Original Article

Differential Diagnosis of Inflammatory Arthropathy Accompanying Active Tuberculosis Infection

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Objective: The study aimed to ascertain the clinical manifestations of inflammatory arthritis accompanying tuberculosis (TB) for the differential diagnosis.
Methods: We retrospectively reviewed patients with active TB who presented with inflammatory arthropathy at Seoul Medical Center. Among 2,872 patients with active TB infection, 47 had inflammatory arthropathy: 14 had crystal-induced arthropathy; 12, TB arthritis; 12, Poncet's disease (PD); 8, Rheumatoid arthritis (RA); and 1, septic arthritis. The clinical characteristics and laboratory and radiographic findings of each group were analyzed.
Results: In TB arthritis, weight-bearing joints were more commonly affected than the elbow and wrist joints. When compared to TB arthritis, PD demonstrated a significantly higher proportion of polyarthritis and involved both large and small-to-medium-sized joints. The duration of arthritis symptoms after anti-TB treatment was significantly shorter in patients with PD (56 days vs. 90 days, p=0.028). When compared to PD, RA flares during active TB infection involved only small-to-medium-sized joints rather than a mixed distribution (62.5% vs. 16.7%, p=0.035). Patients with PD more commonly had fever at onset and showed a good response to nonsteroidal anti-inflammatory drugs alone or were in remission within 3 months after anti-TB treatment. The presence of rheumatoid factor or anti-cyclic citrullinated peptide and radiographic progression after 12 months was frequently observed in patients with RA flares.
Conclusion: The differential diagnosis of inflammatory arthritis accompanying active tuberculosis infection is challenging. Comprehensive history taking and physical examination, synovial fluid analysis, and a high level of clinical suspicion are essential to avoid delayed diagnosis and to reduce the significant morbidity involved.

Keywords: Tuberculosis, Arthritis, Poncet's disease, Rheumatoid arthritis

INTRODUCTION

Tuberculosis (TB) is a serious infectious disease caused by *Mycobacterium tuberculosis* that has become a global public health problem. It usually affects the lungs, but it can affect any part of the body. Extrapulmonary TB is defined as the occurrence of TB at sites other than the lungs, and it accounts for ap-

proximately 15% of all active TB infections [1].

Bone and joint TB, which accounts for approximately 10 to 11 percent of extrapulmonary TB, occurs as a result of the seeding of organisms in bone and/or synovial tissue [2]. TB arthritis, a form of skeletal TB affecting peripheral joints, is generally a monoarticular disease that typically involves large weight-bearing joints, such as the hip and knee [3]. However, its diagnosis

is often delayed or inaccurate due to a lack of awareness and the disease's ability to mimic other inflammatory arthritides, such as rheumatoid arthritis (RA) and crystal-induced arthropathy. Poncet's disease (PD), a reactive polyarthritis in the presence of active TB, may also arise without bacterial involvement of the joint [4]. TB arthritis, PD, and RA may have similar clinical characteristics, including a chronic course with swollen joints and radiologic findings, rendering differential diagnosis difficult [5].

RA is known to be associated with an increased risk of serious infection [6], which appears to be greater in patients with higher RA disease activity than in those with low disease activity or remission [7]. In addition, recent developments in the treatment of RA, particularly biologic agents such as tumor necrosis factor (TNF)-α inhibitors, are associated with the reactivation of TB [8,9]. Accordingly, detection of inflammatory arthritis associated with TB is notably important during the clinical course of RA. However, only few studies have focused on a comprehensive review of inflammatory arthritides that occur with active TB infection. Herein, we reviewed cases of active TB infection associated with joint pain to ascertain the clinical manifestations of inflammatory arthritis accompanying TB and to aid in the differential diagnosis of RA.

MATERIALS AND METHODS

Study design and subjects

This was a retrospective cohort study conducted at Seoul Medical Center, a public hospital in South Korea. We reviewed the electronic health records of patients with active TB who presented with inflammatory arthropathy. Between January 2012 and December 2020, 2,872 patients with active TB infections were included (Figure 1). Of these, 149 (5.2%) had experienced joint symptoms such as pain, swelling, and stiffness during anti-TB treatment and were referred to a rheumatologist; 68 of these patients with joint problems were found to have non-inflammatory conditions due to underlying osteoarthritis, trauma, or side effects of anti-TB treatment. Thirty-four patients were excluded because of insufficient records for proper diagnosis. Finally, 47 patients (1.6%) with inflammatory arthritis involving peripheral joints were included.

Synovial fluid analysis was performed on all subjects suspected of having inflammatory arthropathy, and the results were confirmed by two rheumatologists. TB arthritis, infective ar-

thritis, and crystal-induced arthropathy were diagnosed by microbiological or microscopic examination of the synovial fluid. Diagnosis of RA was based on the 2010 American College of Rheumatology (ACR)/European League Against Rheumatism (EULAR) rheumatoid arthritis classification criteria. PD was diagnosed based on following clinical features: 1) inflammatory, non-erosive, and non-deforming arthritis; 2) exclusion of other causes of inflammatory arthritis; and 3) complete response to anti-TB treatment [10]. This study was approved by the Institutional Review Board of Seoul Medical Center, Seoul, South Korea (IRB No. 2020-10-019).

Data collection

We investigated demographic characteristics (age and sex), underlying clinical conditions (past TB history, comorbidities, and concomitant medication use), and data on TB infection such as presenting symptoms, site of infection (pulmonary vs. extrapulmonary), date of diagnosis of TB from arthritis onset or their temporal relationship, confirmation test, and treatment outcomes. We also collected information on the number and distribution of involved joints (either tenderness or swollen joints), the duration and outcomes of arthritis, synovial fluid analysis (white blood cell count, microbiological results, and polarized microscopic results), and radiographic findings. The serum levels of uric acid and C-reactive protein (CRP), erythrocyte sedimentation rate (ESR), and presence of rheumatoid
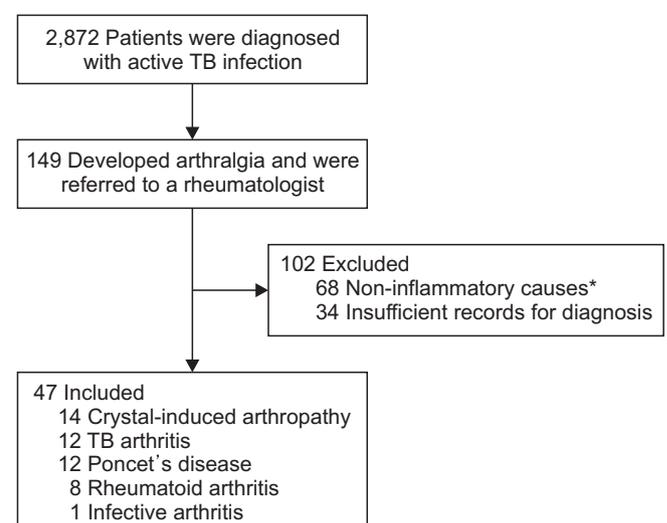
Figure 1. Flow chart of study selection. TB: tuberculosis. *Non-inflammatory conditions include osteoarthritis, trauma, or side effects of anti-TB treatment.

factor (RF) or anti-cyclic citrullinated peptide (anti-CCP) antibodies were assessed.

Diagnostic methods of TB infection

Diagnosis of TB infection was based on bacteriological examinations or clinical diagnosis by pulmonologists or infectious disease physicians. The microbiological tests included acid-fast bacilli (AFB) smear and culture tests with polymerase chain reaction (PCR) of TB and/or histopathological confirmation by the presence of caseating granuloma. Based on the site of infection, TB was classified as pulmonary or extrapulmonary TB. Since approximately 10%~50% of patients with extrapulmonary TB had concomitant pulmonary involvement [11], cases with both pulmonary and extrapulmonary TB were classified as pulmonary TB.

Statistical analysis

Continuous variables were expressed as medians with interquartile ranges (IQRs), and the majority of categorical data were presented as numbers (n) and percentages (%). Group comparisons were made using the Mann–Whitney U-test or Kruskal–Wallis test for continuous variables and the chi-squared test or Fisher's exact test for categorical variables. The comparisons between PD and RA flares are presented as odds ratios (ORs) and 95% confidence intervals (CIs) using chi-square test or Fisher's extract test. A p-value <0.05 was considered statistically significant. All analyses were performed using SPSS Statistics (version 25.0; IBM Co., Armonk, NY, USA).

RESULTS

Among 2,872 patients with active TB infection, 47 had inflammatory arthropathy: 14 had crystal-induced arthropathy; 12, TB arthritis; 12, PD; 8, RA; and 1, septic arthritis. Clinical characteristics of each inflammatory arthritis are summarized in Table 1.

In TB arthritis, the weight-bearing joints such as the hip, knee, and ankle were more commonly involved than the elbow and wrist joints (66.7% vs. 30%). When compared to TB arthritis, PD demonstrated a significantly higher proportion of polyarthritis (75% vs. 0%) and involved both large and small-to-medium-sized joints (83.3% vs. 16.7%). Arthritis symptoms

Table 1. Clinical characteristics of inflammatory arthropathy in 47 patients with active TB infection

Characteristics	TB arthritis (n=12)	Poncet's disease (n=12)	Rheumatoid arthritis (n=8)	Crystal-induced arthropathy (n=14)	Infective arthritis (n=1)
Age (yr)	61 (41~82)	63 (35~85)	68 (59~88)	72 (46~89)	75
Sex, male	8 (66.7)	7 (58.3)	4 (50.0)	11 (78.6)	0
Extrapulmonary TB	6 (50.0)	4 (33.3)	0	2 (14.3)	1
Number of affected joints*					
Monoarthritis	10	0	0	10	1
Oligoarthritis	2	3	5	4	0
Polyarthritis	0	9	3	0	0
Distribution of affected joints					
Large-sized joints	8	0	0	4	1
Small-to-medium-sized joints	2	2	5	8	0
Mixed[†]	2	10	3	2	0
ESR (mm/hr)	72 (48~120)	68 (39~110)	82 (55~108)	47 (36~76)	92
CRP (mg/dL)	5.6 (1.2~8.9)	5.9 (1.4~13.5)	7.4 (1.8~18.3)	7.8 (1.6~19.5)	31.7
Arthritis development predating the diagnosis of TB	11 (91.7)	9 (75.0)	8 (100)	1 (7.1)	0
Duration of arthritis symptoms after anti-TB treatment (d)	90 (45~120)	56 (14~90)	120 (30~180)	12 (5~21)	60

Values are presented as median (interquartile range), number (%), or number only. CRP: C-reactive protein, ESR: erythropoietin sediment rate, TB: tuberculosis. *Monoarthritis is defined as involvement of a single joint, oligoarthritis involvement of two to four joints, and polyarthritis involvement of five or more joints. [†]The pattern of joint involvement affecting small-to-medium-sized joints as well as large joints.

antedated the diagnosis of TB infection in both TB arthritis and PD (91.7% and 75%, respectively). The duration of arthritis symptoms after anti-TB treatment was significantly shorter in patients with PD (56 days vs. 90 days, p=0.028). Age, sex, primary site of TB infection (pulmonary vs. extrapulmonary TB), and serum ESR and CRP levels were not statistically different between TB arthritis and PD. One patient with TB arthritis and one with PD had underlying RA prior to the onset of active TB infection. The initial manifestations were mono- or oligoarthritis mimicking an RA flare-up without respiratory symptoms. Despite screening for latent TB infection, pulmonary TB occurred within 3 months after treatment with a TNF-α blocker.

Among the total number of active TB patients in the present study, the prevalence of RA was 1.08% (31/2,872), and its median duration was 5.8 (IQR, 2.5~14.2) years. The median prednisolone equivalent daily dose was 7.5 (IQR, 0~25) mg at the diagnosis of active TB infection. Of the 31 RA patients, 8 had a flare-up of arthritis during active TB infections. Although all of them had been diagnosed with pulmonary TB, constitutional symptoms such as fever and chills or respiratory symptoms were absent at the onset of RA flares. Consequently, it took a median of 3.4 (IQR, 1.25~5) months from the onset of arthritis symptoms to diagnose active TB infection. RA flares during active TB infection were oligo- or polyarthritis, such as PD (Table 1). There was no significant difference in ESR or CRP levels between patients with RA flares and those with PD. However, the RA flares involved only small-to-medium-sized joints rather than a mixed distribution when compared to PD (62.5% vs. 16.7%, p=0.035). In PD, a significantly higher number of patients had fever at disease onset (OR, 24; 95% CI, 1.78~248.1), showed a good response to non-steroidal anti-inflammatory drugs (NSAIDs) alone (OR, 35; 95% CI, 2.63~465.4), and had relief from arthritis symptoms within 3 months of anti-TB treatment (OR, 33; 95% CI, 2.45~443.6) (Table 2). In contrast, the presence of RF or anti-CCP and radiographic progression after 12 months was frequently observed in patients with RA flares.

Crystal induced arthropathy was the most common inflammatory arthritis associated with active tuberculosis (Table 1). Among the 14 patients with crystal-induced arthropathy, 11 (78.6%) had gout and 3 (21.4%) had pseudogout. Male patients (90.9%) or younger age groups (<65 years, 54.5%) were more common in gout cases than in pseudogout cases (both p<0.05). Similar to TB arthritis, crystal-induced arthropathy mainly presents as monoarthritis. However, small-to-medium-sized joints were predominantly involved in crystal-induced arthropathy when compared to TB arthritis (57.1% vs. 16.7%, p=0.013). Arthritis attacks developed after the diagnosis of TB infection, except for one patient with pseudogout, which coexisted with TB arthritis at the same joint (knee). Hyperuricemia (serum uric acid >7 mg/dL) was detected in 8 of 11 patients (median, 9.8 mg/dL; IQR, 7.4~14.8) who had experienced an attack of gouty arthritis before active TB infection, but only 1 patient had taken a uric acid-lowering agent (allopurinol 200 mg daily). In all gout cases, arthritis attacks occurred 4 weeks after the start of anti-TB treatment.

DISCUSSION

TB infection and its associated conditions can cause uncommon but clinically significant problems in the musculoskeletal system [2,8,12]. In the present study, 149 (5.2%) of 2,872 patients with active TB infection only complained of arthralgia,

Table 2. Comparisons between Poncet's disease and rheumatoid arthritis in active TB patients

Characteristics	Poncet's disease (n=12)	Rheumatoid arthritis (n=8)	Odds ratio*	95% confidence interval
Fever (≥38.3°C) at onset	9 (75.0)	1 (12.5)	24.00	1.78~248.1
Symmetric involvement	8 (66.7)	4 (50.0)	2.00	0.32~12.51
The presence of RF	2 (16.7)	6 (75.0)	0.20	0.03~1.33
The presence of anti-CCP	1 (8.3)	5 (50.0)	0.05	0.01~0.66
Response to NSAIDs alone	10 (83.3)	1 (12.5)	35.00	2.63~465.4
Remission within 3 months after anti-TB treatment	11 (100)	2 (25.0)	33.00	2.45~443.6
Radiographic progression after 12 months	1 (8.3)	4 (50.0)	0.09	0.01~1.08

Values are presented as number (%). Anti-CCP: anti-cyclic citrullinated peptide, NSAIDs: non-steroidal anti-inflammatory drugs, RF: rheumatoid factor, TB: tuberculosis. *Statistics were performed using chi-square test or Fisher's extract test.

with one-third of them having inflammatory arthropathy. There were 12 patients with TB arthritis, accounting for 0.4% of all cases. TB arthritis is rare and often misdiagnosed as a degenerative condition or an inflammatory arthropathy such as RA due to its chronic and indolent disease course, as well as a lack of isolation of the organism in routine culture of the synovial fluid [13,14]. Moreover, it is difficult to distinguish TB arthritis from various forms of inflammatory arthropathy during active TB infection. We found that the majority of the inflammatory arthritis cases, except crystal-induced arthropathy, revealed joint symptoms prior to the diagnosis of active TB infection. In this regard, recognition of TB infection has become a challenge for physicians when patients with active TB infection have arthralgia as an initial or primary symptom.

The typical pattern of TB arthritis is monoarthritis involving weight-bearing joints such as the hip and knee [2]. Although TB arthritis occurring in the upper extremities is rare [15], one-third of the TB arthritis cases in the present study occurred in the elbow, wrist, or metacarpophalangeal joints. To confirm TB arthritis, synovial fluid aspiration or synovial biopsy is necessary for microbiological tests, such as AFB smear/culture and TB PCR or histopathologic examination [16]. However, synovial fluid analysis, such as white blood cell counts, shows variable or nonspecific findings; it may present predominance of either neutrophils or lymphocytes [17]. Moreover, the AFB smear of synovial fluid for TB arthritis has been reported to be positive in only 20% of cases, but it was much higher for cultures, with up to 80% of cases being positive [14]. Molecular diagnostic assays using PCR have proven to be valuable for the rapid diagnosis of pulmonary TB, but their application in extrapulmonary TB remains limited. A previous study showed limited sensitivity (62.5%) and specificity (91.66%) in non-spinal samples, including various synovial tissues and synovial fluid [18]. Histopathologic findings such as granuloma formation alone may be insufficient for the diagnosis of TB [19]. It requires professional expertise and takes over two weeks to obtain the report. Nevertheless, the histological examination may allow for an early presumptive diagnosis until AFB culture results are confirmed, which takes 4~6 weeks to be reported but have a high yield to confirm the histologic finding. In this respect, patients with unexplained chronic monoarthritis, even in the upper extremities, should be considered for a synovial biopsy for histopathologic examination and AFB culture to rule out TB arthritis.

PD has been described as inflammatory polyarthritis in the presence of active visceral TB without bacterial involvement of the joints. The most commonly affected joints are the ankle and knee, but wrists and small joints in the hand can also be involved [10,20]. As the diagnosis is largely based on clinical findings and exclusion of other causes of arthritis in patients with a history of active TB, PD can often be misdiagnosed as RA. Sharma et al. [10] suggested the diagnostic criteria for PD (Sharma and Pinto's criteria): essentially including inflammatory, non-erosive, and non-deforming arthritis and excluding other causes of inflammatory arthritis. Their major criteria include a concurrent diagnosis of extra-articular tuberculosis and complete response to anti-TB treatment. In this study, we diagnosed PD in a patient who satisfied both the aforementioned essential and major criteria. PD was also diagnosed in a patient with underlying RA, when the joint symptoms improved with anti-TB treatment despite the disease-modifying antirheumatic drugs (DMARDs) being discontinued. In patients with RA flare, joint symptoms did not improve even after adequate anti-TB treatment, but were relieved after starting a new DMARD. Patients were excluded if there was insufficient evidence for diagnosis.

In our study, PD exhibited a higher proportion of polyarthritis and involved small-to-medium-sized joints as well as large joints (mixed distribution) when compared to TB arthritis. Patients with pre-existing RA developed oligo- or polyarthritis, but the disease was more likely to affect small-to-medium-sized joints, rather than a mixed distribution as seen in patients with PD. The duration of the resolution of arthritis symptoms after starting anti-TB treatment was significantly shorter in patients with PD than in those with TB arthritis. In addition, when compared to RA patients, there were more PD patients with arthritis remission within 3 months after anti-TB treatment and a good response to NSAIDs alone. The presence of anti-CCP may also be useful in distinguishing RA flares from PD. However, serological tests for RF or anti-CCP can be positive among active TB patients [21,22]. We diagnosed two patients with positive RF or anti-CCP as PD because arthralgia was spontaneously relieved with anti-TB treatment.

In all gouty cases, the arthritis attacks developed early after the diagnosis of TB infection and was associated with the initiation of anti-TB treatment. The incidence of gouty attacks on anti-TB treatment has been reported to be rare, and it is well known that gouty attacks have been associated with patients taking pyrazinamide or ethambutol, both of which induce hyperuricemia by decreasing renal uric acid clearance [12,23]. In our cohort study,

the incidence of gouty attack accompanying active TB infection was only 0.4% of all cases, and approximately three-quarters of patients had a prior history of gout but did not regularly take uric acid-lowering agents. Furthermore, the majority of patients experienced the gouty attack within 2 months of initiating anti-TB treatment, which might be because anti-TB medications such as pyrazinamide or ethambutol affects the rapid increase or fluctuation of serum uric acid levels. A recent study revealed that higher body mass index, chronic kidney disease, and pre-treatment hyperuricemia were independent risk factors for gouty attack while taking anti-TB medication [12]. It is generally unnecessary to prevent or treat drug-induced hyperuricemia because it is often asymptomatic, and the change in serum uric acid levels is reversible after the withdrawal of pyrazinamide or ethambutol. However, our findings indicate that gouty patients with untreated hyperuricemia may be prone to an acute arthritis attack after initial intensive combination treatment against active TB infection. When initiating anti-TB treatment in patients with a previous gouty attack, physicians should consider proper management of pre-treatment hyperuricemia to prevent acute attack of gouty arthritis during anti-TB treatment.

We found several patients who experienced RA flare-ups accompanying active TB infection, one of whom was newly diagnosed with RA. Treatment of RA patients with biological agents can promote active TB infection or reactivate latent TB infection [8,9], but it is unclear whether TB infection aggravates autoimmune diseases such as RA. Based on the evidence of molecular mimicry between the pathogen and host antigens, previous studies have reported that autoimmunity can be triggered by TB infection [24]. TB infection may also be associated with the emergence of various autoantibodies such as RF, anti-nuclear antibody, anti-neutrophil cytoplasmic antibodies, and anti-phospholipid antibodies [25]. An epidemiological survey in Taiwan showed associations of TB infection with an increased prevalence of RA but was unable to establish a causal relationship [26]. A previous study reported that in a collagen-induced mouse model, arthritis phenotypes are enhanced by the administration of killed *M. tuberculosis*, which promotes interleukin-6 expression in macrophages via toll-like receptor 2 [27]. Most published studies are based on epidemiological surveillance or experimental models. Further prospective cohort studies are needed to clarify whether active TB infection can exacerbate or promote RA.

The association of TB arthritis in patients with RA has been reported for several decades [28,29]. However, due to similar radiographic findings and clinical manifestations, it can be difficult to distinguish between RA and TB arthritis [5]. We observed that fewer TB patients with RA flare-ups had complained of fever than TB patients with PD. One of the contributing factors may be the unintended antipyretic effect of anti-inflammatory agents such as NSAIDs and glucocorticoids (GCs), which improve the symptoms of active RA. Furthermore, GC use not only increases the risk of developing TB but also masks constitutional symptoms suggestive of active infection [30,31]. Active TB infection should be considered as the cause of fever of unknown origin, but the fever can sometimes be low-grade or absent, particularly in the elderly [32]. Unlike septic arthritis from other etiologies, TB arthritis usually presents as a slowly progressive monoarthritis, which can be easily mistaken for disease flare-up in RA patients taking NSAIDs or GCs. The interferon-gamma release assay (IGRA), one of the screening tests for latent TB infection, may report false-negative or indeterminate results despite its high specificity. A meta-analysis reported that approximately 8%~19% of patients had a negative IGRA result when presenting with active TB [33], and it revealed suboptimal sensitivity in patients with immunodeficiency, advanced age, and extrapulmonary TB [34-36]. In our cohort study, three RA patients who were diagnosed with active TB infection showed negative IGRA and no evidence of constitutional or respiratory symptoms. They were treated with a more intensive combination of DMARDs, including TNF-α inhibitors, to control arthritis symptoms, which resulted in rapid activation of TB. As culture-positive pulmonary TB with a normal chest X-ray (CXR) is not uncommon [37], repeated sputum studies and serial follow-up CXRs can be helpful in the detection of active TB in RA patients with highly susceptible conditions when considering the biologics for arthritis control, even with negative IGRA results.

The main limitation of our study was its single-center retrospective design. We excluded 34 patients who had joint pain but had insufficient data for diagnosis, and only 47 patients with inflammatory arthritides accompanying active TB infection were identified and analyzed, resulting in low statistical power. Nevertheless, to our knowledge, this is the first study to analyze the prevalence of inflammatory arthritides in all active TB patients in a single center. In addition to our systematic analysis of the clinical features of TB-associated arthritis, we also compared it with RA. Further multicenter prospective observational studies are needed to determine the association between TB and RA.

CONCLUSION

In conclusion, TB arthritis and PD, arthropathies caused by mycobacterial infection, are uncommon disease entities even in patients with active TB, and other inflammatory conditions such as RA flare or gouty attack can potentially develop. However, the differential diagnosis of inflammatory arthritis accompanying active TB infection is challenging, as the use of TNF-α inhibitors or GCs predominantly contributes to TB reactivation in endemic areas. Comprehensive history taking and physical examination, synovial fluid analysis, and a high level of clinical suspicion are essential to avoid delayed diagnosis or misdiagnosis and to reduce the significant morbidity involved.

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CONFLICT OF INTEREST

No potential conflict of interest relevant to this article was reported.

AUTHOR CONTRIBUTIONS

Y.J. and B.Y.C. designed the study, collected and analyze data, and wrote the manuscript.

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REFERENCES

1. Sharma SK, Mohan A. Extrapulmonary tuberculosis. Indian J Med Res 2004;120:316-53.
2. Malaviya AN, Kotwal PP. Arthritis associated with tuberculosis. Best Pract Res Clin Rheumatol 2003;17:319-43.
3. Pigrau-Serrallach C, Rodríguez-Pardo D. Bone and joint tuberculosis. Eur Spine J 2013;22(Suppl 4):556-66.
4. Dall L, Long L, Stanford J. Poncet's disease: tuberculous rheumatism. Rev Infect Dis 1989;11:105-7.
5. Choi JA, Koh SH, Hong SH, Koh YH, Choi JY, Kang HS. Rheumatoid arthritis and tuberculous arthritis: differentiating MRI features. AJR Am J Roentgenol 2009;193:1347-53.
6. Listing J, Gerhold K, Zink A. The risk of infections associated with rheumatoid arthritis, with its comorbidity and treatment. Rheumatology (Oxford) 2013;52:53-61.
7. Mehta B, Pedro S, Ozen G, Kalil A, Wolfe F, Mikuls T, Michaud K. Serious infection risk in rheumatoid arthritis compared with non-inflammatory rheumatic and musculoskeletal diseases: a US national cohort study. RMD Open 2019;5:e000935.
8. Singh JA, Wells GA, Christensen R, Tanjong Ghogomu E, Maxwell L, Macdonald JK, et al. Adverse effects of biologics: a network meta-analysis and Cochrane overview. Cochrane Database Syst Rev 2011;2011:CD008794.
9. Lee SK, Kim SY, Kim EY, Jung JY, Park MS, Kim YS, et al. Mycobacterial infections in patients treated with tumor necrosis factor antagonists in South Korea. Lung 2013;191:565-71.
10. Sharma A, Pinto B, Dogra S, Sharma K, Goyal P, Sagar V, et al. A case series and review of Poncet's disease, and the utility of current diagnostic criteria. Int J Rheum Dis 2016;19:1010-7.
11. Lee JY. Diagnosis and treatment of extrapulmonary tuberculosis. Tuberc Respir Dis (Seoul) 2015;78:47-55.
12. Ha YJ, Chung SW, Lee JH, Kang EH, Lee YJ, Song YW. Clinical features and risk factors for gout attacks during anti-tuberculosis treatment: a case-control study in South Korea. Int J Rheum Dis 2019;22:1905-11.
13. Broderick C, Hopkins S, Mack DJF, Aston W, Pollock R, Skinner JA, et al. Delays in the diagnosis and treatment of bone and joint tuberculosis in the United Kingdom. Bone Joint J 2018;100-B:119-24.
14. Wallace R, Cohen AS. Tuberculous arthritis: a report of two cases with review of biopsy and synovial fluid findings. Am J Med 1976;61:277-82
15. Davidson PT, Horowitz I. Skeletal tuberculosis. A review with patient presentations and discussion. Am J Med 1970;48:77-84.
16. Lewinsohn DM, Leonard MK, LoBue PA, Cohn DL, Daley CL, Desmond E, et al. Official American Thoracic Society/Infectious Diseases Society of America/Centers for Disease Control and Prevention clinical practice guidelines: diagnosis of tuberculosis in adults and children. Clin Infect Dis 2017;64:e1-33.
17. Allali F, Mahfoud-Filali S, Hajjaj-Hassouni N. Lymphocytic joint fluid in tuberculous arthritis. A review of 30 cases. Joint Bone Spine 2005;72:319-21.
18. Pandey V, Chawla K, Acharya K, Rao S, Rao S. The role of polymerase chain reaction in the management of osteoarticular tuberculosis. Int Orthop 2009;33:801-5.
19. Shah KK, Pritt BS, Alexander MP. Histopathologic review of granulomatous inflammation. J Clin Tuberc Other Mycobact Dis 2017;7:1-12.
20. Rueda JC, Crepy MF, Mantilla RD. Clinical features of Poncet's disease. From the description of 198 cases found in the literature. Clin Rheumatol 2013;32:929-35.
21. Isenberg DA, Maddison P, Swana G, Skinner RP, Swana M, Jones M, et al. Profile of autoantibodies in the serum of patients with tuberculosis, klebsiella and other gram-negative infections. Clin Exp Immunol 1987;67:516-23.
22. Elkayam O, Segal R, Lidgi M, Caspi D. Positive anti-cyclic citrullinated proteins and rheumatoid factor during active lung tuberculosis. Ann Rheum Dis 2006;65:1110-2.
23. Ben Salem C, Slim R, Fathallah N, Hmouda H. Drug-induced hyper-

uricaemia and gout. Rheumatology (Oxford) 2017;56:679-688.

24. Shoenfeld Y, Isenberg DA. Mycobacteria and autoimmunity. Immunol Today 1988;9:178-82. Erratum in: Immunol Today 1988;9:277.

25. Machado Ribeiro F, Goldenberg T. Mycobacteria and autoimmunity. Lupus 2015;24:374-81.

26. Shen TC, Lin CL, Wei CC, Chen CH, Tu CY, Hsia TC, et al. Previous history of tuberculosis is associated with rheumatoid arthritis. Int J Tuberc Lung Dis 2015;19:1401-5.

27. Kanagawa H, Niki Y, Kobayashi T, Sato Y, Katsuyama E, Fujie A, et al. Mycobacterium tuberculosis promotes arthritis development through Toll-like receptor 2. J Bone Miner Metab 2015;33:135-41.

28. Latief W, Asril E. Tuberculosis of the wrist mimicking rheumatoid arthritis - a rare case. Int J Surg Case Rep 2019;63:13-8.

29. Seung OP, Sulaiman W. Osteoarticular tuberculosis mimicking rheumatoid arthritis. Mod Rheumatol 2012;22:931-3.

30. Galloway J, Cope AP. The ying and yang of fever in rheumatic disease. Clin Med (Lond) 2015;15:288-91.

31. Jick SS, Lieberman ES, Rahman MU, Choi HK. Glucocorticoid use, other associated factors, and the risk of tuberculosis. Arthritis Rheum 2006;55:19-26.

32. Korzeniewska-Kosela M, Krysl J, Müller N, Black W, Allen E, FitzGerald JM. Tuberculosis in young adults and the elderly. A prospective comparison study. Chest 1994;106:28-32.

33. Sester M, Sotgiu G, Lange C, Giehl C, Girardi E, Migliori GB, et al. Interferon-γ release assays for the diagnosis of active tuberculosis: a systematic review and meta-analysis. Eur Respir J 2011;37:100-11. Erratum in: Eur Respir J 2012;39:793.

34. Doan TN, Eisen DP, Rose MT, Slack A, Stearnes G, McBryde ES. Interferon-gamma release assay for the diagnosis of latent tuberculosis infection: a latent-class analysis. PLoS One 2017;12:e0188631.

35. Hang NT, Lien LT, Kobayashi N, Shimbo T, Sakurada S, Thuong PH, et al. Analysis of factors lowering sensitivity of interferon-γ release assay for tuberculosis. PLoS One 2011;6:e23806.

36. Song KH, Jeon JH, Park WB, Kim SH, Park KU, Kim NJ, et al. Usefulness of the whole-blood interferon-gamma release assay for diagnosis of extrapulmonary tuberculosis. Diagn Microbiol Infect Dis 2009;63:182-7.

37. Marciniuk DD, McNab BD, Martin WT, Hoeppner VH. Detection of pulmonary tuberculosis in patients with a normal chest radiograph. Chest 1999;115:445-52.