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Case Report

Gastric outlet obstruction secondary to adult hypertrophic pyloric stenosis: case report and literature review

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ABSTRACT

Adult hypertrophic pyloric stenosis is a rare entity; the most common type is secondary to underlying gastrointestinal pathologies, such as peptic ulcer, malignant tumors, and certain inflammatory diseases. A rare case of hypertrophic pyloric stenosis is presented in an adult patient secondary to acid peptic disease, presenting with gastric outlet obstruction, treated in the first instance with pyloric dilation successfully, however, the clinical picture has recurred now with 90% hypertrophic pyloric stenosis, it was decided to perform a surgical procedure with roux-en-Y gastrojejunal diversion, progressing with adequate evolution. There is no consensus on which is the most effective treatment. However, most authors seem to favor gastric resection due to the risk of malignancy secondary to long-standing hypertrophic pyloric stenosis.

Keywords: Adult hypertrophic pyloric stenosis, Gastric outlet obstruction

INTRODUCTION

Adult hypertrophic pyloric stenosis (AHPS) is a rare entity and presents as a pyloric obstruction secondary to hypertrophy of the circular fibers of the pyloric canal.[1]

There are various classifications, however the most common type of AHPS is secondary to underlying gastrointestinal pathologies, such as peptic ulcer, malignant tumors, and certain inflammatory diseases.[2]

Chronic peptic ulcer resulting in gastric outlet obstruction is the least common complication, occurring in approximately 2 percent of cases.[3]

Patients with secondary AHPS often do not present any symptoms.[4]

Diagnosing hypertrophic stenosis in adults remains a challenge; differentiation between primary and secondary pyloric stenosis is made by histopathological report. The main treatment is surgical, although endoscopic dilation can also be performed. However, there is still no consensus on what the best surgical approach is.[4]

A rare case of hypertrophic pyloric stenosis is presented in an adult patient secondary to acid peptic disease, which presents with gastric outlet obstruction, successfully treated surgically. This article discusses the various etiologies of AHPS, diagnostic methods, and possible surgical treatments.

CASE REPORT

A 51-year-old male patient, who was admitted from the emergency department with a history of systemic arterial hypertension of 18 years of diagnosis under treatment with Losartan 50 mg every 12 hours, type 2 diabetes of 10 years of diagnosis under treatment with Metformin 850 mg every 24 hours, appendectomy 18 years ago without complications and left JJ catheter placement a month ago without complications.

The patient began symptoms 24 hours prior to his admission, reporting pain in the epigastrium, intensity 7/10 according to the visual analogue scale (VAS), oppressive type, accompanied by heartburn and vomiting on 5 occasions of gastro-alimentary content, so he was admitted to the service of emergencies. His vital signs are within normal parameters. A physical examination was carried out, finding a globose abdomen at the expense of the adipose panniculus, peristalsis present with normoactivity, soft, depressible, painful on deep palpation in the epigastrium, dull on percussion, negative rebound, with no evidence of peritoneal irritation. The rest of the examination without alterations. Admission laboratories within normal parameters.

An upper gastrointestinal endoscopy (UGI) was performed, finding chronic erosive gastropathy and a stenosing Forrest III prepyloric gastric ulcer (Figure 1).

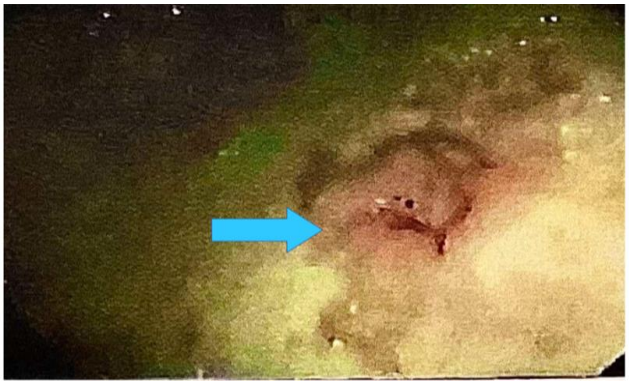

Figure 1: UGI showing chronic erosive gastropathy, stenosing Forrest III prepyloric gastric ulcer (arrow).

Manometry was requested, which reported an upper esophageal sphincter with normal tone and incomplete relaxation, effective esophageal motility, a lower esophageal sphincter with normal basal pressure, and normal relaxation of the gastroesophageal junction.

Gastric biopsies were taken, with the following histopathological report: chronic gastropathy with activity and mild atrophy, scarce bacillary flora (+/+++) compatible with *H. pylori*, active ulcer, negative for metaplasia and/or dysplasia.

Medical treatment is given; however, the patient persists with symptoms presenting dysphagia to solids, a new endoscopy and pyloric dilation with balloon is scheduled, first pyloric dilation at 200 atmospheres for 1 minute and second pyloric dilation at 350 atmospheres for 1:30 minutes corroborating adequate permeability of the pylorus (Figure 2).

The patient is discharged after presenting adequate progress; however, three weeks later the patient goes to the emergency room after presenting vomiting of gastro-alimentary content on more than 10 occasions, dysphagia to solids and liquids as well as hydro electrolyte imbalance with mild hyponatremia (130 mmol/l), moderate hypokalemia (2.72 mmol/l) and mild hypochloremia (90 mmol/l).

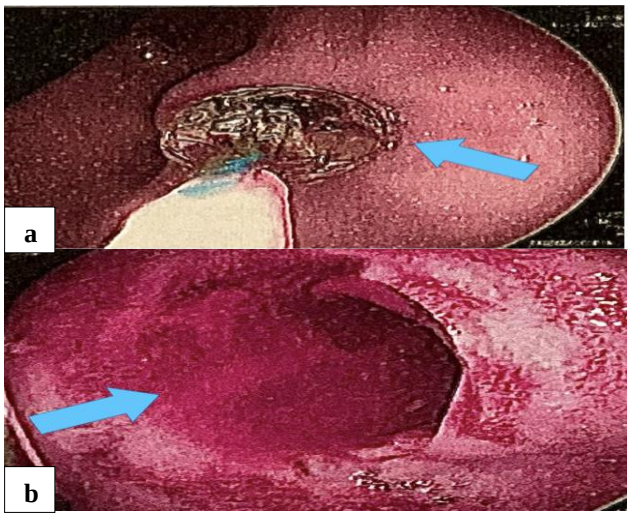

Figure 2: (a) Pyloric dilation with a balloon is shown, and (b) corroborating the adequate permeability of the pylorus.

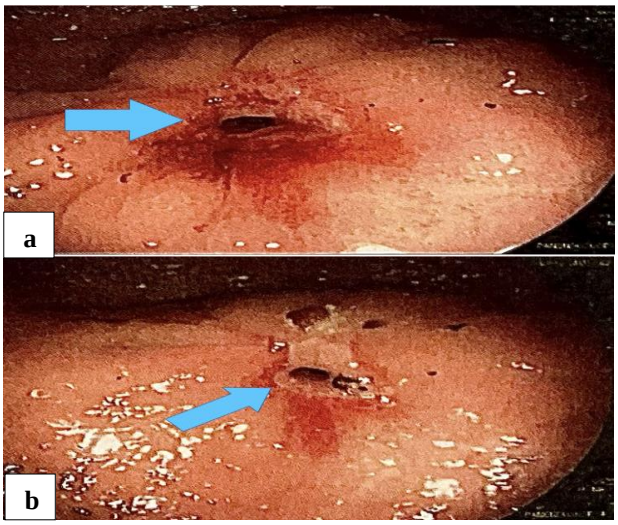

Figure 3: (a) and (b) UGI 90% pyloric stenosis is observed, indicated by the arrows.

A new upper gastrointestinal endoscopy (UGI) was performed, finding 90% pyloric stenosis (Figure 3), without being able to visualize the distal portion of the

stenosis; since its permeability was unknown, it was scheduled to perform derivative surgery.

Laparoscopic roux-en-Y gastrojejunal diversion was performed with anastomosis 75 cm from the angle of Treitz, without complications.

Surveillance continued in the outpatient clinic, the patient presented an adequate post-surgical evolution, without pain, and was satisfied with the surgical medical treatment provided.

DISCUSSION

Approximately 300 to 400 cases of AHSP have been reported in the literature.[5]

The first description of this entity was in 1842 by Curveilhier and later confirmed by Maier in 1885.[5,6]

The AHPS has many classifications by various authors. The most universally accepted classifications are those of Danikas et al, and Zarineh et al classified AHPS into three types: type 1, which is childhood AHPS diagnosed at a late stage; type 2, which is the most common type, which occurs during adult life and is probably secondary to underlying gastrointestinal pathologies, such as peptic ulcer, malignant tumors and certain inflammatory diseases; and type 3, which is idiopathic adult-onset AHPS.[9,10]

Zarineh et al divided AHPS into primary, which has no underlying cause and under which idiopathic AHPS would be classified, and secondary, caused by an underlying disorder such as excessive healing of gastric or duodenal ulcers, malignant tumors, GISTs, postoperative intra-abdominal adhesions, bezoars, and increased vagal activity, causing hypertrophy of the pylorus.[2] The secondary type of AHPS usually demonstrates predominantly localized replacement by fibrous tissue, with little or no smooth muscle hypertrophy.[5]

Secondary AHPS often does not present any symptoms. The clinical symptoms of idiopathic AHPS are like the clinical symptoms of gastric outlet obstruction induced by other causes including epigastralgia, early satiety, and postprandial nausea or vomiting.[1,4-6,10] The exact etiology of AHPS is unclear, and it is usually difficult to diagnose AHPS before surgical resection.

The differential diagnosis includes malignancy and diabetic gastroparesis, both of which may present similarly to idiopathic AHPS.[2] Although recognizing carcinoma is usually straightforward, spindle cell neoplasms, such as gastrointestinal stromal tumor, may be more difficult to differentiate from idiopathic AHPS. Diabetic gastropathy shows the characteristic hydropic neural degeneration with a severe reduction in the density of unmyelinated axons, vasculopathy with thickening of the vessel walls and smooth muscle degeneration and fibrosis, with eosinophilic inclusion bodies (M bodies) that appear be exclusive to this condition.[5]

In patients with AHPS, gastrography usually shows an elongated and narrow pyloric canal and delayed gastric emptying due to pyloric stenosis.[1] While abdominal tomography often shows thickening of the distal gastric wall.[2,6] Endoscopic ultrasound is diagnostic of AHPS when the pyloric muscle wall is greater than 10 mm, the wall of a normal pyloric muscle varies from 3.8 to 8 mm.[1,2] Measurements of up to 3 cm have been recorded in AHPS.[6]

In AHPS gastroscopy secondary gastric ulcers, the mucosa of the gastric sinus or pyloric hilum shows obvious ulcerative erosion, mucosal thickening around the ulcerated surface, loss of the pyloric duct, and local ulceration.[4] The classic finding on gastroscopy is a fixed, markedly narrowed pylorus with a smooth edge.[7]

The histopathological result of idiopathic AHPS shows a very elongated and thickened pylorus. On microscopy, there is marked hypertrophy and hyperplasia of the gastric muscularis propria without marked inflammatory cells or malignancy.[2,6]

The histopathological result of secondary AHPS gastric ulcers shows persistent infiltration of inflammatory cells and hyperplasia of scar tissue in the pyloric mucosa and submucosa.[4]

Most authors agree that histological confirmation of the disease is recommended to safely rule out malignancy.[7]

Asymptomatic patients or patients with minimal symptoms do not require treatment while patients with clinical evidence require surgical treatment.[10]

Multiple treatments have been proposed for AHPS, including endoscopic dilation, pyloromyotomy with or without pyloroplasty, and gastrectomy with Billroth I gastroduodenostomy.[2,4-6]

Among the various surgical techniques, pylorectomy with minimal gastrectomy is the one that seems to obtain the best results.[5,10] Most authors favor a limited distal gastric resection with Billroth I or II anastomosis. This technique is especially recommended if the pylorus wall is very thick, making pyloroplasty technically difficult.[5,7]

Laparoscopic pyloroplasty is a less invasive option. Endoscopic dilation has a high recurrence rate and only provides temporary relief of symptoms. It is an option in high-risk surgical patients or those who reject surgery.[2,4,6,10] Regarding endoscopic dilation, surgery is indicated if the pylorus is obstructed and cannot be safely dilated, or if the obstruction persists or recurs despite endoscopic dilations to achieve a diameter of 15 mm, symptoms usually improve considerably with successful dilation of 12 to 15 mm.[3] In the case presenting with the recurrence of AHPS and the impossibility of dilating it

safely, it is decided to perform a derivative surgical treatment.

Some authors prefer partial gastrectomy with Billroth I reconstruction because carcinoma can be a complication of long-standing pyloric hypertrophy.[5,6] Approximately 25 percent of gastric cancer patients have a history of gastric ulcer related to Helicobacter pylori infection.[4] Which justifies the aforementioned procedure.

Currently, there is no evidence that one surgical technique is superior to another. Further research on AHPS is warranted before a method can be finalized as a standard of care.[2]

CONCLUSION

A rare case is presented, whose diagnosis can only be established after the exclusion of more common causes of gastric outlet obstruction. Successful pyloric dilation was performed in the first instance, however, as stated in the literature, the procedure has a high recurrence rate, which occurred. The histopathological result is what confirms the diagnosis, in this case it was negative for metaplasia and dysplasia, confirming the etiology of AHPS secondary to acid-peptic disease, gastric obstruction is a rare complication of this entity. There is no consensus on which is the most effective treatment. However, most authors seem to favor gastric resection due to the risk of malignancy secondary to long-standing AHPS.

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REFERENCES

1. Hattori A, Kawabata H, Umeda Y, Tsuboi J, Yamada R, Hamada Y, et al. Adult hypertrophic pyloric stenosis that improved by spontaneous double channel pylorus formation. JGH Open. 2020;5(2):317-9.
2. Hassan SM, Mubarik A, Muddassir S, Haq F. Adult idiopathic hypertrophic pyloric stenosis - a common presentation with an uncommon diagnosis. J Community Hosp Intern Med Perspect. 2018;8(2):64-7.
3. Zare E, Raeisi H, Honarvar B, B. Lankarani K. Long-term results of endoscopic balloon dilation for gastric outlet obstruction caused by peptic ulcer. Middle East J Dig Dis. 2019;11:219-25.
4. Zhou J, Liu G, Song X, Liu H, Wang D, Kang Q. Pyloric stenosis secondary to peptic ulcer disease in pediatric patients: A case report and review of the literature. Medicine (Baltimore). 2023;102(12):e33404.
5. Bayramoğlu Z, Başsorgun İC, Ünal B, Akın M, Elpek GÖ. Focal pyloric hypertrophy in adults: a diagnostic pitfall-a case report and review of the literature. Clin J Gastroenterol. 2020;13(1):60-5.
6. Lin HP, Lin YC, Kuo CY. Adult idiopathic hypertrophic pyloric stenosis. J Formos Med Assoc. 2015;114(7):659-62.
7. Hellan M, Lee T, Lerner T. Diagnosis and therapy of primary hypertrophic pyloric stenosis in adults: case report and review of literature. J Gastrointest Surg. 2006;10(2):265-9.
8. Danikas D, Geis WP, Ginalis EM, Gorcey SA, Stratoulias C. Laparoscopic pyloroplasty in idiopathic hypertrophic pyloric stenosis in an adult. JSLS. 2000;4(2):173-5.
9. Craver WL. Hypertrophic pyloric stenosis in adults. Gastroenterology. 1957;33(6):914-24.
10. Fernández Laso AB, Ganchegui Aguirre I, Campos Ruiz A, Urtasun Arlegui L, Gorroño Zamalloa I, Martinez Blazquez C, et al. Estenosis hipertrófica pilórica del adulto (Hypertrophic pyloric stenosis in adults). Gastroenterol Hepatol. 2016;39(9):643-5.

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