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The natural history of chronic Hepatitis B and C infection in the United States

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Chronic viral hepatitis secondary to hepatitis B virus (HBV) and/or hepatitis C virus (HCV) is an important cause of liver disease worldwide. Specifically, in the United States, HBV or HCV-related chronic liver disease are major etiologies for liver transplantation (OLT), not only due to the development of end-stage liver disease (ESLD) and associated complications, but also because of the increased risk of hepatocellular carcinoma (HCC) associated with both of these viruses. Although the prevalence of chronic HBV in the US has been increasing, primarily due to emigration from other parts of the world (Eastern Europe, Asia, Pacific Islands), the use of direct acting antiviral agents (DAA's) such as tenofovir and entecavir, both associated with low rates of viral resistance or breakthrough, has likely changed the natural history of the virus in the US. Fewer patients with HBV are presenting with ESLD and thus listed for OLT, although the problem of HBV-associated HCC still remains. Because many patients with HBV-related HCC present with advanced disease, they are not candidates for OLT. This discussion will focus on diagnosis of HBV and identification of appropriate candidates for antiviral therapy, as well as the important of HCC screening. Chronic HCV infection prevalence is highest among middle-aged and older Americans, the so-called birth cohort, and the CDC initiative to screen these individuals is ongoing. Along with identification of individuals infected with HCV comes the necessity of linkage to care. The advent of the DAAs for treatment of HCV will inevitably impact its natural history and should ultimately result in potential eradication of this infection, and reduction in OLT in this population. However, given the long disease span of chronic HCV infection from its acquisition to the development of cirrhosis, the burden of chronic HCV-related liver disease will continue to increase before it ultimately plateaus and declines. The increasing incidence of HCC which has occurred over the last three decades is primarily due to the development of cirrhosis from HCV infection. Although sustained virologic response (SVR) or cure is now possible for over 90% of individuals infected with HCV, the cost of the DAAs at present is prohibitive, making it a challenge to initiate treatment in many infected individuals. Ultimately, we should see a trend in favor of less ESLD due to both chronic HBV and HCV, along with an upward trend for those non-viral diseases, for example NASH, which will become the challenge of the future.

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Advances in hepatic stem/progenitor cell biology and their therapeutic potential for liver diseases-current scenario

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The liver is famous for its strong regenerative capacity employing different modes of regeneration according to type and extent of injury. Mature liver cells are able to proliferate in order to replace the damaged tissue allowing the recovery of the parenchymal function. In more severe scenarios hepatocytes are believed to arise also from a facultative hepatic stem/progenitor cell (HSPC) compartment. In human, severe acute liver failure and liver cirrhosis are also both important clinical targets in which regeneration is impaired where the role of this stem cell compartment seems more convincing. In animal models, the current state of ambiguity regarding the identity and role of liver progenitor cells in liver physiology dampens the enthusiasm for the potential use of these cells in regenerative medicine. The aim of this talk is to give the basics of liver progenitor cell biology and discuss recent results vis-à-vis their identity and contribution to liver regeneration. In these last couple of years we have learned much about the pathways and conditions involved in HSPC activation thanks to sophisticated genetically modified mouse models. These same models are currently in conflict about the existence and function of HSPCs during liver injury and regeneration. Perhaps novel mouse liver injury models, more representative of human disease need to be developed to fully unravel the existence, identity and function of HSPCs.

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