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Obstructive Shock due to Cardiac Tamponade Secondary to Tubercular Massive Pericardial Effusion in a Patient with Ankylosing Spondylitis on Tofacitinib Therapy: A Case Report

Richmond R Gomes[1], Siam Moazzem[2], Tohura Sharmin[3], Abir Bin Sajj[4]

[1]Professor and Head, Department of Medicine, Ad-din Women's Medical College Hospital, Dhaka, Bangladesh; [2]Assistant Professor, Medicine, Ad-din Women's Medical College Hospital, Dhaka, Bangladesh; [3]Assistant Professor, Community Medicine, Ad-din Women's Medical College Hospital, Dhaka, Bangladesh; [4]Consultant, Department of Cornea and Refractive Surgery, Vision Eye Hospital, Dhaka, Bangladesh

Abstract

Pericardial effusion is an abnormal accumulation of fluid in the pericardial cavity. Because of the limited amount of space in the pericardial cavity, fluid accumulation leads to an increased intrapericardial pressure which can negatively affect heart function. A pericardial effusion with enough pressure to adversely affect heart function is called cardiac tamponade. Pericardial effusion usually results from a disturbed equilibrium between the production and re-absorption of pericardial fluid, or from a structural abnormality that allows fluid to enter the pericardial cavity. Tuberculosis involvement of the pericardium is well-known and can result in pericardial tamponade apart from other sequelae like constrictive pericarditis. Tuberculous pericarditis (TBP) is due to hypersensitivity to tuberculin protein produced by *Mycobacterium tuberculosis* and develops in 1-2% of pulmonary TB cases, representing about 1-2% of extrapulmonary tuberculosis. Complications occur in the form of acute pericarditis (4%) and cardiac tamponade (7%), which may require life-saving invasive procedures. Risk factors include diabetes, substance use disorder, HIV-positivity, renal insufficiency, biological or immunosuppressive therapy, and exposure to regions with a high prevalence of tuberculosis. Latent tuberculosis infection (LTBI) reactivation is a well-known risk associated with immunosuppressive therapies employed in the treatment of ankylosing spondylitis (AS). Tofacitinib, an approved medication for AS that inhibits Janus kinases, has been associated with an elevated risk of TB reactivation. Here we report a case of 40 years old male, who is a known case of ankylosing spondylitis on tofacitinib therapy presented with short duration of fever and acute onset breathlessness. Urgent echocardiography shows cardiac tamponade. Pericardiocentesis was performed immediately and more than one-liter hemorrhagic fluid drained. Patient was put on anti-tubercular treatment with oral steroid after adenosine deaminase positivity and gene X pert TB positivity in exudative pericardial fluid. [*Bangladesh Journal of Infectious Diseases, June 2025;12(1):167-173*]

Keywords: Pericardial effusion; cardiac tamponade; tuberculous pericarditis; ankylosing spondylitis, latent tuberculosis

Correspondence: Dr. Richmond Ronald Gomes, Professor and Head, Department of Medicine, Ad-din Women's Medical College Hospital, Dhaka, Bangladesh; **Email:** rrichi.dmc.k56@gmail.com; **Cell no:** +8801819289499; **ORCID:** https://orcid.org/0000-0002-2511-7972

Introduction

Tuberculosis, an infectious disease predominantly caused by *Mycobacterium tuberculosis*, is a major global health concern. Despite efforts towards eradication, it continues to burden many regions of the world, particularly in low and middle-income countries[1]. Primarily a pulmonary disease, tuberculosis can disseminate to involve other parts of the body, demonstrating a wide array of clinical manifestations[2].

One such extrapulmonary manifestation is tuberculous pericarditis (TBP), a form of tuberculosis that affects the pericardium, the dual-layered sac that envelops the heart. Although tuberculosis is a prevalent disease worldwide, pericardial involvement is relatively uncommon, accounting for approximately 1.0% to 2.0% of all cases of tuberculosis[3-4]. The pathogenesis typically involves the hematogenous spread or direct extension of tubercle bacilli from a neighboring focus of infection to the pericardium. Symptoms are usually insidious in onset, with patients often presenting with chest discomfort, dyspnea, and fatigue. The vague and nonspecific nature of these symptoms can pose diagnostic challenges. In certain cases, TBP can progress to cardiac tamponade, a severe, life-threatening condition resulting from the rapid accumulation of pericardial fluid. This impedes cardiac filling, leading to hemodynamic compromise. Although classical presentation includes hypotension, distended neck veins, and muffled heart sounds, known as Beck's triad, atypical presentations are not uncommon, further complicating the diagnostic process[5]. Echocardiography plays a crucial role in this context. This noninvasive imaging modality provides key insights into cardiac structure and function, aiding in detecting pericardial effusions, assessing their hemodynamic impact, and guiding pericardiocentesis if required[5-6].

Ankylosing Spondylitis (AS) is a long-term autoimmune disorder that impacts nearly 0.1% to 1.4% of people globally. The condition is marked by inflammation of the spine, resulting in reduced quality of life, disability, and pain. In the past few years, there has been a significant advancement in the management of AS due to the development of specific therapies like biological Disease-modifying antirheumatic drugs (DMARDs) and Janus kinase (JAK) inhibitors. These medications can suppress the immune system and pose a higher risk of infections in susceptible individuals. RA patients, especially in countries with a high prevalence of tuberculosis, are particularly vulnerable to TB infections[7]. Tofacitinib further elevates the risk of tuberculosis by suppressing the production of interferon-gamma, a crucial component of the body's defense against tuberculosis[8-9].

This case report highlights an unusual presentation of TBP leading to cardiac tamponade. Our aim was to enhance understanding of this rare manifestation of widespread disease and emphasize the importance of early recognition and timely intervention. This case report also highlights a patient with Ankylosing Spondylitis who developed tuberculosis (TB) while on tofacitinib treatment. It underscores the challenges of managing tuberculosis in patients on immunosuppressive therapy, stressing the importance of careful screening, monitoring, and prompt treatment of TB in RA patients receiving such therapies, along with regular screening for latent TB infection (LTBI).

Case Presentation

A 40-year-old married Bangladeshi male Muslim service holder presented to us with the complaints of fever for 3 days, chest tightness for 2 days, and breathlessness for 1 day. According to the statement of the patient, he is a known case of Ankylosing Spondylitis (radiographic ax-SpA, HLA-B27 positive) for the last 03 years and is on regular rheumatological follow-up. His joint symptoms were in remission with Tofacitinib 5mg twice daily. The patient was admitted to our hospital with a 3-days history of high-grade, intermittent fever not associated with chills and rigors. The maximum documented temperature was 101^0 F, which subsided by taking of antipyretics. The patient also reported a compressive type of chest tightness, but not chest pain, especially on the left side for 2 days. But he denied any radiation. It was associated with nausea but not with vomiting. It was aggravated by lying supine and deep inspiration, also partially relieved by sitting upright and leaning forward. The patient also reported progressive shortness of breath for 1 day. Initially, it occurred during moderate exertion, and subsequently it progressed to such an extent that now patient is now feeling shortness of breath even at rest. Orthopnea was present as well. He denied any history of headache, body ache, cough, palpitation, weight loss, altered consciousness, recent thoracic trauma, oral ulcers, photosensitivity, or rash. His bowel and bladder habits were normal. He is a nonsmoker, nonalcoholic, and denies any substance abuse. He gave no history of contact with a patient with active tuberculosis. All his family members are in good health. He comes from a middle-class family, lives in a brick-built building with a filtered water supply

and proper sanitation. On examination, he appeared ill-looking, dyspneic with average body build (BMI 23.81kg/m^2). Anemia, jaundice, cyanosis, edema were absent. There were no skin or nail changes, but the periphery was cold and sweaty to the touch. Lymph nodes were not palpable, and the thyroid gland was not enlarged. Jugular venous pressure was elevated, about 8 cm from the sternal angle. Hepatojugular reflux was present. Blood pressure 80/70 mm of Hg, pulse 110 beats/min, regular rhythm, low volume, respiratory rate 26 breaths/min, temperature 100^0 F, SaO2 87.0% in room air. On cardiovascular system examination, apex beat was impalpable with muffled both first and second heart sound. Other systemic examinations revealed no abnormalities. On Investigations, complete blood count revealed normocytic normochromic anemia (Hb% 10 gm%, MCV 86fL, MCH 29 pcg) with normal white blood cell and normal platelet count. ESR was raised to 95 mm of Hg in 1st Hour. Pro BNP was mildly raised 605.3 pg/ml (normal less than 400 pg/ml), CRP 110.3 mg/L (normal less than 5 mg/L). Troponin -I, s. creatinine and SGPT came normal. Dengue NS1 antigen was negative. On ABG there was normal pH with hypoxia (PO2- 58 mm of Hg) and hypocapnia (PCO2-31 mm of Hg). ECG showed evidence of electrical alternans (Figure I). Chest x-ray showed enlarged cardiac shadow with fullness of pulmonary conus (Figure II).

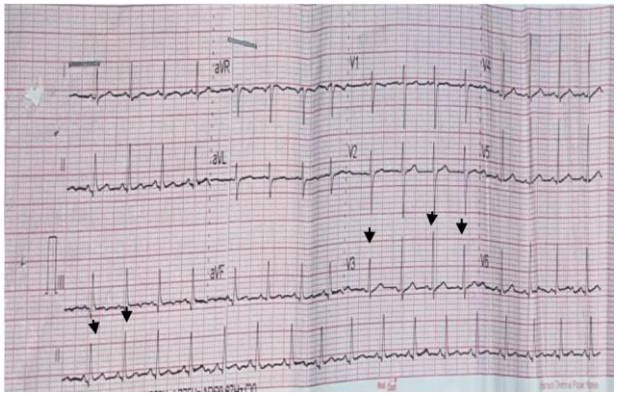

Figure I: ECG showing Beat to Beat Variation in the Amplitude of R wave (Black Arrow Head) (Electrical Alternans)

Urgent transthoracic echocardiography was arranged, which revealed Severe pericardial effusion (40 mm along the posterior wall) with diastolic collapse of the right atrium and right ventricles, suggestive of cardiac tamponade (Figure III and Figure IV).

Immediate pericardiocentesis was done. About 1400 mL of hemorrhagic pericardial fluid was aspirated through a sub-xiphoid approach, and the pericardial fluid was sent for protein, glucose, cell count, Gram stain, ZN stain, ADA, malignant cell, and gene X-pert TB. Pericardial fluid study revealed RBC-plenty/cmm, WBC 500/cmm (lymphocyte 55.0%), protein- 7.1g/dL (normal less than 2.8 g/dL), glucose- 23.4 mg/dL (normal 80 to 140 mg/ dL), ADA: 41.89U/L (normal less than 12 U/L), Gene X-pert TB: detected with no resistance to rifampicin, no malignant cells were seen. So final diagnosis of obstructive shock due to cardiac tamponade secondary to tubercular massive pericardial effusion S/P Pericardiocentesis like possible reactivation of Latent Tuberculosis due to Tofacitinib therapy with Ankylosing Spondylitis (Radiographic ax-SpA, HLA B27 positive) was made. The patient was started on anti-tubercular chemotherapy in a fixed dose combination form according to weight, along with prednisolone (1 mg/kg/day) and pyridoxine. There is a plan to give anti-TB treatment for 6 months (2 months initial intensive phase with 4 drugs, followed by continuation phase for 4 months with 2 drugs.

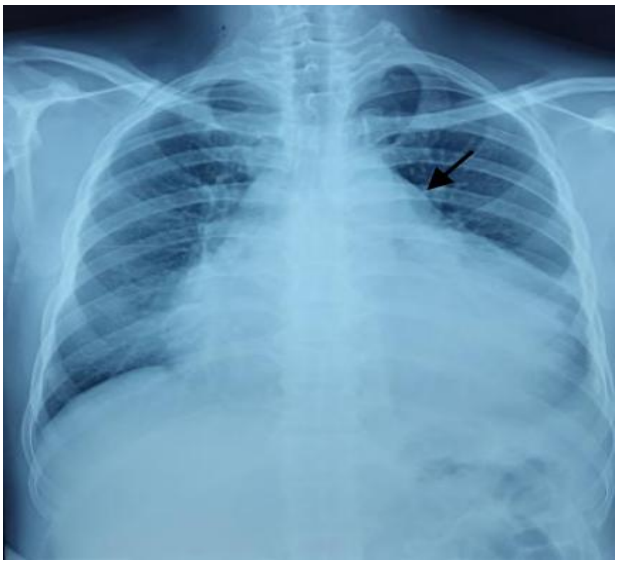

Figure II: Chest x-ray showing Enlarged Cardiac shadow with Fullness of Pulmonary Conus (black arrow head)

Steroids will be continued for 3 weeks with gradual tapering over the next 5 weeks. Tab. Tofacitinib was temporarily stopped. There is a plan to restart it after six months of completion of anti-tubercular therapy. After 5 days of initiating anti-tubercular therapy, the patient showed significant clinical improvement with improved well-being, increased appetite, subsidence of fever, supported by a reduction in CRP (22.3 mg/dl). On discharge ECG and chest x ray was normal. Echocardiography showed only 5 mm effusion (Figure V).

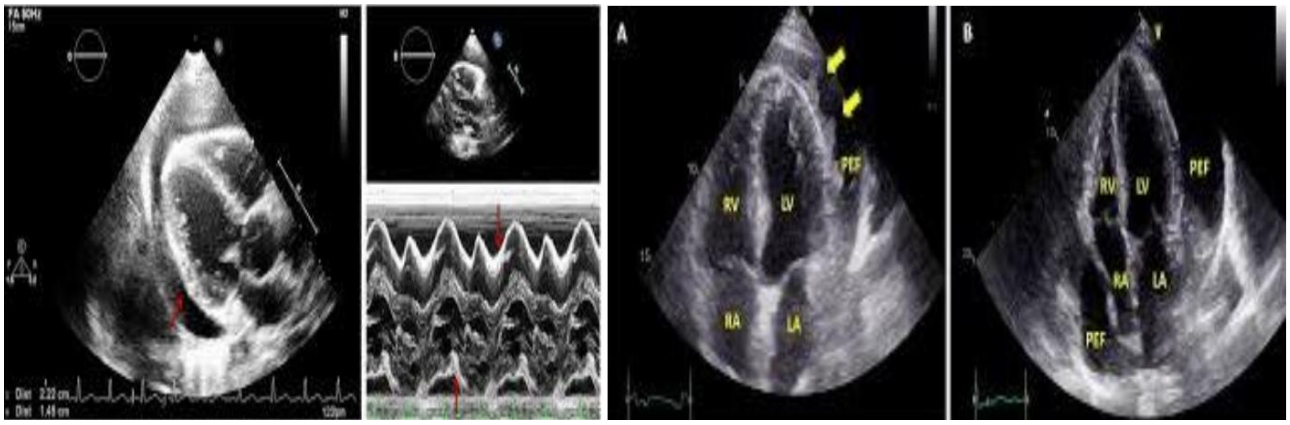

Figure III and Figure IV: Transthoracic Echocardiography Showing Severe Pericardial Effusion (40 Mm Along Posterior Wall) with Diastolic Collapse of Right Atrium and Right Ventricles

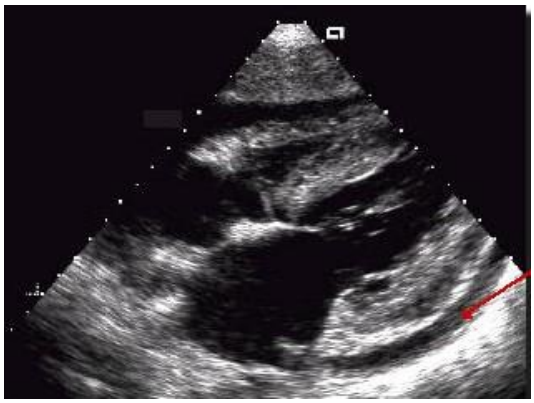

Figure V: Echocardiography showing only 5 mm Effusion Along Posterior Wall (red arrow)

Discussion

Tuberculosis (TB) is a disease caused by Mycobacterium tuberculosis, which is highly transmissible and can pose a serious threat to health if not properly treated. While TB can affect any part of the body, pulmonary TB is the most frequently observed form. Latent TB infection (LTBI) occurs when the bacteria are dormant and do not produce any symptoms or signs of active TB disease. However, LTBI can become an active TB disease, especially in individuals with weakened immune systems, such as those receiving immunosuppressive therapies for autoimmune diseases like ankylosing spondylitis (AS)[9-10].

The recurrence of tuberculosis (TB) in patients with ankylosing spondylitis (AS) is influenced by multiple factors. Firstly, immunosuppressive therapies used in the management of AS, such as corticosteroids and disease-modifying anti-rheumatic drugs, can compromise the immune system's ability to control Mycobacterium tuberculosis infection. Additionally, the presence of comorbidities, such as diabetes mellitus and chronic kidney disease, may increase the risk of TB recurrence in AS patients.[11].

Tuberculous pericarditis is a significant complication of tuberculosis, often presenting diagnostic challenges that can lead to delayed or missed diagnoses. This delay increases the risk of late complications such as constrictive pericarditis and higher mortality[12].

Tuberculous pericarditis results from Mycobacterium tuberculosis spreading to the pericardium through direct extension from adjacent structures like lungs, lymph nodes, or bones or via hematogenous dissemination during miliary TB. It often represents the reactivation of a latent infection, with the primary focus frequently undetectable.

The disease progresses through four stages: 1) fibrinous exudation with granuloma formation, 2) serosanguineous effusion with lymphocytic exudate, 3) absorption of effusion with fibrosis, and 4) constrictive scarring, which can lead to calcification and impaired cardiac filling [13,14]. These stages may occur sequentially or independently, with the effusive stage often being the earliest detectable phase, reflecting a hypersensitivity reaction to tubercular antigens. Without treatment, effusions may resolve spontaneously in about 50.0% of cases within 2 to 4 weeks, but constriction can develop unpredictably[15].

Tuberculous pericarditis presents with nonspecific symptoms, such as fever, weight loss, and night

sweats, often preceding cardiopulmonary complaints. Common symptoms include cough, dyspnea, pleuritic chest pain, and orthopnea, though their frequency varies[13]. Physical findings may include fever, tachycardia, elevated jugular venous pressure, hepatomegaly, ascites, peripheral edema, and a pericardial friction rub[8-9].

Complications include constrictive pericarditis like 30.0% to 60.0% of cases[16], effusive-constrictive pericarditis persistent constriction after effusion drainage[17], and myopericarditis which is pericarditis with myocardial involvement, often linked to HIV[18]. Cardiac tamponade, marked by pulsus paradoxus and hypotension, occurs in about 10.0% of cases[19]. Constrictive pericarditis may present with Kussmaul's sign and elevated jugular veins, while effusive-constrictive pericarditis is challenging to diagnose and is often identified during pericardiocentesis.

Diagnosis is confirmed by detecting *Mycobacterium tuberculosis* in pericardial fluid (smear/culture) or caseating granulomas on histology[14]. Presumptive diagnosis is supported by TB elsewhere, lymphocytic exudate with elevated ADA, or response to anti-tuberculous therapy. Initial evaluation includes chest radiography (cardiomegaly, pleural effusions), echocardiography (effusion/ tamponade), and sputum AFB analysis. Computed tomography (CT)/ magnetic resonance imaging (MRI) may show pericardial thickening, effusion, or lymphadenopathy. Pericardiocentesis is key for fluid analysis like exudative, lymphocytic predominance[15];

AFB smears are positive in 40.0% to 60.0% of cases, with higher yields from culture[15,20]. Polymerase chain reaction (PCR) for mycobacterial deoxyribonucleic acid (DNA) is also useful in diagnosis; however, the utility for diagnosing extrapulmonary TB is supported by small studies in endemic areas, but its effectiveness in nonendemic regions remains understudied. Measurement of pericardial adenosine deaminase (ADA) levels (cutoff 30-60 units/L) aids diagnosis, though ADA sensitivity may be lower in HIV patients[13]. A pericardial biopsy (granulomas in ~53% of cases) is useful if fluid analysis is inconclusive[14].

Each part of this therapeutic strategy plays a vital role in combating TBP. Addressing the tuberculosis infection aggressively with a potent antibiotic regimen forms the cornerstone of TBP treatment. Rifampicin, isoniazid, ethambutol, and pyrazinamide, which comprise the first-line anti-tuberculosis medications, are typically administered concurrently for an intensive 2-month period. The treatment then follows for 4 more months with isoniazid and rifampin[1]. In situations with large pericardial effusions or tamponade, pericardiocentesis can be lifesaving. It also provides the opportunity for further analysis of the pericardial fluid, which could lead to a definitive diagnosis in some cases.

The role of corticosteroids in the treatment of TBP warrants further elaboration. These potent anti-inflammatory drugs are used with the aim of preventing the progression to constrictive pericarditis, a severe complication that can lead to diminished heart function and even death. Corticosteroids function by reducing the inflammatory response in the pericardial space, thereby potentially preventing fibrosis and consequent constriction[14]. However, their use must be balanced with the potential for side effects, and thus they are typically employed when there is a high risk of constriction or when other treatment methods have not produced satisfactory results.

Prior to initiating disease-modifying anti-rheumatoid drugs, it is recommended that screening including a history of TB exposure or infection, chest X-ray, and QuantiFERON testing be performed. This allows at-risk patients to be identified and treated with chemoprophylaxis. While our patient had no risk factors for prior exposure, the importance of screening is highlighted in this instance.[21]

Tuberculosis (TB) reactivation in ankylosing spondylitis (AS) patients undergoing tofacitinib treatment, underscoring the necessity for increased vigilance within the medical community. Given the potential for drug interactions, meticulous monitoring is essential when managing TB in AS patients receiving immunosuppressive therapies.[22] Therefore, it is recommended to screen for latent TB infection (LTBI) before initiating such treatments in AS patients.

Additionally, clinicians should consider implementing appropriate prophylactic measures, such as administering isoniazid, in patients with evidence of LTBI prior to initiating tofacitinib. These recommendations aim to minimize the risk of tuberculosis reactivation and enhance patient safety during tofacitinib therapy for AS. By adhering to these guidelines and advancing our understanding of this potential risk, clinicians can optimize the management and outcomes of RA patients while minimizing the risk of tuberculosis reactivation.

Conclusion

Tuberculous pericarditis is a serious tuberculosis (TB) complication that can be difficult to diagnose and often goes undetected, leading to late complications such as constrictive pericarditis and cardiac tamponade, which lead to increased mortality. This current case illustrates a young female patient presenting with isolated TB pericarditis complicated by cardiac tamponade. She had massive improvement following pericardiocentesis and *anti*-TB treatment. In Bangladesh, tuberculous pericarditis should be considered as a differential diagnosis in any patient presenting with moderate to massive pericardial effusion. A high index of suspicion is required for the diagnosis of extrapulmonary TB pericarditis, especially in patients with known risk factors.

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Conflict of Interest
None

Financial Disclosure
None

Authors' contributions
All co-authors are responsible for data compilation and management plan and writing of discussion part with referencing.

Data Availability
Any inquiries regarding supporting data availability of this study should be directed to the corresponding author and are available from the corresponding author on reasonable request.

Ethics Approval and Consent to Participate
Written inform consent has been taken from the patient.

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ORCID
Richmond R Gomes: https://orcid.org/0000-0002-2511-7972
Siam Moazzem: https://orcid.org/0009-0007-9634-1529
Tohura Sharmin: https://orcid.org/0009-0001-1136-9351
Abir Bin Sajj: https://orcid.org/0000-0002-2326-3378

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References

1. Garcia-Garcia-de-Paredes A, Rodriguez-de-Santiago E, Aguilera-Castro L, Ferre-Aracil C, Lopez-Sanroman A. Fecal microbiota transplantation. Gastroenterol Hepatol. 2015;38(3):123-134
2. Zumla A, Raviglione M, Hafner R, von Reyn CF. Tuberculosis. N Engl J Med. 2013;368(8):745-755
3. Mayosi BM, Burgess LJ, Doubell AF. Tuberculous pericarditis. Circulation. 2005;112(23):3608-3616.
4. Syed FF, Mayosi BM. A modern approach to tuberculous pericarditis. Prog Cardiovasc Dis. 2007;50(3):218-236
5. Roy CL, Minor MA, Brookhart MA, Choudhry NK. Does this patient with a pericardial effusion have cardiac tamponade? JAMA. 2007;297(16):1810-1818
6. Lange RA, Hillis LD. Clinical practice. Acute pericarditis. N Engl J Med. 2004;351(21):2195-2202
7. Song YJ, Cho SK, Kim H, et al. Risk of tuberculosis development in patients with rheumatoid arthritis receiving targeted therapy: a prospective single center cohort study. J Kor Med Sci. 2021;36(10):e70
8. Winthrop KL, Park SH, Gul A, et al. Tuberculosis and other opportunistic infections in tofacitinib-treated patients with rheumatoid arthritis. Ann Rheum Dis. 2016;75(6): 1133–1138
9 Kiazyk S, Ball TB. Latent tuberculosis infection: an overview. Can Commun Dis Rep Releve des maladies transmissible au Canada. 2017;43(3-4):62–66
10. Schwartz DM, Kanno Y, Villarino A, Ward M, Gadina M, O'Shea JJ. JAK inhibition as a therapeutic strategy for immune and inflammatory diseases. Nat Rev Drug Discov. 2017;17(1):78.
11. Cantini F, Nannini C, Niccoli L, Petrone L, Ippolito G, Goletti D. Risk of tuberculosis reactivation in patients with rheumatoid arthritis, ankylosing spondylitis, and psoriatic arthritis receiving non-anti-TNF-targeted biologics. Mediators of inflammation. 2017;2017(1):8909834.
12. Diagnostic Standards and Classification of Tuberculosis in Adults and Children. This official statement of the American Thoracic Society and the Centers for Disease Control and Prevention was adopted by the ATS Board of Directors, July 1999. This statement was endorsed by the Council of the Infectious Disease Society of America, September 1999. Am J Respir Crit Care Med. 2000;161(4 Pt 1):1376-95
13. Ortbals DW, Avioli LV. Tuberculous pericarditis. Arch Intern Med. 1979;139:231-4
14. Mayosi BM, Burgess LJ, Doubell AF. Tuberculous pericarditis. Circulation. 2005;112(23):3608-16
15. Permanyer-Miralda G, Sagristá-Sauleda J, Soler-Soler J: Primary acute pericardial disease: a prospective series of 231 consecutive patients. Am J Cardiol. 1985;10:623-30
16. Cegielski JP, Lwakatare J, Dukes CS, et al.: Tuberculous pericarditis in Tanzanian patients with and without HIV infection. Tuber Lung Dis. 1994;75:429-34
17. Gooi HC, Smith JM: Tuberculous pericarditis in Birmingham. Thorax. 1978;33:94-6
18. Nahid P, Dorman SE, Alipanah N, et al.: Official American Thoracic Society/Centers for Disease Control and Prevention/Infectious Diseases Society of America clinical practice guidelines: treatment of drug susceptible tuberculosis. Clin Infect Dis. 2016;63:e147-95
19. Maisch B, Seferović PM, Ristić AD, et al. Guidelines on the diagnosis and management of pericardial diseases executive summary: the Task Force on the Diagnosis and Management of

Pericardial Diseases of the European Society of Cardiology. Eur Heart J. 2004;25:587-610
20. Strang JI, Kakaza HH, Gibson DG, et al.: Controlled clinical trial of complete open surgical drainage and of prednisolone in treatment of tuberculous pericardial effusion in Transkei. Lancet. 1988;332:759-64
21. Reuter H, Burgess L, van Vuuren W, Doubell A. Diagnosing tuberculous pericarditis. QJM. 2006;99:827-39
22. Schepers GW. Tuberculous pericarditis∗. The American Journal Of Cardiology. 1962;9(2):248-76.