

Tenosynovial giant cell tumor mimicking an ankle monoarthritis

Francisco Vilchez-Oya , Ana Pros 

A 24-year-old woman was referred to our clinic for suspected monoarthritis located in her left ankle (Figure 1). She denied any previous trauma and personal or familial history of rheumatologic or infectious diseases.

Examination of the affected foot revealed swelling overlying the ankle joint, with limited of the range of motion and without redness or warmth in the examined area. There was no swelling in other locations.

Initial blood test revealed normal levels of C-reactive protein and erythrocyte sedimentation rate, negative antinuclear antibody, rheumatoid factor, anticitrullinated protein antibodies, and HLA-B27. Besides, an osteoarticular ultrasound was performed, which showed a marked thickening of the tibiotalar synovial membrane, which widely distends the anterior recess of the joint, showing a pseudotumor (Figure 2). After the clinical and initial imaging findings, nuclear magnetic resonance imaging (MRI) of left foot and ankle was performed (Figure 3). Because of the broad differential diagnosis of the detected mass, a biopsy was needed to confirm the diagnosis and rule out malignancy. The histopathological findings were compatible with a tenosynovial giant cell tumor (TGCT). Subsequently, because of the extension and the bone involvement, maximal resection of pathological tissue was done through open surgery.



Figure 1. Swelling overlying the left anterolateral ankle joint. Longitudinal scar (*) secondary to biopsy.

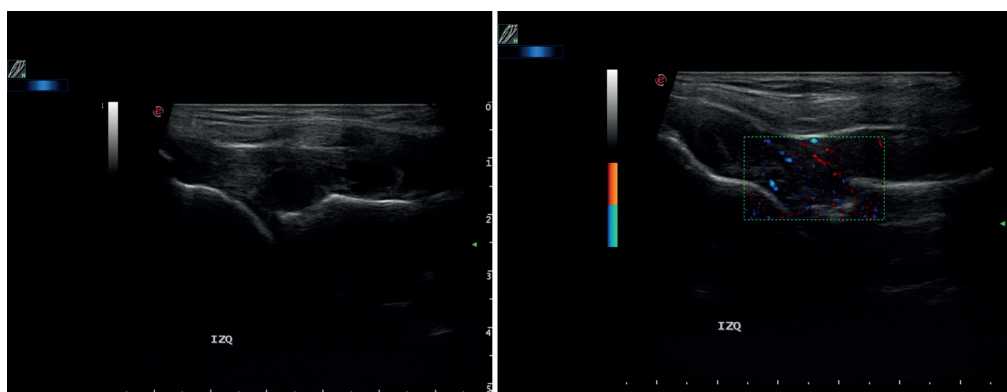


Figure 2. Marked thickening of the tibiotalar synovial membrane, with heterogeneous and hypoechoic appearance and diffuse increase in vascularization pattern with Doppler study. The synovial membrane widely distends the anterior recess of the joint, leading a mass effect on the extensor tendons.

ORCID iDs of the authors:

F.V.O. 0000-0002-0304-7993;
A.P. 0000-0003-0266-0531.

Cite this article as: Vilchez-Oya F, Pros A. Tenosynovial giant cell tumor mimicking an ankle monoarthritis. *Eur J Rheumatol* 2020; 7(4): 203-4.

Department of Rheumatology, Parc de Salut Mar/Hospital del Mar, Barcelona, Spain

Address for Correspondence:
Francisco Vilchez-Oya, Department of Rheumatology, Parc de Salut Mar/Hospital del Mar, Barcelona, Spain

E-mail: fvilchez@outlook.es

Submitted: November 13, 2019

Accepted: December 11, 2019

Available Online Date: April 28, 2020

Copyright©Author(s) - Available online at www.eurjrheumatol.org.

Content of this journal is licensed under a Creative Commons Attribution-NonCommercial 4.0 International License.





Figure 3. Sagittal T1FS before and after contrast: synovial thickening is evident in anterior and posterior recess, hyperintense in study without contrast and with marked relapse after its administration. Intralesional signal gaps persist, which identify the existence of hemorrhagic foci.

Follow-up was necessary because of the risk of recurrence in this case.

TGCT is a rare (1) but well-recognized proliferative lesion that involves the synovium, bursae, and tendon sheath. The pathogenesis is not well understood, although it has been observed that a chromosomal translocation involving 1p13 chromosome causes overexpression of the CSF1 (macrophage colony stimulating factor 1), which binds to the receptor (CSFR1) on the tumoral cell surface, leading to the expression of cells of mononuclear phagocyte lineage that constitute the tumor mass (2-4).

MRI is quite useful and shows a characteristic pattern that helps differentiate the neoplasm

from other masses. Nevertheless, a definitive diagnosis should be possible through biopsy. Regarding the treatment, classically, a surgical approach with resection has been the preferred treatment, but nowadays, medical treatment is also proposed with monoclonal antibodies inhibiting CSF1 receptors overexpressed in TGCT (2, 4, 5).

The advance in the knowledge of the etio-pathogenesis of the TGCT has opened up a greater range of possibilities for a therapeutic approach beyond the classic surgical approach.

Informed Consent: Written informed consent was obtained from the patient who participated in this study.

Peer-review: Externally peer-reviewed.

Author Contributions: Concept - F.V.O., A.P.; Design - F.V.O.; Supervision - A.P.; Analysis and/or Interpretation - F.V.O.; Writing Manuscript - F.V.O.; Critical Review - A.P.

Conflict of Interest: The authors have no conflict of interest to declare.

Financial Disclosure: The authors declared that this study has received no financial support.

References

1. Elnstein V, Andersen SL, Qazi I, Sankar N, Pedersen AB, Sikorski R, et al. Tenosynovial giant cell tumor: Incidence, prevalence, patient characteristics and recurrence. A registry-based cohort study in Denmark. *J Rheumatol* 2017; 44: 1476-83. [\[Crossref\]](#)
2. Lucas DR. Tenosynovial giant cell tumor. Case report and review. *Arch Pathol Lab Med* 2012; 136: 901-6. [\[Crossref\]](#)
3. Nilsson M, Höglund M, Panagopoulos I, Sciort R, Dal Cin P, Debiec-Rychter M, et al. Molecular cytogenetic mapping of recurrent chromosomal breakpoints in tenosynovial giant cell tumours. *Virchows Arch* 2002; 441: 475-80. [\[Crossref\]](#)
4. West RB, Rubin BP, Miller MA, Subramanian S, Kaygusuz G, Montgomery K, et al. A landscape effect in tenosynovial giant-cell tumour from activation of CSF1 expression by a translocation in a minority of tumour cells. *Proc Natl Acad Sci USA* 2006; 103: 690-5. [\[Crossref\]](#)
5. Brahmi M, Vinceneux A, Cassier PA. Current systemic treatment options for tenosynovial giant cell tumor/pigmented villonodular synovitis: Targeting the CSF1/CSF1R Axis. *Curr Treat Options Oncol* 2016; 17: 10. [\[Crossref\]](#)