Signs of pericardial constriction in rupture of ventricular septum complicating myocardial infarction

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Two patients with rupture of the ventricular septum following myocardial infarction are described. In addition to the usual clinical features, both patients had signs of pericardial constriction, including raised venous pressure with a dominant systolic descent and absence of the third heart sound. There was also equilibration of right and left ventricular diastolic pressures at cardiac catheterization.

It is postulated that in some patients with ruptured ventricular septa, the pericardium does not distend in response to the acute volume loading of the heart, and cardiac constriction results.

Rupture of the ventricular septum is an uncommon complication of myocardial infarction, occurring in 1.5 to 2 per cent of cases coming to necropsy (Barnard and Kennedy, 1965). Its importance lies in its poor prognosis (Oyamada and Queen, 1961) and that appropriately timed surgical intervention may improve this prognosis (Barnard and Kennedy, 1965; Iben *et al.*, 1969; Oldham *et al.*, 1969).

Rupture of the septum most commonly follows anterior myocardial infarction (Saunders, Kern, and Blount, 1956) and is manifested by the sudden development of a cardiac murmur, usually within one week of the infarction, and associated with clinical deterioration and the development of cardiac failure or shock (Selzer, Gerbode, and Kerth, 1969). The murmur has been described as a loud, harsh systolic murmur, loudest at the lower left sternal border (Saunders *et al.*, 1956; Swithinbank, 1959; Boicourt *et al.*, 1962), though it may be best heard at the apex, making difficult the clinical distinction from ruptured papillary muscle causing severe mitral regurgitation (Selzer *et al.*, 1969).

The role of the pericardium in producing constriction in ventricular septal rupture after myocardial infarction has not previously been described. However, in the analogous situation of acute volume loading of the heart by

acute mitral regurgitation such a situation has been recognized (Bartle and Hermann, 1967). This report presents the clinical features of 2 patients with rupture of the ventricular septum after myocardial infarction, in whom there was evidence of pericardial constriction.

Case Reports

Case 1 A woman of 57 developed acute anterior myocardial infarction in June 1968. On admission, there was peripheral vasoconstriction, blood pressure was 150/90 mmHg, and she had a sinus bradycardia of 60 a minute. The central venous pressure was not raised, and there were no other signs of cardiac failure. The heart sounds were normal, without any murmurs. She was treated with rest and anticoagulants. Forty-eight hours later, a loud, harsh systolic murmur became audible, maximal at the left sternal edge. The second heart sound appeared to be single. Simultaneously, she developed left ventricular failure. The venous pressure became raised, and the liver became enlarged. She was treated with digoxin and diuretics and was transferred to the Brompton Hospital in July. On examination, the venous pressure was raised 20 cm above the sternal angle at 45°, with a dominant systolic descent. The arterial pulse was small, sharp, and without paradox. A third heart sound was not present.

Phonocardiography indicated wide splitting of the second heart sound, with an accentuated pulmonary valve closure sound (P₂) relative to the aortic valve closure sound (A₂) which was lost in the pansystolic murmur even in the pulmonary area. An electrocardiogram showed sinus rhythm, with ST segment elevation and T wave inversion

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TABLE Cardiac catheterization findings

	Case 1			Case 2	
	Pressure	Oxygen satn (%)		Pressure	Oxygen satn (%)
Superior vena cava		41			48
Right atrium	a = 20 x' = 12 v = 16 y = 14 mean = 15	39		a = 17 x' = 7 v = 14 y = 11 mean = 13	45
Inferior vena cava		42			49
Right ventricle	80/14-25	75		70/10-20	84
Pulmonary artery	80/30 mean = 48	77		70/25 mean = 48	81
Wedge	a = 25 x = 18 v = 30 y = 23 mean = 24	—		a = 28 x = 19 v = 44 y = 19 mean = 26	—
Left ventricle	100/15-25	92		110/10-30	92
Aorta	100/74	92		110/65	92
Pulmonary flow (Qp) (l./min/m ²)	7.8			5.1	
Systemic flow (Qs)	2.4			1.3	
Left-right shunt (Qp:Qs)	3.2:1			3.9:1	
Pulmonary arteriolar resistance	3.0 units × m ²			3.5 units × m ²	

in leads V₁ to V₄, with ST segment depression in leads II, III, aVF, and V₅-V₆. Chest x-ray showed pulmonary plethora and pulmonary oedema. Cardiac catheterization (Table) confirmed the wave form of the venous pulse (Fig. 1) and simultaneous left and right ventricular pressure recordings showed equalization of diastolic pressures (Fig. 2). Right ventricular systolic pressure was 80 mmHg. There was a left-to-right shunt at ventricular level, demonstrated by increased oxygen saturations, with a pulmonary-systemic

flow ratio of 3.2:1. Angiography was not performed.

Eight weeks after the acute infarction, a 3-3½ cm ventricular septal defect was closed with cardiopulmonary bypass using a Teflon patch. There was no ventricular aneurysm. As the pericardium was opened, the prominent systolic

FIG. 2 Simultaneous left ventricular (LV) and right ventricular (RV) pressure tracings in Case 1 showing equalization of pressure during diastole.

FIG. 1 Right atrial (RA) pressure tracing of Case 1 showing dominant systolic descent (X'). Simultaneous electrocardiogram shown.

FIG. 3 Phonocardiogram showing wide splitting of second heart sound in the pulmonary area (PA) and the left sternal edge (LSE). High frequency (HF) filters used. Aortic valve closure (A_2) is identified at the end of the pansystolic murmur by reference to the dirotic notch (DN) of the externally recorded carotid pulse (CAR). P_2 = pulmonary valve closure sound; INSP = inspiration; EXP = expiration; II = lead II of the electrocardiogram. Time-markers 0.04 sec.

descent of the right atrial pressure disappeared. The patient made an uneventful recovery and remains well 42 months later.

Case 2 A woman of 61 had an acute anterolateral myocardial infarct in December 1970. There were no signs of heart failure, the heart sounds were normal, and there were no murmurs. Forty-eight hours later a loud, harsh systolic murmur was heard, maximal at the lower left sternal edge. The second heart sound appeared single. The patient rapidly deteriorated, with heart failure, and treatment with digoxin and diuretics was started. The patient remained in severe heart failure and was transferred to the Brompton Hospital 7 weeks after the initial infarct.

On examination, she had a sinus tachycardia of 120 a minute, with small, sharp arterial pulses, without paradox, and blood pressure was 100/60 mmHg. The central venous pressure was raised 15 cm above the sternal angle at 45° and showed a dominant systolic descent. There was a harsh, decrescendo systolic murmur, maximal at the left sternal border, which was pansystolic to A_2 . The second sound was widely split and P_2 was accentuated. There was no third heart sound. These findings were confirmed by phonocardiography (Fig. 3). Chest x-ray showed pulmonary plethora and congestion, with moderate cardiac enlargement. Electrocardiogram showed ST seg-

ment elevation in leads V2-V4, and absence of R wave from V1-V4.

At cardiac catheterization there was a left-to-right shunt, demonstrated by increase in oxygen saturations, at ventricular level (Table) with a pulmonary-systemic flow ratio of 3.9:1. Right atrial pressure tracings confirmed the dominant systolic descent (Fig. 4) and ventricular pressure tracings were similar to those in Case 1 (Fig. 5), with a right ventricular systolic pressure of 75 mmHg. Left ventricular cineangiography showed good left ventricular contraction apart from a small apical aneurysm and confirmation of the ventricular septal defect by opacification of the right ventricle from the left ventricle.

At this time the patient seemed to be improving. Operation was withheld as it is known that the chances of successful repair are greater with the passage of time (Barnard and Kennedy, 1965;

FIG. 4 Right atrial (RA) pressure tracing of Case 2 showing dominant systolic descent.

Iben *et al.*, 1969; Oldham *et al.*, 1969). However, the patient suddenly deteriorated in January 1971, with severe chest pain and heart failure, and died. Necropsy showed only 10 ml pericardial fluid to be present. The pericardium was normal. There was external evidence of anterior myocardial infarction.

Discussion

These patients exhibited many of the typical features of rupture of the ventricular septum after myocardial infarction. Forty-eight hours after the development of anterior myocardial infarction a systolic murmur appeared, maximal at the lower left sternal border. Both patients deteriorated with severe heart failure. The chest x-rays showed pulmonary plethora and pulmonary oedema. The additional presence of a ventricular aneurysm in Case 2 is a common finding associated with post-infarction ventricular septal rupture, Selzer *et al.* (1969) reporting an incidence of up to 50 per cent.

Phonocardiography in these two patients, and in two others we have seen with rupture of the ventricular septum but without features of constriction, showed a pansystolic murmur which drowned the aortic component (A_2) of a widely split second heart sound (S_2). The length of the murmur and the relative softness of A_2 accounted for the clinical impression of a single S_2 , a feature also typical of many patients with congenital ventricular septal defects (Leatham and Segal, 1962). P_2 was louder than A_2 as is usual with pulmonary hypertensive ventricular septal defects, though wide splitting in this situation in congenital defects is unusual (Sutton, Harris, and Leatham, 1968). Left ventricular ejection time in the patients described was shorter than predicted normal values allowing for sex and heart rate (Weissler, Harris, and Schoenfeld, 1968) – this may in part account for the widely split S_2 .

Unusual findings in these patients were the following features, usually associated with pericardial constriction. The raised central venous pressure had a dominant systolic descent, confirmed by direct recording of right atrial pressure at cardiac catheterization (Fig. 1 and 4). Such a venous pulse and the absence of a third heart sound were described by Gibson (1959) in cases of severe pure pericardial constriction without significant myocardial involvement. Pressure tracings showed equilibration of left and right ventricular diastolic pressures (Fig. 2 and 5), a feature of constriction (Shabetai, Fowler, and Guntheroth, 1970) and not described as a feature of congenital ventricular septal defect despite

FIG. 5 Simultaneous left and right ventricular pressure tracings in Case 2 showing equalization of pressures in diastole.

free communication between the ventricles. In Case 1 the dominant systolic descent of the venous pressure disappeared as the pericardium was opened, suggesting that the constriction was truly pericardial and not due to myocardial disease.

The reason for such constriction is not proven, but it seems likely, as Bartle and Hermann (1967) suggested, that though the myocardium of an acutely volume loaded ventricle is able to distend rapidly the pericardium may be less distensible, resulting in effective constriction. Other cases of rupture of the ventricular septum after myocardial infarction which we have seen have not shown features of constriction, presumably because the relative pericardial/myocardial compliance has been different in these cases.

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