SIR, The presence of hepatic arteritis is a common finding in the hepatic biopsies of patients with PAN. Nevertheless, its impact on other collagen-vascular diseases (CVDs) is much lower. In an extensive pathological series of 120 autopsies and 40 hepatic in vivo biopsies taken from patients with a history of CVD, Matsumoto found signs of liver arteritis in 100% of patients with PAN, but in only 18% of patients with SLE, the last all belonging to autopsy samples [1]. So far the association between hepatic arteritis and autoimmune hepatitis (AIH) has not been published. We report a patient with a diagnosis of AIH and a simultaneous histological finding of hepatic vasculitis.

The patient was a 62-year-old woman with a medical history of a mild self-limiting neutropenia episode during the post-operative period after a hysterectomy due to myomas 10 years earlier. The patient was admitted to our hospital due to a 1 week clinical picture of intense asthaenia, jaundice and coluria. She was not taking medicines or herbal products, and was not suffering from dry eye syndrome, photosensitivity, oral aphthae, alopecia or Raynaud’s disease. Physical examination highlighted cutaneous-mucous jaundice and a 3-cm painful hepatomegaly. The analysis revealed thrombopenia (platelet count of 44 000/μl). The PT and activated partial thromboplastin time were 18 and 21.6 s, respectively. Complement component 4 was decreased (3.6 mg/dl). Bilirubin was 14.8 mg/dl, aspartate aminotransferase was 1272 IU/l, alanine aminotransferase was 1272 IU/l, alkaline phosphatase was 1120 IU/l, ALP was 711 IU/l and γ-glutamyl transpeptidase was 123 IU/l. The γ-globulin levels were elevated with IgG 2790 mg/dl. Antibody studies were positive for ANA (1/320, nucleolar pattern), anti-dsDNA (1/320) and anti-Ro/SSA, and negative for RF, ANCA, AMA, ASMA, lupus-like circulating anti-coagulant, antibodies to liver kidney microsome, anti-cardiolipin, β2-glycoprotein I, soluble liver antigen and CCP. Cryoglobulins were negative. Serologies for hepatotropic virus were negative and ceruloplasmin and α1-anti-trypsin levels were normal. Quantification of proteins in urine was 0.24 g/24 h. Abdominal US showed a normal-sized liver. We did a transjugular biopsy of the liver, and the histopathological study showed the

(a) Interface hepatitis with erosive hepatocellular necrosis. Numerous plasma cells (arrows), a common finding in AIH, are seen in the infiltrate. (b) An arteriole with a focus of necrotizing vasculitis, which is characterized by fibrinoid necrosis of its wall accompanied by a neutrophilic infiltrate with nuclear dust.
presence of parenchymal nodules wholly or partially limited by fibrous bands in which we noted significant ductular proliferation and a lymphohistiocytic inflammatory infiltrate with some eosinophils and neutrophils, as well as plasma cells and hepatocytic changes such as focal foamy degeneration, parenchymal cytolysis and multinucleation. We identified a medium-calibre artery with transmural neutrophilic infiltrate, endothelial damage, slight leucocytoclasia and incipient fibrinoid necrosis in one of the septa (Fig. 1). We began treatment with prednisone 0.5 mg/kg/day and AZA, and observed a rapid clinical response. Analytical parameters gradually improved and remained normal after 2 years of follow-up.

Our patient has a defined AIH according to both the 1999 reviewed diagnostic criteria from the International Autoimmune Hepatitis Group (score 23) [2] and the 2010 simplified criteria by the same group (score 7) [3]. The rapid response to steroidal treatment, the presence of a prominent interface hepatitis with lymphocytic erosive necrosis and a significant component of plasma cells in the inflammatory infiltration of the liver biopsy reinforced this diagnosis.

Anti-dsDNA positivity and the presence of hepatic arteritis, which is sometimes described in liver biopsies from patients with lupus, can make us suspect SLE [4]. Nevertheless, our patient did not meet the classification criteria of the ACR for this entity, and although hypertransaminasenaemia is common in patients with lupus, clinically significant liver disease is rare and the association between AIH and SLE is very unusual (1.3–1.7%) [5]. In addition, positivity for anti-dsDNA, which is classically associated with SLE, is also seen in up to 23% of patients with type 1 AIH [6].

The description of devastating cases, complicated with intra-abdominal haemorrhage or ruptured liver, is striking in the literature review of PAN or SLE cases with hepatic arteritis [7–9]. However, our patient achieved complete normalization of liver enzymes and immunoglobulin levels 6 weeks after the initiation of treatment with CSs and AZA, and remained in remission without further flare in 2 years of follow-up.

Rheumatology key message

- Hepatic arteritis can be seen in patients with AIH.

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Gastric ulcer and gastroenteritis caused by Epstein–Barr virus during immunosuppressive therapy for a child with systemic juvenile idiopathic arthritis

Sir, EBV and CMV are often reactivated in immunosuppressed patients and cause organ-specific complications. In contrast to CMV, EBV rarely causes gastrointestinal (GI) diseases, particularly in children. Here we report a case of gastric ulcer and enterocolitis caused by EBV in a paediatric patient undergoing immunosuppressive therapy for systemic juvenile idiopathic arthritis (sJIA).

A 3-year-old girl with sJIA who had been treated with tacrolimus and prednisolone presented with a 2-week history of anorexia followed by melena. Her medical history included trisomy 21, an atrial septal defect, congenital hypothyroidism and surgically treated Hirschsprung’s disease. On admission, the patient was afebrile with slightly