A subgroup of patients with HIV and PHT without liver disease has recently been identified. These patients are characterised by preserved liver function and a lack of histological cirrhosis.[9] Significant splenomegaly is also typical. It is noteworthy that these patients present normal or slightly elevated HVPG values in the haemodynamic study, which underestimates the actual portal venous pressure. This is due to increased presinusoidal hepatic resistance and the presence of intrahepatic venous collaterals.[1] Despite the fact that our patient manifested unequivocal signs of PHT (oesophageal varices, splenomegaly, collaterals), an HVPG of 10 mmHg was found. Liver biopsy is vital to rule out cirrhosis and other causes of PHT. Histological abnormalities are varied and are not pathognomonic. Those that typically manifest include portal vein sclerosis, sinusoidal dilatation and nodular regenerative hyperplasia.[1,10]

There is no specific treatment for the condition, which is limited to managing the complications of portal hypertension.[7] Although the natural history of this condition is unknown, mortality in these patients is usually caused by variceal bleeding. Mortality rates are lower than for cirrhosis, probably due to preserved liver function.

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Dysphagia lusoria as a differential diagnosis in intermittent dysphagia☆

Disfagia lusoria como diagnóstico diferencial de la disfagia intermitente

The term dysphagia lusoria refers to symptoms that arise from vascular compression of the oesophagus.[1] This clinical entity was first reported by Bayford in 1787 in a woman with a long history of dysphagia, in whom an aberrant right subclavian artery (ARSA) was found during autopsy.[2] This artery abnormality was known as *luxus nature* before the term dysphagia lusoria was adopted. The prevalence of ARSA in the general population is estimated to be between 0.4% and 0.7%. Dysphagia lusoria is caused by the abnormal regression or failure to develop of the right fourth aortic arch during embryonic development.[3] In these cases, the right subclavian artery of the aortic arch runs towards the right arm behind the oesophagus and trachea in 80% of cases, between the trachea and the oesophagus in 15% of cases and in front of both in 5%.[4]

It may be asymptomatic in childhood or manifest with respiratory symptoms during ingestion owing to tracheal compression, as its elasticity is greater in infancy. Symptom onset is rare in adults and tends to manifest in the form of dysphagia arising from greater oesophageal rigidity and arteriosclerosis. Treatment will depend on symptoms, age, associated comorbidity as well as other concomitant vascular anomalies.[5]

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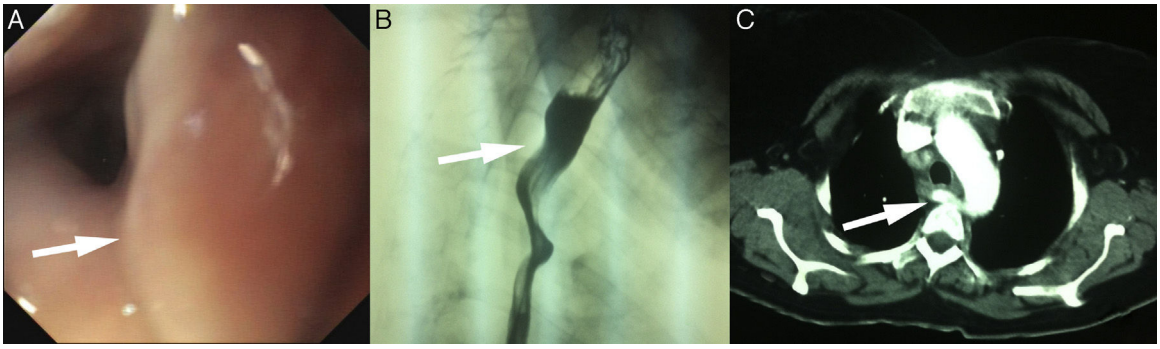

Figure 1 (A) Upper gastrointestinal endoscopy. The arrow indicates a throbbing area suggestive of a vascular anomaly. (B) UGI. The arrow indicates the oesophageal impression secondary to an aberrant right subclavian artery (ARSA). (C) Computed tomography. The arrow indicates the ARSA and how it displaces the oesophagus to the left.

We present the case of a 45-year-old woman who attended our practice with a four-year history of intermittent dysphagia for both liquids and solids, together with occasional vomiting. The patient was in psychiatric follow-up for a behaviour disorder and was being seen by an endocrinologist for multinodular goitre, with no other medical history of interest. The patient reported that the dysphagia had intensified in the last few months, accompanied by weight loss of 68 kg in the last three months. An upper gastrointestinal endoscopy (UGE) was performed, which revealed a throbbing area that slightly occluded the lumen of the upper third of the oesophagus. The study was completed with an upper gastrointestinal series (UGI) and computed tomography (CT) scan. The UGI revealed an oesophageal impression suggestive of extrinsic compression in the upper third of the oesophagus. ARSA was confirmed in the CT scan (Fig. 1). Changes to diet and swallowing were sufficient to improve the patient's symptoms, and the patient is currently asymptomatic after four years of follow-up.

Although symptoms rarely manifest in the reported cases of ARSA, prevalence ranges from 7 to 40% depending on the author.[3,6] Patients with dysphagia lusoria usually present latent symptoms that hinder its diagnosis. It is extremely common practice to conduct multiple examinations as an aberrant artery is not usually suspected as the cause of dysphagia. In this case, the examinations were performed years after the onset of symptoms, given both the patient's relatively mild intermittent dysphagia and her psychiatric medical history. The patient underwent an annual neck ultrasound to monitor her goitre and this was ruled out as being the cause of the dysphagia owing to its small size. Other symptoms associated with dysphagia may manifest, such as weight loss (as in our case), chest pain, occasional vomiting, etc., which may lead us to suspect an organic disease.[4] Given its general frequency, it is also important not to assume that the psychiatric disorder is the cause of the dysphagia.

The UGE, which must be performed to rule out other causes of dysphagia, may fail to diagnose the condition (in up to 20% of cases).[1] In this case, a throbbing area suggestive of a vascular anomaly was identified, which was subsequently confirmed with the UGI and CT scan. The angiography identified this aberrant artery and enabled the vascular map of the area to be more clearly seen. Given that angiography is an invasive procedure, we believe that it should only be used if the vascular anomaly is to be surgically corrected. In this case, the CT scan, which has demonstrated a sensitivity of 100% in some cases, confirmed the presence of the aberrant artery.[7] The initial treatment of dysphagia lusoria largely depends on the severity of symptoms. In mild-moderate cases, as with our patient, initial treatment should comprise changes to the diet, with an increased consumption of soft foods and smaller portion sizes, coupled with improved swallowing technique by eating more slowly and chewing more thoroughly.[3] Some studies have shown dysphagia improvement by also adding proton-pump inhibitors and/or prokinetics.[8] Surgery may be necessary if symptoms do not improve despite dietary changes, or in the event of an aneurysmal dilatation close to the route of the artery.[9,10] Age is also an influential factor, as surgery is the treatment of choice in children with respiratory symptoms or oesophageal compression to prevent symptoms from deteriorating.[5] Conservative treatment (changing the diet and swallowing habits) improved our patient's symptoms, who has remained practically asymptomatic throughout the four years of follow-up.

In conclusion, when persistent intermittent dysphagia presents, dysphagia lusoria secondary to an ARSA should be considered in the differential diagnosis. Conservative treatment comprising a correction of dietary habits could significantly improve the symptoms of these patients.

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Difficult management of triple therapy for chronic hepatitis C in a patient on haemodialysis☆

Difícil manejo del tratamiento con triple terapia en paciente con hepatitis crónica C y hemodiálisis

We present the case of a 52-year-old patient with a 26-year personal medical history of chronic hepatitis C (HCV) infection. The patient was diagnosed with kidney failure secondary to non-proliferative glomerulonephritis and underwent a kidney transplant in 1991 with delayed graft failure in 2007, since which point he has been on haemodialysis. He has subsequently undergone surgery on three separate occasions due to iliac artery aneurysm and mitral valve disease, complicated by endocarditis in 2011, which is being treated with acenocoumarol.

He has been attending the gastrointestinal outpatient clinic since 2009. The patient is infected with genotype 1b and a liver biopsy revealed stage 3/6 fibrosis. Monotherapy was started with pegylated interferon (IFN) alpha-2a 135 μg/week, obtaining a biochemical and virological response throughout the 48 weeks of treatment, with recurrence 6 months after treatment was withdrawn.

Triple therapy was first contemplated in 2012. The degree of fibrosis was staged by elastography, which revealed 16.6 kPa (corresponding to F4 on the METAVIR score), and the IL28B polymorphism was identified as the CC genotype.

Triple therapy with telaprevir was started in February 2014 with the dose adjusted due to kidney failure: IFN 135 μg/week, ribavirin (RBV) 200 mg/day and telaprevir 750 mg/8 h. The lab tests at the start of treatment revealed thrombocytopaenia of 114×10^3/l, GPT 67 and creatinine of 10.7 mg/dl. The complete blood count conducted in week 4 showed a 5 g/dl fall in haemoglobin (Hb) levels (previous Hb level 15.6 g/dl), together with normal transaminase levels and a viral load (VL) <15 IU/ml.

Two packed red blood cell units and two platelet units were required for the first time in week 8 following a fall in Hb levels to 8.6 g/dl and thrombocytopaenia of 18×10^3/l, which led to IFN administration being postponed for one week (Fig. 1). The patient had an undetectable viral load at 12 weeks. Four further packed red blood cell units were transfused in the following weeks due to new episodes of anaemia (with Hb levels falling to 6.7 g/dl), and RBV was finally withdrawn in week 18.

Five months after initiating treatment, the patient was admitted with *Staphylococcus aureus* bacteraemia caused by the tunnelled haemodialysis catheter, with resolution of symptoms upon changing the catheter and administering intravenous antibiotics. Given the patient's clinical status, antiviral treatment was permanently withdrawn in week 21.

In the 3- and 6-month post-treatment follow-ups, the patient's blood levels had completely recovered, with normal Hb levels, an undetectable viral load and sustained viral response that the patient maintained.

The prevalence of chronic HCV infection is higher in patients with chronic kidney disease than in the general population. It currently stands at 5.6% in Spain, and prevalence increases as the number of years on haemodialysis increases.[1] As such, the Spanish Society of Nephrology recommends anti-HCV screening in these patients.[2]

The survival of haemodialysis patients is compromised by HCV,[3,4] which represents the fourth-highest cause of mortality and the main cause of post-kidney transplantation liver dysfunction due to the earlier onset of cirrhosis and the greater risk of liver cancer.[3,5,6]

Use of antiviral treatment in dialysis patients has been limited due to the adverse effects of IFN and RBV-induced haemolytic anaemia.[7,8]

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