International Journal of Research in Medical Sciences
Huwae TECJ et al. Int J Res Med Sci. 2021 Oct;9(10):3206-3208
www.msjonline.org
pISSN 2320-6071 | eISSN 2320-6012

DOI: https://dx.doi.org/10.18203/2320-6012.ijrms20213960

Case Report

Management in patient with rheumatoid arthritis and tuberculous arthritis: a case report

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Received: 09 August 2021
Accepted: 08 September 2021

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ABSTRACT

Rheumatoid arthritis (RA) is mainly treated with immunosuppressive drugs, which affects the immunological system. Therefore, the risk of tuberculosis was increased two to ten times in RA patients. Moreover, immunosuppressant is contraindicated in patients with tuberculosis arthritis. A 51-year-old male was presented with pain in his left elbow after he slipped on the floor. He was diagnosed with RA for 16 years and only took prednisone for six years. Six months before, he came to a rheumatologist and was given corticosteroid for six months for RA. The left elbow radiograph and joint aspiration revealed a tuberculosis infection. The patient treated with chloroquine and oral antituberculosis for one year and showed good clinical outcomes. Other diseases should be suspected in RA with uncommon symptoms. Chloroquine is the drug of choice in RA patients with tuberculosis who are contraindicated in immunosuppressant therapy because chloroquine has no immunosuppressant effect.

Keywords: Chloroquine, DMARDs, Rheumatoid arthritis, Tuberculosis, Tuberculosis arthritis

INTRODUCTION

Rheumatoid arthritis (RA) is an autoimmune disorder characterized by chronic synovial inflammation.[1] Autoimmune reaction namely types III hypersensitivity involving antibodies, complement, antigen-antibody complexes, macrophages, T cells, and B cells, is the cause of RA. This reaction can lead to inflammation and tissue damage. The most commonly affected joints are those in the hands and feet. These sites, however, may differ from person to person. The main symptoms of RA are joint stiffness and edema as a result of synovial inflammation. Although RA primarily affects the joints, it could be also recognized as a condition with extraarticular symptoms.[2] This syndrome has a significant negative impact on performing daily activities, including work and household tasks and health-related quality of life.[3]

Rheumatological diseases such systemic lupus erythematosus (SLE) and RA are attributed to a four-fold increase in tuberculosis infection risk.[4] The elevated risk may be due to the disease's immunological dysfunction as well as treatment with immunosuppressive drugs.[4] As previously discussed, rheumatological disease can increase the risk of tuberculosis infection, and immunosuppressant therapy use to treat RA is also thought to increase the risk of tuberculosis infection. Patients with RA who use 15–20 mg of prednisone per day for a year have been demonstrated to have a higher risk of getting active TB.[5]

Disease-modifying antirheumatic drugs (DMARDs) can be divided into two categories, biologic and synthetic. The biologic agents work by inhibiting cytokines that stimulate the inflammatory cascade that causes RA symptoms. Unless contraindicated or not tolerated, methotrexate is recommended as the first-line treatment in patients with active RA.[6] Even though it has more gastrointestinal adverse effects than methotrexate, leflunomide can be utilized as an alternate treatment. In patients with mild

symptoms, hydroxychloroquine or sulfasalazine is advised as monotherapy. In this case report, we presented a patient diagnosed with RA and tuberculosis infection who received chloroquine as a drug of choice and showed good clinical outcomes. This study has been reviewed by the Universitas Brawijaya, Faculty of Medicine Review Board, and the patient had given written consent.

CASE REPORT

A 51-year-old male was presented to our outpatient department with pain in his left elbow after he slipped on the floor; a clinical appearance of elbow and hand depicted in Figure 1. He was diagnosed with RA in 2004, and during that time, the patient only took prednisone for the first six years, while eight years later, the patient did not take the medication of his own accord. Six months before coming to our clinic, the patient consulted with a rheumatologist. From laboratory findings, the rheumatoid factor (RF) was positive, and the C-reactive protein (CRP) level was elevated, then RA flare diagnosis was made and the patient was given steroid for six months. We found swelling, tenderness, and limited range of motion in his left elbow during the physical examination. Laboratory examination showed rheumatoid factor (RF) of 520 IU/ml, erythrocyte sedimentation rate (ESR) of 99 mm/hour, CRP of 119 mg/l, and positive tuberculosis immunochromatography (TB-ICT). The radiograph examination revealed the bone destruction of his distal humerus and proximal ulna and narrowing of joint space. The X-ray radiograph of the elbow is presented in Figure 2. The patient underwent a joint aspiration, and the results represented tuberculosis infection.

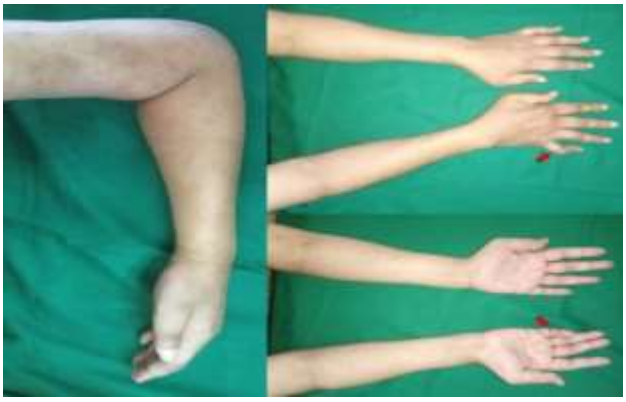

Figure 1: Clinical appearance of patient's hands. Boutonniere deformity is seen on the right hand (red arrow).

After the diagnosis was confirmed, the patient was given 400 mg chloroquine for 24 weeks as a DMARD and antituberculosis drugs to treat tuberculous arthritis. The patient experienced an improvement in his condition characterized by reduced swelling and stiffness and increased range of motion of the elbow joint after six months of therapy. ESR and CRP levels have returned to normal ranges of 12 mm/hour and 7.43 mg/l, respectively. The patient showed a better improvement after the complete treatment for one year.

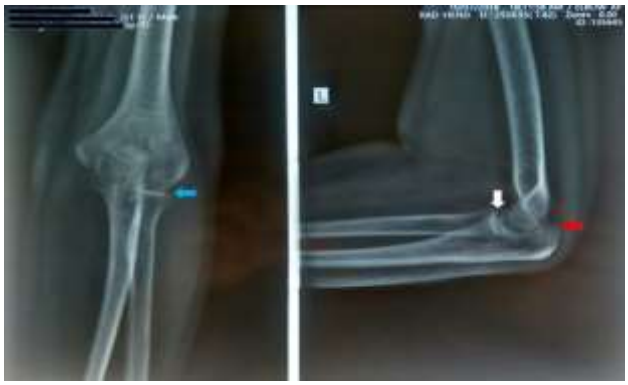

Figure 2 AP/Lateral X-ray of the left elbow. There is juxta-articular osteopenia/osteoporosis (white arrow), peripheral osseous erosions (red arrow), and narrowing of joint space (blue arrow) seen in the X-ray.

DISCUSSION

RA is a symmetrical chronic inflammatory autoimmune disease that needs immunosuppressant therapy. Treatment for RA aims to minimize joint inflammation and pain while also maximizing joint function and preventing joint degeneration and deformity. Nonsteroidal anti-inflammatory (NSAIDs) medications and corticosteroids are the first-line treatments for RA.[7] On the other side, the patient needs a good immunity system to fight infection, especially tuberculosis. In Japan, patients with RA have 3.2 times increased risk of tuberculosis, and this is suggested caused by immunological dysfunction of the disease itself and immunosuppressive therapy that alter the immunity system.[4] Moreover, Keane et al also reported that tumor necrosis factor – α neutralizing agent also increase the risk of tuberculosis in RA.[8] This controversy creates problems in deciding the therapy protocol for RA and tuberculosis patients.

As we know, RA had a symmetrical and polyarthritis clinical appearance; meanwhile, if a patient had unilateral and monoarthritis clinical appearance, we must suspect any other diseases that may emerge with RA condition. In our case, the patient had RA but with unilateral and monoarthritis clinical appearance. After a thorough examination with radiograph and arthrocentesis, tuberculous arthritis was diagnosed on the left elbow. To treat RA with tuberculous arthritis, we need to choose the management carefully. In this patient, we gave chloroquine as the second-line DMARD for RA. The overall goal of this treatment is to suppress the inflammatory responses to promote remission in order to stopping or slowing the joint destruction. Chloroquine is one of the antimalarial drugs that can be used for RA treatment for a long period. Chloroquine accumulates in human organelles and acts by increasing pH, inhibiting antigen processing and presentation, prevent dimerizing of

the α and β chains of the major histocompatibility complex (MHC) class II, and minimalized the response of inflammation. This drug also suppresses the secretion of monocyte-derived proinflammatory cytokines without immunosuppressant effects, so it did not aggravate the tuberculous arthritis condition.[9] Hence, we choose chloroquine as the drug of choice in RA patients with tuberculosis infection.

The successful treatment of RA can be evaluated by ESR and CRP level. In this patient, the ESR and CRP level was decreased from 99 mm/hour to 12 mm/hour and from 119 mg/l to 7.43 mg/l, respectively, after six months of therapy. The CRP level is still higher than the normal references because of the existing RA condition. Another parameter that can be used to evaluate the progression of RA disease is the disability of arm shoulder and hand (DASH) self-assessment. In patients who suffer from upper limb musculoskeletal disorders, the physical function and symptoms can be assessed by this DASH self-assessment questionnaire. The questionnaire was created to help people with upper-limb disabilities explain their disability and track changes in symptoms, functional disability, and quality of life over time.[10] DASH score was decreased from 56 to 19.2 after completing treatment. In patients who suffer from upper limb musculoskeletal disorders, the physical function and symptoms can be assessed by this DASH self-assessment questionnaire.

CONCLUSION

We can summarize that, uncommon clinical appearances must be recognized to diagnose and evaluate RA. Other diseases like tuberculous arthritis should be suspected in RA with monoarthritis and unilateral symptoms, given the high risk of TB in RA. Chloroquine, as DMARD, can attenuate the inflammatory process in patients with RA without immunosuppressant effects. Therefore, this drug is the primary treatment choice in RA patients with tuberculosis who are contraindicated in immunosuppressant therapy. This medicine can be given together with oral antituberculosis. The best clinical outcomes are achieved when chloroquine is used shortly after the onset.

ACKNOWLEDGEMENTS

Authors would like to thank all the colleagues and residents in Orthopaedic and Traumatology Department, Faculty of Medicine whom I have had the pleasure to work during this and other related projects.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: No. 400/005/CR/302/2021

REFERENCES

1. Catrina AI, Svensson CI, Malmström V, Schett G, Klareskog L. Mechanisms leading from systemic autoimmunity to joint-specific disease in rheumatoid arthritis. Nat Rev Rheumatol. 2017;13(2):79-86.
2. Smolen JS, Aletaha D, Barton A, Burmester GR, Emery P, Firestein GS, et al. Rheumatoid arthritis. Nat Rev Dis Prim. 2018;4:1-23.
3. Singh JA, Saag KG, Bridges SL Jr, Akl EA, Bannuru RR, Sullivan MC, et al. 2015 American College of Rheumatology Guideline for the Treatment of Rheumatoid Arthritis. Arthritis Rheumatol. 2016;68(1):1-26.
4. Yamada T, Nakajima A, Inoue E, Tanaka E, Hara M, Tomatsu T, et al. Increased risk of tuberculosis in patients with rheumatoid arthritis in Japan. Ann Rheum Dis. 2006;65(12):1661-3.
5. Kim HA, Yoo CD, Baek HJ, Lee EB, Ahn C, Han JS, et al. Mycobacterium tuberculosis infection in a corticosteroid-treated rheumatic disease patient population. Clin Exp Rheumatol. 1998;16(1):9-13.
6. Eisenberg Center at Oregon Health & Science University. Choosing Medications for Rheumatoid Arthritis. Infection. 2008.
7. Bullock J, Rizvi SAA, Saleh AM, Ahmed SS, Do DP, Ansari RA, et al. Rheumatoid arthritis: A brief overview of the treatment. Med Princ Pract. 2019;27(6):501-7.
8. Keane J, Gershon S, Wise RP, Mirabile-Levens E, Kasznica J, Schwieterman WD, et al. Tuberculosis associated with infliximab. React Wkly. 2001;874:3.
9. da Silva JC, Mariz HA, da Rocha Júnior LF. Hydroxychloroquine decreases Th17-related cytokines in systemic lupus erythematosus and rheumatoid arthritis patients. Clinics. 2013;68(6):766-71.
10. Aktekin LA, Eser F, Başkan BM, Sivas F, Malhan S, Öksüz E, Bodur H. Disability of Arm Shoulder and Hand Questionnaire in rheumatoid arthritis patients: Relationship with disease activity, HAQ, SF-36. Rheumatol Int. 2011;31(6):823-6.

Cite this article as: Huwae TECJ, Santoso ARB, Heifan A, Siahaan LD. Management in patient with rheumatoid arthritis and tuberculous arthritis: a case report. Int J Res Med Sci 2021;9:3206-8.