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Tako-tsubo cardiomyopathy following complete atrioventricular nodal heart block during transcatheter radiofrequency ablation of atrioventricular nodal reentrant tachycardia

Zespół tako-tsubo u chorego poddawanego ablacji częstoskurczu węzłowego powikłanej wystąpieniem bloku całkowitego przedsionkowo-komorowego

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Abstract

We report a case of tako-tsubo cardiomyopathy after an unintentional atrioventricular (AV) block during an ablation procedure in a 77 year-old woman. This intriguing case explores three possible reasons that could have triggered the disease: (1) slow pathway destruction; (2) AV nodal complete heart block; (3) the overall stress the patient had experienced.

Key words: tako-tsubo cardiomyopathy, catheter ablation, atrioventricular block

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INTRODUCTION

Non-ischaemic transient apical ballooning is a heart disorder that remains of interest to a broad spectrum of the medical world. This is not only because of its unique form of development, mimicking an acute coronary event, but also because of its unknown origin. It was first described by Sato et al. [1] and named tako-tsubo because of its resemblance to a Japanese fishing pot in ventriculography. Although a definitive cause has yet to be established, catecholamine-releasing stress seems to play a crucial role [2].

CASE REPORT

A 77 year-old woman with a history of longstanding hypertension, and a subtotal thyroidectomy as a result of nodular goitre, was admitted to our department for another atrioventricular nodal reentrant tachycardia (AVNRT) ablation approach. The patient had complained only of palpitations. No symptoms of heart failure, angina pectoris or any structural heart disease were confirmed during interview or initial examination. She was treated with aspirin, indapamidum and ramipril. A 24-hour Holter monitoring showed a narrow complex tachycardia at 180/min. Transthoracic echocardiography revealed a mild tricuspid valve dysfunction (RSVP = 35 mm Hg), but no ventricular hypertrophy or contraction abnormalities. Left ventricular ejection fraction (LVEF) was 55%. A 12-lead ECG showed sinus rhythm 66/min with inverted T waves in leads III, aVF and V1. Thyroid hormone blood levels were normal. During electrophysiological study, a typical slow-fast AVNRT was induced. Slow pathway ablation was performed using a 4-mm tip catheter (temperature controlled 55°C/30 W). A total of 24 applications (11 min) were delivered resulting in junctional rhythm during 11 applications. No arrhythmia was provoked 20 min after the last radiofrequency (RF) delivery. Eight weeks after discharge, the procedure was repeated because of recurrence of the arrhythmia. Another 25 RF applications (15 min) using a 4 mm tip

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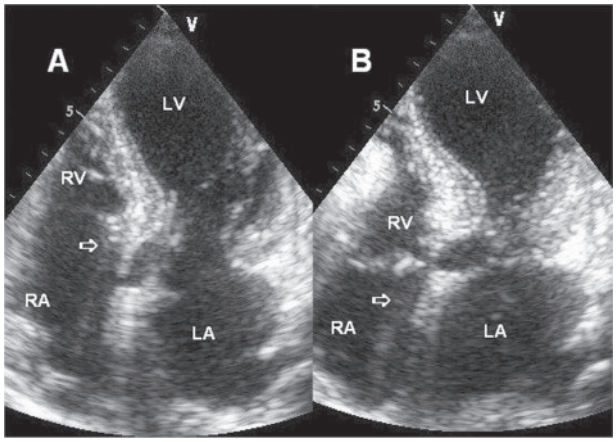

Figure 1. Transthoracic echocardiographic images in four-chamber apical view in diastole (**A**) and in systole (**B**) exhibiting akinetic dilated apical region and hyperkinetic basal part of the left ventricle (LV). In the right atrium (RA), and the right ventricle (RV), a pacing lead is visible (arrows); LA — left atrium

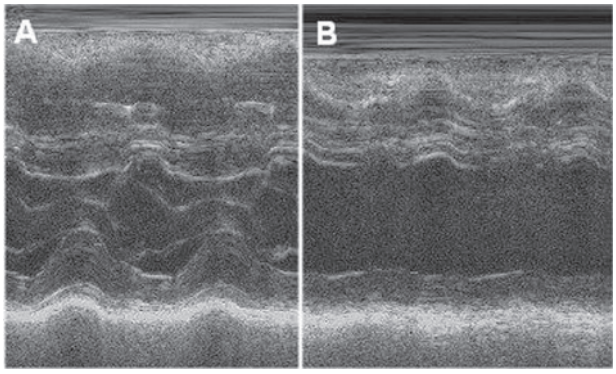

Figure 2. Transthoracic echocardiographic images in M-mode parasternal view exhibiting hyperkinetic movement of basal segments (**A**) and hypokinetic movements of middle segments of the left ventricle (**B**)

catheter (55°C/30 W) were delivered. No general anaesthesia or isoproterenol infusion were used (in either ablation procedure). While the junctional rhythm occurred, a gradual AV conduction delay was observed, resulting in a complete heart block. Though normal conduction reverted after approximately one minute, a permanent AV block was diagnosed the next day. A dual chamber pacemaker was implanted. No history of chest pain, shortness of breath or light-headedness was confirmed during the whole six days of hospitalisation.

The day after discharge, she came back to the emergency department complaining of generalised weakness, dyspnoea and moderate chest pain. Troponin I level was 2.37 ng/mL (normal < 0.1 ng/mL). Despite pacemaker stimulation, the ECG revealed a clear positive inversion of T waves in leads V5–V6 (negative in prior ECG examination). Echo imaging demonstrated isolated apical and midventricular akinetic segments with hyperkinetic basal segments. The LVEF was 35% (Figs. 1, 2). Selective coronary angiography showed minimal coronary artery disease. After seven days of conservative treatment, the patient became stable and asymptomatic with LVEF of 50%.

DISCUSSION

To the best of our knowledge, this is the first report of tako-tsubo cardiomyopathy after an unintentional AV block during ablation procedure. The combination of symptoms, blood test results and imaging findings described above allows us to diagnose tako-tsubo cardiomyopathy. This unusual case is associated with three possible reasons that could have triggered the disease: (1) slow pathway destruction; (2) AV nodal complete heart block; (3) the overall stress the patient had experienced.

Increased sympathetic activation is probably the main cause of most tako-tsubo cardiomyopathy cases. Furthermore, it is also closely associated with modifying target sites of most ablation procedures (AV nodal ablation, slow pathway ablation and destroying the left atrial autonomic ganglinated plexi ablation) [3–5]. The case described by Wadi Mawad suggests no correlation between time and number of applications in regard to tako-tsubo effect. The RF application time (26 min) in our case is comparable with the RF time delivered by Latacha et al. (22 min 46 s) [5]. However, Latacha et al. [5] did not use isoproterenol, and his patient experienced many stress factors (general anaesthesia, radial artery puncture described as very painful) which mean that we need to take into consideration ablation as the possible cause of tako-tsubo cardiomyopathy. On the other hand, these two cases bear an interesting similarity — accelerated junctional rhythm was present during a large number of applications (100% *vs* 41.6%).

In conclusion, tako-tsubo cardiomyopathy may occur as the result of modifying some ablation target sites, a possibility that should not be underestimated by electrophysiologists.

Conflict of interest: *none declared*

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