Herniation Through the Foramen of Winslow *

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In 1834 Blandin [1] gave the first description of herniation into the lesser sac through the foramen of Winslow. This was discovered as an incidental feature at a postmortem examination. Since then 80 cases have been reported [2-5, 8] The case to be reported is the third which has been treated at The London Hospital. Treves [9] performed the first recorded laparotomy for this condition in 1888.

The first London Hospital patient, a 26-year-old man, had suffered from acute intestinal obstruction for 8 days. At laparotomy the hernia was irreducible and the patient died soon afterwards. Postmortem examination showed that the lesser sac contained the cecum, the entire ascending colon and part of the transverse colon. The lesser omentum had given way at one point so that the appendix lay on the anterior aspect of the lesser curve of the stomach. The second London Hospital case was treated by Russell Howard in 1911 (reported by Lane Roberts [4]). The patient, a 33-year-old man had an acute intestinal obstruction for 24 hours. At laparotomy, 8 inches of strangulated but viable ileum was reduced through the foramen. Unfortunately the patient died 3 days later, apparently as a result of metabolic alkalosis and dehydration.

The present patient presented after 4 days of complete intestinal obstruction. At operation an irreducible hernia containing strangulated ileum, was found. Resection was performed and the patient made a good recovery.

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Case Report

A 34-year-old man was first seen at The London Hospital on April 11, 1963 complaining of severe, constant epigastric pain of sudden onset a few hours previously. While sitting in an armchair shortly after eating a large meal, he made a sudden twist to the left. He then vomited all that he had eaten and there was a progressive increase in the severity of the pain. His bowels had been open normally once since the pain began. There was no history of abdominal pain. On examination there were no signs of dehydration, but slight epigastric tenderness was present. Gut sounds were present and considered to be normal. A diagnosis of gastritis was made, the patient was sent home and advised to return if his symptoms persisted. He returned 4 days later, having suffered continuous epigastric pain, vomiting and complete constipation. On examination he was found to be grossly dehydrated, with generalized abdominal tenderness and distention. Bowel sounds were absent. A diagnosis of high intestinal obstruction due to retroperitoneal herniation was made and confirmed by plain x-rays of the abdomen (Fig. 1). The patient was admitted. A naso-esophageal tube was passed, an intravenous infusion of normal saline begun and the patient was taken to the operating room.

Operation (D. G. A. E.). On entering the peritoneal cavity, enormously distended loops of small intestine were found filling the upper abdomen. The cause of the obstruction was found to be herniation of a loop of proximal ileum into the lesser sac through the foramen of Winslow (Fig. 2). The lesser sac was entered via an incision in the lesser omentum and approximately 18 inches of gangrenous small intestine was discovered. This loop could not be reduced through the foramen of Winslow and so resection with end-to-end anastomosis was performed *in situ*. The anastomosed ileum was then easily passed through the foramen and after suture of the defect in the lesser omentum, the abdomen was closed without drainage. No attempt was made to narrow the foramen of Winslow.

The patient's recovery was complicated by

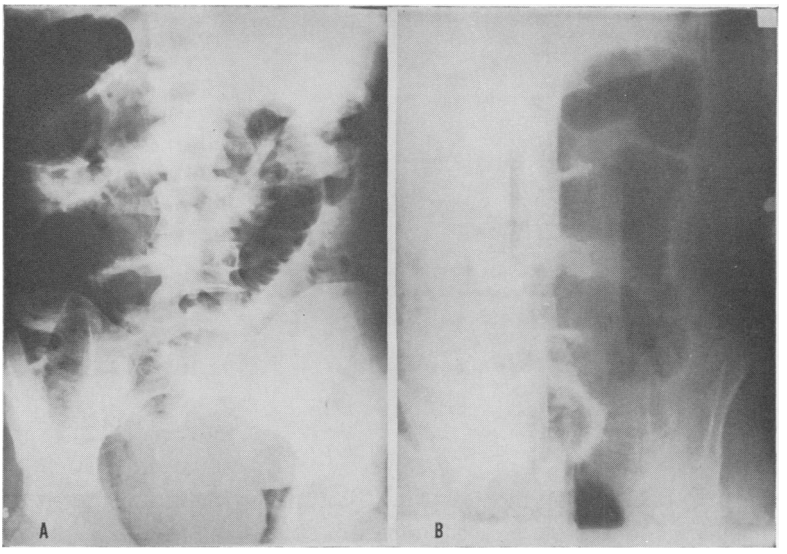

FIG. 1. A. plain x-ray film of abdomen showing distended loops of small bowel. B. Left lateral decubitus, showing distended loops of small bowel with multiple fluid levels.

profound metabolic alkalosis leading to uremia (Fig. 3). The patient was, of course, already alkalotic on admission and due to the continuing high gastric aspirations the large quantities of normal saline which were given intravenously were barely adequate to prevent the situation from deteriorating further. Only when the amount of gastric aspiration diminished was the electrolyte balance restored to normal. After this, progress was normal and the patient was discharged on the 14th postoperative day. Since discharge he has remained in excellent health.

Discussion

Anatomy. The lesser sac of peritoneum is formed as a result of the rotation of the foregut which occurs at about the middle of the third month of embryonic life. In the early embryo the stomach has both a ventral and a dorsal mesogastrium. The ventral mesogastrium contains the liver which is a foregut appendage. When the stomach rotates in the vertical plane through 90 degrees the space which formerly lay to its right now lies posteriorly, closed on the right side by the bulk of the liver and on the left side by the dorsal mesogastrium (Fig. 4). A further rotation of 90 degrees in the horizontal plane places the ventral mesogastrium (lesser omentum) and liver above and the dorsal mesogastrium (greater omentum) below. The resultant space behind the stomach (the lesser sac) is open only on the right side where the ventral mesogastrium ends as a free edge. This is the free edge of the lesser omentum and it forms the anterior border of the foramen of Winslow. In it lie the common bile duct, hepatic artery and portal vein. The posterior border of the foramen is the inferior vena cava covered by peritoneum of the posterior abdominal wall. Above lies the lower surface of the caudate process of the liver. Below, the anterior and posterior

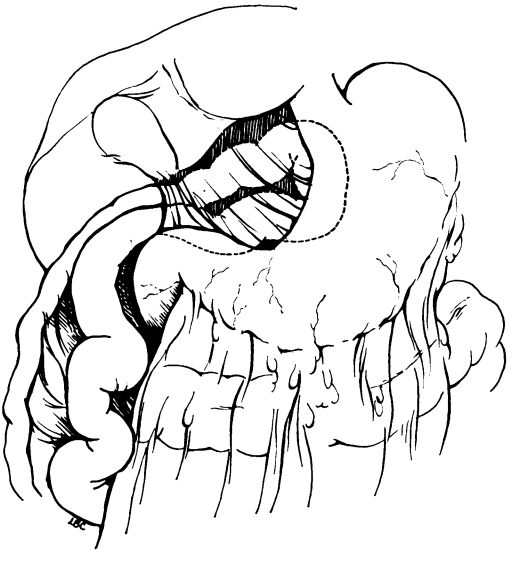

FIG. 2. Diagrammatic representation of findings at operation.

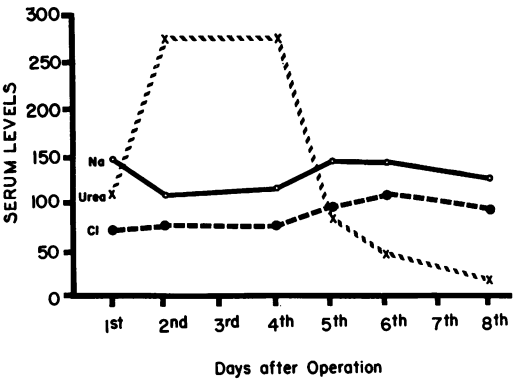

FIG. 3. Serum levels: urea (mg./100 ml.), Cl and Na (mEq./l.).

walls of the foramen meet as the structures in the free edge of the lesser omentum pass behind the first part of the duodenum. In life the anterior and posterior walls are in contact, making a slit approximately 3 cm. long. The foramen was described by Jacobus Benignus Winslow (1669–1760).

Symptomatology. It is obvious that once intestine has passed through a foramen of this size, intestinal obstruction is likely to result. The obstruction may be acute or subacute, though the onset of symptoms is nearly always sudden. The severity of the symptoms will depend on whether or not strangulation is present. A past history of similar, milder attacks is not uncommon, indicating that spontaneous reduction can occur. The most remarkable example of this phenomenon was reported by Neve [7] in 1892. A 17-year-old Indian boy, had suffered from incomplete intestinal obstruction for 2 weeks. A 4-pint enema was given and the patient suspended by the heels and well shaken. This failed to bring relief. At operation the hernia could not be reduced, but spontaneous reduction occurred 2 days later. In a large proportion of cases the onset of symptoms follows soon after eating and coincides with a sudden muscular effort. As will be discussed later, this may be of etiologic significance.

Symptoms are characteristic of upper intestinal obstruction: pain, vomiting and constipation. However due to the high level of the obstruction the patient often has his bowels open once after the onset of the pain. Unless spontaneous reduction occurs, in the typical case, symptoms will persist until surgical relief is obtained or death ensues. Lane Roberts [4] found that the extremes of survival from onset of symptoms to death recorded in the literature were 2½ hours and 17 days. Much less commonly the symptoms are those of subacute obstruction and the patient may survive with symptoms for several months. The usual physical signs are those of upper intestinal obstruction and the only (inconstant) specific physical sign is an indefinite epigastric fullness. Pressure on the structures in the free edge of the lesser omentum does not seem to cause any deficiency in liver function as measured by the standard liver function tests.

It is impossible to make more than a shrewd guess at the diagnosis on the basis of history and physical signs, but radiologic investigations may provide the answer. A firm preoperative diagnosis has been made by performing both barium meal and barium enema x-ray studies, but it is extremely doubtful whether more than erect and supine plain x-ray films are justified in an acutely ill patient. The clinical picture, together with information obtained from x-ray studies, will determine the need for laparotomy and, if an erect lateral x-ray study is added, may even provide the diagnosis by showing distended loops of bowel and fluid levels lying behind the stomach in the lesser sac. In cases of recurrent attacks, where immediate laparotomy is not indicated, more extensive investigations are justified. McKail [5] describes such a case in which results of barium enema x-ray studies were negative but barium meal x-ray film showed that the stomach was displaced forwards by a gas-filled mass. After some delay, barium then entered a distended duodenum and on passing into the small intestine was seen to move upwards into the gas-filled area which was now seen to consist of coils of dilated small intestine.

Etiology. In more than half the recorded cases, the hernia contained only small intestine; in a minority of cases cecum, colon or omentum herniated. This fact requires some explanation since the greater omentum usually forms a barrier between the upper and lower parts of the greater sac of peritoneum, preventing the small intestine from coming into relationship with the foramen. In some cases there seems to have been an anatomic abnormality in the shape of a short mesocolon, atrophic greater omentum or nonrotation of the midgut allowing small intestine access to the upper peritoneal compartment. In other cases the omentum had been adherent to the gallbladder, not only destroying the barrier but acting as a funnel directing small intestine towards the foramen. An etiologic factor of particular importance in females is an unusually mobile cecum and ascending and descending colon. The cecum passes through the foramen first, dragging ascending colon and distal ileum in its wake. This type of hernia is much less common in males. However in many of the recorded cases, particularly in young or middle aged males, no obvious anatomic abnormality was present, and the intestine presumably passed to the right of the greater omentum. Having reached this position some force must impel the intestine through the foramen and into the lesser sac. Much discussion has been devoted to the size of the foramen when herniation occurs but this does not seem relevant—and hardly could be estimated after being stretched for perhaps several days. The lesser sac is normally a potential rather than an actual space, with two layers of serous membrane everywhere in apposition. A force tending to separate these two surfaces would produce a lowering of pressure in the same way that variations of pressure in the pleural space are produced. Normally such changes of pressure in the lesser sac would be of no significance since they would be equalled by changes in the greater sac. If, however, pressure in the lesser sac relative to that in the greater sac was lowered, then

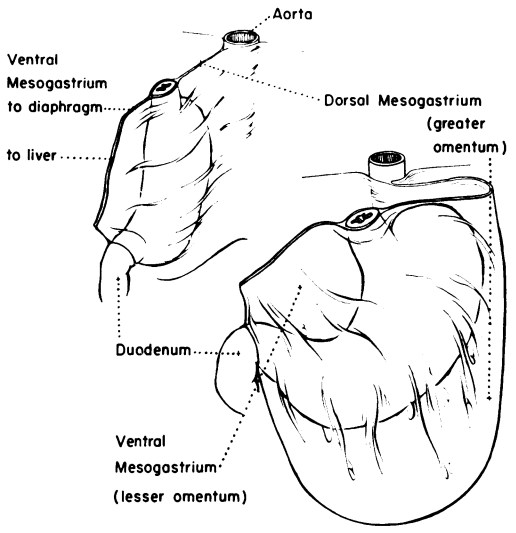

FIG. 4. Rotation of the foregut. After Anatomy, Descriptive and Applied, R. J. Last.

an object placed at the foramen would tend to force the foramen open and enter the sac, propelled by the higher pressure within the greater sac. A force tending to distract the anterior and posterior walls of the lesser sac (the stomach and posterior abdominal wall, respectively) would produce such a pressure drop. The latter is immobile, but the stomach, particularly when full, easily can be imagined as falling forwards when the trunk is suddenly flexed or rotated. We suggest that this is the mechanism which causes the intestine to enter the lesser sac.

Treatment. Treatment is by operation and there should be no delay beyond that necessary to prepare the patient. All that is usually necessary is to reduce the contents of the hernia; resection is rarely required. In only 6 of 55 operations reviewed by Lane Roberts [4] was resection necessary. When strangulation has occurred reduction inevitably will be difficult. When, as in the present case, resection cannot be avoided and reduction is impossible, resection and anastomosis should be performed in the lesser sac, following which the reconstituted intestine can be reduced easily. Occasionally the bowel may be impossible to reduce through the narrow foramen but may appear viable. The hernial orifice then

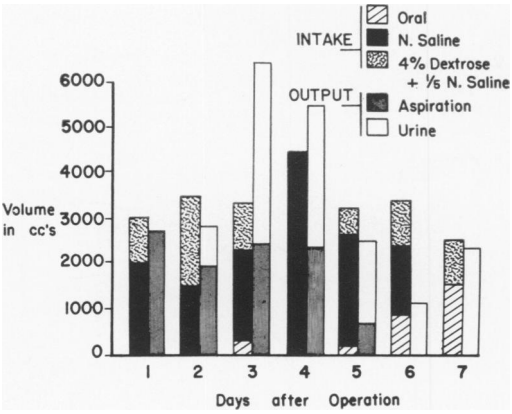

FIGURE 5

may be enlarged by mobilizing the duodenum forwards, after dividing its lateral peritoneal reflection, and then lifting the free edge of the lesser omentum forwards. Though this maneuver has been described often, it never seems to have been indicated. No recurrence has ever been reported after operation, even in those patients where there has been no attempt made to narrow the foramen.

Postoperative management. The problem in management of this condition is almost entirely one of metabolic alkalosis. Continuous vomiting of gastric contents results in dehydration together with a loss of hydrogen and chloride ions and, to a lesser extent, loss of sodium and potassium ions. Uremia follows. This is partly due to dehydration and consequent reduction in extracellular fluid volume, but apparently there is also some renal tubular damage. Despite the low volume of urine, its specific gravity is frequently below 1.020 and proteins and casts may be present.[6] The present case illustrates that, regarding renal deficiency, addition of adequate quantities of fluid is not sufficient. This patient excreted large volumes of urine without a fall in blood urea concentration, and it was not until the large quantities of electrolytes which had been lost in gastric suction and vomitus were replaced that the blood urea fell. Large quantities of 0.9% saline are needed; in the present patient 14 liters were given over a 6-day period. When giving such large quantities of fluid it is essential that frequent estimates of serum and urinary electrolyte concentrations be obtained (Fig. 5).

Summary

The eighty-first recorded case of herniation into the lesser sac through the foramen of Winslow is described.

The anatomy and clinical features of the condition are given. There are two distinct clinical entities: acute intestinal obstruction, with or without strangulation, and subacute obstruction, often recurrent. In the first type early operation is essential and radiologic investigations should be kept to a minimum. In the second type more extensive radiologic investigations may provide the diagnosis preoperatively.

An etiologic mechanism, based on pressure changes within the lesser sac, is proposed.

Acknowledgments

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