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CHAGAS' DISEASE ACHALASIA - LAPAROSCOPIC PRIMARY REPAIR OF ESOPHAGEAL PERFORATION AFTER LAPAROSCOPIC HELLER'S MYOTOMY

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Background

Chagas disease, caused by the parasite Trypanosoma cruzi, is a major cause of secondary esophageal achalasia in endemic areas of Central and South America. It has clinical consequences in the heart and digestive tract. The most important changes in the digestive tract occur in the esophagus and colon. Chagas esophageal disease, also known as megaesophagus, is a severe manifestation of Chagas disease. The parasite is primarily transmitted through the feces of triatomine bugs, commonly known as "kissing bugs," which bite humans and deposit the parasite near the bite wound. Esophageal achalasia results from the destruction of the esophageal intramural nerve plexus, leading to impaired lower esophageal sphincter (LES) relaxation and esophageal aperistalsis. As a result, patients experience severe dysphagia, regurgitation of food, chest pain, and weight loss. The esophagus becomes dilated and elongated, forming what is known as a megaesophagus. Diagnosis of Chagas esophageal disease involves a combination of clinical evaluation, serological tests to detect antibodies against Trypanosoma cruzi, and imaging studies such as barium swallow radiographs and esophageal manometry. Treatment options for Chagas esophageal disease are primarily aimed at relieving symptoms and improving the patient's quality of life. Pharmacological treatments include the use of nitrates and calcium channel blockers to relax the LES. Surgical treatment options for achalasia, including laparoscopic Heller's myotomy (LHM) and pneumatic dilation (PD), are commonly used. LHM involves cutting the muscle fibers at the LES to facilitate food passage and is often combined with fundoplication to reduce the risk of post-procedural gastroesophageal reflux disease (GERD). This surgical technique is considered the gold standard. While both PD and LHM are effective in the long- term control of achalasia symptoms, LHM has been associated with a significantly lower risk of esophageal perforation. Pneumatic dilation, which involves inserting and inflating a balloon at the LES, carries a higher risk of perforation, reported in up to 5.1% of cases compared to 0.9% for LHM. While both LHM and PD effectively alleviate achalasia symptoms, GERD remains a notable post-surgical complication. LHM with fundoplication reduces this risk significantly, with post-procedural GERD rates ranging from 9% to 23%, depending on the type of fundoplication used. In contrast, PD, though effective, has a slightly higher risk of GERD, with up to 31.5% of patients developing reflux in the absence of fundoplication. More recently, peroral endoscopic myotomy (POEM) has emerged as a minimally invasive option, particularly beneficial for patients with type III achalasia. POEM offers success rates over 90% in the short term, but GERD remains a frequent complication due to the lack of anti-reflux measures.

Aim

Reporting a case of a patient with chronic chagasic achalasia, which was complicated by esophageal perforation after Heller's myotomy and was successfully managed by laparoscopic esophageal suturing.

Methods

A 59-year-old patient from Chile was admitted with progressive dysphagia, vomiting and megaesophagus. The endoscopy showed a dilated esophagus with stenosis of the distal third and retained food. A barium swallow study and high-resolution esophageal manometry confirmed esophageal dilatation and achalasia. The patient underwent an elective laparoscopic Heller myotomy with Dor fundoplication. On postoperative day 2, he developed severe intermittent chest and abdominal pain and high drain fluid amylase concentration (49.000 U/L). A CT scan demonstrated small pneumomediastinum, small bilateral pleural effusion, and suspicion of oral contrast leakage. The patient underwent an urgent diagnostic laparoscopy that revealed a 4cm esophageal longitudinal perforation at the posterolateral wall, after the Dor fundoplication reversal. A 36-Fr orogastric tube was inserted across the GE Junction into the stomach and primary repair was performed with interrupted 3–0 Vicryl-plus stitches. Then, an omental patch was sutured over the perforation area. A feeding jejunostomy was inserted for postoperative nutritional support, and a nasogastric tube was inserted to the stomach. Drains were placed intrabdominal and posterior mediastinal. The patient was discharged home in good condition 12 days post op.

Conclusions

Although surgical treatments like LHM, PD, and POEM effectively manage esophageal achalasia in Chagas disease, the choice of procedure must weigh the risk of complications like esophageal perforation and GERD. Laparoscopic Heller myotomy and fundoplication is considered as treatment of choice for idiopathic and chagasic achalasia. Laparoscopic intervention with primary repair of perforated esophagus may be challenging, but it can be a good and life-saving choice instead of esophagectomy.

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