SPONTANEOUS RUPTURE OF OESOPHAGUS

REPORT OF THREE SUCCESSFUL CASES TREATED SURGICALLY

BY

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Spontaneous rupture of the oesophagus, until 1947 a fatal disease, can now be effectively treated by surgical means. The criterion for success requires that such a lesion be known about and kept in mind, that it may be recognized early and treated promptly. The general practitioner is as able to make the diagnosis as the specialist; and, like perforated duodenal ulcer, early recognition and treatment gives satisfying results (Borrie, 1953).

This paper reports three cases seen within an eight-months period at one Thoracic Surgical Unit dealing with a regional area of 250,000 people. In none of the three cases was the diagnosis at first suspected; in all, however, it was later established and operation was followed by complete recovery.

Case 1

A married woman aged 65 was an emergency admission to the medical wards of Dunedin Hospital on December 15, 1952, under Professor J. E. Caughey, with a diagnosis of haematemesis. Earlier that evening, while hungrily eating a fish meal, she tried to make herself sick because she thought she had swallowed a fishbone. She vomited copiously 1 pint (570 ml.) of bright blood-stained gastric contents, and was immediately seized with severe chest pain.

On examination she complained of pain behind the lower third of the sternum. It was severe and gripping, and came in spasms. There were no abnormal respiratory, cardiovascular, or abdominal physical signs, and no cervical emphysema. Her blood pressure was 180/110 mm. Hg, and her haemoglobin 90% (Sahli).

A diagnosis of haematemesis secondary to a foreign body in the lower third of the oesophagus was made, and she was referred to the ear, nose, and throat department. It was decided to keep her under close observation, with nothing by mouth, a rectal drip, penicillin, and "omnopon" to relieve her pain.

On the morning of December 16 she had ceased vomiting, looked better, but still had retrosternal pain. Later that day she could not swallow her sputum, and regurgitated blood-stained mucus. Oesophagoscopy was performed that evening by Mr. Alan Wardale, surgeon in charge of the ear, nose, and throat department. He found a considerable amount of dark blood lying in the oesophagus. The upper two-thirds of the oesophageal mucosa in particular was haemorrhagic. There was no foreign body. The possibility of rupture of the oesophagus at the junction of the middle and lower thirds was entertained, and on December 17 she was referred to the thoracic surgical department.

Clinically there was still no pleural effusion or cervical surgical emphysema. Radiopaque oil was given, and fluoroscopy showed a vertical rent on the left side of the lower third of the oesophagus, lying 1 in. (2.5 cm.) above the diaphragm. The diagnosis of spontaneous rupture of the oesophagus was established and left thoracotomy advised.

Operation.—Pre-operative oesophagoscopy confirmed haemorrhagic oedema of the entire oesophageal mucous membrane together with a vertical ragged area in the lower third of the oesophagus. Under general anaesthesia, the left

chest was opened through the bed of the resected left eighth rib. There was an extensive extravasation of blood-stained fluid under the mediastinal pleura and extending from the level of the oesophageal hiatus in the diaphragm up to the left inferior pulmonary vein, and midway between the necks and angles of the lower left ribs. Through a 3-in. (7.5-cm.) incision in the mediastinal pleura, the oesophagus was mobilized and retracted with a Jaques rubber catheter sling. On its left border was the vertical tear, approximately $\frac{1}{4}$ in. (1.9 cm.) in length. The points of dissecting scissors were easily introduced through the tear into the oesophageal lumen. The tear was closed with four interrupted twenty-day No. 1 chromic catgut sutures passed through all layers. The mediastinal pleura was not sutured. The left chest was closed over water-seal drainage, using interrupted catgut for the muscle layers and fine nylon for the skin.

The patient was allowed sips of water by mouth and over a period of three days gradually increased her intake by mouth to a fluid diet. She took solid food a week after operation. The intravenous fluid was discontinued on the third post-operative day, and the intercostal drainage was removed on the fifth post-operative day.

She was discharged, convalescent, three weeks after admission, swallowing normally. Subsequent fluoroscopy and oesophagoscopy showed no abnormality of the foregut. When seen 18 months after operation she remained well.

Comment.—This patient illustrates the typical onset following violent vomiting, and shows how a small tear need not be associated with rupture into the pleural cavities or with detectable emphysema. The constant retrosternal pain following vomiting alone suggests the diagnosis, which is further suspected after unusual findings at oesophagoscopy, and convincingly established by careful fluoroscopy with radiopaque oil. Untreated, she would undoubtedly have developed a serious mediastinal abscess.

Case 2

A bachelor aged 53 was brought to the casualty department of the Dunedin Hospital at 9 p.m. on June 29, 1953. Earlier that day he was well, and between 5 and 6 p.m. drank six pints (3.4 litres) of beer in a hotel. When the bar officially closed at 6 p.m. he staggered into the street and vomited copiously into the gutter. He started to walk away, but had just gone five paces when he suffered an agonizing retrosternal pain which radiated to the left shoulder and leg. He drove his car home with great difficulty, the pain becoming more intense and breathlessness more acute. There was no further vomiting.

When examined on admission he still had excruciating pain, was restless, and quite unable to find relief either by sitting up or lying down. He was convinced he would die. His pulse was 120 and blood pressure 170/110 mm. Hg. The casualty officer found the apex beat normal. What was believed to be a pericardial friction rub, crackling in quality, was detected. There were left basal crepitations. The abdomen was board-like, but with no definite tenderness. Although acute pancreatitis or rupture of the gall-bladder was entertained, these diagnoses were not supported by the general surgeon who was called to see him.

An E.C.G. showed T-wave inversion, with tall R waves in V_5 strongly suggesting left ventricular strain. With the diagnosis still in doubt, a chest radiograph was ordered. At this stage surgical emphysema above the suprasternal notch was noted, the correct diagnosis established, and the patient admitted to the thoracic surgical department. The chest radiograph showed left hydro-pneumothorax with air around the aortic arch and soft tissues of each side of the neck.

Operation.—At midnight, under general anaesthesia, the left chest was opened through the left ninth rib bed. The pleural cavity contained 1½ pints (850 ml.) of brown alkaline fluid. The mediastinum was emphysematous and the lacerated pleura had lifted from off the aorta and oesophagus. A catheter sling was passed around the oesophagus,

which was then lifted up for inspection. On its left side was a vertical rent 1 in. (2.5 cm.) long, and starting 1 in. (2.5 cm.) above the oesophageal hiatus in the diaphragm. Through this rent protruded oesophageal mucosa. After inserting mucosal stay sutures, the tear was closed with a series of interrupted chromic catgut sutures passed through all oesophageal layers. The chest wall was closed as in Case 1.

On July 1, because of sputum retention, aspiration bronchoscopy was performed and gave relief. The intercostal drainage was removed on the third post-operative day, and the skin sutures on the seventh day. The patient was discharged fit three and a half weeks after admission. The post-operative feeding regime was the same as in Case 1.

Subsequent barium study of the foregut revealed no pleural or oesophageal abnormality, but suggested a deformity of the duodenal cap without any definite evidence of active ulcer. Eleven months after operation the patient continued well and was working.

Comment.—This case illustrates the classical onset of symptoms first graphically described by Boerhaave in 1723 (Barrett, 1946). It illustrates their confusing severity, and how tell-tale cervical emphysema gave the key to diagnosis. Physical findings in the chest had been difficult to elicit and interpret, and an E.C.G. record was definitely misleading. It further shows how prompt surgical intervention materially altered the course of a catastrophe, with minimal convalescence and a most gratifying result.

Case 3

A farmer aged 52 was admitted as an emergency case to a county town hospital on July 20, 1953, suffering from severe epigastric and right chest pain. For years he had had an active duodenal ulcer; for four days before admission there had been an acute exacerbation of his ulcer symptoms, and he took only milk diet until 7 p.m. on July 20, when he ate a pear. Half an hour later he vomited twice and suddenly developed severe pain in the right chest, shoulder, and upper abdomen. The vomit was "coffee ground" material.

On admission he was distressed, with grunting respirations and slight cyanosis; pulse 120 and blood pressure 140/90 mm. Hg. The tongue was dry and furred. There was generalized abdominal rigidity, and absent bowel sounds. Examination of the chest at that time revealed no obvious abnormalities. A diagnosis of perforated duodenal ulcer was logically made, and the patient was prepared for operation—a Ryle tube being passed into the stomach and aspirated, and an intravenous drip being started.

First Operation.—At 11.30 p.m. that same night, under general anaesthesia with endotracheal intubation, laparotomy through an upper right paramedian incision showed the presence of a chronic duodenal ulcer in the first part of the duodenum and firmly adherent to the pancreas. There was no perforation, and the pylorus was patent. The large dilated stomach was further aspirated, 42 oz. (1,200 ml.) of brown fluid being removed. The abdomen was closed and the patient returned to the ward.

On the morning of July 21 a chest radiograph revealed a right-sided hydropneumothorax without tension phenomena. Spontaneous rupture of the oesophagus was established. Throughout the morning his condition gradually deteriorated, nor was it improved by aspirating a few millilitres of that same brownish fluid from the right pleural cavity. His pulse was 140 and blood pressure 55 mm. Hg. Plasma infusion, oxygen, and penicillin were given with no great improvement.

After telephone consultation with the thoracic surgeon in Dunedin, a Malécot catheter was inserted into the right pleural cavity and connected to water-seal drainage. His blood pressure steadily rose to 90 mm. Hg. A pint (570 ml.) blood transfusion was started and the patient was transferred 70 miles by ambulance.

At 11 p.m. on July 21 he was admitted to Dunedin Hospital, having withstood the journey well. Both the blood drip and the intercostal drainage were working satisfactorily. His temperature was 100.5° F. (38° C.), pulse 114, respirations 48, and blood pressure 110 mm. Hg systolic. A further chest radiograph showed little alteration, and the right lung was still partially collapsed.

Second Operation.—An hour later general anaesthesia was induced and the right chest opened through the ninth intercostal space. The pleural cavity contained $\frac{1}{2}$ pint (280 ml.) of brownish fluid. With positive pressure, the right lung was made to re-expand. Below the azygos arch was an irregular tear in the mediastinal pleura, with surrounding oedema. The oesophagus was isolated from the mediastinum and a catheter sling passed round it. Inspection revealed a vertical rent $1\frac{1}{2}$ in. (3.8 cm.) long, starting 2 in. (5 cm.) above the diaphragm, and extending upwards to the azygos arch. This was closed with a series of No. 4 silk sutures, placed $\frac{1}{4}$ in. (3 mm.) apart and passed, as before, through all oesophageal layers. The chest was closed over water-seal drainage.

Post-operatively, bronchoscopy was performed and he was returned to the ward, where graduated continuous suction was applied to the water-seal drain. Fluids by mouth were restricted for four days, during which time intravenous therapy was continued. He was given penicillin and streptomycin.

A chest radiograph on July 24 confirmed that the right lung had re-expanded and was aerating satisfactorily. On July 27, however, there was evidence of a loculated basal pleural effusion, and on July 31 this was drained. The presence of a fistula and also staining after swallowing methylene blue solution confirmed that there was a leak from the oesophageal suture line.

On August 1 a second anterior pocket of pus in the right pleural cavity was cleared by intercostal drainage, and a Stamm gastrostomy performed. Progress thereafter was slow. The partially collapsed lung required a third apical intercostal drain and continuous suction for two weeks before it had re-expanded.

By September 30, almost two months after admission, the oesophageal fistula had healed and normal swallowing was again allowed.

On October 8 the gastrostomy tube was removed, by November 5 the anterior pleural sinus was healed, and by November 18, as the posterior sinus was negligible, he was discharged. He was last seen on March 10, 1954, when his sinuses were well healed and his swallowing was satisfactory.

Comment.—This is an interesting case of cause and effect. It demonstrates a further and unusual complication of duodenal ulcer—a complication which must always be kept in mind when sudden and severe abdominal symptoms follow vomiting. One can readily appreciate how the rupturing of the oesophagus into the right pleural cavity, with associated epigastric pain and rigidity, led to the possible diagnosis of perforated duodenal ulcer. Undoubtedly a pre-operative chest radiograph would have revealed the true diagnosis. This case, and two others not yet in the literature but known by me to have been explored as acute pancreatitis, induce me to plead for a pre-operative chest radiograph in all difficult acute abdominal cases. Nowadays, in such circumstances, a preliminary abdominal radiograph is common practice. The same film can well be adjusted to include the diaphragm and the lower chest, and thus avoid needless laparotomy. The case further illustrates how a patient in civilian practice, with pneumothorax and empyema made safe and temporarily relieved by intercostal drainage and by blood transfusion, can travel 70 miles by ambulance and be improved at the end of his journey.

Although Susman (1953) wisely points out that some cases of spontaneous rupture can recover without suture of the rent, and that Barrett's (1948) case of suture in fact leaked after surgical closure to produce a further oesophageal fistula and empyema, yet such conservative measures must

include adequate treating of the accompanying empyema. In a late case, with fully established empyema, to attempt to free the oesophagus and perform suture would be undoubted folly. In an early case, however, possibly up to 48 hours after perforation, the right attitude should be to explore and if possible suture the rent.

In this third case—with a long rent—there was a late leak. By performing a gastrostomy as an oesophageal bypass, and thus restricting swallowing purely to saliva, local pleural drainage together with gastrostomy ultimately gave sound closure.

Summary and Conclusions

Spontaneous perforation of the oesophagus is an acute emergency which must always be kept in mind and which may simulate many lesions. It can masquerade as coronary thrombosis, or acute abdominal catastrophes, especially perforated gastric ulcer, acute pancreatitis, and acute cholecystitis. Frequently the patient is too distressed to give a rational history.

As the oesophagus may perforate into the mediastinum alone, into the right or left pleural cavities, or into both, there is a diversity of signs and symptoms. Convincing chest clinical signs are often hard to elicit. Not all cases have cervical emphysema, but, if present, it is diagnostic. Chest radiographs, including, if necessary, the swallowing of radiopaque oil, confirm the diagnosis.

Pre-operative management embodies prompt relief of any tension pneumothorax, restriction of all food by mouth, intravenous fluids, passage of a Ryle tube to the stomach, and antibiotics.

Thoracotomy is then required, and wherever possible the rent should be sutured. In early cases, healing is sound and convalescence greatly shortened.

A plea is made to include a chest radiograph when faced with a difficult diagnosis of the acute abdomen.

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An observing station for measuring the smoke and sulphur dioxide pollution in the London atmosphere has been established in the Science Museum by co-operation with the Fuel Research Station of the Department of Scientific and Industrial Research. The station is one of a number that are being established to permit a more detailed examination of atmospheric pollution in London and elsewhere. The sampling equipment is displayed as a working exhibit in the Meteorology gallery. The smoke particles are captured on filter paper through which air is drawn from just above museum roof level by means of a small electric pump. As a result a grey stain appears on the filter paper, the intensity of which is a measure of the degree of smoke pollution. The same air is bubbled through a weak solution of hydrogen peroxide, where the atmospheric sulphur dioxide is dissolved to form an acid solution, the strength of which is measured by standard chemical methods. Observations of smoke and sulphur dioxide pollution are made daily by a member of the museums staff and reported to the Fuel Research Station, who are in charge of the investigation.

SPONTANEOUS RUPTURE OF OESOPHAGUS

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The following case of spontaneous rupture of the oesophagus is placed on record, even though treatment was unsuccessful, as this condition is still thought to be rather rare. The records of this hospital for the past six years, covering 28,725 admissions, show only one comparable case—that of an eclamptic patient who died thirty-two hours after admission—in which two perforations involving the lower end of the oesophagus were found; unfortunately the data about this case are too scanty for an accurate diagnosis to be made.

Increasing awareness of the condition seems to be bringing more cases to light. Mallam, Whitelocke, and Robb-Smith (1940) stated that the total number of published cases was 40. Barrett (1946), in a comprehensive review of the subject, put the figure at about 50 cases. Mauro (1952) discussed 103 cases in the literature. Barrett (1947) was the first to describe recovery following correct diagnosis and deliberate operation to repair the rupture. Frink (1947) published details of a successfully treated case of 1941, and Samson (1951) recorded two such cases mentioned by Graham in 1944. Nanson and Milnes Walker (1953) added the twenty-third recovery to the literature.

It would be wrong to publish only the successes in the treatment of this condition if a correct record of mortality rate is the aim. The mortality rate may be even higher and the disease commoner than the records would suggest, as up to 1949 only 16 cases had been diagnosed in life and the remainder at necropsy (Russell, 1953), and it may be possible to miss a ruptured oesophagus at necropsy unless one is looking especially for the condition (Barrett, 1946). Many cases of death in a period of hours to a few days after the onset of retrosternal pain, collapse, and perhaps vomiting must have been certified as being due to coronary thrombosis without necropsy confirmation, and these might well include a number of cases of spontaneous rupture of the oesophagus.

Summary of Case Notes

A 73-year-old widow, about to begin her tea, complained of severe upper abdominal pain which rapidly spread to both sides of the lower chest and back. After 30 minutes she vomited a little blood-stained fluid and then collapsed with pain. She was admitted to the medical wards, and after several hours was transferred to the surgical side. A long history of dyspepsia which she relieved by making herself vomit was obtained.

Examination.—Grossly obese, very shocked, and in great pain. Respirations 24, pulse 80, temperature taken rectally 99.8° F. (37.7° C.); blood pressure 150/90 mm. Hg. Tongue stained with altered blood. Marked surgical emphysema of front of neck. Equal aeration of both lungs and no detectable effusion or pneumothorax. Abdomen very fat, with large irreducible umbilical hernia; tender in right epigastrium but no guarding. A Ryle tube was passed and 90 ml. of thick bloody material was aspirated.

A provisional diagnosis of spontaneous rupture of the oesophagus was made and the patient was transferred to the x-ray department. Films of the chest were taken and then three mouthfuls of barium mixture were given to her to swallow before repeating the pictures. The following