

Case Report

Adult hypertrophic pyloric stenosis due to peptic ulcer disease: a rare presentation

Shameer Deen^{1*}, Vipin Kumar Bakshi², Siddharth Jai Singh¹, Satish K. Bohara¹, S. C. Dutt¹

¹Department of General Surgery, Mahatma Gandhi Medical College and Hospital, Jaipur, Rajasthan, India

²Department of Radio-diagnosis, Mahatma Gandhi Medical College and Hospital, Jaipur, Rajasthan, India

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*Correspondence:

Dr.Shameer Deen,

E-mail: shameerdeen@gmail.com

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ABSTRACT

Primary adult hypertrophic stenosis is uncommon with an uncertain etiopathogenesis and associated gastric outlet obstruction mimics gastric carcinoma. We present a case of AHPS as sequel of peptic ulcer disease in a 72 year old male. With the advent of proton pump inhibitors as a mainstay of medical therapy, complication into gastric outlet obstruction is a rare disease today. Upper GI endoscopy revealed a distended stomach, residual food and a hyperemic bulky pylorus not accommodating the endoscope. Barium meal follow-through revealed a dilated stomach and minimal barium passing through the pylorus. Histological analysis revealed mild dysplasia at the focus with dense inflammatory infiltrates composed of lymphocytes and eosinophils in the lamina propria. No evidence of malignancy was noted, favouring chronic gastritis. The condition mimics other forms of proliferative disorders like carcinoma, gastrointestinal stromal tumors. We present the clinical findings, imaging analysis and discuss etiopathogenesis and management.

Keywords: Adult hypertrophic pyloric stenosis, Peptic ulcer, Gastric outlet obstruction

INTRODUCTION

With the advent of proton pump inhibitors and H-2 receptor antagonists adult hypertrophic pyloric stenosis (AHPS) as a primary disorder and a sequel of peptic ulcer disease is a rare entity today. Although CHPS is well explained, primary AHPS is rare and not described in recent literatures. AHPS is an uncommon entity with approximately 200 cases reported.¹ In CHPS, hypertrophy involves the entire circumference of the pylorus without associated gastro-intestinal pathology, whereas in the adult form only a localized segment of the pylorus is involved associated with peptic ulceration, gastritis and polyposis.² 80% of the cases reported have occurred in males with an extremely wide age of presentation.^{3,4}

Skoryna et al classified pyloric hypertrophy into primary and secondary forms.⁵ Primary forms included focal forms, diffuse forms with proximal lesion and diffuse forms without a proximal lesion whereas secondary hypertrophy was associated with a distal obstructive lesion. We present a diffuse form without a proximal lesion of AHPS in a 72 year old male with history of chronic peptic ulcer disease.

CASE REPORT

A 72 year old male had complaints of abdominal fullness after meals since 5 months, vomiting after meals since 4 months. Distension occurred after taking meals and relieved with vomiting; it was associated with anorexia, not associated with pain and no significant weight loss. Vomitus consisted of undigested food, non-bilious.

Patient had history of peptic ulcer disease since 10 years. Patient kept no pets.

On physical examination, the patient was lean and thin with no pallor, oedema, cyanosis or clubbing. Abdominal inspection revealed fullness in the upper abdomen, visible left to right peristalsis. Palpation revealed a swelling in the epigastrium at the duodenal line. Succussion splash was present and ausculto-precussion test was positive below umbilicus.

Erect abdominal X-ray abdomen and barium meal follow through (Figure 1) revealed a massively dilated stomach, absent duodenal cap and no external compression. A CT revealed gastric dilatation, circumferential thickening of pylorus (4 cms), no loss of fat planes. Upper GI endoscopy (Figure 2) revealed retained food, a hyperemic, bulky and oedematous pylorus and unable to negotiate scope beyond the pylorus. Multiple biopsies were taken from pylorus and surrounding mucosa. Gross analysis revealed grey white soft tissue. Microscopically, gastric mucosa had mild dysplasia, no evidence of fibrosis and malignancy, favouring chronic gastritis.

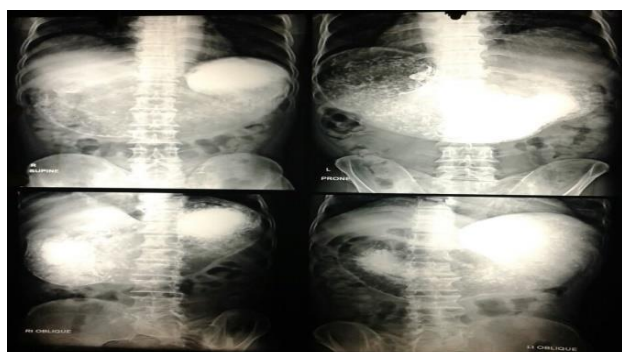


Figure 1: Erect abdominal X-ray abdomen and barium meal follow through.

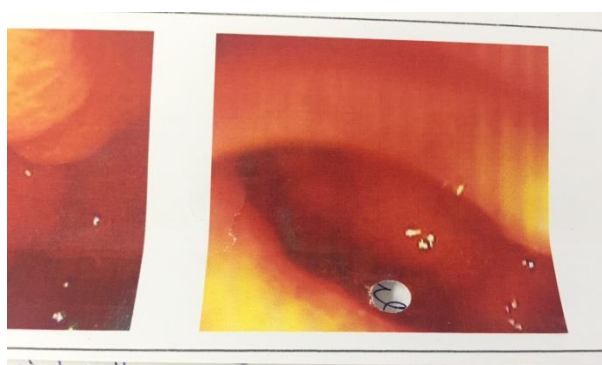


Figure 2: Upper GI endoscopy.

Laboratory analysis revealed a haemoglobin level of 12.50 g/dl, total leukocyte count of 8.20 ($10^3/\mu\text{l}$). His blood sugar levels were 75 mg%, liver and renal function tests were well within normal range and an electrolyte analysis revealed hypokalemia (3.40 mmol/L).

The patient was managed conservatively with IV fluid administration; a nasogastric aspiration was performed and approximately 2.5 liters of residual food was evacuated. On the basis of clinical and radiological analysis we derived the diagnosis of gastric outlet obstruction due to pyloric stenosis. Surgical exploration was considered as the patient did not improve with medical management.

A upper midline incision was used, stomach was greatly dilated, no evidence malignancy. A soft mass was palpable in the pylorus. Bilroth I was performed and subpyloric lymph nodes were biopsied. Post-operative course was uneventful. Patient tolerated post-operative feeding regimens well and barium meal follow-through revealed a homogeneous distribution in the stomach and passed without obstruction. Post-operative histological analysis revealed chronic inflammatory cells multiple sections showed the features of chronic gastritis. Proliferation marker Ki-67 was negative. Follow-up after 6 months revealed good weight gain and no complaints of vomiting, nausea or dyspepsia.

DISCUSSION

AHPS with gastric outlet obstruction as a sequel of peptic ulcer disease is a rare. Few cases been reported in literature and adult forms have occurred secondary to local diseases and less commonly idiopathic causes.⁷ persistence of mild manifestation of juvenile form into adulthood have been proposed but not proved.⁸

Classification by Skoryna et al suggests primary forms of the disease have no underlying disorder and secondary forms demonstrate localized replacement by fibrous tissue with little or no smooth muscle hypertrophy and associated with healing peptic ulcer disease, carcinoma, gastro intestinal stromal tumors, post-operative adhesions, bezoars and muscular hypertrophy due to vagal hyperactivity.^{5,9} One reported cases was due to crohns' disease and one due to mucosal diaphragm.^{10,11} We classify our case to be of a primary diffuse form without a proximal lesion and segmental smooth muscle hypertrophy due to peptic ulceration and no fibrosis. Differential diagnosis on the basis of diffuse circumferential morphology may be a stromal tumor and in our case other causes are less likely.

The preferred mode of management is surgery with gastric resection and Billroth I anastomosis.^{2,4,7-9} Literature reveals pyloroplasty and vagotomy may be performed with success but later presents with recurrence.¹² Resection and Billroth I anastomosis was performed in our patient with satisfactory results.

Gastric outlet obstruction due to AHPS as a sequel of peptic ulcer disease is a rare benign condition in current surgical practice. It has an uncertain aetiology and proper clinical, radiological and histopathological analysis is necessary to differentiate from other diseases such as

gastrointestinal stromal tumors or other spindle cell neoplasms. Biopsy, frozen sections and post-operative histopathological analysis is essential to identify interstitial metaplasia and carcinoma in situ.

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