

Ventricular free wall rupture: sudden, subacute, slow, sealed and stabilized varieties

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Six cases of acute myocardial infarction with blood in the pericardial sac are described. In one case rapid death followed myocardial rupture leaving no time for the possibility of intervention. Of two other cases acute symptoms developing after myocardial rupture, one was operated on promptly and the other, whose condition improved on pericardiocentesis, after a delay of a few hours. Both are now long term survivors. A fourth patient probably had two episodes of rupture which apparently sealed off. He underwent cardiac catheterization, but no epicardial leak was found. Subsequently at operation a sealed myocardial rupture was detected and sutured over. The fifth patient suffered a silent myocardial rupture. A false aneurysm was diagnosed four months later and he withstood successful surgery. In the sixth patient, the course was similar to that of case 1, namely rapid death with a clinical picture suggestive of tamponade. Postmortem examination showed a covert rupture with some evidence of attempts to plug the opening. The purpose of this report is to emphasize the varying course which myocardial rupture can take.

Introduction

Since the first report of successful surgical repair of a rupture of the free wall of the ventricle during acute myocardial infarction in 1969^[1], there have been further reports of similar success^[2-4]. However, the numbers appear to be small^[5] for a condition which, after ventricular arrhythmia and cardiogenic shock, is the most frequent cause of death in acute myocardial infarction^[6].

The possibility of surgical intervention depends on the time interval between the onset of myocardial rupture and the complete collapse of the cardiovascular system. The availability of an appropriately equipped and trained surgical team is an essential precondition, while the time available for intervention is determined by the promptness of diagnosis. The latter depends to a large extent on

a high degree of suspicion in the presence of, at best, suggestive symptoms and signs.

It is known from pathological studies that myocardial rupture is not necessarily a sudden and catastrophic event, for thrombus formation is often seen at the site of rupture suggesting a more gradual evolution to rupture^[7]. Since within one year, 4 cases of free wall rupture following myocardial infarction were successfully operated on in our department, it appeared of interest to document and to confirm this variable time course of myocardial rupture.

Case reports

CASE 1

A 70-year-old man with a history of untreated mild hypertension of many years duration was admitted to the coronary care unit having experienced recurrent chest pain with nausea over a period of 5 h. Physical examination did not reveal any abnormality. ECG showed the signs of an acute inferior infarction. On the chest X-ray, grade I congestion was noted, and through a Swan-Ganz catheter only

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moderately raised pulmonary artery pressures were recorded. Hence the patient was treated with ouabain intravenously and given diuretics. Intravenous anticoagulation with heparin was commenced. About 15 h after admission he suddenly developed bradycardia with respiratory arrest. The blood pressure was unrecordable. With the use of atropine and isoprenaline there was some increase in heart rate but there was no evidence of restoration of blood circulation. Resuscitation was unsuccessful although it was begun within minutes of the onset of collapse. Pericardial puncture and transthoracic pacing were attempted. The patient expired with electro-mechanical dissociation followed by asystole.

Postmortem examination revealed an inferior infarction with myocardial rupture, and blood distending the pericardium with evident tamponade.

Comment

Here the clinical presentation of myocardial rupture was sudden and catastrophic. Recurrence of chest pain, which is said to be a frequent precursor of rupture^[4], was not noted in this patient. From the onset of tamponade there was a total suppression of the circulation with no margin left for possible intervention.

CASE 2

A 58-year-old man who was not known to be suffering from any cardiovascular disease was admitted to the coronary care unit with chest pain of 2 h duration. Physical examination did not reveal any abnormality but the ECG indicated an acute antero-septal infarction with right bundle branch block. Serial enzyme estimations confirmed the presence of an acute infarction. Intravenous anticoagulation with heparin was started and after 3 days continued with a coumarin derivative. On the day after admission, temporary pacing was instituted via a transvenous catheter and continued for 2 days because of periods of asystole. After the acute stage mobilization was slow due to evidence of cardiac decompensation. On the 21st day after infarction the patient suddenly developed ventricular fibrillation. He was promptly defibrillated and resuscitated. Cardiac asystole was noted again but subsequently nodal rhythm ensued. The circulation remained poor with a systolic pressure of about 60 mmHg. Further periods of asystole and bradycardia followed and the blood pressure was scarcely recordable. Patient was transferred back to the coronary care unit. Echocardiography with the

minivisor^[8] showed the presence of fluid in the pericardial sac. Pericardial puncture was performed and some blood withdrawn. An intra-aortic balloon pump was inserted via the right femoral artery using a percutaneous approach and counterpulsation commenced. However, the circulation remained very poor with a barely recordable blood pressure. The heart rhythm alternated between sinus tachycardia and periods of bradycardia. The patient was moved, within 2 h of the onset of collapse, to the operating theatre and an emergency operation performed. The heart was approached via a median sternotomy. On opening the pericardium a considerable quantity of dark blood was encountered. A large infarcted area was noted in the anterior wall of the left ventricle extending almost to the base of the heart. Within this was a smaller area much softened, which showed numerous small perforations. The opening was enlarged and sutures were placed between two strips of Teflon felt such that a part of the necrotic muscle was 'reefed' and excluded from the circulation. The operation was then concluded in the usual manner.

Postoperatively the patient remained haemodynamically stable. It was evident that he had developed a right hemiplegia, with aphasia and rather severe paresis of the right arm. Twenty-three days after operation the patient was discharged from hospital.

He is alive and well 20 months after his infarction without angina pectoris. His hemiplegia has lessened considerably and speech function is almost normal. He works part time.

Comment

In this case the rupture developed over a period of time and in place of one large opening there were numerous small perforations within an area of myomalacia. As a result the bleeding was not catastrophic; nevertheless, it left very little time for intervention, which in this case was fortunately successful.

CASE 3

A 68-year-old man, who was being treated with beta-blockers for hypertension but with no other known cardiovascular abnormality, was admitted to the coronary care unit after he had had recurrent attacks of pain in the chest accompanied by sweating and nausea within a few hours. He had a pulse rate of 40 min⁻¹ and his blood pressure was 90/70 mmHg. The heart sounds were normal.

Lungs were clear and there were no signs of decompensation. The ECG showed 3rd degree AV block and an acute infero-postero-lateral infarction. Chest X-ray showed grade I lung congestion. Temporary pacing was instituted and the blood pressure rose to 130/80 mmHg. AV conduction returned the next day with a prolonged PR interval. Serial enzyme determinations showed the typical rise and fall of acute myocardial infarction. Intravenous anticoagulation with heparin was started and after 4 days continued with a coumarin derivative. During staged mobilization he complained of dyspnea and hence he was treated with diuretics. On the 15th day after infarction, while watching a football match on television, patient felt that he was becoming too excited and so he withdrew to his room and lay on his bed. He did not experience any pain. A short while later he was found by a nurse, looking pale and sweating. He was cold and clammy but not unconscious. He was promptly transferred back to the CCU, where he was found to have a heart rate of 104 min^{-1} and a blood pressure of 85/0 mmHg. There were no murmurs, nor was a rub to be heard. Echocardiography showed fluid in the pericardial sac. Pericardial puncture was performed under ECG monitoring by the xiphisternal approach and 70 ml blood withdrawn. The blood pressure rose quickly to 125/65 mmHg and the patient felt better. As the staff were busy with other patients no immediate action was taken at the time. Three hours later there was once more a rapid fall of blood pressure. The patient was rushed to the operating theatre and emergency operation performed.

The heart was approached via a median sternotomy. On opening the pericardium a large quantity of old and fresh blood and clots were found. After these had been evacuated, a myocardial rupture with systolic jets of bleeding could be seen centrally placed in an infarcted area in the infero-posterior aspect of the left ventricle. The rupture was closed with sutures over Teflon felt and the operation completed in the usual manner. During the post-operative period there were episodes of paroxysmal atrial fibrillation, but haemodynamically the patient remained stable. Nineteen days after the operation, the patient was discharged from hospital in a satisfactory condition. He is now alive and well without angina or cardiac decompensation 10 months later.

Comment

In this patient, too, pain did not precede rupture. Here the rupture clearly occurred 15 days after

myocardial infarction. The bleeding into the pericardial sac was slow (it was seen to be so) and the classic signs of extreme tamponade, namely sinus bradycardia and nodal rhythm^[9], did not occur, although the sudden collapse in blood pressure provided the tell-tale sign. The adverse effects could be relieved by a relatively small (70 ml) pericardiocentesis, but this was of only temporary benefit as the bleeding persisted. Nevertheless, the rate of bleeding was sufficiently slow to permit urgent surgical treatment without lasting damage to other organs.

CASE 4

A 68-year-old man without a previous history of cardiovascular disease suffered an unprovoked manhandling and received a kick on the front of his chest. He felt severe pain which subsided somewhat in half an hour. However, he continued to experience dull pain with discomfort and breathlessness which confined him to his home. Nine days later he suffered an epileptiform fit which led to his admission to another hospital with bradycardia and signs of shock. As the ECG showed an inferior infarction he was admitted to the coronary care unit. A pacing catheter and pulmonary artery Swan-Ganz catheter were introduced. The pulmonary artery pressure was 35/13 mmHg, pulmonary capillary wedge pressure 12 mmHg and right atrial pressure 12 mmHg. Chest X-ray showed cardiothoracic ratio of 18 : 35. The patient was not on anticoagulants, but after admission heparin anticoagulation was started and continued for 7 days, until the second episode of collapse. During the next few days of observation no clinical events of note developed. Serial enzyme estimations ruled out a very recent myocardial infarction. The patient was mobilized in stages. He complained of dyspnea. One week after admission the patient collapsed suddenly. He was found to have a bradycardia and was resuscitated. Immediate echocardiography was performed and pericardial fluid demonstrated. The patient was then transferred to this hospital. On admission here, he was seen to be dyspneic and in distress. The heart rate was 100 min^{-1} and the blood pressure 110/70 mmHg with a pulsus paradoxus of about 20 mmHg. The central venous pressure was raised. Heart sounds were muffled. There was no murmur nor rub to be heard. The lungs were clear. ECG showed an inferior infarction. A Swan-Ganz catheter was introduced and the following pressures noted: right atrium 14 mmHg, pulmonary artery 26/16 mmHg, pulmonary capillary wedge

16 mmHg. Echocardiography once more demonstrated fluid in the pericardial sac. Pericardial puncture under ECG monitoring was performed via the xiphisternal approach and 250 ml blood withdrawn. Patient immediately felt relieved. His blood pressure went up to 150/70 mmHg and pulsus paradoxus disappeared. Heart rate declined to 90 min⁻¹, pulmonary capillary wedge pressure to 12 mmHg and right atrial pressure to 9 mmHg. Cardiac catheterization was then performed. Significant two vessel coronary artery disease was demonstrated in the left anterior descending artery and the right coronary artery. Left ventricular angiography showed hypokinesia of the septum and inferior wall, but no pericardial extravasation of contrast^[10] could be visualized. The next day the heart was approached by a median sternotomy. On opening the pericardial sac a large quantity of old blood and blood clots was encountered. These were evacuated and it was seen that there was no fresh or continued bleeding evident. A recent infarction could be clearly seen in the inferior wall of the left ventricle. After a close search a rupture was detected in the centre of the infarct area. This was closed with sutures over Teflon felt and the operation concluded in the usual manner. The post-operative phase was without problems. Twelve days after operation, the patient was discharged from hospital in a fair condition.

Three months later the patient was admitted with congestive cardiac failure. After investigation, he was operated on for a true aneurysm of the left ventricle. Although the aneurysm was resected successfully, the patient died 24 h after surgery.

Comment

In view of the history of chest trauma and the absence of enzymatic confirmation of myocardial infarction, the possibility that the myocardial necrosis, seen at operation, was due to contusion has to be entertained^[11]. The two episodes of collapse that the patient suffered may have been due to arrhythmia, but on both occasions bradycardia was noted. Hence one must consider the possibility of bleeding into the pericardial sac with acute tamponade, which subsided due to stoppage of bleeding. This sequence of events confirms that a myocardial rupture can seal off spontaneously^[12].

CASE 5

A 51-year-old man was admitted to another hospital with dyspnea and dizziness of a few hours

duration. Two weeks prior to admission he had suffered an attack of compressing chest pain which lasted about an hour. There was no history of cardiovascular disease. The patient was not on anti-coagulants. On examination, systolic and diastolic murmurs were heard. The ECG showed signs of an old lateral infarction. Serial enzyme estimations did not yield evidence of an acute myocardial infarction. A diagnosis of recent myocardial infarction with decompensation and possible pericarditis was made and the patient was treated with digoxin and diuretics. During this time, the patient who had been complaining of hoarseness was found to have a carcinoma of the larynx and was initially treated with radiotherapy. He was transferred to the ENT department for laryngeal biopsy and laryngectomy. The patient, who was then seen by a cardiologist, did not suffer from dyspnea, orthopnea or angina pectoris. Physical examination revealed an enlarged heart and a to-and-fro murmur in the precordium.

The X-ray showed a grossly enlarged heart shadow. Echocardiography displayed a large echo free space posterior to the heart. Cardiac catheterization was performed and left ventricular angiography revealed a false aneurysm opening from the lateral wall of the left ventricle. Coronary angiography revealed obstruction in the left circumflex artery. It was decided to deal with the laryngeal carcinoma initially and, if this were successful, to resect the false aneurysm of the left ventricle. Accordingly laryngeal biopsy was performed followed by a successful laryngectomy. No cardiovascular problems were encountered. Two months later open heart surgery was carried out. A very large false aneurysm of the left ventricle was evacuated and partially excised. The opening in the left ventricle was closed by sutures over felt.

The postoperative course was smooth. He was discharged from hospital after the customary stay and is now alive and well for 15 months after resection of the false aneurysm.

Comment

The symptoms suffered during acute infarction were not dramatic enough for the patient to visit a doctor and hence it remained unrecognized. The myocardial rupture leading to the false aneurysm^[13] appears to have occurred silently although it may have been the myocardial infarction that was silent with myocardial rupture producing the pain. As a result the false aneurysm was neither suspected nor recognized until it had grown greatly in size.

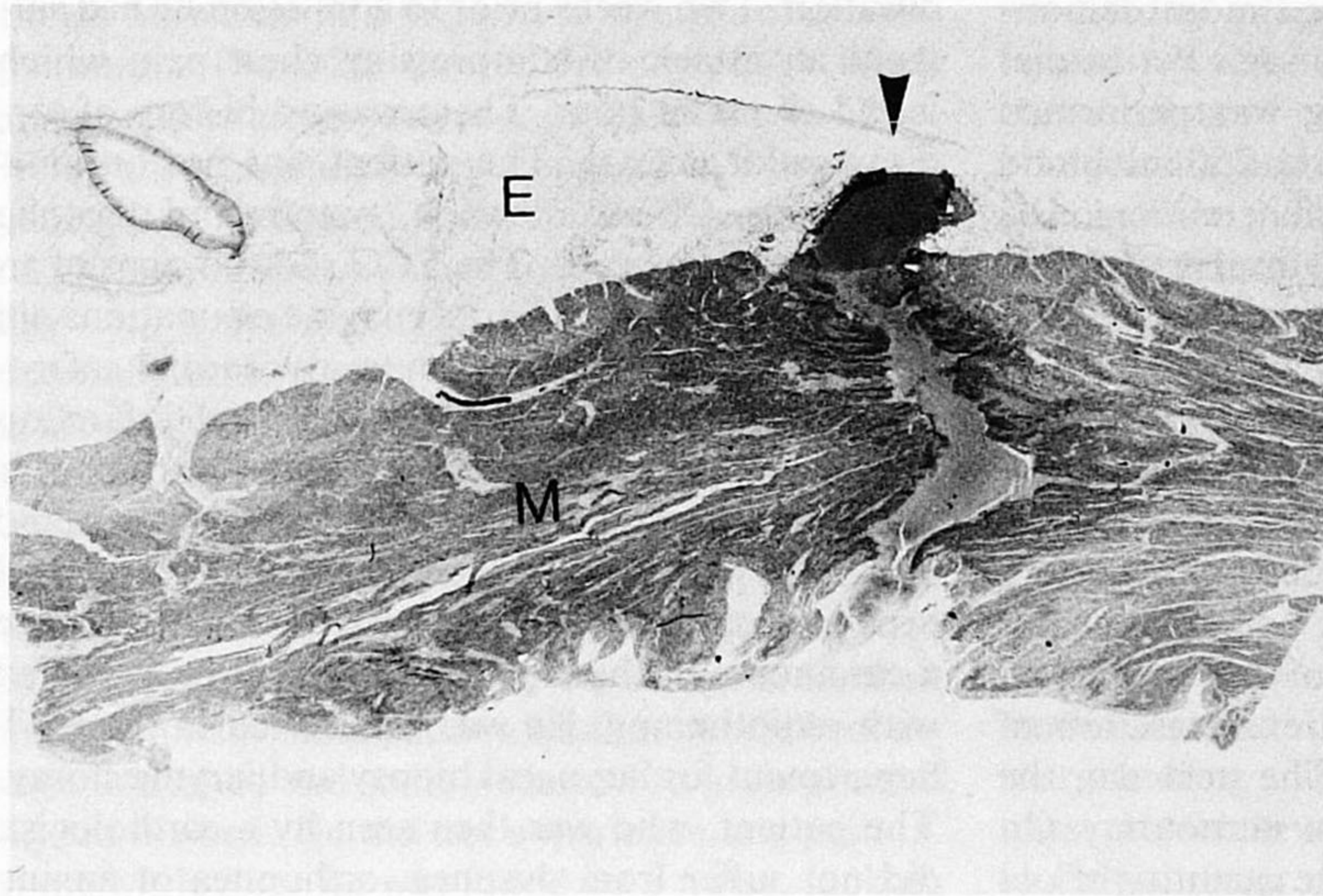


Figure 1 Section through the infarcted myocardium of case 6 (M—myocardium, E=epicardium). Note the tortuous rupture through the myocardium and the thrombotic plug (arrow) at its epicardial end.

Furthermore the development of a false aneurysm meant that though the rupture remained open, the haemodynamic situation had stabilized.

CASE 6

A 65-year-old man, without any previous history of cardiovascular disease, experienced severe retrosternal cramp-like pain with radiation to the throat and left arm at rest. He felt nauseated at the same time. The pain remained more or less constant for about 12 h and then subsided. The next day he consulted his general practitioner who ordered some lab investigations. On the strength of these a diagnosis of acute myocardial infarction was made and patient was admitted to the CCU 2 days after the onset of symptoms. On admission the patient did not have any pain but he complained of tiredness and dyspnea. Physical examination showed a regular pulse at a rate of 72 min^{-1} and a blood pressure of 105/60 mmHg. There were no murmurs and no signs of decompensation. The ECG showed evidence of a recent antero-septal myocardial infarction. The patient was not treated with anticoagulants. Five hours after admission, he suddenly became unconscious, cyanotic and apneic. The jugular venous pressure was raised, the pulses were not palpable and sinus rhythm at 90 min^{-1} was noted. Resuscitation was unsuccessful

and the patient died without having shown any sign of circulatory recovery. At postmortem about 250 ml of blood with clots were found in the pericardial sac. The myocardium showed signs of a recent transmural antero-septal infarct but despite careful examination no evidence of myocardial rupture could be identified, suggesting haemopericardium without rupture^[4]. However, on sectioning the myocardium a narrow zig-zag cleft could be identified, extending from endocardium to epicardium (Fig. 1). This cleft was filled with blood and fibrin and showed thrombus formation at both ends, suggesting an attempt at plugging the leak. There was a subtotal obstruction of the anterior descending branch of the left coronary artery and atherosclerosis in the other arteries. There was also early thrombus formation on the endocardial surface of the infarct.

Comment

This patient apparently died of cardiac tamponade due to haemopericardium 2 days after having suffered an acute myocardial infarction. At postmortem examination, superficially there appeared to be no signs of myocardial rupture. Yet a narrow rupture with perhaps attempts to seal the opening could be demonstrated by sectioning the myocardium.

Table 1 Summary of cases showing variability of presentation and development of haemodynamic effects, possibility of diagnosis and response to therapy.

	Case no.					
	1	2	3	4	5	6
Age (years)	70	58	68	68	51	65
Interval— infarct to rupture	15 h	21 days	15 days	9 days? and 16 days	14 days	2 days
Presentation	Sudden	Sudden	Gradual	Sudden	Gradual	Sudden
Course	Catastrophic	Rapid	Progressive	Recovery Relapse	Non- progressive	Catastrophic
Response to peri- cardial puncture	No im- provement	Not noticeable	Good	Good	Not performed	No im- provement
Diagnosis	Clinical? Postmortem	Echocardi- ography Pericardial puncture Surgery	Echocardi- ography Pericardial puncture Surgery	Echocardi- ography Pericardial puncture Surgery	Clinical Catheter- ization	Clinical? Postmortem
Interval rupture to operation	—	2 h	4 h	24 h after pre- sumed 2nd rupture	8 months	—
Operative result	Did not come to surgery	Haemodynamic recovery, Hemiplegia	Haemodynamic recovery, No immediate sequelae	Haemodynamic recovery, No immediate sequelae	Haemodynamic recovery, No immediate sequelae	Did not come to surgery

Discussion

Even this small set of cases should make clear the eminently treatable nature of a proportion of cases of free wall rupture which occur in a hospital with thoracic surgical facilities. In view of this, promptness of diagnosis assumes great importance. The diagnosis depends on a combination of some of the following features:

1. clinical—often recurrent chest pain followed by collapse or loss of consciousness, absent pulses or low blood pressure, raised central venous pressure and equalization of right and left filling pressures (as opposed to cardiogenic shock, where the right and left filling pressure usually move away from one another), and absent or muffled heart sounds;
2. electrocardiographic—appearance of sinus

bradycardia followed by nodal rhythm or the presence of electromechanical dissociation;

3. echocardiographic—the demonstration of an echofree space between pericardium and myocardium;

4. interventional—the presence of blood on pericardial puncture. The clinical improvement which may follow withdrawal of blood from the pericardial sac is extremely suggestive of the diagnosis. Where a diagnosis is made with reasonable certainty, emergency thoracotomy should be performed as soon as possible thereafter. No time should be lost in carrying out cardiac catheterization. However, as case 4 suggests, an occasional patient may be subjected to thoracotomy where pericardial aspiration would have sufficed^[12].

This series also demonstrates the variable time course of the clinical events following rupture which is not to be likened to the bursting of a pricked balloon but seems to involve more or less gradual weakening and infiltration of the infarct with subsequent leaking of blood into the pericardial sac. What is of importance in the first instance is the size of the leak. If this is large, tamponade and death will ensue rapidly giving no time for therapeutic intervention (case 1). If the leak is small, the crucial factor is probably the compliance of the pericardium. If the pericardium is sufficiently compliant, although tamponade develops and the circulation is depressed, death may be postponed sufficiently long for echocardiography and then pericardial puncture to avoid fatality (cases 2 and 3). Such patients should be operated on as soon as possible. If the leak is small, thrombus plugging becomes important in slowing down the clinical course, or even terminating its development, as happened in case 4. If the rupture is very gradual, the space into which the rupture opens could be confined by epicardium, thrombus formation, pericardial adhesions or surrounding structures^[13]. Furthermore, in such circumstances should two-way flow through the rupture be established, the stage would be set for the development of a false aneurysm. The rupture is then stabilized and relatively prolonged survival becomes possible (case 5).

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