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AN ACUTE ABDOMEN MIMIC IN A YOUNG BOY

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ABSTRACT Brunner's gland hyperplasia usually presents itself clinically without any symptoms. Brunner's gland hyperplasia is mostly an incidental finding during upper gastrointestinal endoscopy. Brunner's glands are specialised duodenal glands present in submucosal region of the proximal part of the duodenum. This specialized Brunner's glands protect the epithelium off the duodenum by secreting fluids which are alkaline in nature. The incidence of these tumours are very rare with an approximated finding in 0.008% in 2,00,000 post mortem studies.1 Here, the discussion is about a young boy diagnosed with Brunner's gland hyperplasia mimicking an acute abdominal pathology.

KEYWORDS : Brunner's gland ; H. pylori ; Hyperplasia; Duodenum ; Upper GI endoscopy

CASE REPORT:

A 13 year old boy brought by his mother, to our hospital with complaints of diffuse abdominal pain, of severe intensity, predominantly over the epigastric and umbilical regions, radiating to the right iliac fossa. He also had intermittent bouts of vomiting which contained food particles and non-bilious in nature. He was tachycardic, but hemodynamically stable otherwise. Haematological investigations revealed no abnormalities, and his renal and hepatic panels were unremarkable. Serum lipase and amylase were within normal limits as well. Abdominal radiographs, USG were unremarkable.

An upper gastro-intestinal endoscopy was planned and it revealed nodular gastric mucosa in the body of the stomach, presence of duodenal nodules and inlet patches in the oesophagus. Multiple biopsies taken from the nodules and various other sites validated the diagnosis of Brunner's gland hyperplasia.

Histopathological evidences confirmed diagnosis of Brunner's gland hyperplasia with lobular proliferation and cystic dilatation of the Brunner's glands. The mucosa overlying the nodules exhibited features suggestive of chronic duodenitis and tested positive for *Helicobacter pylori* infection. The evidence of dysplasia was not present.

Patient was treated symptomatically with empirical IV antibiotics, proton pump inhibitors, and *H.pylori* eradication therapy, and was discharged a week later without any significant complications.

Fig 2 – Photographs of nodular gastric mucosa and duodenal nodules observed in this case.

DISCUSSION:

In the cluster of the benign duodenal tumours, incidence of Brunner's gland hyperplasia is estimated to be 10.6%.[2] They are observed equally in both genders but are usually diagnosed in the sixth decade of life. A well understood histopathological definition and classification system of Brunner's gland pathologies are still needed. It was said that Lesions smaller than 1 cm are likely hyperplasia, those larger than 1 cm are considered adenomas, and those containing a mix of muscular and fatty tissues are classified as hamartomas.[3,] We feel that the term hyperplasia is more applicable in in respect to our case.

Various hypotheses have been elucidated in regard to the aetiology of Brunner's gland hyperplasia. Since these glands secrete alkaline fluid composed of mucin, increased acid secretion was thought to play a part in the pathogenesis, but there was no reduction in size or regression of these lesions with proton pump inhibitors.[4,8]H pylori was established in 71% of Brunner's gland hyperplasia. Due to utmost rareness of this condition with ubiquity of *H. pylori* infections in the Indian subcontinent does not paint a clear picture.

Although mostly these are benign lesions, dysplastic and malignant Brunner's gland lesions have been reported in a few case .[5,6]

The common site distribution of Brunner's gland hyperplasia is in the duodenal bulb present in the proximal duodenum (70%), following in the second part of the duodenum (26%). Pedunculated lesions (88%) are composed of the majority and are usually 2-3cm in the largest diameter.[2,7]

Although usually asymptomatic, the common variations of symptoms include upper GI bleed, intestinal obstruction, pancreatitis due to periampullary obstruction, intussusception. Cases presenting as an acute abdomen are quite rare.

Histopathological examination by mode of upper endoscopy is usually sufficient for the establishment of a diagnosis. Endoscopic ultrasound examination can reveal extent and infiltration of structures adjacent to the lesion.

Upper GI endoscopy with excision of a bleeding polyp seems to be the absolute option because it is minimally invasive, less expensive, and with less complications. Open laparotomy should be restrained for lesions more than 10cm, sessile lesions, and failure of an endoscopic approach.

Pertaining to our case, a conservative approach proved to be effective due to the lack of a bleeding polyp. After time observant monitoring, with cessation of symptoms, the patient was discharged with the advice to follow-up, judicial compliance of *H.pylori* eradication therapy and explanation of signs and symptoms of an acute GI bleed.

CONCLUSION:

Brunner's gland pathologies presenting with symptoms of acute abdomen are rarely reported in literature as they are an incidental finding in upper gastrointestinal endoscopies. The need for close monitoring and follow-up is emphasized upon to prevent complications such as an acute gastrointestinal haemorrhage.

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