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Liver Involvement is Frequently Found in Patients with Primary Sjogren's Syndrome

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Abstract

There may be liver involvement in primary Sjogren's syndrome patients. Our objectives were to correlate the presence of abnormal hepatic biochemistries with other clinical and laboratory features in patients with primary Sjogren's syndrome and to determine the prevalence of clinical liver disease.

Methods: The study looked at 95 patients who had been diagnosed with primary Sjogren's syndrome. Tests for inflammation and autoimmunity, as well as concomitant diseases, liver biochemistry, gender, age and clinical features were gathered.

Results: A total of 42 patients (44 percent) had abnormal hepatic biochemistries and among those 42 patients, 19 (20 percent) had clinical liver disease. The frequency of autoimmune hypothyroidism, arthritis, vasculitis, Raynaud's phenomenon, a higher sedimentation rate and a higher frequency of antinuclear and antimitochondrial antibodies was higher in patients with abnormal hepatic biochemistry than in patients with normal liver biochemistry (P, 0.05 for each). Patients with clinical liver sickness had higher recurrence of joint inflammation, vasculitis and higher recurrence of antimitochondrial antibodies than patients without clinical liver illness. Patients with primary Sjogren's syndrome frequently have liver involvement, which is linked to systemic disease features and autoimmunity and inflammation markers. A subset of patients may have liver involvement as a result of the primary Sjogren's syndrome.

Keywords: Primary Sjogren's syndrome; Abnormal hepatic biochemistries; Liver disease; Sedimentation; Inflammation

Introduction

Although primary SS is an autoimmune exocrinopathy, liver involvement has been reported. Primary Sjogren's Syndrome (primary SS) is the combination of keratoconjunctivitis sicca, xerostomia and salivary gland swelling in the absence of any other rheumatologic condition [1]. Primary Biliary Cirrhosis (PBC), autoimmune cholangitis and primary SS have been linked to liver diseases. Autoimmune Hepatitis (AIH) and the infection with the Hepatitis B and C Virus (HBV, HCV). Primary sclerosing cholangitis, nodular regenerative hyperplasia and pathological labial salivary gland examinations of patients with various types of liver cirrhosis have revealed the presence of chronic lymphocytic sialadenitis in a significant number of cases [2]. The prevalence of abnormal hepatic biochemistries and clinical liver disease in patients with primary SS is evaluated in this retrospective analysis of prospectively acquired data. Additionally, we establish a correlation between the presence of abnormal hepatic biochemistries and clinical liver disease and various other systemic characteristics and indicators of autoimmunity and inflammation [3].

Between January 1985 and December 2005, 156 medical clinical charts of patients with a primary SS diagnosis who visited our institution were examined. These patients were retrieved from our institutional biostatistics database, the rheumatology outpatient clinic; rheumatology consults records, and various outpatient services [4]. We only included 95 of the 156 Sjogren's syndrome patients who met the European Epidemiology Center Criteria (EECC) for primary SS. Because the EECC were created in 1993, the clinical records of patients who attended prior to this year were examined retrospectively to determine whether or not they met these criteria. We also didn't include any patients who showed signs of other rheumatic diseases because this would have made them a secondary Sjögren's syndrome. The histopathological examination of the salivary glands of fifty patients, or 53%, confirmed the diagnosis of Sjogren's syndrome [5].

When the following signs and symptoms were observed in conjunction with abnormal hepatic biochemistry, clinical liver disease was considered: Jaundice, ascites, palmar erythema, spider angiomata, evidence of collateral circulation, asterixis, hepatomegaly or other portal hypertension symptoms like esophageal or gastric varices and splenomegaly. The presence of autoimmune hypothyroidism, Raynaud's phenomenon, vasculitis, arthritis, pancreatitis, peripheral neuropathy and glomerulopathy are among the systemic features analyzed. The student t-test was used for continuous variables in large samples with similar variances and the *Chi-square* and Fisher exact tests were used for categorical measures to compare patients with and without abnormal hepatic biochemistry and clinical liver disease. For small samples, the nonparametric Mann-Whitney U test was used. The

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equation (a × d) 3(c × b) was used to calculate Odds Ratios (OR) with 95% Confidence Intervals (CI), where a denoted patients who had abnormal hepatic biochemistries or clinical liver disease and/or markers of autoimmunity or inflammation and b denoted patients who did not have abnormal hepatic biochemistries or clinical liver disease and/or markers of autoimmunity or inflammation.

Description

According to the study, patients with primary SS are more likely to have liver involvement and the presence of liver involvement is associated with systemic disease features and certain autoimmune and inflammatory markers. In particular, we found a relationship between liver contribution and the presence of immune system hypothyroidism, joint pain, vasculitis, Raynaud's peculiarity, raised globular sedimentation rate, ANA and AMA. Only one previous study had found a higher prevalence of abnormal liver biochemistries and clinical liver disease in patients with primary SS. These studies had found a prevalence of liver affection in patients with primary SS ranging from 6% to 58%; the frequency of liver involvement in our study is higher compared to other reports. However, the definition of liver involvement in the majority of these studies was either subjective or ambiguous. Using clinical, biochemical, immunological, histological data and the largest study of primary SS patients found that 7% of patients had evidence of liver disease and the same percentage had AMA detected by immunofluorescence. Additionally, AMA positive patients had characteristics of chronic cholangitis similar to stage I PBC. However, the lower prevalence of subclinical liver involvement in primary SS patients may be partially explained by this study's higher cut-off value for defining the presence of abnormal liver biochemistries.

On the other hand, primary SS has long been thought to be caused by viral infection, and more recently, a link between primary SS and HCV has been suggested. There is proof that shows a relationship of trademark histological changes of Sjögren's condition in the salivary organs and HCV infection. In this study we tracked down a predominance of 12% of HCV contamination in our patients with essential SS and there are reports that showed frequencies up to 19%, being fundamentally higher than the pervasiveness of disease detailed in everyone. Additionally, the prevalence of abnormal hepatic biochemistries and clinic liver disease was higher in patients with primary SS and HCV infection. It is remarkable that we found a significant association between vasculitis and liver involvement, pointing out a possible link between the hepatic and vascular damage. Since it has been suggested that the target tissue involved in the autoimmune damage of primary SS might be the epithelium, the tissue damage observed in some patients with primary SS may be related to an abnormal interaction between lymphocytes and various epithelial tissues, including the liver. In primary SS, epithelial cells appear to be active participants rather than passive targets in the chronic immune response; however, in order to establish the role of liver epithelial cells in the pathogenesis of this disease, additional research is required, possibly including an analysis of HLA expression and cytokine profiles.

Conclusion

We discovered that liver involvement was present in a significant number of primary SS patients for no apparent reason; as a result, this subset of patients may have immunologic hepatic damage as a result of primary SS. Due to the retrospective nature of this study, the true prevalence of liver involvement cannot be precisely determined, and not all laboratory, radiological and histological studies were performed on all patients. Patients with primary SS typically have liver involvement, which appears to correlate with systemic disease clinical features like arthritis, vasculitis and Raynuad's phenomenon as well as autoimmune and inflammatory markers like ANA, AMA and globular sedimentation rate. A significant number of patients with primary SS who also have subclinical and clinic liver disease cannot be explained by any other diagnosis.

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