Conclusion

1. A case of primary diffuse lymphosarcoma of stomach with polypoid masses has been described.
2. Metastasis has been shown to occur in liver, small intestine, pancreas, spleen, kidneys, and bone marrow.
3. The important points in the differential diagnosis of the condition have been indicated.

My thanks are due to my colleague Dr. S. P. Gupta for his help in taking the photomicrographs.

REFERENCES

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STRANGULATION OF TRAUMATIC DIAPHRAGMATIC HERNIA OCCURRING NINE MONTHS AFTER ORIGINAL ACCIDENT

BY

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(With Special Plate)

In view of the increasing number of cases of strangulation of traumatic diaphragmatic herniae occurring both at the time of the accident and even years later, the following case appears to be of interest. It exemplifies Sir James Mackenzie's saying that "pain is the most important of complaints and the most instructive diagnostic sign," and illustrates the value of Zachary Cope's dictum that "the majority of severe abdominal pains which ensue in patients who have been previously fairly well and which last as long as six hours are caused by conditions needing surgical intervention."

Case Report

In June, 1935, a married woman, aged 52, was admitted to a medical ward of the Hull Royal Infirmary for obscure abdominal symptoms. She was incoherent, and a definite history was difficult to obtain. She complained of constant abdominal pain, obstinate vomiting of four days' duration, and also of considerable pain in the top of the left shoulder and between the shoulder-blades. Her bowels "had not moved" for a week, and she vomited immediately after she took anything. An enema had been given on the morning of admission with no result of flatus or faeces.

The patient gave a history of a motor accident in September, 1934, as a result of which several left ribs were fractured and her left leg had to be amputated. She had been troubled with bilious attacks from childhood, and had been perfectly fit since the accident until the present symptoms developed.

EXAMINATION

The patient looked extremely ill, and was evidently in great pain. She was slightly cyanosed, with some icteric tinge and was extremely restless, tossing about in bed, but she obtained a little relief by sitting up and leaning forwards. She was vomiting copiously. The symptoms were augmented when she lay down, so much so that examination in the supine position was made extremely difficult by her constant desire to resume "the sitting up and leaning forward" position. The abdominal pain was made worse when deep respirations were attempted, as was the pain on the top of the left shoulder.

Examination of the abdomen disclosed no visible mass and no distension. The organ moved very poorly with respirations, and on palpation appeared tender all over, with maximum tenderness just below the left costal margin. There was some rigidity of the upper left rectus, slight diminution of liver dullness, and a tympanitic note in the right flank. No sounds were audible on auscultation, and nothing abnormal was revealed per rectum.

Chest.—Diminished expansion of left chest; slight dullness left base; diminished air entry whole of left chest, and absent breath sounds left base. No adventitia. *Heart*.—Sounds distant and foetal in character, but no evidence clinically of displacement. Well-marked basal pericardial rub. Patient complained of pain in precordial region on attempt to palpate the apex beat.

This factor and the pain on palpation of the lower left rib margin in front and behind I unfortunately discounted.

An enema was given, but without result. The urine contained a trace of albumin. The pulse was 120, respirations 25, and temperature 98° F.

DIAGNOSIS

This presented great difficulty. Coronary occlusion, acute intestinal obstruction, and acute pancreatitis were all suggested. I considered diaphragmatic hernia, but discarded this diagnosis, having an erroneous idea that hernia would not occur so long after the accident. It was evident that operative intervention was urgently required, and a tentative diagnosis of acute pancreatitis was made. No x-ray examination of the chest and no laboratory investigation was carried out in view of the patient's poor condition, and of the need for immediate operation. Loewi's adrenaline test was negative.

OPERATION

This was performed, under light gas and oxygen anaesthesia and local muscle infiltration, within four hours of the patient's admission. There was no evidence of pancreatic necrosis. It was found that the fundus and proximal half of the stomach, the spleen, and a piece of omentum had passed through an opening in the posterior part of the left side of the diaphragm about two inches from the oesophagus and admitting three fingers. After the passage of a stomach tube and aspiration of the contents from the upper compartment of the stomach, reduction was accomplished. The stomach was distended and its wall congested and "purplish" in colour—but viable. The spleen and piece of omentum were successfully delivered through the opening. The opening in the diaphragm was then closed. The condition of the patient throughout the operation was poor, respirations ceasing on three occasions, but she left the table in definitely better condition than before operation. Continuous intravenous saline and carbon dioxide and oxygen inhalation was instituted, but the patient died two hours after operation.

POST-MORTEM EXAMINATION

Post-mortem examination disclosed collapse of the left lung with blood in the left pleural cavity, probably a post-operative occurrence. There were old healed fractures of the left second and third ribs, with adhesions of parietal pleura to the chest wall in this region, and old healed fractures of the eighth, ninth, and tenth ribs on the left side and about two inches from the vertebral column. There was a sutured tear in the posterior leaf of the left diaphragm about two inches from the oesophageal opening, which, on undoing sutures, admitted three fingers. Polypoid fleshy masses surrounded the chest surface of this opening like a frill, probably old organized blood clot. There was no evidence of a hernial sac. The heart was healthy. The small and large intestines were completely collapsed except for the upper few feet of the small bowel. The stomach was ballooned and purplish in colour, with excoriations on its visceral peritoneum, probably as a result of the nipping. The spleen was healthy. Nothing else of note was discovered.

Comments

It is now evident that the diagnosis should have been possible on admission, but the similarity to acute pancreatitis and the fact that due recognition was not given to the patient's history of an accident rather obscured the picture.

Harrington, in a résumé of sixty cases of diaphragmatic hernia, stresses the importance of: (1) the shoulder pain; (2) the cyanosis; (3) the obscure chest findings; and (4) the augmentation of the symptoms on lying down—all of which criteria this case presented. There may be

nothing in the patient's history to indicate the presence of a hernia until strangulation occurs. Polson records a case occurring twelve and a half years after the original accident, and only found at necropsy after death from acute strangulation.

Tixier, Patel, and Pollosson record a case diagnosed one month after a motor accident, confirmed by barium visualization of the stomach, and treated surgically three months later, the patient meantime suffering from dyspeptic symptoms whilst being kept under close observation, with numerous x-ray visualizations of the stomach to estimate the state of affairs. Boyce reports a successful case of operation for traumatic diaphragmatic hernia the day following the accident.

It is probable that traumatic diaphragmatic hernia is commoner than is generally realized, the chief causative factor being car accidents. It would appear that routine visualization of the stomach with barium is desirable in all cases of crushing injuries to the chest if the surgeon is to receive the case early, and not when some catastrophe such as acute strangulation has rendered his help imperative, but has vitiated the chance of a successful outcome to a difficult operation.

I wish to express my indebtedness to Dr. L. Lavine for permission to record this case, and to Mr. H. Upcott, who performed the operation.

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ANTE-MORTEM DIGESTION OF THE OESOPHAGUS

BY

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(With Special Plate)

The following is a case of ante-mortem digestion of the oesophagus, a condition which appears to be but little recognized in this country, although described by Pringle and Teacher some time ago.

Case History

A married woman, aged 48, was admitted to the Glasgow Royal Infirmary on November 21st, 1933, as a case of perforated duodenal ulcer. Operation was performed seven hours after the onset of symptoms, and a perforation of a duodenal ulcer on the anterior aspect of the first part of the duodenum was sutured. The abdomen was closed without drainage. The case progressed favourably until November 24th, when the patient had a slight haematemesis. From then until her death two days later she was frequently sick and vomited up small amounts of coffee-ground material. Just before death she vomited about one pint of black material.

POST-MORTEM REPORT

The body was that of a well-nourished middle-aged woman. There was coffee-ground material around the mouth. The heart appeared normal except that it contained a lesser quantity of blood than is usual. The lungs were normal except for some venous congestion behind. Oesophagus: The lower 8 inches showed a black discoloration which ceased in a straight line at the cardiac sphincter of the stomach, but faded off gradually in an upward direction, where the colour became more of a brownish black. The discoloured area was ulcerated and typical of ante-mortem digestion of the organ, the blackness being due to the soaking of the necrotic tissue

with haemolysed blood. The stomach contained a small quantity of coffee-ground material. There were two ulcers in the first part of the duodenum, one on the anterior wall being 7 mm. in diameter and the other, on the posterior wall, being 1.3 cm. in diameter. That on the anterior wall had perforated about its centre, but had been efficiently sutured. That on the posterior wall had as its base peritoneum at one part and pancreatic tissue at another. The bases of both were firm and indurated, showing them to be very chronic in type. Careful examination of the base of each disclosed no bleeding vessel. The rest of the intestine contained a small quantity of altered blood. All the other abdominal organs were normal.

Sections of the ulcers and of the oesophagus were preserved for further examination. Sections of the ulcers showed the typical appearances of chronic peptic ulcer. No bleeding point was found.

Sections from the affected area of the oesophagus (see Special Plate) showed complete destruction of the mucous membrane, which was replaced by polymorphonuclear leucocytes closely packed together, many of which showed degenerative changes. Superficial to this, and corresponding to the dark colour of the affected area macroscopically, there was a thin layer of brown altered blood pigment. Deep to the leucocytic layer necrotic debris with an occasional thrombosed vessel was seen. The cellular infiltration extended to a lessening degree through the muscularis mucosae, the superficial part of which showed patchy necrosis. There was a slightly increased cellularity of the fibrous layer between the muscularis mucosae and the muscular coats. The muscular coats themselves appeared normal.

Commentary

This interesting case is one of ante-mortem digestion of the oesophagus, which appeared to be almost unknown in British literature until described by Pringle and Teacher in 1919 and later in 1921. Before that a very complete account of the condition was given in Kaufmann's *Spezielle Pathologische Anatomie*. Teacher and Pringle in 1921 described a series of sixteen cases which they had observed over a period of seven years. Modern post-mortem experience suggests that the condition is now much less common than this.

During life the affection is characterized by the frequent vomiting of small quantities of dark brown sanguineous material, and in a small number of cases pain between the shoulders or behind the sternum is recorded, and is said to be very characteristic when present.

Post mortem the lower end of the oesophagus shows varying degrees of dark discoloration and necrosis, and in certain extreme cases described there has been perforation into a pleural sac. A characteristic feature is the contrast between the affected area of the oesophagus and the cardia of the stomach; for, whereas the former shows the varying degrees of necrosis and dark discoloration, the stomach itself has the normal post-mortem appearances, the ulceration ceasing abruptly at the cardiac sphincter. This was a notable feature of our own case and is a point of differentiation from post-mortem digestion, which affects the oesophagus and stomach alike and, of course, shows no vital reaction. These appearances suggested a possible explanation of the condition. Post-operative vomiting of very acid fluid is fairly common, and patients often say that it burns their mouths and lips. It was suggested that the lower end of the oesophagus was in an atonic state, so that it failed to empty itself completely in vomiting and some of the highly acid material was retained in its lower third, helped by spasm of the cardiac sphincter. This fluid acted on the devitalized mucous membrane, digesting it and causing a general oozing of blood, which was changed to brown and black by the acid present. The presence of the acid and the oozing instead of a sudden flow from a punctured vessel of some size distinguishes the condition from other oesophageal haematemeses, which are bright red in colour.

**R. D. B. WRIGHT: STRANGULATION OF TRAUMATIC
DIAPHRAGMATIC HERNIA NINE MONTHS AFTER
ORIGINAL ACCIDENT**

Post-mortem appearances. "A" points to tear in diaphragm, with coin in opening.

**ALEXANDER LYALL: ANTE-MORTEM DIGESTION
OF THE OESOPHAGUS**

Section of the oesophagus $\times 100$; (a) Layer of brown blood-stained necrotic material; (b) leucocytic infiltration.

W. O. LODGE: MASTOID RADIOLOGY

FIG. 1.—Normal mastoid region.

FIG. 2.—Bezold's mastoiditis.