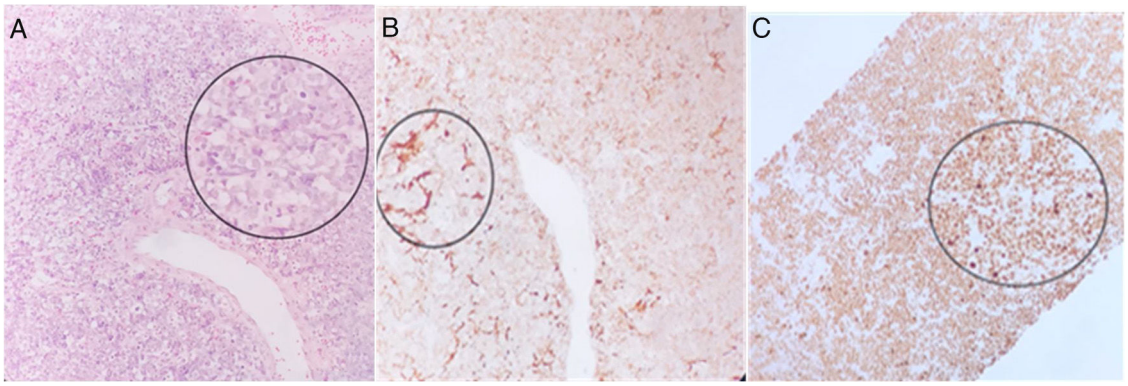


**Figure 2** a) Liver biopsy. Slide stained with H&E at ×40, identifying a lesion with a solid pattern of small, round, blue cells, with monotonous nuclei substituting the liver parenchyma. b) Immunohistochemistry CD20 slide at ×40, identifying an immune reaction in the cells substituting the liver parenchyma. c) Immunohistochemistry Ki-67 slide at ×40, identifying an intense immune reaction in 100% of the cells, consistent with Burkitt's lymphoma. The figure can only be seen in color in the online version of the article.

ethics committee. It contains no personal information that could identify the patient.

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## Conflict of interest

The authors declare that there is no conflict of interest.

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D.S. Morgan[a], A.R. Guzmán[a], B. Barranco[a,*], M. Salamanca[b], A.T. Archundia[c]

[a] *Departamento de Gastroenterología, Centro Médico Nacional 20 de Noviembre ISSSTE, Mexico City, CDMX, Mexico*
[b] *Departamento de Patología Clínica, Centro Médico Nacional 20 de Noviembre ISSSTE, Mexico City, CDMX, Mexico*
[c] *Departamento de Radiología e Imagenología, Centro Médico Nacional 20 de Noviembre ISSSTE, Mexico City, CDMX, Mexico*

*Corresponding author at. Ave. Félix Cuevas 540, Col del Valle Sur, Alcaldía Benito Juárez, CDMX CP 03229, México. Tel.: +5255005003 ext. 14253.
*E-mail address:* betbarranco@yahoo.com.mx (B. Barranco).



# Endoscopic approach to Brunner's gland hamartoma: A case report☆

## Tumor de Brunner, abordaje endoscópico. Reporte de caso

A 45-year-old man, with a past medical history of type 2 diabetes mellitus of 12-year progression, currently with good treatment adherence, and no other remarkable past medical history, was referred to the gastroenterology service because of chronic anemia under study. Hypochromic microcytic anemia due to probable iron deficiency was reported (CBC: Hgb 6.8 mg/dl, Ht: 28.7%, MCV: 57.9 fl, MCH: 13.7%). The patient was asymptomatic at diagnosis. Upper gastrointestinal (GI) endoscopy was ordered for the diagnostic approach to the anemia under study.

Upper GI endoscopy showed a pedunculated polyp in the duodenal bulb, measuring approximately 4 cm in diameter, in the first part of the duodenum, with signs of active bleeding. The decision was made to resect the polyp through the panendoscopy approach. Adrenaline was injected into the base of the polyp and 2 hemoclips were placed facing each other in the pedicle. Hot snare polypectomy was performed with no complications and the specimen was sent for histopathologic study (Fig. 1).

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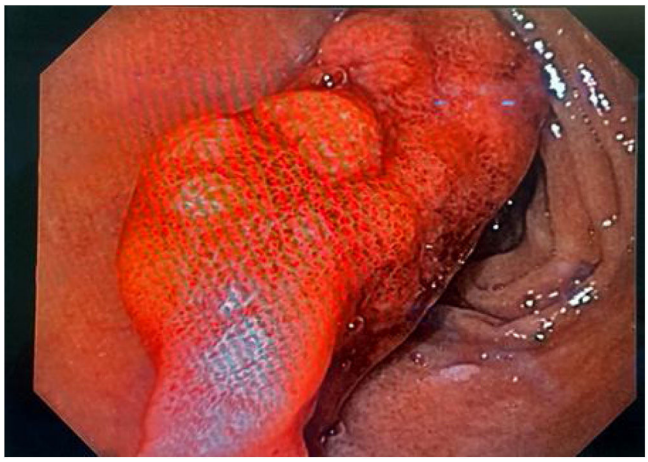

**Figure 1** Upper GI endoscopy, in which a pedunculated polyp, approximately 4 cm in diameter, can be seen in the duodenal bulb in the first part of the duodenum, with signs of active bleeding.

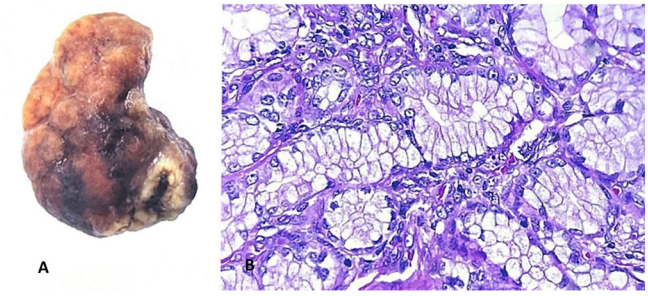


**Figure 2** (A) Macroscopic description: a dark brown duodenal polyp with a wrinkled surface and 4 × 2 × 2 cm nodular formation. (B) Microscopic description: nodular proliferation of Brunner's glands that includes stroma and ducts.

The histopathology report stated a dark brown nodular formation, measuring 4 × 2 × 2 cm, with a wrinkled surface. When cut, its consistency was soft and the interior consisted of light brown uniform tissue and a central area of bleeding, with a nodular proliferation of Brunner's glands that included stroma and ducts. Immunohistochemical staining was positive for Ki67. Given those data, the conclusion was Brunner's gland adenoma, with no signs of atypia (Fig. 2).

Branched acinotubular glands, called Brunner's glands, are found in the mucosal and submucosal layers.[1] The Swiss anatomist, Johan Conrad Brunner, first described them, identifying their main function as that of digestive juice secretion. The incidence of Brunner's gland adenoma predominates in the fifth and sixth decades of life. It is considered an extremely rare tumor, with an incidence below 1% in endoscopic approaches.[2] Brunner's gland hyperplasia tends to be an incidental finding, but a symptomatic presentation can be related to malignant potential.[3]

Its clinical presentation is diverse, with physical examination, clinical imaging studies, or endoscopy resulting in the incidental finding in asymptomatic patients, as well as nonspecific abdominal complaints in symptomatic patients.[4] The typical symptomatic presentation includes iron-deficiency anemia, gastrointestinal bleeding, ampullar obstruction, duodenal obstruction, or intestinal intussusception secondary to the presence of the tumor.[5]

The patient described herein had no apparent signs of upper or lower gastrointestinal bleeding, intestinal occlusion, or signs of intussusception, but did have the incidental finding of iron-deficiency anemia, and so was referred to the gastroenterology service because of the chronic anemia under study.

The pathophysiology of the origin of Brunner's gland hyperplasia has not yet been determined. The main theory is that it is the result of excessive hypersecretion of hydrochloric acid, *Helicobacter pylori* coinfection, or inflammation due to the secretion of alkaline by Brunner's glands.[6] A stool antigen test for *H. pylori* was performed and its result was negative, ruling out an association with said infection.

Polypectomy through the panendoscopy approach is the procedure of choice for small lesions. Laparoscopy or laparotomy are reserved for tumors with sessile characteristics, when upper GI endoscopy cannot be performed or if there are complications, such as patients with hemodynamic instability due to gastrointestinal bleeding.[2]

In the present case, upper GI endoscopy plus hot snare polypectomy was performed, removing the specimen through the snare and endoloop, for its histologic study. The result was nodular proliferation of Brunner's glands that included stroma and ducts. The analysis was completed using immunohistochemistry, with Ki67 positivity confirming the diagnosis.

## Ethical considerations

No informed consent was required, nor approval by an ethics committee, given that no personal data that could identify the patient were published.

## Financial disclosure

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## Conflict of interest

The authors declare that there is no conflict of interest.

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L.O. Olivares[a], P.C. Gómez[a], J.S. Tamayo[a], A.K. Verdiales[b,*], F.Y. Mendoza[b]

[a] *Servicio de Gastroenterología y Endoscopia, Centro de Investigación y Docencias en Ciencias de la Salud, Universidad Autónoma de Sinaloa, Culiacán, Sinaloa, Mexico*

[b] *Centro de Investigación y Docencias en Ciencias de la Salud, Universidad Autónoma de Sinaloa, Culiacán, Sinaloa, Mexico*

* Corresponding author at: Av. Álvaro Obregón, Colonia Tierra Blanca, Código Postal 80030, Culiacán, Sinaloa, México. Tel.: +6871748893.
*E-mail address:* Anakverdiales@gmail.com (A.K. Verdiales).



# Autoimmune pancreatitis in pediatrics, a rare disease☆

## Pancreatitis autoinmune en pediatría, una enfermedad poco frecuente

Autoimmune pancreatitis (AIP) is a systemic fibroinflammatory disease that affects the pancreas and other organs[1]. Prevalence in the pediatric population is low and the most frequent clinical presentation is abdominal pain, obstructive jaundice, and weight loss[2].

We present herein the case of a 15-year-old adolescent girl, with an unremarkable past medical history, who was seen by her family physician for chronic, intermittent, colicky abdominal pain of moderate intensity, located in the right upper quadrant, accompanied by nausea of one-month progression. Ultrasound imaging identified multiple stones, measuring 2–3 mm, that occupied 70% of the gallbladder lumen, for which she was referred to the pediatric surgery department. She underwent laparoscopic cholecystectomy, revealing a fibroinflammatory process with an epiploic mass. The patient presented with postoperative intense abdominal epigastric pain, with vomiting and elevated pancreatic enzymes (amylase 1,006 U/l and lipase 1,271 U/l), and a computed tomography scan showed edema of the pancreas (Balthazar B). Magnetic resonance cholangiography revealed no evidence of stones and endoscopic retrograde cholangiopancreatography showed no evidence of stones in the pancreatic duct. The patient also presented with biloma that merited drainage performed by an interventional radiologist. The patient continued to receive parenteral nutrition and antibiotics, and lastly, was discharged with no apparent complications. One month later, she again arrived at the emergency department due to abdominal pain and vomiting. Laboratory test results revealed elevated pancreatic enzymes (amylase 1,248 U/l and lipase 3671 U/l), and an abdominal computed tomography scan showed pancreatic edema and a single peripancreatic collection (Balthazar D). Given the progression time, a new pancreatitis event was diagnosed. A recurrent pancreatitis approach was started, with a new magnetic resonance cholangiography study showing the single finding of segmental irregularity of the pancreatic duct (Fig. 1). Due to the suspicion of AIP, immunoglobulin subclasses were ordered, resulting in IgG4 levels of 221 mg/dl (normal value: <140 mg/dl) and negative antinuclear antibodies. The patient was started on immunosuppressive therapy with prednisone (prednisone 0.6 mg/kg/day). At follow-up at more than 6 months, she presented with clinical improvement and has developed no other events of pancreatitis.

Recurrent acute pancreatitis is characterized by the presentation of 2 episodes of acute pancreatitis, with complete pain resolution and pancreatic enzyme normalization between the two episodes. Recurrent acute pancreatitis occurs in 15–20% of patients after a first event of the disease, and even though AIP is rare in the pediatric population, it should be considered an etiologic possibility[3].

Two forms of AIP are currently recognized. Type 1 AIP is a multisystemic disease that affects the pancreas (IgG4-related systemic disease) and type 2 AIP appears to be an autoimmune disease that specifically affects the pancreas[4]. In pediatrics, the study of AIP is limited to case series, and so its prevalence is unknown. Our patient presented with abdominal pain, which is found in up to 91% of children with AIP, but she did not have obstructive jaundice, which is reported in 42%[2], signifying that clinical presentation can be heterogeneous. Gallstones are not seen in AIP, and a study that evaluated biliary tract involvement in adults with AIP reported the presence of bile duct stricture and the thickening of the bile duct wall and gallbladder wall as the most frequent findings[5]. Likewise, only 22% of children are likely to have elevated IgG4 levels[2]. That finding guided the diagnosis of our patient, in addition to the magnetic resonance images of the pancreas. She presented with the remission of pain after the administration of the steroid, thus meeting 3 of the criteria for the diagnosis of AIP, according to the International Consensus Diagnostic Criteria for Autoimmune Pancreatitis[6], which includes one or more of the following 5 characteristics:

1. Imaging studies: (a) pancreatic parenchyma (on computed tomography or magnetic resonance imaging) and

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