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Perspective

Pharmacotherapy Interference for the Treatment of Liver Fibrosis

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DESCRIPTION

Chronic liver disease is due to a variety of causes of persistent liver damage, ultimately leading to the development of liver fibrosis. If treatment is not initiated, the condition can progress to cirrhosis and hepatocellular carcinoma. Current treatments include lifestyle changes, abstinence, and coping with causes of injuries such as antivirals. However, treatment based on such etiology is often inadequate in patients with late-stage fibrosis/cirrhosis, thus perpetuating the need for efficient anti-fibrotic drug therapy interventions.

Fibrosis is an important consequences of chronic liver disease and an important determinant of disease progression, including mortality. Due to the decrease in the world's population and the ongoing obesity epidemic, NAFLD/NASH is recognized as the leading cause of chronic liver disease and is considered the leading cause of liver-related morbidity and mortality. The various pathways associated with liver fibroblast formation (eg: myofibroblast activation, ECM deposition, inflammation) are partially reversible when the damaging agent is eliminated. Key drug mechanisms/targets for achieving antifibrotic effects include cellular stress and subsequent death, inflammation, metabolic pathways, intestinal hepatic axis and myofibroblasts, and associated ECM production. Combination therapies using compounds that target multiple mechanisms of NAFLD/NASH etiology are most likely to provide an overall antifibrotic effect.

Liver fibrosis is an abnormal wound healing reaction caused by a variety of chronic liver injuries characterized by excessive deposition of diffuse Extra Cellular Matrix (ECM) and abnormal connective tissue hyperplasia, cirrhosis, liver failure, or. It may progress further to liver cancer. To date, chronic liver disease associated with liver fibrosis has been on the rise, with significant morbidity and mortality worldwide. Although early cirrhosis has been reported to be reversible, the detailed mechanism for reversing cirrhosis remains unclear and there is a lack of effective treatment for cirrhosis. Therefore, it remains a top priority in the research and development of anti-fibrotic drugs. In recent years, as an important drug that inhibits the onset and onset of liver fibrosis, such as anti-inflammatory and liver protection, inhibition of Hepatic Stellate Cell (HSC) activation and proliferation, reduction of ECM overproduction, and promotion of ECM degradation. Many strategies are emerging. Gene therapy has also been shown to be a promising anti-fibrotic method. This section provides an overview of related targets and drugs under development. Our aim is to classify and summarize their potential role in the treatment of liver fibrosis and discuss challenges and the development of antifibrotic drugs.

Oxidative stress is an important factor in liver damage and cirrhosis. The Oxidative Stress Response (ROS) produces excess reactive oxygen species and active free radicals in the liver. This weakens antioxidant function, increases active free radicals in hepatocytes, and reduces hepatocyte removal and destruction. These results affect the function of hepatocyte synthesis and degradation, leading to hepatocyte necrosis and apoptosis. In addition, ROS also promotes activation of HSC and liver fibrosis by causing peroxidative damage to Kupffer cells and neutrophils, upregulating type I collagen alpha 2 expressions in the liver, and inducing inflammation. The combination of liver disease and cirrhosis is the 12th leading cause of death, accounting for 40,545 cases, or 1.5% of all deaths in the United States in 2016. Chronic Liver Disease (CLD) is a major public health concern around the world, and liver fibrosis is a broad protective response to CLD of a variety of etiologies, including: B. Persistent viral hepatitis B and C, Non-Alcoholic Fatty Liver Disease (NAFLD), alcohol overload and autoimmune liver disease. When the injury or inflammation becomes chronic and untreated, the cell's response becomes dysregulated. The imbalance between increased synthesis and decreased degradation causes excessive deposition of Extra Cellular Matrix (ECM) proteins and the development of final scar tissue formation or fibrosis, ultimately cirrhosis and associated complications.

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