CASE REPORT

Poncet’s Disease (Reactive Arthritis Associated with Tuberculosis)

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Abstract:
An 82-year-old man with miliary tuberculosis was admitted to our hospital. Approximately six weeks after starting anti-tuberculosis treatment, he complained of pain in the fingers, wrists, and ankles. A histopathological examination of the synovial biopsy revealed nonspecific chronic inflammation with no granulomas. Culture of the biopsy specimen yielded no acid-fast bacilli. Poncet’s disease was diagnosed based on the clinical presentation, with no findings suggestive of other diseases. His joint pain rapidly improved with steroid therapy. Tuberculosis can cause arthritis through immune-mediated mechanisms without direct invasion in an entity known as Poncet’s disease.

Key words: Poncet’s disease, tuberculosis, reactive arthritis

(Intern Med 61: 3245-3249, 2022)
(DOI: 10.2169/internalmedicine.9241-21)

Introduction
Tuberculosis is one of the most prevalent infectious diseases worldwide, leading to substantial morbidity and mortality (1, 2). While pulmonary tuberculosis is the most common form of tuberculosis, it can directly invade multiple organs as extrapulmonary tuberculosis, including lymphadenitis, pleuritis, enteritis, meningitis, peritonitis, spondylosis, and arthritis (1-3). Furthermore, tuberculosis can cause arthritis via immune-mediated mechanisms without direct invasion. This clinical entity is known as Poncet’s disease (3). Given the rarity and scarcity of awareness of the disease, it may sometimes be underdiagnosed. Furthermore, the optimal treatment of the disease is yet to be established.

We herein report a patient who developed polyarthritis after the commencement of anti-tuberculosis treatment. A histopathological examination of the synovial biopsy specimen was performed, and Poncet’s disease was diagnosed.

Case Report
An 82-year-old man presented to our hospital with a 4-month history of cough, sputum, anorexia, and unintentional weight loss of 5 kg. He had been smoking 5 cigarettes per day for approximately 60 years. He had a history of pulmonary tuberculosis, which had been treated with medication in his 60s.

Chest radiography showed patchy shadows in both upper lungs and a mass lesion in the right middle lung (Fig. 1). Chest computed tomography (CT) revealed multiple bilateral nodules in a random distribution overlapping with patchy opacities and a calcified mass in the right upper lung lobe (Fig. 2). The sputum smear was positive for acid-fast bacilli, and the loop-mediated isothermal amplification for Mycobacterium tuberculosis was positive. He was admitted to our hospital with a diagnosis of miliary tuberculosis. Table 1 shows the results of laboratory tests on admission. He started receiving a combination of isoniazid, rifampicin, and ethambutol. The sputum culture confirmed M. tuberculosis, and the strain was sensitive to all of these drugs.

Approximately 6 weeks after the commencement of anti-tuberculosis treatment, he complained of pain in the fingers, wrists, and ankles. Hand radiography showed neither bone erosion nor joint space narrowing (Fig. 3). Magnetic resonance imaging (MRI) of the right hand revealed mild...
synovial fluid retention without bone erosion or destruction of the cartilage and joints (Fig. 4). The anti-cyclic citrullinated peptide antibody, rheumatoid factor, and matrix metalloproteinase-3 levels were not elevated (Table 2).

Joint pain was treated with loxoprofen, and subsequently, a combination of acetaminophen and tramadol was added. However, these drugs were only partially effective, and the pain worsened. Concurrently, the C-reactive protein (CRP) level was elevated (Fig. 5). A synovial biopsy was performed for a further evaluation. A histopathological examination revealed nonspecific chronic inflammation with infiltration of histiocytes and lymphocytes (Fig. 6). No granuloma was detected. A culture of the biopsy specimen yielded no acid-fast bacilli. Based on the clinical presentation of polyarthritis that arose during the anti-tuberculosis treatment with no findings suggestive of tuberculous arthritis and rheumatoid arthritis, a diagnosis of Poncet’s disease was made.

The patient started receiving prednisolone (15 mg/day), and the joint pain rapidly ameliorated, with the serum CRP level decreasing to the normal range. He was discharged home with sputum smear and culture negativity for acid-fast bacilli and completed the 12-month course of anti-tuberculosis treatment. The prednisolone dose was tapered to 5 mg/day.

Hand radiography and MRI performed six months after the completion of the anti-tuberculosis treatment revealed neither bone erosion nor destruction of the cartilage and joints (data not shown). The patient was lost to follow up as he was transferred to another hospital due to traumatic head injury.

**Discussion**

The present patient developed Poncet’s disease approximately six weeks after the commencement of anti-tuberculosis therapy for miliary tuberculosis. Although nei-
Table 1. Results of the Laboratory Tests on Admission.

| Variable                        | Value | Normal range (in our hospital) |
|---------------------------------|-------|--------------------------------|
| Hemoglobin (g/dL)               | 12.4  | 13.7-17.4                      |
| Hematocrit (%)                  | 38.2  | 40.2-51.5                      |
| White-cell count (per mm$^3$)   | 6,600 | 4,000-8,000                    |
| Differential count (%)          |       |                                |
| Neutrophils                     | 78.3  | 43.0-73.0                      |
| Lymphocytes                     | 7.9   | 19.0-50.0                      |
| Monocytes                       | 12.5  | 2.0-6.9                        |
| Eosinophils                     | 1.1   | 0.0-3.8                        |
| Basophils                       | 0.2   | 0.0-1.0                        |
| Platelet count (per mm$^3$)     | 309,000 | 120,000-300,000               |
| Red-cell count (per mm$^3$)     | 4,490,000 | 4,310,000-5,650,000         |
| Mean corpuscular volume (fL)    | 85    | 86-104                         |
| Sodium (mmol/L)                 | 135   | 135-147                        |
| Potassium (mmol/L)              | 4.0   | 3.3-4.8                        |
| Chloride (mmol/L)               | 98    | 98-108                         |
| Glucose (mg/dL)                 | 87    | 70-110                         |
| Creatinine (mg/dL)              | 0.6   | 0.3-1.1                        |
| Urea nitrogen (mg/dL)           | 13    | 9-20                            |
| Protein (g/dL)                  |       |                                 |
| Total                           | 6.9   | 6.7-8.3                         |
| Albumin                         | 3.6   | 3.8-5.3                         |
| Aspartate aminotransferase (U/L)| 15    | 14-36                           |
| Alanine aminotransferase (U/L)  | 8     | 8-45                            |
| Total bilirubin (mg/dL)         | 0.5   | 0.1-1.2                         |
| Uric acid (mg/dL)               | 2.1   | 2.8-7.5                         |
| Lactate dehydrogenase (U/L)     | 193   | 136-240                         |
| Creatine kinase (U/L)           | 39    | 42-207                          |
| C-reactive protein (mg/dL)      | 5.68  | 0.00-0.30                       |

with tuberculosis. Oligoarthritis or polyarthritis is a common clinical presentation of the disease, while tuberculous arthri-
tis usually causes monoarthritis (8, 9). Poncet’s disease fre-
quently affects small joints in the hand, including the meta-
carpophalangeal joints, knees, wrists, and ankles (8, 9). Pon-
cet’s disease has been reported to be frequently accompa-
ied by extrapulmonary tuberculosis, especially lymphadeni-
tis (9-11).

Classically, Poncet’s disease was reported as arthritis de-
voping in the acute onset of tuberculosis and ameliorating
with anti-tuberculosis treatment without causing joint destruction (9, 10). However, a subsequent report showed that Poncet’s disease can develop after commencement or even after completion of anti-tuberculosis therapy (8).

While Sharma et. al. proposed the diagnostic criteria for the disease, the diagnosis is challenging, as there are no specific findings for the disease (12). It is crucial to rule out other diseases, including tuberculous arthritis and rheumatic diseases. A synovial biopsy is useful for ruling out tuberculous arthritis, given its high sensitivity (3). However, a synovial biopsy should be cautiously considered, as it may be complicated with a fistula (3). In addition, smear and culture of the synovial fluid are important to rule out tuberculous arthritis. However, in many previous reports, the diagnosis was made based only on the diagnosis of tuberculosis, and characteristic clinical presentation, and a histopathological or microbiological examination of the joint was rarely performed (10). In the present case, we performed a synovial biopsy for the following reasons: (a) we considered that we should be cautious in making a diagnosis of Poncet’s disease, given its rarity; (b) we wished to ascertain the diagnosis for considering steroid therapy, methotrexate, and anti-TNF biologics, which are associated with substantial risk of side effects; and (c) the amount of synovial fluid was too small to allow aspiration. In the present case, neither the rheumatoid factor level nor the anti-citrullinated protein antibody level was elevated, and bone erosion was not detected on follow-up with steroid monotherapy, which is incompatible with rheumatoid arthritis. Furthermore, a histopathological examination of the synovial biopsy revealed no direct invasion of the tuberculosis. These findings made the diagnosis of Poncet’s disease more convincing.

The pathophysiology of this disease has not yet been clarified. Previous reports that Poncet’s disease can develop even after the commencement of anti-tuberculosis treatment suggest that it may be caused by immune-mediated mechanisms (8). Bacillus Calmette-Guérin immunotherapy for bladder cancer is known to cause polyarthritis as a side effect (13). In a rat model, injection of heat-inactivated *M. tuberculosis* (complete Freund’s adjuvant) induced generalized or focal arthritis, synovitis, periostitis, and tendonitis. These symptoms are considered to be developed through the same mechanisms underlying Poncet’s disease (14). Poncet’s disease may be caused by cross-reactivity, as there is structural similarity between the components of *M. tuberculosis* and those of human cartilage (15, 16). HLA-B27 has been associated with the development of Poncet’s disease (17, 18).

The optimal treatment for Poncet’s disease has not yet been established. It may subside with only NSAIDs and continuation of anti-tuberculosis therapy (8). Previous reports have suggested that steroids and methotrexate are effective (8). The optimal dose of corticosteroid for the dis-

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**Table 2.** Results of the Laboratory Tests during the Development of Polyarthritis.

| Variable                              | Value | Normal range (in our hospital) |
|---------------------------------------|-------|--------------------------------|
| Anti-cyclic citrullinated peptide antibody (U/mL) | 0.9   | <4.4                          |
| Rheumatoid factor (IU/mL)             | 1     | 0-15                          |
| Matrix metalloproteinase-3 (ng/mL)    | 91.8  | 36.9-121                      |

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**Figure 5.** Schematic diagram of the clinical course. H: isoniazid, R: rifampicin, E: ethambutol, PSL: prednisolone, CRP: C-reactive protein
A publication of this case report. The pathological examination and long-term follow-up. Steroid therapy. Our report highlights the strength of anti-tuberculosis therapy, and it rapidly ameliorated with ease approximately six weeks after the commencement of and low-dose prednisolone (19).

Patient with Poncet's disease that was refractory to NSAIDs et al. reported that adalimumab therapy was effective in a rifampicin can attenuate the efficacy of corticosteroid. Endo ease has not been established. It should also be noted that nontuberculous arthritis: clinical aspects and medical management. Rheum Dis Clin North Am 35: 21-44, 2009.

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Written informed consent was obtained from the patient for publication of this case report.

The authors state that they have no Conflict of Interest (COI).

Acknowledgement

The authors wish to thank Masanori Kikui, M.D., Ph.D., and Yae Masuda, M.T., for their contribution to the pathological examination.

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