Liver cirrhosis: Chronic liver damage.

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Description

Cirrhosis is well-defined as the histological progress of re-forming nodules enclosed by fibrous bands in response to chronic liver damage, which leads to portal hypertension and end-stage liver ailment. Recent improvements in the understanding of the natural history and pathophysiology of cirrhosis, and in management of its complications, have ensued in improved management, quality of life, and life expectancy of patients. Liver transplantation remains the only curing option for a selected group of patients, but pharmacological treatments that can halt development to decompensated cirrhosis or even reverse cirrhosis are now being developed. Liver cirrhosis is the final pathological result of numerous chronic liver ailments, and fibrosis is the precursor of cirrhosis. Symptoms may include loss of Fatigue, bleeding or bruising, Loss of appetite, Weight loss, Yellow discoloration in the skin and eyes. Many types of cells, cytokines and miRNAs are involved in the origination and development of liver fibrosis and cirrhosis. Stimulation of hepatic stellate cells (HSCs) is a pivotal event in fibrosis. Defenestration and capillarization of liver sinusoidal endothelial cells are major causative factors to hepatic dysfunction in liver cirrhosis. Activated Kupffer cells abolish hepatocytes and stimulate the activation of HSCs. Repeated cycles of apoptosis and renaissance of hepatocytes contribute to pathogenesis of cirrhosis. At the molecular level, many cytokines are involved in mediation of signaling pathways that control activation of HSCs and fibrogenesis. Recently, miRNAs as a post-transcriptional regulator have been found to play a key role in fibrosis and cirrhosis. Robust animal models of liver fibrosis and cirrhosis, as well as the newly identified critical cellular and molecular factors involved in the progress of liver fibrosis and cirrhosis will facilitate the improvement of more effective therapeutic approaches for these conditions. The end stage of several forms of chronic hepatitis of different etiologies is a diffuse process characterized by fibrosis and the translation of normal liver architecture into structurally irregular nodules surrounded by annular fibrosis. This chronic advanced clinical condition, leads to liver cell failure and portal hypertension. Liver biopsy is presently considered the best existing standard of reference but it has some limits, so different tools have been established to substitute liver biopsy when assessing liver fibrosis. Serum markers offer a cost-effective alternative to liver biopsy being less invasive and theoretically without difficulties. They can be classified into direct and indirect markers which may be used alone or in amalgamation to produce composite scores. Diagnostic imaging includes a number of instruments and techniques to evaluate liver fibrosis and cirrhosis like ultrasound (US), US Doppler, contrast enhanced US and Elastography. US could be used for the diagnosis of innovative LC while is not able to evaluate progression of fibrosis, in this case Elastography is more reliable.

Risk factors

- Drinking too much alcohol. Excessive alcohol consumption is a risk factor for cirrhosis.
- Being overweight. Being obese growths your risk of conditions that may lead to cirrhosis, such as nonalcoholic fatty liver ailment and nonalcoholic steatohepatitis.
- Having viral hepatitis. Not everyone with chronic hepatitis will develop cirrhosis, but it's one of the world's leading causes of liver disease.

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