Table 1 Main laboratory data (biochemical evolution) from the case report.

	ALT	AST	GGTP	AP	Bilirrubin	ANA	ASMA	Ig G
2015-06-08 (*)	23	25	14	70	0.4	-	-	-
2015-10-30	201	112	22	-	-	-	-	-
2015-11-10(**)	233	-	-	-	0.46	160	(-)	10.31
2015-11-18	252	140	30	69	0.7	-	-	-
2015-11-25	266	151	30	72	0.8	(-)	(-)	10.45
2015-12-02	228	119	31	70	0.8	-	-	-
2015-12-07	179	92	30	79	0.6	(-)	80	10.54
2015-12-21	121	64	27	79	0.5	80	(-)	9.74
2016-01-13	81	58	25	73	0.5	(-)	(-)	10.47
2016-02-19	37	35	20	85	0.44	80	(-)	10.38
2016-04-22	34	33	15	73	0.67	(-)	(-)	10.92
2016-06-21	27	36	15	71	0.73	(-)	(-)	10.4
2016-10-18	26	28	13	72	0.51	(-)	(-)	9.88
2017-03-24	28	34	13	-	0.76	(-)	(-)	-

Baseline (*) and (**) one day after drug-cessation laboratory. ALT (N: 0-41 U/L)-alanine aminotransferase; AST (N: 0-37 U/L)-aspartate aminotransferase; GGTP (N: 10-71 U/L)-gammaglutamil-transferase; AP (N: 40-129)-alkaline phosphatase; bilirrubin (N: 0-1.1mg/dL); ANA: antinuclear antibodies; ASMA: anti smooth muscle antibodies; Immunoglobulin G (N: 7-16g/L).

values started to show a slow improvement. Anti-smooth muscle antibodies were present (title 1:80) as well as antinuclear antibodies (1:80). ALT and AST values continued decreasing and 2 months after stopping Rosuvastatin intake auto-antibodies became negative. Finally, during the following 2 weeks (3 months after discontinuing Rosuvastatin) AST-ALT levels dropped back to normal and stayed that way for the next 12 months' follow up, when the patient was discharged. Our patient remained asymptomatic during the whole process and no corticotherapy was used.

Several cases of autoimmune hepatitis associated with statins have been reported.[3–7] Two different scenarios may be distinguished: Drug induced (DI)-AIH, a selfperpetuating disease, and immune-mediated (IM)-DILI, an acute or chronic process that disappears after drug withdrawal[8] (which we presume our patient's case to be).

Usually symptoms of acute liver injury occur within the first months after starting therapy but a longer latency period is possible[1] (as shown in our patient). A spontaneous remission after drug cessation is expected in IM-DILI but sometimes hepatocellular or mixed type liver damage may appear.[8] If so immunosuppressive (IS) treatment is mandatory.[8] Response to corticoesteroids therapy is usually outstanding with complete remission and no relapse even after discontinuing immunosuppressive therapy.[1,8] Therefore IS treatment is not forever and this distinguishes IM-DILI from idiopathic autoimmune hepatitis and DI-AIH.[8]

Statins are responsible of an important number of cases with DI-AIH (IM-DILI) and DILI with positive autoantibodies[3,8]. It is very difficult to differentiate between this two clinical scenarios[8,9]. The weaknessess of our case are the absence of liver biopsy, the normal Ig G values, and the absence of typical HLA-DR alleles. None of them is mandatory for the diagnosis of DI-AIH, although they are very helpful[6,9,10]. We have not performed a liver biopsy because of the good evolution and the mild clinical features of the patient. Higher Ig G values are associated with DI-AIH, but normal values may be present in these patients[6,10]. The presence of positive autoantibodies (ANA, ASMA) and the loss of ANA positivity after cessation of the drug are more likely to be associated with DI-AIH. The latency period of more than 2 months, generally longer than DILI patients, and hepatocellular liver injury pattern, are tipically associated with this disease. Recovery time is expected to be longer for DI-AIH than for DILI (8-10 weeks vs 5-7 weeks)[6,10]. All of these criteria were fulfilled by our patient. Morover, the transaminases values do not decrease more than 50% in the first month after drug cessation, and, although CIOMS score was possible for DILI, AIH international score revealed probable for AIH. Therefore, we think that this case report is a very probable IM-DILI due to statins.

Conflict of interest

No concflicts of interest

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Protein-losing enteropathy resolved after ventral abdominal hernia repair☆

Enteropatía pierde-proteínas resuelta tras la reparación de una eventración abdominal

Protein-losing enteropathy (PLE) is characterised by an excessive loss of serum proteins through the gastrointestinal tract, primarily causing hypoproteinaemia with hypoalbuminaemia and oedema. When a patient has hypoalbuminaemia with oedema, other causes of decreased serum protein levels, such as malnutrition, kidney disease with proteinuria or liver impairment with alteration in protein synthesis, should first be ruled out.[1]

There are different causes of PLE, including metabolic, intestinal, inflammatory and infectious disorders, and alterations in lymphatic drainage, such as occurs in intestinal lymphangiectasia.[1]

We report the case of a 57-year-old woman who had two episodes of idiopathic acute pancreatitis in less than a year. In that year, the patient developed palpebral and lower extremity oedema with persistent hypoalbuminaemia, with serum albumin levels <2 g/dl (normal 3.30–5.20), a decrease in the concentration of γ-globulins and short half-life proteins, without diarrhoea. In the investigations performed at that time, faecal elastase and faecal fat tests were normal. Alpha-1-antitrypsin clearance was 797 ml/24 h (normal 0.00–12.50) and the bacterial overgrowth test, stool cultures and stool analysis for parasites were negative. Nephrotic syndrome was also ruled out as a cause of hypoalbuminaemia.

The patient subsequently had a further episode of acute pancreatitis with splenic vein thrombosis for which she was started on anticoagulation; treatment was also started with corticosteroids, as autoimmune pancreatitis was suspected despite normal IgG4. She showed no improvement but instead had another exacerbation of pancreatitis. A hypercoagulability study was performed with factor V Leiden, proteins c and S, antithrombin III, homocysteinaemia and prothrombin 20210, which were all within normal limits. A month later, positron emission tomography scan showed an increase in uptake in the tail of the pancreas, suspected to be of malignant origin. In the end, a distal pancreatectomy with splenectomy was performed via laparotomy; pathology was compatible with chronic pancreatitis (inflammation and fibrosis associated with areas of necrosis).

As the hypoalbuminaemia persisted, the patient was started on parenteral nutrition, leading to a slight improvement in blood test parameters (serum albumin 2.6 g/dl) and improvement clinically. Upper gastrointestinal endoscopy identified duodenal and jejunal lymphangiectasia. Causes of secondary lymphangiectasia were ruled out, except for chronic pancreatitis, which the patient already had. Colonoscopy was also performed, showing mucosa with normal appearance. One year after starting the parenteral nutrition, the patient developed pancreatic pain resistant to first- and second-step analgesics, with no diarrhoea or weight loss. MRI and endoscopic ultrasound showed severe chronic calcifying pancreatitis in the head of pancreas and a cyst measuring 35 mm in the wall of the duodenum. She was started on octreotide and FNA confirmed the inflammatory nature of the contents of the cyst. In view of the poor pain control, a coeliac plexus block was performed, which provided pain control for a year. After that same period of time, an abdominal CT detected a subcostal incisional hernia related to the surgical wound (Fig. 1). The hernia was repaired laparoscopically, after which the patient experienced an improvement in her pain. At the same time, her serum albumin levels improved and she was able to stop the parenteral nutrition. Six months after the intervention, serum albumin was 4.21 g/dl and alpha-1-antitrypsin clearance was 37.96 ml/24 h.

Resolution of PLE following laparoscopic hernia repair is not a common event. In our case, intestinal lymphangiectasia was caused by chronic pancreatitis and aggravated by the subcostal hernia. This event, secondary to distal

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