CASE REPORT

Syncope due to Paroxysmal Complete Atrioventricular Block in a Patient with Aortic Valve Stenosis

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Abstract

A 66-year-old man was admitted due to repeated syncope, and the electrocardiogram showed complete atrioventricular block (CAVB). He had moderate aortic valve stenosis (AS) with a severely calcified valve. This case indicates that if calcification spreads into the cardiac conduction system, it may cause CAVB. Although CAVB is not typically considered a main cause of syncope in AS patients, it should nevertheless be considered in the differential diagnosis.

Key words: aortic valve stenosis, complete atrioventricular block, degenerative calcified valve, syncope

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Introduction

The westernization of lifestyles influences the etiology of aortic valve stenosis (AS). Previous studies reported that rheumatic heart disease was a major cause of AS, but degenerative calcified valves due to atherosclerosis is a major cause of AS in the modern era (1). In addition to the westernization of lifestyles, the large aging population in Japan has led to an increased number of patients with arteriosclerotic disease. Because of the recent transition in AS pathophysiology, it is necessary to re-evaluate the causes of symptoms in AS patients.

Case Report

We herein describe the case of a 66-year-old man who was admitted to our hospital with a one-month history of syncopal episodes. He had type 2 diabetes mellitus and hypertension treated with an oral hypoglycemic agent and an antihypertensive agent, respectively. On admission, the patient’s consciousness was clear, and his vital signs were stable. The physical examination did not reveal any abnormal neurological findings. The patient’s electrocardiogram (ECG) showed a sinus tachycardia (heart rate 102/min) and atrial premature contractions. There were no findings of acute cerebral infarction on brain magnetic resonance imaging, and blood tests showed no hypoglycemia or electrolyte abnormalities.

After admission, the patient was followed with ECG monitoring in the intensive care unit. On the afternoon of the admission day, he developed a generalized convulsion. Paroxysmal complete atrioventricular block (CAVB) with pauses of more than 10 seconds was observed on ECG (Fig. 1). A temporary pacemaker was inserted for emergency treatment, and a permanent pacemaker was implanted the next day. On day 13 of the patient’s hospitalization, coronary angiography was conducted to evaluate ischemic heart disease, and a significant stenosis of >75% according to the American Heart Association guideline was observed in the left anterior descending artery, with mild stenoses in the right coronary artery and the left circumflex coronary artery. For the left anterior descending artery lesion, elective percutaneous coronary angioplasty was planned, and the patient was discharged on hospital day 15. His chest computed tomography (CT) scan showed a severely calcified aortic valve (AV) (Fig. 2), and transthoracic echocardiography revealed moderate AS with an AV area of 1.1 cm².

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Discussion

Three classical symptoms (angina, syncope, and dyspnea) play an important role in predicting the prognosis of patients with AS (2). The focus on the causes of AS has been shifting from rheumatic fever or the presence of a congenital bicuspid valve, to arteriosclerosis-induced degenerative calcification of valves. Along with this shift in focus, the number of patients with associated ischemic heart disease has increased. When AS patients present with symptoms of angina, physicians should consider symptoms of angina due to coronary artery stenosis, as well as imbalance of myocardial oxygen demand and supply from AS.

The changing pathophysiology of AS necessitates the reevaluation of the cause of syncope in AS patients. Low cardiac output, vagal reflex, or ventricular arrhythmia have been thought to trigger the main mechanism of syncope in AS patients (3), and the incidence is thought to depend on AS severity (4). However, syncope onset does not relate to AS severity when accompanied by CAVB, as in the present case.

The existence of AV calcification has been reported to trigger CAVB since the 1960s (5). If AV calcification in AS patients spreads as far as the His bundle around the non-coronary cusp and invades the cardiac conduction system, it might cause CAVB (6). The present patient had moderate AS, but the calcification in his AV and coronary arteries was substantial (Fig. 2). Therefore, the above-mentioned mechanism was assumed to have induced CAVB. However, in addition to calcification, CAVB can also be triggered by transient myocardial ischemia induced by decreased blood pres-
sure or coronary artery spasm.

The most common cause of AS is today considered to be degenerative valve calcification induced by underlying atherosclerosis. If AV calcification spreads into the cardiac conduction system, it may cause CAVB, as in the present case. CAVB has not traditionally been considered a major cause of syncope in AS patients, but it should be included in the differential diagnosis process in the modern era. When repeated syncope is observed, as in the present case, paroxysmal CAVB should be considered, and a work-up should include Holter ECG. Furthermore, if the cause of syncope cannot be identified using Holter ECG, the use of implantable loop recorders should be considered for further work-up.

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References

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