



Case Report

Brunner's Gland Hyperplasia Masquerading Carcinoma Stomach: A Diagnostic Dilemma

Neha Tyagi, Akansha, Himani Bhankar, Sufian Zaheer, Ashish Kumar Mandal

Department of Pathology, VMMC and Safdarjung Hospital, Delhi, India

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ABSTRACT

Brunner's gland hyperplasia is a rare benign lesion of the duodenum and is usually found incidentally. We report an unusual case of Brunner's gland hyperplasia in a 67 year old female presenting as gastric outlet obstruction.

***Corresponding author:**

Dr Neha Tyagi, E-60 Anand Niketan New Delhi 110021 New Delhi, 110052 India

Phone: +91 -9873696631

E-mail: drnehatayagi@gmail.com

Introduction

Brunner's gland, first described by anatomist Brunner's in 1688 are found mostly in the duodenal bulb and proximal part of duodenum. Their size and number decreases in the distal part¹. Brunner's gland hyperplasia also known as Brunneroma or Brunner's hamartoma is a rare benign lesion accounting for 10.6% of benign tumours in the duodenum². Only 144 cases have been reported in the literature³. It is mostly an incidental finding and are usually asymptomatic⁴. Brunner's gland hyperplasia rarely can cause gastrointestinal bleeding, abdominal pain and obstruction². We report here a case of Brunner's gland hyperplasia resulting in severe gastric outlet obstruction.

Case Report

A 67 year old female presented to surgery OPD with complains of hematemesis and vomiting since 2 days. Patient was a referred case and was under treatment for gastric outlet obstruction for past one and a half years. Upper GI endoscopy was done many times. First report revealed carcinoma stomach with gastric outlet obstruction with secondary esophagitis. Another endoscopy report showed multiple deep ulcers at the pylorus of stomach. Latest upper GI endoscopy showed gastric outlet obstruction subsequent to pyloric ulcer with narrowing. At that time patient had complains of recurrent vomiting, pain abdomen, constipation and hematemesis. Ultrasound abdomen showed pyloric wall thickening which was circumferential with luminal narrowing. CECT abdomen revealed grossly dilated stomach with symmetric mural thickening in pyloro-duodenal junction most likely a stricture with inflammatory pathology. Patient continued to have recurrent bouts of vomiting and hematemesis. Gastrojejunostomy was done and stomach was found to be distended and nodularity was present in the pylorus region. Biopsy was taken and sent for histopathological examination. Grossly a single grey brown tissue measuring 0.6X0.2X0.1 cm. Microscopically severe proliferation of Brunner's gland was seen (Fig 1). Glands were arranged in large sheets, groups and clusters. The cells were monomorphic with watery clear cytoplasm. These glands were seen occupying whole thickness of mucosa and submucosa. There was no pleomorphism and no mitosis were seen. Therefore a final diagnosis of Brunner's gland hyperplasia was given.

Discussion

Brunner's gland are submucosal mucin secreting acinar glands of the duodenum which secrete mucus, pepsinogen and urogastrone as a response to stimulation by acid³. Brunner's in 1688 anatomically described these glands

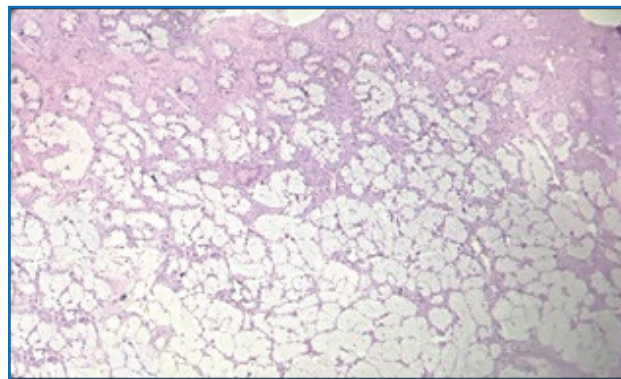


Fig 1: Hyperplasia of Brunner's Gland H and E stain 40X

and gave them the term 'pancreas secundarium'. The name Brunner's gland was proposed by Middeldorph in 1846⁵. The pathogenesis of Brunner's gland hyperplasia is still unknown². Initially it was thought that gastric hyperacidity leading to hyperplasia. On the contrary to this hypothesis 20% may have low gastric acidity³. According to recent studies hyperactivity of exocrine modulating factors such as vagus nerve, intestinal mucus membrane factor is the cause of Brunner's gland hyperplasia⁴.

The incidence of benign tumours of duodenum is 0.008% in patients at autopsy. Lesions smaller than 1cm are counted under hyperplasia while larger than 1cm are called adenomas⁶. Distribution of Brunner's gland hyperplasia is predominantly on the duodenal bulb(57%) followed by second part of duodenum (27%), third part (7%), rarely at sites like pylorus (5%), jejunum (2%) and terminal ileum (2%)⁴. Brunner's gland hyperplasia is usually found in 5th and 6th decade and its size can range from 0.5-12cm⁷. Various conditions like uremia, chronic pancreatitis and Helicobacter pylori infection are found to be associated with this lesion⁸⁻¹⁰.

In 1934, Feyrter classified the abnormal proliferation of Brunner's gland into three types namely diffuse nodular hyperplasia (type 1), circumscribed nodular hyperplasia (type 2) and adenomatous hyperplasia (type 3). Diffuse nodular type is mostly seen as multiple sessile projections in most of the duodenum and can often mimic malignancy⁴. Circumscribed nodular type, the most common type is mainly seen at the duodenal bulb and is usually less than 1cm in size. Adenomatous hyperplasia are generally polypoidal with size ranging from 0.7 to 12 cm¹¹.

Most of the cases of Brunner's gland hyperplasia are asymptomatic⁸. When symptomatic these lesions can cause hemorrhagic or obstructive symptoms. Hemorrhagic manifestations being gastrointestinal

hemorrhage caused by ulceration of the lesion. Obstructive features supervene when hyperplasia diffuses or a giant adenoma which causes vomiting, epigastric bloating and discomfort⁵. Levine et al described clinical features in three categories including asymptomatic group that are diagnosed incidentally (11%), patients with upper gastrointestinal bleeding (40-50%) and those with obstructive symptoms (50%)¹¹.

Various diagnostic modalities such as ultrasonography, barium contrast studies and endoscopy can aid in the diagnosis of Brunner's gland hyperplasia⁶. In barium meal findings are non-specific and we can see sessile or pedunculated filling defect at duodenal bulb¹². Endoscopy can not only aid in diagnosing the condition but can also help in the removal of the lesion simultaneously⁵. Sensitivity of endoscopy is 72-89% but can be non-diagnostic in few cases⁴. The reason being submucosal location of these lesions which makes it difficult to sample in a punch biopsy². The sensitivity of endoscopic biopsy is 83-92% and diagnostic accuracy is 91-97%⁴. In few cases abdominal CT might be helpful. The clinical picture and radiology can be non specific at times therefore arises the need of histopathology which still remains the gold standard for diagnosing this entity.

If the patient is asymptomatic conservative treatment is given. In case of symptomatic patients treatment is resection which can be done via endoscopy, laparoscopy or during laparotomy. If the lesion is pedunculated and less than 5cm in size then endoscopic polypectomy is done. In case of lesions larger than 5cm open surgical excision is preferred⁵. Our case report highlights that in evaluation of patients with gastric outlet obstruction a benign pathology i.e Brunner's gland hyperplasia should be kept in the differential diagnosis.

Conclusion

Our case report highlights that a benign pathology can sometimes mimic a malignant one clinically. So a combined clinic-radiological workup and histopathological analysis is needed to give a correct diagnosis.

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Competing Interests

Not Declared

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