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Iron and liver fibrosis: Mechanistic and clinical aspects

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Abstract

Liver fibrosis is characterised by excessive deposition of extracellular matrix that interrupts normal liver functionality. It is a pathological stage in several untreated chronic liver diseases such as the iron overload syndrome hereditary haemochromatosis, viral hepatitis, alcoholic liver disease, non-alcoholic fatty liver disease, non-alcoholic steatohepatitis and diabetes. Interestingly, regardless of the aetiology, iron-loading is frequently observed in chronic liver diseases. Excess iron can feed the Fenton reaction to generate unquenchable amounts of free radicals that cause grave cellular and tissue damage and thereby contribute to fibrosis. Moreover, excess iron can induce fibrosis-promoting signals in the parenchymal and non-parenchymal cells, which accelerate disease progression and exacerbate liver pathology. Fibrosis regression is achievable following treatment, but if untreated or unsuccessful, it can progress to the irreversible cirrhotic stage leading to organ failure

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