An ominous complication of acute myocardial infarction

MM El-Omar, K Ray, M Rosin, M Been

A 68-year-old man was admitted to hospital with severe cardiac chest pain. An electrocardiogram (ECG) showed evidence of an acute myocardial infarction with ST segment elevation in the inferior leads. He was treated with streptokinase and made an uneventful recovery. At routine follow-up six weeks later, he gave a history of intermittent chest pains and exertional dyspnoea since leaving hospital. An exercise treadmill test was positive in stage one of the Bruce protocol. Coronary angiography revealed occlusion of the left anterior descending and right coronary arteries and left ventricular angiography revealed an unexpected finding (figure 1).

Question

What is the most likely diagnosis?
Answer

Ventricular pseudoaneurysm (VPA). The left ventricular angiogram shows a large sac filled with contrast medium, arising from the inferior aspect of the heart. Of note is the characteristically narrow base compared to the much wider fundus in contradistinction to a true ventricular aneurysm. The patient was referred urgently for cardiac surgery. At thoracotomy, he was found to have a large thin-walled pseudoaneurysm communicating with the left ventricular cavity via a small muscular defect in the posterior left ventricular wall (figures 2 and 3). The false aneurysm was resected and the left ventricular wall repaired with Teflon pledgets. Venous grafts were fashioned to the left anterior descending and posterior descending arteries. The patient made a good recovery.

Comment

Cardiac rupture is a catastrophic complication of acute myocardial infarction responsible for up to 20% of all in-hospital deaths. It can take several forms including free wall rupture, ventricular septal rupture, and papillary muscle rupture. VPA, found in 0.5% of patients referred for cardiac catheterisation in one series, is the result of ventricular free wall rupture followed by containment of the resulting haemopericardium by adhesions between the pericardium and epicardium. With time, the periphery of the haematoma becomes organised into fibrous tissue devoid of myocytes or coronary arteries. Box 1 lists the commonest causes and box 2 the commonest presentations of a VPA. It is worth noting that a VPA may remain silent for many years.

<table>
<thead>
<tr>
<th>VPA: causes</th>
</tr>
</thead>
<tbody>
<tr>
<td>myocardial infarction</td>
</tr>
<tr>
<td>cardiac surgery</td>
</tr>
<tr>
<td>chest trauma</td>
</tr>
<tr>
<td>endocarditis</td>
</tr>
</tbody>
</table>

Box 1

Features that help distinguish false from true ventricular aneurysms are listed in box 3. Diagnosis of a VPA can be made by transthoracic or transoesophageal echocardiography and radionuclide ventriculography but cardiac catheterisation offers better definition of the site and extent of the pseudoaneurysm and provides an accurate assessment of left ventricular function and coronary anatomy. Cardiac catheterisation may help distinguish between a false and a true ventricular aneurysm by demonstrating coronary arteries draping the surface of the latter. Although true ventricular aneurysms usually have a wide base compared to the width of the fundus, some have the reverse pattern, making them indistinguishable from false aneurysms. In such cases, the ultimate distinction between the two can only be made by pathological examination. If, however, a clinical diagnosis of VPA is likely, then surgical resection should be per-
Main differences between true and false ventricular aneurysms

<table>
<thead>
<tr>
<th>Pseudoaneurysm</th>
<th>True aneurysm</th>
</tr>
</thead>
<tbody>
<tr>
<td>rare</td>
<td>common</td>
</tr>
<tr>
<td>myocardium ruptured</td>
<td>myocardium locally stretched</td>
</tr>
<tr>
<td>narrow base</td>
<td>wide base (most, but not all)</td>
</tr>
<tr>
<td>no myocardial elements in wall</td>
<td>myocardites +/- coronary arteries in wall</td>
</tr>
<tr>
<td>high risk of rupture</td>
<td>low risk of rupture</td>
</tr>
<tr>
<td>surgical resection essential</td>
<td>surgery indicated if resistant symptoms</td>
</tr>
</tbody>
</table>

Box 3

formed without delay since these aneurysms, unlike true ventricular aneurysms, have a propensity to rupture regardless of size or symptoms.7,8

Conclusions

VPA formation is an ominous complication of myocardial infarction. If diagnosed, surgical resection, with coronary artery bypass grafting if indicated, should be performed without delay. If left untreated, a VPA is likely to rupture with an invariably fatal outcome.

Final diagnosis

Left ventricular pseudoaneurysm.

Keywords: ventricular pseudoaneurysm, myocardial infarction


Spontaneous aortic rupture in a 22-year-old

Muhammed Ashraf Memon, Caroline Mary Nicholson, Jill Clayton-Smith

A previously healthy 22-year-old Caucasian man was admitted following a sudden onset of an acute abdominal and lower back pain, collapse and hypotension and was rushed to theatre with suspected acute haemorrhage of unknown origin. There was no history of any trauma, past or present and the patient was not engaged in any strenuous activity prior to experiencing the above episode. Urgent exploratory laparotomy revealed rupture of the anterior aspect of the infra-renal abdominal aorta. During repair it was found that all the vessels, including the abdominal aorta, were of very small calibre and were very friable. This caused immense problems during the anastomosis of the graft and also led to damage to the inferior vena cava during the initial dissection. Successful graft anastomosis and satisfactory haemostasis was eventually achieved and mass closure of the abdomen was carried out.

Per- and post-operatively the patient received 40 units of blood, 18 units of fresh frozen plasma and seven units of platelets. The patient developed disseminated intravascular coagulopathy postoperatively and died within 38 hours of surgery.

Post-mortem examination raised the suspicion of some 'connective tissue disorder' because the aortic wall and skin were found to be very fragile during dissection. Specimens from the aorta were sent for histopathological examinations and the opinion of a geneticist. The patient's family received genetic counselling to rule out the prevalence of disease in other close family members.

Question

What is the most likely diagnosis?
An ominous complication of acute myocardial infarction.

M. M. El-Omar, K. Ray, M. Rosin and M. Been


doi: 10.1136/pgmj.72.847.309