

An unusual cause of acute coronary syndrome: Left ventricular outflow tract pseudoaneurysm 🎥

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Introduction

Left ventricular outflow tract (LVOT) pseudoaneurysm is an uncommon but life-threatening pathology. It is generally observed after cardiovascular surgery or infective endocarditis. Its course ranges from an asymptomatic situation to severe complications including rupture, thromboembolism, fistula formation, and compression to the surrounding tissue (1-4). LVOT pseudoaneurysm usually presents with non-specific symptoms including chest pain, dyspnea, and cough. Until now, few cases with stable angina pectoris caused by LVOT compression on the left coronary arteries have been reported. However, in this case report, we present a patient with non-ST-elevation acute coronary syndrome caused by LVOT pseudoaneurysm.

Case Report

A 64-year-old male patient presented to our emergency department with newly perceived chest pain. He had undergone aortic bioprosthesis replacement due to infective endocarditis and single vessel coronary by-pass for second obtuse marginal artery stenosis eight months ago. The initial examination identified ongoing anginal chest pain along the mid-sternal region for

two and half hours. His blood pressure was 100/60 mm Hg, and his pulse was 70 beats/min. His cardiac troponin T blood levels were higher than normal. Thus, the patient was diagnosed with non-ST-elevation acute coronary syndrome and underwent emergency coronary angiography. During the coronary angiography, severe stenosis was detected in the left main coronary artery (LMCA), the left anterior descending (LAD), and the left circumflex (CX) arteries (Fig. 1a). We intended to perform coronary revascularization via a percutaneous coronary intervention. However, immediately after this procedure, we realized that there was a pulsatile variability in the severity of the coronary stenosis (Video 1) and a silhouette of a round-shaped mass under the aortic valve. Left ventriculography revealed an LVOT pseudoaneurysm (Fig. 1b and Video 2). Then, we selectively imaged the LVOT pseudoaneurysm (Fig. 1c and Video 3) to evaluate its suitability for percutaneous closure. The patient immediately underwent computed tomography (CT), which clearly revealed the nature of the pseudoaneurysm and its compressive effect on the LMCA, LAD, and CX (Fig. 2). He was given percutaneous or surgical closure options. The patient chose surgery, and unfortunately he died after surgery from sudden cardiac arrest.

Discussion

In this case report, we presented a patient with non-ST-elevation acute coronary syndrome caused by severe coronary stenosis due to the external compressive effect of an LVOT pseudoaneurysm.

The clinical presentation of LVOT pseudoaneurysms is mostly ambiguous and varies from an incidental asymptomatic pathology to severe but non-specific symptoms such as dyspnea, chest pain, and palpitation. Until now, stable angina pectoris, heart failure, ventricular tachycardia, complete atrioventricular block, cerebro-

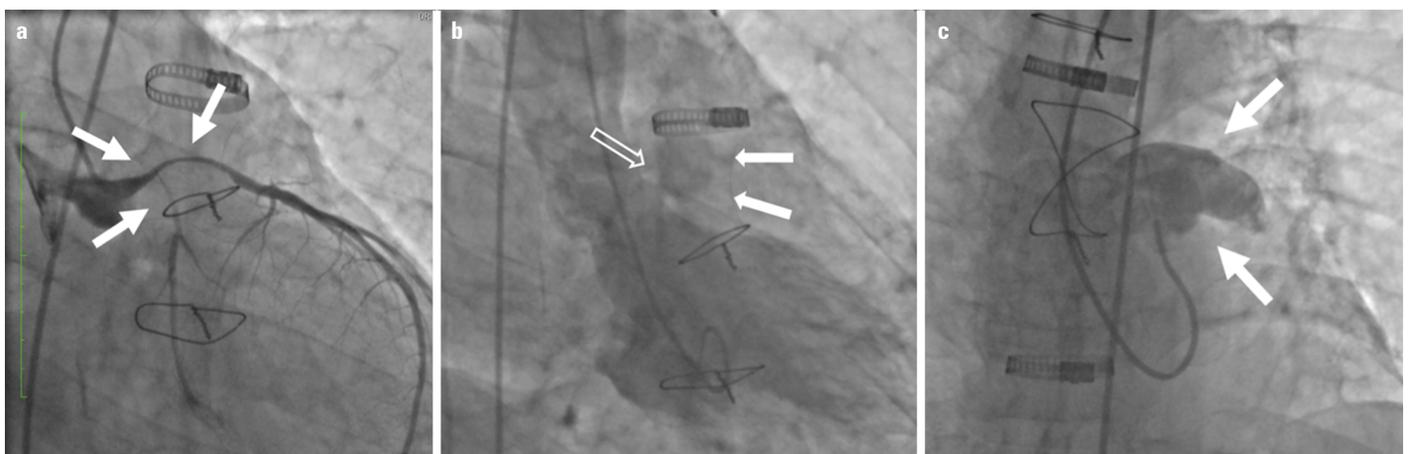


Figure 1. (a) Severe coronary stenosis in the left main coronary artery, left anterior descending artery, and left circumflex arteries on coronary angiography; (b) a left ventricular outflow tract (LVOT) pseudoaneurysm was revealed by left ventriculography; (c) the LVOT pseudoaneurysm was selectively imaged



Figure 2. A left ventricular outflow tract pseudoaneurysm and its compressive effect on the left coronary arteries are shown by computed tomography

vascular events, and prosthetic valve thrombosis have been reported to be associated with LVOT pseudoaneurysm (1-7).

A few cases in the literature have described LVOT pseudoaneurysms that presented with anginal chest pain (1, 2, 8, 9). All of these cases were associated with stable angina pectoris, except for the case described by Alani et al. (9). Cheng et al. (1) presented a patient with stable angina pectoris caused by dynamic left coronary artery compression of the LVOT pseudoaneurysm that developed from leptospirosis-induced myocarditis (1). Jha et al. (8) reported a patient who had dyspnea and stable angina pectoris for six months. Romaguera et al. (2) published a case that had stable angina pectoris because of severe LMCA stenosis caused by an LVOT pseudoaneurysm after aortic valve replacement. In this case, they performed a percutaneous stent implantation for LMCA stenosis but detected in-stent restenosis after four months. At that time, they discovered the presence of external compression on the LMCA. Later, they revealed the compressive effect of the LVOT pseudoaneurysm by CT. Finally, in a poster presentation, Alani et al. (9) presented a patient with intermittent fever and worsening exertional chest pain who had undergone prosthetic aortic valve replacement because of infective endocarditis associated with intravenous drug use. In this case, there was no angina at rest, and acute ischemic ECG changes occurred despite elevated troponin. In addition, they did not report whether the patient was considered to have acute coronary syndrome. The main differences between the present case and the previously reported cases can be summarized as follows: first, to our knowledge, this is the first case presenting with LVOT pseudoaneurysm-induced non-ST-elevation acute coronary syndrome; second, all of the left coronary tree, including the LMCA, LAD, and CX, was influenced by the LVOT pseu-

doaneurysm in this case. Therefore, we think our patient is the first in the literature with these conditions.

The development of coronary vasculopathy induced by prolonged repetitive mechanical compression can be proposed as a possible pathophysiological mechanism explaining the relationship between LVOT pseudoaneurysms and non-ST-elevation acute coronary syndrome. Both Saito et al. (10) and Renard et al. (11) reported on patients with cerebrovascular accidents caused by repetitive mechanical compression-induced carotid vasculopathy. Similarly, in this case, prolonged repetitive compression on the left coronary system may have caused a type of vasculopathy. In this situation, an additional trigger such as tachycardia and hypertensive attack may activate the vasculopathy plaque, which in turn starts a thrombotic process associated with acute coronary syndrome.

Finally, we selectively imaged the LVOT pseudoaneurysm with contrast media in this case. Notably, this imaging carries the risk of perforation. However, our case had non-ST-elevation acute coronary syndrome with ongoing chest pain, which requires urgent intervention. In this case, only coronary stenting would not have been sufficient to overcome the problem due to the pulsatile compressive effect of the LVOT pseudoaneurysm. Thus, we thought that the percutaneous closing of the LVOT pseudoaneurysm may eliminate its compressive effect on the coronary arteries, which in turn may relieve the coronary stenosis without additional coronary stenting. Both the pseudoaneurysm's suitability for percutaneous closure and the appropriate closure method such as coil embolization or an occluder device should be assessed before performing percutaneous closing. Therefore, we performed a selective imaging of the LVOT pseudoaneurysm despite its perforation risk. The risks of perforation should be considered when performing selective imaging of LVOT pseudoaneurysms.

Conclusion

LVOT pseudoaneurysms can cause acute coronary syndrome. It is extremely important to consider the presence of an LVOT pseudoaneurysm if there are any doubtful findings such as pulsatility in the severity of stenosis on coronary angiography, particularly in risky patients who have a history of cardiovascular surgery or infective endocarditis.

Informed consent: Written informed consent was obtained from the patient.

Video 1. Selective left coronary angiography showing severe pulsatile coronary stenosis on the left coronary arteries.

Video 2. A left ventricular outflow tract pseudoaneurysm was revealed by left ventriculography.

Video 3. A left ventricular outflow tract pseudoaneurysm was selectively imaged.

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Atrial fibrillation and atrial flutter ablation using mirror image in a patient with dextrocardia with situs inversus

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Introduction

Several studies have reported successful atrial fibrillation (AF) ablation with the help of intracardiac echocardiography (ICE) using different techniques in patients with dextrocardia with situs inversus (1-3). However, ICE may not always be available, as that observed in the present study. We successfully performed AF and atrial flutter (Afl) ablation in our patient using a 180° mirror image and performed catheter manipulations in the opposite manner as that of usual maneuvers. To the best of our knowledge, this is the first report that presents both AF and Afl ablations in the same session in a patient with dextrocardia with situs inversus.

Case Report

A 50-year-old man with dextrocardia with situs inversus was admitted for symptomatic, drug-refractory paroxysmal AF attacks that were persistent for 2.5 years (Fig. 1). After obtaining written informed consent, the patient was transferred to the catheter laboratory for AF ablation. At the beginning of the procedure, we changed the setting of the angiographic system (Artis zee, Siemens, Healthineers, Muenchen, Germany) and achieved a 180° mirror image. Thereafter, we inserted a 6-F decapolar catheter (St. Jude Medical) into the coronary sinus (CS) via the right internal jugular vein. We made a single transseptal (TS) puncture from the right femoral vein using a TS needle (BRK-1™, St. Jude Medical) through a 8.5-F TS sheath (SLO, St. Jude Medical). We performed the TS puncture in the opposite manner as that of usual catheter maneuvers in the same orientation as the normal fluoroscopic images. We directed the whole system medially and posteriorly by pointing the needle tip to the 10–11 o'clock position at the patient's leg in the anteroposterior view (a usual TS puncture requires a needle orientation at the 4–5 o'clock direction) (4). The interatrial septum was punctured by advancing the needle in the left anterior oblique view. Additionally, the TS sheath was replaced with a 12-F steerable sheath (FlexCath Advance, Medtronic Inc.) over the wire.

A second-generation 28-mm cryoballoon (CB) catheter (Arctic Front®, Medtronic CryoCath LP, Kirkland, Canada) was inserted into the left atrium (LA) and a spiral catheter (Achieve Advance™ mapping catheter 20 mm, Medtronic) was used both to maneuver the CB into the pulmonary vein (PV) and to record the PV signals. After entering the targeted PV with the spiral catheter, the balloon was inflated in the LA and directed to the PV ostium. An intravenous contrast material was injected through the central lumen of the balloon to confirm the complete closure of the PV ostium. A 180-s freeze cycle was performed for each vein (Fig. 2). If PV isolation was not achieved within 60 s after freezing, then the cycle was applied for additional 120 s. The diaphragmatic contraction was manually monitored for all the PVs using intermittent fluoroscopic imaging by pacing with another 6-F decapolar catheter (St. Jude Medical) placed in the