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A Rare Interesting Case of Brunner's Gland Hyperplasia Causing Gastrointestinal Tract Obstruction.

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Abstract: Brunner glands are exocrine glands that secrete mucus to protect against acid secretion. Brunner gland hyperplasia is an expansion in size and shape of glands due to excess mucus secretion which occurs in cases where acid is secreted in excess notably duodenal ulcer or gastritis. Usually asymptomatic as Brunner gland hyperplasia appears as small nodular growth noticed during endoscopic examination. Some Brunner gland hyperplasia causes gastric outlet symptoms that are seen in our case study, as we present a rare and interesting case of Brunner gland hyperplasia causing blockage of the gastric outlet (rare symptom). Although endoscopic removal was done in suspicion of malignancy and turned out to be benign Brunner gland hyperplasia which we are discussing in our case as an elaborate discussion. The importance of considering Brunner gland hyperplasia as a differential diagnosis for gastric outlet blockage, as well as the use of endoscopic biopsy for ruling out cancer, are reviewed in depth. There have been many reports in the adult literature of Brunner's gland hyperplasia causing the bleeding, blockage, or forming intestinal or duodenal folds. Similar cases of Brunner gland hyperplasia impeding stomach outflow are highly uncommon and should be reported.In cases of gastric outlet blockage, Brunner gland hyperplasia should be considered as a distinguishing a particular disease or condition from others that present with similar clinical features, and endoscopic assessment with histopathological microscopy may be a valuable tool in preventing needless surgical intervention. This case report research adds to the necessity of endoscopic biopsy.

Keywords: Brunner gland hyperplasia, Gastrointestinal obstruction, Duodenum, Histopathology, Polypoidal lesion.

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1. INTRODUCTION

Brunner's glands are exocrine glands that are found in the duodenum beneath the lining epithelium and empty into the Lieberkühn crypts. In addition to acid stimulus, they secrete mucus, pepsinogen, and urogastrone, which are most abundant in the duodenal bulb. Hyperplasia is the proliferation with an increase in the size of the Brunner's glands, which are typically asymptomatic and only rarely identified. The cause of these lesions is yet unknown [1]. It is frequently asymptomatic and discovered by chance during esophagogastroduodenoscopy (EGD). Just 144 cases of hyperplasia of these glands have been reported in the scientific literature [2,3]. Feyrter identified three forms of pathological glandular proliferation in 1934: type 1, type 2, and type 3[4]. Many sessile projections can be observed in the duodenum in Type 1 diffuse nodular hyperplasia, Type 2 has duodenal bulb-limited circumscribed nodular hyperplasia, Type 3 is characterized by glandular adenoma and polypoid lesions[4]. It's uncertain if these three histological findings are related. Feyrter's definition, on the other hand, is divisive, with some scholars arguing that all modes should be included[5]. Brunner gland hyperplasia normally appears in age group where, men or women are neither old nor young and has no sex preference [5]; however, cases have been recorded as early as childhood and as late as 80 years of age. Pathology of the Brunner gland generating symptoms of gastric outlet blockage is a rare occurrence. Because Brunner gland hyperplasia is typically benign, surgical or endoscopic excision of the polyp is a safe option [6]. An ocular micrometre was used to estimate the thickness of Brunner's glands in 75 instances of surgically excised duodenal ulcers and 75 postmortem cases as a control in a clinical investigation on the incidence of Brunner's gland hyperplasia in individuals with duodenal ulcers. The Brunner's glands become hyperplastic in duodenal ulcer patients, especially near the ulcer, according to the findings[7]. Acidity contributes to the occurrence of duodenal ulcers, and the relationship between acidity and Brunner's gland hyperplasia emphasises the relevance of acidity as an etiological component. Although Brunners gland hyperplasia is a benign disease, our study emphasises the significance of surgical removal and histological investigation of polypoidal masses in the duodenum.

2. CASE REPORT

We studied a case report of a 65-year-old male presented with complaints of vomiting more than 10 episodes for the past 2 days, complaints of upper abdominal pain on and off for the past 5 days, and complaints of early satiety. He is non-diabetic and has no history of systemic hypertension. Written & Oral informed consent was obtained from the patient. His HbA1c is 6.2, and his fasting and postprandial blood sugar were 80 mg/dl and 103 mg/dl, respectively. He was a smoker and had on and off upper abdominal pain. MRCP did which showed duodenal polyp- Malignancy of duodenum. Upper GI endoscopy shows gastric outlet obstruction with nodularity of the duodenum. No luminal mass. The lesion was identified. Biopsies were taken as well as the removal of the polypoidal mass was done and sent for histopathology. For histological investigation, biopsy samples were fixed in 10% formalin and then embedded in paraffin blocks. These blocks were cut into 4m (microns) serial portions. The tissue slices were stained with haematoxylin and eosin after deparafinization. A pathologist with light microscopy skills reviewed the slides. Histopathology from the polypoidal lesion from the duodenum shows pedunculated duodenal mucosa. Submucosa shows lobules of benign Brunner glands which are hyperplastic with increased mucin secretion (Figure 1). The Hyperplastic Brunner glands were separated by fibrovascular septa. The glands are made up of neutral mucin-secreting cuboidal to columnar cells. The proliferative glands have peripherally placed nuclei because of an increase in alkaline mucinous secretion inside the cells (Figure 3).There is also ulceration of Duodenal mucosa due to erosive duodenitis and excess acid secretion noted in our case (Figure 2).

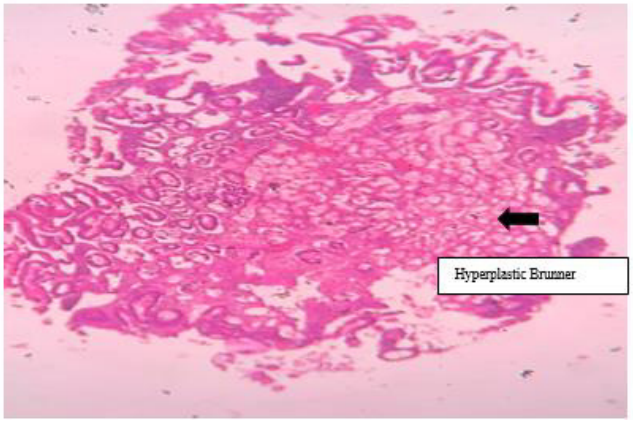

Fig: 1 Scanner view of Brunner gland hyperplasia (arrow pointed) seen in the submucosa of duodenum

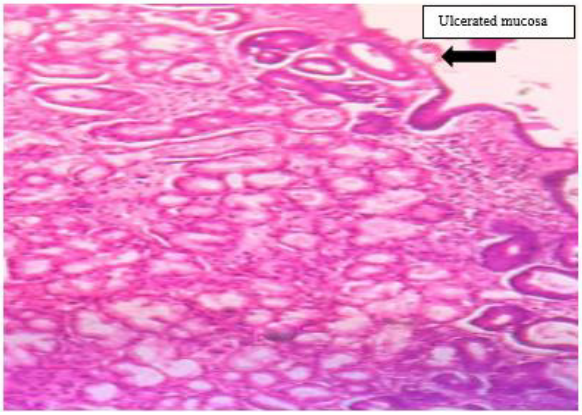

Fig: 2 Low power view (10x)-ulcerated mucosa (arrow pointed) with Brunner gland seen in submucosa.

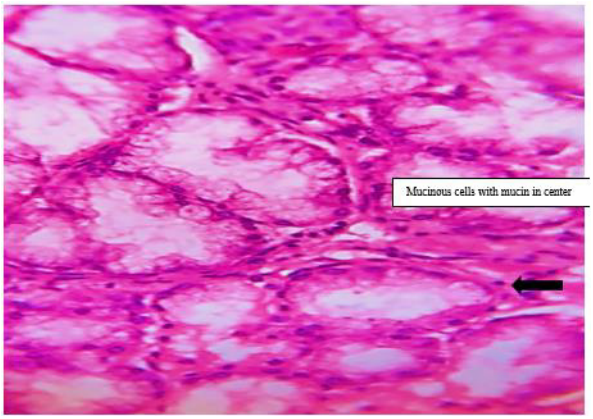

Fig: 3 High power view (40 x) showing Brunner gland hyperplasia with mucinous dilated glands having mucinous cells with a peripherally placed nucleus (arrow pointed).

3. DISCUSSION

Gastrointestinal obstruction following Brunner gland hyperplasia is rarely seen. The Brunner's gland, which is located in the duodenum, secretes mucus that includes anti-acid protective covering epithelium. As a result, when there is an excess of acid and infection with Helicobacter pylori, these glands proliferate or increase in size [8]. Brunner gland hyperplasia accounts for more than 4% and less than 10% of all benign duodenal lesions [9]. The origin of Brunner gland hyperplasia is uncertain, however a link to hyperacidity and Helicobacter pylori infection has been suggested [11,12]. The Giemsa stain revealed that the H.pylori bacterium was not present in our instance. Most Brunner gland tumors are usually presenting without any symptoms sitting dormant. Brunner gland hyperplasia can cause haemorrhage [10,11], such as blood in the stools or vomitus, as well as other symptoms such as stomach discomfort, vomiting, and satiety. Some Brunner gland hyperplasia manifests as a lesion in the second half of the duodenum at the cystic duct opening [12], while others progress to biliary obstruction with an increase in pancreatic parameters in the blood [13]. Some may seem to be malignant neoplasms, necessitating biopsy or surgery with histopathology and immunohistochemistry to distinguish the tumor [14]. An MRCP was performed, which revealed occlusion of the duodenal gastric outflow, and an endoscopic biopsy was performed. Despite the fact that endoscopic biopsies and polypectomy were performed endoscopically. Although the polypoidal mass revealed primarily benign Brunner gland hyperplasia, the depth of tissue excision may not be sufficient to assess the duodenal tissue and rule out malignancy [15,16]. Adenomatous polypoid mass, which is adenomatous polyposis coli and gene associated with familial adenomatous polyposis coli, may be linked to polypoidal lesions in the duodenum. There's also the risk of pancreatic pedunculated carcinoma infiltrating the duodenum. Despite the fact that the histopathological findings from endoscopic removal of polyp and MRI findings were inconclusive, a surgical resection of the polypoidal tumor must be used to determine the depth of tissue invasion [17,18]. Polypoid hamartoma, mass-forming hamartoma, and circumferential infiltrative hamartoma are the three types of Brunner's gland hamartoma. Obstructive symptoms are most commonly produced by the big polypoid type or the mass-forming type, and are extremely seldom connected with the circumferential type, based on their physical appearance[19]. Apart from imitating pancreatic adenocarcinoma, there is a chance of malignant change in very uncommon cases. In research done by Brookes et al., there is considerable dysplasia and malignant transformation with diffuse cytological alterations found in microscopic inspection of this Brunner gland. The occurrence of malignancy in Brunner gland hyperplasia is rare, Brunner gland hyperplasia and Brunner gland hamartoma account for 1 percent of small intestinal tumors[16,20]. However, there is no dysplasia or cytomorphological alterations in our instance. Each condition, such as Brunner gland hyperplasia and lipomatous pseudohypertrophy of the pancreas, occurs individually. Long Cong Nguyen et al. found an incidence of both -Brunner gland hyperplasia and pancreatic lipomatous pseudohypertrophy in one of their studies, raising the possibility of Brunner gland

hyperplasia causing pancreatic lipomatous pseudohypertrophy[21].The MRCP does not reveal any pancreatic lipomatous pseudohypertrophy in our case.The mild hypoglycemia seen in our case is due to a mild increase in insulin secreted from GLP1 receptors located in Brunner glands hyperplasia. Mucus and bicarbonate secretion are stimulated by incretins (glucagon, GLP-1, vasoactive intestinal peptide, secretin, and cholecystokinin) and neuronal factors (acetylcholine)[22]. The GLP-1 receptor in the duodenal mucosal layer appears to be critical for neuralglucoregulation, gut lipid sensing, pathogen defence, and mucosal repair[23,24]. Matthias Hepprich et al. conducted research on a post-bariatric surgery patient who had hypoglycemia and discovered a probable link between gastric bypass surgery, GLP1 receptors, and Brunner gland hyperplasia in the development of hypoglycaemia[25]. To distinguish it from insulinoma, immunohistochemistry markers for GLP1 receptors and insulin were employed in this investigation. GLP 1 receptors were found to be strongly positive, suggesting Brunner gland hyperplasia, but insulin was found to be negative, indicating the absence of insulinomas[25].In our study we did not use immunohistochemical markers for distinguishing it from insulinomas, the slight decrease in sugar levels was satisfactory to above said results. To rule out malignancy, any polypoidal tumour in the duodenum must be surgically removed, even if it is ruled out as benign Brunner hyperplasia. The patient was asymptomatic in our scenario after endoscopic removal of a polypoid lesion. It's critical to emphasise the significance of regulating acidity with pharmaceuticals like proton pump inhibitors, sucralfate, antacids, and other supportive medications. The patient must be followed up on a regular basis as recurrence can happen and the need for surgery if its causing obstructive symptoms. The histopathological workout in our case is very useful in ruling out malignancy.

4. CONCLUSION

In our case study we demonstrate that Brunner's gland hyperplasia, a benign condition, should be included in the differential diagnosis of individuals with gastric outlet blockage. Even if benign Brunner hyperplasia is ruled out, surgical removal of any tumour causing blockage in the duodenum is required. According to several studies mentioned in our case report, surgical removal via endoscopy or open abdominal laparotomy relieves the blockage or obstruction, resulting in symptom-free living, and we insist on histopathological examination with appropriate immunohistochemical markers to differentiate benign from malignant causes. In addition, we emphasise the reduction in sugar levels caused by Brunner gland hyperplasia, which is a new occurrence reported in recent research, as well as the use of immunohistochemical markers for GLP 1 receptors for a more accurate and timely diagnosis. Predisposing factors such as Helicobacter pylori and other sources of increased acidity should be checked out since these etiological factors are indirectly favouring the development of Brunner gland hyperplasia. To treat acidity, which is thought to be the cause of Brunner gland hyperplasia, it's important to stress the need of taking pharmaceuticals like proton pump inhibitors, sucralfate, antacids, and other supportive medications to control acidity. Because there is a chance of recurrence, the patient must be followed up with on a regular basis.

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6. AUTHORS CONTRIBUTIONS STATEMENT

Dr. Azeem I produced this case report under the supervision of Dr. Vindhu and Dr. Natarajan Suresh. Dr. Vindhu and Dr. Natarajan Suresh evaluated and led me through every step of my assignment, including photographing the H and E slide section of the case study. The final version of the work was reviewed and approved by all authors.

7. CONFLICT OF INTEREST

Conflict of interest declared none.

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