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Case Report

Alcohol septal ablation gone wrong- case report of a complication of hypertrophic obstructive cardiomyopathy treatment

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Abstract

Alcohol Septal Ablation (ASA) is an interventional procedure that involves injecting ethanol into a targeted septal artery, inducing localized tissue necrosis, and subsequently reducing septal thickness. Our experience highlights this method as an effective therapeutic intervention for symptomatic HOCM patients, providing relief from symptoms and enhancing cardiac function, reducing Left Ventricular Outflow Tract (LVOT) gradient. However, potential complications such as arrhythmia, Left Anterior Descending (LAD) dissection, cardiac tamponade, and coronary artery spasm are rare but necessitate careful patient selection and vigilant post-procedural monitoring. It is a reasonable therapeutic option especially in older patients with favourable coronary anatomy in the absence of mitral valve disease and if it is performed by an experienced operator.

We present a case of a 68-year-old man with Hypertrophic Obstructive Cardiomyopathy (HOCM), elevated LVOT gradient, and symptoms despite optimal medical therapy. He underwent alcohol septal ablation which was complicated by alcohol leakage likely via collateral circulation into the distal left anterior descending artery and obliteration of the vessel.

Abbreviations

HOCM: Hypertrophic Obstructive Cardiomyopathy; LVOT: Left Ventricular Outflow Tract; TTE: Transthoracic Echocardiogram; LVEF: Left Ventricular Ejection Fraction; IVS: Interventricular Septum; AICD: Automatic Internal Cardiac Defibrillator; ASA: Alcohol Septal Ablation; LAD: Left Anterior Descending; TIMI: Thrombolysis in Myocardial Infarction

Introduction

Hypertrophic cardiomyopathy is a genetic cardiac disorder characterized by excessive thickening of the heart muscle, resulting in impaired ventricular function and outflow obstruction [1]. Septal reduction therapy is reserved for patients who remain severely symptomatic despite optimized drug therapy and consists of two main options: surgical and percutaneous treatment. It is mainly indicated by both the American and European guidelines that myectomy is the primary treatment for HOCM with drug-refractory limiting symptoms due to LV outflow obstruction in the last 60 years [2]. ASA is a minimally invasive modality for the treatment of HOCM, compared to surgical myectomy. The first three cases of ASA were reported in the Lancet in 1995, by Sigwart, et al, and in 1998, Faber, et al. published a report in Circulation on selective septal myocardial ablation using Myocardial Contrast Echocardiography (MCE). MCE-guided ASA is now recognized as the standard method of PTSMA in many countries and

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regions, including Europe, North America, and Asia [3]. ASA has advantages over surgery such as faster recovery with much shorter hospital stay and avoidance of sternotomy, general anaesthesia, and cardiopulmonary bypass. It is mainly indicated by both the American and European guidelines by the Class I Recommendations for pharmacological management as the first step therapy of HOCM are the maximum dose of beta-blockers that are tolerated or nonhydropyridine calcium antagonists such as disopiramide and mavacamten and it is an initial strategy used to control symptoms of HF due to obstruction [1-3] The introduction of PTSMA into clinical practice has enabled the reduction of LVOT PG and improvement of heart failure symptoms in elderly and high-risk patients with symptomatic, drug-refractory HOCM. Our experience highlights ASA as an effective therapeutic intervention for symptomatic HOCM patients, providing relief from symptoms and enhancing cardiac function, reducing LVOT gradient.

Case presentation

A 68-year-old man presented with mild exertional chest pain, dizziness, and syncope. His blood pressure was 140/70 mmHg, his heart rate was 71/min, and on physical examination, he had a high-pitched systolic murmur over the whole precordium.

Medical history

The patient had a history of hypertension, hyperlipidemia, and diabetes mellitus.

Investigations

Transthoracic Echocardiogram (TTE) revealed preserved Left Ventricular Ejection Fraction (LVEF) of 55%, basal Interventricular Septum (IVS) diameter of 21mm and systolic anterior motion of anterior mitral valve leaflet. Left Ventricular Outflow Tract (LVOT) peak and mean gradients were 57 mmHg and 32 mmHg respectively at rest and increased to 86mmHg and 43mmHg with the Valsalva maneuver (Figure 1A, B). Cardiac magnetic resonance showed preserved LVEF of 64% and basal IVS diameter of 23 mm. There were fields of intramyocardial fibrosis of non-ischemic etiology in anterior, basal anteroseptal, and inferoposterior segments. The patient was diagnosed with Hypertrophic Obstructive Cardiomyopathy (HOCM) and was treated with a beta blocker and implantation of an Automatic Internal Cardiac Defibrillator (AICD) due to recurrent episodes of syncope and non-sustained ventricular tachycardia on Holter monitoring. He remained symptomatic with chest pain, dizziness, and poor exercise tolerance on the cardiopulmonary exercise stress test, so he was referred for an Alcohol Septal Ablation (ASA) procedure.

Management

The patient underwent an alcohol septal ablation procedure via right radial access. Due to the presence of AICD a temporary pacemaker did not have to be placed. We engaged the left coronary ostium with a 6-Fr EBU 3.5 catheter and advanced a 0.014'' coronary guidewire into the distal first Septal branch (S1) of the Left Anterior Descending artery (LAD). We then

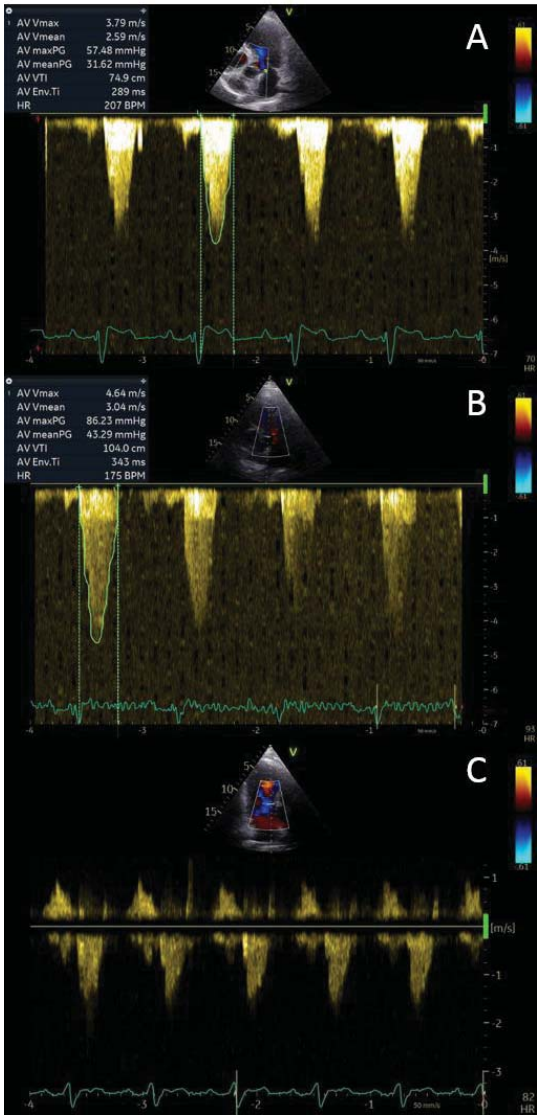

Figure 1: LVOT Doppler gradients. Images show LVOT gradients on continuous wave Doppler from the apical four-chamber view obtained in resting conditions (panel A), after Valsalva (panel B), and following ablation of the septum (panel C). LVOT- Left Ventricular Outflow Tract.

advanced a 1.5x8mm over-the-wire balloon into the S1 branch and inflated at 8 atmospheres. We removed the guidewire and injected echocardiographic contrast through the balloon. Opacification of basal IVS at the point of contact with anterior mitral valve leaflet was visualized (echocardiographic control), after which we injected radiographic contrast to distal S1 to confirm the absence of alcohol back leak into the LAD (angiographic control) (Video 1). This was followed by a slow injection over 5 minutes of 2.5 ml of 99% ethanol. TTE showed a significant reduction in LVOT gradient to a peak gradient of 21 mmHg. Following the injection of alcohol patient experienced chest pain and EKG changes which were expected, however, ten minutes after the alcohol injection control angiogram was made which showed occluded S1 and slow flow phenomenon in the distal segment of the LAD (TIMI 1-2) (Video 2). We placed a microcatheter into the distal LAD and administered 250 µg of intracoronary adenosine, 250 µg of verapamil, and 200 µg of sodium nitroprusside but distal LAD continued to have slow flow (TIMI 1). We gave a bolus of tirofiban and started an

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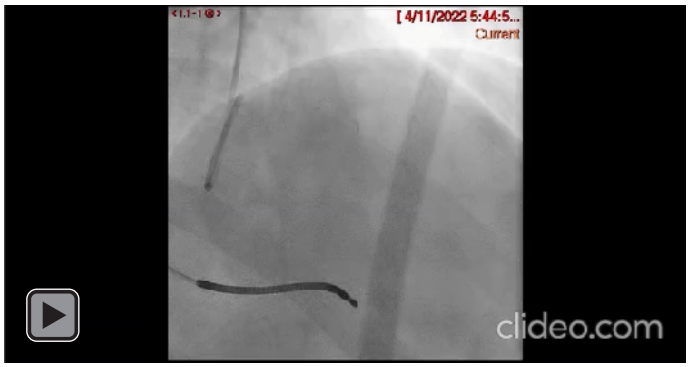
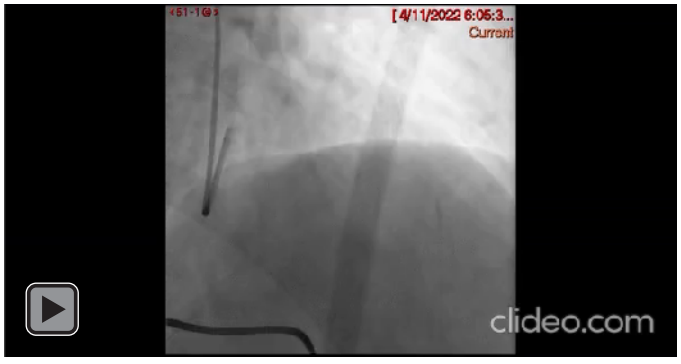
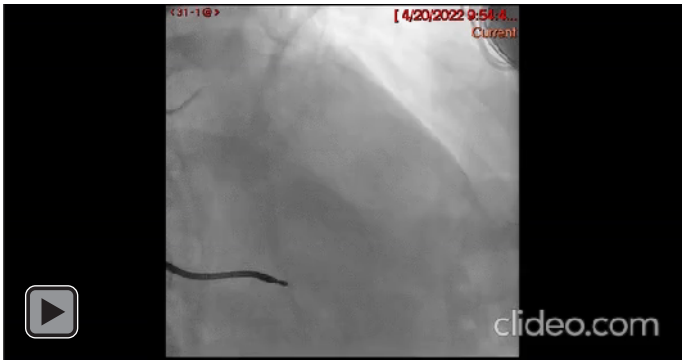
infusion, after which the patient was transported to the Cardiac Intensive Care Unit, where he was observed. Two hours later the patient continued to experience chest pain with ST-segment elevation in anterior leads and TTE revealed apical akinesia and reduction of LVEF to 40%. Repeat coronary angiography showed progression of slow flow phenomenon in distal LAD (TIMI 0) (Video 3). We advanced a coronary guidewire into the distal segment of the LAD and performed balloon angioplasty with 1,25 x 15 mm and 1,5 x 15 mm semi-compliant balloons which were inflated at their nominal pressures but without restoration of flow. Intravascular ultrasound showed a marked reduction in distal LAD lumen (diameter 2,2 mm, area 4,34 mm^2) and minimal atherosclerotic changes in medial (diameter 3,4 – 4,1 mm, area 11,3 mm^2) and proximal (diameter 3,9 – 4,2 mm area 12,5 mm^2) segments of LAD. The patient was treated with an intraaortic balloon pump and inotropes and observed for eight days. Cardiac enzymes were measured daily with a peak 18 hours after injection of alcohol (CK 3923 IU/L, CKMB 370 IU/L, hs Troponin I 88082.4 pg/ml). The last angiogram was performed nine days later and TIMI 0 flow in the distal LAD persisted. The patient was discharged from the hospital on post-procedural day ten in stable condition.



Video 1: S1 contrast injection. Contrast injection through the OTW balloon into S1 with no back leak of contrast into the LAD.
LAD- left anterior descending artery; OTW- Over The Wire.



Video 2: Slow flow in distal LAD. First angiogram 10 minutes after alcohol injection demonstrating slow flow (TIMI 1-2) in the distal LAD. LAD- left anterior descending artery; TIMI- Thrombolysis in Myocardial Infarction.



Video 3: No flow in distal LAD. The final angiogram at the end of the second procedure showed no flow (TIMI 0) in the distal LAD. LAD- left anterior descending artery; TIMI- Thrombolysis in Myocardial Infarction.

Discussion

Coronary artery occlusion is an infrequent complication of alcohol septal ablation occurring in 1% - 2% of procedures [3,4]. The main causes of LAD occlusion during ASA procedure are alcohol leakage, dissection, and vasospasm, whereby discovering the causes of these complications is of crucial importance for choosing an adequate treatment [4-7]. The most feared complication is alcohol leakage leading to anterolateral ischemia and consequent myocardial infarction in the LAD territory [8,9]. We repeatedly checked the position of the OTW balloon during the procedure and confirmed that it did not move throughout and that it was intact after the completion of the intervention. The possible cause of the no-flow phenomenon in distal LAD in our patient could be the opening of the collaterals between the S1 branch and septal branches of the distal LAD due to prolonged balloon inflation in S1. After careful analysis of angiograms obtained at ASA, there is a small amount of contrast going from S1 to distal LAD via collaterals in the AP cranial view (Figure 2). To avoid this scenario, besides adequate balloon sizing and contrast injection to check for collaterals, it is necessary to perform the procedure in a relatively rapid sequence of steps to avoid prolonged balloon inflation in the septal branch that would cause prolonged ischemia in the septal myocardium, consequently the opening of collaterals to this territory not seen on initial contrast injection through OTW balloon. If the preparation for alcohol injection takes more time than anticipated, a brief deflation of the OTW balloon followed by reinflation might be the way to avoid collateral circulation opening and unintended embolization. While the possibility of alcohol embolization via the opening of dormant collaterals had been theoretically considered before, we believe this is the first case report of this phenomenon occurring in clinical practice.

Follow up

At a one-month follow-up visit, the patient reported feeling well, with minimal chest pain. Echocardiography showed decreased LVEF of 40%, the septal thickness of 12 mm, akinesia of basal septum and apical segment of LV with LVOT peak and mean gradients of 19 mmHg and 9 mmHg respectively (Figure 1C).

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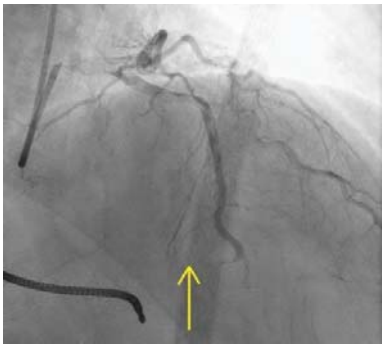

Figure 2: Collateral circulation filling distal LAD. A still frame of the angiographic image shows the collateral connection between the first septal branch circulation and a more distal septal branch (arrow). LAD- Left Anterior Descending artery.

Conclusion

We present here a case of distal LAD obliteration likely due to alcohol embolization via collateral circulation during alcohol septal ablation for the treatment of symptomatic patients with HOCM and high LVOT gradient. While septal ablation is generally safe, we want to raise awareness of the possibility of distal alcohol embolization via collateral circulation and the need to perform the procedure in rapid sequence thus avoiding prolonged balloon inflation and opening of dormant collaterals to the septal myocardium treated with alcohol. Our experience highlights ASA as an effective therapeutic intervention for symptomatic HOCM patients, providing relief from symptoms and enhancing cardiac function, reducing LVOT gradient.

Learning objectives

1. Reasons for LAD occlusion during alcohol septal ablation are dissection, vasospasm, and alcohol leakage.

2. Careful balloon sizing, and angiographic confirmation of the absence of contrast back leak or presence of collateral circulation in a rapid, controlled sequence of steps are some of the techniques that can prevent alcohol leakage via dormant collaterals.

Informed consent statement

Informed consent was obtained from the patient

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