Case Reports

RUPTURE OF THE ŒSOPHAGUS SIMULATING MYOCARDIAL INFARCTION

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RUPTURE of the Œsophagus has been known for approximately the past 240 years and has been more of historic interest than practical importance. Up to 1947, of the cases reported probably not more than 5 are known to have recovered (McPhedran, 1961), and it is only in recent years that surgical treatment has been carried out successfully, the first being in 1947 (Barrett, 1946, 1947). All writers agree that rupture of the Œsophagus, generally spoken of as spontaneous rupture, is an acute surgical emergency with a high mortality. It is probable that the condition is commoner than is generally supposed, and that the diagnosis is frequently missed, because it may not have been seriously considered, or have been forgotten as an acute surgical emergency. Even among those patients coming to necropsy, the underlying cause of death may have been missed owing to the presence of an empyema or other secondary changes in the chest.

Case Report

On 29th May, 1962, a farmer, aged 51, while at work experienced severe pain in the back and right hip, and then in the chest, and found that the pain was aggravated by bending forward. He had previously complained of pain in his chest on several occasions, and for a long time had been known to suffer from dyspepsia, and from varicose veins though he had never had phlebitis. He went to see his doctor, was given some antispasmodic medicine and went back to work. The pain, however, became so severe that he was obliged to return home and go to bed. On the 30th May, the pain in the chest was still present and a crushing sensation was felt beneath the sternum. At 5 p.m. on the same day, the doctor made a diagnosis of pulmonary embolus and administered morphine, which gave some relief, although the pain never completely disappeared. About this time, a cupful of bright red, frothy blood was coughed up. Following this hæmoptysis the pain became very severe, and the patient was sent into hospital.

On arrival, he was restless, cyanosed, and dyspnoeic, and complained of severe substernal pain, and preferred to be propped up in bed and leaning forward. The temperature was 101°F., pulse 100, regular, B.P. 110/80 mm. Hg. The patient was obese, respiration was rapid and shallow, and deep breathing was impossible on account of severe pain. The heart sounds were clear and triple rhythm was present. Crepitations were noticed at the lung bases. No

abnormality was detected in the abdomen. At 11.30 p.m. the pain, which had been constant, became more severe and the dyspnoea increased. Sweating also occurred, and the B.P. fell to 110/70. About this time, a quantity of offensive, greenish coloured sputum was coughed up, and this was followed by an irritating cough.

On the 30th May, an X-ray of the chest showed congestive changes in the lung fields; the appearances of a failing hypertensive heart, and elevation of the left lobe of the diaphragm, and a small effusion in the left costo-phrenic angle. An ECG showed a semi-vertical heart with clockwise rotation, but no evidence of myocardial infarction. The temperature at this time was normal, but cough was still troublesome. RBC normal, WBC 9,000 cu. mm., ESR 10 mm./hr. (Wintrobe), S.G.O.T. 36 units/ml.

On the 1st June, the patient had improved, and this was maintained until the 2nd June, when pleural friction was heard on the right side of the chest. On the 4th June, there were signs of diffuse bronchitis throughout the chest, but the pulse was stronger and the patient did not have any pain. On the 6th June, after a recurrence of pain the patient suddenly died.

Autopsy

A massive left hæmothorax was found with collapse of the left lung and the presence of fluid and blood and a quantity of laminated clot in relation to a postero-lateral tear 1½" long near the lower end of the Œsophagus. Granular food material was also found in a small amount in the pleural cavity. The heart, which was fatty and dilated and showed evidence of LVH was surrounded by an excess of clear fluid. The coronary arteries, though atherosclerotic, were patent. The abdominal viscera were markedly congested, and the liver was friable and fatty. The kidneys, apart from some engorgement, were normal. No evidence of a hiatus hernia was found. The stomach and duodenum appeared to be healthy.

Discussion

The patient's doctor had made a diagnosis of pulmonary embolus, and, on admission, the signs and symptoms suggested myocardial infarction. Anticoagulant therapy was started, but was later stopped and antibiotics substituted since respiratory symptoms became more pronounced and indicated pulmonary infection. There had been no previous history of hæmatemesis, and the patient was sure that he had coughed up rather than vomited up blood, although it was known that

there had been a previous history of long-standing dyspepsia.

Spontaneous rupture of the œsophagus is a rare condition and a surgical emergency with a high mortality. The word "spontaneous" has been criticised as a misnomer on the grounds that it suggests an underlying pathological condition in the œsophagus itself, whereas the rupture is generally caused by powerful physical forces giving rise to greatly increased pressure inside the œsophagus, such as may occur in the act of vomiting, or after excessive eating or drinking.

It is said that a history of previous dyspepsia is obtained in between 36% and 44% of cases where rupture has occurred. Marston and Valk (1959) suggest that peptic ulceration may be a factor in the ætiology, though in such cases perforation does not appear to take place at any particular site.

Other activities which have given rise to this complication have been weight-lifting, childbirth, defæcation, a long-continued asthmatic attack, and an epileptic convulsion. Cases in which spontaneous rupture has been preceded by vomiting following excessive indulgence in food or alcohol, or both, have been designated post-emetic, and rupture from mechanical causes nearly always results in a longitudinal tear in the postero-lateral aspect of the left side of the œsophagus, where the anatomical structure predisposes to this kind of injury. It was in this site that the lesion was found in the present case.

The clinical signs and symptoms of spontaneous rupture, though they may not all be found together, consist of:

1. Severe pain in the chest, followed by a small hæmatemesis.
2. Pain in the back. In the present case, pain was felt in both these sites.
3. Subcutaneous emphysema.
4. The development of pleural friction.

The differential diagnosis is a wide one and the following conditions have to be excluded:

Where the signs and symptoms are predominantly thoracic, spontaneous pneumothorax, coronary thrombosis, dissecting aneurysm, ruptured aneurysm, pulmonary embolus, have to be considered. Whereas, where abdominal signs preponderate, rupture of a viscus, splenic infarction, intestinal obstruction, pancreatitis, and mesenteric thrombosis have to be excluded.

Hodgkin's disease has given rise to rupture of the œsophagus, and 17 cases of this have been reported.

McPhedran described a series of spontaneous rupture of the œsophagus in 9 cases in Toronto General Hospital, and divided them into two groups—a cerebral group, where there had been no signs or symptoms except hyperthermia and in no case was the correct diagnosis made before death; and a mechanical group, in which all cases gave a history of violent vomiting at the time when they experienced severe substernal pain. All

patients noticed an intensification of pain by deep breathing, coughing, or further vomiting, and little relief was obtained from drugs.

A number of cases have been reported by Cushing, including patients with cerebral lesions. In McPhedron's series of cases, none of these had subcutaneous emphysema, but all were in a considerable degree of surgical shock when they reached hospital. All were restless, some slightly cyanosed and had grunting respirations and were most comfortable in the upright or sitting positions. All patients had signs in the chest where the œsophagus had ruptured and the site was found to be almost always on the left side of the diaphragm and the lesion about 2-5 cms. in length. All these signs were found in the present case.

Spontaneous rupture of the œsophagus occurs more particularly in middle age, and is five times commoner in men than in women, and, according to Mackler, low thoracic pain and emphysema in the neck are diagnostic features. Bunch has stated that every case of suspected perforated ulcer should have an X-ray of the chest taken in the erect position, since if there is rupture of the œsophagus, there will be an abnormality in the chest film in 90% of cases, and no free air will be seen beneath the diaphragm. Anderson, Chamberlain and Bryerley state that free air beneath the diaphragm has never been reported in this condition. A characteristic V sign has been reported by Nacherio as occurring in spontaneous rupture of the œsophagus. In our own case, congestive changes were found in the chest and there was elevation of the diaphragm on the left side and a small effusion. Chest X-ray confirmation of spontaneous rupture of the œsophagus has been obtained, by finding gastric juice in the pleura on aspiration of the chest, and the administration of Methylene Blue and its recovery in the pleural fluid has also been used for diagnostic purposes in suspected cases.

The possibility of spontaneous rupture of the œsophagus has to be considered where substernal pain occurs in association with vomiting and with signs and symptoms suggesting both respiratory and abdominal disease, and if the diagnosis can be correctly made, the outlook for the patient depends upon the promptness with which surgical treatment is instituted, since without this, patients inevitably die. It is stated that 25% of cases of spontaneous rupture of the œsophagus die within 12 hours of rupture, 60% within 24 hours, and 10% within 48 hours, and that none live longer than a week. The overall mortality is shown by a 1952 summary of 134 cases, among which there were only 27 survivals, and even at the present day, after operation under the most favourable conditions, the mortality is still about 30%.

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A CASE OF NON-FAMILIAL DEGENERATIVE CHOREA

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DEGENERATIVE changes in the central grey matter of the brain as well as in the cortex give a wide range of the so-called extra-pyramidal syndromes which affect mainly the posture and sometimes the motion of the afflicted persons. Different forms of involuntary movements, namely, static tremor, chorea, choric-athetosis, dystonic and intentional tremors are described. Groups of these are often classified into clear-cut clinical syndromes and further attempts at their classification into clinico-pathological syndromes have often been attempted. Examples are paralysis agitans, Huntington's chorea and olive-ponto-cerebellar degeneration. Yet in analysing the reports of such syndromes, it is noted that the pathological pictures are not often identical as regards the site or nature in all members of one group. If we consider the syndrome of Huntington's chorea, it is noted that the different reported cases show protean pathological changes and a variety of sites for the degenerative changes. They are reported in the lentiform nucleus or only the putamen (Lannois and Paviot, 1898, Alzheimer, 1914, Bielschowsky, 1922, Dunlop, 1927). In some cases lesions appear in the putamen and caudate nucleus (Marie and Lhermitte, 1914; Kleist, 1912; Vogt, 1920; Hunt, 1916; V. Santha, 1931; Jacob, 1923). The cortex is reported to be affected in several cases (Pfeiffer, 1913; V. Santha, 1931; Lind, 1927).

The changes usually occur in the 3rd, 5th and 6th cortical layers (Pfeiffer, 1913; Santha, 1931). In quite a number of cases the meninges are also affected, showing chronic inflammation and/or thickening of the arachnoid (Grimby and Wilson, 1926; Frederick Back, 1926; Marie and Lhermitte, 1914). Clinically these cases are typified as presenting gradual and progressive choreic movements starting usually in middle age. The movements are coarse and involve the face and tongue. The disease terminates with progressive mental deterioration. It is often stressed that the disease is hereditary and transmitted as a Mendelian dominant.

It is therefore of interest to report this case of degenerative chorea which shows atypical chronological and pathological pictures.

Case Report

A. M., male of 40 years admitted to hospital on 21.11.61. The condition started with choreic movements in right upper limb followed one month later by movements in the left lower limb which then became generalised. It was of three years duration and of progressive course. No family history of similar disease could be obtained from the patient, and mental changes were absent. The patient related the onset of the disease to an attack of fever of undetermined nature. On examination, there were generalised choreiform movements, tremors of the tongue and slight generalised hyper-reflexia.