A case of right ventricular infarction due to acute Type A aortic dissection with anomalous aortic origin of the right coronary artery from the left sinus of Valsalva

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Abstract

Anomalous aortic origin of a coronary artery from the opposite sinus is a rare congenital condition that can cause sudden death in young people. When it is associated with acute aortic dissection, acute myocardial infarction can occur due to enlargement of the sinus of Valsalva. We report the case of a 71-year-old man with anomalous origin of the right coronary artery from the left sinus of Valsalva, who developed right ventricular infarction due to the compression of the right coronary artery between the aorta and pulmonary artery trunk.

Introduction

Anomalous aortic origin of the right coronary artery (AAORCA) from the left sinus of Valsalva is an uncommon condition that reportedly causes sudden cardiac death. It involves the compression of the coronary arteries due to dilatation of the aorta or pulmonary artery trunk. The enlargement of the sinus of Valsalva in acute aortic dissection (AAD) possibly led to acute myocardial infarction (AMI) by a similar mechanism, discussed below.

Case report

A 71-year-old man, who presented with dyspnea and severe back pain, was referred to the emergency department. His medical history included percutaneous catheter intervention (PCI) for AMI, caused by left circumflex artery stenosis 11 years ago. At that time, AAORCA from the left sinus of Valsalva was noted, but was followed up in the outpatient clinic without intervention.

On admission, his vital signs were stable, and all four limbs had adequate pulses. Laboratory data revealed no elevation of cardiac enzymes. An electrocardiogram showed inferior wall ST-elevation without bradycardia or atrioventricular block. Transthoracic echocardiogram revealed a wall motion abnormality in the right ventricle. Enhanced computed tomography (CT) showed a type A AAD that extended from the aortic root to the descending thoracic aorta with a thrombosed false lumen. The left coronary artery exhibited contrast enhancement, but the right coronary artery (RCA) did not (Figure 1). An emergency surgery was performed. Since there were only right ventricular infarction findings, the dissection presumably did not extend into the sinus of Valsalva. Rather, the compression occurred in the coronary artery after the RCA bifurcation.

Upon administering anesthesia, the patient developed ventricular fibrillation, which improved after 20 seconds of chest compressions. The surgery was performed via median sternotomy. Transesophageal echocardiogram showed mild to moderate aortic valve regurgitation. On surgical inspection, the sinus of Valsalva was enlarged, and right ventricular movement decreased. Cardiovascular bypass was established between the

left femoral artery and bicaval vena cava. Cardiac arrest was achieved by selective anterograde cardioplegia and retrograde cardioplegia. After circulatory arrest, the space inside the aorta was observed, and the absence of an entry in the aortic arch was confirmed. Thus, ascending aorta replacement was performed. The dissection cavity was glued using Bovine serum albumin-glutaraldehyde glue (BioGlue®), and proximal stump construction was done with inner and outer banded felts. After spontaneous circulation had returned, the right ventricular movement improved, and the procedure was completed without bypass surgery.

He was extubated on postoperative day (POD) 2 and recovered quickly. A coronary CT scan on POD 10 confirmed the absence of RCA stenosis or compression (Figure 2A and 2B). He was discharged without complications on POD 17. Coronary angiography (CAG) was performed one month postoperatively, but there was no RCA stenosis.

Discussion

Anomalous aortic origin of coronary artery (AAOCA) reportedly occurs in 0.2% to 1.3% of patients undergoing CAG and are often encountered in clinical practice. The prevalence of the AAORCA was reportedly between 8% to 16% in AAOCA It is approximately six to 10 times more common than anomalous aortic origin of the left coronary artery (AAOLCA). AAOCA from opposite sinuses can cause sudden cardiac death in young people. When it originates in the opposite sinus and travels between the aorta and pulmonary artery trunk, it can cause ischemia. Its mechanism has been attributed to the enlargement of the aorta and pulmonary artery trunk, which interferes with coronary blood flow, especially during exercise. Acute AAD is lethal and requires emergency surgery. AMI occurs in 1–2% of patients with AAD and is due to extrinsic compression of the coronary ostium by an enlarged false lumen or occlusion by an intimal flap. Bypass surgery is required in most cases.

In this case, AAORCA from the left sinus of Valsalva was observed preoperatively, and there were findings of right ventricular infarction. A compressed RCA between the enlarged sinus of Valsalva and pulmonary artery trunk was assumed to cause AMI. This was supported by the resolution of ischemia after careful stump construction. There have been few reports of AAD complicated by AMI exhibiting a similar mechanism.

The necessity of bypass surgery in this case was debatable. The expert consensus recommended surgical intervention for symptomatic AAOCA and asymptomatic patients with AAOLCA. However, surgery for asymptomatic patients with AAORCA remains controversial. The possibility of the future enlargement of the sinus of Valsalva was considered in this case. Thus, bypass surgery may have been performed. As soon as the surgery began, parts were harvested for the bypass already. However, considering the urgency of the surgery, the short operating time required, and the possibility of PCI if the sinus enlarged postoperatively, bypass surgery was not performed. As a result, the right ventricular movement normalized, and there were no abnormal findings on the postoperative CAG.

This case illustrated that AAOCA from the opposite sinus of Valsalva could become symptomatic due to AAD, and early intervention should be initiated, especially in individuals with a high risk for AAD, such as bicuspid valve or Marfan syndrome patients. AAOCA from the opposite sinus of Valsalva combined with acute AAD may lead to AMI by a new mechanism.

Figure legends

Figure 1. Preoperative enhanced CT shows enlargement of the false lumen and interruption of the blood flow in the RCA (arrow).

Figure 2A. Postoperative enhanced CT reveals the reduction of the sinus of Valsalva and blood flow in the RCA (arrow).

Figure 2B. In the 3-dimensional image, the RCA is confirmed to originate from the left sinus of Valsalva.

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