Chronic Liver Diseases and Hepatocellular Carcinoma: An Update for 2013

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The 10th Korea-Japan Liver Symposium was held on July 7–8, 2013, in Busan, Korea, in conjunction with the 4th Asia Pacific Primary Liver Cancer Expert Meeting on July 5–7, 2013.

Nishida et al. [1] reported 4 cases of Budd-Chiari syndrome with complete or incomplete obstruction of the inferior vena cava and hepatic vein. Within 1 month of performing balloon angioplasty, improved liver function was evident. They therefore concluded that it is crucial to manage this rare disease before it progresses to liver cirrhosis.

Yada et al. [2] reviewed the characteristic features of IgG4-related disease and discussed its association with autoimmune hepatitis. They concluded that some patients with autoimmune hepatitis present symptoms of IgG4-related disease and respond effectively to steroid treatment.

Sugimoto et al. [3] reported that the interferon (IFN) and ribavirin (RBV) resistance-determining region with 6 or more mutations correlated with IFN- λ 1 and was the only significant predictor of a sustained virological response (SVR) in patients with a high viral load of hepatitis C genotype 1b. High serum levels of IFN- λ 1 may therefore be conducive to pegylated IFN and RBV combination therapy via effects on the immunomodulatory system.

Kim et al. [4] investigated the predictive factor of response to pegylated IFN plus RBV combination therapy for chronic hepatitis C genotype 2a and 2b with high viral load. They investigated the impact of host genetics repre-

sented by the single nucleotide polymorphism (SNP) of the IL28B gene and viral genetic variations within the NS5A on the outcome of pegylated IFN and RBV treatment. They concluded that the NS5A sequence heterogeneity and IL28B SNP are useful factors to predict the sensitivity to pegylated IFN plus RBV therapy in HCV-2a and HCV-2b infections.

Sugimoto et al. [5] reported that double-filtration plasmapheresis plus IFN treatment in nonresponders to pegylated IFN plus RBV combination therapy in patients with a high viral load of hepatitis C genotype 1b achieved SVR in 15% (10/40) of patients, which represents a relatively good result. The significant factors associated with SVR were interleukin-28B major type and a rapid virological response at week 4.

Sakurai et al. [6] reported that hypothermia has a direct protective effect on hepatocytes in a mouse model of fulminant hepatitis that is realized through reducing the production of reactive oxygen species. This finding may lead to the development of novel therapeutic methods for fulminant hepatitis in humans.

Nishida and Kudo [7] described the role of oxidative stress and epigenetic instability in hepatocarcinogenesis. They induced epigenetic instability via two types of DNA alterations: hypermethylation of the promoter of tumor suppressor genes (TSGs) and hypomethylation of non-promoter CpG sites, such as repetitive elements and satellite DNA. The former causes transcriptional inactivation of TSGs, while the latter induces chromosomal instability and abnormal activation of oncogenes as well as mobile

genetic elements. These mechanisms act in concert and induce epigenetic instability, leading to hepatocellular carcinoma (HCC).

Kim et al. [8] reported that activation-induced cytidine deaminase is a nucleotide-editing enzyme and that its aberrant expression induced by the inflammatory response contributes to hepatocarcinogenesis via the accumulation of genetic alterations in various tumor-related genes.

Nishida et al. [9] reported that oxidative stress alters chromatin status, which leads to abnormal methylation of TSGs and contributes to hepatocarcinogenesis in chronic hepatitis C patients.

Tsuji et al. [10] reported that CD34 expression in the capillaries and sinusoids of noncancerous hepatic tissue is a risk factor for multicentric recurrence of HCC after surgical resection. They concluded that histologic assessment of hepatic tissue with CD34 immunohistochemistry might be useful for the prognostic evaluation of HCC patients after surgery.

Inoue et al. [11] reported previous treatment for HCC and hyperintensity on T2-weighted magnetic resonance (MR) images as risk factors for the hypervascular transformation of hypovascular nodules that show hypointensity in the hepatobiliary phase image of Gd-EOB-DTPA-enhanced MR imaging. Accordingly, these nodules require careful and extensive follow-up, even though they are hypovascular.

Minami et al. [12] examined whether ablation of the needle tract can prevent bleeding after percutaneous ra-

diofrequency ablation (RFA) for malignant tumors. They found that ablation of the needle tract seems to have no effect on preventing iatrogenic hemorrhage after RFA.

Makino et al. [13] reported the development of a novel variation of the extracted-overlay function in computed tomography (CT)/MR-ultrasonography (US) fusion imaging for RFA, in which only the tumor extracted from CT/MR images with a virtual ablative margin of arbitrary thickness is overlaid on US images. They found this function extremely useful for treatment planning and guidance for RFA as it allows the extracted tumor with an ablative safety margin to be visualized on US images, even during and after ablation.

Kudo et al. [14] performed a multicenter retrospective study to clarify the survival benefits of nontransplant treatments for patients with HCC with associated Child-Pugh C cirrhosis. They found that nontransplant treatments such as transarterial chemoembolization [15], hepatic arterial infusion chemotherapy [16], percutaneous ethanol injection therapy [17] and RFA [18] are independent prognostic factors in these HCC patients. They recommended that medical treatment be considered for these patients, especially those with low Child-Pugh scores. They state that this fact confirms the validity of the recommendation by consensus-based treatment algorithm proposed by the Japan Society of Hepatology [19].

The symposium was highly successful and I firmly believe the findings presented will benefit the entire readership of *Digestive Diseases*, especially readers who are interested in chronic liver diseases and HCC.

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