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Hepatology Highlights

Kevork M. Peltekian

Department of Medicine, Dalhousie University and Division of Digestive Care & Endoscopy,
Queen Elizabeth II Health Sciences Centre, Halifax, Nova Scotia, Canada.

In this issue of the *Annals of Hepatology* the readers are invited to look closely at four articles from four different continents all of which are attempting to identify clinical predictors to facilitate management of patients with liver disease and avoid costly unnecessary interventions.

High age and low sodium urine concentration are associated with poor survival in patients with hepatorenal syndrome

Most of us dealing with decompensated liver disease agonize when managing patients with hepatorenal syndrome (HRS) since its outcomes remain poor in spite all the advancement we have made in hepatology. When considering relatively expensive therapies such as intravenous terlipressin, predicting futile cases as early as possible will help reintroduce compassion to a technologically driven medicine.

Hinz M, *et al.*[1] From Germany, retrospectively evaluated 21 patients with HRS treated with combination of intravenous terlipressin and albumin infusion. Even with this expensive intervention, only 3 (14%) survived, and an extra 2 (10%) survived after undergoing liver transplantation, while 16 (76%) died. Older patients showed significantly lower response to treatment (cut off for responsiveness at around age of 60 years) and low urinary sodium levels prior to treatment were also a significant predictor of mortality (cut off at around 20 mmol/L). Although the importance of age as predictor of responsiveness has been reported,[2] baseline urinary sodium as predictor of outcomes in HRS has not been documented.[3] It is obvious more research is required in management of HRS.

Impact of the severity of end-stage liver disease in cardiac structure and function

Significant time and effort is spent on assessment of cardiac disease in patients with end-stage liver disease (ESLD) prior to listing them for liver transplantation. Unfortunately, cirrhotic cardiomyopathy may not be identified before liver transplantation contributing to intra-operative complications with cardiac causes accounting for approximately 7-15% of deaths in the post-operative period.[4]

Silvestre OM, *et al.*[5] From Brazil, provide more insight into cardiac structure and function by studying echocardiographic parameters in 184 patients with ESLD. Over 49% of patients were found to have diastolic dysfunction but the latter did not correlate with the Model for End-Stage Liver Disease (MELD) score. On the other hand, patients with more advanced ESLD (MELD $\geq$ 16) had larger left-atrial diameter, larger diastolic left-ventricular diameter and higher pulmonary artery systolic pressure. Can these parameters identify patients undergoing liver transplantation who may face intra-operative or post-operative cardiac remodeling events?

Correspondence and reprint request: Kevork M. Peltekian, M.D.
QEII-HSC Victoria General Site, 1276 South Park Street, Halifax, NS, Canada B3H 2Y9.
Telephone: 1-902-4737833. Facsimile: 1-902-4732783;
E-mail: Kevork.peltekian@dal.ca

Patients younger than forty years old with hepatitis C virus genotype-1 chronic infection had treatment responses similar to genotype-2 infection and not related to interleukin-28B polymorphism

Predictors of sustained viral response (SVR) in treatment of chronic hepatitis C virus (HCV) infection to pegylated-interferon and ribavirin have been recently reviewed in the *Annals of Hepatology*.[6] The major SVR predictors include HCV genotypes, stages of liver fibrosis, baseline viral load and age. Currently, interleukin-28B (IL-28B) polymorphism is becoming critical SVR predictor in clinical practice especially for genotype-1 HCV infected patients.

Lin C-Y, *et al*.[7] From Taiwan, studied baseline demographic parameters from 380 treatment-naïve patients as well as CC allele specific primers for rs12979860 (IL-28B SNP) correlating these to SVR following pegylated interferon and ribavirin therapy. In this analysis, patients were divided into two age groups (Age < 40 years *vs.* ≥ 40 years). In genotype-1 patients younger than 40 years, lower body mass index was the only SVR predictor; in fact IL-28B SNP was not a predictor. On the other hand, in the older group with HCV, the three independent predictors of SVR with highest odds ratio (OR) included adherence (OR 12.77; 95% CI: 3.31-49.38), genotype (OR 5.88; 95% CI: 2.80-12.35) and CC allele with OR 5.43; 95% CI: 1.97-15.01. If these results are confirmed in other HCV patient populations, IL-28B polymorphism may need to be determined only in genotype-1 patients older than 40 years that are being treated with pegylated-interferon and ribavirin.

Diagnostic value of fibronectin discriminant score for predicting liver fibrosis stages in chronic hepatitis C virus patients

Attalah AM, *et al*.[8] From Egypt, provide us with another attempt at finding the "holy grail" of hepatology: finding an alternative to liver biopsy to predict stage of liver disease in chronic HCV infection.[9] This is a well executed study with both a derivation group (n = 145) and validation group (n = 180) with appropriate liver biopsy specimens comparing AST to Platelet Ratio Index (APRI) to a newly derived fibronectin discriminant score (FDS) which incorporates serum fibronectin levels (mg/L), APRI and serum albumin levels (g/L). In the validation group FDS cut off ≥ 0.35 had sensitivity of 77% and specificity 82% for predicting patients with fibrosis stages F2-F4. FDS cut-off ≥ 0.55 showed sensitivity of 74% and specificity of 73% in predicting those with advanced fibrosis, early cirrhosis or cirrhosis. FDS better than APRI. The only way, FDS will get a broader acceptance is comparing it to all the other markers of fibrosis.

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