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CLINICAL COMMUNICATIONS

Abdominal pain, *pneumatosis intestinalis* and *aeroportia* in a hemodialyzed patient

Dolor abdominal, *pneumatosis intestinalis* y *aeroportia* en un paciente en hemodiálisis

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Hemodialyzed patients frequently experience various symptoms intrinsic to the dialysis session, some reflecting acute complications of the procedure. Hypotension is the most common complication, affecting 25–55% of patients,¹ and is not infrequently accompanied by nausea, vomiting, headache, and chest and/or abdominal pain. We present a 51-year-old man with end-stage renal disease receiving hemodialysis, that presented with abdominal pain due to non-occlusive mesenteric ischemia, with *aeroportia* and *pneumatosis intestinalis*, unusual findings, but increasingly found in dialysis patients.

Case report

A 51-year-old man with diabetic nephropathy on hemodialysis for the past four years presented to our emergency department because of sudden onset of excruciating abdominal pain, with bilateral dorsal irradiation, nausea and vomiting. He was otherwise asymptomatic until one hour after beginning his regular hemodialysis session, when the

pain suddenly began. The dialysis session was interrupted and he was brought to our hospital. Physical examination revealed arterial hypotension (blood pressure 80/64 mmHg), tachycardia (130/min), painful abdominal palpation with signs of peritoneal irritation, and absence of bowel sounds. The patient was afebrile and had a normal pulmonary auscultation. The rectal touch was negative for bloody stools or melena.

The results of laboratory examinations were unremarkable, except for a mildly elevated C-reactive protein (3.43 mg/dL) and elevations in plasma creatinine and urea (6.3 mg/dL and 61 mg/dL, respectively). Both chest X-ray and plain radiography of the abdomen were normal. The electrocardiogram revealed sinus tachycardia, and no signs of ischemic myocardial disease. A computed tomography revealed large amounts of air within the portal venous system along its extension, from the colic veins to the distal branches of the intra-hepatic portal vein (Fig. 1). We also found areas of *pneumatosis intestinalis* in terminal ileum and ascending colon, suggesting intestinal ischemia (Fig. 2). Computed tomography multiplan and volumetric reconstructions of mesenteric arteries were performed, and were negative for significant local atherosclerotic disease or thrombosis, suggesting that the pathophysiological pro-

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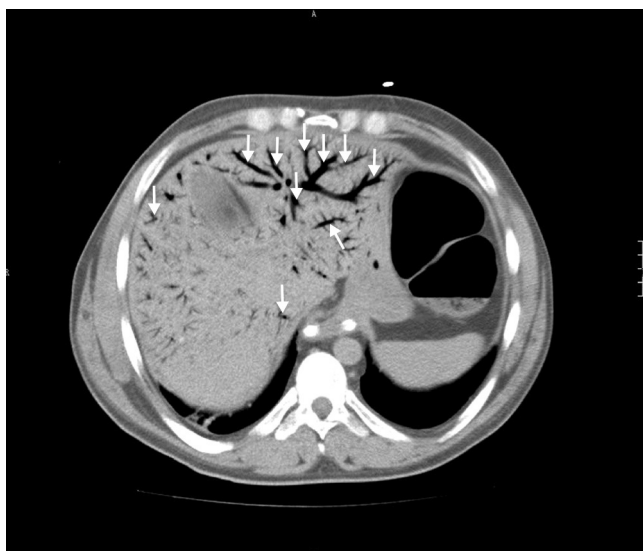


Figure 1 Abdominal contrast-enhanced computed tomography showing large areas of hepatic portal venous gas – *aerportia* (white arrows).

cess underlying the intestinal ischemia was non-occlusive intestinal hypoperfusion (probably secondary to intradialytic hypotension). The patient was hemodynamically stabilized. An exploratory laparotomy evidenced necrosis of ileum, ascending, transverse and descending colon. A diagnosis of mesenteric ischemia and necrosis was established. Subtotal colectomy was performed with removal of all colonic segments and the distal 20 cm of small intestine. There were no signs of perforation or peritonitis. The surgery had no complications and the hospitalization was held with no intercurrents.



Figure 2 Abdominal contrast-enhanced computed tomography showing *pneumatosis intestinalis* in ascending colon and terminal ileum (white arrows).

Discussion

The presence of *pneumatosis intestinalis* is a rare finding in clinical medicine, and gas accumulation in portomesenteric vessels, or *aerportia*, is an even rarer finding. They are both consequence of serious conditions and carry high mortality.² The primary cause is mesenteric ischemia, accounting for about 70% of all cases. Other causes of *pneumatosis intestinalis* and *aerportia* are ulcerative colitis, gastric ulcers, diverticulitis, acute pancreatitis and following invasive procedures.³ *Pneumatosis intestinalis* can also arise in the context of less serious conditions like asthma or emphysema.

In patients on chronic hemodialysis there have been some case reports of *pneumatosis intestinalis* with *aerportia*.^{4–6} In these patients the pathophysiological mechanism is prolonged central hypoperfusion during dialysis, leading to non-occlusive intestinal necrosis. In addition to this, hemodialyzed patients frequently have membrane changes in enterocytes, with greater predisposition to bacterial translocation into bloodstream.⁵ In this particular group of patients the microbial flora is heterogeneous, comprising not only aerobic agents, but also anaerobic agents.⁵ Other factors contributing to intestinal ischemia in the hemodialyzed patient appear to be the early and aggressive erythropoietin therapy and vascular calcifications in mesenteric vessels, resulting from deregulated phosphocalcic metabolism.⁷

In those patients in which mesenteric ischemia coexists with *aerportia*, the clinician should expect probabilities of transmural necrosis and mortality of, respectively, 91 and 75%,⁸ reason why the clinical suspicion, early diagnosis and precocious treatment are fundamental. Nonocclusive mesenteric ischemia is thus a recognized and often lethal complication in hemodialysis patients, and its frequency is increasing in this group of patients,⁹ being about 1.9% per patient-year, compared to 0.09–0.2% per patient-year in the general population.^{10,11}

Portal venous gas and *pneumatosis intestinalis* are rare entities, with increasing incidence in hemodialyzed patients. The pathogenic pathways and risk factors are known, and are almost always preventable with simple measures such as avoiding prolonged periods of intradialytic hypotension and controlling inter-dialytic weight gain. They often cause significant morbidity and mortality, and should always be suspected in patients suffering abdominal pain during or after dialysis sessions, since prompt diagnosis and treatment are key factors for a good outcome.

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