Oozing Type Cardiac Rupture Repaired With Percutaneous Injection of Fibrin-Glue Into the Pericardial Space

— Case Report —

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Two patients, a 56-year-old man and an 81-year-old woman who were admitted to hospital because of anterosep-tal acute myocardial infarction, were initially treated successfully with direct percutaneous transluminal coronary angioplasty. However, both patients later developed sudden cardiogenic shock due to cardiac tamponade caused by left ventricular free wall rupture (LVFWR). Prompt, life-saving pericardiocentesis was performed, then fibrin-glue was percutaneously injected into the pericardial space. After the procedure, there was no detectable pericardial effusion on echocardiography and the hemodynamic state became stable. The surgical treatment was the standard procedure for LVFWR, but percutaneous fibrin-glue therapy can also be considered for oozing type LVFWR. (Jpn Circ J 2000; 64: 312–315)

Key Words: Acute myocardial infarction; Fibrin-glue; Left ventricular free wall rupture

The incidence of left ventricular free wall rupture (LVFWR) following acute myocardial infarction (AMI) is about 3%1 and is usually rapidly fatal due to cardiac tamponade and hemodynamic deterioration. Boshour et al2 classified LVFWR into 3 types: (i) 'blow out'; (ii) 'oozing', characterized by a small rupture or leakage through a friable aneurysm; and (iii) 'false aneurysm', characterized by chronic rupture. Patients with the blow-out type of LVFWR are very difficult to save, but those with the oozing type can be saved by urgent management. It is generally considered that surgical intervention is necessary and the majority of reports on such cases indicate that such measures have been successful in saving the patient's life2. Recently, percutaneous fibrin-glue injection therapy was reported by Ogiwara et al3,4. We successfully used this method to repair the oozing type of cardiac rupture in 2 cases, which are presented here.

Case Reports

Case 1

A 56-year-old man was admitted to hospital with chest pain and nausea in December 1998. He had been on hemodialysis for chronic glomerulonephritis since 1988. His medical history revealed nothing precursory to his symptoms; he had no history of chest pain. The onset of chest pain occurred 1 h prior to admission. Neither electrocardiogram nor echocardiogram revealed anterosep-tal AMI; cardiac enzymes were not elevated. We performed urgent coronary angiography, which revealed 90% stenosis in the middle segment of the left anterior descending artery (LAD) and 90% stenosis in the proximal segment of the first diagonal branch (D1). Subsequently, direct percutaneous transluminal coronary angioplasty (PTCA) was performed for those 2 stenotic lesions. The guidewire was easily passed across the target lesions, and residual stenosis was 25% without dissection. The procedure was completed in 90 min, at which point hemodialysis was begun. One hour later, the patient complained of severe chest oppression. Suddenly, systolic blood pressure dropped to about 60 mmHg, and his pulse rate was 150 beats/min. The echocardiogram revealed cardiac tamponade (Fig 1). Pericardiocentesis was immediately performed using a 22 g long needle. Bloody fluid was obtained (approx 50 ml), and vital signs recovered slightly. The patient was transferred to the coronary care unit (CCU) where echocardiography was performed again, revealing some residual fluid in the pericardial space. Repeat pericardiocentesis, using a 16 g single lumen IVH catheter, obtained 200 ml of bloody fluid. It was then decided to inject fibrin-glue into the pericardial space. The fibrin-glue was composed of 10 ml of Solution A (800 mg human fibrinogen + 750 units coagulation factor XIII + 10,000 KIE aprotinin solution) and 10 ml of Solution B (2,500 units thrombin + 10 ml calcium chloride). Solutions A and B were injected separately, and the lumen of the catheter was flushed with approximately 2 ml of saline. During the procedure we used a vasodilator to maintain systolic blood pressure within 80 mmHg. After this treatment, the patient’s hemodynamic state remained stable and the echo-free space in the pericardium was no longer observed. One month later, the patient was discharged without any complications and has remained asymptomatic.

Case 2

An 81-year-old woman was admitted to hospital with
chest pain in March 1999. Prior to admission, she had been healthy with no history of chest pain. The electrocardiogram on admission showed persistent elevation of the ST segment in the anterior chest leads, and cardiac enzymes were elevated; namely, creatine kinase of 667 IU/L, aspartate aminotransferase 791 IU/L, and lactate dehydrogenase 671 IU/L. Anteroseptal AMI 10h after onset was the diagnosis. Urgent coronary angiography revealed total occlusion in the proximal segment of the LAD and PTCA was performed. The guidewire was easily passed across the target lesion and after the balloon angioplasty the residual stenosis was 75% with slightly dissection. Stenting was then performed successfully. The procedure was completed in 2h without complications and the patient was transferred to the CCU. The next day she suddenly lost consciousness. Her systolic blood pressure was approximately 40 mmHg.

Fig. 1. Echocardiography of case 1. Cardiac tamponade developed 1h after PTCA (A). After fibrin-glue therapy, there was no echo-free space in the pericardium (B).

Fig. 2. Echocardiography of case 2. Cardiac tamponade developed 1 day after PTCA (A). After fibrin-glue therapy, there was no echo-free space in the pericardium (B).

Fig. 3. Cut surface of the heart shows transmural myocardial infarction in the anteroseptal wall. A small amount of serous fluid was found in the pericardial space. No adhesions were found in the pericardial space. The site of the tear in the myocardial muscle could not be seen.
and her pulse rate was 130 beats/min. The emergency echocardiogram revealed cardiac tamponade (Fig2). Pericardiocentesis was immediately performed and fibrin-glue injected into the pericardial space as described in case 1. After this treatment, the patient’s hemodynamic state remained stable and the echo-free space was no longer observed in the pericardium. An intra-aortic balloon pump was introduced to unload systolic blood pressure, which was maintained below 80 mmHg by using vasodilators for 10 days. The patient did not develop any signs of organ failure, such as renal dysfunction, due to the hypotension, but unfortunately she did develop severe pneumonia and died of sepsis and acute respiratory distress syndrome 1 month later. At autopsy, the heart weighed 350g and its gross appearance is shown Fig 3. No adhesion was found in the pericardial space, but there was a small amount of serous fluid. The site of the oozing rupture could be seen in the anteroapical wall, which was very thin. Microscopy revealed red blood cells in the fatty tissue and a layer of fibrin covering the surface of the pericardial membrane (Fig4).

**Discussion**

LVFWR is a catastrophic, usually fatal, complication that frequently occurs in patients recovering from AMI. In the past, the diagnosis of LVFWR was based on the patient’s physical appearance and vital signs; namely, sudden loss of consciousness, jugular distension, hypotension and/or tachycardia. However, LVFWR does not always present such a dramatic course and echocardiography is the most sensitive method for diagnosis. Both cases in this report were the oozing type rupture. Although surgery is generally recommended for all 3 types of LVFWR, it is considered hazardous soon after myocardial infarction because the fragility of the tissue can make suturing difficult. Various surgical techniques for LVFWR have been reported: closing the tear with teflon-reinforced sutures, combination of infarct excision and closure of the created defect with interrupted sutures, and covering the ventricular rupture and surrounding infarcted muscle with a patch. As a variation of the third method, fibrin-glue is injected under the pericardium to reinforce the repair and prevent leaking through the suture line. Despite various revisions to the surgical procedure, the mortality rate is still high, ranging from 40 to 70%.13

Recently, Ogawa et al reported successful treatment of the oozing type of cardiac rupture using percutaneous fibrin-glue therapy. Prior to the clinical application of this therapy, they experimented on a dog model and established its efficacy. Yagi et al reported 4 patients with oozing type LVFWR, all of whom survived after receiving percutaneous fibrin-glue therapy.13 We informed the present patients’ families of this new method, including the merits and risks, and obtained their consent. The procedure was performed as described. Because we inserted the catheter from the subxiphoid process, the entry point of the catheter must have been near the cardiac apex. Initially, the fibrin-glue would probably pool at the bottom of the pericardium due to the gravity and then be scattered all over the pericardium by the beating heart; autopsy of case 2 revealed that the fibrin-glue was equally distributed over the surface of the myocardium. After injection, it is important to maintain the systolic blood pressure below 80 mmHg to decrease the pressure load on the LV wall.

The autopsy findings were very interesting, and there are no reports of the pathology of fibrin-glue therapy. The surface of the pericardial membrane was very smooth and no adhesions were detected. The bleeding is stopped not by adhesions in the pericardial space but by the thin layer of fibrin that covers the pericardial membrane. From these pathological findings, we can infer that fibrin-glue therapy is a physiological method; that is, we consider that diastolic dysfunction, such as constrictive pericarditis, will not be observed in the future. This therapy may not be sufficient for the treatment of the blow-out type of LVFWR, but it can effectively treat the oozing type.

Jaume et al reported 19 LVFWR patients treated medically; that is, bed rest and control of systolic blood pressure with a β-adrenergic blocking agent. Pericardiocentesis was carried out in 15 patients. They concluded that the long-term survival of selected patients with prompt hemodynamic recovery after LVFWR was possible without surgical repair. In our 2 cases, fibrin-glue may have been unnecessary; pericardiocentesis alone may be sufficient to cure the oozing type of LVFWR. We did not
compare 2 patient groups (ie, with or without the fibrin-glue therapy), so we can not conclude that fibrin-glue therapy truly contributed to the outcome. Further studies are needed to confirm the efficacy of this therapy.

In summary, we used percutaneous fibrin-glue therapy to successfully treat 2 LVFWR patients. The principal therapy for LVFWR is surgical, but as cardiovascular surgeons may not always be available, fibrin-glue therapy can be performed by a physician. Thus, fibrin-glue therapy can be an alternative treatment for LVFWR, though it is important to remember that the surgical option may be required to manage the case in which this method is not effective.

References