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Brunner's Gland Hamartoma: Masquerading as a Duodenal Mass

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ABSTRACT

Brunner's gland hamartoma is a benign mass of the duodenum often asymptomatic in presentation and typically identified endoscopically. Treatment is dependent on how the mass manifests. If asymptomatic, then no treatment is necessary. If there is concern for malignant transformation or obstruction, then treatment is endoscopic or surgical resection.

1 | Introduction

Brunner's glands are mucosal or submucosal glands most commonly found in the duodenum [1, 2]. Brunner's gland hyperplasia or hamartoma is a benign growth of the mucosal or submucosal layers of the duodenum. The pathogenesis is unknown. There have been a few proposed etiologies ranging from high gastric acid secretion to *Helicobacter pylori* infection, the result of inflammation, and chronic pancreatitis [1, 2]. However, given the rarity of the presentation, a clear link has been difficult to establish. The manifestation of Brunner's gland hamartoma is estimated to be present in approximately 10% of benign duodenal tumors and typically presents in the fifth and sixth decade of life [1, 2]. The hamartoma characteristically forms from polypoid pedunculated duodenal masses as they grow [2]. Brunner's gland hamartomas often present without symptoms. If symptoms are present, they are nonspecific obstructive symptoms such as nausea, vomiting, abdominal pain, or gastrointestinal bleeding. Therefore, the diagnosis is usually made endoscopically [2]. We present a rare case of Brunner's gland hamartoma, initially thought to be a gastrointestinal stromal tumor, presenting with anemia.

1.1 | Case History and Examination

A 53-year-old male presented to his primary care clinic with fatigue and shortness of breath. His past medical history was only notable for hyperlipidemia, and he had no prior history of endoscopic procedures. His vital signs were stable. He was found to be anemic, with a hemoglobin of 8.6 g/dL, and the rest of his labs were within normal limits. On physical examination, his abdomen was non-tender, and there was no palpable mass.

1.2 | Differential Diagnosis, Investigations, and Treatment

He underwent Esophagogastroduodenoscopy (EGD) and endoscopic ultrasound (EUS) with biopsy. Results demonstrated a 4×4 cm round hypoechoic mass with some cystic features within the duodenal bulb. Endoscopically (Figure 1), the lesion appeared most likely to be a gastrointestinal stromal tumor (GIST). However, histopathology of the EUS biopsy showed non-diagnostic blood and benign enteric cells read as a benign neoplasm of the duodenum. Subsequent computed tomography

(CT) of the abdomen and pelvis with contrast showed a 2.5 × 7 cm soft tissue mass in the duodenal bulb and descending duodenum consistent with the known mass (Figure 2). No additional masses or abdominal lymphadenopathy were identified.

The patient followed up with a general surgery team. Given the non-diagnostic result on initial biopsy, a repeat EUS with biopsy was recommended to rule out the possibility of malignant neoplasm that could require neoadjuvant therapy. Repeat EUS showed an intramural (subepithelial) lesion in the duodenal bulb. The lesion appeared to originate from within the submucosa (Figure 3). Repeat sampling with fine needle biopsy was read as "excessive Brunner gland tissue," consistent with Brunner's gland hyperplasia. (Figures 4 and 5).

Given that the lesion was benign, treatment options favored a flexible approach for watchful waiting.

1.3 | Outcome and Follow-Up

His overall clinical presentation was stable in the setting of his anemia; however, tentative surgery was planned for 6 months in the future if alarm symptoms manifested. The ensuing surgical

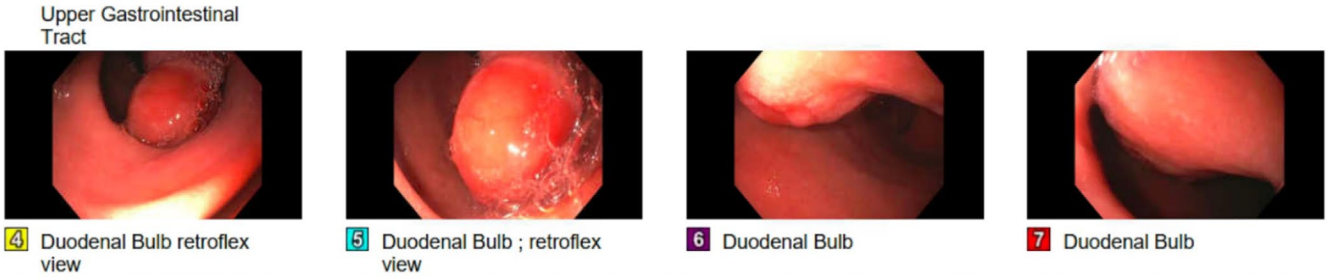

FIGURE 1 | Esophagogastroduodenoscopy (EGD) of mass in the duodenal bulb.

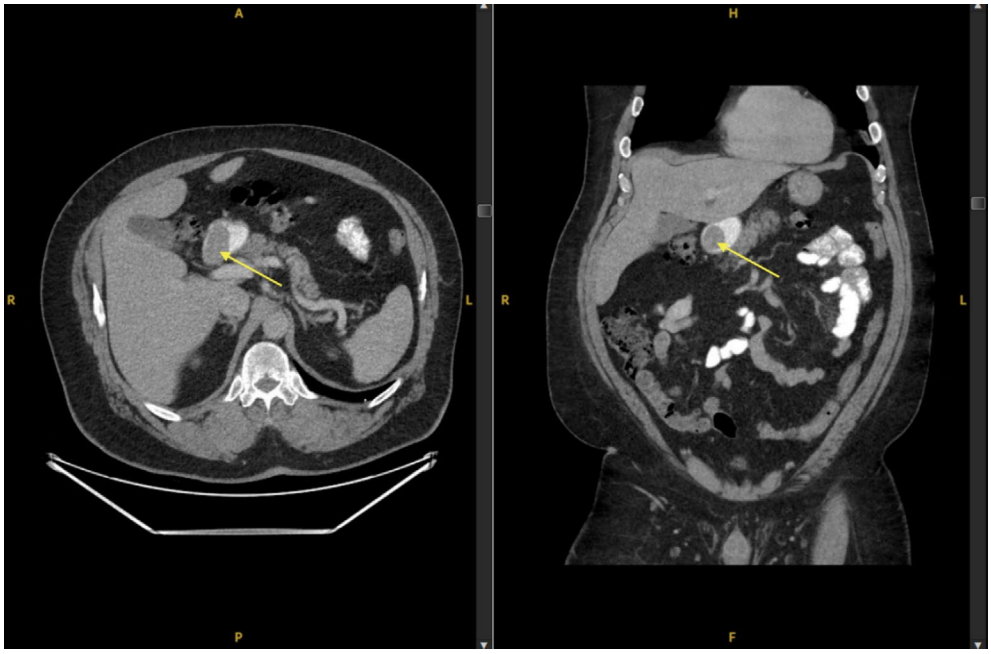

FIGURE 2 | Computed tomography (CT) showing 2.5 × 7 cm soft tissue mass in the duodenal bulb and descending duodenum.

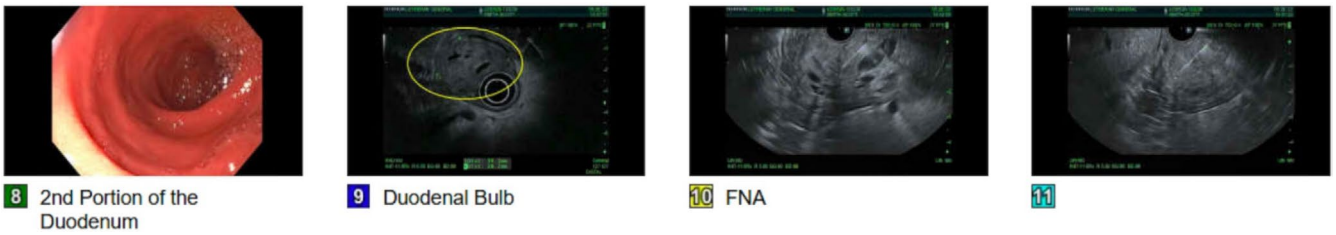

FIGURE 3 | Endoscopic ultrasound (EUS) showing hypoechoic intramural (subepithelial) lesion in the duodenal bulb. The lesion appears to originate from within the submucosa (Layer 3).

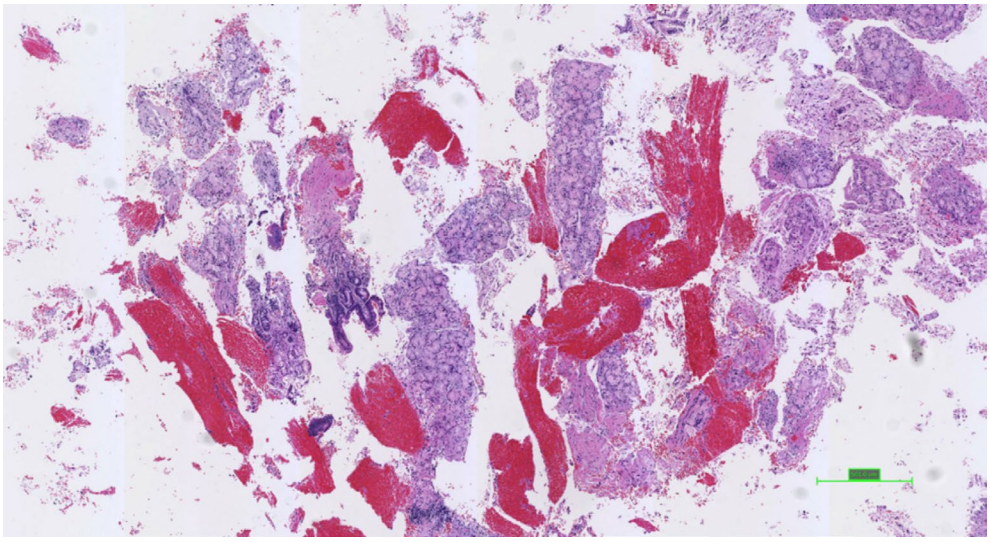

FIGURE 4 | Histopathological appearance of the duodenal lesion. The biopsy specimen shows an abundance of Brunner gland tissue (H&E, x40). In the absence of more significant clinicopathologic findings, this pathology is consistent with Brunner gland hyperplasia.

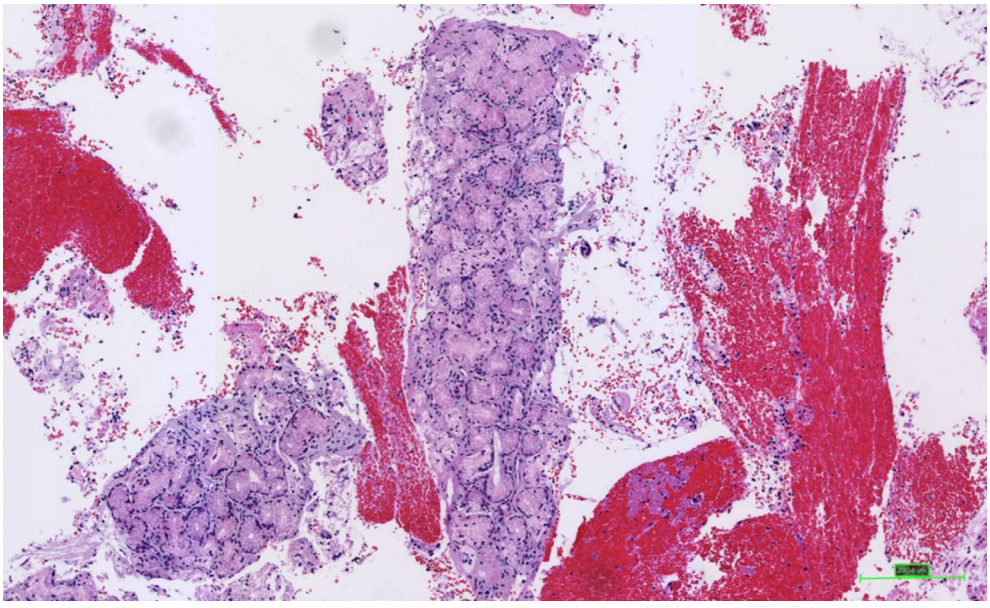

FIGURE 5 | At high power magnification, the glands consist of closely packed clusters of cuboidal cells with flat basal nuclei and foamy cytoplasm (H&E, x100). There were no intermingled smooth muscle or adipose tissue elements. There was no cytological atypia, nuclear enlargement or mitotic activity.

plan would be to proceed with the option of laparoscopic robotic-assisted duodenal resection of the mass if the patient experienced any additional significant hemoglobin drop resulting in instability, melena, hematochezia, or obstruction during the 6-month waiting period. During the 6-month waiting period, the patient did not have any alarm symptoms. Repeat CT of the abdomen at the end of the waiting period showed a stable lesion. Therefore, no intervention occurred for his benign lesion.

2 | Discussion

Nomenclature surrounding Brunner's gland hyperplasia versus hamartoma and adenoma is not very well defined and has been a source of contention when identifying this rare manifestation. Brunner's gland hyperplasia was first reported in 1835 by Cruveilheir [1, 3]. Later, Feyrter categorized Brunner's gland proliferation in 1934 into Type 1 diffuse nodular hyperplasia, Type 2 circumcised nodular hyperplasia, and Type 3 glandular hamartoma/adenoma [1, 4].

Another classification differentiates between hyperplasia and hamartoma based on size. Brunner's gland hyperplasia is described as solitary or multiple nodules <0.5 cm in diameter [5]. On endoscopy, a heterogeneous hypoechoic mass is located in either the mucosal or submucosal layer [5, 6]. In contrast, Brunner's gland hamartomas are thought to be >0.5 cm in size [5]. The nomenclature is often described as arbitrary. However,

given these definitions, it can be thought that our patient had a true Brunner's gland hamartoma.

Brunner's gland hamartomas are most commonly found in the duodenal bulb (57%–70%), followed by the second portion of the duodenum, and lastly, the third portion of the duodenum [5, 7]. Although typically found in the proximal duodenum, they can also appear in the distal duodenum and sometimes extend into the proximal jejunum [1, 7, 8]. They have even been reported to extend to the proximal ileum in a well-documented case in 1983, where a Brunner's gland hamartoma appeared as an ileal obstruction [9].

The pathogenesis of the Brunner proliferation is unknown [1, 2, 7]. The current literature suggests that Brunner's gland proliferation may be due to *Helicobacter pylori* infection, high gastric acid secretion, dysplasia of the duodenum, mucosal injury, chronic pancreatitis, or other inflammatory stimulation [1, 2, 7, 10, 11]. In the case of our patient, the inciting factor was also unclear.

It is also essential to rule out differential diagnoses when looking at duodenal lesions. These include but are not limited to gastric heterotopia, inflammatory fibroid polyp, lipoma, leiomyoma, carcinoid, gastrointestinal stromal tumor, lymphoma, non-ampullary sporadic adenoma, familial adenomatous polyposis, Peutz-Jeghers syndrome, and solitary Peutz-Jeghers polyp [11]. Brunner's gland hamartoma typically presents in the 50-60th decade of life and does not have gender predominance. This helps differentiate Brunner's gland pathology from other duodenal lesions, which may present earlier [7]. Biopsy of Brunner's gland may initially be non-diagnostic because the biopsy does not penetrate the submucosa [5]. It was initially thought that our patient had a gastrointestinal stromal tumor solely based on the earlier endoscopic findings. Additionally, the initial biopsy was non-diagnostic. As a result, endoscopic sonography or endoscopic ultrasound (EUS) is often helpful in allowing for both visualization and identification of Brunner's gland hamartoma. The lesion is frequently described as having variable echogenicity and may produce a more heterogeneous pattern [6].

Brunner's gland hamartomas are often asymptomatic. If they do present with symptoms, they manifest as obstruction characterized by abdominal distention and vomiting [9]. They also occasionally present with symptomatic anemia [11, 12] such as in a well-documented case by Cai et al. where a patient presented with gastrointestinal bleeding and was found to have a Brunner's gland hamartoma [13]. In contrast, our patient presented with non-complicated anemia without signs of active bleeding. Lastly, there is a possibility of malignant transformation. Specifically, Brunner's gland hamartomas have been known to transform to dysplasia in 2.1% of cases and to adenocarcinoma in 0.3% of cases [14].

Ultimately, the treatment for Brunner's gland hamartomas depends on how the tumor manifests. If asymptomatic, no treatment is necessary unless there is concern for transformation to malignancy [14]. If symptomatic, such as causing hemorrhage or obstruction, then the tumor should be removed and is typically done either endoscopically or surgically [15]. If there is a compromise of the surrounding structures, sometimes surgical resection with a more aggressive approach may be explored. More specifically, aggressive surgical methods such as Whipple surgery may be offered but are often reserved for cases where there is a concern for malignancy [16].

Author Contributions

Dirin Ukwade: conceptualization, data curation, formal analysis, resources, validation, visualization, writing – original draft, writing – review and editing. **Sana Hussain:** conceptualization, writing – review and editing. **Saaed Ali:** conceptualization, data curation, formal analysis, supervision, writing – review and editing. **Brian Boulay:** data curation, formal analysis, supervision, writing – review and editing.

Ethics Statement

All procedures performed were in accordance with the ethical standards. The examination was made in accordance with the approved principles.

Consent

Written informed consent was obtained from the patient to publish this report in accordance with the journal's patient consent policy.

Conflicts of Interest

The authors declare no conflicts of interest.

Data Availability Statement

The authors have nothing to report.

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