Medical Memoranda

Primary Traumatic Coronary Thrombosis

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Traumatic coronary thrombosis is a very rare condition, and in the present case it was associated with traumatic rupture of the liver. Both conditions were caused by a non-penetrating blow on the chest, and proved fatal some 44 hours after the original injury.

Case Report

A wood machinist aged 42 was admitted to the surgical wards of the Southern General Hospital, Glasgow, at 1.45 p.m. on 17 December 1951 approximately three-quarters of an hour after sustaining an injury to his chest. He was feeding a piece of wood (1 by 7 in. by 4 ft.; 2.5 by 18 by 122 cm.) into a circular saw which "kicked back," and the wood, end on, struck him forcibly on the lower left side of the chest and upper left quadrant of the abdomen. Though winded and unable to speak for about five to ten minutes afterwards, he was not unconscious and did not vomit. Dull substernal pain had persisted since its onset at the time of the accident. There was no personal or family history of heart disease.

The patient, a well-built man, was severely shocked, cold, sweating, with ashen countenance and greyish cyanosis of the lips. Oedema, congested neck veins, and dactylopathy were absent. There was an abrasion over the left lower ribs, extending from the base of the body of the sternum to the tip of the tenth costal cartilage. There was also a dark bluish, tender, fluctuant swelling, 9 by 4 in. (23 by 10 cm.), in the subcutaneous tissues deep to the abrasion.

Blood pressure was 62/40 mm. Hg. The pulse showed a regular irregularity due to group beating of variable strength. Auscultation over the brachial artery with variable sphygmomanometric pressure confirmed that arterial pulsation was in groups of five to six beats of gradually diminishing strength, the first and second beats of each group being palpable at the radial pulse. The heart rate appeared to be 68 a minute. Pulsation was not present on the chest wall and the heart was not definable by palpation or percussion. There was generalized abdominal tenderness and borborygmi were present. The chest was somewhat barrel-shaped with a hyperresonant percussion note, but adventitious sounds were absent, and further systematic examination did not show any abnormality.

The patient was sedated with ¼ gr. (16 mg.) of intramuscular morphine sulphate, and three-quarters of an hour later, after 1½ pints (850 ml.) of plasma, the blood pressure was 90/60 mm. Hg. The heart sounds, which had become audible, were faint and of poor quality but pure at all areas. *X*-ray examination of the chest and abdomen showed nothing abnormal. The haemoglobin was 86% (Sahli). An electrocardiogram at 5 p.m. showed myocardial damage and a defect of conduction. A blood transfusion was started but had to be discontinued because of acute pulmonary oedema. A further electrocardiogram at 12 noon the next day still showed myocardial damage and revealed that the conduction defect was due to right bundle-branch block. The patient's condition gradually deteriorated, and in the evening he became orthopnoeic and presented the classical signs and symptoms of acute left ventricular failure, with a pulse rate of 140 a minute. Treatment with digoxin and morphine was carried out, but further deterioration took place and the patient died at 8.30 a.m. the next day.

Electrocardiograms.—Two electrocardiograms were taken: the first, four hours after the accident, was of standard leads only (Fig. 1) and the second, 22 hours after the accident, included standard leads, unipolar limb leads, and precordial leads V2, V4, and V6 (Fig. 2). In all records time = 0.04 and 0.2 second, and amplitude 1 mm. = 0.10 mV. The changes in the first electrocardiogram (Fig. 1) are indicative of acute myocardial damage and show an intraventricular conduction defect. It is noteworthy that Q waves are absent and ST segments raised in all three leads. Comparison of Fig. 1 with the standard leads of Fig. 2, taken 18 hours later, shows rapid improvement, which suggests that the myocardial damage is, at least in part, due to a somewhat transitory process. In aVF (Fig. 2) Q waves are absent, but in V6 small Q waves are present. Though the changes are not diagnostic they are consistent with the presence of an acute posterolateral wall infarction. The intraventricular conduction defect is now recognizable as a right bundle-branch block.

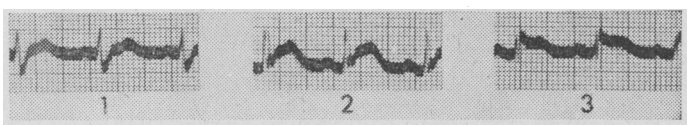

FIG. 1.—Standard lead electrocardiograms, four hours after injury, show acute myocardiai damage and an intraventricular conduction defect.

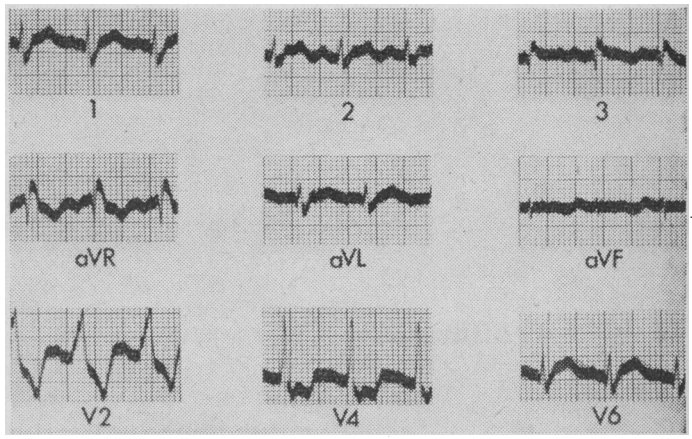

FIG. 2.—Standard, unipolar limb, and chest lead electrocardiograms, 22 hours after injury, are consistent with acute posterolateral wall infarction. The intraventricular conduction defect is now recognizable as a right bundle-branch block.

Necropsy Findings

External Examination.—There was marked bruising of the anterior surface of the body in the region of the xiphisternum with a large haematoma immediately superficial to the upper end of the rectus adominis muscle.

Internal Examination.—Some effusion of blood was observed between the outer layers of the anterior surface of the pericardium,

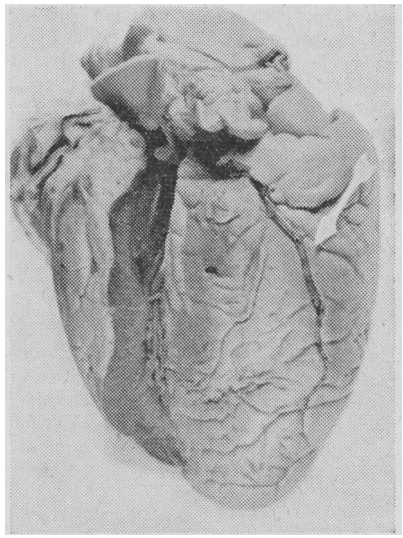

FIG. 3.—Photograph of heart showing, at arrow, a large organizing thrombus in circumflex branch of the left coronary artery.

and the sac contained 30 ml. of blood-stained fluid. The heart (460 g.) was commensurate in size with that of the body. The myocardium appeared healthy and there was no evidence of dilatation of either ventricle. All valves were normal. In the coronary arteries there was no macroscopic evidence of atheroma, but a large organizing thrombus was present in the circumflex branch of the left coronary artery (Fig. 3). Both lungs showed fairly marked congestion, with slight oedema. There was no evidence of bronchitis. Pleurae and trachea were healthy. The aorta was elastic and quite free from atheroma. There was marked bruising of the anterior surface of the upper peritoneum, and the cavity contained about 2 litres of blood mixed with bile. In the liver (1,630 g.) there were several extensive ragged lacerations in the under surface of the left lobe. The gall bladder was healthy but was quite empty. There was a small laceration in the common bile duct. The entire alimentary tract, pancreas, suprarenals, spleen (190 g.), and kidneys (120 g. each) were healthy.

Microscopical Examination.—A section of the circumflex branch of the left coronary artery, 0.5 cm. proximal to the proximal end of the thrombus, showed minimal atheroma.

COMMENT

This is a case of primary traumatic coronary thrombosis. It is apparent that the thrombosis occurred from injury to a previously healthy heart, for the onset of symptoms was contemporaneous with the injury. Further evidence is provided by clinical data at three-quarters of an hour, by electrocardiograms at four hours, and by necropsy after death at 44 hours.

Most cases claimed to be similar are secondary to advanced heart disease or posttraumatic—that is, with an interval between the injury and the thrombosis. It is becoming widely accepted that traumatic heart disease is more common than had been previously recognized. This case, together with that described by Meessen (1940), establishes that primary traumatic coronary thrombosis is an entity.

It is a pleasure to acknowledge my indebtedness to Professor W. A. Mackey for permission to report this case; to Dr. M. M. O'Hare for her report on the electrocardiograms; to Dr. A. Dick for the necropsy report; and to Dr. J. H. Wright for his comments on the manuscript.

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Fenfluramine Overdosage

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Fenfluramine hydrochloride (Ponderax), superficially a compound of an amphetamine-type structure, was reported by Munro *et al.* (1966) to be an effective appetite depressant devoid of stimulant effects on the central nervous system. The only side-effect observed was severe diarrhoea in 2 of the 25 patients who received the drug. This lack of stimulation, together with the lack of hypertensive effect (Le Douarec and Schmitt, 1964; Lambusier, 1965), is in contrast with observed effects with amphetamines. In view of the present concern over the dangers of drug habituation and the widespread use of anorectic drugs this is not without importance.

We report a case of self-poisoning with fenfluramine hydrochloride, together with some laboratory findings.

CASE REPORT

A 13-year-old girl was admitted to New End Hospital half to one hour after having taken approximately 15 tablets of Ponderax (20 mg.)—that is, 300 mg. of fenfluramine hydrochloride. On arrival she was very drowsy and uncooperative, refusing to say what had happened. She had vomited twice, was shivering, and said she felt cold. On examination the extremities were cold, the pulse was of good volume and regular at 88 per minute, and the blood pressure was 110/70 mm. Hg. The pupils were widely dilated and did not react to light until three hours after admission. The cardiorespiratory system and abdomen were normal. The central nervous system was normal apart from sluggish tendon reflexes. This combination of symptoms and signs is most uncommon in other forms of poisoning. A specimen of blood was taken for drug estimation and gastric lavage performed. The patient made an uneventful recovery.

The specimen of blood and a sample of the stomach washout were analysed at Chelsea College of Science and Technology by gas-liquid chromatography. The blood sample was found to contain 0.24 μg. of fenfluramine and 0.04 μg. of norfenfluramine, a metabolite of fenfluramine, per ml. The stomach washout contained approximately 3 mg of fenfluramine hydrochloride.

COMMENT

Previous work has been carried out in these laboratories on excretion patterns of this drug after various oral and intravenous doses to humans. An oral dose of 180 mg. of fenfluramine hydrochloride has been shown to give blood levels of 0.08 and 0.16 μg. of fenfluramine per ml. one hour and two hours respectively after taking the dose. Extrapolating from these results, the level of fenfluramine in the blood specimen from the patient indicates that approximately 360 mg. of fenfluramine hydrochloride—that is, 18 tablets—was taken, an estimate in agreement with the report from the patient.

Though the samples were received and analysed two weeks after being taken, storage tests over four weeks show that deterioration is negligible and that no interfering peaks occurred on the chromatogram if the samples were refrigerated. The analysis was carried out on a Perkin–Elmer F.11 gas chromatograph with a flame-ionization detector; the method used was similar to that for the detection and identification of amphetamine (Beckett and Rowland, 1965).

Ten cases of fenfluramine overdosage (unpublished) in France have been reported to the manufacturer, but no fatalities have occurred. Of these 10 patients three took 30 tablets, five took 40, one took 80, and one took 90. For most of these cases full details are not available, but the patient who took 80 tablets presented with symptoms of trismus and transient spasm of other muscles and was semiconscious. Recovery was spontaneous and rapid but there was loss of appetite for one month afterwards.

We are grateful to Dr. Cecil Symons for permission to publish this case; to Dr. L. T. Newman, the patient's family doctor, for preadmission details; and to Selpharm Laboratories Limited for information from sources in France and for supplying fenfluramine hydrochloride for experimental studies.

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