ACUTE PANCREATITIS: ITS CAUSES, SYMPTOMS, AND TREATMENT.

WITH A DESCRIPTION OF THREE CASES.*

By ARTHUR J. EVANS, F.R.C.S. EDIN.,

HONORARY SURGEON, STANLEY HOSPITAL, LIVERPOOL; VISITING
SURGEON, BROWNLOW HILL INFIRMARY, LIVERPOOL.

The rapid development of abdominal surgery in recent years, especially as regards the treatment of diseased organs lying in the upper abdomen, has in a large measure been due to the pioneer work of Mayo Robson and Moynihan, and to this must be added the no less important pathological, chemical, and experimental work of Cammidge, Opie, Flexner, and others.

We have now, therefore, a very precise knowledge of the intimate inter-relationship between the biliary passages, the duodenum and the pancreas, and how disease of one of these structures may affect the other, more especially so if it be of a catarrhal or microbic nature.

It has been my lot within the past eighteen months to operate on three cases of acute pancreatitis, a condition which, not many years ago, often passed unrecognized, even on the operating table and sometimes in the *post-mortem* room.

The recognition of acute pancreatitis from a clinical standpoint during life is still an exceedingly difficult matter, and in only one of my cases was the diagnosis suspected before the abdomen was opened.

CASE I.

E. J., male, aged 35, a dock labourer, was brought into hospital in a state of collapse. He had been drinking heavily during the previous ten days, and had hardly been sober during that period.

He had been vomiting frequently for some sixteen hours, and complained of intense paroxysmal pain in the epigastric region. The conjunctivae were slightly tinged yellow; there was no jaundice of the skin; the symptoms were typical of an "acute abdomen."

He vomited some black altered blood during my examination.

The abdomen was retracted, and the recti firmly contracted in their upper half. The patient, at intervals, lapsed into delirium; the pulse was exceedingly rapid and thready, the temperature subnormal.

There was no loss of liver dullness. A perforation of the stomach or duodenum was suspected, and an immediate laparotomy was performed under open ether, the region of the stomach being explored by the usual median incision. The peritoneum and omentum was found to be oedematous and swollen and of a deep red colour.

Recent adhesions were evident in the anterior abdominal wall.

The stomach and duodenum were carefully examined, and there was found no evidence of perforation.

The omentum was separated from the anterior abdominal wall, to which it was adherent, and on its under-surface were several well-marked white areas, varying in size from that of a millet seed to a shilling. It was then at once recognized that the pancreas must be the seat of the disease.

On exploring the small peritoneal sac with the finger, a large mass of black necrotic tissue, together with blood clot, was dislodged. A large drainage tube was passed down to the pancreas and the wound closed hurriedly, owing to the collapsed condition of the patient.

He died six hours after the operation.

The autopsy disclosed extensive inflammation of the common bile duct and upper part of the duodenum. There were no calculi in the biliary passages; the pancreas was to all intents and purposes unrecognizable as a definite organ. The head consisted of a large necrotic cavity, filled with blood clot and debris; the body and tail were swollen, red, and oedematous, and exceedingly friable, so much so that they could not be removed except in small portions.

CASE II.

A. F., aged 51, female, was brought into hospital in March, 1912. She had been vomiting for some days previously, and had suffered severe pain in the upper abdomen, which came on in paroxysms, for the past four months.

This culminated in a very severe attack previous to admission. She had had several "bilious attacks" during the past seven years, but had never had jaundice to her knowledge. She had been in the habit of drinking alcohol, but not heavily. The patient was of the fat, plethoric type, and was exceedingly collapsed on admission. There was no jaundice. On examination of the abdomen there was no undue distension, the recti, especially in their upper half, were firmly contracted,

and she complained of excruciating pain on palpation over the region of the stomach and transverse colon. The liver was not enlarged, but there was tenderness over the region of the gall bladder. No other abnormality could be distinguished, but she was a difficult case to examine owing to her obesity.

The pulse was 130, and the temperature subnormal. Laparotomy was immediately performed, a vertical incision being made in the upper part of the right rectus, which would command the exploration of the bile passages, and the pyloric region of the stomach. On opening the parietal peritoneum the gastro-colic omentum was exposed. This was seen to be somewhat matted together and covered with white, glistening plaques of various sizes and irregular shapes, standing out in marked contrast to the yellow, fatty membrane in which they were embedded. "Fat necrosis" was at once recognized.

The whole omentum was then drawn through the wound and its under surface examined; this was studded over with shining clusters of "fat necrosis," and presented a picture never to be forgotten. Exploration of the small peritoneal sac with the finger released some blood-stained fluid, and beneath the finger the pancreas was felt to be swollen and oedematous. The peritoneum in front of it was gently torn through and a quantity of dark-brown-stained fluid was evacuated; a large drainage tube was inserted down to the pancreas through the gastro-colic omentum. The gall bladder, which was distended, was then opened through the upper part of the incision. It was found to contain a large quantity of thick, viscid bile and several small calculi. A drainage tube was sutured in and the wound closed.

The patient made a steady and uneventful recovery and is alive and well to-day.

CASE III.

D. C., male, aged 52. I am indebted to Dr. Touch, of St. Helens, for the notes on this case. The patient had been a very healthy man up to the illness. He had had pneumonia fourteen years previously and had entirely recovered from it. He was extremely temperate as regards alcohol but was a heavy smoker; he was very regular in all his habits and lived a generally healthy life. There was no history of any biliary trouble; he had put on flesh very rapidly during the past six months.

On November 6th, 1912, he became suddenly ill, with severe agonizing epigastric pain, the pain being entirely localized to the epigastric region. This pain lasted some twenty-four hours and was accompanied by frequent vomiting of some brownish material. The abdominal muscles were not rigid, but the tenderness made palpation difficult. Pulse-rate 120, small and regular. After the pain disappeared, deep palpation gave the impression of a fairly large tumour behind the stomach, but, as the acute symptoms passed off, it was decided to watch the case further.

There was obstinate constipation for the first few days; this was followed by diarrhoea, six to eight stools a day being passed up to the date of operation. They were greenish in colour and extremely offensive. The urine was found to contain sugar, and was of a high specific gravity, 1025. The patient remained in a more or less quiescent state for nearly a month, but on December 2nd he was again seized with an attack, the feature of which was precisely the same as in the first instance. Some abnormal condition of the pancreas was then suspected.

I saw him in consultation on December 4th, and an operation was decided upon. This was performed at St. Helens Hospital the next day.

On opening the abdomen extensive adhesions were found between the omentum and the anterior abdominal wall; the peritoneum and omental fat were stained a dirty red colour. No fat necrosis could be seen owing to the general discoloration of the tissues. An irregular mass could be felt behind the stomach, lying in relation to the small peritoneal sac. The foramen of Winslow was occluded by adhesions. The finger was introduced into the small sac above the stomach, via the gastro-hepatic omentum; and a large quantity of brown "muddy" fluid was evacuated through the opening, followed by brown-coloured debris and blood clot. The whole of the posterior portion of the upper abdominal cavity appeared to be filled with the same material. As much as was possible was removed by gauze swabbing, but, owing to the extremely collapsed condition of the patient, a large quantity had to be left behind. A large drainage tube was passed down to the pancreas, and the wound sutured.

The condition of the gall bladder and common bile duct could not be ascertained owing to the extensive adhesions around and the critical condition of the patient.

The patient showed marked improvement immediately after the operation. The sugar, which previously had been found regularly in the urine, entirely disappeared. On the seventh day, however, he showed signs of delirium, which came on at intervals. This gradually increased, with other signs of internal toxæmia. He died ten days after the operation. An autopsy could not be obtained. Two interesting features marked this case: First, the occurrence of two separate acute attacks, with an interval of twenty-six days between. Secondly, the appearance of glycosuria after the first attack, and its immediate disappearance after the operation.

Acute pancreatitis is a disease of adult life, and is more frequently found in males than in females. It occurs very rarely in children, as a metastatic manifestation following mumps.

* Read before the North Wales Branch of the British Medical Association.

Cases have also been reported as being due to traumatism. It has been produced experimentally by injecting bile and bile salts into the pancreas of dogs; and it has been found to have occurred in the human subject by the impaction of a gall stone in the ampulla of Vater, thus deviating the bile, which has been previously infected by cholangitis, into the duct of Wirsung, and thence into the head and body of the pancreas.

It has again been shown that the injection of the secretions of the duodenum alone into the pancreas has been followed by acute necrosis; this coincides with the *post-mortem* conditions sometimes found after death from this disease, where the duodenum is inflamed, and in some cases the seat of ulcer. It has, however, been clearly shown from the existing literature that at least 50 per cent. of all cases of pancreatitis are complicated by gall stones or cholangitis.

The histological structure of the gland, as Mayo Robson and Cammidge have pointed out, are of importance in view of the acute changes which may take place as the result of microbic or inflammatory invasion. The gland itself has no true capsule; its lobes and lobules are but loosely held together by delicate connective tissue, while the blood vessels and ducts are much thinner and finer than those found in the salivary glands. The digestive ferments also which it secretes are the most powerful in the body, thus rapidly producing, when set free, auto-digestion in the gland itself.

It follows, therefore, that when the organ becomes invaded—via its ducts—by any irritant, and more especially so by infective material, a rapid and extensive inflammatory reaction results.

Acute haemorrhagic pancreatitis, gangrenous pancreatitis, and purulent pancreatitis are simply phases of the same disease. Haemorrhage probably follows the initial inflammation, the haemorrhage causes a rapid disintegration of the friable organ, and rapid destruction of its structure takes place. Added to this, its powerful digestive ferments are let loose in the abdominal cavity, and one striking evidence of their potency is found in the presence of so-called "fat necrosis." This latter condition is due to the "steapsin," or fat ferment, splitting the fat of the gland itself into fatty acid and glycerine, together with any other fatty structure it may meet. The soluble glycerine is absorbed, while the fatty acids are deposited in needle-like crystals in the fat cells. These altered fat cells are arranged in clusters or patches, and are of a striking dull-white colour, and stand out in unmistakable contrast to the surrounding yellow fatty tissue.

Symptoms.

These are at first vague in the early stages. There is often gastric uneasiness, flatulence, obstinate constipation, and persistent vomiting.

After some hours—sometimes several days—the patient is struck down by agonizing unbearable pain in the epigastric region, often shooting towards the back. This is followed by vomiting and collapse.

The pain is continuous, with periods of paroxysmal increase, and it may be so intense as to cause the patient to faint.

There is also acute tenderness of the upper abdomen, especially so in the middle line.

The pulse becomes rapid and thready, and assumes the abdominal type. The temperature is usually normal or subnormal, but as inflammation increases it may become irregular. In two of my cases there was marked cyanosis, in striking contrast to the pale, pinched aspect of ordinary peritonitis.

Constipation is a marked feature, so much so that this condition has been mistaken for intestinal obstruction.

Delirium is very constant, and was particularly marked in my third case, alternating with perfect mental clearness.

Jaundice may be present. It was noticed in Case 1. Its presence and its intensity are entirely dependent upon whether there is obstruction in the bile passages or not.

Sugar is rarely found in the urine, although it was present in my third case.

If the disease has progressed for some days a definite mass can be felt in the epigastric region; it is well defined, circumscribed, and limited to the upper abdomen. In

front of it the stomach and colon can be percussed. The mass is due to localized peritonitis, and the accumulation of blood and inflammatory exudate in the small peritoneal sac in front of the pancreas.

Diagnosis.

This is exceedingly difficult, and in most cases impossible. Given, however, a patient who has been previously healthy and who begins to complain of flatulence and dyspepsia, this being followed rapidly by the acute symptoms already referred to, with the formation of a definite circumscribed mass in the upper abdomen, the presence of pancreatitis is probable. The differential diagnosis must be made from a perforating gastric or duodenal ulcer, intestinal obstruction, acute inflammatory affections of the bile passages, and acute appendicitis.

The very nature of such symptoms warrant, however, an immediate exploratory laparotomy, and in most cases it is only by these means that the diagnosis can be made with certainty.

Cammidge's test, which depends upon the demonstration of pentose and other substances in the urine, has proved highly successful in his own hands, but its complexity, together with the length of time involved in performing it, do not warrant its use in so rapid and lethal a disease as pancreatitis in its acute form.

Treatment.

The treatment is wholly surgical—immediate laparotomy, with exposure of the pancreas, and its free drainage. If the patient's condition will allow it, drainage of the gall bladder also should be established. If this is not possible at the time, the gall bladder should be drained as soon as the patient's condition improves sufficiently. The mortality is exceedingly high. Fully 60 per cent. of the recognized cases succumb after operation; without operation it is almost certain death.

The intensity of the disease depends in a large measure on the amount of gland destroyed, the actual cause of death being trypsin poisoning.

This protein-splitting enzyme has only a feeble digestive action of itself, but when brought into contact—as shown by Pawlow—with the ferment "enterokinase" found in the succus entericus, its digestive action is rapidly brought into play. When, therefore, following the rapid disruption of the gland, it is thrown into the free abdominal cavity, its action on the tissues is not of a digestive nature, but it is absorbed, and produces that intense "lethal" condition, which is so characteristic of this interesting but deadly disease.

It is stated that there are now some 250 legally qualified women practising medicine in Japan, and that there is an almost equal number of women studying medicine in the University of Tokyo. One of the chief private hospitals in the Japanese capital is that carried on under the direction of Dr. Yoskoka Yacoi, one of the leading medical women of Japan; it has an out-patient department where the average attendance of patients is about eighty a day. The movement for the repeal of the law forbidding women to practise medicine was started by a lady named Hagino Yosci-Ko in 1884.

HEISER, in the *Philippine Journal of Science*, vol. viii, Section B, No. 2, April, 1913, describes the recent outbreak of plague in Manila. He states that after an absence of six years in human beings, and five years among rats, plague was again found in the Philippine Islands on June 9th, 1913. Manila is in almost daily communication with badly plague-infected foreign ports, which are within a few days' steaming distance for the average vessel, and therefore passengers, crew, rodents, and vermin may arrive well within the incubation period of the disease. This being the case, it seems remarkable that the Philippines should have remained free from plague for so many years. During the period of its absence, however, cases of the disease have been detected from time to time amongst human beings upon incoming vessels, but such infections have always been intercepted at quarantine stations. As the outbreak was still going on when Heiser's paper was written, he has confined himself strictly to a statement of facts, and for the present has not attempted to draw any conclusions as to the source of origin of the outbreak.