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Acute electrocardiographic correlates post heart transplant in a non-compacted myocardium obese patient: A case report

Correlaciones electrocardiográficas agudas tras el trasplante cardíaco en un paciente obeso con miocardio no compactado: reporte de caso

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SUMMARY

This work describes a patient with non-compacted myocardium with a very low ejection fraction, type I obesity, with a history of atrial septal defect (ASD) correction, who underwent heart transplantation and the immediate electrocardiographic changes after the procedure. The immediate alterations included atrial fibrillation with a high ventricular response, bradycardia requiring pacing, and alterations due to right ventricular pressure overload (right bundle branch block). After the initial perioperative course, we observed good evolution after transplantation and clinical treatment. At the one-year follow-up, the patient remained without signs of rejection and with grade I functional capacity, free of cardiovascular events.

Keywords: *Heart transplant, electrocardiographic changes, atrial fibrillation, right bundle-branch block, arrhythmias.*

RESUMEN

Este trabajo describe un paciente portador de miocardio no compactado con fracción de eyección muy baja, obesidad tipo I, con antecedentes de corrección de comunicación interauricular (CIA), sometido a trasplante cardíaco y los cambios electrocardiográficos inmediatos tras el procedimiento. Entre las alteraciones inmediatas destaca la fibrilación auricular con alta respuesta ventricular, bradicardia

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con necesidad de marcapaso y alteraciones debidas a sobrecarga de presión ventricular derecha (bloqueo de rama derecha). Tras el curso perioperatorio inicial, observamos buena evolución tras el trasplante y el tratamiento clínico. En seguimiento al año, permaneció sin signos de rechazo y con capacidad funcional grado I, libre de eventos cardiovasculares.

Palabras clave: *Trasplante cardíaco, alteraciones electrocardiográficas, fibrilación auricular, bloqueo de ramo derecho, arritmias.*

INTRODUCTION

Heart transplantation is the treatment of choice in patients with end-stage heart failure (1). In the specific case of non-compacted cardiomyopathy, heart transplantation currently provides the highest survival rate in these patients (2). On the other hand, although the pathology is associated with genetic causes, recent meta-analyses show that it has the same heart failure prognostic degree compared to dilated heart disease, despite having a higher number of hospitalizations (3). The reversible cardiac rhythm alterations in the postoperative period have been described as signs of acute rejection, generally reverted with a steroid pulse and normalization of the right ventricle pressures (4). Among all arrhythmias, the authors describe a worse prognosis per year in patients who had previously atrial fibrillation before transplant (5). Moreover, no literature has been reported that includes perioperative electrocardiographic changes addressing this particular cardiomyopathy.

Case report

We present a male patient from Belém with a history of late surgical correction of an atrial septal defect (ASD) at 26 years, diagnosed in a hospital routine evaluation. At that time, he had an excellent postoperative course and reported improvements in his ability to make usual efforts, which he had previously interpreted as normal for his lifestyle. During a late follow-up, and forty-two years old, the patient again noticed dyspnea and edema in the lower limbs over the last twelve months. He sought a cardiologist in his region, who diagnosed heart failure according to New York Heart Association (NYHA) classification as functional class IV, difficult to control despite optimized medication, which at the time was sacubitril-valsartan 49/51 mg, furosemide 20 mg, spironolactone 25 mg, and carvedilol 25 mg twice a day.

Due to the continuation of symptoms, the patient sought treatment at the Beneficência Portuguesa Hospital for a more thorough assessment of the problem. An echocardiogram showed a significant increase of cavities, left ventricular (LV) diastolic diameter: 72 mm and systolic: 63 mm, left atrium: 50 mm, septum: 9 mm, and posterior wall 10 mm. Myocardial motility severely affected: diffuse hypokinesia of the LV with an ejection fraction of 27 %, systolic displacement of the tricuspid annulus (TAPSE): 12 ms and S' wave:7 cm/s, pulmonary arterial pressure: 60 mmHg, in the presence of significant tricuspid insufficiency, and moderate mitral insufficiency, with thrombus located in the right ventricle (RV) apical inferior wall and the apical portion of the LV basal inferoseptal wall, in addition to signs suggestive of non-compacted myocardium (ventricular trabeculation). There was a lack of familial history relationship with the cardiomyopathy.

A Magnetic Resonance Imaging (MRI) upon admission showed significant cardiac chambers enlargement, right ventricle (RV) systolic dysfunction (9 %) and left ventricular (LV) at 10 %, presence of interatrial septal patch with a residual flow, increased RV trabeculation and LV lateral wall trabeculation. Furthermore, thrombus in the apical-inferior wall of the RV, as well as the apical septal and apical-inferior walls of the LV. A cardiopulmonary test was also performed, with a VO_2 maximal of 14 mL/kg/min.

The patient initiated anticoagulant therapy and achieved therapeutic optimization. After 15 days of hospitalization and clinical improvement, the patient was discharged, however, had new hospital admission eight days later, requiring intensive care and inotropic support. In this new hospitalization, a transesophageal echocardiogram showed the absence of intracavitary thrombi, and the patient underwent right heart catheterization with vasoreactivity tests with sodium nitroprusside, obtaining a good response. We found a significant reduction in pulmonary hypertension (60->40 mmHg),

pulmonary capillary (32->18 mmHg), and a drop in almost 50 % of systemic vascular resistance: from 2.88 ->1.79 dyn.sec.cm^{-5}.

The patient remained in the intensive care unit since admission on Milrinone 0.5 μg/kg/min with difficult weaning and was evaluated by the heart transplant team, based on the clinical picture and complementary exams above, in conjunction with the approval perspective of the patient as the best path, indicated heart transplantation.

The transplant was performed on February 28, 2020, with a young donor organ, 18 years old, without previous disease, the victim of a car accident. Total ischemia time was 168 minutes (113 min cold and 55 min hot). In the immediate postoperative period, the patient evolved with a high ventricular response atrial fibrillation (reversed with amiodarone) and right bundle branch block, with deep S at V5-V6, lengthening the QRS complexes and denoting acute right ventricular overload (Figure 1).

Subsequently, the patient became pacemaker dependent due to severe sinus bradycardia but without hemodynamic instability (Figure 2). In the subsequent days, without further pacemaker need, the QRS complex narrowed, and the S on the precordial leads disappeared (Figure 3), coinciding with a drop in the pulmonary artery pressure from 24 to 19 mmHg and a cardiac output increase, from 4.6 to 6.1 by a Swan-Ganz catheter assessment.

The pathological examination confirmed the hypothesis of non-compacted myocardium with involvement of the lateral and posterior walls of the left ventricle, more evident in the apical half, and mural thrombosis in the organization process in the anteroseptal wall with accumulation of hemosiderin pigment, besides moderate myocardial hypertrophy. On March 12, 2020, the patient undergoes an endomyocardial biopsy (8 fragments), verifying the absence of histological and immunohistochemical findings or humoral rejection.

The patient was discharged from the hospital in good clinical condition after 38 days of transplantation, reversing the QRS narrowing and keeping a right branch block (Figure 4). After one year of a heart transplant and local clinical follow-up, the patient had no criteria for organ rejection or other cardiovascular events, evolved in NYHA functional class I, and was referred to his original city for further continuous health monitoring.

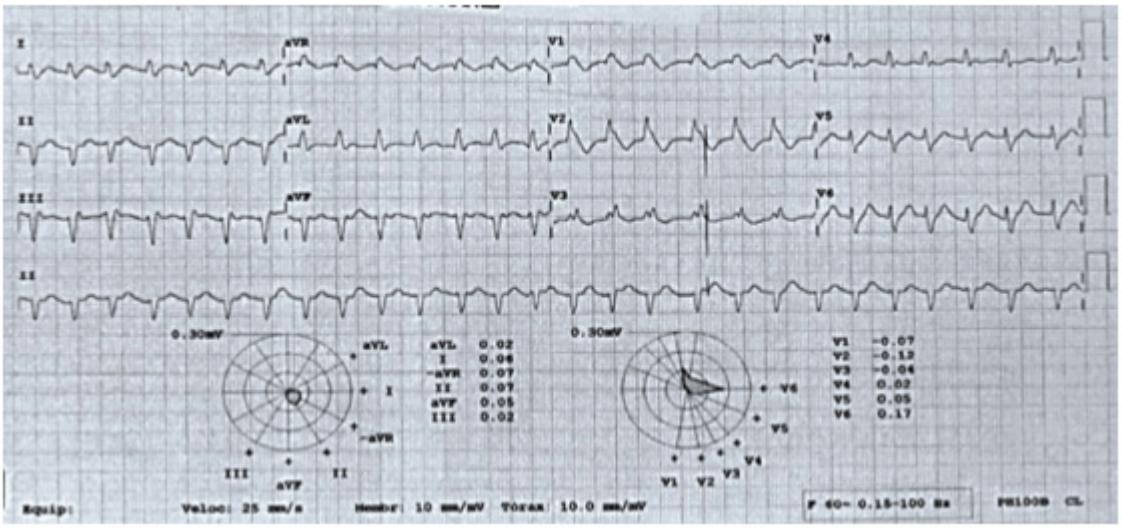

Figure 1. Immediate postoperative electrocardiogram showing atrial fibrillation.

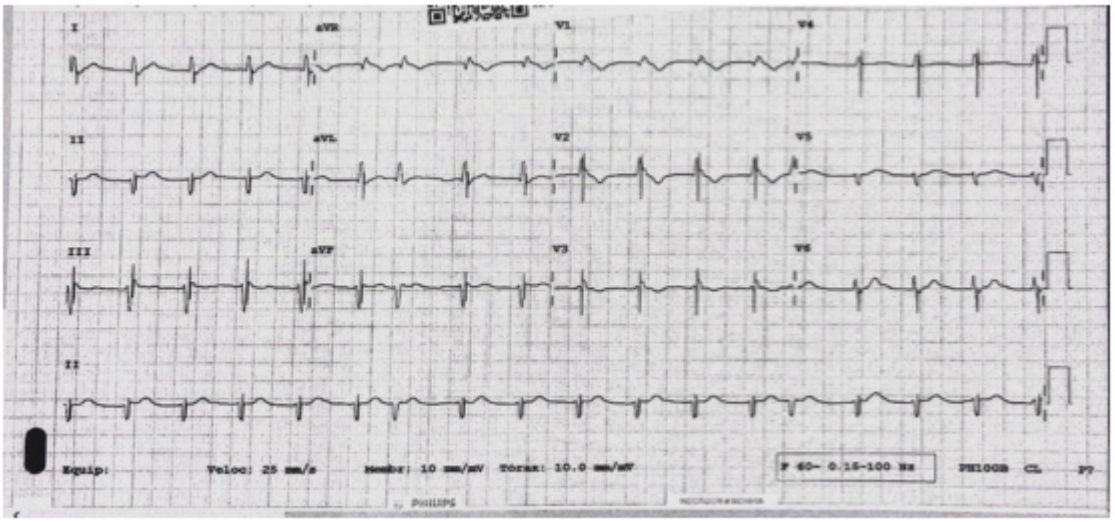

Figure 2. Electrocardiogram in pacemaker rhythm.

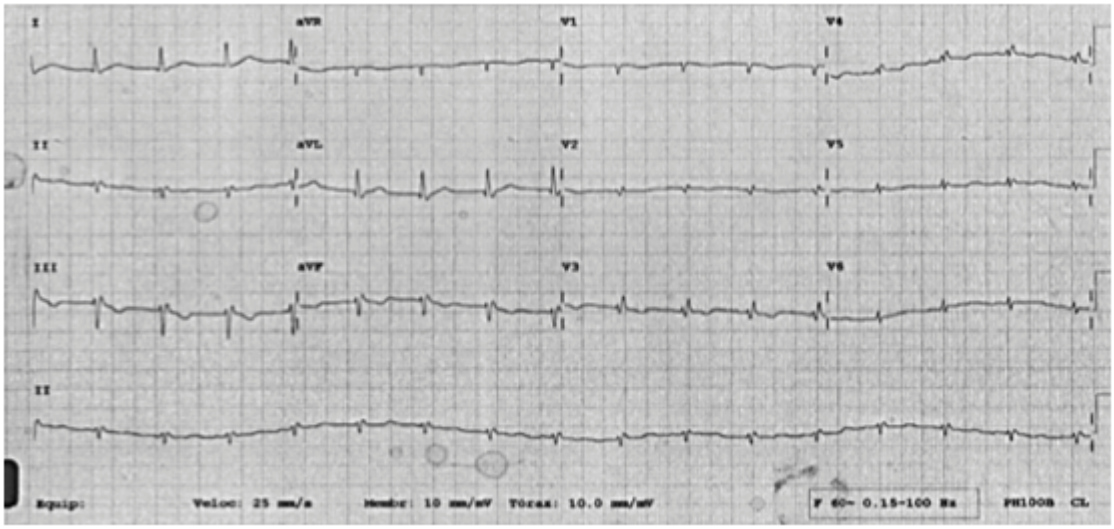

Figure 3. Electrocardiogram with QRS narrowing in concordance with pulmonary pressure fall.

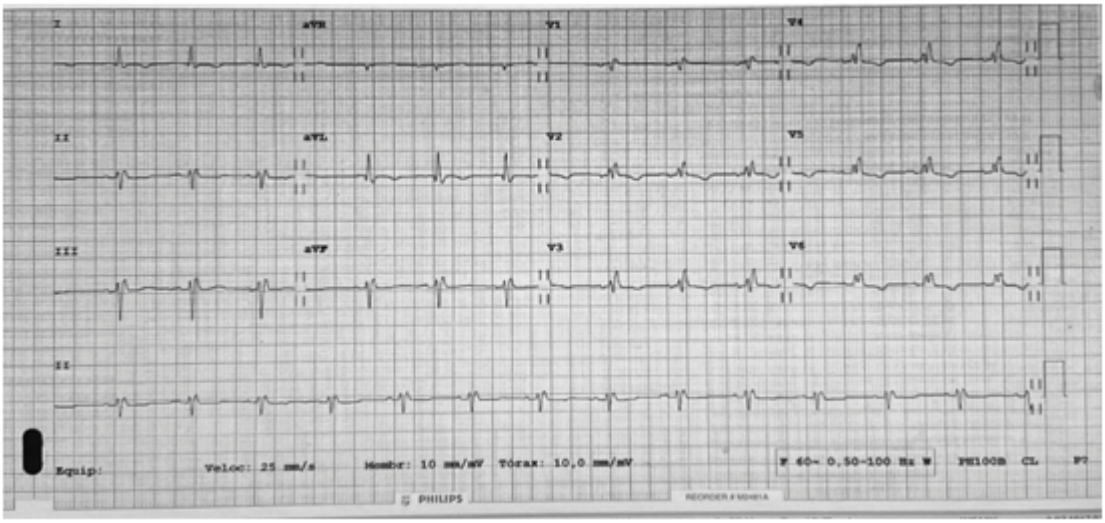

Figure 4. Electrocardiogram at the time of hospital discharge.

DISCUSSION

Uncompacted myocardium is a heart disease that can manifest with lethal arrhythmias, thrombotic events, and heart failure with treatment similar to other causes of heart failure from dilated myocardiopathy (6).

Heart transplantation remains the treatment of choice in patients with end-stage heart failure (1). In the peculiar case of non-compacted myocardiopathy, heart transplantation currently offers prolonged survival in these patients.

The abnormal regime where the native heart was in (pulmonary arterial hypertension, low cardiac index, and high pulmonary resistance), besides several drugs in use, and when the donor's heart faces this adverse regime, it suffers acute consequences, which was the reason for many deaths in the past including with false attribution to acute rejection when in reality the cause was acute right ventricular dysfunction and severe cardiac arrhythmias. With the improvement of the postoperative quality, the knowledge and training curve the better postoperative drug management, there was a significant reduction in these complications (7).

Electrocardiographic abnormalities, mainly rhythm disturbances and conduction abnormalities, are frequently observed after orthotopic heart transplantation, with right bundle branch block being the most frequent and not associated with adverse prognosis (8). The explanation for its occurrence is controversial, but researchers relate it to the position of the new heart (posterior rotation on its long axis), the result of the surgical technique, along some initial degree of right ventricular dysfunction (8,9).

There is an increase in bradyarrhythmia and atrioventricular blockages in the early postoperative period (10). Such changes are mainly secondary to sinus node dysfunction, usually transient and with a rare need for pacemaker implantation (11).

Tachyarrhythmias often occur in the first postoperative month of heart transplantation and are mostly supraventricular tachyarrhythmias, with atrial flutter as the most common (12,13). Some authors found no relationship between the prevalence of such arrhythmias with organ rejection, justifying their occurrence by the prolonged ischemia time of the graft, surgical trauma, antiarrhythmic drugs used preoperatively, cardiac denervation, and hypersensitivity

to catecholamines (12,13). However, other studies have correlated the occurrence of Atrial Flutter with acute rejection, justifying that the inflammatory process of the myocardium during rejection would cause conduction disturbances resulting in arrhythmogenic reentrant circuits (7,14).

Regarding pre-operatory risk in cardiac transplant, we point out some considerations in this specific case heart surgery antecedent could account for the patient having a long-term post-operatory period nonetheless, the shunt after surgery was residual. In another scenario, in obesity, a retrospective analysis of 4910 cases with type I obesity and undergoing orthotopic transplantation showed that it does not carry a higher mortality risk per year compared to other Body Mass Index (BMI) groups (15). However, all patients undergoing heart transplant programs must have their nutrition monitored.

In the reported case, the electrocardiographic alterations were diverse, such as right bundle branch block, atrial fibrillation, and bradycardia, with the need for a transitory pacemaker, fortunately without signs of rejection.

Heart transplantation is a life saving procedure and the gold standard in end-stage heart failure, especially in non-compacted myocardium. The measure benefits patients with reversible pulmonary hypertension, regardless of the cause.

Several comorbidities such as obesity, the type of underlying pathology, ischemia time, pulmonary hypertension, and vasoactive drugs, among others, are risk factors for the onset of arrhythmias and electrocardiographic changes in the immediate postoperative period.

The present case illustrates the electro-cardiographic alterations associated with the perioperative period of bicaval orthotopic heart transplantation in a patient with non-compacted myocardium and no association with acute graft rejection.

CONCLUSION

The various comorbidities such as obesity, the type of underlying pathology, ischemia time, pulmonary hypertension, and vasoactive drugs, among others, are risk factors for the appearance of these electrocardiographic changes in the immediate postoperative period. The identification of acute right ventricular dysfunction, its electrocardiographic correlate, and its correct management nowadays increase the efficiency of heart transplantation treatment, reducing mortality, which until then was attributed mainly to acute rejection processes.

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