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CASE REPORT

WHEN INFLAMMATION DISRUPTS CARDIAC CONDUCTION: COMPLETE HEART BLOCK IN ANKYLOSING SPONDYLITIS REQUIRING PERMANENT PACING

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Abstract

Introduction. Chronic inflammatory diseases are risk factors for cardiovascular diseases. We present the case of a patient with HLA-B27 positive ankylosing spondylitis associated with inflammatory myocardial involvement and transient complete atrio-ventricular (AV) block.

Case presentation. A 42-year-old man presented with a four-day history of generalized weakness and fatigue, occurring both at rest and during physical exertion. The patient had a history of HLA-B27 positive ankylosing spondylitis (AS) diagnosed in 2015 and treated with immunotherapy. The outpatient electrocardiogram (ECG) demonstrated a complete heart block, with a ventricular rate of 55 bpm, and narrow QRS complexes. Upon admission, ECG revealed sinus rhythm with first-degree AV block and narrow QRS complexes. Laboratory evaluation demonstrated elevated inflammatory markers. Echocardiography revealed dilation and calcification of the aortic root. A 48-hour Holter ECG monitoring demonstrated recurrent daytime episodes of transient complete AV block with narrow escape rhythm. The cardiac magnetic resonance imaging (MRI) showed myocardial edema, hyperemia, and non-ischemic late gadolinium enhancement

Résumé

Quand l'inflammation affecte la conduction cardiaque: bloc cardiaque complet dans la spondylite ankylosante nécessitant une stimulation cardiaque permanente

Introduction. Les maladies inflammatoires chroniques constituent des facteurs de risque de maladies cardiovasculaires. Nous présentons le cas d'un patient atteint de spondylarthrite ankylosante (SA) HLA-B27 positive, associée à une atteinte myocardique inflammatoire et à un bloc auriculo-ventriculaire (BAV) complet transitoire.

Rapport du cas. Un homme de 42 ans s'est présenté avec une asthénie et une fatigue généralisées évoluant depuis quatre jours, survenant au repos comme à l'effort. Ce patient avait des antécédents de SA HLA-B27 positive, diagnostiquée en 2015 et traitée par immunothérapie. L'électrocardiogramme (ECG) réalisé en ambulatoire avait mis en évidence un BAV complet, une fréquence ventriculaire de 55 bpm et des complexes QRS étroits. À son admission, l'ECG a révélé un rythme sinusal avec un BAV du premier degré et des complexes QRS étroits. Les analyses biologiques ont montré une élévation des marqueurs inflammatoires. L'échocardiographie a révélé une dilatation et une

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compatible with inflammatory myocardial involvement. Coronary angiography demonstrated no significant coronary artery disease. Because of symptomatic transient complete AV block and persistent conduction abnormalities, a dual-chamber rate-responsive pacemaker (DDDR) was implanted.

Conclusions. This case highlights the importance of recognizing cardiac involvement in AS patients, in whom persistent inflammatory-fibrotic myocardial changes may lead to significant conduction anomalies requiring permanent pacing, even when AV block appears transient.

Keywords: ankylosing spondylitis, complete heart block, cardiac MRI, pacemaker, fibrosis.

List of abbreviations:
AS – ankylosing spondylitis
AV – atrioventricular
MRI – magnetic resonance imaging
ECG – electrocardiogram
DDDR – dual-chamber rate-responsive pacemaker
HLA-B27 – human leukocyte antigen B27

calcification de la racine aortique. Un enregistrement Holter ECG de 48 heures a mis en évidence des épisodes diurnes récurrents de bloc AV complet transitoire avec rythme d'échappement étroit. L'imagerie par résonance magnétique cardiaque (IRM) a révélé un œdème myocardique, une hyperhémie et un rehaussement tardif non ischémique au gadolinium, compatibles avec une atteinte myocardique inflammatoire. La coronarographie n'a pas mis en évidence de coronaropathie significative. En raison du bloc AV complet transitoire symptomatique et des troubles de la conduction persistants, un stimulateur cardiaque double chambre à fréquence adaptative (DDDR) a été implanté.

Conclusions. Ce cas souligne l'importance de reconnaître l'atteinte cardiaque chez les patients atteints de spondylarthrite ankylosante, chez lesquels des modifications myocardiques inflammatoires et fibrotiques persistantes peuvent entraîner des troubles de la conduction significatifs nécessitant une stimulation cardiaque permanente, même lorsque le bloc AV semble transitoire.

Mots-clés: spondylarthrite ankylosante, bloc AV complet, IRM cardiaque, stimulateur cardiaque, fibrose.

Introduction

Ankylosing spondylitis (AS) is a chronic immune-mediated inflammatory disorder belonging to the spectrum of HLA-B27-associated spondyloarthropathies[1]. Cardiovascular involvement represents an important extra-articular manifestation associated with increased morbidity and mortality. Aortic root dilation, valvular disease and disturbances of the cardiac conduction system have been described as the most common cardiac complications in AS[1,2]. Conduction abnormalities in AS are thought to be the result of chronic inflammation and fibrosis affecting the interventricular septum and atrioventricular conduction system[1,2]. These inflammatory changes may promote myocardial remodeling and electrical instability. Although first-degree atrio-ventricular (AV) block is commonly observed, transient complete heart block remains an uncommon manifestation[3]. Cardiac magnetic resonance imaging (MRI) has become a valuable noninvasive technique for evaluating myocardial fibrosis and inflammatory changes[4]. Myocardial edema combined with non-ischemic late gadolinium enhancement may indicate active inflammation occurring on a background of chronic fibrotic remodeling, thereby contributing to the development of clinically important conduction abnormalities [4,5]. We report the case of a patient with HLA-B27 positive ankylosing spondylitis presenting with transient complete AV block and MRI evidence of inflammatory myocardial involvement requiring permanent dual-chamber pacemaker implantation.

Case presentation

A 42-year-old male presented to our cardiology clinic complaining of a four-day history of generalized weakness and fatigue, during both physical exertion and rest.

On his medical history, the patient had been diagnosed with HLA-B27 positive AS in 2015. Initial therapy consisted of methotrexate 2.5 mg (2×2 tablets weekly) for six months. Due to inadequate clinical remission, treatment was switched to infliximab 400 mg administered intravenously with induction dosing at weeks 0, 2, and 6, followed by maintenance therapy every eight weeks. In 2024, the patient was transitioned to etanercept 50 mg, administered subcutaneously every two weeks.

His family history was notable for AS in his brother, who was also HLA-B27 positive and receiving immunotherapy.

On physical examination, the patient was alert and hemodynamically stable. Cardiac auscultation was normal, and pulmonary auscultation revealed vesicular breath sounds bilaterally and no pulmonary crackles. The abdomen was soft and non-tender, with no peripheral edema. The musculoskeletal

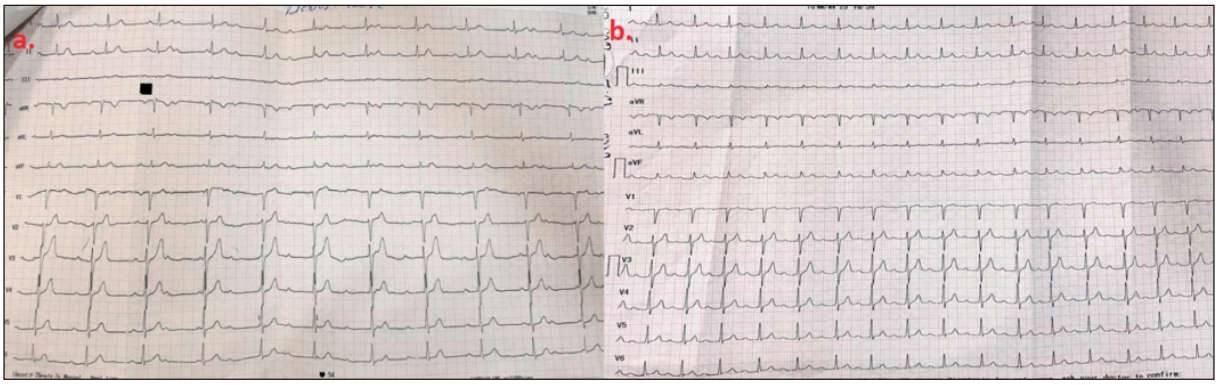


Figure 1. Electrocardiogram. a) ECG showing complete heart block with ventricular rate of 55 beats per minute, narrow QRS complex. b) ECG showing sinus rhythm, with a heart rate of 75 beats per minute, first degree atrio-ventricular block and narrow QRS complex.

Table 1. Laboratory work-up. Blood count analysis and biochemistry panel, elevated inflammatory biomarkers.

Complete Blood Count			
Parameters	Reference range	Units	Patient's values
RBC	4-5.5	X 10^6/uL	5.35
HCT	36-52	%	47.3
HB	12-16.5	15.9	13.7
WBC	4-10.5	K/uL	7.9
PLT	150-400	K/uL	336
Biochemistry Panel			
Urea	21-43	mg/dL	25.4
Creatinine	0.57-1.11	mg/dL	1
Na	136-145	mmol/L	138
K	3.5-5.1	mmol/L	4.8
Cl	98-107	mmol/L	100
Total bilirubin	0.3-1.2	mg/dL	0.48
ALT-SGPT	<55	U/L	22
AST-SGOT	5-34	U/L	16
CK	29-168	U/L	106
CK-MB	<5.2	ng/mL	0.5
Troponin-I	<0.016	ng/mL	**0.023**
NT-proBNP	<125	pg/mL	79.8
LDL-chol	<130	mg/dL	66
TG	50-180	mg/dL	87
CRP	<0.5	mg/dL	**2.83**
D-Dimer	200-400	mg/dL	0.33
Glucose	82-115	mg/dL	121
Fibrinogen	200-400	mg/dL	**517**

examination demonstrated loss of the normal cervical and lumbar spinal curvature.

Prior to admission, the outpatient electrocardiogram (ECG) demonstrated a complete heart block with a ventricular rate of 55 beats per minute and narrow QRS complexes (Figure 1a). At admission, resting ECG showed sinus rhythm with first-degree AV block and narrow QRS complexes, with a heart rate of 75 bpm (Figure 1b).

Laboratory work-up demonstrated an active inflammatory state, with elevated inflammatory markers, including C-reactive protein and fibrinogen, while complete blood count, renal function, liver enzymes, electrolytes, and cardiac biomarkers remained largely within normal limits, except for minimal troponin elevation, as summarized in Table 1.

Transthoracic echocardiography demonstrated normal left ventricular dimensions and preserved

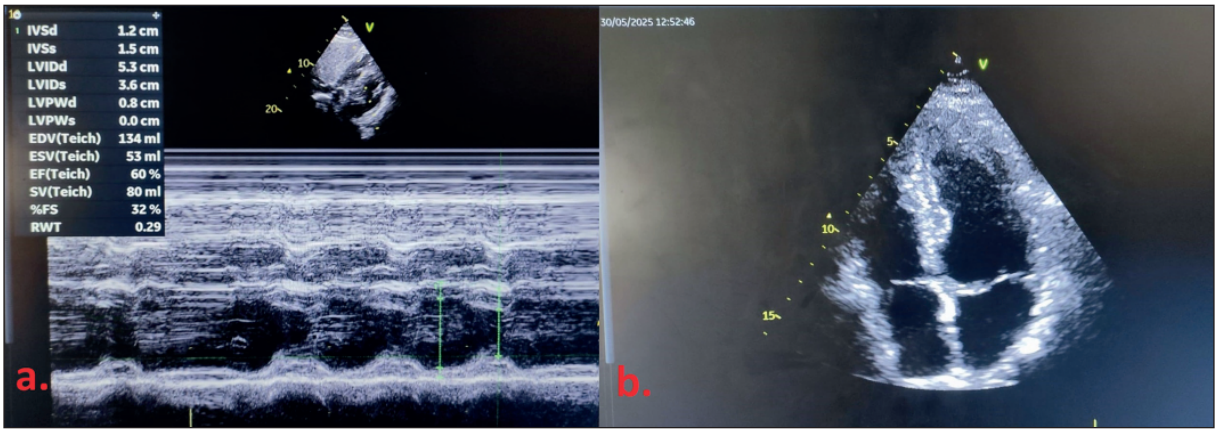


Figure 2. Echocardiography. a) Transthoracic echocardiography, M-Mode demonstrating preserved left ventricular systolic function. b) Transthoracic echocardiography, apical 4-chambers view showing septal hypertrophy.

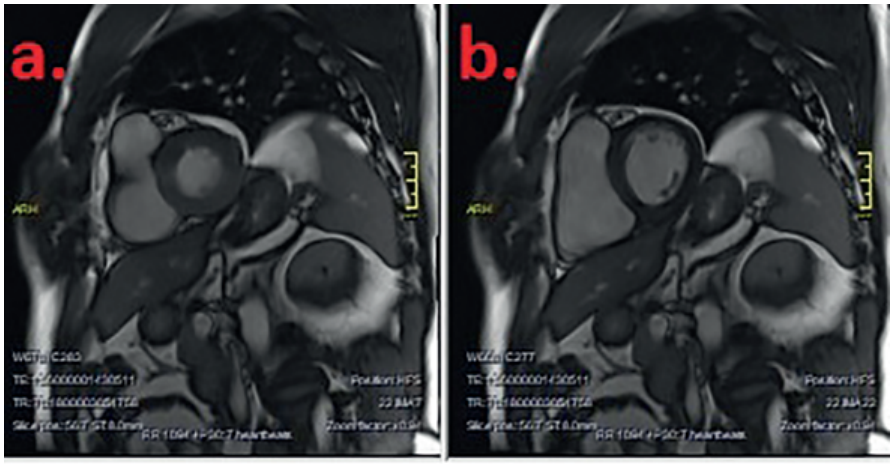


Figure 3 Cardiac MRI. a) Cardiac MRI demonstrating myocardial edema involving predominantly the interventricular septum and inferior wall, compatible with active inflammatory myocardial involvement. b) Cardiac MRI late gadolinium enhancement sequence demonstrating non-ischemic myocardial fibrosis involving the interventricular septum.

systolic function, without regional wall motion abnormalities (Figure 2a). Trace mitral regurgitation and minimal tricuspid regurgitation were present, with an estimated pulmonary artery systolic pressure of 25 mmHg. Apical 4-chamber view demonstrated septal hypertrophy, without pericardial effusion (Figure 2b). Dilation and calcification of the aortic root were noted.

A 48-hour Holter ECG monitoring demonstrated recurrent transient complete AV block predominantly during daytime hours, characterized by narrow QRS escape complexes with ventricular rates between 45 and 55 bpm. During nighttime hours, sinus rhythm with first-degree AV block predominated. No pauses longer than 2 seconds were observed. No supraventricular or ventricular arrhythmias were detected. The minimum heart rate was 45 bpm, average heart rate 67 bpm, and maximum heart rate 124 bpm.

Cardiac MRI demonstrated normal left ventricular and right ventricular volumes, with preserved global systolic function. Myocardial edema involving predominantly the septal, inferior, and lateral walls was observed, associated with hyperemia and minimal pericardial effusion (Figure 3a). Non-ischemic late gadolinium enhancement was present within the same myocardial territories, compatible with inflammatory myocardial involvement superimposed on chronic fibrosis (Figure 3b).

The coronary angiography demonstrated coronary arteries without significant fixed stenosis. A myocardial bridge involving the mid left anterior descending artery was identified.

Given the presence of symptomatic transient complete AV block associated with persistent conduction abnormalities and MRI evidence of inflammatory-fibrotic myocardial involvement, the arrhythmology team decided to implant a dual-chamber rate-responsive pacemaker (DDDR) (Figure 4a). The procedure was technically challenging because of the patient's loss of normal cervical and lumbar spinal curvature related to advanced ankylosing spondylitis, which limited

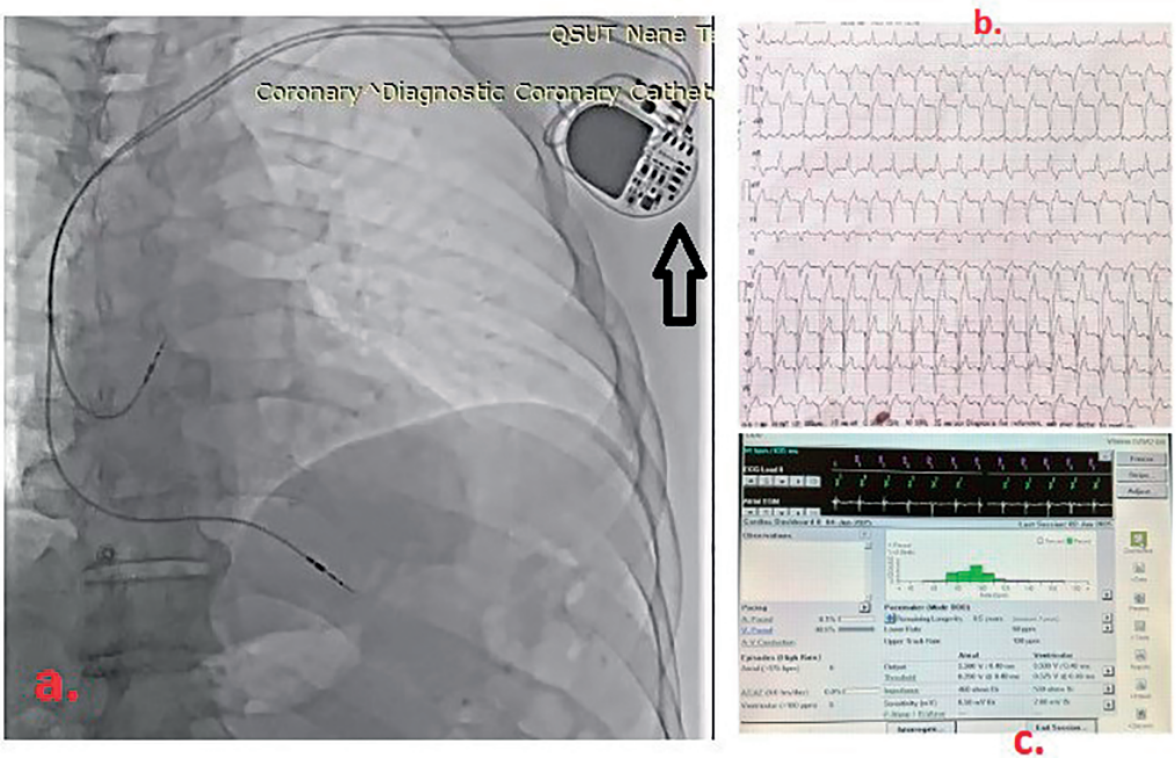


Figure 4. Pacemaker implantation and device interrogation. a) Chest fluoroscopy demonstrating dual-chamber pacemaker implantation, black arrow showing the device. b) Electrocardiogram showing ventricular paced rhythm after pacemaker implantation. c) Pacemaker interrogation demonstrating DDDR pacing with high ventricular pacing burden consistent with persistent atrioventricular conduction disease.

positioning during implantation. Post-implantation pacemaker interrogation demonstrated a high ventricular pacing burden (98.8%), consistent with persistent advanced atrioventricular conduction disease (Figure 4c). The patient had an uncomplicated post-procedural course and was discharged in improved clinical condition. Nebivolol 5 mg once daily was initiated. Follow-up with the arrhythmology and rheumatology teams was recommended.

Discussion

Cardiac involvement in AS is uncommon, but may significantly contribute to morbidity and mortality[6,7]. Chronic inflammation may extend into the interventricular septum and AV conduction pathways, leading to fibrosis and progressive conduction disease[7]. In our case, the transient complete AV block with a narrow QRS escape rhythm suggested predominantly supra-Hisian conduction disease.

Cardiac MRI is an important noninvasive tool for identifying myocardial inflammation and fibrosis in inflammatory diseases[8,9]. The coexistence of myocardial edema and fibrosis in our patient suggested ongoing inflammatory activity superimposed on chronic structural remodeling rather than a purely acute reversible myocarditis process. Echocardiographic findings of aortic root dilation and calcification further supported chronic cardiac involvement related to AS[6,7].

Although transient AV block may occasionally regress, persistent conduction abnormalities together with MRI evidence of inflammatory-fibrotic myocardial involvement supported the decision for permanent pacing[10,11]. Post-implantation pacemaker interrogation demonstrated a high ventricular pacing burden, confirming persistent clinically significant conduction disease.

This case highlights the importance of cardiovascular evaluation in patients with AS presenting with conduction abnormalities and emphasizes the role of permanent pacemaker implantation in preventing clinically significant bradyarrhythmias associated with persistent inflammatory-fibrotic conduction disease.

Conclusions

AS may rarely present with advanced AV conduction disturbances associated with inflammatory myocardial involvement. Cardiac MRI may provide important diagnostic and prognostic information by identifying active inflammation and chronic fibrosis.

Even when complete AV block is transient, permanent pacemaker implantation may be necessary due to persistent inflammatory-fibrotic involvement of the cardiac conduction system.

Author Contributions:

O.SH., E.SH., M.G., and A.GJ. contributed to the conception and design of the study, project supervision, provision of resources and materials, data collection and processing, analysis and interpretation of the results, and preparation of the manuscript. O.SH. and E.SH. performed the literature review. O.SH., E.SH., M.G., and A.GJ. contributed to writing the manuscript and critically reviewed it for important intellectual content. All authors have read and approved the final version of the manuscript.

Compliance with Ethics Requirements:

"The authors declare no conflict of interest regarding this article".

"The authors declare that all the procedures and experiments of this study respect the ethical standards in the Helsinki Declaration of 1975, as revised in 2008(5), as well as the national law. Informed consent was obtained from the patient included in the study"

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None

AI use disclosure:

The authors confirm that no artificial intelligence (AI)–assisted technologies (including large language models, chatbots, or image generation tools) were used in the preparation, analysis, or writing of this manuscript

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