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Challenging diagnostic and therapeutic problems in a high-risk patient with mixed aortic valve disease and atrial tachycardia

Trudności diagnostyczne i terapeutyczne u pacjenta z grupy wysokiego ryzyka ze złożoną wadą aortalną i częstoskurczem przedsionkowym

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Abstract

We present the case of a 80-year-old female with multiple comorbidities, mixed aortic valve disease and heart failure decompensation in the course of atrial tachycardia. The introduced treatment with beta-blockers and subsequent heart rate reduction resulted in a negative haemodynamic response, as shown by elevated biochemical markers of renal and liver failure. This observation, together with clinical presentation, indicated pathophysiology of aortic regurgitation (AR) as the cause of this phenomenon.

This report shows the significance of clinical features in determining the severity of AR and the complexities surrounding the therapeutic decision-making process in a high-risk patient.

Key words: mixed aortic valve disease, atrial tachycardia, high-risk patient

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Introduction

The primary treatment of atrial tachycardia (AT) is rate control with beta-blockers or calcium channel blockers, and electrical cardioversion in cases of haemodynamic instability. However, in patients with structural heart disease, pharmacological treatment may lead to haemodynamic deterioration due to myocardial depression or peripheral vasodilation.

Moderate tachycardia improves cardiac performance in aortic regurgitation (AR) by reducing regurgitant volume and end-diastolic load without significantly reducing left ventricular (LV) filling volume [1, 2]. The opposite is the case in aortic stenosis (AS), where tachycardia shortens the time for optimal filling of stiffened LV.

Case report

We present the case of a 80 year-old female previously diagnosed with severe, asymptomatic AS and mild AR of bicuspid aortic valve (BAV) referred to our department from another hospital with heart failure decompensation in the course of AT. The patient had a history of diabetes mellitus, dyslipidaemia, smoking, stroke with left hemiparesis and mid stage vascular dementia, uterine fibroids, endometrial hyperplasia, and moderate anaemia [haemoglobin (Hb) 8 g/dL]. Coronary angiography showed stenosis of one, narrow (< 2 mm) artery.

On admission, the clinical condition of the patient was stable [New York Heart Association (NYHA) III] on dopamine infusion. Cardiovascular examination showed

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collapsing pulse, forcible apical impulse, basal harsh ejection systolic and blowing diastolic murmurs, and blood pressure 105/40 mm Hg. AT with irregular ventricular rhythm (140 bpm) (Figure 1) and left ventricular hypertrophy (LVH) were demonstrated by electrocardiography (ECG). Transthoracic and transoesophageal echocardiography showed severe mixed disease of BAV (valve area by planimetry — 0.7 cm^2, maximal velocity — 3.3 m/s, mean gradient 24 mm Hg) (Figure 2), mild mitral regurgitation, LVH with no LV enlargement or segmental asynergy, ejection fraction (EF) 60%, mild diastolic dysfunction, thrombus in left atrial appendage (LAA), no aortic dilatation or signs of infective endocarditis. The only significant difference between the current and previously performed echocardiograms was exacerbation of AR (Figure 3). However, quantification of AR was challenging in the presence of

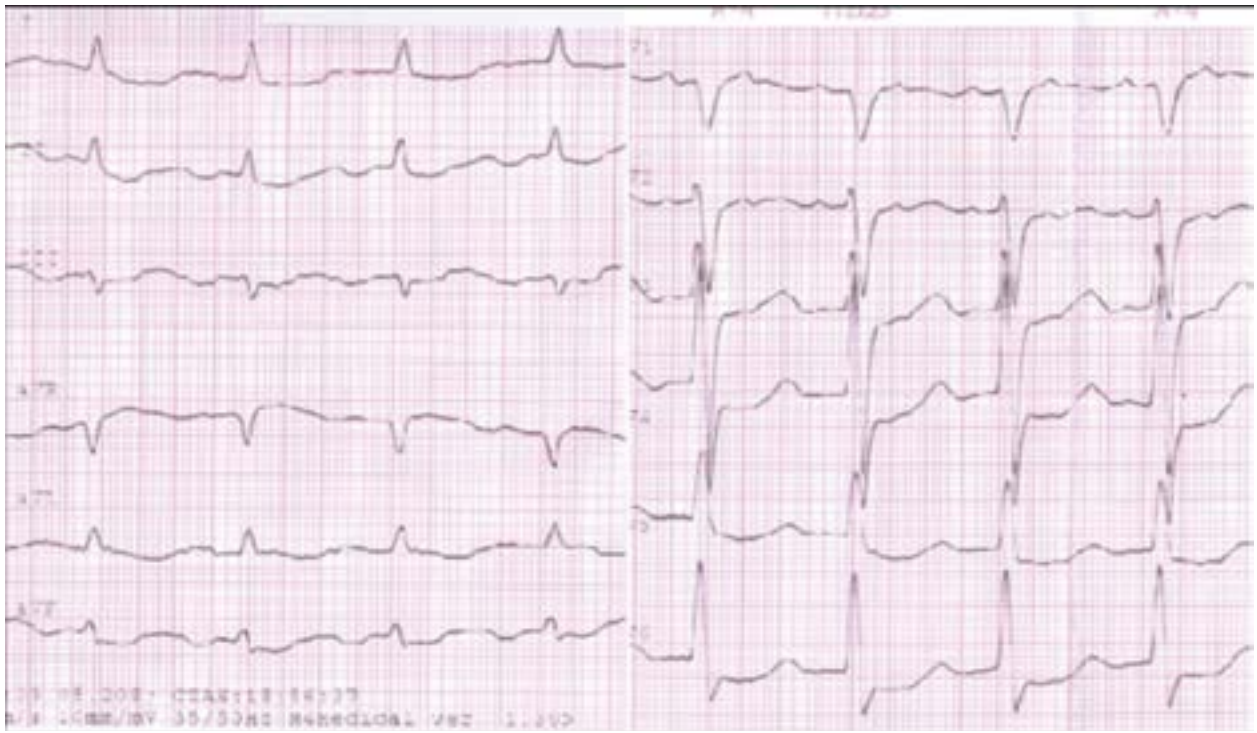

Figure 1. Electrocardiogram on admission — atrial tachycardia with ventricular rhythm 140 bpm (paper speed — 50 mm/s)

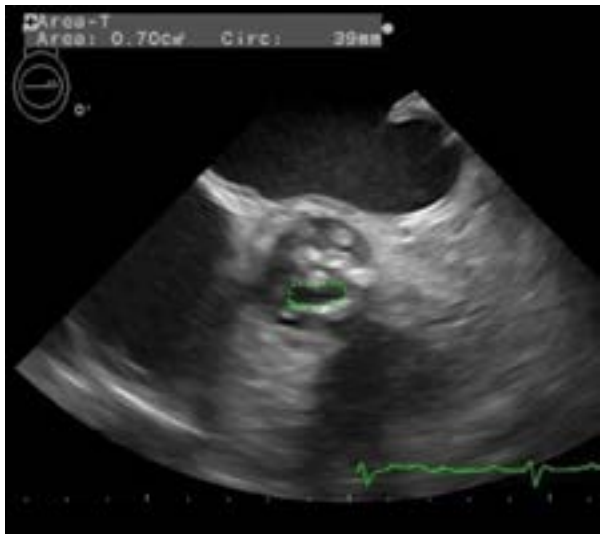

Figure 2. Mid-oesophageal short axis transoesophageal echocardiographic view at 0° showing aortic valve area

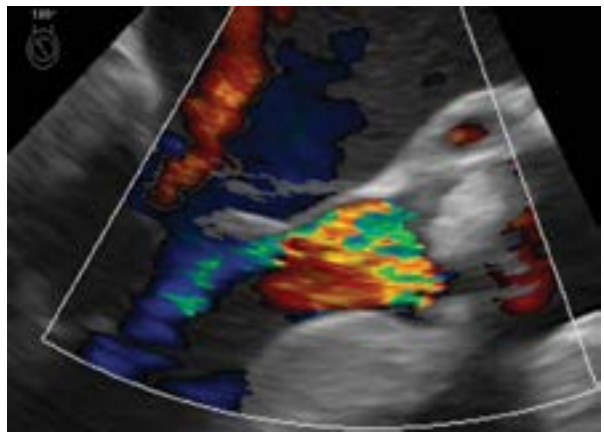

Figure 3. Mid-oesophageal long axis transoesophageal echocardiographic view at 130° showing aortic regurgitation

mixed valve disease and eccentric regurgitant jet typical for BAV and AT. Peripheral signs of AR indicated AR to be the predominant valve defect. Moreover, a negative haemodynamic response to heart rate reduction below 100 bpm with beta-blockers was observed, as shown by the elevation of biochemical markers of renal and liver failure. Pathophysiology of AR was assumed to be the cause of this phenomenon. The impact of negative inotropic action of beta-blockers on LV function seemed irrelevant on the basis of echocardiographic EF monitoring.

During hospitalisation, anticoagulation and diuretic therapy was continued while dopamine infusion was gradually stopped. Due to the low blood pressure, vasodilator therapy was unlikely to significantly reduce regurgitant volume and was not considered clinically beneficial [3]. Surgical or transcatheter aortic valve replacement was not selected by the Heart Team for this high-risk, low life expectancy patient. We considered cardioversion if the thrombus in LAA had resolved and further pacemaker implantation to control heart rate or transcatheter ablation of AT, but the patient refused any invasive treatment. Relative clinical improvement was achieved on beta-blockers, with a heart rate of 100–120 bpm.

Conclusion

This report is important on two counts. It shows the significance of clinical features in determining the severity of AR in mixed aortic valve disease and it illustrates the complexities surrounding therapeutic decision-making in high-risk patients [4].

Conflict interest(s)

The authors declare no competing interest.

Streszczenie

Opisano przypadek 80-letniej pacjentki obciążonej wieloma chorobami ze złożoną wadą aortalną oraz dekompensacją niewydolności serca w przebiegu częstoskurczu przedsionkowego. Zastosowane leczenie lekiem beta-adrenolitycznym i zwolnienie czynności serca spowodowało hemodynamicznie negatywną odpowiedź, na co wskazywały podwyższone wartości markerów biochemicznych nerek i wątroby. Ta obserwacja łącznie z obrazem klinicznym wskazała na patofizjologię niedomykalności aortalnej (AR) jako przyczynę powyższego zjawiska. Zaprezentowany przypadek ukazuje istotność objawów klinicznych w ocenie ciężkości AR oraz kompleksowego podejścia w procesie podejmowania decyzji terapeutycznych u pacjentów z grupy wysokiego ryzyka.

Słowa kluczowe: złożona wada zastawki aortalnej, częstoskurcz przedsionkowy, pacjent z grupy wysokiego ryzyka

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