EJR European Journal of Rheumatology

DOI:10.5152/eurjrheum.2025.23035

Case Report

Tuberculous Necrotizing Fasciitis in a Patient with Rheumatoid Arthritis on Anti-TNF Treatment: A Case Report

Juan Carlos Cataño[1], Pablo Arango Guerra[2]

Abstract

Tuberculous necrotizing fasciitis (NF) is an exceedingly rare condition, particularly in patients undergoing immunosuppressive therapy for autoimmune diseases such as rheumatoid arthritis. This case describes a 69-year-old male with a history of rheumatoid arthritis treated with adalimumab, who presented with severe pain, swelling, and redness in the right upper limb. Despite initial treatment for presumed bacterial NF, histological examination and tuberculosis (TB) polymerase chain reaction (PCR) confirmed tuberculous fasciitis. The patient exhibited respiratory symptoms, and imaging revealed a cavitary lesion suggestive of pulmonary tuberculosis, which was also confirmed by sputum PCR. Unfortunately, the patient succumbed to an acute myocardial infarction during treatment. This case highlights the importance of considering TB in the differential diagnosis of necrotizing fasciitis, particularly in immunocompromised individuals, to ensure timely and appropriate management.

Keywords: Tuberculosis, fasciitis, soft tissue infection, musculoskeletal

Introduction

Tuberculosis (TB) is a leading cause of illness and death globally, affecting both immunocompetent and immunocompromised individuals. Sharma et al[1] report that approximately 20% of extrapulmonary TB infections occur in immunocompetent individuals, while 50% occur in immunocompromised individuals. Among the least common manifestations of TB is extra-spinal tuberculosis, which has a relative frequency of about 1%-2%. De Backer et al[2] state that the most common extra-spinal manifestations are tuberculous arthritis and osteomyelitis, accounting for 50% and 48%, respectively, while the remaining 2% refer to tenosynovitis and bursitis but not fasciitis.

Necrotizing fasciitis (NF) is a deep fascial infection that rapidly spreads and can cause skin necrosis. Immunosuppression is a significant risk factor for this condition, usually caused by virulent streptococci, gram-negative organisms, and anaerobes.[3] However, only a few case reports describe tuberculous fasciitis, emphasizing its rarity as a cause. In this paper, we describe the case of a patient who presented with necrotizing fasciitis caused by TB. Informed consent for the preparation of this manuscript was obtained from the patient.

Case Presentation

A 69-year-old man who had a history of rheumatoid arthritis since 2000 and who had been taking adalimumab since 2015 was admitted to our hospital due to severe pain in the right upper limb accompanied by enlargement and redness in the arm and forearm, which had started 3 weeks ago (Figure 1). He denied any history of TB or known close contact but reported 8 months of malaise, fever, night sweats, cough, and a 10-km weight loss, for which he had not sought medical attention. Upon examination, he appeared malnourished and chronically ill. However, he was afebrile, with a blood pressure at 100/60 mm Hg, a pulse at 93 beats/min, a respiratory rate of 22/min, and an oxygen saturation of 96% on room air. The right arm was very painful, with noticeable edema, areas of erythema, and minimal movement of fingers and wrist. The blood chemistry showed hemoglobin 11.9 g/dL (normal range: 13-15 g/dL); leukocytes 22.000 mm^3 (normal range: 4.500-11.000 mm^3); platelet count 410 000 mm^3 (normal range: 150.000-450.000 mm^3); C-reactive protein 32 mg/dL (normal range: 0-0.5 mg/dL); creatine phosphokinase 2522 U/L (normal range: 28-174 U/L); serum sodium 134 mmol/L (normal range: 135-145 mmol/L); serum glucose 105 mg/dL (normal range: 70-100 mg/dL); kidney and liver tests were unremarkable, and an LRINEC score of

ORCID iDs of the authors:
J.C.C. 0000-0002-0150-5487;
P.A.G. 0000-0002-2254-5082.

Cite this article as: Cataño JC, Arango Guerra P. Tuberculous necrotizing fasciitis in a patient with rheumatoid arthritis on anti-TNF treatment: A case report. *Eur J Rheumatol.* 2025, 12(1), 1-4, 0035, doi: 10.5152/eurjrheum.2025.23035.

[1] Infectious Diseases Section, Internal Medicine Department, University of Antioquia School of Medicine, Medellín, Colombia

[2] Department of Internal Medicine, CES University, CES Clinic, Medellin, Colombia

Corresponding author:
Juan Carlos Cataño
E-mail: kataju@hotmail.com

Received: May 15, 2023
Revision Requested: May 23, 2024
Last Revision Received: July 2, 2024
Accepted: September 13, 2024
Publication Date: February 17, 2025

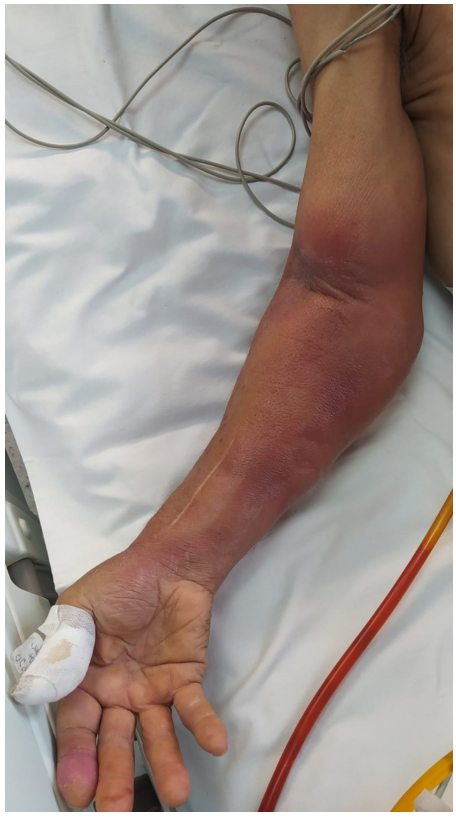

Figure 1. Right forearm with enlargement and inflammatory changes.

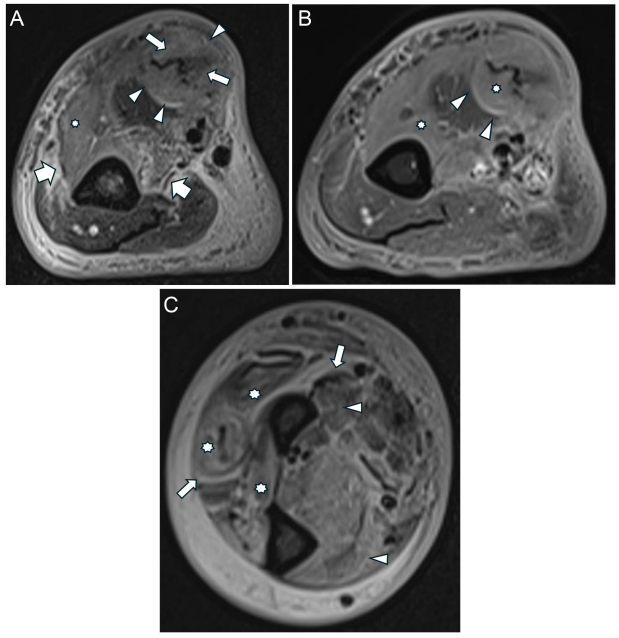

Figure 2. A. Magnetic resonance imaging shows axial T2 of the right arm with myositis of brachial biceps muscle (arrows), brachial muscle (asterisk), fasciitis (arrowhead), and fluid was observed in the space of the lateral and medial intermuscular septa of the arm (gross arrow). B. Magnetic resonance imaging shows axial contrast-enhanced T1 of the right arm with myositis of brachial biceps and brachial muscle (asterisk), and signs of fasciitis (arrowhead). C. MRI shows axial T2 of the right forearm with myositis of extensor muscles (asterisk), deep flexor muscles (arrowhead), and signs of fasciitis (arrows).

8 points was calculated. Due to the suspicion of NF, antibiotic treatment with vancomycin and piperacillin-tazobactam was initiated, and it was decided to perform contrast-enhanced magnetic resonance imaging (MRI), which revealed signs of fasciitis and myositis in the flexor and extensor muscles of the forearm, without abscesses or synovitis (Figure 2A-C). Orthopedic surgeons performed urgent surgery on the patient, during which they conducted debridement and tissue sampling.

Main Points

- Tuberculous NF, an infrequent presentation of extrapulmonary tuberculosis, is more frequently observed in patients with autoimmune diseases undergoing immunosuppressive therapy, underscoring the importance of suspecting it.
- Early diagnosis of tuberculous NF in patients with autoimmune diseases can be challenging due to its atypical presentation; however, considering tuberculosis in the differential diagnosis is crucial, at least to order basic microbiological studies for timely intervention and treatment.
- Tuberculous NF can occur even in the absence of pulmonary manifestations.

Gram stain and traditional cultures for aerobic and anaerobic bacteria were negative. However, histological examination revealed liquefaction necrosis, and Ziehl–Neelsen staining demonstrated the presence of acid-fast bacilli. The TB polymerase chain reaction (PCR) (GeneXpert) test was positive, confirming the diagnosis of tuberculous fasciitis. We also conducted a test for the human immunodeficiency virus accordingly, and the results came back negative. Due to respiratory symptoms, we conducted a contrast-enhanced chest contrasted tomography that showed consolidation in the posterior-apical segment of the left upper lobe, with areas of cavitation suggestive of TB (Figure 3A and B), and this was confirmed by Ziehl–Neelsen staining and a positive TB PCR in spontaneous sputum. The patient then began first-line treatment with rifampicin, isoniazid, pyrazinamide, and ethambutol according to local guidelines, but unfortunately, he passed away a few days later due to an acute myocardial infarction.

Discussion

Extraspinal musculoskeletal TB comprises less than 2% of cases of TB.[4,5] Tuberculous NF is a type of mycobacterial soft tissue infection secondary to the hematogenous spread of mycobacteria into the fascia. There are few cases reported in the literature, and those over the past 20 years can be found in Table 1. Most of them strikingly have underlying autoimmune diseases, including systemic lupus

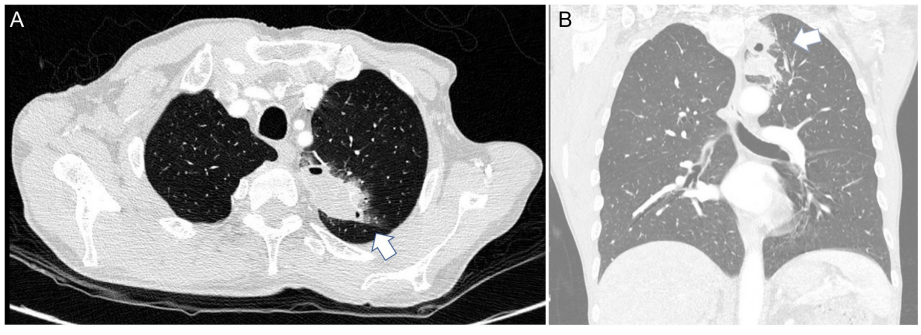

Figure 3. Chest computed tomography (CT) shows coronal (A) and axial (B) windows with consolidation of the posterior apical segment of the left upper lobe with cavitation (arrows).

Table 1. Cases of Tuberculous NF Reported in the Literature Over the Past 20 years

Author/Year	Age/Sex	Immunosuppresive Therapy	Concomitant or Previous Pulmonary Tuberculosis	Symptom Duration Prior to Diagnosis
Lee et al (2004)[7]	25/F	Yes	No	20 days
Yoshida et al (2004)[19]	69/M	Yes	No	7 weeks
Salgado et al (2006)[16]	50/M	Yes	No	8 weeks
Liu et al (2008)[17]	46/F	Yes	No	2 weeks
Cho et al (2008)[20]	79/M	No	No	2 weeks
Hefny et al (2010)[9]	46/M	No	No	4 days
Hefny et al (2010)[9]	55/M	No	Yes	5 days
Kwon et al (2010)[5]	65/F	Yes	Yes	Not mentioned
Verma et al (2014)[21]	39/M	No	Yes	5 days
Nagayama et al (2016)[4]	71/F	Yes	Yes	4 weeks
Meena et al (2021)[8]	22/M	No	No	3 days
Bayileyegn et al (2023)[22]	50/M	No	Yes	Not mentioned
Chen et al (2023)[23]	52/M	No	Yes	8 weeks
Our case	69/M	Yes	Yes	3 weeks

F, female; M, male.

erythematosus,[6] rheumatoid arthritis,[5] systemic sclerosis,[7] dermatomyositis,[4] and all with immunosuppressive therapy as a basis, not limited only to biological therapy, as in our case, but even in patients with corticosteroid use exclusively. This entity appears not limited to profound immunosuppression states, as cases describe it in immunocompetent patients.[8-10] In one of the case series by Wang et al[11] involving 35 patients, only 8.5% used immunosuppressive drugs, even though one-third had an underlying chronic disease. Our case demonstrates an uncommon type of disseminated TB in an immunocompromised patient because of anti-TNF treatment. There is a similarity in the cases described in the literature of tuberculous myofasciitis. Most affected individuals tend to come from areas with high (300 cases per 100 000) and intermediate (25-300 cases per 100 000) TB incidence rates. Our country, with 2021 data showing 26 cases per 100 000 inhabitants,[12] also falls into this category, and patient immunosuppression further adds to the risk factor. Rheumatoid arthritis patients have 4 times the risk of TB infection compared to the general population (134 cases per 100 000 vs 23 cases per 100 000), as reported by Carmona et al.[13] When patients receive anti-TNF therapy, this risk exponentially increases, reaching 1893 cases per 100 000, as Gomez et al[14] reported.

Clinical signs of TB in individuals treated with anti-TNF agents are unusual or have extrapulmonary presentations, which is consistent with our situation.

It seems remarkable that in some small case series of myofasciitis, such as Puttick et al[15] and Wang et al,[11] radiographic findings, at least in chest radiography, were abnormal between 36.3% and 51.4%; however, we cannot claim that this number is entirely conclusive and say that they did not have pulmonary involvement, given that more sophisticated diagnostic images such as chest tomography were not performed. Hematogenous spread is the most frequently cited cause of myofasciitis in the literature, but there is also evidence of contiguous dissemination and traumatic inoculation. It is worth noting that the physiological conditions of the tissue (high lactic acid concentrations, high vascularization, and a lack of the mononuclear phagocytic system) contribute to the low incidence of myofascial tuberculous infection, as these conditions create an unfavorable environment for the growth of *M. tuberculosis*.[16]

The length of the symptoms and the decreased likelihood of a fatal evolution set tuberculous fasciitis apart from the typical progression of a microorganism-produced necrotizing fasciitis infection. Contrary to pyogenic bacteria-caused necrotizing fasciitis, which can last from hours to days, the duration of muscle symptoms in tuberculosis is typically between weeks and months. However, when the patient presents with an acute symptomatic deterioration, confusion can arise. If medical professionals do not perform a robust interrogation, they may miss the data on the chronicity of the symptoms. Consequently, the unusual presentation typically causes a delay in the diagnosis.[4] Despite advances and the availability of imaging methods, it takes an average of 11 weeks to reach the correct diagnosis because of the non-recognition of TB as a potential cause of fasciitis or myositis.[4,17]

Although NF caused by pyogenic bacteria may be fatal in 70% of cases, TB exhibits a different behavior, with a maximum fatality rate of 14.3%, according to one of the case series.[11] Unfortunately, the patient perished in our situation, although the reason was not TB.

The possibility of a large inoculum suggests that this illness necessitates surgical intervention, and limited series have confirmed this.[10,11,15,18] However, in rare instances when no surgical therapy was provided, satisfactory outcomes were still achieved. Even though so few cases are reported that it is challenging to make a recommendation in this regard, cases of pyogenic bacteria-caused necrotizing fasciitis almost entirely support the need for intervention.

Tuberculous NF is an extremely rare condition that can occur even in the absence of pulmonary manifestations. Especially in immunocompromised patients, such as those seen by rheumatology specialists, the appearance of acute or chronic muscular symptoms should raise suspicion of this condition, particularly in high-endemic areas. It is crucial to focus on early surgical and antituberculous treatment to achieve good outcomes.

Informed Consent: Written informed consent was obtained from the patient who agreed to take part in the study.

Peer-review: Externally peer-reviewed.

Author Contributions: Concept – P.A.; Design – P.A.; Supervision – P.A., J.C.; Resources – P.A., J.C.; Materials – P.A., J.C.; Data Collection and/or Processing – P.A.; Analysis and/or Interpretation – P.A., J.C.; Literature Search – P.A.; Writing – P.A.; Critical Review – P.A.

Declaration of Interests: The authors have no conflict of interest to declare.

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

References

1. Sharma SK, Mohan A, Kohli M. Extrapulmonary tuberculosis. *Expert Rev Respir Med*. 2021;15(7):931-948. [CrossRef]
2. De Backer AI, Mortelé KJ, Vanhoenacker FM, Parizel PM. Imaging of extraspinal musculoskeletal tuberculosis. *Eur J Radiol*. 2006;57(1):119-130. [CrossRef]

3. Salati SA. Necrotizing fasciitis a review. *Pol Przegl Chir*. 2022;95(2):1-8. [CrossRef]
4. Nagayama I, Nagatoya K, Kurahara Y, et al. Tuberculous fasciitis in polymyositis: a rare case of extrapulmonary tuberculosis. *Intern Med*. 2016;55(21):3205-3209. [CrossRef]
5. Kwon HH, Baek SH, Park SH. Miliary tuberculosis and necrotizing tuberculous fasciitis--an unusual coexistence in a rheumatoid arthritis patient. *Int J Rheum Dis*. 2010;13(2):171-174. [CrossRef]
6. Akagi T, Hirano H, Fujita S, Morita Y. Tuberculous fasciitis in a patient with systemic lupus erythematosus. *BMJ Case Rep*. 2019;12(7):e230845.
7. Lee CH, Shim JC, Lee YW. Tuberculous fasciitis in scleroderma. *Clin Rheumatol*. 2004;23(1):66-68. [CrossRef]
8. Meena SP, Acharya N, Kala PC, Rohda M, Meena SP, Acharya N. Isolated chest wall necrotizing fasciitis: an unusual fatal manifestation of extrapulmonary tuberculosis. *Cureus*. 2021;13(12):e20585. [CrossRef]
9. Hefny AF, Abu-zidan FM. Necrotizing fasciitis as an early manifestation of tuberculosis: report of two cases. *Ulus Travma Acil Cerrahi Derg*. 2010;16(2):174-176.
10. Kabani AM, Yao JDC, Jadusingh IH, Lee BC. Tuberculous fasciitis and tenosynovitis an unusual presentation of miliary tuberculosis. *Diagn Microbiol Infect Dis*. 1993;16(1):67-71. [CrossRef]
11. Wang JY, Lee LN, Hsueh PR, et al. Tuberculous myositis: a rare but existing clinical entity. *Rheumatology (Oxford)*. 2003;42(7):836-840. [CrossRef]
12. Serna-Trejos JS, Agudelo-Quintero E, Serna-Trejos JS. Contexto epidemiológico de la tuberculosis en Colombia. *MediSur*. 2022;20(5):802-804.
13. Carmona L, Hernández-García C, Vadillo C, et al. Increased risk of tuberculosis in patients with rheumatoid arthritis. *J Rheumatol*. 2003;30(7):1436-1439.
14. Gómez-Reino JJ, Carmona L, Valverde VR, Mola EM, Montero MD, BIOBADASER Group. Treatment of rheumatoid arthritis with tumor necrosis factor inhibitors may predispose to significant increase in tuberculosis risk: a multicenter active-surveillance report. *Arthritis Rheum*. 2003;48(8):2122-2127. [CrossRef]
15. Puttick MP, Stein HB, Chan RM, Elwood RK, How AR, Reid GD. Soft tissue tuberculosis: a series of 11 cases. *J Rheumatol*. 1995;22(7):1321-1325.
16. Salgado Ordóñez F, Fúnez Liébana R, Pérez Nadal F, García González E. Fascitis necrosante como primera manifestación de tuberculosis en un paciente inmunodeprimido. *Reumatol Clin*. 2006;2(4):212-216. [CrossRef]
17. Liu CH, Liu WC, Chen LW, Chen JS. Tuberculous myofasciitis in dermatomyositis. *Clin Rheumatol*. 2008;27(suppl 1):S7-S9. [CrossRef]
18. Stebbings AE, Ti TY, Tan WC. Necrotizing fasciitis--an unusual presentation of miliary mycobacterium tuberculosis. *Singapore Med J*. 1997;38(9):384-385.
19. Yoshida Y, Nakayama J, Furue M, Matsuda T. Dermatomyositis with tuberculous fasciitis. *Eur J Dermatol*. 2004;14(2):123-124.
20. Cho WC, Pai YC, Hsiung Y, Choi WM. Cutaneous tuberculosis presenting as necrotizing fasciitis in an elderly patient. *Int J Gerontol*. 2008;2(2):79-82. [CrossRef]
21. Verma R, Shah A, Gaikwad K, Sayed ZK, Halgaonkar PS. Necrotizing fasciitis secondary to tuberculosis in a middle aged man. *J Evol Med Dent Sci*. 2014;3(24):6675-6678. [CrossRef]
22. Bayileyegn N, Mengiste DT. Necrotizing fasciitis of the chest wall caused by empyema necessitans following tuberculosis: case report and literature review. *Int J Surg Case Rep*. 2023;106:108300. [CrossRef]
23. Chen L, Zhu Y, Fan D. Necrotizing fasciitis due to mycobacterium tuberculosis: a case report. *Heliyon*. 2023;9(10):e20733. [CrossRef]