Inflammation in alcohol-associated liver disease progression

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Abstract

Chronic alcohol consumption induces stress and damage in alcohol metabolising hepatocytes, which leads to inflammatory and fibrogenic responses. Besides these direct effects, alcohol disrupts intestinal barrier functions and induces gut microbial dysbiosis, causing translocation of bacteria or microbial products through the gut mucosa to the liver and, which induce inflammation indirectly. Inflammation is one of the key drivers of alcohol-associated liver disease progression from steatosis to severe alcoholic hepatitis. The current standard of care for the treatment of severe alcoholic hepatitis is prednisolone, aiming to reduce inflammation. Prednisolone, however improves only short-term but not long-term survival rates in those patients, and even increases the risk for bacterial infections. Thus, recent studies focus on the exploration of more specific inflammatory targets for the treatment of severe alcoholic hepatitis. These comprise, among others interference with inflammatory cytokines, modulation of macrophage phenotypes or targeting of immune cell communication, as summarized in the present overview. Although several approaches give promising results in preclinical studies, data robustness and ability to transfer experimental results to human disease is still not sufficient for effective clinical translation.