

Henry Ford Health

Henry Ford Health Scholarly Commons

Nephrology Meeting Abstracts

Nephrology

7-2022

Predicting prognosis in large cohort of decompensated cirrhosis of liver (DCLD)- a machine learning (ML) approach

Ashok Choudhury

Vinod Arora

Kajali Mishra

Harshvardhan Tevethia

Vishal Kaushik

See next page for additional authors

Follow this and additional works at: https://scholarlycommons.henryford.com/nephrology_mtgabstracts

Authors

Ashok Choudhury, Vinod Arora, Kajali Mishra, Harshvardhan Tevethia, Vishal Kaushik, Babita Prasad, Manoj Kumar, and Shiv Kumar Sarin

Conclusion: Ammonia is an independent predictor of ACLF and its associated mortality in clinically stable outpatients with cirrhosis. An ammonia level over 1.48 times the ULN defines the high-risk group for ACLF.

OS066

Predicting prognosis in large cohort of decompensated cirrhosis of liver (DCLD)- a machine learning (ML) approach

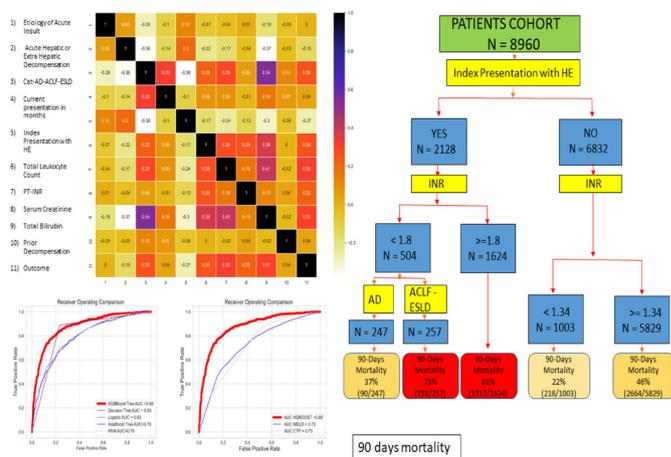
Ashok Choudhury¹, Vinod Arora¹, Kajali Mishra², Harshvardhan Tevethia¹, Vishal Kaushik¹, Babita Prasad¹, Manoj Kumar¹, Shiv Kumar Sarin¹. ¹Institute of Liver and Biliary Sciences, New Delhi, India; ²Henry Ford Hospital, Detroit, United States
Email: doctor.ashokchoudhury@gmail.com

Background and aims: Onset of decompensation in cirrhosis is associated with poor outcome. The current clinico-biochemical tools have limited accuracy in predicting outcomes reliably. Identifying the predictors with precision model on the big data using artificial intelligence may improve predictability. We aimed to develop a machine learning (ML) based prognostic model for predicting 90 day survival in patients of cirrhosis presenting with decompensation.

Method: We analysed electronic medical records retrospectively of hospitalised cirrhosis patients at the ILBS, with a complete 90-day follow-up. Clinical data, laboratory parameters and organ involvement were serially noted. AI-modelling was done after appropriate mining, feature engineering, splitted randomly into train and test-sets (20:80). The class imbalance problem was handled by random over-sampling technique, to make balanced 50:50 ratios. After 10-fold cross validation, 3 repetitions and grid search for optimal hyper parameters, the XGB-CV model was chosen. AUC was the primary selection criteria and confusion matrix was used to compare AUCs between AI-models and existing indices; CTP and MELD-score.

Results: Total of 6326 patients [mean age 48.2 ± 11.5 years, 84% male, Mean CTP 10.4 ± 2.2 and MELD Na-30.4 ± 11.9, alcohol 49.4%] were included. Ninety day mortality was 29.2%. Acute insult was identified in 80% cases; of which extra-hepatic 49%, hepatic 46% and unknown 5% cases respectively. The XGB-CV model had the best accuracy for prediction of 90 days event in the train set 0.90 (0.90–0.93), validation set 0.80 (0.79–0.81) and for overall dataset 0.80 (0.79–0.81). The AUC of the XGB-CV model was better than CTP and MELD Na-score by 16% and 15% respectively. The prediction model considered 43 variables; 18 of which predicted the outcome, and 10 maximum contributors are shown in concordance classifier. The most contributors to poor outcome included, index presentation as HE, diagnosis of AD/ACLF/ESLD, PT-INR, serum creatinine, total bilirubin, acute insult etiology, prior decompensation, acute hepatic or extrahepatic insult, leukocyte count and present duration of illness. In the Decision Tree Model, the presence of HE, PT-INR and syndromic diagnosis of AD or ACLF/ESLD was able to stratify the patients into low (22%), intermediate (23–46%) and high risk (>75%) of mortality at 90 days.

Conclusion: The AI based current model developed using a large data base of CLD patients presenting with decompensation immensely adds to the current indices of liver disease severity and can stratify patients at admission. Simple ML algorithms using HE and INR besides syndromic presentation, could help treatment decisions and prognostication.



Liver Immunology

OS067

Differential changes in global and antigen-specific B cell frequencies and function associate with the outcome of HBV nucleos (t)ide analog treatment withdrawal

Sabela Lens^{1,2}, Alice Burton¹, Jessica Davies¹, Mireia García-López², Anna Jeffery-Smith¹, Nikolai Novikov³, Simon Fletcher³, Sofía Pérez-del-Pulgar⁴, Xavier Forn², Mala Maini¹. ¹University College of London, Division of Infection and Immunity, United Kingdom; ²Hospital Clinic, Liver Unit, Barcelona, Spain; ³Gilead Sciences, United States; ⁴Liver Unit, Hospital Clínic, University of Barcelona, IDIBAPS, CIBERehd, Barcelona, Spain
Email: slens@clinic.cat

Background and aims: Despite HBsAg loss and the development of anti-HBs being the hallmark of natural resolution of HBV or achievement of ‘functional cure’, humoral immunity has not yet been examined in the setting of nucleos (t)ide analog (NA) discontinuation. We analysed HBeAg and HBsAg-specific frequencies and functional potential longitudinally in HBeAg-negative chronic Hepatitis B (CHB) patients stopping NA therapy to assess whether HBsAg loss ± off-treatment viral control associated with a recovery of the defective B cell response characteristic of CHB.

Method: Twenty-one HBeAg- CHB patients with complete viral suppression (>3 years) and without cirrhosis were studied prospectively. 16-colour flow cytometry was used to quantitate and phenotype circulating HBsAg- and HBeAg-specific B cells (dual fluorochrome-labelled antigen baits), global B cells, plasmablasts and Tfh at baseline, 12 and 48 weeks after stopping NA (W12 and W48). Cultured ELISPOTs were used to longitudinally evaluate antibody-secreting cells against HBsAg and HBeAg.

Results: After a median follow-up of 34 months (IQR 26–37), 16 (76%) patients remained off-therapy, with 5 (24% of the total cohort) losing HBsAg, whilst 5 (24%) required NA reintroduction. Frequencies of HBsAg and HBeAg-specific B cells were comparable between groups at baseline and did not correlate with levels of qHBsAg or HBeAg respectively, but did show differential temporal dynamics according to clinical outcome. HBeAg-specific B cells increased at W12 in all groups in association with viral rebound, but decreased to baseline values by W48 only among those achieving HBsAg loss or being re-treated. By contrast, patients remaining off-therapy had a significant increase in HBsAg-specific B cells by W48 compared to re-treated patients. Compared to patients remaining HBsAg+ after NA discontinuation, B cells in patients with HBsAg loss were enriched for the