Penetration of the left ventricular myocardium by benign peptic ulceration: two cases and a review of the published work

C PORTEOUS, D WILLIAMS, A FOULIS, BA SUGDEN

From the Departments of Surgery and Pathology, Western Infirmary, Glasgow

SUMMARY Two cases of penetration of the left ventricular myocardium by benign peptic ulcer are reported. Twenty five similar cases in the world published work are reviewed. The condition is only possible when there are fibrous adhesions between the stomach and diaphragm and the pericardium. In addition, the left lobe of the liver may be small. Alternatively, an ulcer within a hiatus hernia may erode into the left ventricle.

Erosion of a benign peptic ulcer into the myocardium of the left ventricle is rare, and fistulae between the left ventricle and stomach are very rare. There are 21 cases in the world published work, each describing a fistula between a benign peptic ulcer of the stomach and the left ventricle of the heart. In addition, there are four cases in which the myocardium of the left ventricle was penetrated but no fistula formed (Table). We report a further two cases in which a peptic ulcer has eroded into the heart. The first had a fistula between a benign gastric ulcer and the left ventricle. In the second case the connection was between a chronic gastric ulcer and the right coronary artery.

Case reports

CASE 1
An 86 year old woman presented with light headedness and haematemesis. She had no acute abdominal pain but had a short history of melena. Seven years previously she had collapsed and results of investigations had shown iron deficiency anaemia, which was thought to be due to chronic blood loss from a fixed sliding hiatus hernia diagnosed by barium meal. Five years later she was again admitted after a collapse. An electrocardiogram showed a subendocardial anterolateral infarction. Twelve months later she had a brisk haematemesis after treatment with naproxen for osteoarthritis. This was followed six months later by a further episode of haematemesis. These gastrointestinal bleeds were all attributed to her hiatus hernia, and after her fourth episode she received cimetidine. On admission, after her fifth haematemesis, she was pale, her pulse rate was 120 beats/min, and her blood pressure was 90/60 mmHg. Her abdomen was not tender. Rectal examination showed melena, and haemoglobin concentration was 7-8 g/dl. She remained in a stable condition for 6 h, but she then vomited bright red blood several times and became profoundly shocked. In spite of aggressive resuscitation she died.

Pathological findings
The oesophagus and stomach were full of blood. There was a hiatus hernia of 6 cm diameter fixed to the left side of the pericardium by fibrous adhesions and also to the left lateral aspect of the heart. The hernia was of the sliding type and was partially reducible. Pressure on the stomach reinflated the hernia and also caused blood to re-enter the heart. A 2-5 cm diameter benign gastric ulcer in the hiatus hernia had eroded through the pericardium and the myocardium of the left ventricle to form a fistula, which entered the left ventricle just anterior to the posterior mitral cusp (Fig. 1). Adjacent cardiac muscle showed old ischaemic fibrosis, the distribution of which was in keeping with the clinical history of an anterolateral infarction. A probe of 2 mm diameter could be passed into the heart through the fistula (Fig. 2). Small coronary vessels in the myocardium surrounding the ulcer showed pronounced endarteritis obliterans.

CASE 2
A 76 year old woman presented as an emergency having collapsed. A recent bowel motion was described as consisting of 1 litre of dark red material. There was no history of pain and at no time did...
Fig. 1  A chronic peptic ulcer on the right hand side of this figure has eroded into the left ventricle just below the mitral valve.

Fig. 2  A probe entering the hiatus hernia and passing through the fistula emerges within the right ventricle.
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She vomited any blood. Four months previously she had had an inferior myocardial infarction. On examination she was pale and breathless at rest. Her pulse rate was 90 beats/min, blood pressure 100/60 mmHg, and she was not feverish. The abdomen was not tender and bowel sounds were active. Rectal examination showed dark red clotted blood. Haemoglobin concentration was 7·6 g/dl and she was therefore transfused six units of packed cells. An electrocardiogram showed the old inferior myocardial infarction with some further acute changes evident in the anterior leads. Diverticulitis was thought to be the cause of this haemorrhage and therefore endoscopy was not performed. Initially, she responded well to conservative treatment, but during the next nine days she had several episodes of melaena and hypotension, which resulted in the deterioration of her condition and her death.

Pathological findings
The left lobe of the liver was small. The fundus of the stomach was adherent to the inferior aspect of the diaphragm normally covered by the liver, and the left ventricle of the heart was joined to this by fibrous adhesions. At that site there was a benign gastric ulcer, 5 cm in diameter, which had penetrated to within 2 mm of the lumen of the left ventricle (Fig. 3). Two vessels were evident in the base of this ulcer and these were identified as branches of the right coronary artery. The ulcer had eroded through these vessels, which were the main source of bleeding. There was altered blood in the ileum and colon. A fibrinous pericarditis with a small effusion was noted. There was an area of organising infarction at the apex of the heart in the distribution of the right coronary artery. The base of the ulcer was fibrous with endarteritis of small vessels.

Discussion
Perforation of the left ventricular myocardium by benign gastric ulceration was first described in 1880 by Oser,1 and a more complete pathological description of the same case was provided by Chiari.2 Since then, there have been only 20 other cases described in the world published work. Four other cases are described in which the left ventricular myocardium was penetrated but no fistula with the lumen of the heart was identified (Table). The presenting symptom in all but two cases in the series was haematemesis, and in these two cases penetration of the myocardium was an incidental finding at necropsy. Melaena was described in only eight cases.2 3 5 10 Contrary to what might be expected, exsanguination and rapid death occurred in only four patients, three of whom survived until admission to hospital. Of the 24 patients admitted to hospital 17 survived more than

Fig. 3 Only 2 mm of myocardium is left between the base of this chronic peptic ulcer and the lumen of the left ventricle of the heart.
<table>
<thead>
<tr>
<th>Author</th>
<th>Year</th>
<th>Age</th>
<th>Sex</th>
<th>Fistula</th>
<th>Period between 1st bleed and death</th>
<th>Size of ulcer</th>
<th>Associated condition</th>
<th>Pathological features</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tylecote</td>
<td>1913</td>
<td>70</td>
<td>F</td>
<td>Yes</td>
<td>3-5 days</td>
<td>1 × 1 cm</td>
<td>Nine month history of left sided chest pain and epigastric pain. Haematemesis. Melaena.</td>
<td>Fibrous adhesion between stomach, diaphragm, and pericardium. Benign gastric ulcer on anterior wall of stomach.</td>
</tr>
<tr>
<td>Askanazy</td>
<td>1926</td>
<td>61</td>
<td>M</td>
<td>No</td>
<td>Incidental finding at necropsy</td>
<td>5-5 × 3-5 cm</td>
<td>Six year history of intermittent melaena. Epigastric seizure just before death.</td>
<td>Fibrous adhesions between stomach, diaphragm, and pericardium. Benign peptic ulcer on lesser curve. No fistula.</td>
</tr>
<tr>
<td>Erhardt</td>
<td>1933</td>
<td>63</td>
<td>F</td>
<td>No</td>
<td>Incidental</td>
<td>3 × 2 cm</td>
<td>Inoperable sigmoid carcinoma leading to death.</td>
<td>Death due to sigmoid carcinoma. Incidental finding: benign peptic ulcer on anterior wall of stomach. Penetration left ventricle. No fistula. Two other gastric ulcers.</td>
</tr>
<tr>
<td>Johannessen</td>
<td>1946</td>
<td>40</td>
<td>M</td>
<td>Yes</td>
<td>5 days</td>
<td>3 × 2 cm</td>
<td>No history. Haematemesis. Melaena.</td>
<td>Adhesions between stomach, diaphragm, and heart. Benign peptic ulcer on lesser curve.</td>
</tr>
<tr>
<td>Bittman (1)</td>
<td>1960</td>
<td>44</td>
<td>M</td>
<td>Yes</td>
<td>60 h to operation. 24 h after operation.</td>
<td>5 × 5 cm</td>
<td>Long peptic ulcer history. Haematemesis resulting in laparotomy. Subtotal gastric resection. Patient died 24 h after surgery.</td>
<td>Adhesion between stomach, diaphragm, and heart. Benign peptic ulcer at resection line.</td>
</tr>
<tr>
<td>Bittman (2)</td>
<td>1960</td>
<td>62</td>
<td>F</td>
<td>Yes</td>
<td>24 h</td>
<td>3-5 × 3-5 cm</td>
<td>18 year history of peptic ulcer. Oesophagogastrectomy for gastric ulcer 12 months before death. Heartburn.</td>
<td>Postoperation fibrous adhesion between stomach, diaphragm, and heart. Benign peptic ulcer on resection line.</td>
</tr>
<tr>
<td>Ritz</td>
<td>1966</td>
<td>69</td>
<td>M</td>
<td>Yes</td>
<td>12 days</td>
<td>2 × 2 cm</td>
<td>Perforated gastric ulcer 9 years before death. Haematemesis resulting in gastrectomy. Death 24 h after operation.</td>
<td>Postoperation fibrous adhesion between stomach, diaphragm, and pericardium. Benign peptic ulcer in fundus of stomach.</td>
</tr>
</tbody>
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</thead>
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<tr>
<td>Meinecke</td>
<td>1967</td>
<td>39</td>
<td>M</td>
<td>Yes</td>
<td>10 h</td>
<td>3 x 3 cm</td>
<td>Long history of peptic ulcer disease.</td>
<td>Adhesions between stomach, diaphragm, and heart. Benign peptic ulcer on lesser curve.</td>
</tr>
<tr>
<td>Kiss</td>
<td>1967</td>
<td>89</td>
<td>F</td>
<td>Yes</td>
<td>10 h</td>
<td>3 x 1-5 cm</td>
<td>Long history of hiatus hernia.</td>
<td>Hiatus hernia with peptic ulcer.</td>
</tr>
<tr>
<td>Maroske</td>
<td>1972</td>
<td>59</td>
<td>M</td>
<td>Yes</td>
<td>20 min</td>
<td>10 x 8 cm</td>
<td>Left phrenic nerve paralysis and left diaphragm elevation after war injury. Long history of left sided abdominal pain.</td>
<td>Left diaphragm raised and adherent to the left lobe of liver, heart, and left lung. Left lobe of liver normal size. Ulcer penetrated liver, diaphragm, and left ventricle. Severe endarteritis resulting in myocardial infarction.</td>
</tr>
<tr>
<td>Lam</td>
<td>1974</td>
<td>67</td>
<td>M</td>
<td>Yes and to left atrium.</td>
<td>2 days</td>
<td>3 x 2 cm</td>
<td>Gastric carcinoma resected. Haematemesis.</td>
<td>No evidence of local recurrence of gastric ulcer. Fistula with left ventricle and left atrium suggested it may have been due to ischaemia.</td>
</tr>
<tr>
<td>Kennedy</td>
<td>1983</td>
<td>81</td>
<td>F</td>
<td>Yes</td>
<td>8 h</td>
<td>3 x 1 cm</td>
<td>Hiatus hernia.</td>
<td>Pericardial cavity obliterated by adhesions. Fibrosis and endarteritis Obliterans in the underlying myocardium.</td>
</tr>
</tbody>
</table>

6 h; 13 of those survived more than 24 h, the mean period of survival being three days. A possible explanation for this remarkable feature was first postulated by Grosse, who thought that hypotension together with the tamponading effect of a stomach turgid with blood would be enough to stop the haemorrhage, at least for a limited period. Despite this, surgery was attempted in only four cases, and in no case was the diagnosis made clinically. Myocardial infarction was diagnosed before death in three cases. Historical cases, however, may have lacked modern techniques for diagnosing myocardial infarction. One other case had pathological evidence of myocardial infarction. Fourteen cases had a previous history suggestive of peptic ulcer disease. The ulcers tended to be large with a mean diameter of 3 cm. All ulcers were histologically benign chronic peptic ulcers. The only unusual ulcer, in which a small piece of glass was found in the base, was described by Chiari. Endarteritis obliterans is a commonly described feature. Only Chiari and Tylecote described fatty change in the myocardium surrounding the ulcer. Twenty cases were associated with fibrous adhesions between the stomach and diaphragm. Only five reports described cases where this was not a feature. In each case the ulcer had developed within a paraoesophageal hiatus hernia. Kissel pointed out that the fundus or lesser curve of the stomach must come into contact with the inferior aspect of the diaphragm for a fistula to develop and that a small left hepatic lobe should be found in each case. In the case described by Grosse and Maroske, however, the left lobe of the liver was normal in size but, in addition, the liver had been penetrated by the ulcer. In the second case presented here the left lobe of the liver was notably small. Absence of fibrous adhesions between visceral and parietal pericardium results in a pneumopericarditis but no fistula. Four cases have been described in which a chronic gastric ulcer penetrated the myocardium of the left ventricle but failed to establish a fistula as in case 2. Three of these were incidental findings at necropsy. The other case reported by Matthews was similar to our second case in that the source of the blood in the haematemesis was a coronary artery. Lam described a case where the perforation was at the atrioventricular junction, resulting in a fistula between the stomach and both the left atrium and ventricle.

Conclusion

Three features allow a peptic ulcer to penetrate the heart. Firstly, the ulcer may be located high in the stomach or within a hiatus hernia; secondly, there must be fibrous adhesions between the stomach,
diaphragm, and pericardium; and, thirdly, the left lobe of the liver may be small, allowing the stomach to come into contact with the diaphragm.

No patient has yet survived this rare condition. The advent of modern fibroptic equipment and routine endoscopy after haematemesis, however, makes early diagnosis feasible. An awareness that a major haematemesis is from a chronic ulcer in the lesser curve or fundus of the stomach, together with a history of pericarditis with or without a myocardial infarction, should alert doctors to the possibility of penetration of the left ventricle.

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Requests for reprints to: Mr C Porteous, University Department of Surgery, Western Infirmary, Glasgow, Scotland.
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C Porteous, D Williams, A Foulis and B A Sugden

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