The most common form of NCPH is regenerative nodular hyperplasia, reported above all in association with systemic diseases or use of medicines, and identified by regenerative nodules in the absence of fibrosis.[2,3] However, incomplete septal cirrhosis represents a much more unusual pathological finding for which there are hardly any references.[1,4,5]

Incomplete septal cirrhosis is characterised by a vague nodularity with thin incomplete fibrous tracts surrounding nodules of regeneration, hypoplastic portal spaces with no inflammation, enlarged efferent veins and sinusoidal dilatation.[5] Sometimes a differential diagnosis with other entities is nearly impossible, especially in fine-needle biopsies, and may only be suggested. It should be noted that its typical characteristics may be missing or incomplete in a simple biopsy and its recognition may be underestimated; therefore, a sample obtained through laparoscopy proves much more useful.

Overlapping traits of this and other forms of NCPH have been reported. Nakanuma et al. re-evaluated 107 liver biopsies with NCPH in Japan, and classic characteristics of incomplete septal cirrhosis were reported in just 25 cases, which in addition presented other traits shared with partial nodular transformation. This raised the question of whether these could be different stages of the same disease.[6] In the same vein, our patient's biopsy also showed areas of regenerative nodular hyperplasia. This, too, raised the possibility of diagnosis of progressive forms of the same entity. The co-existence in a single biopsy of regenerative nodular hyperplasia and incomplete septal cirrhosis has been previously reported in the literature in a patient with autoimmune hepatitis treated with azathioprine. The authors believe that incomplete septal cirrhosis could be the result of regression of fibrosis following hepatic aggression.[7] Recently a case was published of septal cirrhosis in a patient with multiple sclerosis treated with corticosteroids, which reversed after treatment was stopped. This supports the notion that this entity represents a dynamic, not necessarily progressive process, if not the notion that in some patients it is postulated as a stage of regression of fibrosis.[4]

In conclusion, our case featured the uncommon finding of 2 different histological entities—regenerative nodular hyperplasia and incomplete septal cirrhosis—in a patient with NCPH. This could bolster the idea that these are in reality different phases of the same disease, related to the process of progression or involution of hepatic fibrosis.

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# Eosinophilic ascites: A case report☆

## Ascitis eosinofílica: a propósito de un caso

A 35-year-old man with a history of atopic rhinitis was admitted due to hypogastric abdominal pain for the past 20 days and gradual abdominal distension. He had no associated diarrhoea or other symptoms. He denied having consumed alcohol or other toxic substances. On physical examination he was found to be afebrile with a distended abdomen and dullness on both flanks, suggestive of ascites. Blood testing revealed: leukocytes 9930/μl, eosinophils 29.9% and no immature forms of myeloid cells. The rest of the complete blood count, amylase, the liver panel, total proteins, albumin, TSH and clotting were normal. A chest X-ray was also normal. An abdominal ultrasound with Doppler showed abundant free peritoneal fluid. The liver, splenoportal axis, pancreas and spleen as well as the rest of the examination were normal. An abdominal computed tomography (CT) scan showed a moderate amount of ascites and thickening of the abdominal mesenteric fat suggestive of oedema (Fig. 1). Diagnostic paracentesis revealed: cytology

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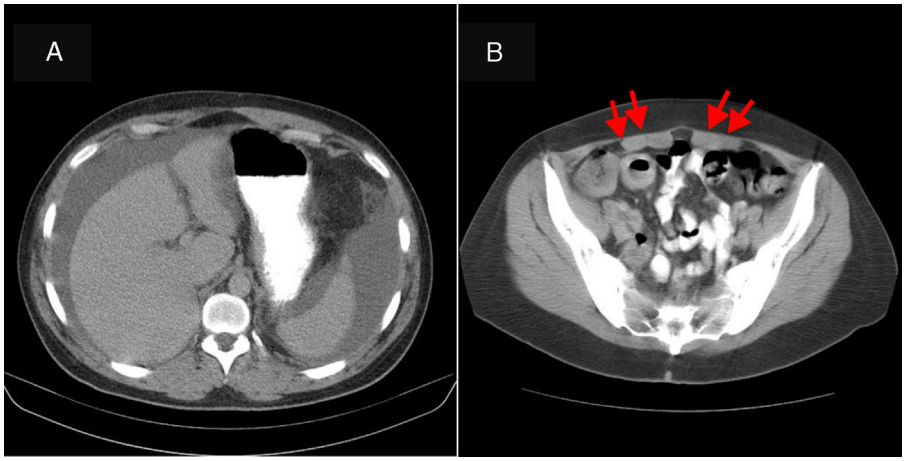


**Figure 1** Computed tomography (CT) scan of the abdomen. (A) Moderate amount of ascites in perihepatic spaces, perisplenic spaces, paracolic gutters and pelvis. Homogeneous liver with regular borders. (B) Rarefaction of mesenteric fat suggestive of mesenteric oedema (arrows).

study with 95% eosinophils, with no evidence of malignant cells; adenosine deaminase (ADA) <40 U/l; culture negative; seroalbumin gradient <1. A study of the usual parasites in faeces came back negative in 3 determinations. The patient did not report having travelled to any area endemic for parasitic infections. As eosinophilic gastroenteritis (EGE) was suspected, gastroscopy was ordered. This showed a normal oesophagus, stomach and duodenum (biopsies by normal segments) as well as normal intestinal transit. A PET-CT scan performed to screen for tumour disease ruled out metabolic abnormalities consistent with peritoneal carcinomatosis. It showed several lymph node formations of non-pathological size and metabolism in a mesentery with a reactive appearance. The study was completed with the following peripheral blood determinations: adenosine deaminase (ADA) normal; C1-inhibitor normal; *Anisakis* IgE negative; serology for human immunodeficiency virus (HIV) and hepatotropic viruses negative; anti-smooth muscle antibodies (ASMAs) 1/640; antineutrophil cytoplasmic antibodies (ANCAs), antimitochondrial antibodies, antinuclear antibodies and anti-transglutaminase antibodies negative; and serum tryptase negative. A Mantoux test came back negative. With a suspected diagnosis of eosinophilic ascites and other causes of eosinophilia having been ruled out, treatment was started with 25 mg of prednisone. A decrease in the patient's ascites was verified within a week. After 2 months, the patient's symptoms had completely disappeared.

EGE is an uncommon entity characterised by infiltration of eosinophils in any segment of the gastrointestinal wall, capable of affecting any layer, in the absence of other causes of eosinophilia.[1]

There are 3 different disease types depending on the layer of the gastrointestinal wall that is affected. Mucosal impairment is the most common type (70%). It manifests as iron-deficiency anaemia, enteropathy, protein loss or malabsorption. Predominately muscular impairment (20%) is characterised by localised or diffuse thickening of the abdominal wall and is capable of causing obstructive symptoms. Finally, subserous impairment (10%) features ascites rich in eosinophils or, in severe cases, peritonitis, perforation or intestinal intussusception.[2] Other less common forms of presentation are obstructive jaundice due to biliary tract impairment and extraintestinal manifestations such as eosinophilic cystitis, eosinophilic splenitis and eosinophilic hepatitis.

Talley et al. identified 3 diagnostic criteria for EGE: (1) gastrointestinal symptoms; (2) gastrointestinal tract biopsies showing eosinophilic infiltration or radiological characteristics with peripheral eosinophilia or ascites rich in eosinophils; and (3) absence of parasitic or extraintestinal disease.[3] Although peripheral eosinophilia is present in most patients, it is present at even higher rates in subserous forms. It may be absent in 30% of cases.

Among patients with EGE, 50% have a concomitant allergic disorder (asthma, rhinitis or a food or drug intolerance).[4] This suggests that EGE may result from immune deregulation in response to an allergic reaction. Parasitic infestation and drugs may also act as triggers.

Diagnosis of subserous EGE may be complicated due to its rarity and the non-specific nature of its signs and symptoms. The most common symptoms are: abdominal pain (90.45%), nausea and vomiting (57.1%), diarrhoea (52.3%), and abdominal distension (38.1%).[5] Eosinophilic ascites should be suspected in all patients with abdominal pain, ascites and peripheral hypereosinophilia.[6,7] The key to diagnosis is confirmation of ascitic fluid rich in eosinophils. Diagnosis requires ruling out parasitic diseases, abdominal tuberculosis, rupture of a hydatid cyst, chronic pancreatitis, vasculitis (Churg–Strauss), hypereosinophilic syndrome, malignancy and Crohn's disease.[8]

The overall prognosis for eosinophilic ascites is good, with an excellent response to oral steroids as a first line of treatment. It is advisable to start with prednisone 20–40 mg per day. This achieves remission of symptoms in 80% of patients within a week and returns eosinophil counts to normal within 2 weeks. However, recurrence may be seen in 50% of patients when treatment is suspended.[1] Surgery is reserved for the few cases with obstructive complications.

As eosinophilic ascites represents the most unusual variety of EGE, and due to the non-specific nature of its symptoms and the potential absence of both peripheral

eosinophilia and infiltration of eosinophils in the gastrointestinal wall, diagnosis represents quite a challenge for the gastroenterologist and requires a high degree of suspicion.

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