HISTOLOGY OF IRON OVERLOAD IN ALCOHOLIC LIVER DISEASE: ARE THERE PROGNOSTIC IMPLICATIONS?

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Iron can be identified in H&E stained sections as dark-brown granules on light microscopy. However, histochemical staining (Prussian blue orPerl's stain) facilitates the detection of iron with much higher sensitivity and specificity. Iron may be present in parenchymal cells as fine siderin granules at the biliary pole of hepatocytes and in cholangiocytes as well as in mesenchymal cells including endothelia, macrophages in portal tracts and fibrous septa and/or Kupffer and fat storing cells in sinusoids. Thus three types of hepatic iron overload designated as parenchymal, mesenchymal or mixed can be distinguished according to the cellular and lobular distribution of iron. In alcoholic liver disease (ALD) iron storage, mostly of mild degree is a frequent histologic finding present in up to 57% of patients with chronic alcohol abuse. In contrast to early stages of hereditary HFE hemochromatosis in which iron is mainly stored in periportal hepatocytes, iron deposition in ALD is frequently present as mixed type siderosis. In alcoholic cirrhosis iron storage can be marked resembling late stage HFE hemochromatosis.

The cause of iron accumulation in the liver in ALD is multifactorial and involves age, diet, race, HFE status, hepcidin synthesis as well as environmental factors. Iron overload may exacerbate liver injury presumably by iron-driven oxidative stress in chronic liver diseases and ALD contributing to disease progression, the development of cirrhosis and hepatocellular carcinoma.