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Case Report

Hepatodiaphragmatic interposition of the colon: anatomical findings of a variant of a Chilaiditi sign associated with a sliding hiatus hernia of the stomach

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Abstract

The present study contains a detailed anatomical description of a hepatodiaphragmatic interposition of the colon that corresponds to the signs of Chilaiditi and that has been found during routine dissection in the anatomical dissection course. The right colic flexure (hepatic flexure), the transverse colon and parts of the greater omentum were located in the left subphrenic recess. The hepatodiaphragmal interposition of the colon was associated with a sliding hiatus hernia of the stomach. A large portion of the stomach together with the corresponding section of the gastrocolic ligament was dislocated in the thoracic cavity.

In addition, postulated mechanisms in the etiology of the hepatodiaphragmatic interposition of the intestine were discussed, especially the malrotation and malfixation of the colon as predisposing factors for Chilaiditi signs. Whether there is a mechanical reason between the herniated stomach and the malrotated/malfixed colon remains to be resolved.

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Key words [Chilaiditi sign] [intestine] [malrotation] [sliding hiatus hernia]

Introduction

In 1910, the Viennese radiologist Demetrius Chilaiditi originally described the temporary interposition of the hepatic flexure of the colon between the right liver lobe and the diaphragma [1]. The term Chilaiditi sign indicates the asymptomatic hepatodiaphragmatic interposition of the intestine typically diagnosed as an incidental radiologic finding. In most cases with Chilaiditi signs, the hepatic flexure or transverse colon is interposed anterior to the coronary ligament, between the anterior-superior aspect of the liver and the diaphragm, whereas the interposition of the small intestine, either alone or combined with the colon, occurs less frequently [2, 3]. The Chilaiditi sign has to be distinguished from Chilaiditi syndrome, in which the anomaly produces gastrointestinal symptoms necessitating surgical treatment [4, 5].

Case Report

The Chilaiditi sign was found in an 81-year-old female cadaver during routine dissection in the anatomical dissection course.

The right colic flexure (hepatic flexure), the transverse colon and parts of the greater omentum were located in the left subphrenic recess delimited inferiorly by the left liver lobe, superiorly by the sternocostal part of the diaphragm, laterally by the falciform ligament and posteriorly by the left coronary ligament (Figure 1). The caecum was located directly below the right liver lobe. The ascending colon retroperitoneally crossed the subhepatic space and traversed the inferior margin of the liver to arrive the left subphrenic recess, where it formed the right colic flexure. In the further course, the transverse colon run along the diaphragmatic surface of the left liver lobe to form a hairpin-like sheet with the convexity directed to the falciform ligament and reached the left colic flexure. The mobile transverse colon was attached at the dorsal abdominal wall with the transverse mesocolon and at the greater curvature of the stomach with the gastrocolic ligament, respectively. Exhibiting a normal anatomical position, the left colic flexure was fixed with the left part of the diaphragm by the phrenicocolic ligament.

No varieties in the arterial blood supply of the ascending, transverse and descending colon could be detected.

After removal of the lungs and the heart, we found a large portion of the stomach together with the corresponding section of the gastrocolic ligament dislocated in the thoracic cavity according to a sliding hiatal hernia.

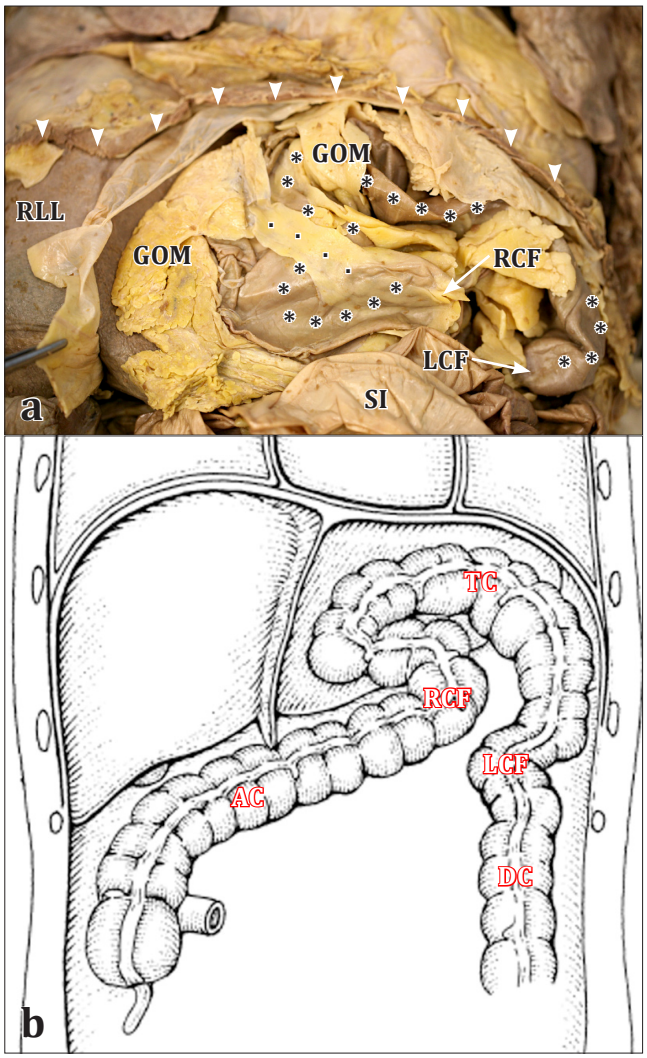

Figure 1. Hepatodiaphragmatic interposition of the colon. **a)** The transverse colon (asterisk line) is interposed between the left liver lobe and the diaphragm. The hairpin-like sheet of the transverse colon is connected by an unusual peritoneal band (dotted line). The falciform ligament is held with a tweezers. **b)** Schematic representation of the in-situ conditions in a. *(**AC**: ascending colon; **TC**: transverse colon; **DC**: descending colon; **GOM**: greater omentum; **LCF**: left colic flexure; **RCF**: right colic flexure; **RLL**: right liver lobe; **SI**: small intestine; **arrowheads**: cut edge of the diaphragm; **asterisk line**: course of the transverse colon; **dotted line**: peritoneal band)*

The blood supply of the stomach normally originates from branches of the coeliac trunk, which forms anastomoses along the curvatures, especially the left and right gastric arteries between the layers of the lesser omentum along the lesser curvature and the left and right gastroomental arteries between the layers of the gastrocolic ligament along the greater curvature. In the present case report, the left gastric artery ramified in a common trunk ("gastric trunk"), which reached the lesser curvature of the stomach, and a hepatic branch, which penetrated the diaphragmatic surface of the left hepatic lobe near the angle between the falciform ligament and the left triangular ligament. The gastric trunk supplied the herniated stomach in the thoracic cavity with a greater epiphrenic branch. A smaller subphrenic arch coursed upwards along the remaining part of the lesser curvature in the abdominal cavity (Figure 2).

Discussion

To the factors that may allow misplacement of a bowel loop into the subphrenic space belong malrotation or abnormal motility of the intestine, reduced liver volume, relaxation of the liver suspensory ligaments and phrenic nerve palsy [6].

The ascending colon remains mobile when the fusion of its dorsal mesentery with the parietal peritoneum of the dorsal

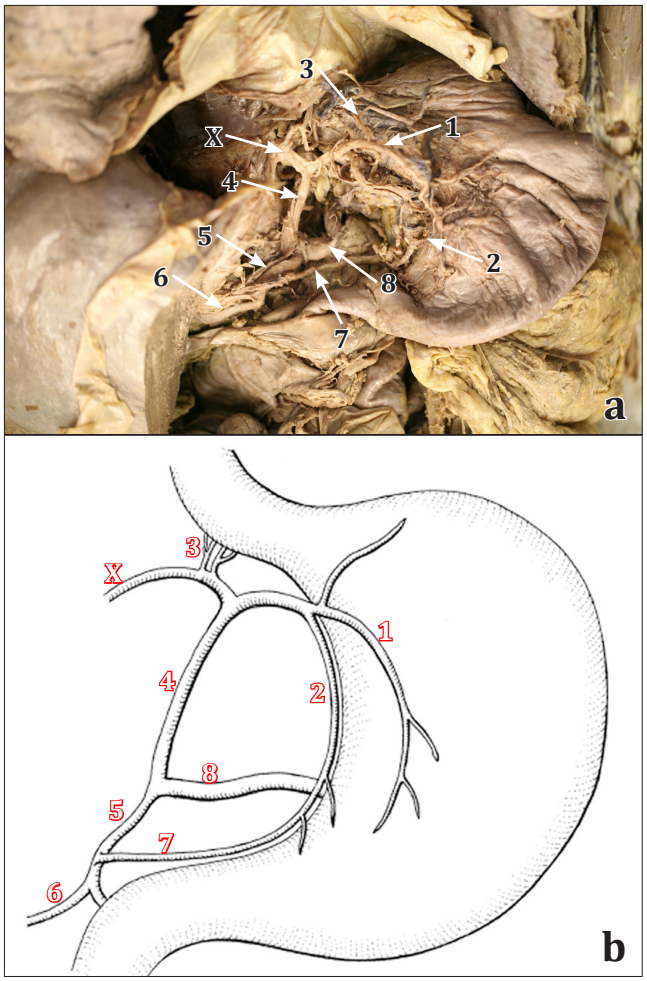

Figure 2. Blood supply of the stomach. **a)** For the preparation of arterial blood vessels, the stomach was completely relocated into the abdominal cavity. The left liver lobe was dissected. **b)** Schematic representation of the in-situ conditions in **a**. *(**1**: epiphrenic branches of the left gastric artery; **2**: subphrenic branch of the left gastric artery; **3**: oesophageal branches; **4**: left gastric artery; **5**: common hepatic artery; **6**: hepatic artery proper; **7**: right gastric artery; **8**: splenic artery; **x**: accessory hepatic artery)*

abdominal wall ceases. During the regular intrauterine development, the midgut is connected to the dorsal mesentery, which continues over the posterior abdominal wall as parietal peritoneum. Due to the rapid growth of the midgut, the intestinal tube forms a sagittally orientated loop with a proximal and a distal segment. Later, the loop rotates in a counter-clockwise manner around its own axis, resulting in a total rotation of 270 degrees. As the colon continues to grow, the caecum descends from the upper quadrant of the right abdominal cavity into the right iliac fossa. The mesenteries of the ascending and descending colon fuse completely with the parietal peritoneum. Several anatomic structures contribute to the fixation of the liver to the anterior and posterior abdominal wall, the diaphragm and other viscera. These include the liver suspensory ligaments, the connective tissue between the bare area of the diaphragmatic surface of the liver and the diaphragm and the suprahepatic veins, through which the liver is connected with the inferior vena cava [7]. The effects of elongated hepatic ligaments on the hepatoptosis were described with Marfan syndrome, when a point mutation of the FBN1 gene is responsible for the defective synthesis of the glycoprotein fibrillin–1 as a major component of the microfibrils in elastic fibers [8].

The term "malrotation" describes developmental abnormalities of the midgut that are associated with abnormal rotation and/or fixation. The non-fixation and a common mesentery result in an increased intestinal mobility. Malrotation may occur as an isolated entity or in association with abdominal wall defects and diaphragmatic hernia [9]. Depletion of elastic fibers in ligaments supporting the gastroesophageal junction provides evidence for a structural basis of hiatus hernias [10].

To our knowledge, this is the first report of a hepatodiaphragmatic interposition of the colon combined with a sliding hiatus hernia. The herniated stomach has probably drawn the colon between the left liver lobe and the diaphragm by means of the gastrocolic ligament, which connects the greater curvature of the stomach with the transverse colon. However, whether the mechanical tension by the dislocated stomach is causative for the intestinal malrotation/malfixation remains under discussion.

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