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SCIENTIFIC LETTERS

Granulomatous gastritis induced by onychophagia: First case report

Gastritis granulomatosa inducida por onicofagia: primer relato de caso

Granulomatous gastritis (GG) is a rare entity that is characterized by the presence of granulomas within the gastric mucosa or submucosa.[1] Common causes of GG include disseminated infections (such as tuberculosis, fungal infections), Crohn's disease, underlying malignancies or foreign bodies.[2] When these conditions have been excluded, the diagnosis of idiopathic granulomatous gastritis (IGG) can be made. Nevertheless, most authors have been able to relate gastric granulomas to a particular etiology and the existence of the idiopathic form has been questioned.[3]

A 27-year-old woman was referred to the Gastroenterology outpatient clinic due to post-prandial vomiting for 10 months with associated weight loss (6 kg). Her past medical history was irrelevant and her usual medication was omeprazole 20 mg/day due to occasional symptoms of epigastric pain with years of evolution. An upper endoscopy did not reveal lesions, but multiple biopsies of the gastric mucosa were taken. Gastric biopsies revealed chronic inflammatory process with formation of lymphoid follicles and evidence of *Helicobacter pylori* infection. Additionally, multiple well-defined and non-necrotizing epithelioid granulomas with multinucleated giant cells were found (Fig. 1A and B). In one of the granulomas it was identified a birefringent material compatible with a foreign body (Fig. 1C and D). No acid-fast bacilli (Ziehl–Neelsen stain) or fungal microorganisms (PAS and PAS-D stains) were observed. No additional microbiological studies were performed. A diagnosis of GG, related to a probable foreign material was made.

After a careful clinical history, the patient reported the habit of biting and ingesting nails since childhood. She denied history of onychomycosis, use of nail polish or other nail beauty item lately. The patient had recently begun her work as a teacher and denied contact with chemicals products in her job or at home. Nevertheless, she underwent

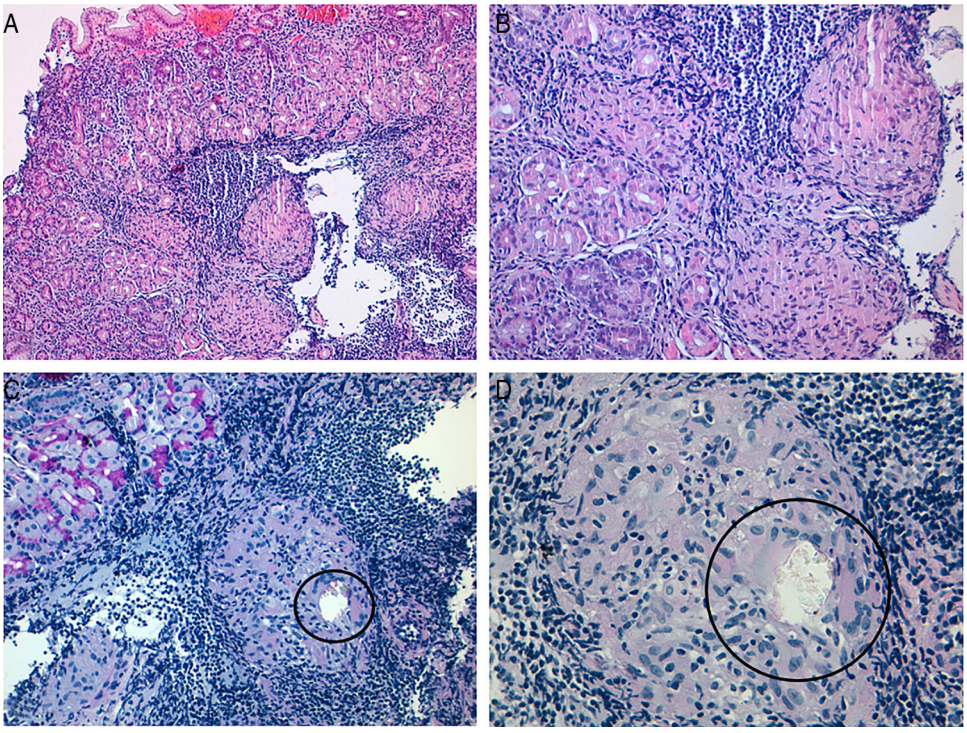

Figure 1 Gastric mucosa with epithelioid granulomas (A – H&E stain, ×100; B – H&E stain, ×200). In the PAS staining a foreign body, highlighted by the circle, was detected in one of the granulomas (C – PAS stain, ×200; D – PAS stain, ×400).

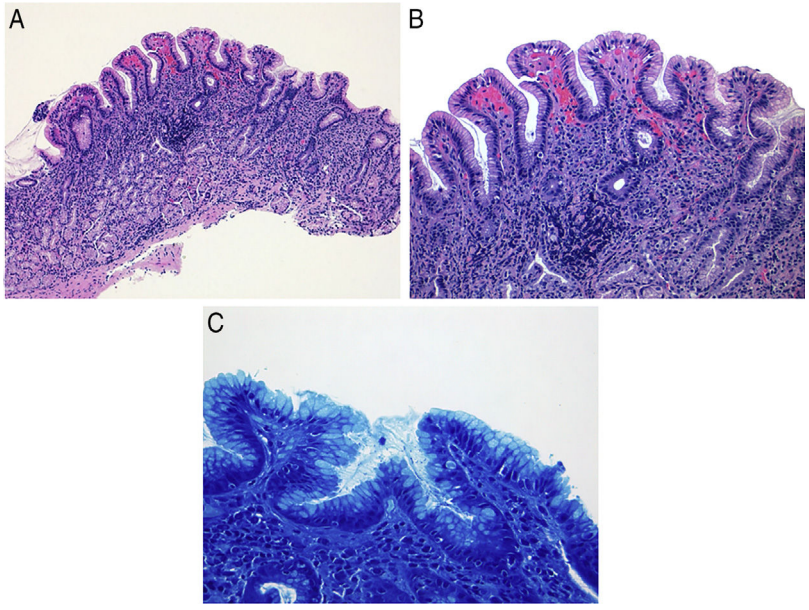

Figure 2 Gastric mucosa with moderate chronic gastritis (A – H&E stain, ×200; B – H&E stain, ×400). *Helicobacter pylori* microorganisms were identified (C – modified Giemsa stain, ×400).

a colonoscopy and videocapsule endoscopy that excluded Crohn's disease. She was referred to the Infectious Diseases outpatient clinic and tuberculosis was excluded with chest X-ray, Mantoux test and QUANTIFERON®-TB Gold test.

After six months of avoiding nail ingestion, the patient became asymptomatic. She underwent a subsequent upper endoscopy. Multiple gastric biopsies were undertaken and disclosed moderate chronic *H. pylori* gastritis but no granulomas or foreign material were seen, confirming resolution of GG (Fig. 2A–C).

Granulomatous gastritis is an uncommon pathological finding, with a reported incidence of 0.08–0.35% in the gastric biopsy samples.[4,5] The most common cause of GG in the western countries is Crohn's disease accounting for approximately half of the cases.[4,6] Other more rare causes include gastric tuberculosis, sarcoidosis, parasitic infections, foreign body reactions, Whipple's disease and adenocarcinoma.[7] Miyamoto et al. also described a possible association with *H. pylori* infection with two reported cases of granulomatous gastritis resolution after successful eradication therapy for *H. pylori*.[8] Nevertheless there is still controversy in relation to the role of *H. pylori* as a causative factor in GG, with larger studies unable to conclusively associate *H. pylori* infection with granuloma formation.[4,9] In our case, since the patient presented with disabling symptoms and weight loss our priority was to exclude Crohn's disease and tuberculosis. The presence of foreign material in the biopsies, granulomatous gastritis and habit of nail ingestion suggested this association. Since the role of *H. pylori* in the development of GG is still controversial, we decided not to eradicate *H. pylori* and evaluate the clinical and histological response to suspension of nail ingestion. In our patient, it is likely that the symptoms of epigastric pain with years of evolution may be related to the habit of nails ingestion. The continuous exposure of the gastric mucosa to a foreign body material probably lead to the development of a granulomatous process with subsequent symptomatic aggravation. Given that the patient became asymptomatic and there was an evident histological improvement, we can conclude that nails ingestion (onychophagia) was the cause of GG in this case.

Conflicts of interest

No conflicts of interest to declare.

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Lesiones psoriasiformes: forma infrecuente de presentación de glucagonoma

Psoriasiform lesions: Uncommon presentation of glucagonoma

El glucagonoma es un tumor inusual, que se origina en las células alfa de los islotes del cuerpo y cola del páncreas. Aproximadamente el 70% están asociados al síndrome del glucagonoma, que se caracteriza por la presencia de eritema necrolítico migratorio (ENM), diabetes mellitus, pérdida de peso, anemia, diarrea, alteraciones neuropsiquiátricas y fenómenos tromboembólicos[1]. El ENM es una dermatosis poco frecuente, que se caracteriza por la presencia de pápulas eritematosas, pruriginosas y dolorosas en periné y áreas intertriginosas. Se agrupan formando placas, alrededor de una ampolla central. El 90% de los ENM aparecen asociados a un glucagonoma[2]. En ocasiones las lesiones cutáneas adoptan un aspecto psoriasiforme, debiendo realizar el diagnóstico diferencial entre psoriasis y ENM ante una psoriasis extensa que no mejora con tratamiento mantenido.

Presentamos el caso de un varón de 52 años, que es estudiado por dermatología por presentar lesiones cutáneas. Es diagnosticado de psoriasis vulgar, pero con el paso del tiempo presenta nuevas lesiones consistentes en placas eritematosas, erosivo-costrosas en miembros inferiores (sobre todo por debajo de las rodillas), durante 3 años en tratamiento sin presentar mejoría. Con la sospecha de que dichas lesiones se correspondieran con un ENM, se solicitó biopsia de piel que informó de dermatitis psoriasiforme con paraqueratosis compacta y pústulas subcorneales, morfológicamente consistentes, con diagnóstico de ENM en fase inicial (figs. 1 y 2). Se realizó una TC abdominal, encontrando una masa de 3,2 cm en el cuerpo del páncreas con adenopatías en tronco celíaco de tamaño significativo y múltiples metástasis hepáticas bilobares. Analíticamente presentó niveles de glucemia normales, hemoglobina de 12,3 g/dl (13,5-17,5), hematocrito del 36% (41-53%), lutropina 12,5 mUI/ml (1,5-9,3), folitropina 5,9 mUI/ml (1,5-12,4), cortisol 14,3 μg/dl (6,2-19,4), somatotropina 1,3 ng/ml (< 10,0), adrenocorticotropina 87,8 pg/ml (< 46,0), enolasa 29 ng/ml (< 16,0), beta-hCG 69 mUI/Ml (< 3,0), antígeno carcinoembrionario 1,4 ng/Ml (< 5,0). Se completa el estudio con OctreoScan® (fig. 3), que muestra una gran masa a nivel del cuerpo pancreático que mide aproximadamente 3 cm, compatible con la existencia de un glucagonoma, además se observan numerosos depósitos patológicos del trazador a nivel hepático, bilobares. Con el diagnóstico de glucagonoma se decidió intervenir quirúrgicamente al paciente, realizando una pancreatectomía córporo-caudal con linfadenectomía. El informe anatomopatológico, concluye que estamos ante un tumor neuroendocrino bien diferenciado G1, compatible con glucagonoma, de cuerpo de páncreas, pT3 pN1. Tras la cirugía, el paciente evolucionó favorablemente, desapareciendo por completo las lesiones en la piel, a los 15 días de la intervención (fig. 4). Actualmente el paciente se encuentra en tratamiento con lanreótido y está pendiente de valoración para transplante hepático.

El 2% de los tumores digestivos son tumores neuroendocrinos de páncreas, de ellos el glucagonoma representa el 4%[3]. Es más frecuente en mujeres a partir de los 45 años. Aproximadamente el 50% de los pacientes diagnosticados con estos tumores, presentan clínica con la sintomatología y signos relacionados con la actividad biológica de las hormonas secretadas por la neoplasia. El 70-80% de los pacientes con glucagonoma presentan la siguiente tríada: diabetes, ENM y anemia. El glucagonoma, como otros tumores neuroendocrinos, expresa receptores de somatostatina en más del 80% de los casos.

La detección de los glucagonomas se puede realizar mediante TC, RMN o ecografía[4]. Al expresar los receptores anteriormente descritos, la gammagrafía con análogos de la somatostatina se utiliza para demostrar la presencia de la neoplasia y su diseminación en los casos en los que esta existe.

El crecimiento de estos tumores es lento, presentando una alta tasa de supervivencia que supera el 50% a los

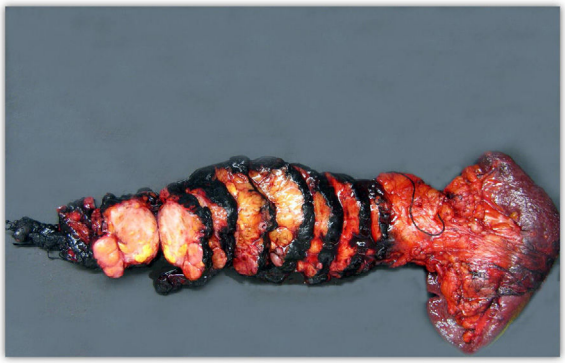

Figura 1 Resección corporocaudal de páncreas y esplenectomía. Se aprecia una lesión blanquecina con áreas amarillentas, firme, de 4,7 cm, en el cuerpo del páncreas.