Br Heart J 1982; 47: 180–2

Survival after subacute cardiac rupture

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SUMMARY A 43-year-old man collapsed suddenly, with pericardial tamponade, seven weeks after an inferior myocardial infarction. Pericardiocentesis disclosed very heavily blood stained fluid. Left ventricular angiography 10 days later showed a left ventricular aneurysm. At operation a left ventricular false aneurysm was resected and the patient recovered uneventfully.

Cardiac rupture occurs in 4 to 24% of all deaths from acute myocardial infarction.[1] It may occur from a few hours to several days after the infarction and is usually fatal. It is not usually an acute "blowout", but a gradual penetration of the necrotic myocardium by a dissecting haematoma,[1] leading to cardiac tamponade. Surgical treatment of this complication has occasionally been successful. We report a case of cardiac rupture that occurred seven weeks after myocardial infarction and was subsequently treated surgically.

Case report

A 43-year-old mechanic was admitted to hospital with a two day history of praecordial pain radiating down both arms and up to the throat. The pain was intermittent but had gradually increased in intensity. There was no significant past medical history.

On physical examination his general condition appeared good. His pulse rate was 80 per minute, regular, and his blood pressure was 160/110 mmHg. There was no evidence of cardiac failure. The electrocardiogram confirmed an acute inferior myocardial infarction. The chest *x*-ray film showed slight cardiomegaly. From day one to day three, his daily aspartate transaminase levels were 23, 38, 50 IU/l (normal <18), and hydroxybutyrate dihydrogenase levels 172, 310, and 290 IU/l (normal <140). He made an uneventful recovery and was discharged home seven days after admission, his blood pressure having settled to 120/70 mmHg. On review as an outpatient four weeks later, he was asymptomatic and his blood pressure was 140/95 mmHg.

Two weeks later, he had a sudden onset of dizziness followed by two transient syncopal episodes. This was soon followed by upper chest pain which radiated to the throat and was aggravated by deep inspiration and lying flat. On examination he was pale, cold, clammy, and dyspnoeic. Pulse was 120 per minute, regular, and of low volume. The systolic blood pressure by palpation was 70 mmHg on expiration, falling to 50 mmHg on inspiration. The jugular venous pressure was raised 8 cm above the sternal angle. The heart sounds were inaudible. The electrocardiogram showed the presence of the previous inferior myocardial infarction. An anteroposterior portable chest *x*-ray film showed probable cardiomegaly. An echocardiogram showed a large echo-free space anteriorly and posteriorly, indicating a pericardial effusion.

A diagnosis of pericardial tamponade was made and pericardiocentesis was carried out using the apical approach. On removal of 67 ml heavily blood-stained fluid, there was a dramatic clinical improvement and the blood pressure rose to 110/70 mmHg. The pericardial fluid haemoglobin was 10·4 g/dl and the circulating blood haemoglobin was 14·4 g/dl; the erythrocyte sedimentation rate was 22 mm/hour. He was transferred by ambulance to the Middlesex Hospital Cardiothoracic Surgical Unit, with a pericardial drain *in situ* but closed off. His general condition continued to improve so operation was deferred.

Left ventricular angiography, 10 days after his collapse, showed a large saccular posteroinferior left ventricular aneurysm (Fig.). This showed paradoxical movement and there was a suggestion of mural thrombus within it. Coronary arteriography showed a normal left coronary artery and a right coronary artery which was totally obstructed distal to the right ventricular branch.

At operation, 17 days after his collapse, excision of

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†Since this paper was written Dr Rissen has died.

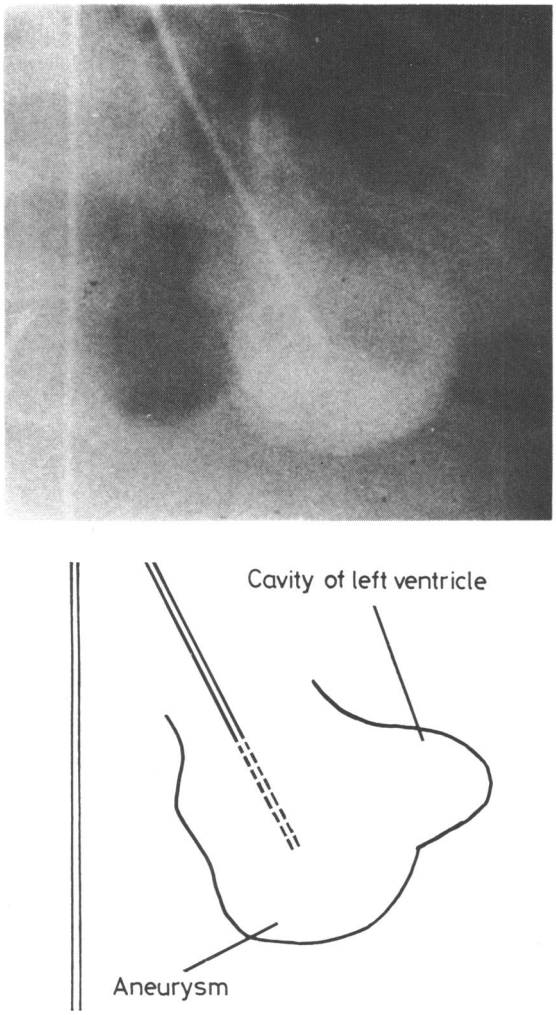

Fig. *Systolic frame from left ventricular angiogram.*

the left ventricular false aneurysm was carried out on cardiopulmonary bypass. The wall of the aneurysm was less than 1 mm thick. The sac was extensive and a defect in the posterior left ventricular wall measuring 7·6 by 2·5 cm was identified. It had smooth and rather soft muscular margins leading into the false aneurysm. The margins of the sac were separated from the heart and the ventricular defect closed with interrupted 2/0 Mersiline sutures and reinforced with a running suture superficially.

Postoperatively he was hypertensive and was treated with a nitroprusside infusion. His subsequent course was uneventful and he was discharged home two weeks after operation. He remains well six months later.

Discussion

Cardiac rupture usually leads to rapid deterioration and death within a period of minutes to hours, because of cardiac tamponade. Efforts to identify the causes of rupture have been inconclusive. Rupture is more common in the elderly, being commonest in the seventh and eighth decades,[2] and rare below 50 years of age.[3] Women are more likely to suffer cardiac rupture than men.[4] About 50% of ruptures occur within the first three days after myocardial infarction.[2]

Cardiac rupture is not usually an acute "blowout" phenomenon but is a gradual process that begins with small epithelial tears, which permit the formation of a haematoma that dissects through the necrotic myocardium. Death results from cardiac tamponade rather than pump failure.[1]

Repeated and prolonged chest pain may frequently precede cardiac rupture[2] and is thought to be the result of slow leakage of blood into the pericardial sac.[5] A possible complication of this is the formation of a left ventricular false aneurysm which, in contrast to a true aneurysm, may rupture and lead to death.[5]

The physical signs consist of sudden hypotension, rapidly increasing venous pressure, pulsus paradoxus, deepening cyanosis, and stupor. Abrupt onset of bradyarrhythmias in patients with acute transmural myocardial infarction showing sudden syncope should arouse suspicion of cardiac tamponade.[6] Echocardiography in this situation is extremely helpful as it is a rapid, non-invasive method of confirming a pericardial effusion.

We have found reports of successful surgical repair in 11 cases of cardiac rupture.[1 7-9] Once the diagnosis is made the most important step is pericardiocentesis. A drain may be left in the pericardial sac during transport to the theatre or even during interhospital transfer.

In this case the improvement occurring after pericardiocentesis was maintained, so emergency surgery was not needed and repair was undertaken as an elective procedure.

We wish to thank Mr M F Sturridge for providing the details of the operative findings.

References

1 Kendall RW, DeWood MA. Post-infarction cardiac rupture: surgical success and review of the literature. *Ann Thorac Surg* 1978; 25: 311–5.
2 London RE, London SB. Rupture of the heart: a critical analysis of 47 consecutive autopsy cases. *Circulation* 1965; 31: 202–8.
3 Sievers J. Cardiac rupture in acute myocardial infarction. *Geriatrics* 1966; 21 (July): 125–30.

4 Lewis AJ, Burchell HB, Titus JL. Clinical and pathologic features of post-infarction cardiac rupture. *Am J Cardiol* 1969; **23**: 43–53.
5 Van Tassel RA, Edwards JE. Rupture of heart complicating myocardial infarction. *Chest* 1972; **61**: 104–16.
6 Mogenson L, Nyquist O, Orinius E, Sjogren A. Studies in myocardial rupture with cardiac tamponade in acute myocardial infarction: II Electrocardiographic changes. *Chest* 1972; **61**: 6–10.
7 Anagnostopoulos E, Beutler S, Levett JM, Lawrence JM, Lin CY, Replogle RL. Myocardial rupture: major left ventricular infarct rupture—treated by infarctectomy. *JAMA* 1977; **238**: 2715–6.
8 O'Rourke MF. Subacute heart rupture following myocardial infarction. *Lancet* 1973; **ii**: 124–6.
9 Mahoney PGC, Slesser BV, Baigent DF, Humber PJB, Slade PR, Lawson CW. Subacute cardiac rupture after myocardial infarction. *Br Med J* 1976; **i**: 747.

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