In chronic hepatitis C (CHC), the interlobular bile ducts, particularly the small bile ducts, show a variety of abnormalities collectively known as hepatitis-associated bile duct damage. This type of bile duct lesion is found in up to 90% of the liver biopsies performed in these patients (1, 2). Usually, these lesions are different from that observed in primary biliary cirrhosis (PBC). However, cases of CHC associated with true PBC, with (3–6) and without (7–9) positive anti-mitochondrial antibodies (AMA), have been reported. In the former cases, interferon (IFN) therapy was ineffective (3) or even triggered cholestatic damage (4–6).

In this paper, we report the case of a female patient with CHC and PBC-like duct lesions, negative AMA and positive anti-nuclear antibodies (ANA), fulfilling the criteria for autoimmune cholangitis (AC).

CASE REPORT

A 49-year-old white woman was studied for asymptomatic changes of liver tests over the previous three years. She was previously healthy, apart from a hysterectomy two years earlier. The physical examination was unremarkable. Laboratory tests showed: eosinophils: 610/μL (normal < 500); erythrocyte sedimentation rate, 44 mm/hr (normal < 20); bilirubin, 0.3 mg/dl (normal 0.2–1.2 mg/dl); aspartate aminotransferase, 53 units/liter (normal 5–45); alkaline phosphatase (ALP), 409 units/liter (normal 98–295); Albumin, 4.4 g/dl; prothrombin activity, 100%. The ANA were positive (1:160–1:2560) and with a speckled pattern. AMA, anti-M2, anti-liver–kidney microsomal, anti-smooth muscle, and anti-neutrophil cytoplasmic antibodies were negative. Serum levels of immunoglobulins were increased [IgG, 1550 mg/dl (normal 644–1436 mg/dl); IgA, 469 mg/dl (normal 160–1280 mg/dl); IgM, 736 mg/dl (normal 65–348 mg/dl)], and the cryoglobulins were positive (2.5%). Serum levels of copper, ceruloplasmin, α1-antitrypsin, glucose, cholesterol, triglycerides, creatinine, and ferritin were normal, as was the saturation of transferrin and urinary excretion of copper and porphyrins. Circulating anti-hepatitis C virus antibodies were positive and hepatitis C virus RNA was detected by the polymerase chain reaction method. Abdominal ultrasonography was normal apart from the presence of gallbladder microlithiasis. However, nuclear magnetic cholangioresonance disclosed a normal biliary tree.

Percutaneous liver biopsies were performed at the time of diagnosis and one year later. Both liver biopsies showed similar features. Liver architecture was distorted by portal fibrosis associated with dense interstitial lymphoplasmocytic infiltrates and sporadic lymphoid aggregates. At the periportal interfaces, mild piecemeal necrosis and ductular proliferation were observed. Several ductal lesions were noted. Small interlobular ducts, frequently located within lymphoid aggregates (lymphocytic cholangitis), displayed nuclear pseudostratification and occasional intraepithelial lymphocytes (Figure 1A, B). Some degree of ductopenia (less than 20%) was also present (Figure 2). In both biopsies, basal membrane of the largest biliary ducts were interrupted by lymphohistiocytic infiltrates with rupture of their basal membrane and vacuolization of the epithelial cells (floridlike ductal lesion) (Figure 3A, and B). Mild lymphoplasmocytic infiltrates and patchy acidophilic degeneration of liver cells were observed in the lobules.
fatty changes, biliary pigment, copper, or hemosiderin were apparent. Considering the importance of the inflammatory bile duct damage, ursodeoxycholic acid (UDCA) was administered (13 mg/kg/day) for one year. During this time, liver tests returned to the normal range, but liver damage and immunological profile remained unchanged.

**DISCUSSION**

Autoimmune cholangitis (AC) is a chronic cholestatic liver disease in which liver biopsy shows changes consistent with PBC, but in which AMA is negative. Generally, other antibodies, such as those directed against nuclear antigens or smooth-muscle antigens, are present. Brunner and Klinge first described this entity in 1987 (10), and numerous cases have been published subsequently (11–13). However, the identity of this disease, as an atypical form of PBC or as an independent entity, remains under discussion (14, 15). In the present case, liver biopsy demonstrated the