

A CASE OF PERFORATED DUODENAL ULCER AND CARDIAC INFARCTION

BY

A. W. BRANWOOD

From the Department of Clinical Medicine, Royal Infirmary, Edinburgh

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The following case was thought to be of sufficient interest to be worth reporting because cardiac infarction seemed to follow within an hour of a perforated duodenal ulcer.

A musician, aged 58 years, was admitted to Edinburgh Royal Infirmary, on 21/1/47. For six months he had experienced attacks of pain over the upper part of the sternum, crushing in character, and radiating to the shoulder and arm as far as the elbow on the left side. The pain was brought on by exertion, over-eating, excitement, and cold; and was relieved by rest, leaving the præcordium and the left arm numb. The attacks became progressively more severe, more frequent, and more easily provoked by exertion.

Three months from the onset he experienced a violent attack of sternal pain while at rest in bed; the pain was very severe, crushing in character, and radiated down the left arm to the fingers. It persisted all night but eased next day. He was not confined to bed at this time but remained in the house resting. During the following months he had several further attacks of pain, usually brought on by very slight exertion, such as washing or dressing. Electrocardiograms in November 1946, showed little abnormality in the resting records apart from left axis deviation, but conspicuous S-T distortion after exercise.

The patient had suffered periodically from indigestion for 20 years. The attacks were characterized by pain and distension in the epigastrium, relieved by food and alkalis. A barium meal examination three years before had shown no abnormality.

The family and social history was irrelevant.

On examination he was of average height and build, anxious and worried, but without cyanosis or œdema. Clinical examination showed slight cardiac enlargement, a blood pressure of 180/110, faint heart sounds with gallop rhythm and a soft localized apical systolic murmur. Radiologically there was slight enlargement of the left ventricle.

Abdominal examination showed no restriction of movement and no palpable mass or tenderness, but revealed guarding of the upper right rectus muscle. No other abnormalities were discovered on physical examination. The blood count was normal, the Wassermann reaction and urine examination negative. An electrocardiogram taken on 22/1/47 showed no gross abnormality.

The clinical diagnosis was angina pectoris with severe coronary arterial disease. The patient was treated with complete rest in bed, a low residue diet, and half a grain of phenobarbitone morning and night, and glyceryl trinitrate tablets, 1/100 of a grain, taken when required.

Progress. One week after admission his condition showed little change: even turning in bed brought on an attack of angina pectoris.

At 12 noon on 29/1/47 he suddenly cried out with pain and lay completely motionless with flexed knees. His pulse was 90 a minute, the temperature 96° F., the respiration rate 28 a minute, and the blood pressure 90/50. Examination revealed entire absence of abdominal

movement on respiration and marked muscular guarding of the upper abdomen with extreme tenderness on palpation in the epigastrium. There was slight diminution of the liver dullness. The heart sounds were faint.

The provisional diagnosis of perforated peptic ulcer was confirmed by a surgeon, but in view of the patient's poor general condition it was decided to adopt conservative rather than operative treatment.

One hour later the patient stated that the pain had spread over the left side of the chest and down the left arm to his fingers. Previously immobile, he was now extremely restless and was bathed in a cold sweat. His pulse had risen to 100 a minute but respiration was unchanged in type and rate. The findings on abdominal examination were similar to those an hour before. Electrocardiograms at 1.20 p.m. and 4.20 p.m. showed changes indicative of recent cardiac infarction (Fig. 1, B and C).

In view of the cardiographic evidence and the history of coronary disease, the provisional diagnosis of perforation was revised. It was assumed the patient had another cardiac infarction. He was treated with large doses of morphia and absolute rest.

During the following six days the patient felt more comfortable, and had little discomfort after the first 48 hours. His pulse, however, continued fast (115–120), his gallop rhythm persisted, and basal crepitations were heard. There was now fever (101° F.) with slight leucocytosis (10,800–11,400) and a fast B.S.R. (24–30 mm. Westergren). Abdominal guarding and epigastric tenderness persisted. The cardiographic changes are discussed later and in more detail in the legend to Fig. 1.

On 6/2/47 the patient began to suffer from cardiac asthma. His pulse rate rose to 130 a minute and he developed coupled beats. The respiratory rate was 36 a minute. The apex beat was 3 cm. outside the mid-clavicular line in the sixth left interspace. The heart sounds were feeble, there were many extrasystoles, and the apical systolic murmur persisted. Dullness on percussion and coarse crepitations were elicited on examination of the bases of the lungs. The patient was treated with continuous oxygen, aminophyllin, and morphia, but death occurred 10/2/47 from left ventricular failure.

Electrocardiographic changes. The routine curve taken at 11.40 a.m. on 29/1/47 is of a type common in cases of myocardial disease of coronary origin, the conspicuous abnormalities being the T inversions in leads I and IVF: it was the same as earlier records.

The record taken at 1.20 p.m. shows marked S–T shifts in chest leads indicative of "injury currents"; the nature of the changes with the history of pain rendered an ischæmic lesion likely. In the record three hours later QRS changes in limbs and chest leads, though slight, are apparent and were read as confirming infarction. The appearance of a small Q in the apical lead, together with the grossly aberrant QRS in leads C₂F suggested an early anterior infarction.

The subsequent records are consistent with a developing infarct and the deep Q in the apical lead is characteristic of such a lesion underlying that electrode. The cardiographic diagnosis was a large coronary infarction involving *inter alia* the apical region.

Necropsy. This was performed on 11/2/47.

The pericardial sac contained 150 ml. of blood-stained serous fluid. The visceral pericardium showed a loss of lustre over the anterior, postero-inferior, and apical regions of the heart. A fibrinous exudate and areas of fibrous thickening were present in the posterior part of the pericardial sac. The heart weighed 400 g.; it was enlarged and showed generalized bulging of the apex and lateral aspect of the left ventricle. The anterior surface, apex, and lower part of the postero-inferior aspect of the heart were the seat of infarction, and internally this extended to the left ventricular wall and the interventricular septum. The myocardium was necrotic, thin, and covered with ante-mortem thrombus. The chambers and auriculo-ventricular orifices were dilated. The valves were healthy. The coronary arteries were

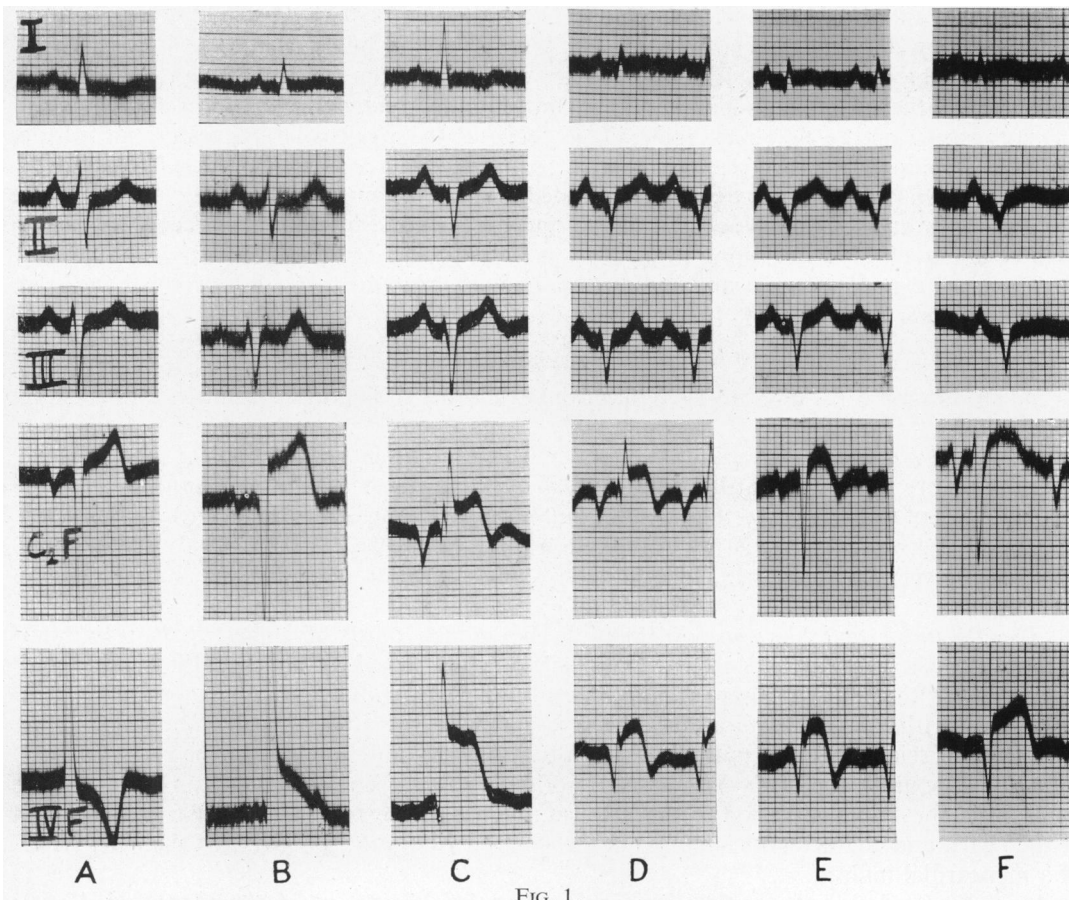


FIG. 1.

- (A) 29/1/47, 11.40 a.m. Sinus rhythm, rate 98 a minute; P-R interval 0.18 sec.: left axis deviation; shallow inversion of T in lead I, upright T waves in leads II and III. The sternal chest lead (C₂F), shows a deep Q, and trace of S-T elevation and upright T waves: the T waves in lead IVF are deeply and sharply inverted.
- (B) 29/1/47, 1.20 p.m. Sinus rhythm persists with P-R of 0.16 sec., and left axis deviation. The T waves in lead I are now low upright. In lead C₂F S-T elevation exceeding 5 mm. in height has developed without changes in QRS or T. In the apical lead a tiny initial Q precedes the tall R, marked S-T elevation is present, and the inverted T waves have disappeared.
- (C) 29/1/47, 4.20 p.m. In this record further changes are apparent, viz.: disappearance of R in lead II; increase in amplitude of R in lead I; diphasic T waves in lead I; gross changes in form of QRS in lead C₂F, with increase in size of vestigial Q in lead IVF; conspicuous "coronary" S-T-T segments in both chest leads.
- (D) 30/1/47. Sinus tachycardia rate approx.: 120 a minute: marked decrease in voltage of QRS in all limb leads; persistence of splintered upright QRS in lead C₂F: development of deep Q as main deflection in lead IVF; beginning inversion of terminal position of T in both chest leads.
- (E) 31/1/47. The principal changes from the last record are increased slurring of QRS in limb leads; shallow inversion of T in lead I; steep downward initial deflection in lead C₂F; and increase in depth of T inversion in both chest leads.
- (F) 10/2/47. This record shows further reduction in voltage in limb leads, involving QRS and T waves; return of initial deflections in lead C₂F to an rS pattern, and conspicuous if in the apical lead, with S-T elevation and upright T waves in both chest leads.

atherosclerotic with greatly narrowed lumina. Recent ante-mortem thrombus was present in the descending branch of the left coronary artery. The aorta showed minimal atherosclerosis in its descending portion only.

An abscess was present in the peritoneal sac, localized in the space between the hepatic

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flexure, liver, and duodenum, walled off from the peritoneal cavity by dense fibrous adhesions and containing 200 ml. of thick, foul smelling, greenish yellow pus. There was no generalized infection of the peritoneum. The abscess had originated from the perforation of a chronic peptic ulcer situated in the duodenum. The duodenum presented a peptic ulcer at the antero-inferior aspect of the first part immediately beyond the pylorus. The crater, measuring 12 mm. \times 5 mm. had a hard sclerotic base and firm edges. A small perforation had occurred at the upper end, the lower extremity had eroded into the head of the pancreas.

The appearance of the myocardial infarct and the localized peritonitis indicated they were both of about 14 days' duration.

There were no other relevant findings apart from bilateral hydrothorax and congested and œdematous lungs.

DISCUSSION

When the case is reviewed in the light of the post-mortem findings a number of interesting points arise.

The Duodenal Ulcer. Despite the complaint of intermittent indigestion for 20 years, the symptoms were mild, and hardly suggestive of duodenal ulcer, still less of an ulcer eroding into the head of the pancreas; the constant dull boring pain characteristic of this complication of peptic ulcer was absent. X-ray examination three years ago had been negative: the ulcer may have developed since then, though its size and character suggested that it was of long standing.

The Diagnosis and Sequence of Events. The sudden onset of epigastric pain without radiation to the arm, as had occurred on each previous attack of angina; the rigidity and tenderness in the upper abdomen; the diminution of liver dullness; the thoracic type of respiration with grunting expiration; the severe shock; the attitude and immobility of the patient; the relatively slow pulse and the furred tongue, all supported a diagnosis of perforated peptic ulcer, especially with a history of indigestion. The occurrence an hour later of pain in the chest with radiation to the left arm, intense restlessness, rapid pulse, enlargement of the heart, and characteristic changes in the electrocardiogram, all indicated the occurrence of a myocardial infarct.

It is suggested that the perforation occurred at the time the patient suddenly cried out; and, due to the shock, low blood pressure, and poor flow blood, thrombosis occurred in the descending branch of the left coronary artery, that was already the seat of gross atherosclerosis. This was the cause of the extensive myocardial infarction, later causing left ventricular failure and leading to death.

The Perforated Duodenal Ulcer. Post-mortem, the abscess was well localized between liver, hepatic flexure, and duodenum, and was completely walled off from the peritoneal cavity. If the patient had not died from coronary thrombosis the perforated ulcer would presumably have healed by conservative treatment, thus supporting the views of Turner, Taylor, and Visick, all of whom have advocated a conservative régime for these cases.

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REFERENCES

- Turner, B. (1945). *Brit. Med. J.*, 1, 457.
 Taylor, H. (1946). *Lancet*, 2, 441.
 Visick, H. (1946). *Brit. Med. J.*, 2, 941.