air in the intra- and retroperitoneal spaces along the inferior vena cava up to the mediastinum and pleura.[4] It is important to mention that the amount of air does not correlate with the severity of the symptoms.[10] Treatment of the perforation should be initially conservative[4,5] and should include bowel rest, a nasogastric tube, hydration and antibiotics. Surgery is indicated for patients with persistent biliary obstruction, cholangitis, sepsis or non-response to conservative treatment.[4] The type of surgery depends on the findings and can include perforation repair, drainage of abscesses, choledochojejunostomy or even pancreatoduodenectomy.[5]

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Cystic Adventitial Disease of the Popliteal Artery

Enfermedad quística adventicial de la arteria poplítea

Adventitial cystic disease (ACD) is a rare vascular disease with an estimated prevalence of one of every 1200 cases of intermittent claudication. Due to its rare nature, it is frequently confused with atherosclerotic disease. It typically presents as intermittent claudication of the calf that is unilateral and rapidly progressive in a young healthy man with no cardiovascular risk factors.[1]

We report the case of a 42-year-old male with a history of smoking and hypercholesterolemia who presented intermittent claudication at 300 m in the right calf region. Physical examination revealed a reduced popliteal pulse and lack of distal pulses in the lower right extremity. Doppler study showed an ankle brachial index of 0.53 in this extremity. With Doppler ultrasound, we observed right popliteal artery ectasia with a maximum size of 11 mm, anechogenic content and high speed flow. Magnetic resonance imaging showed an abrupt occlusion of approximately 4 cm in length at the second right popliteal portion. We decided to perform surgical correction using the posterior approach, where we observed an enlarged popliteal artery and the discharge of a slightly yellowish gelatinous material. The second portion of the popliteal artery was removed and reconstructed with a popliteal–popliteal reconstruction using an inverted saphenous vein graft. Samples sent for pathological examination were reported to be adventitial cystic disease (Figs. 1 and 2). The immediate postoperative period was uneventful and the

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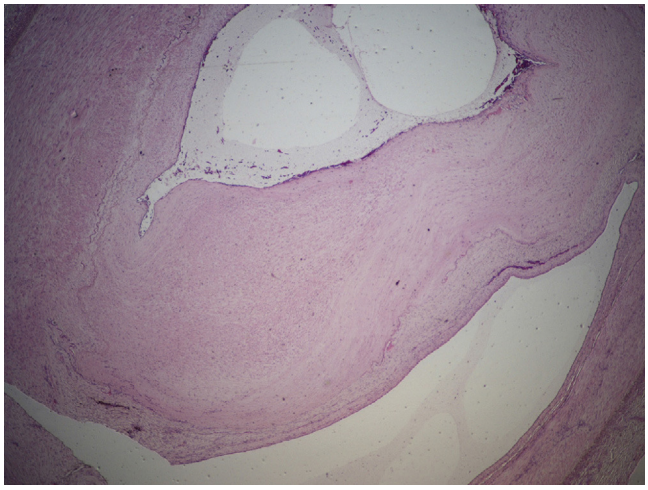

Fig. 1 – Large adventitial cyst protruding into the arterial lumen (hematoxylin–eosin stain, 2×).

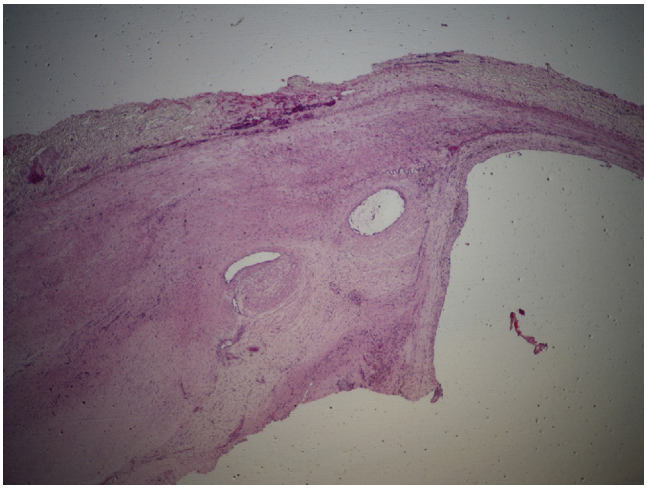

Fig. 2 – Small cysts of the arterial adventitia (hematoxylin–eosin stain, 2×).

patient was discharged 3 days later with palpable distal pulses. At the 3-month follow-up, the patient was asymptomatic and presented good perfusion of the limb.

ACD involves the formation of mucinous cysts in the adventitia of the artery walls that grow slowly, protrude into the lumen and produce localized stenosis with increased flow velocity in the segment. Over time, they can cause total occlusion. They are located in the popliteal artery in 85%–90% of cases and the involvement is often unilateral. ACD mainly affects men (15:1) between the ages of 40 and 50. It is not related to atherosclerosis or its risk factors.[1]

There are several theories describing the origin of cyst formation: the degeneration of the adventitia related to systemic connective tissue diseases or repeated microtraumas; the lymph node theory that suggests that cysts originate from synovial structures implanted in the adventitia; and the development theory that proposes that ACD is a manifestation of mucin-secreting cells derived from the mesenchyme of the proximal joints during embryonic development.[1,2]

Clinically, ACD typically presents as sudden-onset intermittent claudication in the calf region that rapidly progresses. It rarely presents in the form of acute ischemia. During physical examination, distal pulses are usually palpable, although they may not be in cases of occlusion. In some patients, passive knee flexion induces the disappearance of distal pulses (Ishikawa sign).[1,3] The ankle-brachial index is usually normal at rest and may decrease after exercise.[1]

Given the nonspecific presentation and the rarity of ACD, the diagnosis is not usually made before surgery. For diagnosis, Doppler ultrasound, computed tomography and magnetic resonance are recommended.[1,3,4] These imaging tests have replaced conventional diagnostic angiography.[1] Because of its availability and low cost, Doppler ultrasound is considered the initial test to perform in cases with suspected ACD. The cysts appear as anechoic or hypoechoic lesions. MRI is considered the imaging test of choice because it provides an accurate portrayal of the cystic lesions and their relationship with vascular structures. Cystic lesions have low-intensity signal in T1 and high-intensity in T2.[1,3,5,6]

There are several treatment options. Conventional surgery involves resection of the affected arterial segment and its reconstruction using an autologous vein graft. This is the technique of choice when the artery is completely occluded or if degeneration of the tunica media of the artery wall is found. When the popliteal artery is permeable, surgical excision of the cyst is a less invasive alternative that offers good long-term results. Percutaneous needle aspiration can result in incomplete evacuation of the cyst with recurrence rates of around 10%. Percutaneous transluminal angioplasty is not an effective option for definitive treatment because, unlike atherosclerosis, the intima is normal and the arterial wall is affected.[1,3,5]

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Obstructive Jaundice Secondary to Liver Hilar Lymph Node Tuberculosis

Ictericia obstructiva secundaria a tuberculosis ganglionar del hilio hepático

Tuberculosis (TB) of the hepatobiliary system is uncommon and it is a rare cause of bile duct stenosis. The symptoms are nonspecific, the most important of which is jaundice. Hepatobiliary TB is difficult to differentiate from other entities such as cholangiocarcinoma, and many cases require surgery, histology and bacteriological confirmation to reach a definitive diagnosis. Treatment does not differ from pulmonary TB and involves the administration of quadruple therapy for one year. In order to resolve the bile duct obstruction, endoscopic retrograde cholangiopancreatography (ERCP) is used to insert a bile stent; meanwhile, some cases may require surgical decompression.

We present the case of a 29-year-old male patient with no prior medical history of interest that came to our Emergency Department due to abdominal pain that had been developing over the previous 3 weeks. The pain was poorly defined, located in the right hypochondrium, and accompanied by jaundice of the skin and mucous membranes with choluria but no other alterations. On admittance, the work-up showed a pattern of extrahepatic cholestasis with bilirubin 4.2 mg/dl and GGT 467 IU/l. Basic biochemistry, complete blood count and coagulation were within normal limits. Tumor marker levels were also normal. Chest and abdominal radiographies presented no alterations. Abdominal ultrasound revealed the presence of a normal-sized gallbladder with biliary sludge, no signs of inflammation and moderate dilatation of the intra- and extrahepatic bile duct, observing multiple lymphadenopathies in the area as well as under the spleno-portal axis. The pancreas had a normal appearance with no observed free fluid. Abdominal computed tomography (CT) (Fig. 1) confirmed the dilatation of the bile duct seen on ultrasound, showing narrowing of the common bile duct at the hepatic hilum. We identified a heterogeneous mass of 30×40 mm with ill-defined edges and internal calcifications responsible for the dilatation of the bile duct as well as the cavernous transformation of the portal vein. In addition, intercaval-aortic and gastrohepatic lymphadenopathies larger than 1 cm were detected. We attempted to use endoscopic ultrasound needle aspiration to biopsy the mass, but this was not possible because of the location and collateral circulation. Using ERCP, we inserted a plastic stent (8.5 F×10 cm) in the common bile duct after the bile duct cytology was negative for malignancy. Since the diagnostic tests did not confirm whether the process was benign or malignant, exploratory laparotomy was indicated. We observed local inflammation of the gallbladder and hepatic pedicle with compression of the middle common bile duct caused by a cluster of at least 3 lymphadenopathies. Intraoperative biopsy of one of these lymph nodes confirmed the existence of a

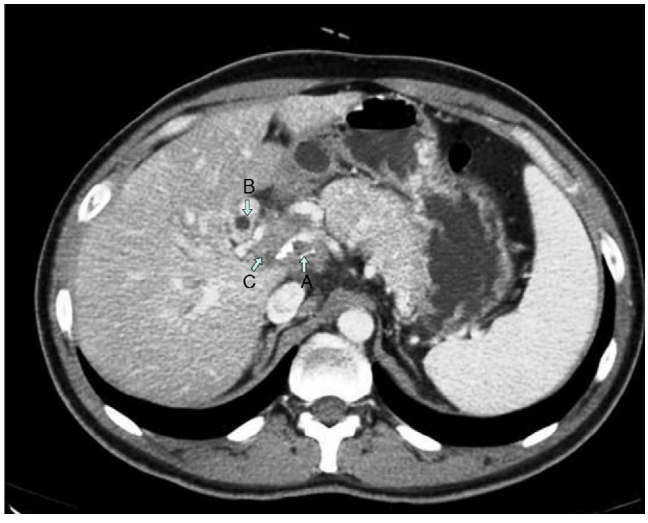

Fig. 1 – Abdominal computed tomography: (A) calcified lymph node mass in the hepatic hilum; (B) dilatation of the bile duct; and (C) portal thrombosis.

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