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Case Report

Giant Cell Tumour of Tendon Sheath (GCTTS) At Ankle Joint - Case Report with Radiological Review

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ABSTRACT

Giant cell tumour of tendon sheath (GCTTS) is a benign proliferative non tumoural lesion that may affect the joints, bursae and tendon sheaths. It is known to be of synovial origin and viewed as an extra articular form of pigmented villonodular synovitis (PVNS). It affects most commonly the hand with rare involvement of foot and ankle joint. Haemosiderin deposits showing blooming on GRE sequence of MRI is characteristic. We report a case of 22 year old male diagnosed with giant cell tumour of tendon sheath of tibialis posterior, flexor digitorum longus and flexor hallucis longus tendons of ankle joint on high resolution ultrasonography and MRI.

Keywords: Giant cell tumour of tendon sheath (GCTTS), Pigmented villonodular synovitis (PVNS), MRI, Osteoclast giant cells, Ultrasonography (USG)

INTRODUCTION

Giant cell tumour of tendon sheath (GCT-TS) is a benign proliferative soft tissue lesion arising from the complex of tendon sheaths and periarticular soft tissue of small joints. It is distinguished from pigmented villonodular synovitis (PVNS) which arises from the synovial membrane of the joint. ^[1] It most commonly affects the tendon sheaths of volar aspect of index and middle finger of hand with peak age of incidence in third to fifth decades of life.^[2] It is less common in foot and ankle but when diagnosed has a very aggressive nature and high recurrence rate (40-50%). ^[3] They may present as localized form (nodular tenosynovitis) or as diffuse or infiltrative

form (Florid proliferative synovitis). Histologically, they are composed of multinucleated giant cells, macrophages, fibroblasts and xanthoma cells with varying amounts of haemosiderin within the lesion. ^[1] Here we present a rare case of young male presenting with diffuse/infiltrative form of giant cell tumour of tendon sheath of ankle joint with histopathological correlation.

CASE REPORT

A 22 year old male complained of gradually increasing painless swelling in the medial malleolus of right leg since 2 years. There was history of trauma 2 year back due to fall from bike. No h/o tingling or

numbness sensation. No overlying redness or raise in temperature of the swelling. No h/o fever, discharging sinus or evening rise of temperature. On physical examination, patient had mild tenderness with overlying soft tissue swelling. There was no history past history of tuberculosis. H/o surgical excision of similar lesion 3 years back.

High resolution ultrasonography of ankle joint was performed using linear probe (8-11 MHz) which revealed mixed echoic solid lesion , predominantly hypoechoic in appearance around the Tibialis posterior, Flexor digitorum longus and Flexor hallucis longus tendons posterior to medial malleolus and extending to foot.(Figure 1 A and B) On colour Doppler, the lesion showed increased vascularity (Figure 1 C). Possibilities of Giant cell tumour of tendon sheath and chronic tuberculosis was considered.

MRI of the right ankle joint was performed without and after administration of intravenous gadolinium contrast. Tibialis posterior, flexor digitorum longus and flexor hallucis longus in the lower third of leg extending posterior to ankle joint were thickened due to a diffuse soft tissue intensity lesion appearing heterogeneously isointense T1WI, heterogeneously on hyperintense on T2WI, STIR and PD fat sat images. Inferiorly, extension of lesion was noted in plantar aspect of foot up to their insertion. (Figures 2 and 3)

Other flexor and extensor tendons at ankle joint appeared normal. Multiple hypointese foci were noted within the lesion in all spin echo sequences showing blooming on GRE images - likely to be hemosiderin deposits (Figure 3C). On contrast study, the lesion showed heterogeneous, predominantly peripheral thick enhancement. (Figure 4)

Histopathological examination on high power view (Figure 5 A and B) showed subsynovial fibrohistiocytic cells with round to oval nuclei and abundant foamy cytoplasm, and on low power view (Figure 5 C) showed numerous villous structures. There were presence of numerous giant cells in groups. These findings confirmed giant cell tumour of tendon sheath.

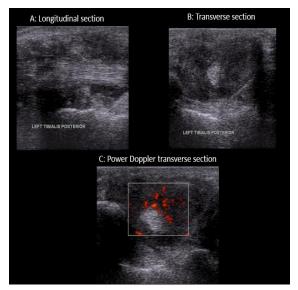


Figure 1 (A,B, C): USG (Longitudinal, transverse section, Power doppler) showing mixed echoic predominantly hypoechoic solid lesion around the tibialis posterior tendon at ankle extending posterior to medial malleolus with extension in foot showing increased vascularity on power Doppler.

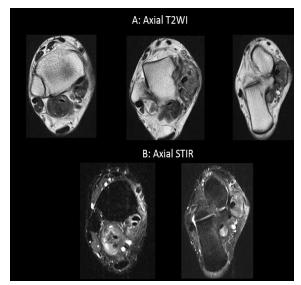


Figure 2: (A- Axial T2WI, B- Axial STIR) Showing diffuse soft tissue intensity lesion around tibialis posterior, flexor digitorum longus and flexor hallucis longus posterior to ankle appearing heterogeneously hyperintense on T2WI and STIR.

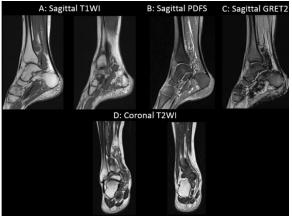


Figure 3: (A- Sagittal T1WI, B- Sagittal PDFS, C- Sagittal GRE T2, D- Coronal T2WI) Showing diffuse soft tissue intensity lesion around tibialis posterior, flexor digitorum longus and flexor hallucis longus posterior to ankle with extension into the foot, appearing isointense on T1WI, heterogeneously hyperintense on T2WI and PDFS showing blooming on GRE due to heamosiderin.

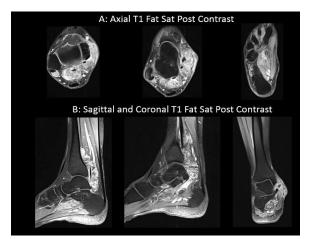


Figure 4: (A- Axial T1 fat sat post contrast, B- Sagittal and coronal T1 fat sat post contrast) Showing heterogeneous enhancement of soft tissue lesion around tibialis posterior, flexor digitorum longus and flexor hallucis longus posterior to ankle with extension into the foot.

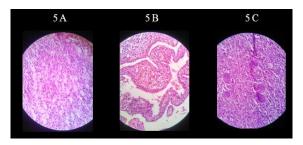


Figure 5 (A,B C)

Figure 5: Histopathological examination A and B: High power view showing subsynovial subsynovial fibrohisticytic cells with round to oval nucleI and abundant foamy cytoplasm. Numerous giant cells are present in groups. C: Low power showing numerous villous structures.

DISCUSSION

Giant cell tumour of the tendon sheath (GCT-TS) comprises approximately 1.6% of all soft-tissue tumors and is characteristically a benign peritendinous fibrous mass. ^[4] It is a slow growing proliferative benign non tumoural mass that may arise from the surrounding soft tissues, bursae, joint capsule, ligaments and tendon sheaths.^[5] GCT-TS was first described by Jaffe et al in 1941 who claimed that GCT-TS had same histological origin as pigmented villonodular synovitis (PVNS) with difference as GCTT-TS growing outward from the tendon sheath whereas pigmented villonodular synovitis growing inward from the synovial lining into the joint. Haemosiderin was found in cytoplasm of all cases of GCT-TS. ^[6] Pathogenesis behind GCT-TS is found to be uncertain. Significant number of cases showed aneuploid cells noted by DNA flow cytometry.^[1] In a study of 52 cases of GCT-TS of hand, Grover et al studied the expression of nm23 using immunohistochemical techniques on paraffin sections and found correlation of absence of this gene in 21% of tumours with significant high risk of local recurrence of tumour.^[7]

Translocations involving chromosomes 1, 2 and 16 have also been noted with GCT-TS.^[1]

GCT-TS may present as a localised form as well defined painless nodular lesion tendons attached to with rare local present recurrence or may as diffuse/infiltrative form which represents an extrarticular subtype of PVNS with very high recurrence rate. ^[5] Localised form can arise within or extrinsic to the joint whereas form originates predominantly diffuse [4] ioint. It develops outside the predominantly more in volar aspect than dorsal aspect of fingers of hands with less incidence in foot and ankle, although foot and ankle are the second commonest site to

be involved. ^[8] Both feet and plantars and dorsal aspects were found to be equally affected with lesion arising in the forefoot predominantly the great toe. ^[1] There may be a female predominance of any age with peak incidence in third to fifth decade. ^[4] In a study of 188 cases of GCT-TS by Jones FE et al 77 cases were found in hand and only 3% were found in foot. ^[9] Ushijima et al, out of 207 cases found 25 cases arising from the toes and 10 cases from the ankle and large joints of foot. ^[10] In 2003 Justin Q Ly et al were the first to describe a case of giant cell tumour of peroneus tendon sheath ^[4]

The mode of presentation and clinical features of GCT-TS in foot were found to be similar to hand. Clinically the lesion presents a slow growing mass over months to years which may be painful or asymptomatic. History of past trauma was observed by some observers significant in development of GCT-TS. ^[1,4] In presence of pain and neurogenic symptoms with a solitary lesion nerve sheath tumour or soft tissue sarcoma should be considered as a differential diagnosis. ^[1] Bony involvement by GCT-TS is rare with 11% reported incidence in hand ^[11]

Radiographically, GCT-TS is difficult to diagnose and may appear as a soft tissue mass or as a normal study. Few cases may show pressure erosions of underlying bone (10-20%),periosteal reactions, cystic degeneration, osteopenia or calcification but these are rare presentations. ^[4] On high frequency transducer ultrasonography, GCT-TS appears as a hypo echoic or hyperechoic homogenous and heterogeneous soft tissue mass with high vascularity on Colour and Power Doppler evaluation.^[12]

On MRI, GCT-TS shows low to intermediate signal intensity on T1WI and T2WI due to paramagnetic effect created by haemosiderin deposits that shortens T1 and T2 relaxation times. There is significant

blooming artifact on gradient echo images (GRE) due to haemosiderin deposits. On suppressed sequence), STIR (fat the paramagnetic effect is exaggerated creating a magnetic susceptibility and producing high signal areas. Post contrast study with gadolinium shows moderate enhancement due to numerous proliferating capillaries in Fibrosis collagenous stroma. and inflammation results in varying enhancement and helps determine the extent of lesion ^[4,12] Histological studies further helps differentiating GCT-TS from similar appearing low signal intensity lesions on MRI such as Desmoid tumour and Pigmented villonodular synovitis (PVNS). Grossly, GCT-TS appears as well encapsulated, multinodular, rubbery, greyish tan, brown, orange or yellow mass with changes in colour proportionate to amount of hemosiderin deposition and presence of foam cells. Histologically, they have histiocyte like foamy or multinucleated cells and fibroblast like cells with hemosiderin deposits.^[7] Hemosiderin deposits in GCT-TS hallmarks its diagnosis from other differentials like Desmoid tumour, fibroma, ganglion cyst. granuloma. cavernous haemangioma, malignant fibrous histiocytoma, granulomatous tenosynovitis, chondroma and granuloma.^[4]

Treatment of GCT-TS is surgical excision with removal of entire lesion to prevent local recurrence. Accurate diagnosis using USG, MRI and histology is important for planning of treatment. USG provides extent of contact and circumferential involvement of tendon and MRI is helpful for extent of lesion, preoperative planning post-operative follow and up and histopathology forms the main basis of diagnosis. In our case MRI and histopathological studies was done to accurately diagnose GCT-TS of foot and was treated with complete surgical excision without any evidence of local recurrence till date.

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