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SCIENTIFIC LETTERS

Emphysematous gastritis: Effectiveness of early antibiotic therapy☆

Gastritis enfisematosa, eficacia del tratamiento con antibioterapia precoz

The presence of air in regions of the abdomen may be due to 2 diseases: gastric emphysema and emphysematous gastritis, which differ in aetiology, clinical presentation, evolution and prognosis.[1,2]

Gastric emphysema was described by Brouardel in 1985 as a generally benign disease caused by disruption of the gastric mucosa and subsequent infiltration of the mucosa by air. It is usually a self-limiting disease and resolves without sequelae.[2]

Emphysematous gastritis is a rare and severe gas-forming infection in the gastric wall. It is caused by the disruption of the mucosa and subsequent invasion of the gastric wall by gas-forming bacteria. This results in acute suppurative inflammation of the wall that compromises the submucosa and muscle layer, with the formation of abscesses, necrosis and marked leucocyte infiltration.[3] Incidence has increased, associated with immunodeficiency syndromes. Aetiology may differ, but a common factor in all cases is increased intragastric pressure caused by, for example, nasogastric intubation, hyperemesis, acute pancreatitis, duodenal obstruction, use of non-steroidal anti-inflammatory drugs, peptic ulcer, alcoholism, acids and alkalis, high dose dexamethasone therapy, instrumentation of the upper gastrointestinal tract, general anaesthesia, and cardiac resuscitation. It may also be associated with recent surgery, trauma, haemodialysis, malignant neoplasms, extensive burns, and gastric infarction.[4]

The diagnostic method of choice is computed tomography (CT), while the preferred treatment is antibiotic therapy and surgery when medical treatment fails. Due to its initial non-specific symptoms and rare occurrence, diagnosis is usually late and the disease often progresses to severe peritonitis with a very poor prognosis.

We present the case of a 75-year-old woman with a history of high blood pressure, non-insulin dependent diabetes mellitus, rheumatic valvular heart disease with mitral valve replacement by a metal stent and tricuspid ring, and moderate/severe aortic stenosis. She reported a 4-month history of diarrhoea, which in the last month was accompanied by vomiting after meals. Five days previously, she presented a new episode of diarrhoea without fever, accompanied by intense epigastric pain irradiating to the back, associated with vomiting and general malaise. In the emergency department, the patient remained hypotensive, with metabolic acidosis. An urgent abdominal CT scan without contrast revealed emphysematous gastritis and gas in the intrahepatic bile duct (Figs. 1 and 2). Fibre-optic endoscopy showed haemorrhagic gastritis. The patient was admitted to the intensive care unit (ICU) and started on treatment with metronizadole and piperacillin-tazobactam, proton pump inhibitors, fluid therapy and total parenteral nutrition. In the ICU, the patient remained haemodynamically stable and did not require vasoactive support. She presented progressive mixed inflammatory/iron deficiency anaemia, for which she received treatment with intravenous iron and vitamin B complex. On the fourth day of admission, right basal consolidation was observed on the chest radiograph. Epigastric and right upper quadrant pain gradually subsided during her stay in the ICU. A follow-up CT scan on the sixth day of admission showed that the cystic pneumatosis of the gastric wall had resolved, but gas remained in the hepatic portal system. Results of blood cultures, urine cultures and sputum culture taken from the patient on admission were negative, while pathological study of the gastric biopsy found histological changes on the base of the ulcer with a small number of bacilli consistent with *Helicobacter pylori*. Given the patient's good progress (and once she was able to tolerate food and liquid by mouth), she was discharged to internal medicine on the 12th day of admission.

This entity was first described by Galen, who named it ''erysipelatous tumour of the stomach''.[5] Decreased acidity or presence of lesions in the gastric mucosa could permit bacterial colonization of the gastric wall. Another mechanism involves dissemination of the bacteria through the blood from a distant septic focus.[5,6] However, the port of entry is often unknown,[6] as in our patient. Gastric mucosa involvement can be superficial gastritis, necrotic gastritis, pangastritis or emphysematous gastritis. The latter is serious and the least common, and is characterized by the

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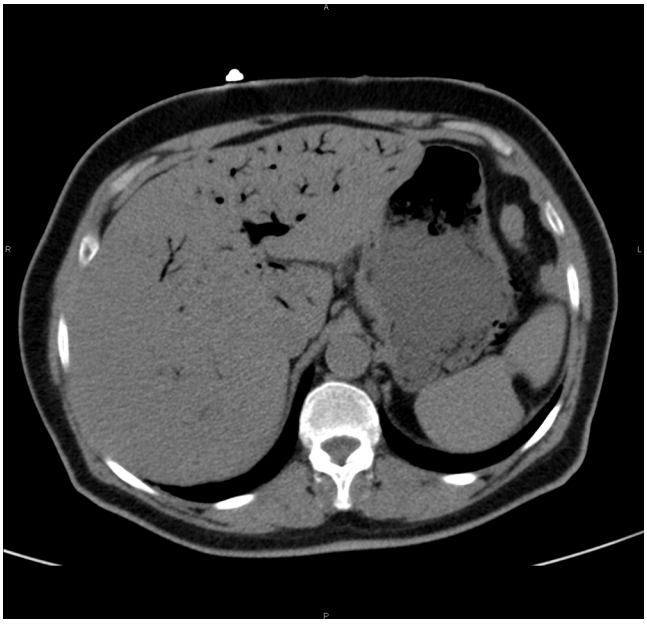

Figure 1 Abdominal CT scan without contrast. Axial slice: air bubbles in the gastric wall and diffuse wall thickening. Abundant gas in the liver parenchyma and in the portal system.

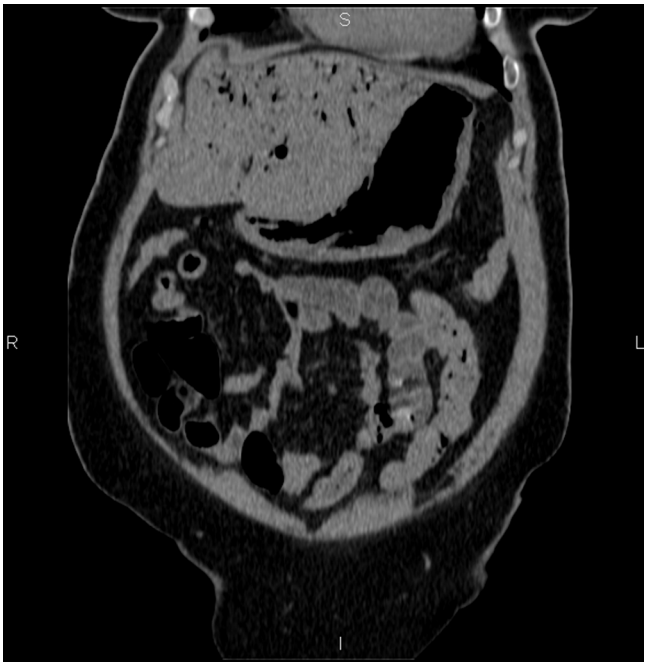

Figure 2 Abdominal CT scan without contrast. Coronal reconstruction: emphysematous gastritis and portal system.

formation of abscesses in the gastric wall (especially in the submucosa), with a tendency to open into the gastric cavity and form gastric cellulitis that can spread to the peritoneal cavity, producing secondary peritonitis. There may be associated spontaneous pneumoperitoneum and air in the portal vein and its intrahepatic lobar or segmented branches. The existence of gas in the portal territory was previously an indication for primary surgery, but due to poor outcomes, early antibiotic treatment is currently preferred[2]; the case of our patient is an example of the presence of gas in the portal territory and favourable progress with antibiotic treatment without requiring surgery.

The microorganisms most frequently isolated are group A β-haemolytic streptococcus (*Streptococcus pyogenes*), staphylococcus (*Staphylococcus aureus*) and *Escherichia coli*. Other pathogens found are *Enterobacter cloacae*, *Pseudomonas aeruginosa*, *Clostridium perfringens* and *welchii* and *Klebsiella pneumoniae*.[1,5,6]

Initial symptoms are very non-specific: abdominal pain, diarrhoea, nausea, vomiting and fever, so early diagnosis is very difficult. In some cases, the clinical presentation is septic shock, although in other cases–like our patient–the presentation is sepsis. Diagnosis is by plain abdominal X-ray and/or abdominal CT scan. In addition to detecting the presence of gas in the abdominal wall, CT can also reveal gastric wall thickening.[5] Less often, diagnosis is made by upper gastrointestinal fibre-optic endoscopy with biopsy sampling.

Current treatment should include antibiotics that cover Gram negative, Gram positive and anaerobic bacteria. Gastric acid secretion inhibitors, fluid therapy and parenteral nutrition should also be added.[7] Surgery should be considered in the event that medical treatment fails or complications such as gastric perforation arise. Prompt diagnosis followed by appropriate antibiotic treatment improves the prognosis. Ideally, these patients should be monitored in ICUs. According to published case series, mortality is greater than 60%.[8,9]

If the patient progresses well, healing occurs with hyperplasia of the conjunctive tissue and residual fibrosis, or chronic cirrhotic gastritis that resembles Brinton linitis plastica in appearance and general characteristics.[3]

In conclusion, phlegmonous gastritis is a rare, rapidly progressive and potentially fatal bacterial infection of the gastric wall; in some cases, such as our patient, early antibiotic treatment can be curative, thus improving survival rates.

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Pancreatic metastasis from a Merkel cell carcinoma diagnosed by ultrasound-guided biopsy☆

Metástasis pancreática de carcinoma de células de Merkel diagnosticada mediante biopsia guiada por ecoendoscopia

Merkel cell carcinoma, also known as trabecular carcinoma of the skin, cutaneous APUDoma, primary small cell carcinoma of the skin or cutaneous neuroendocrine carcinoma, is a rare, aggressive skin cancer with a marked tendency towards locoregional and distant recurrence. Few cases of pancreatic metastases from Merkel cell carcinoma have been reported in the literature. We present the case of a 75-year-old man who had been diagnosed and treated for localised Merkel cell carcinoma with pancreatic recurrence.

The patient had a history of high blood pressure and hypercholesterolaemia, and had been diagnosed with Merkel cell carcinoma localised in the right forearm that was treated with extensive surgical resection. Six months later, he presented local recurrence, for which he received treatment with surgery and adjuvant radiotherapy. A follow-up computed tomography (CT) scan performed 6 months later revealed right axillary lymphadenopathy and flattening of the D11 vertebral body, as well as a new 38-mm solid lesion at the level of the pancreatic tail. The patient was asymptomatic, and laboratory tests showed no alteration in liver function tests or pancreatic enzymes. In view of these findings, endoscopic ultrasound was requested, in which a 47-mm hypoechogenic mass with poorly defined borders was detected in the pancreatic tail and fine needle biopsy was performed with a 22G ProCore biopsy needle (Cook Endoscopy Inc., Limerick, Ireland) (Fig. 1). The histopathology study was consistent with Merkel cell carcinoma, with expression of neuroendocrine markers (synaptophysin and chromogranin) and positive staining for cytokeratin 20 (CK20) (Fig. 2). After confirming disease progression, aspiration of the axillary lymphadenopathies was considered unnecessary and the patient commenced palliative chemotherapy with carboplatin-etoposide.

Merkel cell carcinoma originates in cells of the same name located in the epidermal basal layer, where they perform a mechanoreceptor function. Recent studies have shown that these tumour cells express epithelial markers such as CK20, which points to an origin in the epithelium rather than the neural crest, as previously thought.[1] It mainly affects Caucasian men older than 70 years,[2] and incidence is increasing due to population ageing. Onset has been related with exposure to both natural and artificial ultraviolet radiation, and also with immunosuppression, especially in HIV-positive patients,[3] solid organ transplant recipients[4] and patients with chronic lymphocytic leukaemia.[5] Merkel cell polyomavirus—a double-stranded DNA virus detected in up to 80% of cases—has recently been implicated in the pathogenesis of this tumour, although the prognostic implications of this finding are controversial.[1]

This type of carcinoma is most often found in sun-exposed areas, especially the head, neck and extremities, manifesting as a single, rapidly growing painless bluish-red nodule.[6] Almost half of patients (49%) present localised disease on diagnosis, and the presence of metastasis at presentation is rare (around 8%). However, the tumour is usually aggressive, with a 10-year survival of 57%.[2] It has a marked tendency towards local recurrence. Similarly, almost half of patients will develop distant metastases during the course of their disease, mainly bone and liver.[2] The occurrence of pancreatic metastases in Merkel cell carcinoma is exceptional, with only 14 cases having been published to date,[7–9] 4 of which were diagnosed by endoscopic ultrasound-guided fine needle aspiration cytology.[9–11]

Treatment is usually extensive resection of the lesion in localised forms, combined with adjuvant radiotherapy if the resection is incomplete or there is a high risk of local recurrence. The current tendency is to biopsy the sentinel lymph node, and to perform dissection if lymph nodes are involved.[12] When distant disease appears, the prognosis worsens drastically, with a mean survival of 9 months. Chemotherapy regimens similar to those used in small cell lung carcinoma (with etoposide, cisplatin, vincristin, doxorubicin or cyclophosphamide) can be combined with radiotherapy or palliative surgery, although none of these strategies significantly increase survival.[12]

In the case presented, the distant disease in the pancreas appeared 1 year after the initial diagnosis, and the endoscopic ultrasound-guided biopsy was key in establishing the differential diagnosis with a possible primary tumour of the pancreatic gland. The therapeutic management of the patient was thus modified, avoiding unnecessary surgical resection. It should be borne in mind that metastases account for only 2% of all malignant lesions detected in the pancreas.[13] Furthermore, this is the first case of pancreatic metastasis from Merkel cell carcinoma diagnosed by endoscopic ultrasound and fine needle aspiration histocytology. The amount of material and type of sample that

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