

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

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**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 melaena. 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

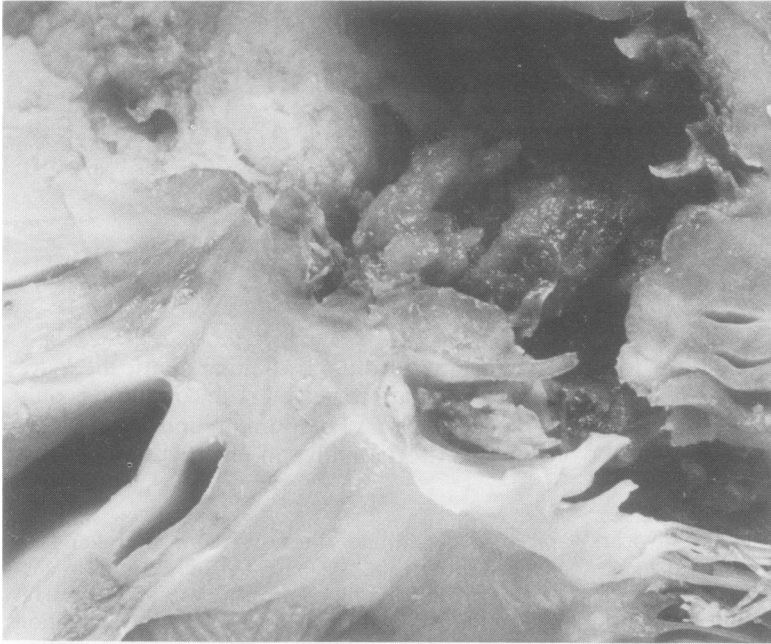
received cimetidine. On admission, after her fifth haematemesis, she was pale, her pulse rate was 120 beats/min, and her blood pressure was 90/60 mmHg. Her abdomen was not tender. Rectal examination showed melaena, and haemoglobin concentration was 7.8 g/dl. She remained in a stable condition for 6 h, 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.*

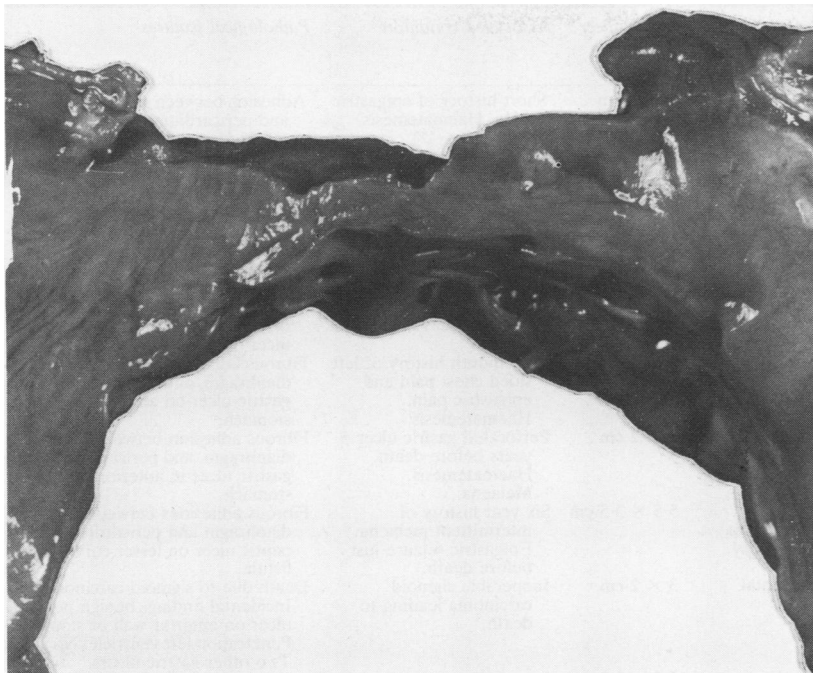


Fig. 3 Only 2 mm of myocardium is left between the base of this chronic peptic ulcer and the lumen of the left ventricle of the heart.

she vomit 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,<sup>1</sup> and a more complete pathological description of the same case was provided by Chiari.<sup>2</sup> 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.<sup>2,3,5,10</sup> 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

<i>Author</i>	<i>Year</i>	<i>Age</i>	<i>Sex</i>	<i>Fistula</i>	<i>Period between 1st bleed and death</i>	<i>Size of ulcer</i>	<i>Associated condition</i>	<i>Pathological features</i>
Oser Chiari <sup>1</sup>	1880	71	F		3-5 days	2 × 2 cm	Short history of epigastric pain. Haematemesis. Melaena.	Adhesion between stomach, diaphragm and pericardium. Fatty changes in myocardium. Small piece of glass in ulcer. Benign ulcer on lesser curve.
Brenner <sup>6</sup>	1881	55	F	Yes	4 days	10 × 8 cm	Long history of symptoms of peptic ulcer. Haematemesis. Melaena.	Adhesion between stomach, diaphragm and pericardium. Benign ulcer on lesser curve.
Finney <sup>3</sup>	1886	19	M	Yes	30 min	2 × 3 cm	Rheumatic fever. Long history of left sided chest pain. Melaena.	Rheumatic pericarditis. Adhesion between stomach, diaphragm and pericardium. Benign ulcer on anterior wall of stomach.
Brunniche <sup>7</sup>	1887	40	M	Yes	20 h	2 × 3 cm	No history of ulcer. Haematemesis. Melaena.	Adhesions between stomach, diaphragm and pericardium. Benign ulcer on lesser curve.
Tylecote <sup>19</sup>	1913	70	F	Yes	3-5 days	1 × 1 cm	Nine month history of left sided chest pain and epigastric pain. Haematemesis.	Fibrous adhesion between stomach, diaphragm, and pericardium. Benign gastric ulcer on anterior wall of stomach.
Salmony <sup>8</sup>	1922	50	M	Yes	Found dead	2 × 2 cm	Perforated gastric ulcer 5 years before death. Haematemesis. Melaena.	Fibrous adhesion between stomach, diaphragm, and pericardium. Benign gastric ulcer in anterior wall of stomach.
Askanazy <sup>20</sup>	1926	61	M	No	Incidental finding at necropsy	5.5 × 3.5 cm	Six year history of intermittent melaena. Epigastric seizure just before death.	Fibrous adhesions between stomach diaphragm and pericardium. Benign peptic ulcer on lesser curve. No fistula.
Erhardt <sup>22</sup>	1933	63	F	No	Incidental	3 × 2 cm	Inoperable sigmoid carcinoma leading to death.	Death due to sigmoid carcinoma. Incidental finding: benign peptic ulcer on anterior wall of stomach. Penetration left ventricle. No fistula. Two other gastric ulcers.
Wassmer <sup>21</sup>	1943	61	M	No	No bleeding noticed	5.5 × 3.5 cm	Long history of peptic ulcer with haematemesis. Haematemesis.	Adhesion between stomach, diaphragm, and heart. Benign ulcer on anterior wall of stomach.
Johannessen <sup>5</sup>	1946	40	M	Yes	5 days	3 × 2 cm	No history. Haematemesis. Melaena.	Adhesions between stomach, diaphragm, and heart. Benign peptic ulcer on lesser curve.
Dietrich <sup>8</sup>	1947	49	M	Yes		2 × 2 cm	Three year history of symptoms of peptic ulcer. Haematemesis.	Paraoesophageal hiatus hernia with benign peptic ulcer. Left lobe of liver normal.
Rappert <sup>23</sup>	1950	71	M	Yes	36 h	2 × 2 cm	No relevant history.	Adhesions between stomach, diaphragm, and pericardium. Benign ulcer in lesser curve of stomach.
Ladanyi <sup>12</sup>	1954	54	M	Yes	24 h	3 × 3 cm	Long history of gastric ulcer. Haematemesis resulted in laparotomy. Fistula diagnosis during operation. Patient died during operation.	Adhesions between stomach, diaphragm, and pericardium. Benign peptic ulcer on lesser curve.
Pendel <sup>15</sup>	1958	79	F	Yes	30 h	3 × 2 cm	Short peptic ulcer history. Recent myocardial infarction. Haematemesis.	Para oesophageal hiatus hernia. Benign peptic ulcer.
Bittman (1) <sup>11</sup>	1960	44	M	Yes	60 h to operation. 24 h after operation.	5 × 5 cm	Long peptic ulcer history. Haematemesis resulting in laparotomy. Subtotal gastric resection. Patient died 24 h after surgery.	Adhesion between stomach, diaphragm, and heart. Benign peptic ulcer at resection line.
Bittman (2) <sup>11</sup>	1960	62	F	Yes	24 h	3.5 × 3.5 cm	18 year history of peptic ulcer. Oesophagogastrectomy for gastric ulcer 12 months before death. Heartburn.	Postoperation fibrous adhesion between stomach, diaphragm, and heart. Benign peptic ulcer on resection line.
Ritz <sup>13</sup>	1966	69	M	Yes	12 days	2 × 2 cm	Perforated gastric ulcer 9 years before death. Haematemesis resulting in gastrectomy. Death 24 h after operation.	Postoperation fibrous adhesion between stomach, diaphragm, and pericardium. Benign peptic ulcer in fundus of stomach.
Kissel <sup>24</sup>	1964	59	M	Yes	20 h	2 × 2 cm	No history. Aspirin abuse. Haematemesis.	Fibrous adhesions between stomach, diaphragm, and pericardium. Small left lobe of liver. Benign peptic ulcer on lesser curve.

Author	Year	Age	Sex	Fistula	Period between 1st bleed and death	Size of ulcer	Associated condition	Pathological features
Meinecke <sup>17</sup>	1967	39	M	Yes	10 h	3 × 3 cm	Long history of peptic ulcer disease. Haematemesis. Melaena.	Adhesions between stomach, diaphragm, and heart. Benign peptic ulcer on lesser curve.
Kiss <sup>25</sup>	1967	89	F	Yes	10 h	3 × 1.5 cm	Long history of hiatus hernia. Haematemesis.	Hiatus hernia with peptic ulcer. Fibrinous pericarditis.
Grosse <sup>9</sup>	1970	69	M	Yes	20 min	3 × 2 cm	Cahexia. Myocardial infarction. Haematemesis.	Adhesions between stomach, diaphragm, and pericardium. Chronic peptic ulcer on anterior surfaces of stomach.
Maroske <sup>16</sup>	1972	59	M	Yes	20 min	10 × 8 cm	Left phrenic nerve paralysis and left diaphragm elevation after war injury. Long history of left sided abdominal pain. Haematemesis.	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.
Lam <sup>26</sup>	1974	67	M	Yes and to left atrium.	2 days	3 × 2 cm	Gastric carcinoma resected. Haematemesis.	No evidence of local recurrence of gastric ulcer. Fistula with left ventricle and left atrium suggested it may have been due to ischaemia.
Matthews <sup>14</sup>	1974	71	F	No	6 days	6 × 3 cm	Previous history of peptic ulcer disease. Hiatus hernia. Myocardial ischaemia. Atrial ectopics. Intermittent left bundle branch block.	Left circumflex coronary artery in bases of ulcer and source of bleeding fibrous adhesions.
Kennedy <sup>10</sup>	1983	81	F	Yes	8 h	3 × 1 cm	Hiatus hernia.	Pericardial cavity obliterated by adhesions. Fibrosis and endarteritis Obliterans in the underlying myocardium.

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,<sup>9</sup> who thought that hypotension together with the tamponading effect of a stomach turged with blood would be enough to stop the haemorrhage, at least for a limited period. Despite this, surgery was attempted in only four cases,<sup>11-14</sup> and in no case was the diagnosis made clinically. Myocardial infarction was diagnosed before death in three cases.<sup>9, 14, 15</sup> Historical cases, however, may have lacked modern techniques for diagnosing myocardial infarction. One other case had pathological evidence of myocardial infarction.<sup>16</sup> Fourteen cases had a previous history suggestive of peptic ulcer disease.<sup>1, 8, 11-13, 15-20</sup> 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.<sup>2</sup> Endarteritis obliterans is a commonly described feature. Only Chiari and Tylecote<sup>19</sup> described fatty change in the myocardium surrounding the ulcer. Twenty cases were associated with fibrous adhesions between the stomach and diaphragm.<sup>1, 3, 5-9, 11-13, 16-24</sup> Only five reports described cases where this was not a feature. In each case the ulcer had developed within a paraoesophageal hiatus hernia. Kissel<sup>24</sup> 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<sup>9</sup> and Maroske,<sup>16</sup> 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<sup>27</sup> 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<sup>14, 18-20</sup> as in case 2. Three of these were incidental findings at necropsy. The other case reported by Matthews<sup>14</sup> was similar to our second case in that the source of the blood in the haematemesis was a coronary artery. Lam<sup>24</sup> 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 fibreoptic 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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