Left Ventricular Aneurysm Perforating into the Right Ventricle: A Rare Complication of a Small Side Branch Occlusion after Elective Percutaneous Coronary Intervention

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INTRODUCTION

The incidence of mechanical complications of acute myocardial infarction (MI) has lessened significantly in the reperfusion era. They most commonly result from complete occlusion of a major coronary artery. We present a unique case of concomitant left ventricular aneurysm (LVA) formation and ventricular septal rupture (VSR) requiring open surgical repair after occlusion of a small (caliber < 1 mm) diagonal branch artery during successful elective stenting of the left anterior descending coronary artery (LAD). Multimodality imaging was integral to the correct diagnosis and therefore treatment of this rare pathology.

CASE PRESENTATION

A 73-year-old woman underwent elective coronary angiography for exertional chest pain and an exercise stress echocardiogram positive for LAD ischemia. She was an ex-smoker with a history of hypertension and dyslipidemia on standard treatment. Her resting electrocardiogram showed mild anterior and inferior ST-segment depression (Figure 1).

On coronary angiography, an 80% mid-LAD stenosis was identified (Figure 2A). Notably, left ventriculography and preceding stress echocardiography did not show any evidence of interventricular communication (Videos 1 and 2). The patient underwent direct stenting of the mid-LAD stenosis with a drug-eluting stent. Slow flow was not present at any stage during the procedure. There was no residual stenosis within the LAD stent, but there was occlusion of a small (caliber < 1 mm) diagonal artery, jailed by the stent, noted at the end of the procedure (Figure 2B). She experienced mild chest pain in the hours after the procedure, in the absence of any electrocardiographic changes. On review the following morning, she was asymptomatic with unchanged electrocardiographic findings but mildly elevated creatine kinase (253 U/L; reference range, 34–145 U/L). She was discharged after a further 24 hours of unremarkable observation.

Three weeks later the patient presented to a rural hospital with chest pain and normal troponin I but anteroseptal T-wave inversion on electrocardiography (Figure 1). Transthoracic echocardiography (TTE) was suggestive of flow from the left ventricle to the right ventricle, and she was transferred to our center for further investigation. Repeat coronary angiography showed a patent LAD stent, but left ventriculography identified opacification of the right ventricle and pulmonary artery with localized dyskinesia in the mid anterolateral wall, consistent with a VSR and suspicious for a coexisting aneurysm (Figure 2C, Video 3). Repeat TTE and transesophageal echocardiography confirmed communication between the left and right ventricles and a provisional diagnosis of VSR (Figure 3, Video 4). Imaging with cardiac computed tomography delineated myocardial rupture at the mid to distal anterior wall and anteroseptum (adjacent to the implanted stent), resulting in a 2.6 x 1.6 x 2.4 cm aneurysm that was communicating with the right ventricular apex (Figure 4). Cardiac magnetic resonance imaging confirmed these findings and showed systolic expansion of the aneurysm and small magnitude, almost continuous, left-to-right shunting (Qp/Qs = 1.3) through the septal perforation (Figure 5, Videos 5 and 6).

The patient continued medical therapy for coronary artery disease. Given the absence of heart failure and recent drug-eluting stent implantation, open surgical repair was deferred for 3 months after her stenting, and she remained well in the intervening period. At open surgery, localized transmural infarction of the diagonal artery territory of the left ventricle was noted, and on exploration of the aneurysm, communication with both the left ventricle and the right ventricle via septal perforations was identified (Figure 6). All defects were sutured closed, and the aneurysm was obliterated by a “double-breast” closure of the wall of the sac (Figure 6). Closure of the interventricular communication was confirmed on intraoperative transesophageal echocardiography. The patient made an uneventful recovery. At 4-month follow-up, the patient remained well with no evidence of heart failure. Absence of a residual interventricular shunt was confirmed on transthoracic echocardiography (Video 7).

DISCUSSION

An LVA is defined as an abnormal bulge in the left ventricular contour in both systole and diastole and is a recognized complication...
of acute (usually Q-wave) MI. Historically the incidence of LVA after a first Q-wave MI was >20%, but this has lessened in the reperfusion era. LVAS usually develop within 3 months of the index MI, and although rupture may occur in the “development” stage, it is rare once the LVA is formed, because of the dense fibrosis of the wall. As in this case, it may present with recurrent angina and electrocardiographic changes. VSR is a rare mechanical complication of acute MI (as low as 0.17% of cases in the reperfusion era) that typically becomes apparent within the first 24 hours. Our case describes a rare combination of subacute LVA formation complicated by rupture at the base of the aneurysm extending into the septum, allowing left-to-right shunting through a forme fruste of VSR. Such mechanical complications classically occur with occlusion of a major coronary artery (particularly the LAD). To our knowledge it has not been described after the occlusion of a diagonal artery or a vessel of this caliber.

Open surgical repair of a VSR is the treatment of choice, and surgery within the first 24 hours is undertaken if cardiogenic shock is present but carries a mortality risk of 60%. Percutaneous device closure may be considered in selected patients, but in-hospital mortality remains high (42% in the largest series). When possible, open surgical repair should be delayed to allow the formation of scar tissue. The optimal delay remains unclear, as it needs to be balanced against the risk of interim rupture extension and long-term complications of left-to-right shunting. Fortuitously, our patient did not develop hemodynamic decompensation, probably as the septal perforation developed subacutely in the presence of a small-territory MI, and the magnitude of shunting was small. At surgery, septal perforations can be closed directly with sutures, but the orifice communicating with the left ventricle is typically larger and requires a patch or a “double-breast” closure of the wall of the sac.

Figure 1 Preprocedure electrocardiogram (A) and on presentation to hospital 3 weeks after elective percutaneous coronary intervention (B) showing new anteroseptal T-wave inversion.

Figure 2 (A) Initial coronary angiogram showing 80% mid-LAD stenosis (arrow) with adjacent small diagonal side branch. (B) Coronary angiogram after stenting with loss of diagonal side branch (circle). (C) Left ventriculogram obtained on second presentation showing LVA (arrow).

Figure 3 Transthoracic echocardiogram parasternal long-axis image of abnormal flow across the interventricular septum from left ventricle to right ventricle.
This case describes a unique complication of elective percutaneous coronary intervention and highlights the necessity for multimodality imaging in the diagnosis of such rare pathology to ensure safe and effective patient management.

**SUPPLEMENTARY DATA**

Supplementary data related to this article can be found at https://doi.org/10.1016/j.case.2017.09.001.
REFERENCES

1. Visser CA, Kan G, Meltzer RS, Koolen JJ, Dunning AJ. Incidence, timing and prognostic value of left ventricular aneurysm formation after myocardial infarction: a prospective, serial echocardiographic study of 158 patients. Am J Cardiol 1986;57:729-32.

2. Tikiz H, Balbay Y, Atak R, Terzi T, Genc Y, Kutuk E. The effect of thrombolytic therapy on left ventricular aneurysm formation in acute myocardial infarction: relationship to successful reperfusion and vessel patency. Clin Cardiol 2001;24:656-62.

3. Jones BM, Kapadia SR, Smedira NG, Robich M, Tuzcu EM, Menon V, et al. Ventricular septal rupture complicating acute myocardial infarction: a contemporary review. Eur Heart J 2014;35:2060-8.

4. Arnaoutakis GJ, Zhao Y, George TJ, Sciortino CM, McCarthy PM, Conte JV. Surgical repair of ventricular septal defect after myocardial infarction: outcomes from the Society of Thoracic Surgeons National Database. Ann Thorac Surg 2012;94:436-43.

5. Calvert PA, Cockburn J, Wynne D, Ludman P, Rana B, Northridge D, et al. Percutaneous closure of postinfarction ventricular septal defect: in-hospital outcomes and long term follow-up of UK experience. Circulation 2014;129:2395-402.

6. Lemery R, Smith HC, Giuliani ER, Gersh BJ. Prognosis in rupture of the ventricular septum after acute myocardial infarction and role of early surgical intervention. Am J Cardiol 1992;70:147-51.