

Brunner's Gland Hamartoma of the Duodenum (Brunneroma)

Stuart A. Becker MD¹ and Nadia Ziv-Sokolovskya MD²

¹Division of Gastroenterology, Tel Aviv Sourasky Medical Center, Tel Aviv, Israel

²Institute of Pathology, Kaplan Medical Center, Rehovot, Israel

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The initial credit for describing Brunner's gland hamartomas is given to Cruveilhier in 1834, with excellent and more readily available reviews subsequently written by Goldman [1] in 1963 and Strutynsky et al. [2] in 1982. Although only 143 cases had been described until 1984, in the last few years new case reports appear almost monthly in the medical literature. Initially felt to be an incidental finding on endoscopy, Brunneromas, when present, often cause upper gastrointestinal bleeding and abdominal colic, and occasionally pancreatic-biliary symptomatology [3]. While classified as non-neoplastic lesions with no malignant potential [2], there is at least one recent report of a Brunneroma with dysplastic changes [4]. In the current report, emphasis is placed on the evolving concept of Brunneromas histopathologically, the importance of endoscopic ultrasound for both diagnosis and treatment, and the suitability of endoscopic resection.

Patient Description

A 29 year old man in moderate good health (apart from symptomatic asthma) developed epigastric pain, without nausea, vomiting or diarrhea, in October 1999. Initial hemoglobin was 12.2 mg/dl. Several days later the abdominal pain intensified, followed by a melenotic stool. Upon admission to the hospital, repeat hemoglobin was 7.4 mg/dl with blood urea nitrogen elevated to 46. In rapid sequence the patient twice underwent gastroscopy, colonoscopy and upper gastrointestinal and small bowel series. Except for a small duodenal diverticulum, no abnormalities were found. After receiving two units of blood, the patient's hemoglobin stabilized at 8

mg/dl and he was released from hospital.

After discharge from the hospital, the patient was seen in routine follow-up in December 1999. He was asymptomatic. His hemoglobin was 13.3 mg/dl, serial stool hemocult examinations were negative, and his serum iron/total iron-binding capacity was normal. Meckel's scan was normal. The patient was discharged from active follow-up but was told to return if he again developed melena, protracted nausea, vomiting, or abdominal pain.

One year later he returned following an episode of melena, with a drop in hemoglobin to 7.7 mg/dl. An emergency red blood cell bleeding scan was performed and was normal. At repeat gastroscopy a large broad-based mass was found in the middle portion of the second part of the duodenum [Figure A], with blood located immediately distal to it. In an attempt to establish the exact location of this polypoid mass, hypertonic duodenography was performed, confirming location in the

distal second portion of the duodenum. Because of the precarious location of the polypoid mass, and because the patient's asthma was problematic, it was decided to remove the mass by formal laparotomy instead of endoscopically. At laparotomy the mass was easily palpated, and had prolapsed distal to the ligament of Treitz causing at that time a small bowel obstruction. At duodenostomy the mass turned out to be a large polyp measuring 3.5 x 3 x 1 cm with a stalk measuring 1.3 x 0.3 x 2 cm. Histology documented that the mass was a Brunneroma [Figure B]. With resection of the Brunneroma, recovery was rapid and complete. Five years later, there have been no further episodes of recurrent gastrointestinal bleeding or abdominal pain.

Comment

It was Feyrter (cited by Goldman) – after examining 2,800 duodenums – who provided the first classification of hypertrophy of Brunner's glands into one of three categories (diffuse nodular hyperplasia, circumscribed hyperplasia, and adenomatous single nodular hyperplasia). Goldman, however, felt that Brunner gland hypertro-



Figure 1. Endoscopic view of Brunneroma in the second portion of the duodenum. B = Brunneroma.

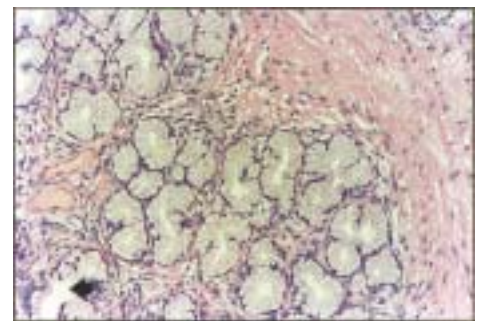


Figure 2. Histology: Brunner's gland hamartoma: lobules of Brunner's glands surrounded by bands of muscles. Arrow indicates dilated gland.

phy, later to be termed Brunneroma, was in fact a spectrum of changes that were neither hyperplastic nor neoplastic but rather proliferative. It was his finding of acinar, adipose and ductular elements together with thickened bundles of muscle tissue, with no surrounding capsule and the distinct absence of atypia, that convinced Goldman that a Brunneroma was in fact a benign process best classified as a hamartoma [1]. Although almost all of the nearly 200 case reports in the medical literature note that Brunner's gland hypertrophy is a benign process, a recent case report by Brookes et al. [4] documented, for the first time, dysplastic elements in an otherwise classic Brunneroma.

Despite their relative rarity, Brunneromas are clinically significant tumors because 45% of them bleed and 51% of them cause partial small bowel obstruction with colic [3]. Not unexpectedly, since most Brunneromas are located in the second part of the duodenum, there have been rare case reports of obstructive jaundice and even recurrent pancreatitis [3]. Radiologic diagnosis, with barium, requires a high index of suspicion and directed radiology utilizing hypertonic duodenogra-

phy. Endoscopy, deep into the second part of the duodenum, is usually successful in locating these lesions as little or no benefit is gained diagnostically from endoscopic biopsies because of their superficial nature. Endoscopic ultrasonography, best described by Weisselberg et al. [5], has proven to be the imaging study of choice to diagnose a Brunneroma and determine the safest method of resection. Classically, on endoscopic ultrasonography, the Brunneroma is a hyperechoic lesion with occasional cysts (due to dilated glands), located entirely in the submucosa, most importantly not involving the underlying muscularis propria. By demonstrating the benign appearance of a Brunneroma, identifying the presence or absence of a stalk and the size of its central vessel, endoscopic ultrasonography provides the information necessary to determine if resection can be done safely endoscopically or if formal laparotomy is required.

Despite their rarity, but because of significant clinical sequelae, Brunner's gland hamartomas of the duodenum (Brunneromas) represent a histopathologic entity that can readily be diagnosed and treated with available endoscopic modal-

ities. Since resection (whether endoscopic or by formal laparotomy) is definitive, intervention is clearly warranted in this potentially troublesome, but easily removed lesion of the proximal duodenum.

References

1. Goldman L. Hamartomatous polyp of Brunner's glands. *Gastroenterology* 1963;44:57-62.
2. Strutynsky N, Posniak R, Mori K. Obstructing hamartoma of Brunner's glands of the duodenum. *Dig Dis Sci* 1982;27:279-82.
3. Waldon DT, Marcon NE. Endoscopic injection and polypectomy for bleeding Brunner's gland hamartoma: case report and expanded literature review. *Gastrointest Endosc* 1998;47:403-6.
4. Brookes MJ, Manjunatha S, Allen CA, Cox M. Malignant potential in Brunner's gland hamartoma. *Postgrad Med J* 2003;79:416-17.
5. Weisselberg B, Melzer E, Liokumovich P, Kurnik D, Koller M, Bar Meir S. The endoscopic ultrasonographic appearance of Brunner's gland hamartoma. *Gastrointest Endosc* 1997;46(2):176-8.

Correspondence: Dr. S.A. Becker, Division of Gastroenterology, Tel Aviv Sourasky Medical Center, 6 Weizmann Street, Tel Aviv 64239, Israel.

Phone: (972-3) 697-4466

Fax: (972-8) 949-4694

email stuart_b@tasmc.health.gov.il

Can't act. Can't sing. Slightly bald. Can dance a little

1928 verdict of the director of screen tests at MGM studios after Fred Astaire's first screen test



If excessive smoking actually plays a role in the production of lung cancer, it seems to be a minor one

1954, W.C. Hueper MD, National Cancer Institute

Capsule

Bacterial antibiotic resistance

The inherent persistence of bacterial populations after exposure to antibiotics or other stress is well known but little understood. Such persistence is distinct from acquired antibiotic resistance and, on regrowth, such bacteria are still antibiotic-sensitive. Balaban et al. investigated the growth dynamics of various mutant and wild-type *Escherichia coli* using a microfluidic device to track individual organisms. At least three different phenotypes were revealed. Those with a normal growth rate were killed. Type I persists exited stationary phase very slowly – hours rather than minutes after nutrients were restored. Type II persists arose by

a spontaneous switch from the normal growth rate to grow consistently more slowly, regardless of growth conditions, and, rarely, could switch back to the normal growth rate. Many pathogens have become resistant to the beta-lactam antibiotics, like penicillin, by a variety of mechanisms, including mutation of penicillin-binding protein genes, destruction of the antibiotic by beta-lactamases, or by inhibition of uptake by the bacterial cells.

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E. Israeli

