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Cardiac Tamponade as a Presenting Manifestation of Rheumatoid Arthritis: Case Report

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Abstract | **Case Report**

Pericarditis is the most common cardiac condition in rheumatoid arthritis. Its clinical expression is rare and is exceptionally complicated by tamponade. Patients with RA who developed hemodynamic compromise as a result of pericardial involvement had a 100% mortality rate**.** We report in this observation the case of a 35-year-old female patient admitted for tamponade on a rheumatoid arthritis background.

Keywords: tamponade, rheumatoid arthritis, pericarditis.

INTRODUCTION

Rheumatoid arthritis (RA) is the most common inflammatory arthritis. While more effective therapies for RA have become available, it has been identified that patients with RA still have a higher mortality compared to the general population.

Pericarditis is considered the most common cardiac manifestation of RA. It usually falls under the specific cardiac involvement of the disease [1].

Its clinical expression is rare, occurring in less than 5% of patients, with signs of pericardial involvement observed in 20-50% of patients by echocardiography [2, 3]. Autopsy series also report that 20-40% of patients show signs of pericarditis, most often fibrinous [2, 4].

CASE REPORT

Mrs. S.K., 35 years old, was treated for seropositive RA with little erosive activity for eight years, and had been on methotrexate for two years.

Admitted with rest dyspnea with orthopnea associated with atypical positional chest pain.

The initial cardiovascular and pleuropulmonary examination showed signs of poor hemodynamic tolerance: tachycardia at 110 bpm, polypnea at 25 bpm, hypotension at 8/6 (baseline blood pressure at 13/8) and turgidity of the jugular veins.

The cardiac echography shows a large pericardial effusion (post=24mm; latero-VG=19mm; latero-VD=21mm; ant=11mm; retro-OD=25mm) with the presence of respiratory variations. As well as the presence of floating hyperechogenic images in the pericardial fluid.

In front of this table of tamponade, the patient benefited from an emergency pericardial drainage, bringing back 1000 cc of serumatic fluid.

The pericardium is thick with a highly inflammatory appearance. Anatomopathological examination of the biopsy fragments of the pericardium concluded suppurative and inflammatory subacute and chronic nonspecific remodeling. The bacteriological study of the pericardial fluid noted an absence of germs on direct examination with sterile culture. The XPERT gene in the pericardial fluid is negative

The biology shows an inflammatory syndrome (VS at 85, CRP at 62 mg/l). Cardiac enzymes (transaminases, CPK, LDH, troponin) are normal. Rheumatoid factor test was positive at 82 IU/l, antinuclear antibody test was negative, viral serologies were negative. Chest X-ray shows no abnormalities.

On electrocardiogram, there is an electrical alternation with repolarization disorders such as negative T waves in ASA and low lateral.

The thoraco-abdomino-pelvic CT scan shows nodular formations of the lingula and postero-basal right with bilateral axillary nodes of juxta and supra centimetric size with sub segmental ventilation disorders of the lingula and integrity of the abdomino-pelvic viscera.

A post-drainage echographic check-up showed a pericardial effusion anteriorly, retro-OD and posteriorly estimated at 12 mm without respiratory variations with an inferior vena cava diameter of 17 mm. LV systolic function was preserved at 60% (SBP).

Corticosteroid therapy at 1 mg/kg per day was introduced with progressive decrease of the doses according to the clinical and echographic data. A treatment with colchicine 1 mg/d was introduced to avoid recurrences. The evolution was favorable on the clinical, biological and echographic levels. Indeed, dyspnea and chest pain disappeared, the biological inflammatory syndrome regressed, and the ultrasound controls did not show any recurrence of the effusion.

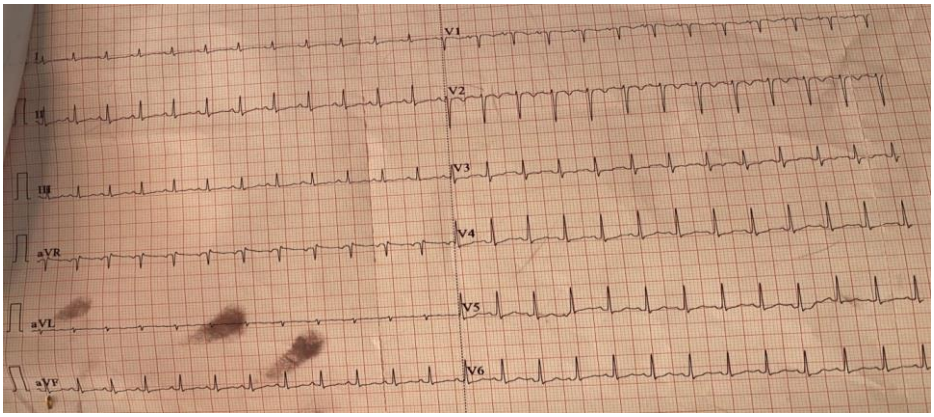

Figure 1: Electrocardiogram showing electrical alternation with repolarization disorder

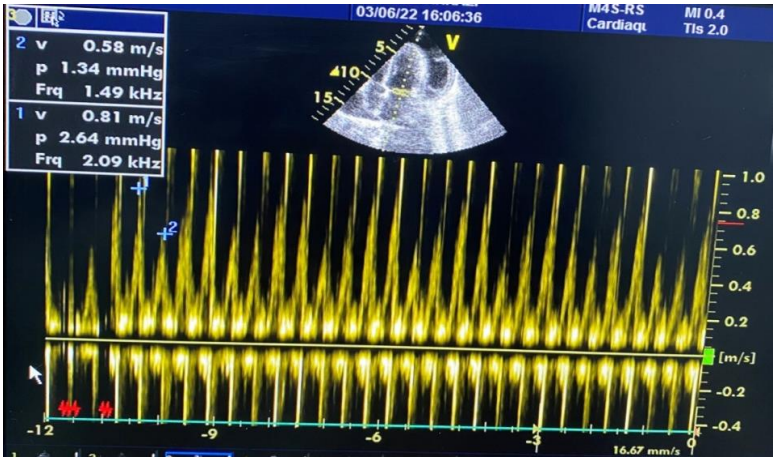

Figure 2

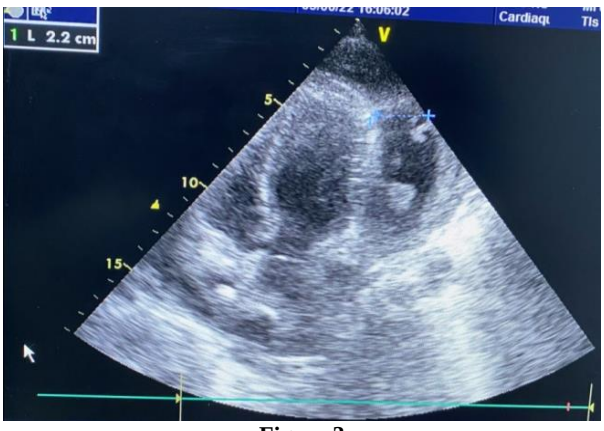

Figure 3

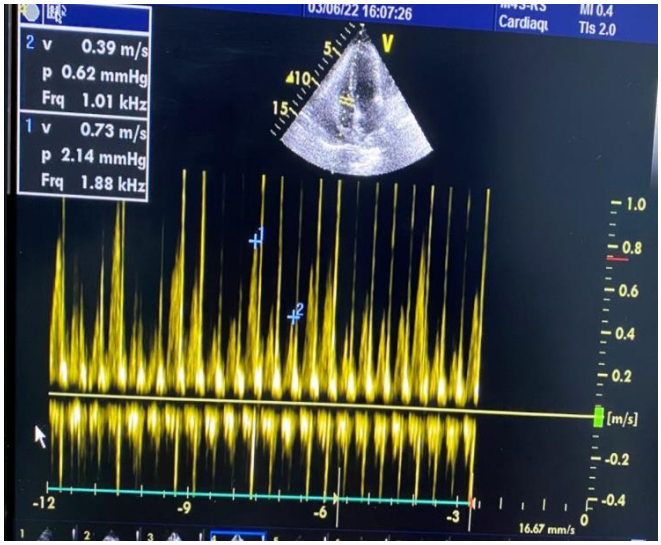

Figure 4

Figure 2, 3, 4: Echocardiography showing pericardial effusion with respiratory variations

DISCUSSION

Pericarditis is frequently seen in male patients, with an estimated annual incidence of 0.34% in women and 0.44% in men [5] in those who are HIV positive and in patients with severe or active disease. Pericarditis may be constrictive or effusive [6].

Pericarditis does not always parallel joint inflammation. Rarely, patients may present with pericarditis in the setting of positive serologies for RA and in the absence of significant joint symptoms.

Pericardial effusions can be caused by a variety of pathologic conditions, including malignancy, viral infection, bacterial infection, tuberculosis, hypothyroidism, and collagen vascular disease [7].

It is reasonable to suspect such a process in the production of the pericarditis that led to tamponade. The absence of bacteria in the tissues, sterile culture of the pericardial fluid, and the excellent results of treatment with steroids and chlorambucil without antibiotic "blanket" provide additional support for the rheumatoid etiology of this pericarditis.

The pathogenesis of pericarditis in RA is unknown, although it is assumed to be the same as the pathogenesis of other extra-articular manifestations. Whether there is a relationship with certain genetic markers is unknown.

Rheumatoid pericarditis most commonly presents with precordialgia. Dyspnea with orthopnea, peripheral edema, hepatic or gastrointestinal congestion may be seen in constrictive forms. Pericardial rubbing is present in 30-40% of cases and pericardial snapping is present in constrictive forms. Other signs (tachycardia, muffled heart sounds) are nonspecific [2]. General signs (fever, asthenia, weight loss) are common.

Hemodynamic compromise due to pericardial disease is uncommon and is seen in approximately 0.5% of patients [8].

The electrocardiogram is quite often normal. X-rays show cardiomegaly and often a uni- or bilateral pleural effusion. Ultrasound is the most useful examination to demonstrate the effusion. This examination also allows the assessment of the impact of the effusion on the cardiac chambers [6].

The pericardial effusion is usually small or medium in size. Exceptionally, it is very abundant and results in tamponade, as was the case in our patient.

Chest CT or right heart catheterization may be necessary to make a diagnosis of pericarditis, especially in cases of constrictive disease. CT may show pericardial inflammation, fluid, or calcification.

CT is most useful in cases of constriction, allowing constrictive pericarditis to be distinguished from restrictive cardiomyopathy [2].

Pericardial fluid analysis classically reveals an exudate rich in protein, lactic dehydrogenase, and leukocytes, while serum complement and glucose levels are lowered [9].

Cholesterol crystals may also be present. These can be seen in tuberculous pericarditis. Staphylococcal pyopericarditis has also been described in RA. Infectious pericarditis thus represents the main differential diagnosis of rheumatoid pericarditis [2].

Histological study usually shows thickening of both pericardial sheets by fibrous tissue and sometimes a cellular infiltrate that is mostly CD8 lymphocytes [9].

Immunofluorescence may show immunoglobulin and complement deposits [10].

Other aspects may be observed such as rheumatoid nodules and vasculitis lesions.

The rarity of this condition is reflected in the paucity of literature documenting the efficacy of different therapeutic approaches and the prognosis of these patients.

For example, there is no evidence-based treatment regimen for rheumatoid pericarditis. According to existing reports, rheumatoid pericarditis has been successfully treated with non-steroidal anti-inflammatory drugs NSAIDs, corticosteroids, and immunosuppressive disease-modifying antirheumatic drugs (DMARDs) or DMARDS biological therapy [11].

There is evidence to suggest that biologic therapy reduces the occurrence of extra-articular manifestations of RA [12].

On the other hand, cases of pericarditis have been reported in patients receiving biologic therapy in this situation, infection and malignancy must be ruled out [13].

However, when RA pericarditis progresses rapidly and results in massive pericardial effusion and subsequent tamponade, corticosteroids are inadequate and pericardiocentesis is warranted. On the basis of our findings, we conclude that surgical drainage of pericardial fluid is indicated in cases of acute life-threatening tamponade, both as a therapeutic and diagnostic modality to confirm rheumatoid pericarditis.

CONCLUSION

Pericarditis is rare in rheumatoid arthritis. Constrictive forms and those complicated by tamponade are serious and may be life-threatening. The rarity of this condition is reflected in the paucity of literature documenting the efficacy of various therapeutic approaches and the prognosis of these patients.

BIBLIOGRAPHY

1. Mohyuddin, T., Elyan, M., & Kushner, I. (2007). Pericarditis: a rare complication of methotrexate therapy. *Clinical rheumatology*, *26*(12), 2157-2158.
2. Kitas, G., Banks, M. J., & Bacon, P. A. (2001). Cardiac involvement in rheumatoid disease. *Clinical Medicine*, *1*(1), 18-21.
3. MODY, G. M., STEVENS, J. E., & MEYERS, O. L. (1987). The heart in rheumatoid arthritis-a clinical and echocardiographic study. *QJM: An International Journal of Medicine*, *65*(2), 921-928.
4. Koivuniemi, R., Paimela, L., Suomalainen, R., & Leirisalo-Repo, M. (2013). Cardiovascular diseases in patients with rheumatoid arthritis. *Scandinavian journal of rheumatology*, *42*(2), 131-135.
5. Jurik, A. G., & Graudal, H. (1986). Pericarditis in rheumatoid arthritis. *Rheumatology International*, *6*(1), 37-42.
6. Barcin, C., Yalcinkaya, E., & Kabul, H. K. (2013). Cholesterol pericarditis associated with rheumatoid arthritis: a rare cause of pericardial effusion. *International Journal of Cardiology*, *166*(3), e56-e58.
7. Goyle, K. K., & Walling, A. (2002). Diagnosing pericarditis. *American family physician*, *66*(9), 1695-1702.
8. Escalante, A., Kaufman, R. L., Quismorio Jr, F. P., & Beardmore, T. D. (1990, December). Cardiac compression in rheumatoid pericarditis. In *Seminars in arthritis and rheumatism* (Vol. 20, No. 3, pp. 148-163). WB Saunders.
9. Travaglio-Encinoza, A., Anaya, J. M., AD, D. D. A., Reme, T., & Sany, J. (1994). Rheumatoid pericarditis: new immunopathological aspects. *Clinical and experimental rheumatology*, *12*(3), 313-316.
10. Bologna, C., Poirier, J. L., Herisson, C., & Simon, L. (1992). Constrictive pericarditis in severe seronegative rheumatoid polyarthritis. *La Revue de Medecine Interne*, *13*(1), 64-68.
11. Imadachi, H., Imadachi, S., Koga, T., Miyashita, T., Izumi, Y., Takayama, H., ... & Migita, K. (2010). Successful treatment of refractory cardiac tamponade due to rheumatoid arthritis using pericardial drainage. *Rheumatology international*, *30*(8), 1103-1106.
12. Nyhäll-Wåhlin, B. M., Petersson, I. F., Jacobsson, C., Geborek, P., Nilsson, J. Å., Nilsson, K., ... & Turesson, C. (2012). Extra-articular manifestations in a community-based sample of patients with rheumatoid arthritis: incidence and relationship to treatment with TNF inhibitors. *Scandinavian journal of rheumatology*, *41*(6), 434-437.
13. Ambrose, N. L., & O'Connell, P. G. (2007). Anti-TNF alpha therapy does not always protect rheumatoid arthritis patients against developing pericarditis. *Clinical and experimental rheumatology*, *25*(4), 660.