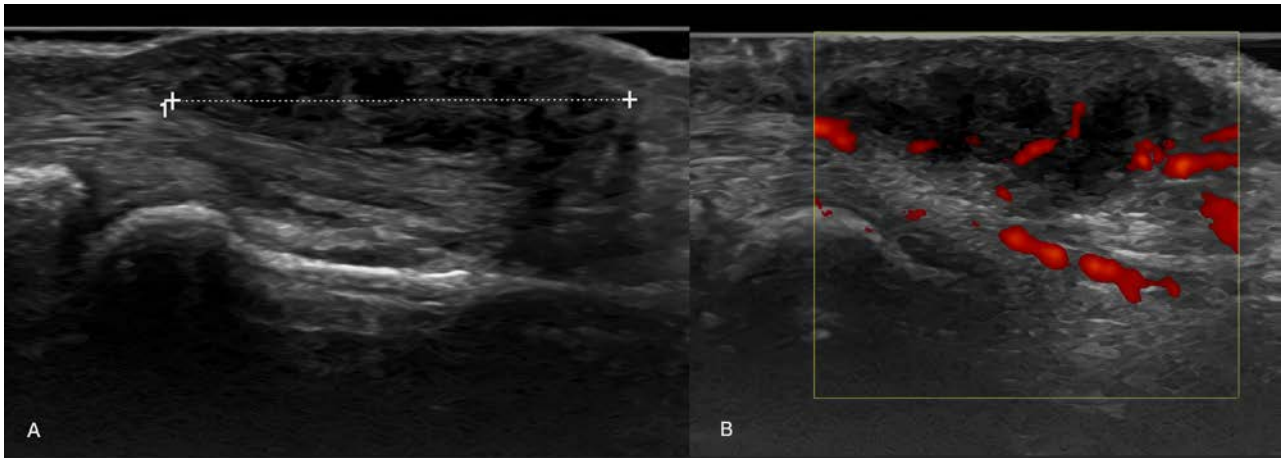

Fig. 11: *Ultrasound of the finger at the level of the proximal interphalangeal joint demonstrates a localized extra-articular TSGCT. The lesion appears as a solid hypoechoic nodular mass adjacent to the tendon sheath (A) with intralesional vascularity on colour Doppler (B).*

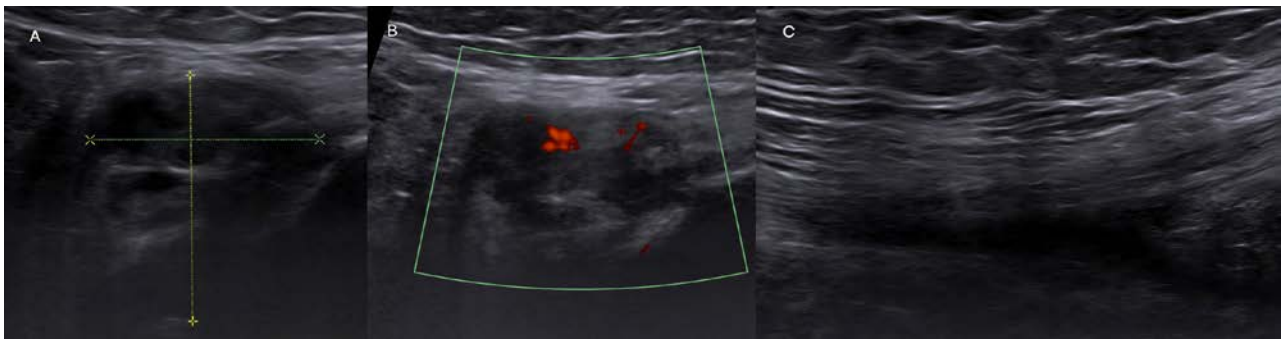

Fig. 12: *Ultrasound of the knee demonstrates a diffuse intra-articular TSGCT with a solid synovial component (A). The lesion appears as an ill-defined hypoechoic mass with intralesional vascularity on colour Doppler imaging (B), associated with joint effusion (star, C).*

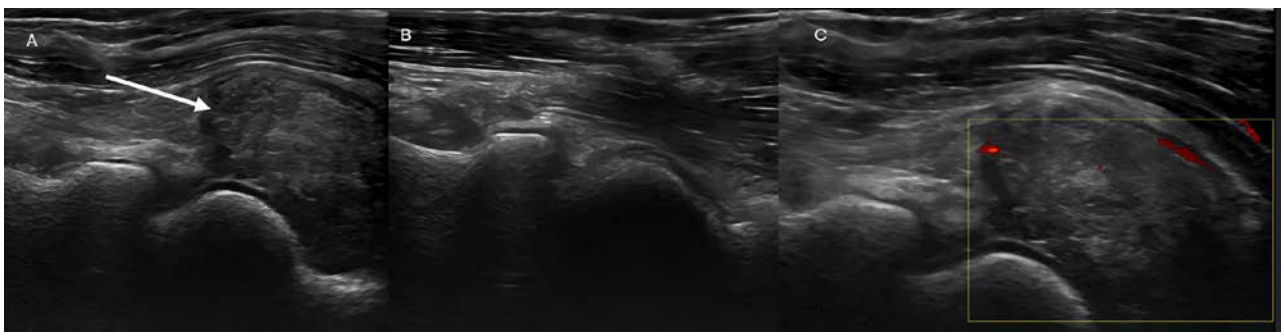

Fig. 13: *Ultrasound of the elbow demonstrates localized intra-articular TSGCT in the anterior fat pad of the elbow (arrow). The lesion appears as a well-defined hypoechoic nodular synovial mass (A) with intralesional vascularity on colour Doppler (C). There is no significant intra-articular fluid.*

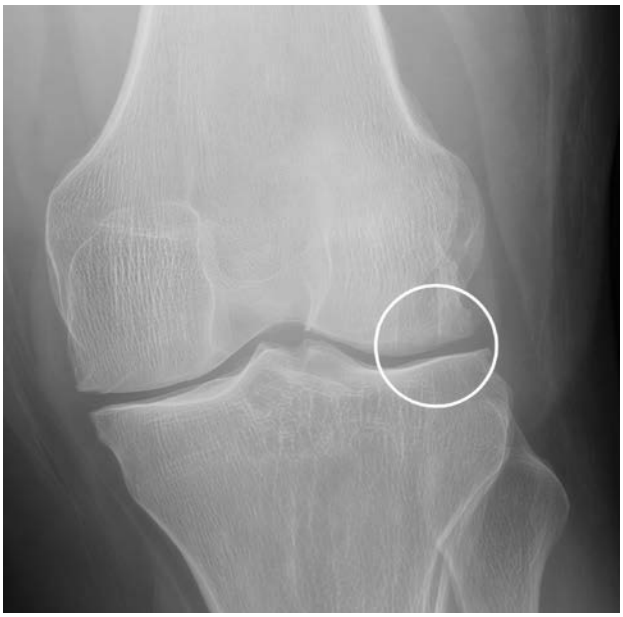

Fig. 14: *Anteroposterior radiograph of the knee demonstrates cortical erosions along the lateral femoral condyle (circle, A). T1-WI shows bone erosions with hemosiderin depositions along the lateral femoral condyle (arrows, B). Although non-specific, such pressure erosions may be seen in diffuse intra-articular TSGCT (as was seen in this case) and reflect chronic synovial proliferation with adjacent bone involvement.*

ease, chronic synovial proliferation may lead to pressure erosions of adjacent bone, particularly in joints with relatively tight capsules [Fig 14].

Importantly, calcifications are typically absent, which may help differentiate TSGCT from other synovial lesions such as synovial chondromatosis.

In advanced cases, radiographs may demonstrate:

- cortical bone erosions
- subchondral cystic changes
- secondary degenerative joint changes

Radiography therefore remains valuable as an initial screening tool and for evaluating osseous changes [6, 8].

C. Computed Tomography

Computed tomography plays a limited but potential complementary role in the evaluation of TSGCT.

On CT, the lesion typically appears as a soft-tissue mass that may demonstrate slightly increased attenuation relative to muscle due to haemorrhage or hemosiderin deposition [Fig. 15].

CT is particularly useful for:

- assessing cortical bone erosions
- evaluating complex anatomical regions
- preoperative planning in selected cases

In diffuse disease, CT may demonstrate irregular synovial thickening and bone erosions [6].

D. PET/CT

FDG PET-CT may demonstrate increased metabolic activity in TSGCT. This uptake reflects the inflammatory cellular composition of the lesion rather than malignant behaviour.

Because FDG uptake is non-specific, PET/CT has a limited role in diagnosis but may be useful in selected situations such as evaluation of disease extent or detection of recurrent disease [6].

E. Key Point

MRI remains central to diagnosis and staging, while ultrasound and radiography provide valuable adjunctive information.

CT and PET/CT have a limited, problem-solving role in selected cases.

Differential Diagnosis

The imaging appearance of tenosynovial giant cell tumour may overlap with several benign and malignant conditions involving the synovium, tendon sheaths, or periarticular soft tissues. The differential diagnosis therefore depends largely on the lesion's anatomical

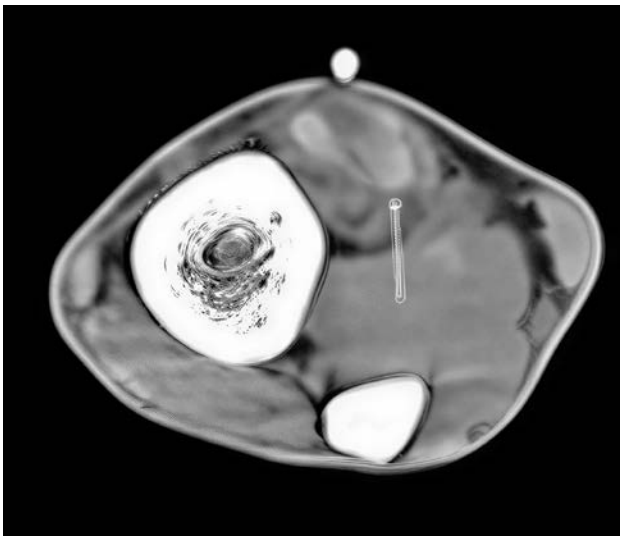

Fig. 15: *Axial CT image demonstrates an extra-articular TSGCT presenting as a soft-tissue mass with areas of increased attenuation (arrow), in keeping with blood degradation products.*

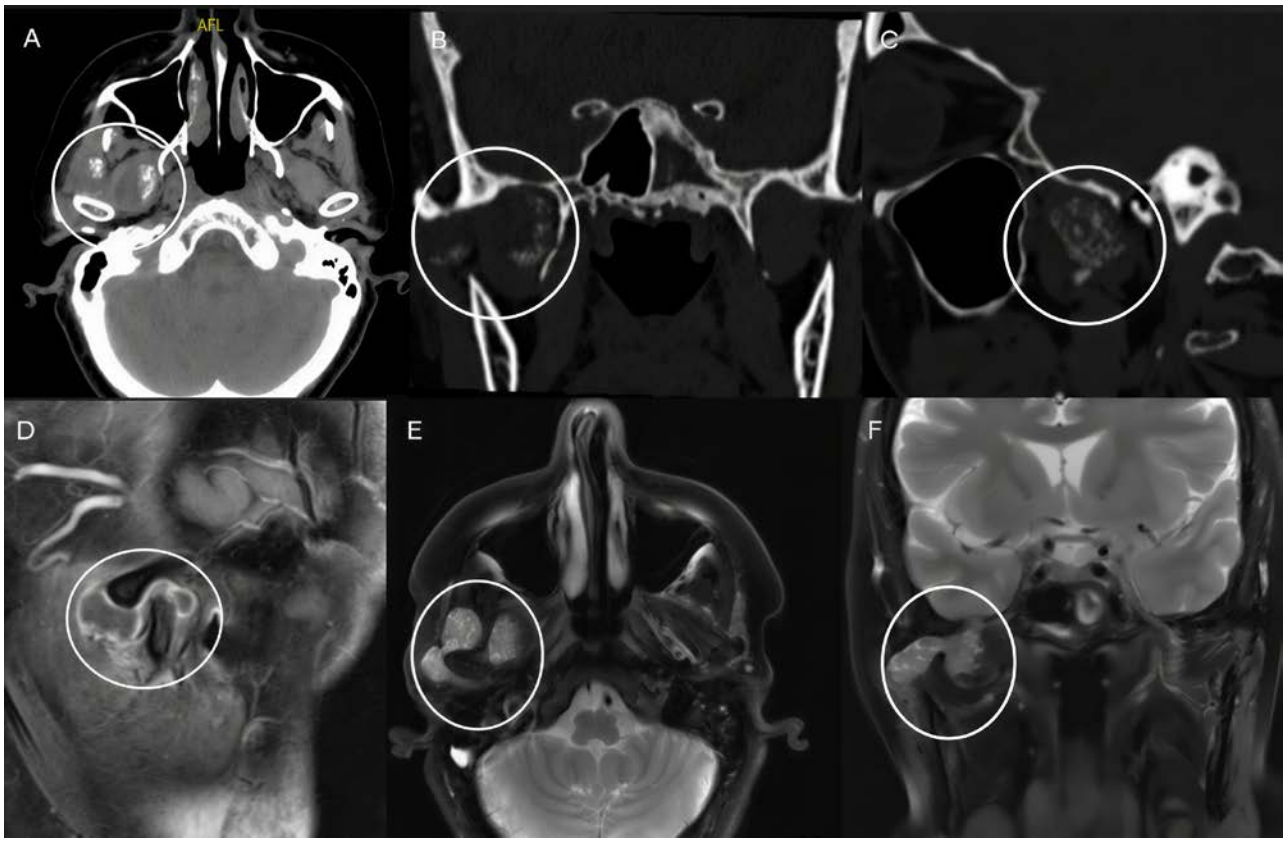

Fig. 16: *Axial CT (soft tissue window) shows multiple soft tissue calcifications (A, red circle) at the right TMJ. Coronal and sagittal CT (bone window) show multiple calcified loose bodies of uniform size and widening of the right TMJ (B-C, red circles). Sagittal post contrast T1-WI of the right TMJ show peripheral wall enhancement representing slightly thickened synovium (D, red circle). Axial and coronal T2-WI with fat suppression show multiple loose bodies in the joint, most of which are of low signal intensity (E-F, red circles).*

Origin: Vanhoenacker F.M., Department of Radiology, AZ Sint-Maarten, Mechelen-Duffel, Belgium

location, growth pattern, and imaging characteristics [10]. In many cases, recognition of the combination of relatively low signal intensity on fluid-sensitive MRI sequences and the presence of susceptibility-related blooming artefacts strongly suggests TSGCT. Nevertheless, several entities may present with partially overlapping features and should be considered during image interpretation [10].

A. Primary Synovial Chondromatosis

Primary synovial chondromatosis [Fig. 16] is a benign proliferative disorder of the synovium, characterized by cartilaginous metaplasia with formation of multiple intra-articular nodules. These nodules may detach over time, resulting in numerous loose bodies within the joint space. The process is typically mono-articular and most frequently involves large joints such as the knee and hip [10-11].

On MRI, the imaging appearance depends on the degree of mineralization. In early stages, non-calcified cartilaginous nodules demonstrate intermediate signal intensity on T1-weighted images and high signal intensity on T2-weighted sequences, reflecting the high-water content of hyaline cartilage. As the disease progresses, nodules may undergo calcification, resulting in areas of low signal intensity on both T1- and T2-weighted images. Ossified nodules may be of high signal on T1- weighted images. A characteristic feature is the presence of multiple similar-sized nodules, often with a lobulated or clustered configuration. After contrast administration, there is typically peripheral or septal enhancement corresponding to synovial proliferation, rather than diffuse solid enhancement [10-11].

Radiography and CT play an important complementary role, as they frequently demonstrate multiple calcified intra-articular bodies with typical “ring-and-arc”

chondroid mineralization. This feature is highly suggestive of a cartilaginous origin and represents a key discriminator from other synovial pathologies. In primary synovial chondromatosis, the nodules are typically multiple and of relatively uniform size, with preservation or even enlargement of the joint space, helping to differentiate it from secondary synovial chondromatosis, where the intra-articular bodies are usually more variable in size and associated with underlying degenerative joint disease. The distinction from TSGCT is usually straightforward when mineralization is present. In contrast to synovial chondromatosis, TSGCT typically lacks calcifications on radiographs and CT.

On MRI, TSGCT demonstrates predominantly low to intermediate signal intensity on both T1- and T2-weighted sequences due to hemosiderin deposition, often accompanied by blooming artefacts on susceptibility-sensitive sequences, which are typically absent in synovial chondromatosis. Furthermore, TSGCT tends to present as a solid synovial mass or diffuse infiltrative synovial thickening, rather than multiple discrete cartilaginous nodules [11-12].

In summary, the presence of multiple nodules with high T2 signal and chondroid mineralization strongly favours synovial chondromatosis, whereas low-signal synovial proliferation with susceptibility artifacts is characteristic of TSGCT.

B. Tophaceous Gout

Tophaceous gout [Fig. 17] may occasionally mimic TSGCT, particularly when periarticular soft-tissue masses are present. Gout results from deposition of monosodium urate crystals in soft tissues and joints and may present with nodular masses known as tophi [10]. On MRI, gouty tophi may demonstrate heterogeneous signal intensity, often with intermediate signal on T1-weighted images and variable signal on T2-weighted sequences. In some cases, low signal intensity on both pulse sequences may be observed due to crystal deposition or associated fibrosis, potentially creating confusion with TSGCT [13]. However, several imaging features may help distinguish gout from TSGCT. Radiographs frequently demonstrate characteristic bone erosions with overhanging edges, which are typical for gouty arthropathy. In addition, soft-tissue calcifications or amorphous mineralisation may be present within tophi [13]. Dual-energy CT has emerged as a particularly useful modality in this context, as it allows direct identification of urate crystal deposits, thereby confirming the diagnosis of gout [13].

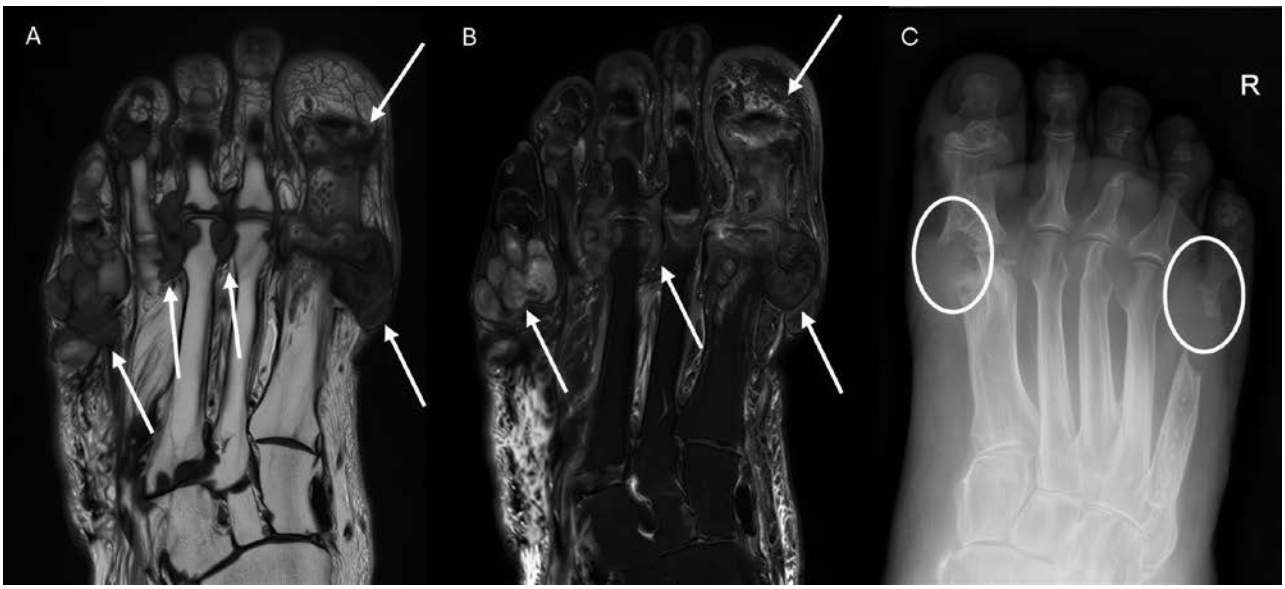

Fig. 17: *Coronal T1-weighted MRI (A) and Dixon water-only sequence (B) demonstrate extensive, lobulated periarticular soft-tissue deposits (arrows) involving multiple joints of the forefoot, with predominantly intermediate to low T1 signal and heterogeneous signal on fluid-sensitive imaging. The lesions show a typical periarticular distribution with associated soft-tissue swelling and edema. Conventional radiography (C) reveals characteristic punched-out erosions (circle) with overhanging edges. In contrast to TSGCT, these lesions do not demonstrate susceptibility-related blooming, while the presence of multifocal erosions and typical radiographic findings strongly support the diagnosis of gout.*

Clinical history also plays a key role in differentiation. Patients with gout often have a history of hyperuricaemia, recurrent episodes of acute arthritis, or involvement of typical sites such as the first metatarsophalangeal joint.

C. Vascular malformation

Vascular malformations [Fig. 18] are a rare benign vascular lesion that most commonly occurs in children and young adults and frequently involves the knee joint. Because these malformations arise from abnormal vascular channels in or around the joint and may present with joint swelling and effusion, they can mimic diffuse intra-articular TSGCT. [10].

MRI findings in vascular malformations, which are most commonly venous malformations and previously known as synovial haemangiomas, typically include markedly hyperintense signal on T2-weighted sequences, reflecting a high water content of vascular channels. Serpiginous vascular structures may be present, while true flow voids are typically absent given the low-flow nature of the lesion [14]. After contrast administration, vascular malformations usually demonstrate strong and homogeneous enhancement. In addition, phleboliths may be present within the lesion and appear as small, rounded calcifications on radiographs or CT. In contrast to TSGCT, vascular malformations usually demonstrate very high T2 signal intensity, and susceptibility artefacts related to hemosiderin deposition are typically absent [14].

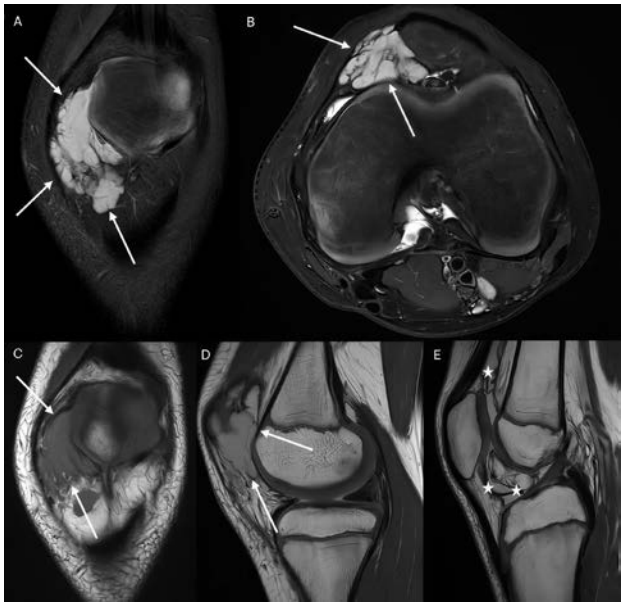

Fig. 18: *Proton density fat-saturated (PD-FS) images demonstrate a lobulated intra-articular mass with markedly high signal intensity, reflecting its vascular / fluid-rich composition (arrows, A-B). On the T1-weighted image, the lesion appears predominantly iso- to hypointense relative to muscle, with subtle intralesional heterogeneity (arrows, C). Proton density (PD) images without fat suppression show hyperintense signal compared to muscle (arrows, D), with better delineation of internal septations. Low signal along the synovial lining on PD images (stars, E) correspond to hemosiderin deposition, related to recurrent intralesional haemorrhage. In contrast to TSGCT, the lesion is predominantly hyperintense on fluid-sensitive sequences and lacks diffuse low signal with pronounced susceptibility effects, supporting the diagnosis of synovial haemangioma.*

D. Rheumatoid Synovitis and Other Inflammatory Arthritides

Inflammatory arthritides [Fig. 19] such as rheumatoid arthritis may produce diffuse synovial thickening, joint effusion, and contrast enhancement, potentially resembling diffuse intra-articular TSGCT [10]. However, several imaging features may help differentiate inflammatory synovitis from TSGCT. Inflammatory synovitis generally demonstrates higher signal intensity on T2-weighted sequences because the inflamed synovium contains abundant fluid and inflammatory tissue. In contrast, the synovial proliferation in TSGCT frequently demonstrates relatively low T2 signal intensity due to hemosiderin deposition [15]. In addition, inflammatory arthritides are usually polyarticular diseases, whereas TSGCT typically affects a single joint. Associated imaging findings such as marginal erosions, periarticular osteopenia, and cartilage loss may also suggest inflammatory arthritis rather than TSGCT [15].

E. Haemophilic Arthropathy

Haemophilic arthropathy [Fig. 20] represents another condition that may mimic TSGCT on imaging. Recurrent hemarthrosis in patients with haemophilia leads to synovial hypertrophy and deposition of hemosiderin, which may produce MRI findings similar to those seen in diffuse TSGCT [10]. MRI may demonstrate diffuse synovial thickening with low signal on both T1- and T2-weighted sequences, as well as susceptibility artefacts due to hemosiderin deposition. Clinical history is usually decisive in distinguishing haemophilic arthropathy from TSGCT. Patients with haemophilia typically present with a well-established bleeding disorder and often multiple joints are involved [16].

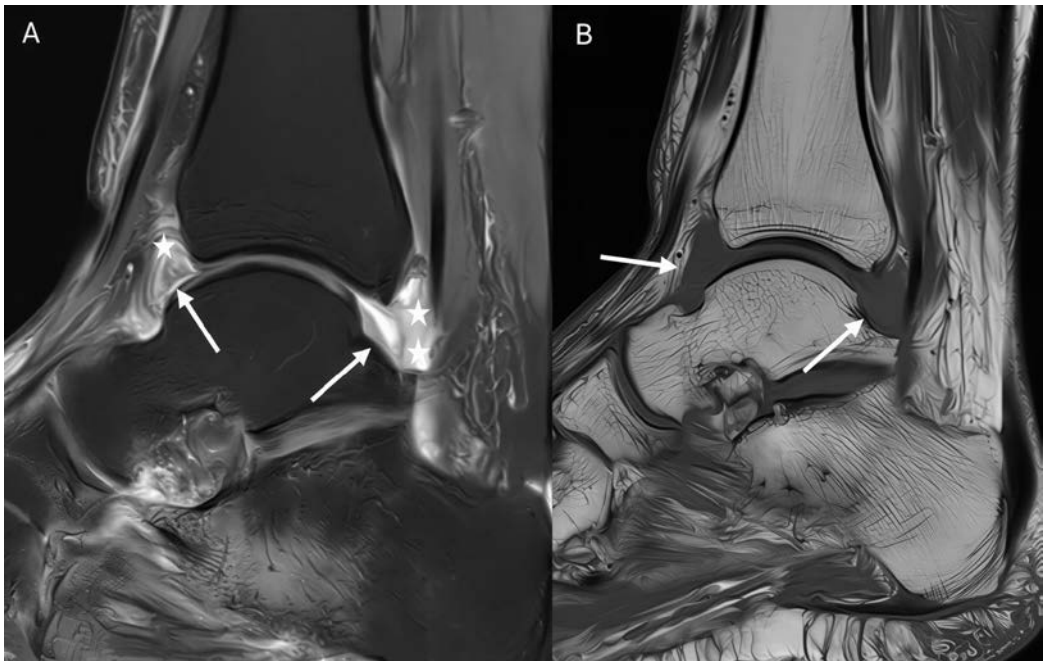

Fig. 19: *Sagittal PD-weighted fat-saturated image (A) demonstrates joint effusion (arrows) and synovial thickening (stars) with high signal. The corresponding T1-weighted image (B) shows low to intermediate signal synovial proliferation (arrows). In contrast to TSGCT, the synovial proliferation lacks diffuse low signal intensity and susceptibility-related blooming, while the prominent fluid signal and surrounding edema favour inflammatory arthritis.*

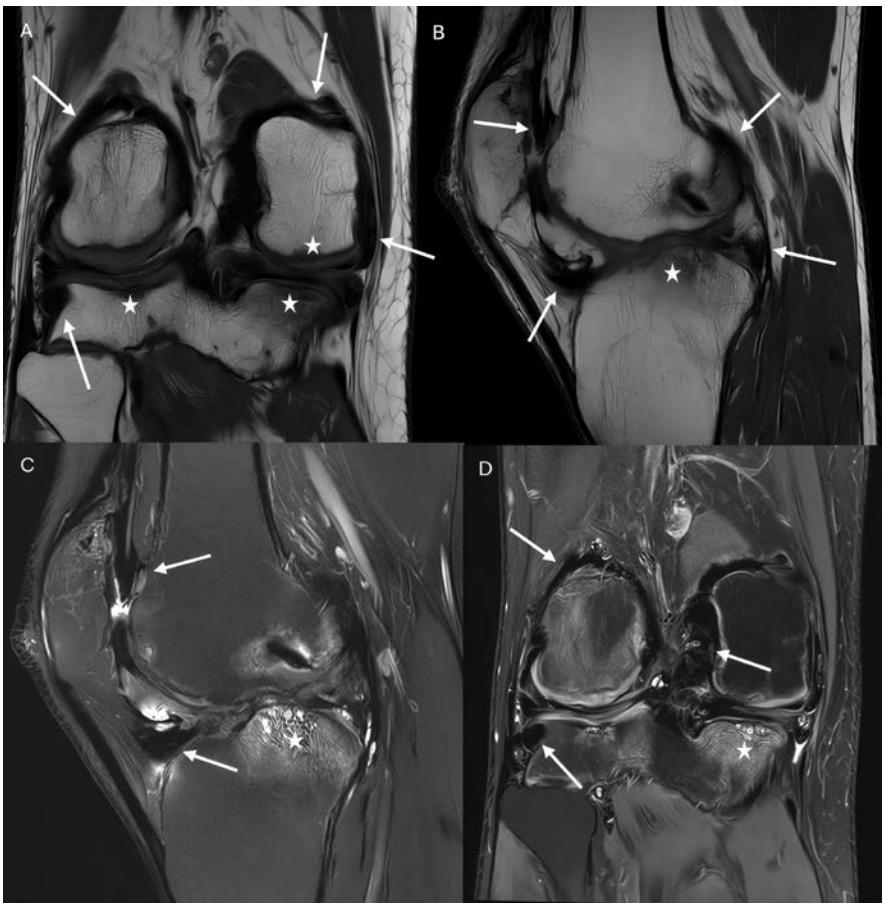

Fig. 20: *Coronal T1-weighted (A) and sagittal PD images (B) demonstrate diffuse synovial thickening with predominantly low signal intensity (arrows), associated with irregular articular surfaces and subchondral changes (stars). PD-FS (C) and T2-weighted FS images (D) show hypointense synovial hypertrophy (arrows), and bone marrow oedema (stars). Marked low-signal synovial deposition on all sequences reflects extensive hemosiderin accumulation due to recurrent hemarthroses. Although the low signal and hemosiderin deposition may mimic TSGCT, the diffuse joint involvement, advanced degenerative changes, and the known clinical history of haemophilia strongly support haemophilic arthropathy.*

F. Synovial Sarcoma

Although relatively rare, synovial sarcoma [Fig. 21] must be considered in the differential diagnosis of periarticular soft tissue masses, because the management of this malignant lesion differs substantially from that of benign synovial lesions [10]. Despite its name, synovial sarcoma is a misnomer, as the tumour does not originate from synovial tissue [17].

Synovial sarcoma typically affects young adults and may arise near joints, often in the lower extremities. On MRI, synovial sarcomas often demonstrate heterogeneous signal intensity on T2-weighted sequences, sometimes producing the so-called "triple sign," which reflects a mixture of high, intermediate, and low signal intensity areas corresponding to cystic, cellular, and fibrous components on histopathology [18].

Unlike TSGCT, synovial sarcomas frequently demonstrate markedly hyperintense areas on T2-weighted sequences, reflecting necrosis or cystic change. In addition, calcifications may be present in approximately 30% of cases and may be visible on radiographs or CT [19]. The presence of a rapidly growing soft-tissue mass, large lesion size, and extensive surrounding oedema should raise suspicion for malignancy [18-19].

G. Amyloid arthropathy

Amyloid arthropathy may mimic a TSGCT on MRI because amyloid deposits typically demonstrate low signal intensity on both T1- and T2-weighted images. However, blooming artefact is usually less pronounced than in TSGCT due to the absence of significant hemosiderin deposition. Associated multifocal subchondral cystic lesions and the clinical context of chronic dialysis or systemic amyloidosis may further support the diagnosis of amyloid arthropathy [20].

H. Practical Diagnostic Approach

When evaluating a suspected synovial lesion, a systematic imaging approach can help narrow the differential diagnosis.

Key imaging features that strongly favour TSGCT include:

- Low or intermediate signal intensity on T1-weighted images
- Relatively low signal intensity on T2-weighted images
- Susceptibility-related blooming artifacts due to hemosiderin deposition
- Synovial-based lesion with nodular or villous morphology
- Absence of calcifications

When these features are present in combination, particularly around the knee or tendon sheaths of the hand, the diagnosis of TSGCT can often be suggested with high confidence.

Conclusion

Tenosynovial giant cell tumours represent an uncommon but important group of synovial lesions that radiologists should be familiar with. Although histologically benign, these tumours may behave in a locally aggressive manner and can lead to progressive joint damage, functional impairment, and a significant risk of recurrence, particularly in diffuse forms of the disease. Early recognition therefore plays a key role in guiding

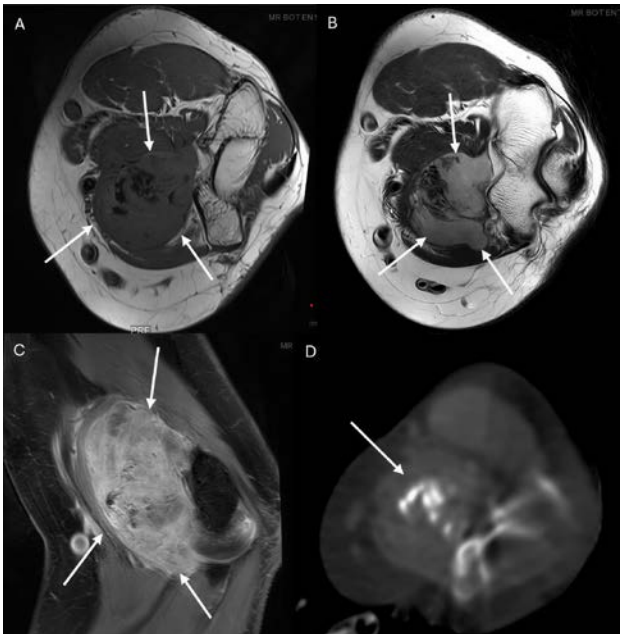

Fig. 21: *Axial T1-weighted image (A) demonstrates a heterogeneous soft-tissue mass with predominantly intermediate signal intensity relative to muscle (arrows). On the T2-weighted image (B), the lesion appears markedly heterogeneous with predominantly high to intermediate signal intensity and intralesional areas of lower signal. Post-contrast Dixon T1-weighted image (C) shows heterogeneous and predominantly solid enhancement with irregular margins. Axial CT image demonstrates internal calcifications within the soft-tissue mass (D, arrows). In contrast to TSGCT, the lesion demonstrates predominantly high T2 signal, lacks a clear synovial-based growth pattern, and contains internal calcifications on CT, a feature highly atypical for TSGCT and more suggestive of synovial sarcoma.*

appropriate clinical management and surgical planning. Magnetic resonance imaging remains the cornerstone modality for the evaluation of TSGCT. The typical MRI appearance reflects the underlying histopathology of the lesion, particularly the presence of hemosiderin deposition within the tumour. The combination of relatively low signal intensity on fluid-sensitive sequences together with susceptibility-related blooming artefacts on gradient-echo or susceptibility-weighted sequences represents a characteristic imaging pattern that should immediately raise suspicion for TSGCT.

In addition to lesion detection, MRI provides crucial information regarding tumour extent, involvement of adjacent structures, and the distinction between localized and diffuse disease. This distinction is clinically relevant because diffuse forms are associated with more extensive synovial involvement and higher recurrence rates, often requiring more complex treatment strategies. Other imaging modalities, including ultrasound, conventional radiography, and computed tomography, may provide complementary information in selected cases. However, familiarity with the characteristic MRI features remains essential for confident diagnosis.

Recognising the typical imaging spectrum of TSGCT and distinguishing it from other synovial and periarticular lesions allows radiologists to play a central role in establishing the diagnosis and facilitating optimal patient care. **R**

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Ethical approval

Ethical approval was not required for this study because only anonymized institutional imaging data were used for educational purposes.

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