aggressive surveillance, starting at age 35–40 and repeating every two years if they have a high rate of polyps or every three to five years if they have a low rate.[8,13] They also recommend gastroscopy at the time of diagnosis and at five-year intervals, or less, depending on the findings.[10]

In conclusion, given the fact that multiple histological types can coexist in intestinal polyposis, it is important to assess the extra-intestinal manifestations that help us determine the end diagnosis, since the surveillance and management will be different in each case.

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Hepatitis B reactivation in a patient with chronic hepatitis C treated with direct-acting antivirals☆

Reactivación de hepatitis B en paciente con hepatitis crónica C tratado con antivirales de acción directa

Direct-acting antivirals (DAA), currently used as a treatment for hepatitis C virus (HCV), are not effective for the hepatitis B virus (HBV). Therefore, in co-infected patients (HCV/HBV) who are inactive carriers, DAA could cause the reactivation of HBV. This situation has been recently described in patients receiving treatment with DAA, although the real risk is unknown.[1–4]

We present the case of a 52-year-old male patient referred to gastrointestinal medicine from nephrology for chronic HBV infection (HBsAg positive, anti-HBc positive, HBeAg negative, anti-HBe positive) and HCV genotype 4. Originally from Cameroon, he had been living in Spain for over 20 years. His only previous medical history was hypertension, with hypertensive retinopathy and mild renal failure.

Viral loads were determined, with HBV-DNA 112 IU/ml and HCV-RNA 965,000 IU/ml. A Fibroscan® was performed to establish fibrosis stage, showing liver stiffness of 10.7 kPa.

The patient was started on therapy with ombitasvir, paritaprevir, ritonavir and ribavirin. His viral load was negative at week 4 of treatment; treatment duration was 8 weeks and he showed a sustained virological response (SVR) at 12 weeks after completion with normal AST and ALT. In week 8 of treatment, having been previously asymptomatic, the patient went to the hospital's Emergency Department with epigastric pain, with blood tests showing total bilirubin of

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4.5 mg/dl with normal ALT. He was discharged as simply epigastric pain and he decided to stop taking the treatment. His symptoms did not subsequently return, so they could be attributed to the treatment.

At post-treatment follow-up in week 24, he had ALT of 295 IU/l with RNA-HCV <15 IU/ml. HBV-DNA was 53,678,800 IU/ml. He was started on treatment with entecavir (0.5 mg/day), with transaminase values back to normal after 12 weeks and HBV-DNA <15 IU/ml at 24 weeks.

Since treatment with DAA for HCV infection began, cases of HBV reactivation (increased HBV-DNA values in patients with HBsAg and/or anti-HBc positive) have been described in co-infected patients.[1] Although reactivation has not caused clinical repercussions in most patients, there are reports of liver failure and even the need for liver transplantation.[2,3] HCV is known to suppress the replication of HBV in co-infected patients.[4]

In the light of the evidence of reactivation in patients treated with the new DAA, in December 2016 the *Agencia Española de Medicamentos y Productos Sanitarios* (AEMPS) [Spanish Agency of Medicines and Medical Devices] recommended performing HBV serology before starting treatment with DAA in all patients who were candidates for the treatment, and in those already being treated.[5] As early as April 2016, the Food and Drug Administration (FDA) had warned about the risk of reactivation of HBV in patients treated with DAA.

Although the current data are limited, they show that reactivation of HBV can occur with any DAA treatment. The reactivation usually occurs within the first 4–8 weeks after starting treatment with DAA, and is related to the rapid reduction of the HCV viral load characteristic of DAA. The recommendations are therefore to always rule out HBV infection before commencing treatment with DAA, and in cases where there is co-infection, monitor patients while on DAA treatment and after the treatment is completed.[6–8]

The reactivation of HBV was not described as an adverse effect of DAA in clinical trials, as co-infected patients were excluded from the studies.

Recent studies assessing the actual risk of reactivation in patients treated with DAA show that reactivation occurs in more than 50% of patients who are HBsAg positive, while the risk is much lower in patients with isolated anti-HBc (less than 2%). In the majority of cases, the reactivation is not accompanied by an increase in transaminases, especially when the HBV-DNA is less than 20,000 IU/ml, which is most often the case. Although rare, cases like ours have been described with repercussions on blood parameters.[6,7,9] In our case, there was no HBV-DNA monitoring and the patient was not diagnosed until his transaminase levels had already risen and his HBV-DNA levels were very high, confirming that, although an uncommon occurrence, the risk of clinically significant reactivation is real.

There is no consensus on the optimal management of prophylaxis and treatment of HBV reactivation by DAA. Some authors suggest that not all patients with reactivation should be treated. If the data from the latest studies are confirmed, one possibility would be to closely monitor and treat if HBV-DNA values are higher than 20,000 IU/ml or transaminases are also increased. The recommendation of the European Association for the Study of the Liver (EASL) of 2017 is to perform HBV serology before treatment. They recommend treating HBsAg-positive or anti-HBC-positive patients with detectable HBV-DNA with nucleoside/nucleotide analogues and monitoring transaminase levels in patients who are anti-HBs and anti-HBc positive.[10] If antiviral treatment is started, there is no consensus on how long it should last.

In conclusion, patients with HCV who are going to be treated with DAA should be screened for chronic HBV infection and, if positive, they should be closely monitored for possible HBV reactivation.

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Recurrent gastrointestinal bleeding secondary to Dieulafoy's lesion successfully treated with endoscopic ultrasound-guided sclerosis☆

Hemorragia digestiva recurrente por lesión de Dieulafoy tratada con éxito mediante esclerosis guiada por ecoendoscopia

We present the case of an 82-year-old male who attended the Emergency Department with haematemesis and melaena. His previous medical history included hypertension, atrial fibrillation on anticoagulant therapy, an operated abdominal aortic aneurysm and a previous episode seven months earlier of upper gastrointestinal bleeding caused by a Dieulafoy lesion (DL) in the gastric fundus, which had been treated by sclerosis and placement of endoclips.

On arrival at our hospital, the patient had blood pressure of 85/50 mmHg, a heart rate of 120 bpm and haemoglobin of 6.5 g/dl, for which he was resuscitated with volume and transfusion of blood products. Subsequently, within the first 8 h of the patient arriving in the emergency department, a gastroscopy was performed in which a large clot was identified adhered to the greater curvature-fundus. It was not possible to clearly visualise the focus of the bleeding, but it was treated by injecting adrenaline and polidocanol through the clot. Another gastroscopy was performed the next day, showing a clot of less than 1 cm in size, as well as small adjacent punctate disruptions in the mucosa, probably corresponding to the sclerosis of the previous day and the endoclips placed in the previous admission. As there were no signs of re-bleeding and it was not possible to determine the exact point of origin of the bleeding, no treatment was carried out this time.

Back on the ward, the patient had melaena and anaemia once again. An endoscopic ultrasound (EUS) was performed in which a 2 mm calibre vessel was identified coming through the stomach wall at the level of the gastric fundus, highly compatible with Dieulafoy's lesion (Fig. 1). The lesion was sclerosed using an endoscopic ultrasound (EUS)-guided needle, injecting 0.5 mg of adrenaline and 2 cc of polidocanol, after which the flow at that level was seen to disappear. After that, over the next 6 months of follow-up, the patient had no external signs of bleeding and did not require further transfusions.

DL is a vascular malformation in the gastrointestinal tract, most commonly occurring in the stomach.[1–4] It consists of a submucosal artery that is histologically normal but follows a tortuous path and does not undergo normal branching, thereby becoming 10 times larger than the normal calibre of the capillaries in the gastrointestinal mucosa.[1,5–7]

Although uncommon, DL is important as a serious cause of upper gastrointestinal bleeding, representing approximately 1–2% of cases.[2,7] The aetiology and pathogenesis are unknown, although it typically affects adult males with multiple comorbidities, such as hypertension, diabetes mellitus and chronic kidney disease.[1,2] The use of NSAIDs has also been associated with DL, probably as a result of the ischaemia and atrophy they cause in the gastrointestinal mucosa, which makes bleeding in these patients more likely.[1,6,8]

DL can remain silent for years, starting in most cases as sudden-onset painless gastrointestinal bleeding, which can have high morbidity and mortality rates, and has a tendency to recur.[2,4,5,8]

Endoscopy is the diagnostic technique of choice in this disease.[4,8] However, in up to 33% of cases, repeat endoscopies are needed to reach a definitive diagnosis,[2,6,7] as DL can be difficult to detect if there is no active bleeding due to the small size of the lesion and the normal appearance of the surrounding mucosa.

From the point of view of treatment, endoscopy has proven to be a safe and effective technique, achieving

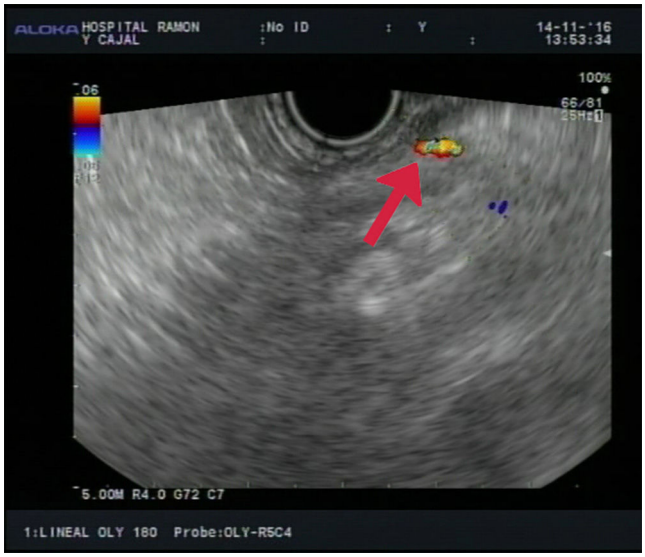

Figure 1 Endoscopy image. Vascular image crossing through the gastric wall compatible with Dieulafoy lesion.

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