Ventricular Septal Defect and Left Ventricular Aneurysm

Late Occurrence as Complications of an Acute Myocardial Infarction

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SUMMARY

Mechanical complications of acute myocardial infarction (AMI) such as a ventricular septal defect (VSD) usually occur within the first week. In the thrombolytic era, the incidence of a VSD has not increased, but has been reported to occur earlier than previously described. We report an unusual case of an elderly Caucasian female with an acute anterior wall myocardial infarction treated with thrombolytic therapy. Her AMI was complicated by pulmonary edema secondary to a VSD and a left ventricular aneurysm five weeks later. Prompt diagnosis, immediate surgical closure of the VSD, and aneurysmectomy resulted in her complete recovery. (Jpn Heart J 2000; 41: 773-779)

Key words: Ventricular septal defect, Ventricular aneurysm, Stroke, Thrombolytics, Acute myocardial infarction

ACUTE myocardial infarction (AMI) can be associated with devastating mechanical complications. These tend to occur more frequently with a large, transmural myocardial infarction (MI). Myocardial rupture is an infrequent, but well described complication of AMI. It can involve the ventricular free wall, interventricular septum, or papillary muscle. Acute rupture of the interventricular septum usually occurs within the first week of AMI. The overall incidence has been reported to occur in 1-3% of patients and is associated with a high mortality if not immediately diagnosed and adequately managed.1-3) Left ventricular aneurysm (LVA) is relatively more common with a reported incidence of 3.5-38%.4-6) Ventricular aneurysms have been associated with myocardial free-wall rupture, congestive heart failure (CHF), left ventricular (LV) thrombus formation, and incessant ventricular tachyarrhythmias. The finding of a VSD and LVA in the
same patient is rare and usually occurs within the first week. We describe an unusual case of an elderly female with an acute anterior wall MI treated with thrombolytic therapy. Her AMI was complicated by pulmonary edema secondary to a VSD and LVA five weeks after her acute infarction. Prompt diagnosis and immediate surgical closure of the VSD and aneurysmectomy resulted in her complete recovery.

CASE REPORT

A 68-year old Caucasian female with hypertension and type II diabetes mellitus presented with a three hour history of retrosternal chest pain and dyspnea. Her electrocardiogram (EKG) revealed ST segment elevation in leads V1-V5, consistent with an acute anterior wall MI. She was treated with accelerated t-PA, aspirin, unfractionated heparin, metoprolol, and captopril. Approximately four hours later, she developed left hemiparesis. Magnetic resonance imaging revealed an ischemic cerebral infarction (CI) involving the right middle cerebral artery territory without an intra-cerebral bleed. Transthoracic echocardiography showed severe antero-septal

![Figure 1](image-url). Transthoracic echocardiography, apical four-chamber view, shows the aneurysmal dilatation of the left ventricle. RA = right atrium; LA = left atrium; RV = right ventricle; LV = left ventricle.
Figure 2. Left ventriculography, left anterior oblique view, illustrates the site of an apical VSD (arrows) with left-to-right flow.

Figure 3. Left ventriculography, left anterior oblique view, shows the contrast-filled dilated RV from the VSD.
hypokinesis, apical akinesis, and moderate LV systolic dysfunction. There was no evidence of a VSD, LVA, or LV thrombus. The remainder of her hospital course was uneventful and she was managed conservatively. After discharge, she was referred for physical therapy.

Two weeks later, she was evaluated for exertional dyspnea. Physical examination revealed no evidence of CHF. A repeat echocardiogram was unchanged and a diuretic was added to her medical regimen.

Five weeks after her AMI, she presented with acute pulmonary edema. Her vital signs were: BP- 142 / 82 mm Hg, Pulse- 126 / min, Respiratory Rate- 32 / min. Physical examination was significant for jugular venous distension, bilateral rales approximately 2 / 3 of the lung fields, and a grade III / VI holosystolic murmur at the left lower sternal border. Her EKG showed sinus tachycardia and an old anterior MI with persistent ST segment elevation in leads V2-V5. A chest radiograph revealed cardiomegaly and pulmonary vascular congestion. She responded well to intravenous lasix. Serial cardiac enzymes were negative. Transthoracic echocardiography performed in the emergency department showed anteroseptal hypokinesis and a large apical aneurysm with no thrombus (Figure 1). On color flow Doppler, an apical VSD with a left-to-right shunt was seen. Cardiac catheterization and coronary angiography were performed, which revealed a step-up between the right atrium and the right ventricle with a pulmonary to systemic flow ratio (Qp / Qs) of 2.4:1, single vessel coronary artery disease with an occluded left anterior descending artery and visualization of the distal vessel filling via collaterals from the right coronary artery. Left ventriculography confirmed the presence of a large VSD and a large apical aneurysm (Figures 2 & 3). There was no mitral regurgitation. She underwent successful surgery with patch-closure of VSD and resection of the left ventricular aneurysm. Her post-operative course was uncomplicated and she remained asymptomatic after six months of follow up.

DISCUSSION

Acute myocardial infarction remains a major public health concern associated with significant morbidity and mortality. Major complications of AMI are summarized in the Table. The combined presence of a VSD and LVA as described in our patient represent a highly lethal and rare complication of AMI that requires immediate recognition and surgical correction. 7,8) Left ventricular thrombus can form within four hours of an AMI.
Yet, there has been no increase in the incidence of cerebral or systemic embolization in the thrombolytic era. However, ischemic CI is a serious complication after AMI with a mortality rate of 54–61%. Since first reported by Latham in 1846, rupture of the interventricular septum is a well established complication of AMI, usually occurring by the third to fifth hospital day. Rupture of the interventricular septum can be categorized as either simple or complex. A simple VSD has through-and-through communication across the interventricular septum. It usually involves the apical septum and frequently occurs with anterior infarctions. Whereas a complex VSD has an irregular and serpiginous tract extending in multiple directions. It usually involves the inferobasal septum and is more frequent with inferior infarctions. A VSD is more commonly seen in elderly patients with transmural, anterior wall MI (particularly first MI), hypertension, non-smoker, multi-vessel disease, and in those lacking an established collateral network. Patients usually present with pulmonary edema or cardiogenic shock. A new loud, harsh holosystolic murmur is usually detected. Presence of a palpable thrill in greater than 50% of cases may help in differentiating this entity from papillary muscle rupture. Echocardiography is a highly sensitive and specific tool in assessing the presence of VSD and is the diagnostic modality of choice. Despite optimal treatment, mortality remains 45% in surgically treated patients and 90% in those managed medically. Predictors of survival following surgery include systolic blood pressure, mean right atrial pressure, and cardiopulmonary bypass time.

Left ventricular aneurysm formation is a relatively more common complication after anterior wall MI. It usually results from infarct expansion, thinning and dilatation of the infarct zone along with fibrosis and

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impaired contractility. This pathophysiological process has been referred to as “expaneurysm”. The majority of LVAs following anterior MI are apparent by the time of hospital discharge. Higher ventricular-afterload and administration of steroids also predispose to the formation of an aneurysm. An overall mortality rate of 67-80% has been reported. Since first described by Beck in 1944, surgical repair of a ventricular aneurysm results in improved survival in this subset of high-risk patients. Indications for surgical resection include refractory CHF and refractory ventricular tachyarrhythmias. Surgery may also be indicated in patients with recurrent thromboembolism. The mean operative mortality remains high and is reported to be 9.9%.

Reperfusion therapy with thrombolytics should reduce the likelihood of mechanical complications such as VSD and LVA by early restoration of coronary blood flow. In GUSTO-1, the incidence of myocardial rupture has been inversely correlated with early TIMI grade 3 flow restoration. Yet, reperfusion therapy also increases the likelihood of hemorrhagic myocardial necrosis, which may lead to myocardial rupture. However, no significant change in the incidence of these complications has been reported in the thrombolytic era, though an earlier “acceleration” of myocardial rupture has been observed, typically within 24-48 hrs following thrombolytic therapy. The median time to the diagnosis of VSD was one day in 84 of the 41,021 patients in the GUSTO-1 study. Also the elevation of C-reactive protein level to ≥ 20 mg / dl following AMI has been associated with an increase risk of cardiac rupture, LVA formation, and cardiac death.

Our patient is unique in that she presented with a ventricular septal defect and left ventricular aneurysm five weeks after an AMI with no evidence of re-infarction. Late occurrence of these complications in the thrombolytic era is rare. Failed reperfusion therapy with an occluded infarct related artery, absence of a collateral network, and the presence of hypertension may have been the contributing factors in our patient.

**Conclusion:** The combined presence of a VSD and LVA is rare and usually occurs within two weeks of an AMI. However, as demonstrated in our patient, these complications may present up to several weeks later. Early recognition, prompt diagnosis, and immediate surgery can result in complete recovery.
REFERENCES


