Liver can get damaged from various injurious factors which, when acting chronically can result in liver injury and inflammation. This leads to repair, healing, and fibrosis, which ultimately can result into cirrhosis. Cirrhosis has its consequences and can result in hepatic insufficiency and portal hypertension, which in turn lead to portal-systemic encephalopathy, gastrointestinal bleeding, ascites, renal dysfunction, and ultimately can lead to hepatocellular carcinoma and death. Various factors like viruses, toxic substances, drugs, auto immune hepatitis, steatosis and alcohol can result in liver injury. In our scenario, hepatitis B and C are the commonest causes of liver fibrosis and cirrhosis. Multiple mechanisms have been described which lead to liver fibrosis. The central factor is activation of hepatic stellate cells, which occurs from multiple reasons, which cause the quiescent cell to become activated. Injury to hepatocytes and cholangiocytes stimulates hepatic stellates cells via various cytokines and thus these cells become activated. When activated, these cells start poring extra cellular matrix, which result in deposition of fibrin resulting in fibrosis (fibrogenesis). Stellate cells result in proliferation, inflammation, contractility, chemotaxis, altered matrix degradation and retinoid loss. Hepatitis B virus X protein (HBx), large isoform of hepatitis delta antigen (LHDAg) and hepatitis C virus derived proteins have been documented to induce stellate cell activation. With ongoing insult and on poring of extra cellular matrix in the sinusoids, this progresses to high grade fibrosis and cirrhosis.

In hepatitis, it takes number of years to cause the liver fibrosis and cirrhosis. Therefore, eradicating the virus before the fibrosis sets in can spare the liver from any further damage. Improvement in Metavir score was reported in patients with hepatitis C who underwent treatment with pegylated interferon. Improvement in inflammation and regression of fibrosis and even cirrhosis occurs in hepatitis B patients after treatment with tenofovir. The detection of fibrosis is important because the treatment of the underlying condition has to be before the fibrosis sets in. Several noninvasive laboratory tests have been described and many radiological tests also can detect the development of fibrosis and high degree of fibrosis and cirrhosis. Liver stiffness was the best non-invasive method to assess the presence of cirrhosis, portal hypertension and esophageal varices. The liver stiffness can be assessed by fibroscan and fibro tests. However, in our setting, the easiest means to detect early fibrosis are AST/ALT ratio and platelet count, which can be easily performed as part of the routine examination. The liver biopsy remains a gold standard for the detection of fibrosis and cirrhosis, but noninvasive tests have their place now because the liver biopsy can be associated with complications, sampling error and inter observer variability.

The prevention of fibrosis involves treatment and elimination of causative agents. In our setting, treatment of hepatitis B and C is the most important and it should be done before the fibrosis sets in. Newer treatments for hepatitis C are making this possible without the multiple side effects and prolonged therapy, even in treatment experienced patients and patients with cirrhosis. Treatment of hepatitis B still remain challenging. However, prolonged viral suppression is possible with presently available anti viral agents, which has been shown to prevent fibrosis, cirrhosis and its associated complications.

Fibrosis and cirrhosis are no longer considered to be static conditions and is a continuous remodeling process and cirrhosis is a dynamic condition and can be reversed (fibrolysis). The most important step in reversing the fibrosis is elimination of inciting factor like virus, alcohol or drugs. Once the fibrosis sets in, even then there are chances that the
fibrosis and cirrhosis can be reversed, as has been shown by several studies in autoimmune hepatitis\textsuperscript{10} and in hepatitis C.\textsuperscript{7} Many agents are under investigation as antifibrotic drugs. Therapeutic targets include interaction between cells, soluble mediators, extra cellular matrix and its receptors, and relevant intracellular signaling.\textsuperscript{8} While all these therapies are in progress, to reverse the fibrosis most important step for us at this point is to focus on its prevention and that involves the timely management and elimination of hepatitis viruses, as they have been so much prevalent in our country.

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