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Case Report

Pyloric hypertrophy in adults: case report

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ABSTRACT

Pyloric hypertrophy is a benign condition characterized by narrowing of the pyloric canal, which may be associated with other pathologies. It is believed to manifest as a mild form of hypertrophy from childhood into adulthood. Symptoms can be nonspecific, such as nausea, vomiting, oral intolerance, and weight loss. Diagnosis can be challenging for physicians, and accurate reporting of cases is difficult due to some patients remaining asymptomatic.

Keywords: Hypertrophy, Pylorus, Adults, Stenosis

INTRODUCTION

Pyloric hypertrophy is relatively rare, primarily involving hypertrophy of the muscular layer, particularly the circular muscle.[1] The incidence of congenital hypertrophy is estimated at 0.25% to 0.5% of live births, with approximately 300 to 400 cases reported in the literature.[2] Pyloric hypertrophy in adults was first described in 1835 by Jean Cruveilheir, and its pathological anatomy was detailed by Maier et al.[3,4] The condition narrows and elongates the pyloric canal, affecting primarily the pyloric sphincter muscle. Although benign, its diagnosis can be challenging, and radiologically it may mimic carcinoma.[5] Adult patients typically do not have a history of childhood vomiting or gastrointestinal symptoms, but current symptoms may include vomiting, weight loss, and abdominal distension.[6]

CASE REPORT

A 67-year-old female patient with a 10-year history of hypertension treated with nifedipine 30 mg every 12 hours and losartan 50 mg every 24 hours. She underwent laparoscopic cholecystectomy in January 2024. Obstetric history includes three pregnancies, one miscarriage, and two deliveries. Eight months prior to admission, she began experiencing epigastric pain radiating to the posterior chest, exacerbated by food and unresponsive to medical treatment. Upon consultation, she was diagnosed with cholelithiasis, and surgical treatment was decided. Laparoscopic cholecystectomy was performed without complications. Post-surgery, she experienced approximately 10 kg weight loss within 1 month, along with nausea, vomiting, and postprandial fullness. She developed oral intolerance with worsening signs and symptoms, leading to hospitalization under the care of the general surgery service.

Conservative management was initiated. Abdominal tomography was performed (Figure 1), revealing significant gastric dilation, prompting an endoscopy (Figure 2), which showed a retained stomach without identification of the duodenal lumen. Biopsies were taken and pathology reported moderate-grade superficial chronic gastritis, negative for intestinal metaplasia or malignant activity. Further investigation included magnetic resonance imaging (Figure 3), demonstrating preserved duodenal lumen with increased pyloric dimensions. Surgical treatment involved Roux-en-Y gastrojejunostomy, performed without complications. Follow-up esophagogastroduodenal series (Figure 4)

showed contrast passage without anastomotic leakage. A liquid diet was started on postoperative day 2, progressing to a soft diet by day 5, with the patient tolerating it well and exhibiting no additional signs or symptoms. Consequently, she was discharged home.

At outpatient follow-up, the patient tolerated oral intake well, without nausea, vomiting, or other complaints.

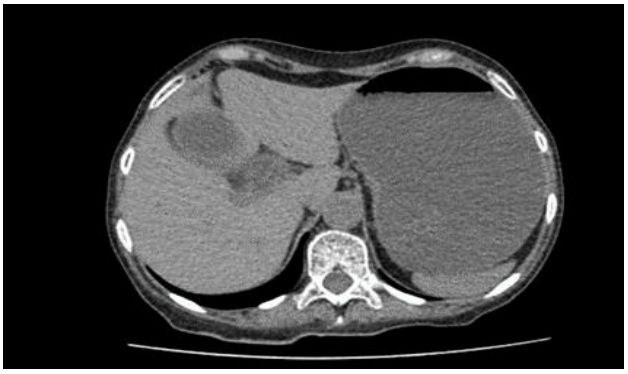

Figure 1: Abdominal CT scan shows stomach dilation without thickening of the wall.

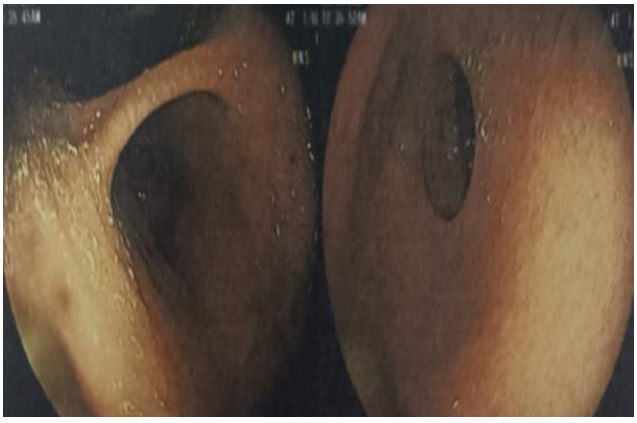

Figure 2: Endoscopy reveals a stomach with retention and duodenal stenosis.

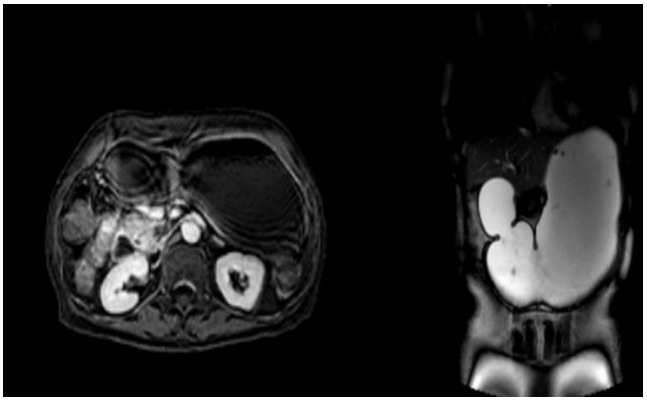

Figure 3: Magnetic resonance imaging shows pyloric widening resulting in decreased amplitude of the pyloric channel, with significant distension of the first and second portions of the duodenum.

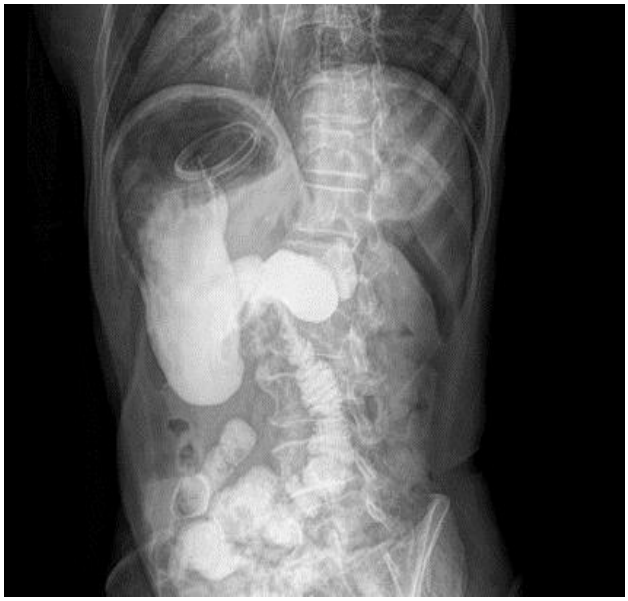

Figure 4: Esophagogastroduodenal study shows no contrast leakage, with contrast passage from stomach to jejunum.

DISCUSSION

In this case illustrated, the patient presents signs and symptoms of upper gastrointestinal obstruction, in addition to rapid weight loss, with a tumor located at the pylorus and first portion of the duodenum, leading to imaging studies and a diagnosis of pyloric hypertrophy.[7] The frequency of pyloric hypertrophy can vary, ranging from 0.5% to 1%. It occurs more frequently in male patients with a ratio of 3:1 and typically affects individuals aged between 30 and 60 years, with the peak incidence around 40 years.[1]

Pyloric hypertrophy is commonly diagnosed in infants but is extremely rare in adults.[7] Clinical symptoms in adults manifest due to gastric obstruction and include epigastric pain, postprandial fullness, nausea, vomiting, and weight loss.[6,7] Literature reports symptoms can occur anywhere from 1 to 420 months; in our patient, symptoms persisted for approximately 5 months. A significant number of cases are asymptomatic and discovered incidentally during post-mortem examinations.[6]

It can be classified into primary or secondary. In cases of secondary pyloric hypertrophy, the most common causes are gastric or duodenal ulcers, carcinomas, gastrointestinal stromal tumors, or vagal hyperactivity. In cases without a predisposing factor, it is classified as primary or idiopathic, as in the case of our patient. It is believed that mild infantile pyloric hypertrophy may persist into adulthood.[8]

The pylorus is composed of a thicker layer of the inner circular muscle, and the pyloric channel measures approximately 0.8 to 1 cm in length with a thickness of 3-

8 mm. In pyloric hypertrophy, the muscle can measure 1 cm or more in thickness and 2 cm in length. It has been reported that these changes are due to inflammatory processes, as previously mentioned, or edema, with degenerative changes of the ganglion cells in the myenteric plexus.[9]

Diagnosing through imaging is challenging because some studies may not show alterations or pathognomonic signs. Kirklin's sign, or the mushroom sign, is described as the protrusion of the pyloric muscle into the duodenum in upper gastrointestinal series and can be found in 50% of pediatric patients. On computed tomography, gastric distension, as seen in our patient, or thickening of the wall may be observed.[4]

Pyloroplasty using the Finney method or Heineke-Mikulicz pyloroplasty has technical disadvantages in patients with very thick muscle.[10] The first laparoscopic pyloromyotomy was performed in children for pyloric stenosis in 1990. It has been shown that the laparoscopic approach is safe, effective, and offers better aesthetics, with earlier postoperative recovery and shorter hospital stays.

Gastric resection may be unnecessary in a benign process; biopsies were taken during endoscopy in our patient, and no malignant process was found.[3] The treatment that can be employed is gastric resection and Billroth I anastomosis. In our case, gastric resection was not performed at the patient's request; risks and benefits of both procedures were explained, hence Roux-en-Y gastrojejunostomy, was performed.[2] Endoscopic treatment through pyloric dilations is also mentioned but with a high recurrence rate and is only used in selected cases such as patients at high surgical risk or those who do not wish to undergo surgery as treatment.[9,10]

CONCLUSION

Pyloric hypertrophy is a rare condition and challenging to diagnose due to its nonspecific signs and symptoms, requiring a high diagnostic suspicion. The pathophysiology of the disease is not well understood, although there are several hypotheses. Treatment can be challenging, but surgical intervention has been shown in the literature to be the best option.

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