Mesenteric artery syndrome associated with HAART onset in a patient with HIV

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DESCRIPTION
A 28-year-old man (body mass index (BMI) of 15.6 kg/m²) with untreated HIV infection since 2004 was admitted to the hospital with a 3-month headache, generalised weakness and neurocognitive deficit. He underwent head MRI and a ring-enhancing lesion was found in subcortical temporal-occipital location. Cerebral biopsy revealed primary central nervous system lymphoma. The patient received chemotherapy with cytarabine, rituximab and high-dose methotrexate without complications. Simultaneously, he was started on antiretroviral therapy (ritonavir, darunavir, raltegravir and tenofovir). Two weeks after highly active antiretroviral therapy (HAART) onset, he developed sudden abdominal pain, distention and profuse biliary vomit. CT angiography revealed severe dilation of the stomach and duodenum until its third portion with a transition zone at the level of the superior mesenteric artery (SMA) (figure 1).

An aortomesenteric angle of 13° and a distance of 5 mm between the aorta and SMA were measured with evident compression of the duodenum between such structures noticed. Initially, a nasogastric catheter was placed in order to relieve the obstruction and total parenteral nutrition was started. Two days later, he underwent upper endoscopy and a nasojejunal catheter was advanced to start enteral nutrition. While on enteral nutrition we observed gradual weight increase, and after reaching BMI of 16.8 he was able to start a soft diet with posterior removal of the catheter (figures 2 and 3).

Superior mesenteric artery syndrome (SMAS) is a condition characterised by an acute or chronic compression of the third portion of the duodenum between the aorta posteriorly and the SMA anteriorly.

Anatomically, it can be explained by the dual embryologic origin of the duodenum and its vascular supply; it is a very unusual disease, its incidence in radiological studies has been described between 0.20% and 0.78%.1

It has classically been associated to specific conditions such as abrupt weight loss, prolonged bed rest, spinal deformities and several catabolic states.2 Studies suggest there may be contribution of anatomical factors such as a short mesentery, an altered anatomy or an abnormally high fixation by the ligament of Treitz for its development.

It has been seldom reported in patients with HIV, and never before in association with HAART onset; this is relevant regarding the well-known effects HAART has on lipid metabolism which are suspected to be dependent on a similarity of an HIV-1 protease and two homologous human proteins (CRABP-1 and LRP1).

About the diagnostic approach, SMAS must be suspected in all patients presenting with symptoms of high intestinal obstruction such as biliary vomit, epigastric pain and oral intolerance. Differential
diagnosis must include internal hernias, intestinal adherences, pyloric stenosis, acute gastritis and acute/chronic pancreatitis. Laboratory tests must be directed to identifying complications as intestinal ischaemia or perforation, with at least C-reactive protein, complete blood count, blood chemistry and lactate levels. Plain radiographs usually show non-specific gastrointestinal dilation, whereas barium studies may add signs of linear extrinsic pressure at the occlusion site. The most effective study is CT, which can measure the distance and angulation between aorta and SMA, as well as give an estimate of retroperitoneal fat status and identify abdominal complications. Studies have shown that aortomesenteric angles of 8°–10° and distances of 5.7–11 mm are highly suggestive of this syndrome.3

In our case, we believe both antiretrovirals and chemotherapy may have contributed to depletion of the lipid reserve of the body, leading to a reduced retroperitoneal fat pad and SMAS. Fortunately, we had obtained a previous CT scan which helped us characterise structural before/after changes associated with this condition.

Currently, there are no concise guidelines on treatment. However, experience has shown us it requires early surgical team involvement to avoid poor outcomes. In general terms, primary management consists of NPO (nil per os), gastric decompression and starting early caloric supply, reserving operative approaches to cases that fail such measures. Duodenal mobilisation (Strong’s technique) is considered the standard surgical procedure whereas minimally invasive approaches have recently shown promising outcomes. Conservative treatment success will be strictly associated with prompt caloric supply and weight recovery.

**Learning points**

- Superior mesenteric artery syndrome is caused by compression of the third portion of the duodenum between the superior mesenteric artery and the aorta.
- It must be considered in the differential diagnosis of patients presenting with symptoms of high bowel obstruction.
- Strong association with catabolic states and depleted lipid reserve is suspected in patients receiving highly active antiretroviral therapy or chemotherapy.

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REFERENCES