may be due to a genetic alteration that is triggered during dialysis. An international study suggested that mutations in the GNAS1 gene could promote the genesis of SS.[4] Evolved SHPT and inappropriate treatment may play an important role in the appearance of SS.[3,4] In our patient, the lack of adherence to treatment that led to graft rejection and his refusal to undergo hemodialysis could have triggered the disease.

Facial changes in hyperparathyroidism are exclusively associated with patients who have advanced CKD, and their degree of association depends largely on the severity of the disease and its duration.[1] Our patient developed deformities in his skull and jaw despite being exposed to maximum doses of cinacalcet. Cinacalcet is an extremely expensive drug, and, in patients whose parathyroid glands show nodular hyperplasia and a volume >500 mm^3, it seems to be associated with resistance to treatment,[3] as occurred with our patient.

According to the Kidney Disease Improving Global Outcomes (KDIGO) clinical guidelines, patients who present moderate deterioration of kidney function (G3a) to kidney failure (G5d), with significant secondary hyperparathyroidism that does not respond to medical treatment, should undergo parathyroidectomy.[7] The postponement of parathyroidectomy for years clearly contributed to the appearance of the functional and cosmetic alterations of his face and hands, and to the presence of difficult to control hungry bone syndrome with a prolonged postoperative stay.

Therefore, in those patients with CKD and SHPT who are inadequately treated,[2] parathyroidectomy should be performed without delay, before the onset or signs of SS.

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Gastric Antral Vascular Ectasia Refractory to Endoscopic Treatment☆

Ectasia vascular gástrica antral refractaria a tratamiento endoscópico

Gastric antral vascular ectasia (GAVE) is a rare cause of upper gastrointestinal bleeding. It causes approximately 4% of upper gastrointestinal bleeding not associated with esophageal varices,[1–5] predominantly in women aged 60–70 years.[1–3]

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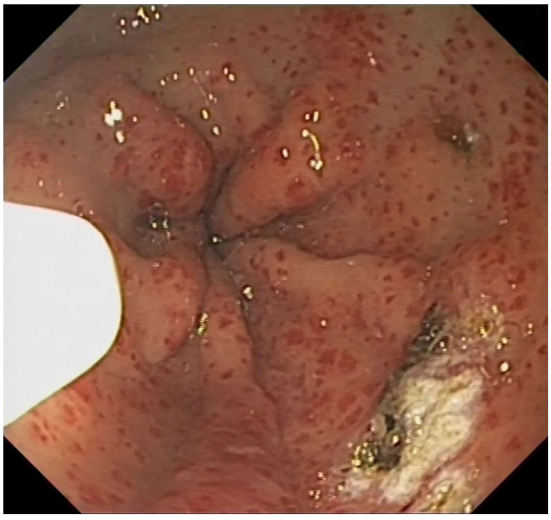

Figure 1 – Endoscopic findings: GAVE.

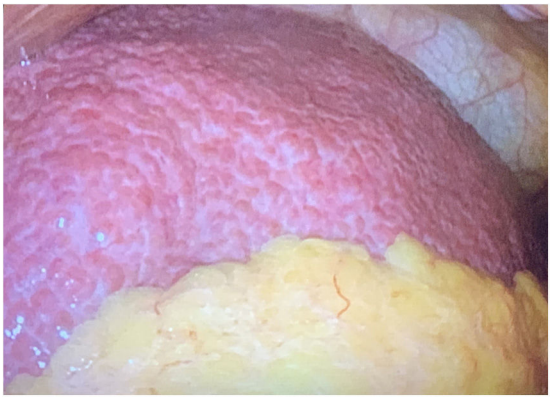

Figure 2 – Intraoperative findings: liver cirrhosis.

It can present as hidden bleeding associated with iron-deficiency anemia or, more rarely, as acute bleeding.[1,2,5,6] Some 60%-70% of patients require periodic blood transfusions despite chronic iron supplementation.[3] The etiology is uncertain, although it is closely related to systemic diseases, such as cirrhosis of the liver (up to 30%).[1–5,7]

The diagnosis is made based on characteristic endoscopic findings, showing 2 well-differentiated GAVE patterns: diffuse stippling, or 'honeycomb stomach'; and linear dots, or 'watermelon stomach'.[1,4,6,7]

It is important to make a correct differential diagnosis with portal hypertensive gastropathy and chronic antral gastritis because their management is very different.[7] Portal hypertensive gastropathy usually affects the fundus and the gastric body. It is more frequent in men with liver cirrhosis, and its treatment (unlike GAVE) is effective with measures that reduce portal pressure.[1,2,5,7] A pathology study is important in these cases because, while not pathognomonic, characteristic findings of GAVE include: vascular ectasia of the capillaries, intraluminal hyaline thrombi and spindle cell proliferation, without signs of inflammation.[1,3,7]

Symptomatic treatment consists of fluid resuscitation and blood transfusions to correct blood loss, either acute or chronic, in addition to appropriate iron supplementation.[1,5] There is not sufficient scientific evidence about medical treatment with estrogen/progestogen, tranexamic acid, thalidomide, or octreotide, so these are only recommended if endoscopic treatment has failed to control blood loss.[1–3,5–7]

The gold-standard treatment options for this disease include endoscopic ablation with YAG laser or argon-plasma coagulation. The latter provides the best benefit–cost ratio and lowest complication rate, although multiple sessions are necessary to reduce bleeding and reliance on transfusions for continued blood loss.[1,2,5–7] Other endoscopic treatments, such as band ligation, have been proposed in recent years, but more studies are needed before providing a definitive conclusion regarding their use.[2,4]

Surgery is an uncommon therapeutic option. Antrectomy should be reserved for refractory cases[4] that, despite medical and endoscopic treatment, present recurrent bleeding and severe anemia.[6] Given the higher morbidity and mortality rates of these patients,[1–3,5,7] surgery is the only curative treatment.[3]

Liver transplantation should be considered in cases of associated liver cirrhosis if the patient is a candidate.

We report the case of a 67-year-old woman with a history of poorly controlled type 2 diabetes, high blood pressure and long-term iron-deficiency anemia treated with high doses of oral iron, who required hospitalization for dyspnea on minimal exertion associated with severe anemia (Hb 7.7 mg/dL). For this reason, lower gastrointestinal endoscopy was conducted, during which colon polyps were observed. Findings from the upper gastrointestinal endoscopy were consistent with chronic antral gastritis.

After diagnosis, significant anemia persisted despite therapy, so outpatient treatment was initiated with intravenous iron, in addition to requiring multiple blood transfusions. For this reason, and due to suspected gastrointestinal blood loss, the study was completed with capsule endoscopy, which revealed GAVE and showed signs of recent bleeding. The diagnosis of 'watermelon stomach' GAVE was confirmed by upper gastrointestinal endoscopy (Fig. 1), and endoscopic treatment was initiated with argon-plasma coagulation for 6 sessions.

Despite the endoscopic treatment and the association of octreotide, as well as an experimental treatment in a hyperbaric chamber, the need for transfusion continued to be high (3–4 units of packed red blood cells per week), and the patient remained highly symptomatic.

As the condition was refractory to treatment, a surgical approach assisted by intraoperative endoscopy was proposed to obtain the most effective resection possible, performing antrectomy with laparoscopic Roux-en-Y reconstruction.

During surgery, we observed a previously unknown micronodular cirrhotic liver (Fig. 2), despite having carried out a preoperative study using thoracoabdominal CT scan, serologies, autoimmunity, tumor markers, laboratory analyses with liver function tests, respiratory function tests and transthoracic echocardiogram, all of which were normal.

The postoperative period progressed favorably, except for poor glycemic control, and the patient was discharged on the eighth day.

In the follow-up visits after 1, 3 and 6 months, the absence of GAVE was confirmed by endoscopy and anemia testing. In

addition, the chronic liver disease study was completed, which provided the diagnosis of Child-Pugh stage A liver cirrhosis, probably caused by steatosis.

The pathological study of the surgical specimen reported the presence of focal ulceration of the mucosa and vascular congestion with fibrin thrombi in the *lamina propria* in the absence of inflammation, all of which were compatible with GAVE.

Our case highlights the importance of a good differential diagnosis with portal hypertensive gastropathy and chronic antral gastritis due to their different management, the frequent association of this disease with liver cirrhosis and the need for surgical treatment in cases that are refractory to endoscopic and pharmacological treatment, since, despite there being no cases reported in the literature, this is the only curative treatment.

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Torsion of a Wandering Spleen☆

Torsión de bazo ectópico

The first detailed description of this clinical entity was by Van Horne in 1667 as an incidental finding during autopsy.[1]

The ectopic spleen is a rare clinical condition characterized by an unusual position of the spleen in the lower abdomen or pelvis. The congenital form may be due to the lack of splenic ligaments or to their incorrect position caused by an abnormally developed dorsal mesogastrium, which generates the suspensory ligaments of the spleen. Cases have also been observed due to progressive splenomegaly caused by diseases such as typhoid fever, lymphoma, and especially malaria.[2]

Acute torsion is the main complication of an ectopic spleen, which is caused by torsion of the vascular pedicle. This leads to splenic infarction due to vascular compromise.[3]

We present the case of a 15-year-old male diagnosed 5 months earlier with an ectopic spleen located in the hypogastrium, who came to the emergency department with abdominal pain that had been progressively intensifying for several days. Initially starting in the hypogastrium, the pain was not alleviated with analgesics and increased with sudden movements. In recent hours, it had spread to the entire abdomen and was accompanied by fever of 38 °C, nausea, little vomiting, asthenia and anorexia.

On physical examination, the patient presented splenomegaly in the hypogastrium and mesogastrium that was immobile, hard in consistency, had smooth edges and was painful on palpation. Laboratory analysis showed a leukocytosis of 12.6×10^9/L, while other values were normal.

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