—**Original**—

Clinicopathological Characteristics of 10 Patients with Rupture of Both Ventricular Free Wall and Septum (Double Rupture) After Acute Myocardial Infarction

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Abstract

Cardiac ruptures after myocardial infarction are classified as ventricular free wall ruptures (FWR), ventricular septal ruptures (VSR), and papillary muscle ruptures (PMR). A combination of any two types of rupture is called "ventricular double rupture;" (VDR) and shows a specific clinical course.

3,284 patients with acute myocardial infarction (AMI) were admitted to the CCU of our hospital between April, 1973 and December, 2001, and 10 patients (8 males and 2 female, aged 54～82 years) with VDR were clinicopathologically evaluated. All were diagnosed as VDR consisting of FWR and VSR. VDR was observed in 0.30% of all patients with AMI, in 3.0% of those with FWR, and in 16.1% of those with VSR. The infarct site was anteroseptal in 3 patients, anterolateral in 3, inferior in 3, and posterolateral in 1. Two patients with inferior infarction complicated RV infarction and a patient with posterolateral infarction had healed inferior infarction.

The risk factors related to VDR were age, a history of hypertension, increased sympathetic tone to improve hemodynamic aggravation after perforation, cardiotonic agents, thrombolytic agents, delayed reperfusion, right ventricular volume overload by shunt and re-infarction. However, these factors might have played only a subsidiary role. The most important factor in VDR was the pathological findings. The site of septal perforation was the apex close to the septum-free wall junction in 9 patients and the site of rupture was also apical in 8 patients.

Four patients already had VSR on admission to our CCU. FWR developed soon after VSR was demonstrated in 4 patients. FWR and VSR occurred simultaneously in one patient. These results suggest that VSR in the apical region is a precursor of VDR and requires the earliest surgical treatment.

Surgical treatment was carried out in the operating room in 5 patients and 3 (60.0%) of them survived for 4 months or more. Two patients with rupture incidentally detected during operation for VSR were discharged and are still alive, though another one with free wall blow out rupture died 129 days after operation. Bedside thracotomy was performed in 3 patients and all of them died. (J Nippon Med Sch 2003: 70; 21–27)

Key words: ventricular double rupture, ventricular septal rupture, free wall rupture, acute myocardial infarction

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Introduction

Cardiac rupture is one of very serious and fatal complication of acute myocardial infarction[1]. Cardiac ruptures can be classified as ventricular free wall ruptures (FWR), ventricular septal ruptures (VSR), and papillary muscle ruptures (PMR). A combination of any two types of rupture is called "ventricular double rupture" (VDR) and the most common combination is FWR and VSR.

The first report of VDR associated with myocardial infarction (AMI) was done by Snyder et al. in 1940[2]. In Japan, a case of double rupture was reported by Kawakami in 1965[3], followed by a study by Sugiura and Okada[4]. In 1977, Vlodaver et al[5] pathologically evaluated 98 patients with cardiac rupture and reported 3 types of double rupture: VSR with FWR, VSR with PMR and FWR with PMR. In 1984, Edwards et al[6] pathologically evaluated VSR and confirmed the co-existence of FWR in 9 patients. Most reports regarding VDR consider just one case and there have been few clinicopathological studies.

To clarify the clinicopathological characteristics of VDR, we analyzed 10 patients with FWR complicated by VSR among patients with AMI admitted to the coronary care unit (CCU) of our hospital.

Subjects and Methods

Between April, 1973 and December, 2001, 3,284 patients with AMI were admitted to the CCU of our hospital. FWR was observed in 77 (2.3%) of all our AMI patients and VSR was observed in 62 (1.9%). The subjects of this study were 10 patients (0.30%) with FWR complicated by VSR. VDR was observed in 13.0% of the patients with FWR and in 16.1% of those with VSR. FWR was confirmed during emergency bedside thoracotomy in 4 patients, during operation for VSR in 4, and during autopsy in 2. VSR was diagnosed by right cardiac catheterization in 4 patients, Doppler echocardiography in 3, and autopsy in 3. Double rupture could be clinically diagnosed in 7 of the 10 patients.

In these patients, the clinical background, the site and size of myocardial infarction, hemodynamics, coronary lesions, treatment before rupture, the process from the onset of infarction to VDR, treatment for VDR, pathological characteristics and prognosis were analyzed.

Results

1) Clinical characteristics

Table 1 summarizes the clinical characteristics of the patients. There were 8 males and 2 females, ranging in age from 54 to 82 years (mean, 69 years). Among previous diseases, hypertension (HT) was most frequently observed (60.0%) and diabetes mellitus (DM), angina pectoris (AP), old myocardial infarction (OMI) and cerebral infarction (CI) were observed in a patient, respectively.

The infarction site was anteroseptal in 3 patients, anterolateral in 3, inferior in 3 and posterolateral in 1. Two patients with inferior infarction had RV infarction as a complication, and the patient with posterolateral infarction had old inferior infarction.

On admission, the condition of 4 patients was the Class I according Killip's classification[7], and the other 6 patients had cardiac failure (Class II～IV), including 3 patients with cardiogenic shock (Class IV). Four patients (No. 2, 7, 8 and 10) already had VSR on admission to our CCU. In 6 patients without VSR on admission, systolic blood pressure was elevated to over 150 mmHg in one patient (No. 9) and heart rate was over 90/min in 5 patients (No. 1, 3, 4, 6, 9).

Soon after admission, hemodynamic parameters were measured with a Swan-Ganz catheter in 7 patients. According to Forrester's classification[8], 3 patients were Subset I, 3 was Subset II (all patients had VSR on admission), and one was subset IV.

Before VDR occurred, arrhythmias developed in 6 patients: ventricular fibrillation (Vf) in No. 1 and 6, ventricular tachycardia (VT) in No. 9, complete AV block (CAVB) in No. 5, atrial fibrillation (Af) in No. 5, atrial premature beats (APBs) in No. 1 and 4 and ventricular premature beats (VPBs) in No. 3 and 4.

The peak level of serum CPK was unknown because of the late admission of 4 patients (No. 2, 4,

Table 1 Clinical data

Case No.	Age	Sex	Site of infarction	Previous diseases	Killip class	Forrester class	max. CK (IU/ml)	Arrhythmias	Treatment before rupure
1	64	M	Anteroseptal		I	I	689	Vf, APBs	UK, Lidocaine, Disopyramide
2 *	63	F	Anterolateral	HT	I	II	(212)		IABP, NTG, Nifedipine
3	72	M	Anterolateral	HT	I			VPBs	Lidocaine, Digoxin
4	79	M	Anterolateral	HT, DM	II	I	(610)	VPBs, APBs	IABP Catecholamine
5	82	M	Inferior (RV)		IV	IV	3,897	CAVB, Af	RV Pacing, Catecholamine
6	54	M	Inferior		II	I	910	Vf, Af	UK, Lidocaine
7 *	67	M	Posterolateral	AP, OMI	IV	II	3,700		UK, Catecholamine
8 *	59	M	Anteroseptal	HT	II		(2,290)		none
9	75	M	Inferior (RV)	HT, CI	I		(1,874)	VT	PTCA, STENT heparin, NTG
10 *	76	F	Anteroseptal	HT	IV	II	(3,099)		PTCA, STENT, heparin, IABP Catecholamine

* VSR on admission
( ); the highest value in measurements, not peak value
M; male, F; female, RV; right ventricle, HT; hypertension, DM; diabetes mellitus, AP; angia pectoris, OMI; old myocardial infarction, CI; cerebral infarction, Vf; ventricular fibrillation, APB's; atrial premature beats, VPB's; ventricular premature beats, CAVB; complete atrioventricular block, Af; atrial fibrillation, VT; ventricular tachycardia, UK; urokinase, IABP; Intraaortic balloon pumping, NTG; nitrogycerin, PTCA; percutaneaous transuluminal coronary angioplasty

9, 10). CPK level could not be measured in one patient with sudden death (No. 3). One patient (No. 8) died before CPK reached the peak. In the other 4 patients, CPK levels varied widely from 689 to 3,897 iu/L.

2) Treatment before VDR

The treatments performed before VDR are shown in the right-hand column of **Table 1**. One patient (No. 5) whose condition was complicated by VSR before admission and who sufferd brow-out immediately after admission had no adequate treatment. Percutaneous coronary intervention (PCI) was performed in 2 patients. One (No. 9) was treated 72.5 hours after the onset of infarction and the other (No. 10) was treated 23 hours after the onset in another hospital. Urokinase (600,000～900,000 units) and catecholamine were administered intravenously to 3 patients (No. 1, 6, 7), and 4 patients (No. 4, 5, 7, 10) respectively. The patient with CAVB (No. 5) underwent temporary pacing. However, when DVR occurred, the pacing catheter had been removed. Intraaortic balloon pumping (IABP) was performed in 3 patients, but double rupture could not be prevented in two patients (No. 2, 10). In another patient (No. 4), FWR occurred after discontinuation of IABP following surgical closure of the septal perforation.

3) The interval from onset of infarction to ruptures

The interval from the onset of infarction to VDR was analyzed (**Table 2**). The interval from the onset of infarction to admission to the CCU was less than 6 hours in 4 patients (No. 1, 3, 6, 8) and more than 24 hours in 5 (No. 2, 4, 5, 9, 10). The mean interval was 25.8 hours.

Concerning the relation between VSR and FWR, FWR clearly developed after VSR in 5 patients (No. 3, 4, 6, 7, 8). FWR occurred 35 minutes after VSR in one patient (No. 6) and after 40 minutes in another one (No. 3) of these 5 patients. In 2 patients, the time of VSR was unclear because it was already present at the time of admission to the CCU, and FWR

Table 2 Time from onset of AMI to rupture

Case No.	from onset to CCU	from onset to VSR (diagnostic method)	from onset to FWR (diagnostic method)	from VSR to FWR
1	5 hr. 30 min	unkown (Autopsy)	123 hr. 30 min (thracotomy in CCU)	unknown
2	70 hr.	noted at admission (Rt catheterization) Qp/Qs=?	within 145 hr. (Operation)	unknown
3	2 hr. 45 min	14 hr. 30 min (Auscultation/Autopsy)	15 hr. 10 min (Autopsy)	40 min
4	68 hr	83 hr (Rt catheterization) Qp/Qs=1.85	21 days (Operation)	17 days (Re-infarction)
5	38 hr	114 hr. 30 min (Autopsy)	114 hr. 30 min (Autopsy)	Simultaneously?
6	1 hr. 35 min	142 hr (Rt catheterization) Qp/Qs=3.28	142 hr. 35 min (thracotomy in CCU)	35 min
7	15 hr.	noted at admission (Rt catheterization) Qp/Qs=2.51	25 hr. (Operation)	within 25 hr.
8	5 hr. 45 min	noted at admission (Color flow Doppler)	7 hr. (thracotomy in CCU)	within 7 hr.
9	72 hr.	74 hr. (Rt catheterization) Qp/Qs=2.61	unknown (Operation)	unknown
10	13 days	10 days * (Rt catheterization) Qp/Qs=2.54	unknown (Operation)	unknown

* VSR occurred in another hospital and the patient was transferred to our CCU
Qp/Qs; shunt ratio

occurred at least within 7 (No. 8) or 25 hours (No. 7) after VSR. In the other patient (No. 4), FWR developed 17 days after surgical closure of the septal perforation, and it was suspected that the FWR was associated with recurrence of infarction based on chest pain and ECG change.

In the remaining 5 patients (No. 1, 2, 5, 9, 10), the relation between the two ruptures was unclear. FWR was detected during operation for VSR in 3 patients (No. 2, 9, 10), and VDR was confirmed by autopsy in the other 2 (No. 1, 5). Though the order of the two ruptures was unclear, they were thought to have occurred simultaneously in one patient (No. 5).

4) Pathological characteristics

The pathological characteristics of this VDR were evaluated based on surgical findings, including emergency bedside thoracotomy. The site and size of VSR are shown in the left-hand column of **Table 3**. The perforation site was recognized in 7 patients (70.0%) in the apical septum, in one case each in the high septum (No. 7), posterior septum (No. 9) and mid septum (No. 10) In the 7 patients with apical perforation, a linear tear was observed near the septum-apex junction at the center of necrosis where the myocardium showed severe thinning. The length of the tear was about 10 mm in many patients.

The site of FWR was the left ventricular wall near the VSR area in all patients excluding two; one (No. 4) had FWR that occurred after operation for VSR, and one (No. 7) had long tear in the septum that appeared to have extended to the longest tear in the free wall. In patient No. 4, perforation and rupture occurred separately at different times, and the free wall rupture site differed from the site of the previous VSR or surgical wound. In patient No. 7, perforation and rupture occurred through a common ostium and had large tears in the high septum and free wall, which suggested that the mechanism of the rupture differed from that in the others.

Tears of the septum and free wall occurred at 2

Table 3 Pathological findings and prognosis

Case No.	Site of VSR (diameter)	Site of FWR (diameter)	Mann's classification	Coronary lesion	Operation	Result
1	Apical septum (unkown)	Apical posterior wall (unkown)	true	LAD#6 (100%)	Thracotomy in CCU	Died
2	Apical septum (10mm)	Apical anterior wall (10mm)	true	LAD#7 (100%)	(+)	Alive
3	Apical septum (7mm)	Apical anterior wall (5mm)	unkown	LAD#6 (100%)	(−)	Died
4	Apical septum (unkown)	Posterolateral wall (unknown)	different site (post-ope) (re-infarction)	LAD#6 (75%)	(+)	Died
5	Apical septum (10mm)	RV apical posterior wall (4mm)	true (RV)	RCA#1 (90%)	(−)	Died
6	Apical septum (10mm)	Apical posterior wall (10mm)	true	RCA#1 (100%)	Thracotomy in CCU	Died
7	High septum (40mm)	Posterior wall (80mm)	different site (extension?)	RCA#2 (100%)	(+)	Died
8	Apical septum (unkown)	Apical anterior wall (5mm)	true	unkown	Thracotomy in CCU	Died
9	Posterior septum (unkown)	Posterior wall (unkown)	unkown	RCA#1 (100%)	(+)	Died
10	Mid septum (unkown)	anteriolateral wall (5mm)	unkown	LAD#7 (100%)	(+)	Alive

different sites in the same infarction area in 8 cases. In one patient (No. 5) who was clinically diagnosed with right ventricular infarction, the right ventricular wall blew out. The type of tears were evaluated according to Mann's classification[11] in 8 patients. The true type was observed in 5 patients, but no examples of the junctional type were not found and the type was undetermined in 3 patients.

The culprit lesion of the coronary artery was determined by coronary angiography in 5 (No. 2, 6, 7, 9, 10) and by autopsy in 4 (No. 1, 3, 4, 5). 7 patients showed complete occlusion of the coronary artery: 4 in LAD and 3 in RCA. PCI (PTCA and Stenting) was performed in 2 patients, one (No. 9) before VSR and the other (No. 10) after VSR. The intervention was done for the purpose of late reperfusion, and TIMI flow grade III was obtained 72.5 and 23 hours after the onset of AMI, respectively. Urokinase was administered intravenously to 3 patients (No. 1, 6, 7) before FWR, but did not bring about effective reperfusion.

5) Treatment for VDR and prognosis

After a diagnosis of VSR was established, emergency operation was performed in 4 patients. In 2 patients (No. 2, 10), FWR was detected during this operation, and they showed a good postoperative course and were discharged and alive. A patient (No. 9) whose FWR was detected during operation died of multi-organ failure, 8 days after operation. The other patient (No. 4) developed cardiac tamponade 17 days after closure of perforation and immediately underwent surgical repair, but died of renal failure 129 days after operation.

In 3 patients (No. 6, 7, 8), although a diagnosis of septal perforation was made, the free wall ruptured before or during operation. Emergency thoracotomy performed for closure was not effective in 2 patients (No. 6, 8). In patient No. 7, cardiac blow out developed in the operating room but we could not save him. In the other 3 patients (No. 1, 3, 5), VSR was not diagnosed clinically but was observed on autopsy after death of blow out. As a result, 2 patients (20.0%) with VDR survived.

Discussion

In 1977, Vlodaver et al.[5] pathologically evaluated 98 patients with cardiac rupture and reported 3 types of double rupture: VSR with FWR in 6 patients, VSR with PMR in one and FWR with PMR in

one. In 1984, Edwards et al.[6] at the same institution pathologically evaluated VSR and confirmed the co-existence of FWR in 9 patients (16.9%). This incidence of VDR in the patients with VSR in their study was similar to ours (16.1%; 10/62). On the other hand, there have been few studies on the incidence of VDR in patients with FWR. The incidence in Vlodaver's study was 10.2%, and that in our study was 13.0% (10/77).

The risk factors of cardiac rupture have been suggested to be advanced age, female gender, first attack, and hypertension. The risk factors of VDR according to our patients were (1) age (average 69 years old), (2) first attack 90% of them, (3) anterior infarction 60% of them and (4) history of hypertension 60%of them but (5) more in males than in females (8: 2). The reason why VDR occurred more in males was unknown. Other possible mechanisms of DVR are as follows: 1) increased sympathetic tone to improve hemodynamic aggravation after perforation (60% more than Killip II), 2) various cardiotonic agents (in 50%), 3) thrombolytic agents (in 30%), 4) delayed reperfusion[9] (in 20%), 5) increase in the right ventricular load by shunt (right ventricular rupture[10] occurred in 10%) and 6) re-infarction (in 10%). These factors may play only a subsidiary role, and the most important factor in VDR is the pathological findings.

Mann et al.[11] classified VDR according to pathological findings into true double rupture and junctional rupture. In the true type, tears are clearly observed both in the ventricular septum and free wall. In the junctional type, rupture is localized in the septal-free wall junctional area (**Fig. 1**). In our study, no patient had the definite junctional type, and most patients showed apical rupture near the junction and different tears in the septum.

Mann et al.[11] also reported that a systolic murmur due to VSR was heard before FWR in 5 of 7 patients, in whom the interval from the onset of infarction to detection of heart murmur (the occurrence of VSR) ranged from 15 hours to 6 days, and that from the onset of infarction to death ranged from 21 hours to 28 days. In our study, VSR occurred in the range of 14.5 hours to 10 days after the onset of AMI, and FWR occurred in the range of

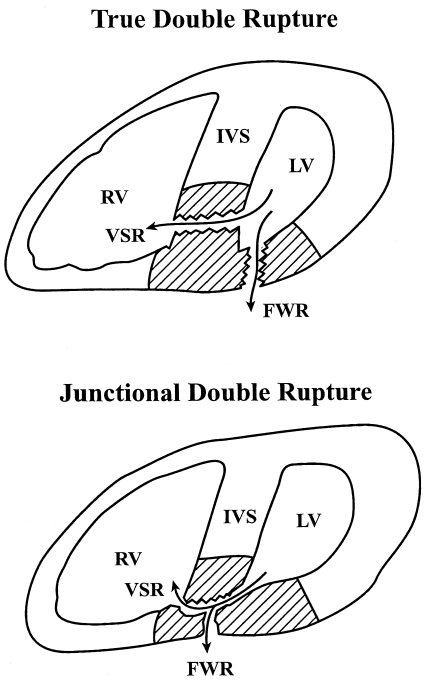

Fig. 1 Classification of the type of double rupture by Mann et al[11].
(Hatched zones indicate sites of infarction. RV; Right Ventricle, LV; Left Ventricle, IVS; Interventricular Septum)

7 hours to 21 days. FWR developed clearly after VSR in 5 patients and within 25 hours after perforation in most of the patients.

Zingone et al.[12] reported 2 patients in whom VSR occurred after repair of FWR. In one of our patients (No. 4), FWR developed after operation for VSR. As described above, we speculated that infarction developed twice in the same patient, and mechanical failure incidentally occurred in each infarct in this patient. This condition might be extremely rare.

The first operation for VDR was reported by Windsor et al. in 1976[13]. However, their 2 patients died 3 days and 16 days after operation. The first patient who survived operation was reported by Pefarre et al.[14] and rupture was incidentally detected during operation for septal perforation in this patient. In most of the subsequently reported survivors, free wall rupture was detected during operation for VSR[15–18]. However, patients with VDR with blow out type successfully treated by operation have recently been reported[19]. We performed surgical treatment in 8 of our 10 patients (including

emergency bedside thoracotomy in CCU). Three (37.5%) of them survived for 4 months or more. Two patients with rupture incidentally detected during operation for VSR were discharged and are still alive, and another one with free wall blow out rupture died 129 days after the operation.

Conclusion

Most of our VDR cases had cardiac apical infarction and the occurrence of septal perforation close to the septum-free wall junction in the apex. Among VSR associated with myocardial infarction, cardiac apical perforation close to the septum-free wall junction often leads to free wall rupture in a relatively early stage. Therefore, such perforation associated with apical infarction was considered as a precursor to double rupture, and surgical intervention should be carried out as soon as possible.

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