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A Rare Case of Rheumatoid Arthritis Complicated by Complete Heart Block and Lead Thrombosis

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ABSTRACT

Rhematoid arthritis is a chronic inflammatory autoimmune disease characterized by progressive and disabling symmetrical polyarthritis and other organ damage. Heart manifestations include myocardial, valvular and conduction tissue abnormalities. Complete heart block is rare and results from a direct involvement of the conducting system with granulomas and subsequent fibrosis or extension of the inflammatory process. When symptomatic, it usually presents with lipothimia or syncope. In which case, the treatment of choice is a permanent pacemaker implantation that may complicate with thrombosis. Several hypotheses have been discussed: heart failure, atrial fibrillation, coagulopathie and the pacemaker lead itself may predispose to thrombosis.

There are no established standard guidelines for determining the therapeutic course of action following the diagnosis of a clot attached to the cardiac device lead. Some researchers suggest that the decision should depend on the clot's size and the signs and symptoms exhibited by the patients.

KEYWORDS: rhematoid arthritis, atrio-ventricular block, pacemaker, thrombosis

INTRODUCTION

Rheumatoid arthritis is a common chronic autoimmune disease with a prevalence of≈1% and a female predominance. It is a systemic disease resulting in extraarticular manifestations, including subcutaneous nodules, ocular, lung and cardiac involvement, peripheral neuropathy, vasculitis, and haematologic abnormalities.

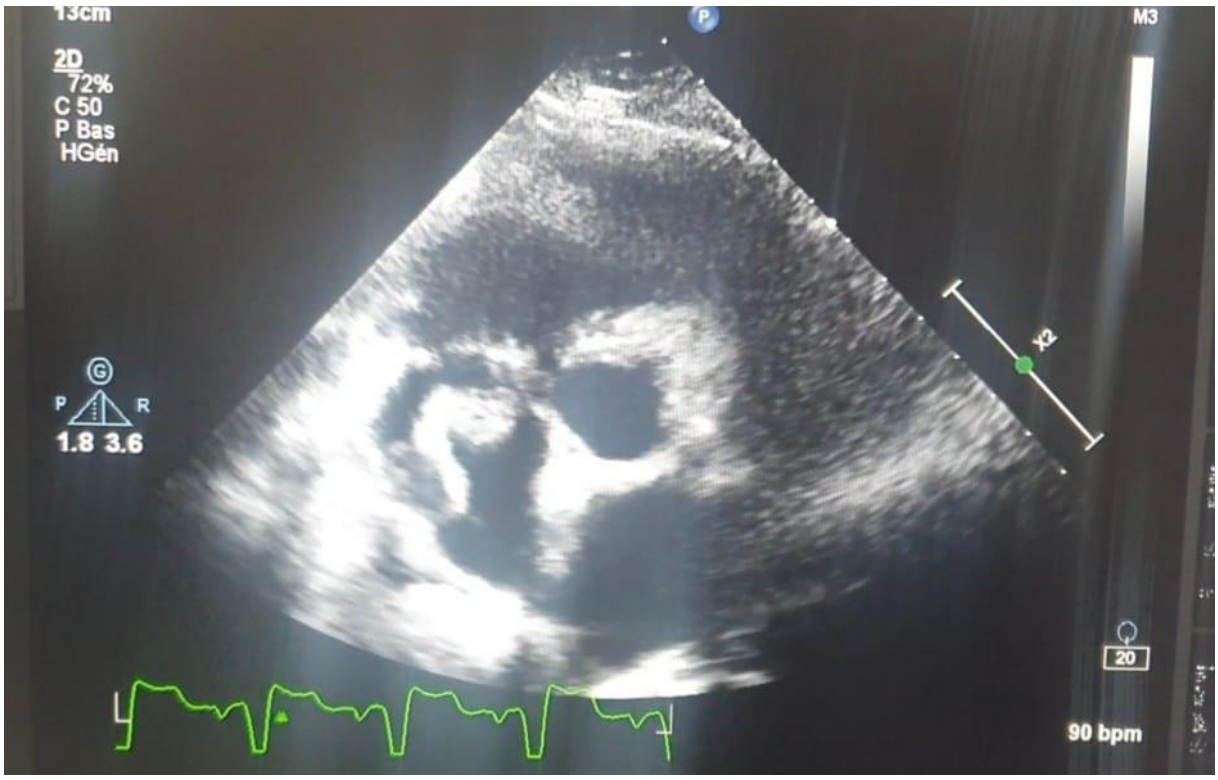
Rheumatoid arthritis has been associated with cardiac damage due to inflammation at all levels,1 mainly in the form of heart disease, but also due to conduction tissue damage with AVB2 and severe arrhythmia. When the heart block is complete, it usually leads to permanent pacemaker implantation which can complicate into infections and less commonly thrombosis (1)

CASE REPORT

A 38yo young women with a history of celiac disease and rheumatoid arthritis treated by corticoids and methotrexate, was admitted in the emergency room with episodes of syncope. An ECG was performed revealed a complete heart block. Blood tests showed no abnormalities. A trans-thoracic echocardiography showed normal sized cavities with a preserved function. Since the heart block was persisting, the patient underwent a pacemaker implantation.

3 months later, the patient presented two episodes of syncope ECG showed a pacemaker rhythm, A pacemaker interrogation was performed and revealed no conduction disturbance. The patient was monitored in the CICU but no heart block was detected. Methotrexate was suspected to be incriminated in stimulation threshold disturbance, it was decided to stop the medicine and the patient was released.

6 months later, the patient presented with electric shock sensation in response to emotional stress with no dyspnea nor syncope nor chest pain. clinical examination was normal. An ECG was unchanged. A transthoracic echocardiography was again performed and revealed a huge mobile mass on the pacemaker lead suggesting either a vegetation or a thrombus. There was no suspicious element nor regurgitation through the valves. C reactive protein and consecutive blood cultures were negative as well as serologic tests for indolent causes of endocarditis. A body CT-scan was performed with no embolic complication. An FDG PET scan did not show any uptake.



Parasternal short axis view showing a mass appended to the pacemaker lead.

Since there was no criteria for endocarditis and given the immunological profile, the patient was put on non fractionated heparin with close echocardiography follow-up that shows a regression of the mass.

DISCUSSION

The increased incidence of cardiovascular disease in patients with RA is well recognized. It usually results in premature development of atherosclerosis, myocardial infarction, and arterial stiffening. Congestive heart failure is likewise independently related to RA, possibly because of impairment of LV diastolic filling.

Arrhythmias had been related in RA especially conduction disturbances. These could be RBBB, hemiblocks or AV blocks of any degree. Complete AV block is rare, with an approximate incidence of 1 in 1000 patients with rheumatoid arthritis. Lesser degrees of block may precede complete heart block for varying periods, sometimes with intervening periods of normal conduction (1,2)

The most restrained causes of complete heart block in patients with rheumatoid arthritis are: a direct involvement of the conducting system with granulomas and subsequent fibrosis or extension of the inflammatory process from the base of the aorta or mitral valves to the conduction pathways. Complete heart block occurs generally in patients with established erosive nodular rheumatoid disease. It usually appears to be sudden and permanent, but progression from minor conduction delays is not uncommon. The characteristic histopathological finding is a rheumatoid granuloma in or near the AV node or bundle of His. (3)

It has been also hypothesized that a secondary amyloidosis or Haemorrhage into a rheumatoid nodule can be of cause. Whereas in some cases, a Coronary arteritis causing ischaemia of the conduction tissue or focal myocarditis due to RA1 and premature coronary artery disease due to accelerated atherosclerosis in patients with RA can complicate with heart block (3)

It can develop suddenly, being discovered after syncope or found unexpectedly on routine physical and electrocardiographic examination.

The management of complete heart block in this condition depends on the clinical features. As in our patient's case, if a symptomatic heart block occurs, the treatment of choice is a permanent pacemaker implantation (4) which may complicate with infection and less likely with thrombosis.

The incidence Venous thrombosis after pacemaker implantation occurs frequently, with a reported annual incidence of 23%, whereas the occurrence of thrombi on cardiac implantable electronic device lead remains extremely rare. The pathogenesis of thrombosis related to implanted devices remains controversial, as well as its clinical significance. An autopsy study reported right atrial PM lead thrombosis in 14% of the patients at 4 years after implantation(5). A recent study has shown that thrombi have been found on 48% of atrial leads and 33% of ventricular leads of cardiac devices in autopsies.

Several thrombus formation risk-factors have been reported, such as heart failure, atrial fibrillation, coagulopathies, thrombocytopenia, polycythaemia, and silicone pacemaker leads (6).

The long-term residence of permanent pacemaker leads may act as a continuous nidus for the formation of thrombus and may cause increased local thrombogenicity by causing endothelial trauma and by increasing blood turbulence like any other intravascular device. This may induce platelet aggregation and fibrin deposition, additionally, In cases of dual-chamber pacing multiple pacemaker wires, pacemaker leads may produce a foreign-body-type reaction with inflammation and fibrosis along the course of the wire .

Other factors associated with pacemaker lead-associated thrombosis include coagulopathies such as protein S and C deficiency,6 oral contraceptive use, heparin-induced thrombocytopenia, polycythaemia vera, the pacemaker material itself and fractured retained pacemaker lead(6,7).

These clots rarely have clinical manifestations such as right-sided heart failure, pulmonary embolism, shock, chest pain, malaise, cyanosis, peripheral oedema ,distended neck veins , and most often mimics infective endocarditis(7,8).Bidimensional transthoracic and transesophageal echocardiography are routinely used to visualize and characterize the mass. F-FDG PET/CT can be of help to exclude endocarditis .

No standard guidelines exist for the therapeutic options after the diagnosis of the clot attached to the cardiac device lead is made (6), based on the size of thrombus. Small sized thrombus can be managed with anticoagulation while larger clots may require additional therapeutic options, such as thrombolysis or a surgical approach (7).

CONCLUSION

Rhematoid arthritis may be complicated with serious cardiac manifestations including conduction disturbance. Complete heart block remains rare and usually managed by permanent stimulation when syncope occurs. Pacemaker implantation rarely complicates with cardiac and leads thrombosis .Anticoagulation is the treatment of choice of small clots whereas surgery may be necessary with larger ones.

REFERENCES:

1. Roman MJ, Salmon JE. Cardiovascular manifestations of rheumatologic diseases. Circulation. 2007 Nov 13;116(20):2346-55. doi: 10.1161/CIRCULATIONAHA.106.678334. PMID: 17998471.
2. Pandit A, Londhey V, Chawla B, Khedkar U, Sundaram S, Asgaonkar DS. Complete heart block in a case of rheumatoid arthritis. J Assoc Physicians India. 2013 Nov;61(11):836-8. PMID: 24974502.
3. Ahern M, Lever JV, Cosh J. Complete heart block in rheumatoid arthritis. Ann Rheum Dis. 1983 Aug;42(4):389-97. doi: 10.1136/ard.42.4.389. PMID: 6882034; PMCID: PMC1001249.
4. Ahern M, Lever JV, Cosh J. Complete heart block in rheumatoid arthritis. Ann Rheum Dis. 1983 Aug;42(4):389-97. doi: 10.1136/ard.42.4.389. PMID: 6882034; PMCID: PMC1001249.
5. Silva Marques J, Varela MG, Ferreira R, Nobre A, Almeida AG, de Sousa J. Intracardiac sterile pacemaker lead thrombosis. Rev Esp Cardiol (Engl Ed). 2012 Feb;65(2):193-4. English, Spanish. doi: 10.1016/j.recesp.2011.04.013. Epub 2011 Jul 23. PMID: 21783309.
6. Mohsin Ansari, Ameya Udyavar, Successful resolution of clots on the pacemaker lead by warfarin therapy, IHJ Cardiovascular Case Reports (CVCR), Volume 2, Issue 1, 2018, Pages 1-3, ISSN 2468-600X, https://doi.org/10.1016/j.ihjccr.2018.01.006.
7. Buttigieg J, Asciak R, Azzopardi CM. Pacemaker lead-associated thrombosis in cardiac resynchronisation therapy. BMJ Case Rep. 2015 Jul 7;2015:bcr2015210314. doi: 10.1136/bcr-2015-210314. PMID: 26153289; PMCID: PMC4499717.
8. Panagiotis MN, Nikolaos MP, St Georgia G, Athanasios KI. Case report: recurrent thrombosis of an old lead of a DDDR pacemaker mimicking lead infection. Eur Heart J Case Rep. 2018 May 26;2(2):yty063. doi: 10.1093/ehjcr/yty063. PMID: 31020141; PMCID: PMC6176961.
9. A RARE CASE OF PACEMAKER LEAD-ASSOCIATED THROMBOSIS IN AN ANTICOAGULATED PATIENT, DINA HAROUN, ABDUL RAHMAN AL ARMASHI, KANCHI PATELL, MOHAMED HOMEIDA, KEYVAN RAVAKHAH
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