BRADYCARDIA-TACHYCARDIA SYNDROME IN A POST-OPERATIVE CARDIAC SURGERY PATIENT

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ABSTRACT

The bradycardia-tachycardia syndrome is a condition characterized by episodes of supraventricular tachycardia followed by sinus bradycardia, resulting in significant heart rate variability. This syndrome can be associated with various cardiac abnormalities, including valve abnormalities and atrial dysfunction. The presented case is of a patient who sought emergency medical attention due to episodes of dyspnea with minimal exertion, orthopnea, and paroxysmal nocturnal dyspnea, associated with lower limb edema. The patient had a mechanical mitral prosthesis due to decompensated heart failure and prosthetic leaflet fracture, with three reoperations. During the physical examination, irregularities in the heart rhythm were observed, and a transthoracic echocardiogram (TTE) revealed moderate tricuspid regurgitation, moderate biatrial enlargement, and estimated pulmonary arterial hypertension of 80 mmHg. Continuous cardiac monitoring recorded prolonged pauses in the context of episodes of supraventricular tachycardia followed by sinus bradycardia, characteristic of bradycardia-tachycardia syndrome. Additionally, a normal ventricular ejection fraction, moderate biatrial enlargement, and pulmonary arterial hypertension were observed. Bradycardia-tachycardia syndrome can lead to hemodynamic changes and is associated with adverse cardiovascular risks. Treatment includes pharmacological control and, in some cases, lung transplantation. In this case, the patient received outpatient pharmacological treatment and underwent pacemaker implantation.

KEYWORDS: RADYCARDIA-TACHYCARDIA SYNDROME; TRANSTHORACIC ECHOCARDIOGRAM; TRICUSPID REGURGITATION; PULMONARY ARTERIAL HYPERTENSION

CASE REPORT

A female patient, 51 years old, with a history of heart disease, sought emergency medical services after mentioning that, over the past 14 days, she had been experiencing periods of dyspnea with minimal exertion, orthopnea, and paroxysmal nocturnal dyspnea, associated with lower limb edema. The patient had a mechanical mitral prosthesis due to decompensated heart failure, as well as a prosthetic leaflet fracture, for which she had undergone three reoperations.

During the physical examination, an irregularity in the cardiac rhythm was observed, and a transthoracic echocardiogram (TTE) was requested. The TTE results showed normal functionality of her mechanical mitral prosthesis and preserved biventricular systolic function. However, moderate tricuspid regurgitation, moderate biatrial enlargement, and an estimated pulmonary arterial hypertension of 80 mmHg were noted.

During the continuous 24-hour cardiac monitoring, a total of nine prolonged pauses were recorded, with an average duration of 6.2 seconds (Figure 1). These pauses were observed in the context of episodes of supraventricular tachycardia followed by sinus bradycardia, characteristic of bradycardia-tachycardia syndrome (Figures 2 and 3).

The patient reported that during the pauses, she experienced a sensation of imminent fainting, accompanied by dizziness and irregular palpitations. The episodes occurred unpredictably, significantly impacting her quality of life.

Figure 1. Bradycardia-tachycardia syndrome. Nine pauses are observed, with higher frequency between 5 and 6 am.

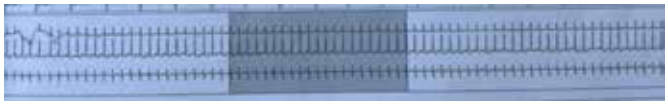

Figure 2. Bradycardia-tachycardia syndrome. A supraventricular tachycardia (SVT) is observed. The maximum recorded frequency in this segment is 156 bpm.

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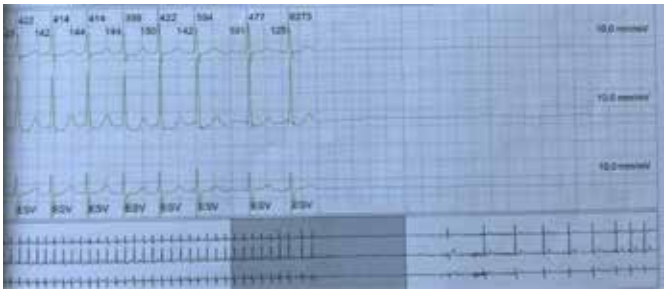

Figure 3. Bradycardia-tachycardia syndrome. In the three observed lines, there is a non-sustained supraventricular tachycardia (SVT) that stops spontaneously. In the first line of the record, a longer sinus pause of 6.2 seconds is observed, followed by sinus bradycardia, with a gradual recovery of the normal sinus node heart rate. The bradycardia is a result of the preceding tachycardia, which decreased the automatic activity of the sinus node.

In the TTE, the prolonged tracing of lead D2 revealed a supraventricular tachycardia that abruptly ceased, followed by a sinus pause and subsequent sinus bradycardia, with gradual recovery of the heart rate. Considering that bradyarrhythmia seemed to be the cause of the syndrome, the medical team diagnosed the patient with sinus node pathology characteristic of bradycardia-tachycardia syndrome and decided to admit her for further investigation of the arrhythmia and the implantation of a cardiac pacemaker.

Continuing with the results, the patient had mild prosthetic regurgitation. The patient's ventricular ejection fraction was 65%, with moderate biatrial enlargement. The normal systolic and diastolic diameters of the left ventricle were 30 mm and 47 mm, respectively, while in the right ventricle, the diastolic diameter was 27 mm. The patient had normal dimensions of the aortic root (31 mm), while the left atrium had a diameter of 47 mm.

After undergoing cardiac pacemaker implantation, the evaluated patient showed improvement in her overall condition. She was breathing normally without discomfort and was undergoing respiratory and motor physiotherapy.

DISCUSSION

The bradycardia-tachycardia syndrome is a condition characterized by episodes of supraventricular tachycardia followed by sinus bradycardia, resulting in significant variability in heart rate. This syndrome can be associated with various cardiac abnormalities, including valvular abnormalities and atrial dysfunction.[1] In this case report, the patient with bradycardia-tachycardia syndrome presented with moderate tricuspid insufficiency, moderate biatrial enlargement, and pulmonary arterial hypertension.

The moderate tricuspid regurgitation observed in this case indicates impairment of the tricuspid valve function, which separates the right atrium from the right ventricle. This type of regurgitation may be related to structural or functional changes in the valve, such as dilation of the valve annulus, rheumatic disease, endocarditis, or right ventricular dysfunction. Scientific literature has shown that moderate tricuspid regurgitation is associated with an increased risk of adverse cardiovascular events, such as heart failure, arrhythmias, and cardiovascular mortality.[2]

The moderate biatrial enlargement observed in this case suggests dilation of the right and left atria. Atrial dilation may be related to volume or pressure overload, as seen in cases of heart failure, valve diseases, pulmonary arterial hypertension, or arrhythmias. In the patient's case, her medical condition as a carrier of bradycardia-tachycardia syndrome may explain this event. Literature indicates that biatrial enlargement is associated with a higher risk of atrial fibrillation, thromboembolic events, and cardiovascular complications.[3]

A previous study by Choi et al.4 (2015) revealed that patients with bradycardia-tachycardia syndrome tend to have an ejection fraction greater than 63%, with manifestations of eccentric left ventricular hypertrophy and increased left atrium observed through transthoracic echocardiography. Cardiomegaly can also occur and is observed in chest radiographs of patients with bradycardia-tachycardia syndrome.

The patient in this case report had estimated pulmonary arterial hypertension of 80 mmHg, with an estimated pulmonary artery systolic pressure of 56 mmHg. Pulmonary arterial hypertension can result from various causes, such as heart disease, chronic lung disease, pulmonary embolism, or genetic diseases. Elevated pulmonary arterial pressure is associated with increased workload on the right side of the heart, leading to complications such as right heart failure and reduced survival.[5,6]

The bradycardia-tachycardia syndrome can lead to hemodynamic changes in the heart and blood vessels, contributing to an increase in pulmonary artery pressure. During tachycardia episodes, the heart may have difficulty pumping blood adequately to the lungs, resulting in an increase in pressure in the pulmonary arteries. Additionally, subsequent bradycardia can lead to a decrease in cardiac output, contributing to an increase in pulmonary arterial pressure.[6]

The treatment of pulmonary arterial hypertension generally involves specific pharmacological approaches, and in some cases, lung transplantation may be considered.[7] The patient in question underwent outpatient treatment with Losartan (50mg), Aldactone (25mg), Aspirin (100mg), furosemide (40mg), and metoprolol (50mg). Additionally, for antibiotic prophylaxis, cefuroxime (750mg) for three days, Tazocin, and Meropenem were prescribed.

According to studies, pharmacological control in patients with bradycardia-tachycardia syndrome can be used to regulate ventricular frequency. Medications such as amiodarone, diltiazem, verapamil, and digoxin may be prescribed, and atrioventricular nodal conduction may be delayed to adjust the ventricular response.[8,9] However,

since such treatments can worsen bradyarrhythmia, it is necessary to insert a pacemaker before attempting pharmacological control.[10] In cases where bradycardia is not a result of drug therapy (such as digitalis or beta-blockers), abnormal sinus node physiology should be considered.[4]

It is worth noting that the presence of bradycardia in atrial disease itself may predispose to the emergence of supraventricular tachycardia. The hemodynamic effects of bradycardia are more significant than those of tachycardia. It is important to emphasize that the supraventricular tachycardia that promotes bradycardia should not involve the use of antiarrhythmic medications, as these may more intensely suppress the dysfunctional sinus node. In the present case, the patient underwent the implantation of a cardiac pacemaker, as recommended by the literature.[7,10,11,12]

CONCLUSION

The report of this case allows us to conclude that bradycardia-tachycardia syndrome is a characteristic arrhythmia of sinus node disease. This case report highlights the importance of identifying and appropriately treating bradycardia-tachycardia syndrome, with therapeutic approaches such as pacemaker implantation and pharmacological therapy. The effective management of this condition can improve the patient's quality of life, reduce the risk of cardiovascular complications, and promote favorable clinical outcomes.

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