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Scientific letter

# Surgical Treatment of Ruptured Mycotic Hepatic Artery Aneurysm☆

## Tratamiento quirúrgico de un aneurisma micótico roto de la arteria hepática

We present the case of an 80-year-old male admitted for fever of unknown origin, presenting only a positive blood culture for *Salmonella* spp. Among the complementary tests ordered, CT angiography identified a common hepatic artery aneurysm with a maximum diameter of 78 mm (Fig. 1), and PET/CT scan demonstrated intense metabolic activity in the region of said aneurysm. After the administration of antibiotic treatment with ciprofloxacin and fever remission, the patient was discharged pending surgery, which was not performed during admission for personal reasons of the patient. Two weeks later, the patient came to the emergency room for abdominal pain and asthenia, and a pulsatile mass was detected in the epigastrium on physical examination. After performing another CT angiography, which showed increased size of the mycotic hepatic artery aneurysm (HAA) associated with signs of contained rupture, emergency surgery was indicated and these findings were confirmed intraoperatively.

Vascular control was achieved of the celiac trunk and superior mesenteric artery. During the clamping of the trunk, the intima was injured, making ligation necessary. We therefore decided to perform a retrograde bypass (with inverted left saphenous vein) from the infrarenal aorta to the bifurcation of the common hepatic artery, and from there to the proximal portion of the common hepatic artery (near the celiac trunk) to ensure retrograde circulation to the left splenic and gastric arteries, finding no signs of visceral hypoperfusion (Fig. 2). The duration of ischemia was approximately 4 h. Patient progress during the postoperative period was satisfactory. The only complication was a postoperative pancreatic fistula, which was managed conservatively, and the patient was discharged 2 weeks after the procedure.

The concept of mycotic aneurysm arose in 1885 following the study of infected aneurysms secondary to bacterial endocarditis.[1,2] They are very rare, with an incidence of 0.8%–3.2% of all aneurysms.[3] In descending order, they are located in the abdominal aorta, peripheral (femoral), cerebral and visceral arteries, with the superior mesenteric being the most frequently involved.[4]

In the pre-antibiotic era, the most frequent cause of its development was bacterial endocarditis with the development of septic emboli that settled on the vessel wall, which could be healthy or have intimal lesions. Other explanations of its genesis are the infection of a previous arteriosclerotic aneurysm (due to infections adjacent to the aneurysm or bacteremia) and the appearance of post-traumatic pseudoaneurysms, with a greater role in recent decades secondary to parenteral drug addiction and endovascular procedures. The pathogenesis is secondary to the septic embolus penetrating the *vasa vasorum*, causing an infection of its wall with the consequent destruction and the development of the aneurysm. This would explain the cases secondary to septic emboli, or an infection adjacent to the vessel that would erode the arterial wall until the aneurysm was formed.[1,4] The most frequently involved germs are *Staphylococcus* and *Streptococcus*.[4,5] The profile of the typical patient is an individual in the 7th decade of life with some degree of immunosuppression, such as neoplasms, corticosteroid treatment, etc.[2,3,6]

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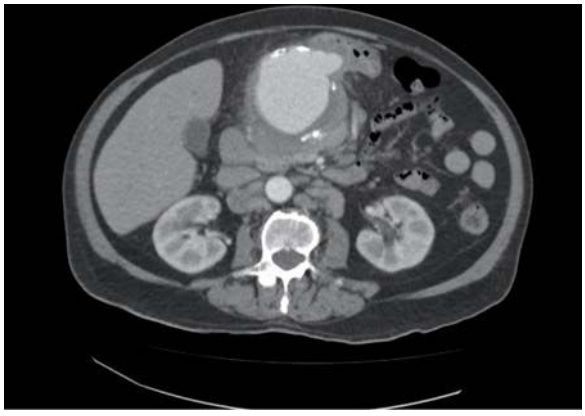

**Fig. 1 – CT angiography showing hepatic artery aneurysm.**

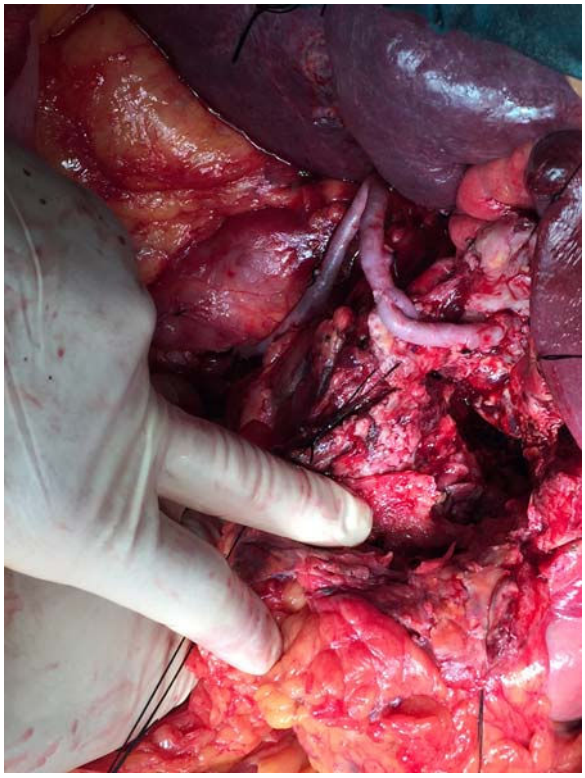

**Fig. 2 – Intraoperative image.**

The most frequent clinical presentation of HAA is abdominal pain in the right hypochondrium. In up to 80% of cases, rupture is the form of presentation of HAA, which can be to the peritoneal cavity, biliary tree or digestive tract. If the rupture occurs in the biliary tree, the classic triad consists of jaundice, abdominal pain and digestive tract bleeding[7,8]; meanwhile, if it occurs in the digestive tract, there will be no jaundice. The diagnosis is based on imaging tests, mainly CT angiography, which shows mycotic aneurysms as lobed vascular masses with irregular arterial walls and perilesional edema.[4]

The treatment of HAA continues to be debated and is a challenge for surgeons. This is based on a series of premises, such as adequate antibiotic treatment or the duration of treatment. Although it seems clear that it should be a minimum of 2 to 8 weeks, there are authors who advocate lifelong treatment and the definitive treatment of the aneurysm, either endovascular or surgical. This is of the treatment of choice in complicated HAA, especially in the scenario of an aneurysmal rupture. Surgery involves excision of the aneurysm and debridement of the perilesional infected tissue associated or not with the arterial re-anastomosis, using prostheses or autologous grafts. Endovascular treatment, with infection permanence rates of up to 23%, entails embolization or stent placement. However, there are few data about their long-term efficacy or the need for subsequent surgical intervention in many cases to definitively treat HAA.[6,9,10]

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# Celiac Trunk Thrombosis as a Presentation of Takayasu Arteritis☆

## Trombosis del tronco celíaco como presentación de la arteritis de Takayasu

Takayasu arteritis (TA) is a granulomatous vasculitis that affects large arteries, primarily the aorta and supra-aortic trunks, although medium-sized arteries may also be involved.[1]

We present the case of a 35-year-old woman, with no family history of interest or known cardiovascular risk factors except obesity and oral contraception, who came to the emergency department due to exacerbation of postprandial epigastric abdominal pain that had been progressing for months. Lab work showed evidence of leukocytosis with neutrophilia, and abdominal ultrasound revealed no significant findings. The patient was hospitalized with suspected erosive gastropathy. Gastroscopy demonstrated changes in coloration of the gastric mucosa suggestive of ischemic disease. Subsequently, emergency abdominal CT scan identified complete thrombosis of the celiac trunk, hepatic hypoperfusion and splenic infarction. Diagnostic arteriography was performed and the findings were compatible with the CT scan (Fig. 1a). Due to the high risk of thrombus migration and signs of hypoperfusion of the abdominal organs, endovascular treatment was ruled out, and we opted to perform surgical treatment.

During emergency exploratory laparotomy, we observed generalized hepatic hypoperfusion and splenic infarction, with no other alterations. Thrombectomy was performed (Fig. 2), followed by angioplasty through the splenic artery to subsequently perform splenectomy. Hepatic reperfusion and restoration of flow to the celiac trunk were confirmed by intraoperative Doppler ultrasound.

On the second day of the postoperative period, a follow-up Doppler ultrasound was performed, which showed the absence of flow in the celiac trunk but no clinical repercussions. CT scan confirmed these findings but showed no alterations in the perfusion of the abdominal organs. On postoperative day 15, the patient was discharged from the hospital with home anticoagulation therapy using low molecular-weight heparin after hypercoagulability studies had shown no alterations.

One month later, the patient came to the emergency department for cyanosis of the fingertips of the right hand; corticosteroid and antihypertensive treatment were prescribed. After one month of treatment and persistence of pain along with claudication of the upper right limb, the patient was hospitalized. CT angiography of the aorta and supra-aortic trunks with 3D reconstruction showed wall thickening at the origin of the right subclavian causing a small stenosis in the segment between the clavicle and the first right rib (Fig. 1b).

Given the symptoms, claudication of the upper right limb, and the difference in systolic blood pressure between both arms greater than 10 mmHg, the diagnosis of Takayasu disease was established. Treatment with prednisone and methotrexate was initiated, and the patient's progress was good.

TA is a chronic, idiopathic inflammatory disease that mainly affects women under the age of 40 and children. The annual incidence is estimated at 1.2–2.6 cases per million inhabitants per year in the western population, although it is much higher in Southeast Asia.[1]

Histopathology revealed adventitial thickening, areas of leukocyte infiltration of the tunica media and intimal hyperplasia. This response predisposes patients to the development of stenoses or arterial occlusion.[2]

The spectrum of presentation, severity and rate of progression of the disease can often lead to an inaccurate evaluation and a delay in diagnosis.[3] Clinically, it presents with general symptoms and specific symptoms of vascular involvement (arterial hypertension, carotidynia and vascular murmurs)[1] related to involvement of the celiac trunk in 18% of cases, according to the literature.[3,4]

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