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CASE REPORT
Coronary heart disease

An unusual complication during alcohol septal ablation: severe left anterior descending artery vasospasm causing cardiac arrest: a case report and review of the literature

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Background Septal reduction therapy can be considered along the lines of hypertrophic obstructive cardiomyopathy patients who have drug-refractory symptoms. This can be applied either surgical myectomy or either alcohol septal ablation (ASA). Alcohol septal ablation has been performed successfully since the first announcement of ASA in 1995.

Case summary We present a case report of coronary artery vasospasm that occurred in the left anterior descending artery (LAD) during ASA. We performed ASA via first septal artery. Two cubic centimetre of 99% ethanol was slowly injected and 10 min later balloon was withdrawn. Then the patient felt severe chest pain; his systolic blood pressure went down quickly and fibrillated. We started the cardiopulmonary resuscitation (CPR). After CPR, the rhythm was achieved total 4 min later cardiac arrest but blood pressure was low. Emergent coronary angiography showed that coronary spasm caused severe occlusion in the LAD segment just after the first septal artery and impaired coronary flow nearly totally in the LAD just after septal artery. At that time, we decided to implant a stent due to the patient's serious condition and a 3.5 × 18 mm drug-eluting stent was implanted. We performed control angiography to patient 3 days later of the procedure and LAD flow was TIMI 3.

Discussion The causes of LAD occlusion are alcohol leakage, dissection, and vasospasm. It is important to detect the correct reason for appropriate treatment. Alcohol leakage impairs and causes coronary flow disruption; this can cause ventricular wall motion abnormalities. In our case, there was severe spasm in the LAD coronary artery and LAD flow was severely impaired. On echocardiogram, there was no myocardial wall motion abnormality. So alcohol leakage was ruled out. Left anterior descending artery image was not typical dissection. As a result of these findings, we concluded that the cause of LAD occlusion was coronary artery vasospasm.

Keywords Case report • Alcohol septal ablation • Coronary vasospasm • Cardiac arrest

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Learning points

- Periprocedural complications of alcohol septal ablation are death, cardiac tamponade, conduction abnormalities, malignant ventricular arrhythmias, and coronary artery occlusion.
- The frequency of complete occlusion of left anterior descending artery (LAD) during alcohol ablation is about 1–2%.
- The causes of LAD occlusion are alcohol leakage, dissection, and vasospasm. It is important to detect the correct reason for appropriate treatment.
- Coronary vasospasm can cause life-threatening arrhythmias and cardiac arrest. Therefore, stent and implantable cardioverter-defibrillator implantation can be required.

Introduction

Septal reduction therapy can be considered in patients with hypertrophic cardiomyopathy who have drug-refractory symptoms.[1] This can be applied either surgically (myectomy) or through percutaneous alcohol septal ablation (ASA). Surgical myectomy should be considered first-line treatment.[1] Alcohol septal ablation has been performed successfully since the first description in 1995.[2] Periprocedural complications of ASA are death ranging from 0% to 4%, cardiac tamponade, conduction abnormalities, and permanent pacemaker implantation nearly 10%, malignant ventricular arrhythmias 1–2%, and coronary artery occlusion 1–2%.[3–5] We present a case of coronary artery occlusion in the left anterior descending artery (LAD) during ASA.

Timeline

Time	Event
12 years prior to procedure	The patient had underwent surgical myectomy.
1 years prior to procedure	The patient started to feel dyspnoea and syncope.
4 months prior to procedure	Increasing burden of symptoms greatly affected the patient's daily life.
Time to procedure [alcohol septal ablation (ASA)] July 2018	We performed ASA, left anterior descending artery vasospasm occurred during ASA and patient had cardiac arrest. So we had to implant a drug-eluting stent and after stent the rhythm was achieved.
3 days later procedure	3° atrioventricular block occurred.
5 days later procedure	DDD-R implantable cardioverter-defibrillator was implanted.
6 days later procedure	The patient was discharged.
1 month later procedure	On routine control the patient was asymptomatic.

Case presentation

A 52-year-old man presented with exertional severe dyspnoea and syncope. He had underwent surgical myectomy 12 years ago. Echocardiography revealed normal left ventricular systolic function; interventricular septum diameter, 25 mm; left ventricular outflow gradient is 75 mmHg at rest, and 105 mmHg at provocation with systolic anterior movement of mitral valve. So we decided to perform ASA. Coronary angiography showed normal LAD and suitable first septal branch for intervention (*Figure 1A*; Supplementary material online, *Video S1*). A temporary pacemaker was placed into right ventricle. Left coronary ostium was engaged with a 6-Fr JL4 guiding catheter and a 0.014-inch floppy guidewire (Asahi Soft) was advanced to distal first septal artery. A 2.5 × 20 mm over-the-wire balloon (OTW) (Milvus, Conic Vascular, Switzerland) was advanced and inflated (*Figure 1B, C*; Supplementary material online, *Videos 2* and *S3*). The guidewire was removed from the balloon; subsequently, contrast material (nearly 2 ml) was injected into the septal artery from the distal part of the balloon (Supplementary material online, *Video S3*). After echocardiographic and angiographic control, we decided that the first septal artery was supplying the septal segment causing occlusion in the left outflow tract. Two cubic centimetre of 99% ethanol was slowly injected and 10 min later balloon was withdrawn. Then the patient felt severe chest pain; his systolic blood pressure went down quickly and fibrillated. We defibrillated the patient but the rhythm was not achieved; and we started the cardiopulmonary resuscitation. Then the patient was intubated. After cardiopulmonary resuscitation (CPR), the rhythm was achieved total 4 min later cardiac arrest but blood pressure was low. Emergent coronary angiography showed that coronary spasm caused severe occlusion in the LAD segment just after the first septal artery and impaired coronary flow nearly totally in the LAD just after septal artery (*Figures 1D, 2A*; Supplementary material online, *Videos S4* and *S5*). At that time, we decided to implant a stent due to the patient's serious condition. A 0.014-inch floppy guidewire was advanced to the distal LAD then 3.5 × 18 mm (Xience Pro, Abbott Vascular, Belgium) drug-eluting stent was advanced to the severe spasm segment without dissection and inflated at a nominal pressure at 10 atm (*Figure 2B, C*; Supplementary material online, *Video S6*). Then cine angiogram showed us that LAD flow was still nearly TIMI 2 probably due to the vasospasm after stenting (*Figure 2D*; Supplementary material online, *Video S7*). After intervention, we checked coronary balloon for any break because of any probability causing alcohol leakage into the LAD. And again, we controlled cineangiograms for any dissection; there was no coronary dissection image in the angiograms during viewing retrogradely.

Subsequently, the patient was monitored in coronary care unit. His peak CK-MB was 189 ng/mL and troponin I was 26 ng/mL. Left ventricular outflow tract (LVOT) gradient decreased to a maximum of 15 mmHg at provocation. We performed control

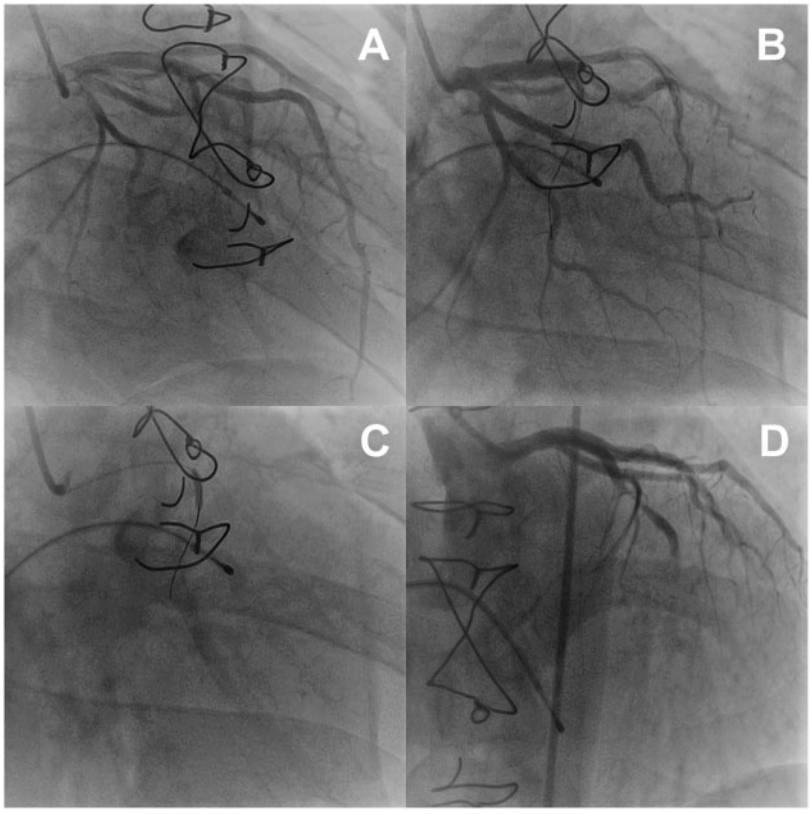

Figure 1 (*A*) Coronary angiography shows normal left anterior descending artery and suitable first septal branch for intervention on RAO view. (*B*) A 0.014-inch floppy guidewire and 2.5 20 mm over-the-wire balloon was advanced to distal first septal artery. (*C*) Over-the-wire balloon was inflated at nominal pressure at 6 atm. (*D*) Cine angiogram showed that coronary spasm caused severe occlusion in the left anterior descending artery segment just after the first septal artery and impaired coronary flow nearly totally in the left anterior descending artery on AP cranial view. AP, anteroposterior; RAO, right anterior oblique.

angiography to patient 3 days later of the procedure and LAD flow was TIMI 3 (Supplementary material online, *Video S8*). Complete atrioventricular block occurred 3 days later ASA and became permanent so we implanted a DDD-R implantable cardioverter-defibrillator (ICD) 5 days later after ASA. The patient was discharged 5 days later procedure. In his 1-month follow-up, he was asymptomatic and LVOT gradient was 15 mmHg at provocation.

Discussion

We present a case of severe spastic occlusion during alcohol septal ablation caused by stiff OTW balloon and resolved following coronary stent implantation. The frequency of complete occlusion of LAD during alcohol ablation is about 1–2%.[4–8] The common causes of LAD occlusion during ASA include alcohol leakage, dissection, and vasospasm. It is important to detect the correct reason for appropriate treatment. Alcohol leakage impairs and causes coronary flow disruption; this can cause ventricular wall motion abnormalities. In our case, there was severe spasm in the LAD coronary artery and LAD flow was severely impaired. On echocardiogram, there was no myocardial wall motion abnormality.[6–8] So alcohol leakage was ruled out. In previous reports, the most frequent reason was LAD dissection.[4–6] Coronary artery damage can be avoided increasing experience and using proper equipment.[4] Ziaee *et al.* reported a LAD dissection case.[6] They decided dissection because they administered intracoronary nitroglycerine and there was no effect but they did not perform intravascular ultrasonography (IVUS).[6] But in our case, we could not administer nitroglycerine and perform IVUS due to the cardiac arrest and LAD image was not typical dissection as well as after stent implantation LAD flow was nearly TIMI 2. As a result of these findings, we concluded that the cause of LAD occlusion was coronary vasospasm. Coronary artery vasospasm can cause life-threatening arrhythmias and cardiac arrest. Therefore, stent and ICD implantation can be required.[9–11]

In our case, vasospasm did not resolve so we had to implant a stent and after stent the rhythm was achieved and LAD flow was provided successfully. Alcohol septal ablation entails the risk of LAD occlusion distal to the septal branch, leaving a wire in the distal LAD for safety reasons can be a thoughtful choice. Now we strongly suggest leaving

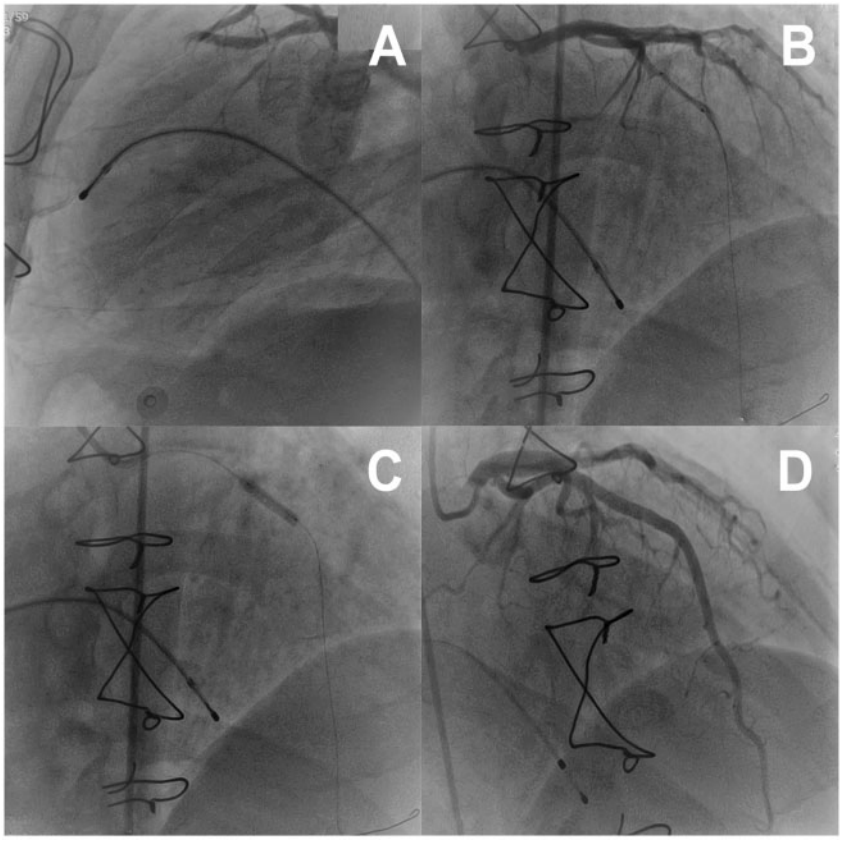

Figure 2 (*A*) Cine angiogram showed that coronary spasm caused severe occlusion in the left anterior descending artery on lateral view. (*B*) A 3.5 × 18 mm (Xience Pro, Abbott Vascular, Belgium) drug-eluting stent was advanced to the severe spasm segment. (*C*) A 3.5 × 18 mm drug-eluting stent was inflated at a nominal pressure at 10 atm. (*D*) After stent there was no dissection and left anterior descending artery flow was TIMI 3 on anteroposterior (AP) cranial view.

a second 0.014″ wire into the LAD in light of this case. In previous reports, only Alam *et al.* and Ziaee *et al.* mentioned about vasospasm but there are no reported cases during ASA in the literature.[5,6] To our knowledge, this is the first case that severe vasospasm was occurred in the LAD causing cardiac arrest during ASA, and stent implantation was required to improve coronary flow.

Conclusion

Alcohol septal ablation is generally safe and efficacious but rarely life-threatening complications can occur. This case emphasizes that interventional cardiologists can encounter severe vasospasm in the LAD during ASA. Vasospasm can cause life-threatening arrhythmias and cardiac arrest. Therefore, stent and ICD implantation can be required. Alcohol septal ablation entails the risk of LAD occlusion distal to the septal branch, now we strongly suggest leaving a second 0.014″ wire into the LAD in light of this case.

Lead author biography

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Supplementary material

Supplementary material is available at *European Heart Journal - Case Reports* online.

Slide sets: A fully edited slide set detailing this case and suitable for local presentation is available online as Supplementary data.

Consent: The authors confirm that written consent for submission and publication of this case report including image(s) and associated text has been obtained from the patient in line with COPE guidance.

Conflict of interest: none declared.

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