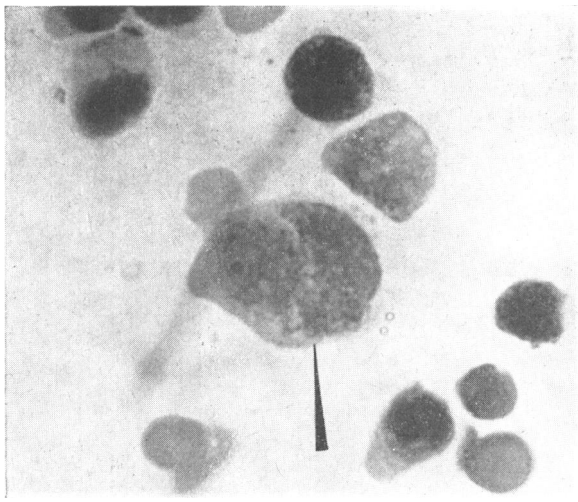

FIG. 1.—Case 4. Typical Halberstaedter - Prowazek inclusion. (× 530.)

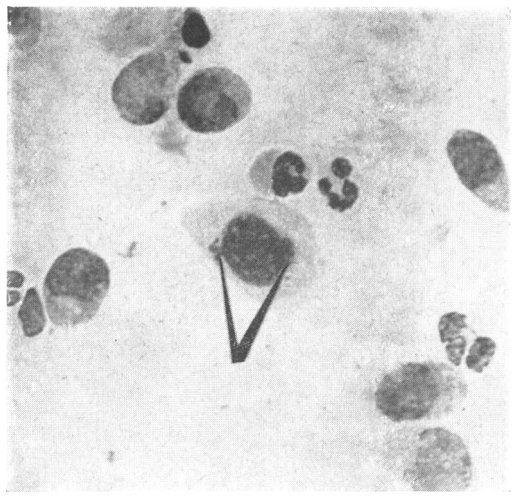

FIG. 3.—The marked cell contains two inclusions, probably early initial bodies. This together with a typical cytology (cf. Fig. 2) is regarded as "very suggestive" of TRIC agent infection. (×340.)

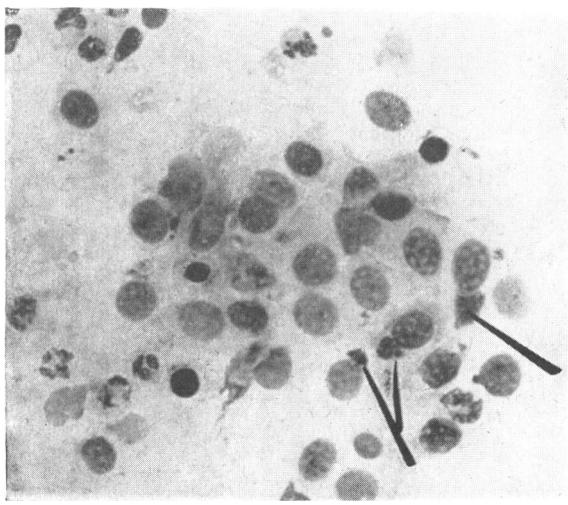

FIG. 2.—Case 1. Cytology typical of TRIC agent infection with polymorphs, lymphocytes, monocytes, and degenerate epithelial cells. Three Halberstaedter-Prowazek inclusions indicated. (×205.)

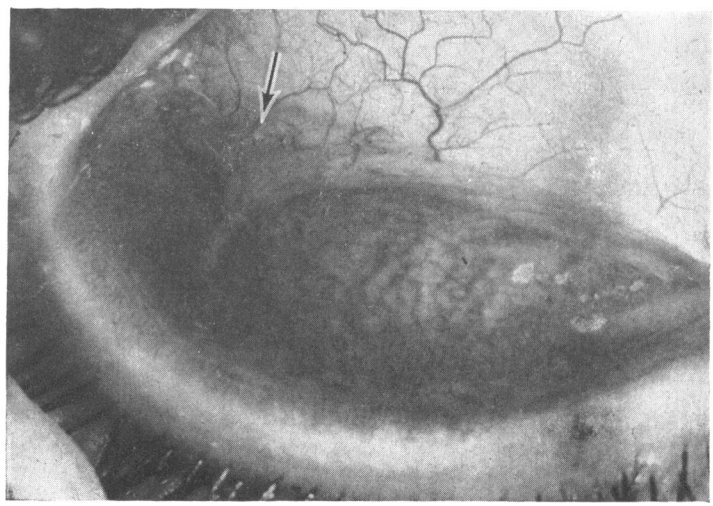

FIG. 4.—Case 5. A "sheet" scar of the conjunctiva of the lower fornix, forming a fold from bulbar to fornix conjunctiva.

I. N. MARKS AND A. D. KEET: INTRAMURAL RUPTURE OF THE OESOPHAGUS

FIG. 1. — Radiograph showing collection of barium outside lumen of the oesophagus just above level of diaphragm.

FIG. 2. — Radiograph showing band-like longitudinal intraluminal filling defects in oesophagus. Note extraluminal collection of barium just above level of diaphragm.

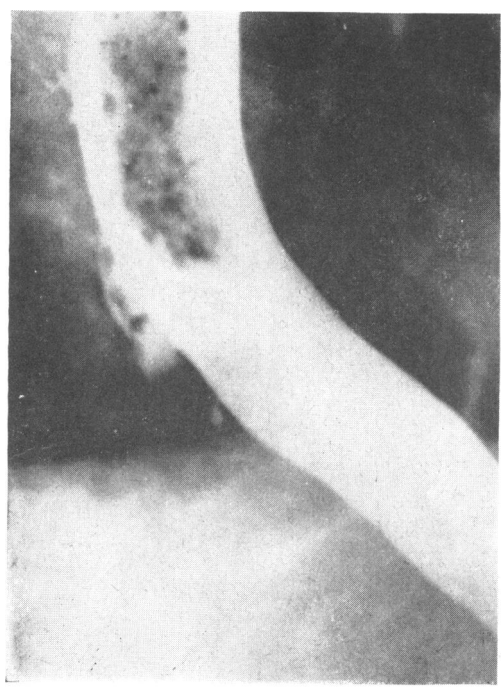

FIG. 1

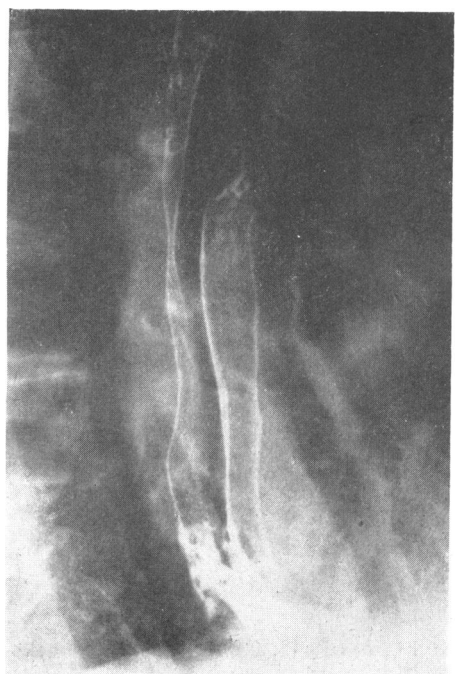

FIG. 2

Medical Memoranda

Intramural Rupture of the Oesophagus

[WITH SPECIAL PLATE FACING PAGE 525]

Brit. med. J., 1968, 3, 536–537

Rupture of the oesophagus is a rare but well-documented emergency which may result from violent vomiting or, less commonly, from other causes of an abrupt increase in intra-abdominal or intrathoracic pressure such as epilepsy, childbirth, defaecation, weight-lifting, or non-penetrating blows on the chest (Moore and Murphy, 1948 ; Mackler, 1952 ; Anderson, 1952 ; Schinz *et al.*, 1954 ; Chamberlain and Byerly, 1957 ; Truelove and Reynell, 1963 ; *Lancet*, 1966 ; Thompson *et al.*, 1967). The rupture usually occurs as a longitudinal slit in the left posterolateral wall of the lower oesophagus within 5–6 cm. of the cardia, and it is generally agreed that this may occur in the absence of underlying disease of the oesophagus. The clinical triad of acute chest pain and shock, surgical emphysema in the neck, and signs at the left base is virtually diagnostic of this potentially lethal condition (Kinsella *et al.*, 1948 ; Mackler, 1952 ; Anderson, 1952 ; Chamberlain and Byerly, 1957 ; Flavell, 1963). Early surgery offers the only real hope of survival.

The purpose of this paper is to draw attention to a most unusual form of rupture involving only the mucosal layer of the oesophagus. The patient presented with acute chest pain and shock which developed during the course of a meal, and the diagnosis of incomplete rupture of the oesophagus was made on radiological examination. The condition settled on conservative measures.

CASE REPORT

A previously healthy 63-year-old woman developed acute lower retrosternal discomfort and difficulty in swallowing during the sweet course of a rather hurriedly eaten meal. Symptoms became increasingly severe over the next 20 minutes, by which time she complained of an excruciating lower retrosternal pain radiating into the right side of the chest and associated with some difficulty in breathing and on attempting to swallow. There was no vomiting, but a sensation of something sticking in the lower gullet caused her to swallow repeatedly in trying to dislodge it. There was no history of heartburn or oesophageal spasm, and she denied having swallowed a bone during the meal.

On clinical examination she was shocked and in extreme pain. Auscultation of the heart and chest was non-contributory, and there was no tenderness on abdominal palpation. No surgical emphysema was found at this or any subsequent stage of the illness. The pain was uninfluenced by two intravenous injections of 5 ml. of Buscopan compositum (hyoscine butylbromide) and was lessened only after an intramuscular injection of 100 mg. of pethidine. An electrocardiogram showed slight flattening of the T wave in lead AVL, but this was considered to be within normal limits. Electrocardiograms 24 and 72 hours later showed no change, and the serum aspartate aminotransferase at 48 hours was within the normal limits. The serum bilirubin, alkaline phosphatase, and amylase levels were normal 12 and 72 hours after the onset. The E.S.R., however, was raised at 26 mm. in the hour (Westergren). The pain diminished progressively, but there was residual lower retrosternal and thoracic discomfort by the end of the third day ; slight dysphagia was still present.

A barium swallow was carried out on the fourth day. During swallowing in the erect position a small accumulation of barium was seen to appear just outside the lumen of the lower end of the oesophagus, a few centimetres about the diaphragm, on the right posterolateral side (Special Plate, Fig. 1). This accumulation was 2 mm. in depth and 6 mm. wide, remained in close apposition to the barium in the lumen, did not track away, and could be demonstrated repeatedly. The diagnosis of an intramural rupture was made. In addition, two broad, band-like, longitudinal intraluminal filling defects were seen to extend through the middle and lower thirds of the oesophagus, causing a partial narrowing of the lumen (Special Plate, Fig. 2). Normal peristaltic waves were seen in the upper third, but there was complete lack of movement in the lower two-thirds. No incoordinated contractions or spasms occurred. In the Trendelenburg position free and persistent gastro-oesophageal reflux occurred, and a few vaguely defined filling defects were seen in the lumen just above the diaphragm. The remainder of the upper gastrointestinal tract was normal.

The patient complained of increasing lower retrosternal discomfort over the next 48 hours. This was associated with a mild fever (99.2° F. ; 37.3° C.) and an increase in the E.S.R. *X*-ray examination of the chest showed no mediastinal emphysema or other abnormality. She was admitted to hospital and treated with ampicillin and cloxacillin intramuscularly, supplemented by intravenous fluids for a four-day period, during which time only mouth gargles and sips of a dilute aqueous solution of neomycin were allowed. She was given oral fluids for the next few days, mashed foods for another few days, and was finally allowed soft foods just before her discharge on the 14th day after admission. The head of the bed was raised to reduce possible gastric reflux. The temperature settled within a few days of admission, but the E.S.R. increased to 50 mm. in the hour by the 10th day despite continued antibiotic cover. It decreased gradually thereafter, and became normal six weeks after the onset of the illness.

A barium swallow at this stage showed considerable improvement in the appearance of the lower oesophagus. There was no evidence of the previous rupture, and the band-like defects in the middle and lower thirds, though still visible, were much less prominent. Primary peristaltic waves were still absent.

The patient has remained well in the two years that have elapsed, and, in particular, has been free of oesophageal symptoms.

DISCUSSION

The acute onset of severe retrosternal pain with dysphagia during the course of a meal was thought to be due to severe oesophageal spasm, but the persistence of symptoms and the development of a mild pyrexia and a raised sedimentation rate indicated a more serious condition. Coronary thrombosis, acute cholecystitis, and other intra-abdominal emergencies were briefly considered, but *x*-ray examination showed an incomplete rupture of the lower oesophagus situated posterolaterally a few centimetres above the diaphragm. The absence of mediastinal or surgical emphysema and lack of chest signs pointed very strongly against a complete oesophageal rupture, and the patient responded satisfactorily to a conservative regimen. Oesophagoscopy was contraindicated, and surgery was never seriously considered.

Incomplete rupture of the oesophagus is probably not as rare as the paucity of the literature on the subject would suggest. The diagnosis should be suspected because of the staggered onset of (1) acute oesophageal pain during the course of a meal which remains severe and persistent, (2) dysphagia, and (3) a mild pyrexia and a raised sedimentation rate, and confirmed by *x*-ray studies with Lipiodol within a few days of the acute onset. This clinical triad was present in two further patients encountered during the past year, but the diagnosis could not be confirmed by *x*-ray studies carried out more than a week after the onset of symptoms ; one of these patients vomited a small amount of unaltered blood almost immediately after the acute onset. *Since the treatment of incomplete rupture of the oesophagus would appear to be conservative, it is obviously*

important to exercise considerable care in excluding a complete rupture of the oesophagus, a grave condition in which surgery usually offers the only hope of survival. Thus patients with a presumptive diagnosis of incomplete rupture must be repeatedly examined to exclude surgical emphysema and signs to the left side of the chest, and a plain *x*-ray film of the chest should be taken to further exclude these stigmata of complete rupture.

The radiological findings in the present case were sufficiently clear-cut to dispel any doubts regarding the diagnosis of an incomplete and intramural rupture of the oesophagus. The small extraluminal collection remained in close apposition to the intraluminal barium, and the unusual band-like vertical filling defects in the middle and lower thirds suggested a severe reaction process, or, just possibly, haematoma formation in the walls. In this connexion it is of interest that a fatal case of intramural oesophageal dissection starting at a mucosal laceration has recently been described by Thompson *et al.* (1967); *x*-ray studies in this patient showed a "double-barrelled" oesophagus due to detachment of the mucosa and submucosa from the muscular wall. Band-like vertical filling defects of the oesophagus have also been recorded by Brombart (1961, 1967), who considered them to be due to oesophagitis.

Gastro-oesophageal reflux noted in the present case suggests that an underlying oesophagitis may have facilitated the rupture (Mackler, 1952). In any event the precipitating factor in the rupture was the forced attempts at swallowing initiated by spasm or a bolus of food impacted in the lower oesophagus. Though severe vomiting is the most common cause of complete rupture, occasional cases of the latter may occur as a result of forced swallowing of an impacted bolus of food (Truelove and Reynell, 1963; Conte, 1966).

The site of the complete rupture in the present case conforms to that in which clinical and experimental complete rupture of the oesophagus occurs. The fact that the incomplete rupture involved only the mucosal layer may be construed as being at variance with the experimental findings of Mackler (1952) and Lion-Cachet (1963). These workers, using different inflation and bursting techniques, found that the muscle layer of the oesophagus ruptures earlier than does the mucosal layer. Lion-Cachet, however, noted that the musculature of the lower end of the oesophagus and the stomach have the capacity to distend beyond the limits of their respective mucosal linings.

Incomplete rupture of the oesophagus should not be confused with the Mallory-Weiss syndrome, a condition characterized by brisk haematemesis due to vomiting-induced mucosal tears of the cardia of the stomach (Mallory and Weiss, 1929). The long axes of these mucosal tears are in line with the oesophagus, and, rarely, they may extend from the cardia into the lower end of the oesophagus. Clinically the accent in this syndrome is on bleeding and not on pain.

We would like to acknowledge the assistance given by Professor J. H. Louw and Dr. S. Bank in management of the case.

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Right Atrial Myxoma with Right-to-left Interatrial Shunt and Polycythaemia

Brit. med. J., 1968, **3**, 537-538

This paper describes a case of right atrial myxoma in which the correct clinical diagnosis led to successful surgical removal and in which polycythaemia was associated with arterial desaturation due to a right-to-left shunt at atrial level.

CASE REPORT

The patient, a 45-year-old married woman, gave a history of progressive effort dyspnoea for 18 months, "tightness in the chest" on effort for six months, and anorexia and nausea for three months. There was no history of rheumatic fever. There had been one episode of near syncope without neurological sequelae three months before admission.

On examination she was afebrile and the positive physical signs were confined to the cardiovascular system. There was central cyanosis and the jugular venous pressure was raised to the angle of the jaw. The wave form of the venous pulse showed a giant "a" wave which was easily palpable; in fact, some observers mistook it for the carotid pulse. On auscultation a soft systolic murmur and a soft diastolic murmur with presystolic accentuation were heard. These murmurs were maximal at the left sternal edge and their intensity increased on inspiration. There was no clinical evidence of pulmonary hypertension.

Investigations.—Blood: Hb 124% (18 g./100 ml.), R.B.C. 6,200,000/cu. mm., platelets 184,000/cu. mm., W.B.C. 9,000/cu. mm., E.S.R. 1 mm./1 hour (Westergren), total serum proteins 7.1 g./100 ml., albumin 4.1 g., globulin 3.0 g. Electrophoresis showed a slight increase in alpha-2 globulin fraction.

E.C.G.: sinus rhythm; P–R 0.16 sec.; P wave in standard leads I and III was tall and peaked, measuring 3 mm.

Chest *x*-ray examination showed normal heart size, no enlargement of right or left atrium, and lung fields normal.

In view of the short history of effort intolerance and one episode of near syncope, and the clinical features, a diagnosis of subacute tricuspid valve obstruction was made. It was thought that the obstruction was probably due to a tumour, ? right atrial myxoma. Emergency admission into hospital was arranged and cardiac catheterization was performed the next day. There was gross increase in the right atrial pressure, primarly due to an "a" wave measuring 21 mm. Hg. The right atrial systolic pressure was higher than the right ventricular systolic pressure, and the pressure in the right atrium exceeded that in the right ventricle at all times except during peak ventricular systole (Fig. 1). The right ventricular, pulmonary arterial, and indirect left atrial (wedge) pressures were normal. There was systemic arterial desaturation (81%) and the cardiac index was 1.6 l./min./sq. m. Angiocardiography showed a large filling defect in the right atrium (Fig. 2). On cine-angiocardiography this filling defect was seen to prolapse through