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Persistent but reversible advanced atrio-ventricular block in a heart transplant man induced by dobutamine stress echocardiography☆

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ABSTRACT

Dobutamine when used for stress echocardiography (DSE), it rarely causes transient atrio-ventricular (AV) block. We report a heart transplant patient with high cardiovascular risk who developed symptomatic advanced AV block during DSE which persisted after termination of dobutamine administration, necessitating pacemaker implantation. To our knowledge, this is the first published case of persistent high grade AV block in a heart transplant patient induced by DSE.

Introduction

Dobutamine when used for stress echocardiography, it rarely induces temporary AV block, usually of first or second degree (incidence 0.6–1.1%). The main cardiovascular adverse reactions are: ventricular premature complexes, ventricular tachycardia, angina pectoris, hypo/hypertension and localized phlebitis [1–3].

Initially, the transplanted heart was thought to be anatomically and functionally denervated. However, since then, many studies have investigated autonomic reinnervation and in our days, sympathetic reinnervation is generally accepted, occurring usually after 1 year post transplantation [4]. Parasympathetic reinnervation seems to begin <1 year after transplantation when the bicaval method is used. Reconnection of the major branches of the vagus nerve may not be related to this early reinnervation [5]. In any case, further investigations are needed to identify the precise mechanism and location of early parasympathetic reinnervation. Atrio-ventricular conduction system electrophysiologic properties are similar pre and post heart transplantation [6]. The precise mechanism of AV block in heart transplants is not well known, however, transplant rejection, transplant coronary heart disease, endocarditis, sepsis, surgical or catheter interventional injury have been suggested as potential causes (Table 1) [7].

Case presentation

A 57 years old heart transplant man with high cardiovascular risk was admitted to our cardiology department for his annual evaluation. A DSE was programmed in order to exclude a coronary heart disease. He was heart transplanted 10 years ago for terminal dilated cardiomyopathy due to alcoholism. He was active smoker, he had arterial hypertension and hypercholesterolemia. His medical history included hepatitis B at the age of 20 years old, left ear squamous cell carcinoma, genital herpes, bilateral kidney cysts, gingival condyloma, appendectomy, surgical excision of chest wall schwannoma (9,5 years ago) and depression. His treatment consisted on cyclosporine 100 mg (morning) - 150 mg (evening), mycophenolic acid 1500 mg 2 times per day, methylprednisolone 2 mg per day, pravastatin 20 mg per day, amlodipine 5 mg per day and alprazolam 0,5 mg per day.

During DSE at the dose of 40 μg/kg/min, he developed a high grade AV block with junctional and ventricular escape rhythm (Figs. 1, 2) that was accompanied by dizziness, somnolence and fatigue. Neither stopping of dobutamine infusion, nor atropine administration or isoprenaline infusion was effective. The advanced AV block persisted.

His blood pressure was 96/50 mmHg and heart rate 35 bpm (despite continuous intravenous infusion of isoprenaline). In the laboratory test, there was a slight neutrophilia (8100/mm^3, normal value: 1890–7200/mm^3), CRP (20 mg/l, normal value: <10 mg/dl) and uremia (61 mg/dl, normal value 15–40 mg/dl). The ion laboratory analysis was normal. During DSE there were no signs of ischemia, no thoracic pain and the regional heart contraction was normal, both at baseline and at 40 μg/kg/min of dobutamine (92% of maximum heart rate). The coronary angiography revealed moderate diffuse atherosclerosis without stenosis. A temporary pacemaker was placed via the right femoral vein. The amount of ventricular pacing was reduced from 100% (day 0 DSE) to 50% (day 2 post DSE). A permanent double chamber pacemaker was implanted 2 days after DSE without any complication (Fig. 3). The evolution of the patient was excellent and he was discharged 48 h after pacemaker implantation.

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Table 1

Potential causes of AV block in heart transplant patients [7]:
- Cellular or humoral transplant rejection
- Transplant coronary heart disease
- Surgical or catheter interventional injury
- Endocarditis
- Sepsis

The amount of pacing 12 months later was <1% and after 18 months was 0%. The advanced AV block was reversible.

Discussion

The P waves in the ECG of our patient are very small (Figs. 1, 2, 3). Orthotopic heart transplantation is related to significant electrocardiographic changes. P waves of donor heart are usually undetectable or tiny (small duration and amplitude) in first month after transplantation, they become bigger after 2 months and then stay relatively steady. P waves of recipient heart have a tendency to become smaller over time [8].

The incidence of AV block after heart transplantation is 10.8%, including incidence of first-degree AV block of 8.3% and second degree of 1.8%. However, the true incidence of post-transplant AV block may be underestimated, because only clinical and electrocardiographic (12 leads ECG snapshots, no ambulatory monitoring) follow-up is used after heart transplantation [7]. There are no predictable factors for its onset with median time to pacemaker implantation ranging from 35 months to 167 months (median of 70.5 months) [7,9]. The majority of patients requires <50% of ventricular pacing and the progression to permanent AV block is rare. The development of late onset AV block (>3 months) seems to not be related to donor or recipient characteristics [7,9]. Chronic right ventricular pacing could be a potential cause of accelerated transplant failure in pacing-dependent heart transplant patients [10].

The precise mechanism of AV block in heart transplant remains unknown. First-degree AV block after heart transplantation has been related to cellular or humoral rejection and transplant coronary artery disease. Second and third-degree AV block have been related to surgical or catheter interventional injury and have been also correlated with transplant coronary heart disease and rejection [7,11]. Allograft rejection

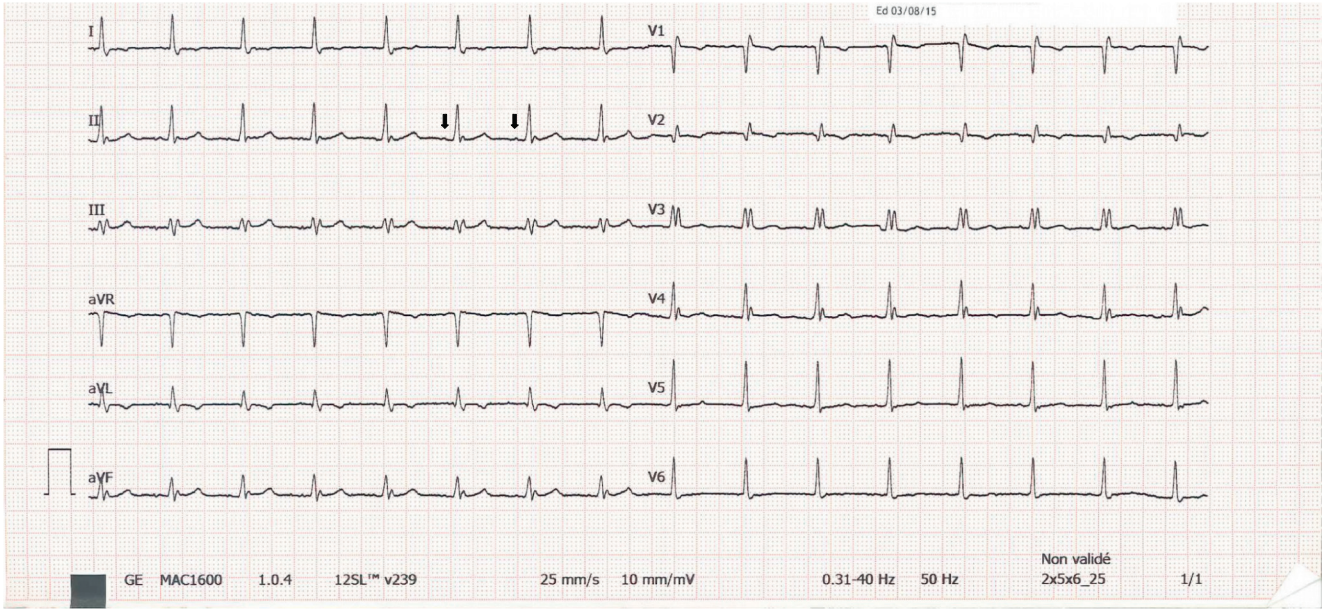

Fig. 1. ECG on admission. Sinus rhythm with incomplete right bundle branch block. (black arrow: p wave)

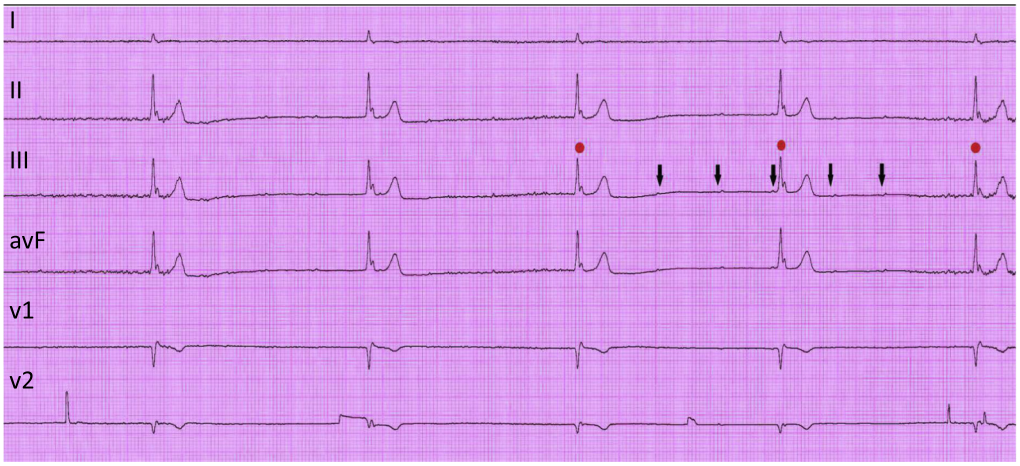

Fig. 2. ECG during dobutamine stress echocardiography (dose of 40 μg/kg/min). High grade AV block with junctional escape rhythm. (black arrow: p wave, red spot: QRS)