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## Should we set-up routine screening for primary sclerosing cholangitis in all IBD patients?

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Primary sclerosing cholangitis (PSC) is an inflammatory stricturing bile duct disease, associated with inflammatory bowel disease (IBD) in up to 80% of cases. It is associated with serious hepatobiliary complications (cirrhosis, hepatocellular carcinoma, cholangiocarcinoma) and an increased risk of colorectal neoplasia, leading ultimately to an increased risk of mortality in patients with IBD and PSC. PSC is usually classified as classic (large duct) PSC with successive dilatation and stricturing of bile ducts, and small duct PSC in cases of normal Magnetic Resonance Cholangiography (MRC) with histopathology findings of PSC on liver biopsy (1-3). In this issue of Digestive and Liver Disease, Belle A. and colleagues report the experience of the Nancy IBD cohort regarding the prevalence of PSC (4). In this cohort, 233 IBD patients underwent MRC and were compared to 187 MRC findings of non-IBD patients. Patients were divided within the IBD group into two groups according to the presence or not of liver function test (LFT) abnormalities. The prevalence of PSC was 9.9% in the IBD cohort with LFT abnormalities, higher than found in the non-IBD cohort. Only one out of 30 (3.3%) patients without LFT abnormalities was diagnosed with PSC. Biochemically only an increase

of gamma-glutamyl transferase (GGT) was independently associated with MRC findings of PSC.

Beside the selection bias and the unclear indication for MRC in all patients of the cohort, including controls, this study underlines the high frequency of PSC in IBD patients with abnormal LFT. In a recent Norwegian study, systematic MRC was performed in IBD patients to screen for PSC and 8.1% of patients (26/322) had associated PSC(5). A recent report at Digestive Disease Week 2018 based on a cohort of patients undergoing systematic liver biopsy during any IBD surgery detected an even higher PSC prevalence (16.5%); by using histological diagnostic criteria this also included small duct PSC(6). Of note, nearly half of these patients had normal LFTs, unlike the Nancy cohort. The natural history outcomes of small duct PSC without MRC abnormalities remain unclear in the current literature. While liver complications appear to be decreased, whether colorectal cancer risk is different between the small duct and classic PSC remains to be determined. Given the known serious natural history outcomes of PSC patients the question of whether all IBD patients should be systematically screened for PSC remains of interest. As underlined by Belle and colleagues, MRC should be performed in all cases of IBD patients with abnormal LFTs. Whether MRC should be performed in all IBD patients with normal LFTs remains questionable, although the results from this study would suggest that this is not necessary. In patients with confirmed PSC recommendations for radiological surveillance with MRC, or ultrasound for gallbladder lesions, also remain to be confirmed. From a luminal viewpoint there is a need to more

higher risks of colorectal neoplasia. This may potentially allow for further individualisation of colonoscopic surveillance recommendations for PSC patients; until such data are produced annual surveillance colonoscopies are recommended.

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