Postinfarction Left Ventricular Rupture Misdiagnosed Ruptured Intramural Hematoma of Aorta

Young-Won Yoon¹, Donghoon Choi, Bon-Kwon Ko², Won-Heum Shim¹, Seung-Yun Cho¹, and Byung-Chul Chang²

¹Cardiology Division, ²Division of Cardiovascular Surgery, Yonsei Cardiovascular Hospital, Yonsei University College of Medicine, Seoul, Korea.

Left ventricular rupture is a fatal complication of acute myocardial infarction, however accurate preoperative diagnosis is still difficult. We experienced a postinfarction left ventricular rupture patient whose symptoms and radiologic findings mimicked those of acute intramural hematoma of the aorta. Upon emergency operation, he was proven to have a postinfarction LV rupture and underwent successful surgery. We herein report the case with a brief review of the literature.

Key Words: Post infarction ventricular rupture, intramural hematoma of aorta

Left ventricular rupture is the second most common cause of death following acute myocardial infarction, behind only pump failure.¹,² While the incidence rate varies greatly, it accounts for 12% to 21% of deaths following myocardial infarctions.³ The wide spread availability and use of cardiac imaging studies, particularly echocardiography, have increased the number of cases diagnosed before death and the rate of successful surgical treatment of this desperate complication. However, because of it’s various clinical presentations, accurate preoperative diagnosis is still a challenge. We recently experienced a patient with a presumed diagnosis of acute intramural hematoma of an ascending thoracic aorta that after emergency operation proved to be a left ventricular posterior wall rupture.

CASE REPORT

A 61-year old male patient experienced a sudden severe back pain one hour prior to arriving at the emergency room. He had a history of hypertension for 3 years without treatment and 50 pack-year smoking. Otherwise, he had no remarkable history of cardiovascular illness. On examination, the patient was drowsy and skin was clammy. The blood pressure barely registered at 60/40 mmHg respectively. The pulse rate was 125/min and neck vein engorgement was noted. The heart sounds were distant and diminished, so the discrimination of murmurs or a gallop sound was difficult. The ECG showed Q waves and ST segment elevations on lead I and aVL, and ST segment depressions on precordial lead V5-V6 suggestive of acute lateral wall myocardial infarction (Fig. 1). Since the chest roentgenogram taken at ER showed a widened mediastinum (Fig. 2) and the patient complained of severe back pain, we

![Fig. 1. Electrocardiogram showed Q waves and ST segment elevations on lead I and aVL, and ST segment depressions on precordial lead V5-V6, suggestive of acute lateral wall myocardial infarction.](image-url)
ruled out acute dissecting aneurysm of the aorta with coronary occlusion leading to myocardial infarction. Computed tomography of the chest showed a marked amount of pericardial effusion and fluid collection around the proximal ascending aorta and aortic arch without definite flap (Fig. 3). The density of the collected fluid was somewhat high, suggestive of hematoma of the ascending aorta and hemopericardium. Emergency coronary angiography revealed a total occlusion of the big obtuse marginal branch and a 70% luminal narrowing of the distal circumflex artery (Fig. 4). During coronary angiography, the patient suffered cardiac arrest and underwent 5 minutes of resuscitation. Further investigation such as ventriculography, aortography and transesophageal echocardiography could not be performed due to the patient’s unstable condition. We presumed that the patient had an acute intramural hematoma of the aorta and cardiac tamponade coexistent with acute lateral wall myocardial infarction. Therefore, the patient underwent emergency surgery. In the operation field, after the pericardium was resected, dark bloody pericardial fluid and blood clots were gushed out. Upon exposure of the great vessels, localized hematoma was found around the ascending aorta, however the remaining portion was unremarkable and there was no evidence of aortic dissection. Careful examination disclosed a discolored ventricular ruptured site at the apicolateral aspect of the left ventricular wall, that was the cause of pericardial effusion and
extending hematoma around the ascending aorta. Ventricular wall repair was done with Teflon felt.
Aorta to mid-RCA, aorta to second and third obtuse marginal branch coronary artery bypass graft was performed. Following the surgery, the patient recovered relatively well. He was discharged 3 weeks after the operation and has done well to date.

**DISCUSSION**

Since the first successful surgical repairs of postinfarction left ventricular rupture reported in 1972, there have been many successes in the management of postinfarction left ventricular rupture. There are several reported operative mortalities of this complication, ranging from 24% to 35%. One thing is clear: the most important contributing factor to a positive prognosis is an accurate preoperative diagnosis. However, as the clinical presentation of this complication varies greatly and patients usually have unstable vital signs, a correct preoperative diagnosis remains difficult.

Certain patients have an increased risk of free wall rupture following myocardial infarction, including those whose age greater than 60 years, females, patients with preexisting hypertension, and those with no prior history of myocardial infarction. These are well known characteristics, but not specific enough for prediction. Oliva et al. reported the characteristic signs and symptoms of ventricular rupture by studying 70 consecutive patients. The symptoms included positional pleuritic chest pain, repetitive and unprovoked emesis, and restlessness and agitation. They reported that 84% of patients with a rupture had two of these three symptoms. However, all the three symptoms are manifestations of cardiogenic shock and provide little help in a practical aspect. In our case, the patient’s symptoms and radiological findings were clinically suggestive of a ruptured aortic intramural hematoma and we didn’t expect left ventricular rupture before the operation.

Cheriex et al. suggested that a myocardial rupture typically occurs in an infarcted area lacking reperfusion. Coronary angiography findings and the ruptured site on the operation field were also consistent in our case. We attempted aortography and ventriculography at the catheterization room, but barely completed diagnostic coronary angiography. Fortunately, the patient was resuscitated well and underwent a successful surgery. In a case such as this, it would be better to perform a simple transthoracic echocardiography or transesophageal echocardiography rather than an invasive study. Currently, echocardiography is becoming the fastest and most timesaving diagnostic test for confirming cardiac rupture. The most consistent finding is pericardial effusion. Other signs such as echogenic masses in the effusion and wall defects are also consistent findings. Pollak et al. reported that these findings have 97% sensitivity and 93% specificity. On the other hand, the role of invasive tests in the diagnosis of left ventricular rupture is unclear. Ventriculography is usually not recommended due to its low diagnostic yield. A confirmative diagnosis is often impossible because it requires continuous contrast leakage through the ruptured site. Some authors recommend coronary angiography before operation of an LV rupture in order to delineate the anatomy of coronary arteries in cases of performing coronary artery bypass graft simultaneously. However, this often provides no further information and is quite time consuming. Pifarre and coworkers suggested that surgery should not be delayed unnecessarily for cardiac catheterization in critically ill patients.

Of interest in our case is the confusing preoperative diagnosis. There are no similar cases reported to our knowledge. Because of the high mortality rate of left ventricular rupture, many cases like this may have expired before operation. When a patient presents cardiac tamponade with hematoma of the ascending aorta, and if there is significant ECG change, one should suspect ventricular rupture among the various differential diagnoses.

**REFERENCES**

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