

CO-ORGANIZED EVENT

13th International Conference on Clinical Gastroenterology & Hepatology & 2nd International Conference on Digestive Diseases

December 07-08, 2017 Madrid, Spain

Acute-on-chronic liver failure: An update

Dong Joon Kim

Hallym University College of Medicine, South Korea

Acute-on-chronic liver failure (ACLF) is an increasingly recognized distinct disease entity encompassing an acute deterioration of liver function in patients with chronic liver disease. Although there are no widely accepted diagnostic criteria for ACLF, the Asian–Pacific Association for the Study of the Liver (APASL) and the American Association for the Study of Liver Disease and the European Association for the Study of the Liver (AASLD/EASL) consensus definitions are commonly used. It is obvious that the APASL and the AASLD/EASL definitions are based on fundamentally different features. Two different definitions in two different parts of the world hamper the comparability of studies. Recently, the EASL-Chronic Liver Failure Consortium proposed new diagnostic criteria for ACLF based on analyses of patients with organ failure. There are areas of uncertainty in defining ACLF, such as heterogeneity of ACLF, ambiguity in qualifying underlying liver disease, argument for infection or sepsis as a precipitating event, etc. The two ACLF definitions result in differences in mortality and patient characteristics among ACLF patients. Although the exact pathogenesis of ACLF remains to be elucidated, alteration of host response to injury, infection, and unregulated inflammation play important roles. The predisposition, infection/inflammation, response, organ failure (PIRO) concept used for sepsis might be useful in describing the pathophysiology and clinical categories for ACLF. The mechanisms of ACLF were described recently. In 1/3 of cases of ACLF, inflammation develops in response to bacterial infection. However, a significant number of cases are not related to obvious bacterial infection. In these cases, the translocation of PAMPs without viable bacteria or the releases of DAMPs by dying cells are likely mechanisms of the ‘sterile inflammation’. Finally, a decrease in the tolerance to inflammation could be involved. Treatment strategies are limited to organ support, but better understanding of the pathophysiology is likely to lead to discovery of novel biomarkers and therapeutic strategies in the future.

djkim@hallym.ac.kr