Intramyocardial dissection with concomitant left ventricular aneurysm as a rare complication of myocardial infarction: a case report

Chang-Cheng LIU1, Liang-Shan WANG1, Zhao-Ping SU2, Ying ZHAO3, Cheng-Xiong GU1
1Academic Department of Cardiovascular Surgery, Beijing Anzhen Hospital, Beijing Institute of Heart Long and Blood Vessel Diseases, Capital Medical University, Beijing, China
2Epidemiology and Health Statistics, Academy of Public Health and Management, Weifang Medical University, Weifang, Shandong, China
3Department of Ultrasound, Beijing Anzhen Hospital, Capital Medical University, Beijing, China

Abstract

We describe a rare case of a 60-year-old woman suffering from intramyocardial dissection and left ventricular aneurysm secondary to acute myocardial infarction. A rare form of ventricular septal rupture resulted from intramyocardial dissection deterioration, which was identified during echocardiographic follow-up. Surgical repair under beating-heart cardiopulmonary bypass was successful.

Keywords: Aneurysm; Complication; Dissection; Left ventricle; Myocardial infarction

1 Introduction

Intramyocardial dissection (ID) is an extremely rare and unusual form of impending heart rupture, overwhelmingly secondary to acute myocardial infarction (AMI). ID is defined as dissection among the spiral myocardial fibers and influx of blood, creating a neo-cavitation.[1] ID is characterized by being entirely contained within an integrated myocardial wall, unlike ventricular pseudoaneurysm, as well as by communication with the ventricular chambers by a serpiginous dissecting tract, with blood flow following the heartbeat.[2,3] We present a case of ID with concomitant left ventricular aneurysm (LVA) complicated by AMI. Our case not only revealed ID coexisting with LVA, but also presented an involved course of ID leading to ventricular septal rupture (VSR).

2 Case Report

A 60-year-old woman with a history of hypertension was admitted to a local hospital complaining of severe chest pain lasting for more than one hour. She was diagnosed as suffering from acute anterior MI and received streptokinase, but her symptoms persisted. She was then transferred to our hospital for cardiac catheterization. ECG showed sinus rhythm, elevated ST-segment with relevant inverted T waves in leads V1–V5, and pathological Q patterns in leads III and V1–V4, suggesting anterior wall MI. Transthoracic echocardiography (TTE) revealed akinesia of the apical and lower anterior ventricular septum with formation of an apical LVA, and ID located in the apical-septal segments (Figure 1A & B). Cardiac function was moderately dysfunctional (ejection fraction of 38%).

Coronary angiography demonstrated that typical three-vessel lesions were also present including total median-distal occlusion of the left anterior descending artery without visible established collateral circulation, diffuse moderate-severe stenosis of the left circumflex artery, and mild narrowing of the right coronary artery, eliminating the opportunity for percutaneous coronary artery intervention. Biochemical blood test results revealed serum levels of high-sensitivity C-reaction protein and troponin of 14.79 mg/L and 2.26 ng/mL, respectively, significantly higher than the normal ranges. Considering that the optimal timing of emergency operation had passed (less than 6 h from initial acute event), and the progressive ischemic myocardial edema, exudation and inflammatory reaction, the effectiveness of surgery was considered to be low. Therefore, pharmacological treatments were commenced to stabilize the patient’s condition during the preoperative period. Three
months later, the patient was admitted to our cardiac surgery department for surgical treatment. Cardiac function was grade III according to the New York Heart Association Classification. Repeat TTE indicated that the ejection fraction was 45%. Unfortunately, ID had also expanded into the right ventricular chamber as a tortuous tract present at several horizontal levels, which led to VSR followed by a small left-to-right shunt (Figure 1C & D). Serum levels of all cardiac markers were normal. After full physical evaluation, the patient underwent cardiac surgery with beating-heart cardiopulmonary bypass grafting (CABG). CABG was performed first. ID and VSR were repaired second as follows. First, two traction sutures were placed at the apex. Second, a 2 cm incision was made between the two traction sutures parallel with the left anterior descending artery. Finally, 2-0 polypropylene sutures were passed through a strip of Dacron felt, the left ventricular ID entry point, apical septum, and the other end of the strip of felt, using a horizontal mattress pattern. The LVA was corrected in a similar way using 2-0 Prolene suture and two strips of Teflon felt in a continuous horizontal mattress pattern (Figure 2). Linear plication paralleled the left anterior descending artery, eliminating the chance of an abnormal contracting scar and restoring normal ventricular size and shape. Postoperative TTE confirmed disappearance of the echo-free space at the dissecting cavity, and no shunting. The patient recovered uneventfully without surgical complications and was asymptomatic at discharge. During a follow up of 6 months, patient has been free of angina pectoris.

3 Discussion

Our case displayed an unusual form of cardiac disease secondary to myocardial infarction: ID coexisting with LVA. During the wait for cardiac surgery, the patient experienced deterioration of the ID, which caused a rare form of VSR. ID is a rare complication of AMI. Dellborg, et al.[4] reported that among 3960 AMI cases, sub-acute cardiac rupture with concomitant ID accounted for approximately 1% of patients. The pathogenesis of ID involves the rupture of intramyocardial vessels in the extracellular matrix, which increases tissue fragility and decreases tensile force in the infarcted area. In cases of microcirculatory obstruction, ischemic reperfusion increases coronary capillary perfusion pressure.[2,5,6] ID can appear in the left ventricular free wall, ventricular septum, or right ventricle, but the majority of cases occur at the ventricular septum next to the left ventricular free wall.[7–9]

Prior to the availability of non-invasive imaging techniques, ID was a postmortem diagnosis.[10,11] However, even with newer diagnostic tools, definitive diagnosis usually involves more than one test. Angiography of the coronary

Figure 1. Transthoracic echocardiographic views of intramyocardial dissection. (A): Parasternal long-axis view showed a dissecting free-echo space (yellow arrow); (B): color Doppler imaging depicted a to-and-fro flow through dissecting tract between the left ventricle and dissecting cavitation; (C): apical four-chamber view showed a serpiginous tract in ventricular septum (yellow arrow heads); and (D): color Doppler imaging presented a small left-to-right shunting via a tortuous tract at ventricle septum. LV: left ventricle; RV: right ventricle.
Figure 2. Operative technique of ID and LVA. (A): Schematic diagram showed an apical aneurysm and a complex cardiac rupture which is defined as an interventricular communication via a serpiginous course; (B): sketch figure displays that the entry of ID serpiginous channel is closed by a horizontal mattress pattern (arrow), and LVA is plicated at the thin rim of scar with the mattress sutures; (C): left ventricular aneurysm located at the apex (ellipse); and (D): intraoperative picture of apical aneurysm placation. An: aneurysm; ID: intramyocardial dissection; LV: left ventricle; LVA: left ventricular aneurysm; RV: right ventricle.

arteries and left ventricle is considered the gold standard for diagnosing coronary lesions, guiding CABG and identifying dissecting sacs in the myocardium and/or silhouettes of ventricular aneurysms.[12] However, during angiography, contrast agent is rapidly injected into the left ventricle under high pressure, which increases the possibility of expanding the dissection and inducing cardiac rupture. TTE is a reasonable first examination because it is non-invasive, inexpensive, and portable. Color Doppler echocardiography can indicate a low-velocity flow profile in the abnormal route, sometimes with a bidirectional to-and-fro pattern.[13,14] Transesophageal echocardiography can evaluate the various myocardial ventricular segments with higher resolution, particularly ID located on the septal and/or right ventricular segments.[14] ID is prone to further extension and rupture, and may be in an evolutionary period before cardiac rupture and formation of a pseudoaneurysm, as described in our report and others.[1,13] Therefore, it is essential to correct ID secondary to AMI. Harpaz, et al.[11] reported that the mortality rate among medically treated patients was 90% and that none of the surgically treated patients died. However, another small case series suggested that of 15 cases, four of six patients died undergoing surgery with simultaneous CABG and ID repair, while in five patients receiving pharmacological treatment, only one died.[16] Sari, et al.[10] also reported a case in which ID resolved spontaneously. Because the data were scattered, derived from case reports and small case series studies, and because of the lack of systematically retrospective reviews of large numbers of consecutive cases, the actual incidence and mortality of ID is not well known. Nonetheless, considering the irreversible progression of coronary arteriosclerosis and the underlying ischemic cardiomyopathy, we emphasize the significance of coronary revascularization at the time of ID repair. In our case, the simultaneous ID and LVA complications of AMI greatly increased the risk of cardiac rupture, heart failure, and the frequency of ventricular arrhythmias related to the LVA. Therefore, our team successfully performed CABG accompanied by ID repair. More importantly, the surgical repair was conducted with a beating heart, differentiating our study from procedures performed under cardiac arrest in other reports. The beating-heart technique not only avoided aortic cross-clamping, but also facilitated identification of the non-contractile zone of the LVA, avoiding excessive reduction of the residual ventricular volume and insufficient removal of abnormal contractions compared with on-pump LVA linear closure.

In conclusion, with rapid developments in percutaneous coronary artery intervention, the incidence of LVA and ID has declined; however, this creates a new challenge for surgeons confronted with more severe coronary disease.[17] Nevertheless, operative risks are also substantial, and comprehensive evaluation of patients’ physical status and appropriate surgical procedures are essential. We believe that complete revascularization combined with surgical correction of concomitant abnormalities is indicated for all patients with a confirmed diagnosis unless there are obvious operative contraindications.

Acknowledgements

This study was founded by National Natural Science Foundation of China (81370436); Beijing Municipal Commission of Science and Technology (Z131107002213003); Beijing Municipal Commission of Education (KZ201410025028).

References

1 Harpaz D, Kriwisky M, Cohen AI, et al. Unusual form of cardiac rupture: Sealed subacute left ventricular free wall rupture, evolving to intramyocardial dissecting hematoma and to pseudoaneurysm formation—a case report and review of the
2 Dias V, Cabral S, Gomes C, et al. Intramyocardial dissecting haematoma: a rare complication of acute myocardial infarction. *Eur J Echocardiogr* 2001; 10: 585–587.

3 Lewis A, Burchell H, Titus J. Clinical and pathologic features of postinfarction cardiac rupture. *Am J Cardiol* 1969; 23: 43–52.

4 Dellborg M, Held P, Swedberg K, et al. Rupture of the myocardium occurrence and risk factors. *Br Heart J* 1985; 54: 11–16.

5 Purcaro A, Costantini C, Ciampani N, et al. Diagnostic criteria and management of subacute ventricular free wall rupture complicating acute myocardial infarction. *Am J Cardiol* 1997; 80: 397–405.

6 Slootweg A, Louwerenburg J, Mecozi G, et al. Obstructive intramyocardial haematoma after percutaneous coronary intervention. *Neth Heart J* 2012; 20: 376–378.

7 Pliam MB, Sternlieb JJ. Intramyocardial dissecting hematoma: an unusual form of subacute cardiac rupture. *J Card Surg* 1993; 8: 628–637.

8 Edwards BS, Edwards WD, Edwards JE. Ventricular septal rupture complicating acute myocardial infarction: identification of simple and complex types in 53 autopsied hearts. *Am J Cardiol* 1984; 54: 1201–1205.

9 Batts KP, Achermann DM, Edwards WD. Postinfarction rupture of the left ventricular free wall: clinicopathologic correlates in 100 consecutive autopsy cases. *Hum Pathol* 1990; 21: 530–535.

10 Sari I, Duvutoglu V, Kucukdurmus Z. Intramyocardial dissection after subacute anterior wall myocardial infarction: an unusual form of myocardial rupture with subsequent spontaneous healing. *Echocardiography* 2008; 25: 228–230.

11 Roberts WC, Burks KH, Ko JM, et al. Commonalities of cardiac rupture (left ventricular free wall or ventricular septum or papillary muscle) during acute myocardial infarction secondary to atherosclerotic coronary artery disease. *Am J Cardiol* 2015; 115: 125–140.

12 Frances C, Romero A, Grady D. Left ventricular pseudoaneurysm. *J Am Coll Cardiol* 1998; 32: 557–561.

13 Vargas-Barron J, Roldán FJ, Romero-Cardenas A, et al. Intramyocardial dissecting hematoma and postinfarction cardiac rupture. *Echocardiography* 2013; 30: 106–113.

14 Vargas-Barron J, Molina-Carrion M, Romero-Cardenas A, et al. Risk factors, echocardiographic patterns, and outcomes in patients with acute ventricular septal rupture during myocardial infarction. *Am J Cardiol* 2005; 95: 1153–1158.

15 Psaltis PJ, Dundon BK, Teo KS, et al. Utility of cardiac magnetic resonance imaging in detection of post-infarction intramyocardial dissection. *Heart Lung Circ* 2008; 17: 415–416.

16 Vargas-Barron J, Roldán FJ, Romero-Cardenas A, et al. Dissecting intramyocardial hematoma: clinical presentation, pathophysiology, outcomes and delineation by echocardiography. *Echocardiography* 2009; 26: 254–261.

17 Ikeda N, Yasu T, Kubo N, et al. Effect of reperfusion therapy on cardiac rupture after myocardial infarction in Japanese. *Circ J* 2004; 68: 422–426.