# APASL-2016
# ABSTRACTS ON ACLF

# INDEX

PL-6

# AARC-ACLF SCORE PREDICTS 30 DAY SURVIVAL BETTER THAN CLIF-SOFA AND MELD SCORES IN PATIENTS WITH ACLF

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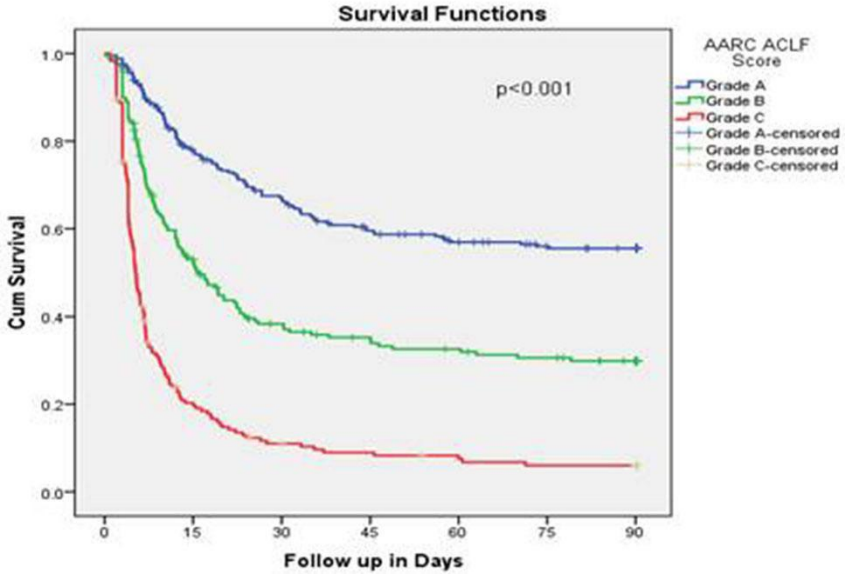
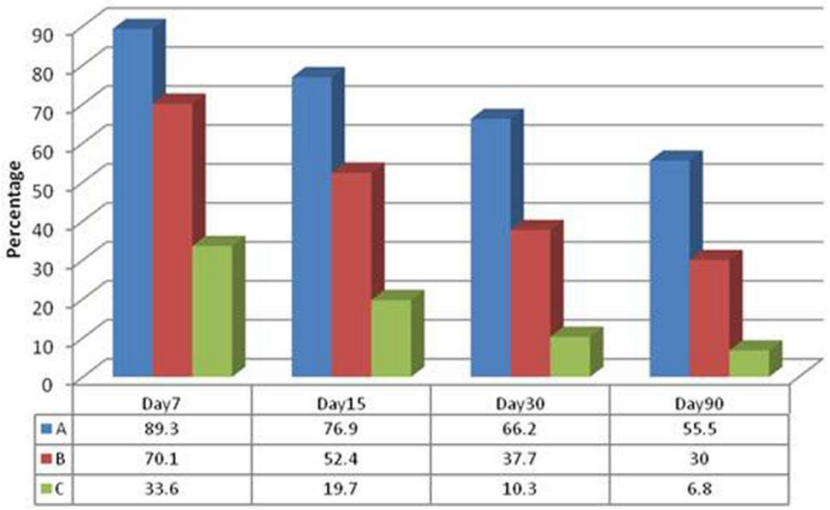
Background and aims: Acute on chronic liver failure (ACLF) is associated with the rapid worsening of liver failure with high mor-tality. Prediction of survival and early intervention can improve the outcome. Aim was to derive a prognostic model in patients of ACLF by APASL deÞnition.

**Fig-1: Survival at time point in different AARC ACLF Grade**



Methods: Total 1021 ACLF cases with 90 days follow up enrolled into the APASL ACLF Research Consortium (AARC) were analyzed. A derivation set of 338 cases analyzed for a prognostic model and calibrated in 683 cases validation set.

**Fig-2: Survival at time points –AARC ACLF Grade wise.**




| | Day7 | Day15 | Day30 | Day90 |
|---|---|---|---|---|
| A | 89.3 | 76.9 | 66.2 | 55.5 |
| B | 70.1 | 52.4 | 37.7 | 30 |
| C | 33.6 | 19.7 | 10.3 | 6.8 |

Results: Of all the baseline independent predictor of mortality, total bilirubin, Creatinine, Lactate, INR and hepatic encephalopathy were considered. AUROC in derivation and validation cohort were 0.797 and 0.793 respectively. AARC ACLF score was developed with a minimum and maximum of 5 and 15. The score was better than the MELD and CLIF SOFA with an AUROC of 0.76, sensitivity 70 %, speciÞcity 67 %, PPV 78 % and NPV of 58 % in predicting 90 days survival. Grading was done with Grade A (5Ð9), Grade B (10Ð11) and Grade C (12Ð15 points). The mortality risk increases by 9.7 % with each unit increase. Score of 11 at baseline or persistence of the same in Þrst week associated with 100 % mortality in 30 days. Overall median survival was 26.3 days and that of Grade B, C being 16 and 5 days respectively and overall survival of 51.8 %.

Conclusion: The AARC ACLF score is dynamic, simple and better to the existing models. The deÞnitive therapies i.e. transplant can be predicted within Þrst week.

**Table-1: AARC ACLF Score**

| Points | Bilirubin (mg/dl) | HE Grade | INR | Lactate (mmol/lit) | Creatinine (mg/dl) |
|---|---|---|---|---|---|
| 1 | <15 | 0 | <1.5 | <1.5 | <1.0 |
| 2 | 15.01-22 | I-II | 1.5-2.5 | 1.5-2.5 | 1.01-1.5 |
| 3 | >22 | III- IV | >2.5 | >2.5 | >1.5 |

**Table-2: AARC ACLF Grade**

| Grade | Score |
|---|---|
| A | 5-9 |
| B | 10-11 |
| C | 12-15 |

Table-3: Comparison with CLIF SOFA, MELD, AARC ACLF Score without creatinine

| Score | AUROC | Cutoff | Sensitivity | Specificity | Positive Predictive Value | Negative Predictive Value |
|---|---|---|---|---|---|---|
| AARC Score | 0.76 | 9.5 | 70.4 | 66.8 | 77.9 | 57.6 |
| MELD | 0.74 | 30 | 69.6 | 69.7 | 80.0 | 57.4 |
| AARC Score without creatinine | 0.73 | 7 | 79.0 | 53.0 | 73.6 | 60.3 |
| CLIF-SOFA | 0.70 | 10.5 | 72.7 | 55.5 | 72.1 | 56.2 |

O-226

# COMPROMISED SDF1 PRODUCTION BY LIVER STROMAL CELLS SUPPRESS HEPATOCYTE SELF REPLICATION IN ACLF

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Background and aims: Acute insult triggers hepatic regeneration in both normal and chronic liver disease. Regenerative response is variable in ALF and ACLF. Regeneration in ALF mainly occurs through hepatocyte self replication. In ACLF, regeneration occurs mainly via activation and differentiation of hepatic progenitor cells. Underlying mechanisms for compromised hepatocyte proliferation in ACLF is not known. We investigated the role of hepatic microenvi-ronment in defective hepatocyte proliferation in ACLF.

Patients and Methods: Liver biopsy/explant and peripheral/hepatic vein blood was collected from ACLF (n = 33) and ALF (n = 21) patients. Plasma growth factors were analysed by Cytokine array. Growth factors levels in tissue was conÞrmed by RT-PCR. Immunohistochemistry was performed for CXCR7 and Ki67.

Results: ACLF plasma showed more than twofold decrease in AFP, HGF, LIF, M-CSF, SDF and increase in EGF and GM-CSF in comparison to ALF. Ki67 signiÞcantly correlated with serum HGF (R = 0.537, p&1t0.001) and SDF1 (R = 0.369, p = 0.006) levels in both ALF and ACLF. HGF and SDF1 protein level in serum (p \ 0.0002, p = 0.002) and mRNA levels (p = 0.002, p = 0.0155) in tissue were signiÞcantly decreased in ACLF as compared to ALF suggesting that decreased HGF and SDF1 might be responsible for poor hepatocyte replication in ACLF. RT-PCR analysis showed sig-niÞcantly reduced CXCR7, Id1 and Wnt2a mRNA levels in ACLF liver tissue in comparison to ALF. IHC shows decreased CXCR7 positive liver endothelial cells in ACLF. Data suggests decreased liver SDF1 might be responsible for defective CXCR7-Id1-HGF/Wnt2a signaling, leading to compromised hepatocyte proliferation in ACLF. Conclusion: SigniÞcantly lower SDF1 level might be responsible for decreased HGF production and subsequent compromised hepatocyte proliferation in Acute-on-chronic liver failure.

O-231

# A PREDICTIVE FORMULA FOR THE PROGNOSIS OF PATIENTS WITH ACUTE-ON CHRONIC LIVER FAILURE

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Background & aim: The prognosis of acute-on chronic liver failure (ACLF) is extremely poor. We aimed to establish methods for its severity to identify the patients with a poor prognosis.

Methods: The laboratory data at admission of 30 ACLF were evaluated. Three established prognosis prediction models (model for end stage liver disease [MELD]; MELD modiÞed by serum sodium concentration, [MELD-Na]; and the Japan hepatic encephalopathy prediction model [JHEPM]) were assessed using area under the receiver operating characteristic curve (AUROC) values.

Results: J-HEPM was able to predict the outcome of the ACLF subjects (AUROC, 0.93) although MELD and MELD-Na presented lower predictive value (AUROC; 0.438 or 0.439, respectively). The high MELD-Na or MELD scores in the deceased patients correlated with the PT-INR value (r = 0.837 or r = 0.719, respectively), while the high scores in the surviving patients correlated with serum crea-tinine level (r = 0.859 or r = 0.849, respectively).

Conclusions: The JHEPM effectively predicted the prognosis of liver failure in patients with ACLF.

P-0194

# COMPARISON OF LDT VS ETV IN PATIENTS WITH HEPATITIS B RELATED ACLF IN BANGLADESH

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Aim: To compare telbivudine and entecavir in treatment naive hep-atitis B virus(HBV)-related acute-on-chronic liver failure(ACLF-B). Methods: Nineteen patients aged 25Ð60 years recruited. Informed, written consent obtained. 7 females and 12 male. Acute insult was HBV ßare in all. All had detectable HBV-DNA (3.2 9 104Ð1.1 9 107 copies/ml). 13/19 (68.4 %) HBeAg-negative and 6/19 (31.6 %) HBeAg-positive. 12/19 (63.2 %) had ascites only, 5/19 (26.3 %) ascites plus hepatic encephalopathy (HE) and 2/19(10.5 %) HE only. Serum bilirubin was 6.5Ð31 mg/dl, ALT 530Ð4235 U/L, albumin 1.0Ð2.8 gm/L and INR [1.9. creatinine [1.5 mg/dL in 10 patients. Treatment at presentation were with tablet telbivudine (600 mg) (10/19) or tablet entecavir (0.5 mg) (9/19) orally daily. S. bilirubin, S. albumin, S. creatinine, INR and blood counts monitored weekly for 2 weeks, at 1 month and monthly for 2 months. HBV-DNA monitored at 2 weeks and at end of 3 months. Results: At 3 months, 8 expired. All had S. creatinine[ 1.5 mg/dL at baseline. Of them 2 presented with ascites only, 4 ascites plus HE and 2 HE only. 7/11 surviving were on telbivudine and 4/11 were on entecavir. Three had complete LFT normalization; all receiving tel-bivudine. LFT improvement was seen in 2 in each arm and remained steady in 4, again 2 each in each arm. HBV-DNA were unde-tectable in 6 after 2 weeks with 5 getting telbivudine. Nine had undetectable HBV-DNA after 3 months with 6 on telbivudine. None experienced HBeAg-seroconversion.

Conclusion: Study has shown that safety and efÞcacy of telbivudine and entecavir in ACLF-B with better survival with telbivudine. Study with large patient pool is warranted.

P-0195

# COMPARISON BETWEEN TDF VS ETV IN PATIENTS WITH HEPATITIS B RELATED ACLF IN BANGLADESH

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Aim: The aim is to compare tenofovir and entecavir in treatment naive hepatitis B related acute-on-chronic liver failure (ACLF-B) patients.

Methods: Thirty four patients aged 14Ð65 years recruited and informed written consent obtained from all. 4/34 (11.8 %) were females and 30/34 (88.2 %) males. Acute insult was HBV ßair in all. None had HCC. HBV-DNA was 1.8 9 104Ð8 9 108 IU/L at base-line. All had ascites and 11/34 (32.4 %) had hepatic encephalopathy (HE) in addition. None had only HE. At baseline, S. bilirubin was 5.3Ð33.5 mg/dl, ALT was 50Ð3028 U/L, S. albumin was 1.8Ð3 gm/L and INR [1.9. Treatment at presentation was with tablet tenofovir (300 mg) (19/34) or tablet entecavir (0.5 mg) (15/34) orally daily, pending HBV-DNA, planning discontinuation of treatment if HBV-DNA was undetectable. Biochemistry and haematology were moni-tored weekly for 2 weeks, at 1 month and monthly for 2 months. HBV-DNA was monitored at end of 3 months of treatment.

Results: At end of 3 months, 12/34 (35.3 %) patients expired. Cause of death was hepato-renal syndrome in 11 and sepsis in 1. Among the survivors, 16/22 (72.7 %) had received tenofovir and only 6/22(27.3 %) were on entecavir. Improvement of MELD score was seen in all survivors on entecavir and in all but 2 on tenofovir at end of follow up. However all who survived had undetectable HBV-DNA after 3 months in both arms.

Conclusion: Present study reconÞrms safety and efÞcacy of both tenofovir and entecavir in ACLF-B with much better survival with tenofovir. However for speciÞc conclusion, further study with larger patient pool is recommended.

P-0196

# SERUM APOA-V IN SURVIVAL PREDICTION OF HEPATITIS B VIRUS-RELATED ACUTE-ON-CHRONIC LIVER FAILURE

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Aim: Hepatitis B-related acute-on-chronic liver failure (HBV-ACLF) is a life-threatening condition and lipid metabolism disorder is com-mon in the development of disease. The aim of this study was to deÞne the characteristics of serum apolipoprotein A-V (apoA-V) concentration in HBV-ACLF.

Methods: A total of 330 HBV-ACLF patients were recruited in this study, and the relationships of serum apoA-V concentration with clinical variables were analyzed. The independent factors associated with the prognosis of HBV-ACLF were assessed by using the binary logistic regression model, and receiver operating characteristic (ROC) curve analysis was performed to assess the accuracy of serum apoA-V in predicting the survival of HBV-ACLF.

Results: Of the 330 patients, high to 209 patients (63.33 %) died in hospital or after being discharged from hospital. As compared to survivors, the non-survivors had signiÞcantly lower concentrations of serum apoA-V; serum apoA-V concentrations were positively corre-lated with PTA, and negatively correlated with interleukin-10, tumor necrosis factora, and iMELD scores. Though serum apoA-V, iMELD score and PTA were all independent factors to predict survival, serum apoA-V had the highest performance for the prediction of the survival of HBV-ACLF and the cut-off value of[480.00 ng/mL had a positive predictive value of 84.68 % and a negative predictive value of 92.23 %.

Conclusion: Serum concentration of apoA-V decreases signiÞcantly in non-survivors of HBV-ACLF, and serum apoA-V may be regarded as an early predictive marker for the prognosis of HBV-ACLF.

P-0197

# COMPARISON BETWEEN TDF VS TDF PLUS GCSF IN PATIENTS WITH ACLF-B IN BANGLADESH

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Aim: The aim is to compare tenofovir and tenofovir-GCSF combi-nation in treatment-naive HBV-related acute-on-chronic liver failure (ACLF-B).

Methods: Nineteen patients aged 18Ð62 years recruited and informed written consent obtained. 2/19 (10.5 %) were females and 17/19 (89.5 %) males. Acute insult was HBV ßair in all. None had HCC. HBV-DNA was 1.8 9 $10^2$Ð5.58 9 $10^6$ IU/L at baseline. All had ascites and 8/19 (42.1 %) had hepatic encephalopathy (HE) in addi-tion. None had only HE. At baseline, S. bilirubin was 6Ð43.4 mg/dl, ALT 36Ð1102 U/L, S. albumin 1.1Ð3.8 gm/L and INR [1.9. Treat-ment at presentation was with tablet tenofovir (300 mg) (9/19) or tenofovir (300 mg) plus GCSF (30 IU) sub-cutaneously daily for 6 days (10/19), pending HBV-DNA, planning treatment discontinu-ation if HBV-DNA undetectable. Biochemistry and haematology were monitored weekly for 2 weeks, at 1 month and monthly for 2 months. HBV-DNA was monitored at end of 3 months of treatment.

Results: At end of 3 months, 3/19 (15.8 %) patients expired. Cause of death was hepato-renal syndrome in 2 and variceal bleeding in 1. Among the survivors, 9/10 (90 %) received tenofovir- GCSF and 7/9 (77.8 %) received only tenofovir. Improvement of MELD score was in 9/10 (90 %) survivors on tenofovir-GCSF and in 7/9 (77.8 %) on tenofovir only at end of follow up. However all who survived had undetectable HBV-DNA after 3 months in both arms.

Conclusion: Present study reconÞrms safety and efÞcacy of both tenofovir and tenofovir-GCSF in ACLF-B with much better survival with tenofovir-GCSF. However for speciÞc conclusion, further study with larger patient pool is recommended.

P-0198

# DOWN-REGULATED MIR-181C WAS RELATED WITH HBV ASSOCIATED ACUTE-ON-CHRONIC LIVER FAILURE BY TNFALPHA

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Background: Hepatocyte apoptosis induced by tumor necrosis fac-tor(TNF)-a/TNFR1 is an important pathway for the incidence of fulminant viral hepatitis. Accumulating evidence suggests that a limited number of microRNAs (miRNAs) are involved in severe exacerbation of hepatitis B. The relationship between circulating miRNAs and HBV associated acute-on-chronic liver failure (HBV-ACLF) need to be further investigated.

Methods: miRNA expression proÞle by miRNA microarray analysis was performed on pooled Peripheral Blood Mononuclear Cell (PBMC) obtained from identiÞed groups of patients with chronic hepatitis B (CHB) or HBV-ACLF, respectively. Selected unnormal expressed miRNAs were veriÞed in more clinical samples by quan-titative real-time PCR (qRT-PCR). Targets were then subjected to a prediction by bioinformatics target prediction software. A luciferase reporter assay was conducted to conÞrm whether TNF-a is a direct target of Hsa-miR-181c.

Results: Our results showed 7 kinds of miRNAs were down-regulated and 9 kinds of miRNAs were up-regulated in the PBMC of HBV-ACLF patients by microarray. Expression of Hsa-miRNA-181c was signiÞcantly down-regulated in these patients by qRT-PCR. TNF-a was experimentally veriÞed as a target of Hsa-miR-181c.

Conclusion: Our data suggest a potential role for Hsa-miRNA-181c in the regulation of TNF-a expression in patients with HBV-ACLF.

P-0200

# THE DYSREGULATION OF ER STRESS RESPONSE IN AOCLF PATIENTS CAUSED BY ACUTE EXACERBATION OF CHB

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Background & Aims: Although endoplasmic reticulum (ER) stress is critical in various liver diseases, its role in acute-on-chronic liver failure (AoCLF) caused by acute exacerbation of chronic hepatitis B (CHB) is still elusive. This study aimed to analyze ER stress responses in the progression of HBV-related AoCLF.

Methods: Normal liver tissues (n = 10), liver tissues of CHB (n = 12) and HBV-related AoCLF patients (n = 19) are used. Electron micro-scopy of the ultrastructure of the ER was carried out on liver specimens. The gene and protein expression levels of ER stress-related genes were measured. We further analyzed the correlation between the expression levels of ER stress-related molecules and liver injury.

Results: Electron microscopy identiÞed typical features of the ER microstructure in AoCLF subjects. Among the three pathway of unfolded protein responses, the PKR-like ER kinase and inositol-requiring enzyme 1 signaling pathway were activated in CHB subjects and inactivated in AoCLF subjects, while the activating transcription factor 6 signaling pathway was sustained in activated form during the progression of AoCLF; the expression of glucose-regulated protein (Grp)78 and Grp94 were gradually decreased in AoCLF subjects compared to healthy indi-viduals and CHB subjects, showing a negative correlation with serum ALT, AST and TBIL; the ER stress-related apoptosis molecules were activated in the progression of acute exacerbation of CHB.

Conclusions: The dysregulated ER stress response may play a complicated role in the pathogenesis of AoCLF, and a severe ER stress response may predict the occurrence of AoCLF caused by acute exacerbation of CHB.

P-0205

# ACUTE ON CHRONIC LIVER FAILURE INDUCED BY HEPATIC INJURY IS CHARACTERIZED BY MASSIVE CELL DEATH

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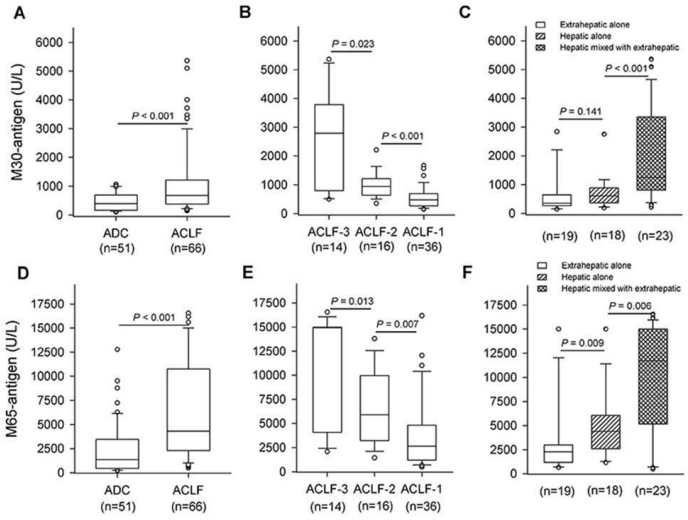
Background: Acute-on-chronic liver failure (ACLF) is a clinical entity which is distinctive from acute decompensated cirrhosis (ADC). Currently, much is unknown about ACLF. This study aims to investigate the role of hepatic cell death in ACLF.




Methods: 66 ACLF patients were identiÞed from a prospective cohort of 117 patients with ADC admitted to Rui-Jin Hospital from February, 2013 to August, 2014. ACLF was diagnosed as per EASL-CLIF criteria. This criteria was also used to subdivided the ACLF into three grade, ACLF-1, ACLF-2 and ACLF-3. The higher the grade is, the more organ fails. Hepatic cell death was assessed by the mea-surements of serum M30 (apoptosis) and M65 (total cell death) levels Results: Serum M30- and M65-antigen were signiÞcantly higher in ACLF than ADC (P \ 0.001). Patients with ACLF-3 presented the highest levels of M30- and M65-antigen followed by ACLF-2 and ACLF-1. SigniÞcant correlations (P \ 0.001) were found between M30- or M65-antigen and disease severity scores, CTP, MELD or CLIF-SOFA. ACLF was further categorized into two distinct groups according to the types of acute insults (hepatic or extrahepatic). Extrahepatic-ACLF group demonstrates relative low level of apop-tosis (P [ 0.05) and

signiÞcant lower total cell death (P \ 0.001) compared with hepatic-ACLF and were even close to that from ADC (P [ 0.05). However, cell death biomarkers markedly elevated in the ACLF group mixed with hepatic and extrahepatic insults .

Conclusions: ACLF demonstrates massive hepatic cell death which is absent from ADC. Acute hepatic rather than extrahepatic injury alone leads to such high level of cell death. But extrahepatic insults help exaggerate the hepatic cell death in ACLF patients with acute hepatic injury.

**Table 1. Correlation between cell death biomarkers and clinical parameters and scores**

| | M30-antigen | | M65-antigen | |
|---|---|---|---|---|
| | *r* | *P* | *r* | *P* |
| ALT | 0.462 | <0.001 | 0.454 | <0.001 |
| AST | 0.558 | <0.001 | 0.562 | <0.001 |
| TB | 0.423 | <0.001 | 0.51 | <0.001 |
| INR | 0.328 | <0.001 | 0.388 | <0.001 |
| Cr | 0.121 | >0.05 | 0.145 | >0.05 |
| MAP | -0.0763 | >0.05 | -0.1 | >0.05 |
| CTP | 0.511 | <0.001 | 0.565 | <0.001 |
| MELD | 0.415 | <0.001 | 0.489 | <0.001 |
| CLIF-SOFA | 0.478 | <0.001 | 0.551 | <0.001 |

Abbreviation: TB, total bilirubin; Cr, creatinine; MAP, mean arterial pressure; Child-Turcotte-Pugh; MELD, Model for end-stage liver disease; CLIF-SOFA, chronic liver failure-sequential organ failure

**Table 2. Categroy of acute insults for all the ACLF patients**

| Events | Frequency |
|---|---|
| Hepatic insults alone | 18(27.3%) |
| Reactivation of HBV | 12(18.2%) |
| Alcoholic hepatitis | 3(4.5%) |
| Flare up of AIH | 1(1.5%) |
| Hepatotoxic drugs | 2(3.0%) |
| Extrahepatic insults alone | 18(27.3%) |
| Bacterial infection | 12(18.2%) |
| UGIB | 6(9.1%) |
| Hepatic mixed with extrahepatic insults | 23(34.8%) |
| Unknown | 6(9.1%) |

Abbreviation: upper gastrointestinal bleeding

P-0207

# ACUTE-ON-CHRONIC LIVER FAILURE DEVELOPMENT CORRELATE WITH BASELINE PORTAL HYPERTENSION

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Background and aim: Acute-on-chronic liver failure (ACLF) is considered as main prognostic event in cirrhosis. Portal hypertension (PHT) and liver disease severity have been accepted as main prog-nostic factors in the development of acute decompensation which is essential precondition of ACLF. The aim of this study is evaluation about the relationship between baseline PHT, liver disease severity with the future ACLF development. Methods: 602 Cirrhotic patients (male 499, 82.9 %) have been prospectively followed for the development of ACLF and ARM. Baseline hepatic venous pressure gradient (HVPG) measurement, serologic tests were performed to all patients. The diagnosis of ACLF was based on EASL/AASLD criteria.

Results: 127 (21.1 %) patients developed more than once of ACLF. ACLF related mortality (ARM) was developed in 98 patients (16.3 %). In the univariate analysis, Child-Pugh score (CPS), MELD score, HVPG, CPS class C, MELD score over 15, clinically signiÞ-cant portal hypertension (CSPH), alcohol, total bilirubin (TB), albumin, INR, sodium, hemoglobin were signiÞcant for ACLF developments (P \ 0.05). In the multivariate analysis modeling with avoiding colinearity, CSPH is always signiÞcant predictor variables for ACLF development in multiple models. In KaplanÐMeier analy-sis, CSPH, MELD score over 15, CPS class C were signiÞcant for ACLF development (P \ 0.001). In univariate analysis, CPS, MELD score, HVPG, CSPH, TB and INR were statistically signiÞcant for ARM (P \ 0.05). In multivariate analysis, CPS, MELD score, CSPH and TB were signiÞcant for ARM (P \ 0.05).

Conclusions: Our data suggests that the development of ACLF and ARM were closely related with underlying liver disease severity (CPS, MELD score) and baseline clinically signiÞcant portal hypertension.

P-0211

# COMPARISON OF THE ACUTE ON CHRONIC LIVER FAILURE SEVERITY SCORE: A NEED FOR SIMPLE AND DYNAMIC ONE

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Background and aim: Acute on chronic liver failure is rapidly progressive liver failure with high short term mortality. A simple and dynamic prognostic model is needed for early and deÞnitive therapy. Aim was to compare the existing disease severity scores for predic-tion of 90 days survival in ACLF patients by APASL deÞnition Method: 1021 ACLF patientsÕ enrolled into the APASL ACLF Research Consortium (AARC) were analyzed. CTP, MELD, SOFA, CLIF SOFA, APACHE II and number of organ failure at baseline and delta change on D4 and D7 were compared with the new onset HE or AKI, change in lactate or bilirubin for prediction of 90 days mortality.

Results: The baseline MELD, CLIF SOFA, APACHE II, SOFA, CTP and number of organ failure as mortality predictor had AUROC of 0.74, 0.72, 0.71, 0.71, 0.68 and 0.68, respectively. Their delta change at D4 or D7 were poor predictor of outcome with AUROC\0.68. The new onset AKI [HR: 2.58 (1.76Ð3.75)], increase in total bilirubin by 6.1 mg/dl [HR: 2.16 (1.77Ð2.61)], new onset HE [HR: 2.14 (1.36Ð3.34)], increase in lactate by 1.48 meq/l [HR: 1.85 (1.45Ð2.34)] on D4 and D7 had a better hazard for mortality than SOFA [HR: 2.13 (1.41Ð3.20)], MELD [HR: 1.63 (1.33Ð1.99)], CLIF-SOFA [HR: 1.42 (1.06Ð1.90)], CTP [HR: 1.35 (1.07Ð1.69)] and APACHE II [HR: 1.81 (1.65Ð2.22)] change.

Conclusion: The existing disease severity score were poor predictor of outcome and lack dynamicity. The change in bilirubin, lactate with development HE

and/or AKI were good predictors. A model con-sidering above is strongly recommended.

**Table 1: Disease severity score at baseline as predictor of 90 day mortality (AUROC)**

| | AUROC | Cut off | Sensitivity | Specificity |
|---|---|---|---|---|
| MELD | 0.737 | 30.5 | 68.1 | 68 |
| CLIF SOFA | 0.720 | 12.5 | 66.7 | 64.2 |
| APACHE II | 0.711 | 14.5 | 70.8 | 64.2 |
| SOFA | 0.708 | 9.5 | 63.9 | 64.1 |
| CTP | 0.683 | 11.5 | 72.2 | 57.5 |
| No of Organ Failure | 0.682 | >2 | 47.5 | 79.2 |

**Table 2: Dynamic Parameters**

| Prognostic Parameters | Day 4 | P Value | Day 7 | P Value |
|---|---|---|---|---|
| New onset AKI | 2.58(1.76-3.74) | <0.001 | 1.95(1.23-3.09) | 0.004 |
| Delta Total Bilirubin | 2.16(1.77-2.61) | <0.001 | 1.68(1.34-2.10) | 0.039 |
| New onset HE | 2.14(1.36-3.34) | <0.001 | 1.87(0.86-2.85) | 0.039 |
| Delta Lactate | 1.85(1.45-2.34) | <0.001 | 1.55(1.14-2.10) | 0.005 |
| | | | | |
| SOFA | 2.13(1.41-3.20) | <0.001 | 1.75(1.08-2.84) | 0.022 |
| APACHEE-II | 1.81(1.65-2.22) | 0.005 | 1.42(1.39-1.90) | 0.171 |
| MELD Score | 1.63(1.33-1.99) | <0.001 | 1.72(1.36-2.15) | <0.001 |
| CLIF-SOFA | 1.43(1.06-1.90) | 0.017 | 1.32(0.90-1.93) | 0.145 |
| CTP score | 1.35(1.07-1.69) | 0.009 | 1.48(1.16-1.90) | 0.002 |

P-0213

# LIVER TRANSPLANTATION FOR PATIENTS WITH ACUTE-ON-CHRONIC LIVER FAILURE IN ASIA

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Aim: Acute-on-chronic liver failure (ACLF) is characterized by high mortality. Liver transplantation (LT) is effective in patients who do not improve with supportive measures. This study examines the outcome of ACLF patients who underwent LT in Asia.

Methods: Prospectively collected data from 17 Asian countries in the APASL ACLF Research Consortium was analyzed. 43 patients who underwent LT for ACLF were compared with 1657 non-transplanted ACLF patients. The variables analyzed include patient demographics, acute insult, background liver disease, severity scores (MELD and SOFA scores) and post-LT outcome.

Results: Mean age of LT patients was 42.1 years and non-trans-planted patients was 43.7 years. 74.4 % of LT patients and 85.1 % of non-LT patients were male. The most common acute liver insult was HBV reactivation (24.4 %) in LT patients, compared with alcohol (49.5 %) in non-LT patients. Three-month survival rate was 76.7 % in LT group, and 52.6 % in non-LT group. Mean MELD scores prior to transplant was (27.7 ± 4.7) and (30.5 ± 8.3) in non-transplant group. In LT patients, baseline renal dysfunction predicted mortality (mean

urea: 1.4 vs. 0.84 mg/dL, p = 0.015) (mean creatinine: 61 vs. 27 lmol/l, p = 0.042). High SOFA score was signiÞcantly associated with mortality in both LT (12.5 vs. 8, P = 0.015) and non-LT (8.3 vs. 10.9, p \ 0.001) patients. In non-LT patients, baseline urea (68.5 vs. 41.2 lmol/l, p \ 0.001), MELD (33.8 vs. 27.5, p \ 0.001) and Child-Pugh score (12 vs. 11, p \ 0.001) were independently associated with mortality.

Conclusion: Baseline renal dysfunction and higher SOFA score predict poorer LT outcome in ACLF patients

P-0214

# PRESENCE OF SIRS AND SEPSIS PREDICTS MORTALITY IN PATIENTS WITH ACUTE-ON-CHRONIC LIVER FAILURE

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Background: Acute-on-chronic liver failure (ACLF) is characterized by organ failure(s) and high short-term mortality. This study develops and validates a speciÞc prognostic score for ACLF patients.

Methods: Data from 1023 patients included in the prospective ACLF-AARC database were used. Prognosis prediction was done using established models of CLIF-SOFA, MELD, MELD-sodium (MELD-Na), and Child-Pugh (CPs) scores. The level of signiÞcance was set at 0.005.

Results: The database has enrolled 1023 patients with mean age 44.2 years (males 88.4 %) with predominant etiology being ethanol. Clinical predictors of mortality at baseline included presence of hepatic encephalopathy [grade 2 [Hazard ratio 4.3 (CI 3.4Ð5.5)], systemic inßammatory response syndrome [HR 2.5 (CI 2.2Ð3.2)], septic shock [HR 6.42 (CI 5.9Ð6.5)]. The presence of spontaneous bacterial peritonitis [HR 1.48 (CI 1.3Ð1.51)] was an independent predictor of mortality, the other sites of sepsis being pneumonia, urinary tract and skin and soft tissue infection. The new prognostic formula elaborated was as follows: ACLF Prognostic Index (API) = 0.02 9 total bilirubin (mg/dl) + 0.29 9 INR + 0.17 9 lac-tate (mmol/l) + 1.3 (if MAP\70 mmHg at baseline) + 0.46 (if serum creatinine [1.5 mg/dl at baseline). The area under the receiver operating characteristic curve of the API in predicting the outcome of patients with ACLF was 0.82, as opposed to 0.71 for the original MELD, 0.72 for MELD sodium and 0.78 for CLIF-C score (P \ 0.05).

Conclusion: The presence of SIRS and sepsis at ACLF diagnosis predicts mortality. A simple clinically relevant prognostic score can be used to stratify the risk of mortality in ACLF patients.

P-0216

# THE PROGNOSIS OF PATIENTS WITH ACUTE ON CHRONIC LIVER FAILURE WHO WERE ADMITTED TO ICU

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Background: ACLF is a characterized by acute decompensation of cirrhosis, organ failures and high short term mortality.
Material and method: From 2013/1/1 to 2013/12/31, patients who were admitted to our specialized GI intensive care unit due to ACLF were enrolled for our studies. Various parameters of seven different time points, including ICU 8Ð14 days were recorded for analysis.

Result: 93 patients fulÞll our inclusion and exclusion criteria. The mean inhospital days were 33.3 ± 28.5 days. The mean follow-up days were 223.9 ± 268.1 days. The 28 day mortality rate was 24.7 %. The inhospital mortality rate was 52.7 %. The diagnosis day 3Ð7 ACLF grade could predict 28 day mortality with AUROC area 0.841. However, for the prediction of inhospital mortality, ICU 8Ð14 day had highest AUROC area 0.844. Acute kidney injury and hepatic encephalopathy grade could predict inhospital mortality independently in addition to CLIF C ACLF scores.

Conclusion: The diagnosis day 3Ð7 ACLF grade could predict 28 day mortality. ICU 8Ð14 day ACLF grade could prediction inhospital mortality the highest among different time points. Acute kidney injury and hepatic encephalopathy grade could predict inhospital mortality independently in addition to CLIF C ACLF scores.

P-0297

# SERUM LEVEL OF MICRORNA IN THE PATIENTS WITH ACLF RELATED HBV INFECTION

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Aim: To identify the signiÞcant miRNAs and the inßuence factors of miRNAs in the patients with acute on chronic liver failure related hepatitis B virus infection.

Method: Sera collected from 41 CHB, 55 HBV-associated acute-on-chronic liver failure (ACLF) patients and 30 chronic asymptomatic carriers (ASC) were included in this study. In order to demonstrate whether miRNAs may have been correlated with the severity of HBV-related disease, we used microarray to investigate the miRNA expression proÞles in serum from ASC, CHB, ACLF patients. Those miRNAs with altered levels were further measured by quantitative reverse transcription polymerase chain reaction (qRT-PCR). The SPSS version 18.0 (SPSS Inc., Chicago, IL, USA) was used for the statistical analyses.

Conclusion: 1. MiRNA-146a-5p, 122-3p, 328-3p can be novel non-invasive biomarkers in HBV infection. The more severe inßammation is, the higher the expression level is. 2. The prognosis of ACLF are associated with INR, $Na^+$, MELD, hepatic encephalopathy, gas-trointestinal bleeding, lung infection, hepatorenal syndrome, 122-3P, 146a-5p, 328-3p. The predictive model of the prognosis of ACLF include four aspects: $Na^+$, INR, gastrointestinal bleeding, 122-3p, better than the MELD Score. The new model is Y=0.4029 $Na^+$ - 1.72 9 INR - 4.963 9 gastrointestinal bleeding N 0.278 9 122-3p + 50.449. 3. Four miRNA in each other have no cooperation in ACLF.

LBO-35

# DIAGNOSIS VALUE AND CORRELATION WITH IMMUNE REGULATORY FACTORS OF MSP IN ACLF PATIENTS

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Objective: To observe the expression of macrophage stimulating protein level in acute-on-chronic liver failure patients, and explore the clinical signiÞcance and correlation with different immune regulatory factors of this cytokine.

Method: Double antigen sandwich method to detect MSP in peripheral blood in 45 cases diagnosed with ACLF and 32 cases with chronic hepatitis B (CHB), the healthy peripheral blood serum as control. Compared the expression of MSP in different prognosis of patients with ACLF, detect the liver function, hepatitis B virus (HBV), peripheral blood $CD4^+$ interferon $c^+$, $CD4^+$ interleukin$4^+$, $CD4^+$ IL-17 $^+$, $CD4^+$ $CD25^+$ $FOXP3^+$ in different groups. Analysis of variance between different groups.

Results: MSP level was signiÞcantly higher in ACLF patients serum 1.65 ± 0.46 ng/mL than that in CHB group 1.43 ± 0.32 ng/mL and healthy control 1.23 ± 0.21 ng/mL P \ 0.01. CHB group and healthy control group had no statistical difference. ACLF survival group (1.82 ± 0.32 ng/m) ACLF death group 1.17 ± 0.22 ng/m, there was signiÞcant difference between the two groups P = 0.042. Lympho-cyte Th2, Th17 cells in the group of ACLF were higher than the other two groups P \ 0.01, while about Th1, Treg cells, there was no dif-ferent compare with the two groups, MSP level and Th2, Th17, Th17/ Treg lymphocyte number changes were positively correlated r = 0.386, 0.644, 0.605, P = 0.032, 0.000, 0.000.

Conclusion: MSP was involved in the progress of ACLF, there is a certain relationship between the level change of MSP, with the clinical outcomes and cellular immune imbalance in ACLF patients, MSP is very important to the future clinical diagnosis and treatment of acute-on-chronic liver failure.

LBP-038

# IL-9 AND IL-10 BUT NOT TH9 CELLS ARE ASSOCIATED WITH SURVIVAL OF PATIENTS WITH ACLF

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CD4+ T cell are reported to be essential for the immune response to HBV infection. Th9 cells are a new subset of CD4+ T cells and function through secreting interleukin (IL)-9 and IL-10. The present study aimed to investigate Th9 cells percentage as well as IL-9 and IL-10 levels in different stages of HBV infection and their relation-ship with progress and prognosis of liver disease. Serum samples were collected from 26 healthy controls (HC) and 85 patients with hepatitis B virus (HBV) infection, including 39 chronic hepatitis B (CHB) patients, 25 HBV-liver cirrhosis (HBV-LC) patients and 21 acute on chronic liver failure (ACLF) patients. The Th9 cells pro-portion and IL-9 and IL-10 levels were determined. The results showed that no difference of Th9 cells proportion as well as IL-9 and IL-10 levels were observed in different patient groups, in patients with different HBeAg status, and in HBV-LC patients with different complications. They were not related with inßammation index as well as prognosis indexes. In CHB patients receiving antiviral treatment, Th9 showed no change while IL-9 and IL-10 levels increased after treatment. Th9 cells showed no difference in ACLF patients survival or not, while IL-9 and IL-10 levels were signiÞcantly higher in non-survival ACLF patients. Furthermore, baseline IL-9 level showed the prognosis prediction with 87.5 % sensitivity and 61.5 % speciÞcity for ACLF patients. Our data indicated that Th9 cells were not involved in the pathogenesis of HBV infection while elevation of IL9 and IL-10 may be related to a bad prognosis of ACLF.

LBP-070

# PERIHEPATIC NODES BY POINT-OF-CARE ULTRASOUND IN ACUTE HEPATITIS AND ACUTE-ON-CHRONIC LIVER DISEASE

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*Aim: To study the manifestations of perihepatic lymph nodes during the episode of acute hepatitis ßare by point-of-care ultrasonography.*

Methods: One hundred and seventy-six patients with an episode of acute hepatitis ßare were enrolled retrospectively. Diagnosis of eti-ology of the acute hepatitis ßare was based on chart records and serological and virological assays. The patients were categorized into two groups (viral origin and non-viral origin) and further deÞned into ten subgroups according to the etiologies. An ultrasonograpy was performed within 2Ð72 h. The maximum size of each noticeable lymph node was measured. Correlation between clinical parameters and nodal manifestations was analyzed.

Results: Enlarged lymph nodes were noticeable in 110 (62.5 %) patients, mostly in acute on chronic hepatitis B (54.5 %). The viral group had a higher prevalence rate (89/110 = 80.9 %) and larger nodal size (median, 7 mm) than those of the non-viral group (21/ 66 = 31.8 %; median, 0 mm). Meanwhile, there were signiÞcant differences in the nodal size between acute and chronic viral groups, and between acute hepatitis A and non-hepatitis A viral groups. In logistical regression analysis, the nodal width still showed strong signiÞcance in multivariate analysis to stratify the two groups. The area under the curve of ROC was 0.805, with a sensitivity of 80.9%, a specificity of 68.2%, positive predictive value of 80.92 %, negative predictive value of 68.18 %, and an accuracy of 76.14 %.

Conclusion: Point-of-care ultrasonography to detect perihepatic nodal change is valuable for clarifying the etiologies in an episode of acute hepatitis ßare.

| | AFP | | | | PIVKA-II | | | | AFP+PIVKA-II |
|---|---|---|---|---|---|---|---|---|---|
| Cut-Offs | >10 | >20 | >100 | >400 | >40 | >48 | >70 | >150 | |
| HBV | | | | | | | | | >10; >70 |
| Sensitivity | 36.4 | 29.5 | 20.5 | 18.2 | 75.0 | 68.8 | 58.3 | 39.6 | 65.9 |
| Specificity | 97.1 | 100 | 100 | 100 | 76.5 | 85.3 | 94.1 | 94.1 | 91.2 |
| DA | 62.8 | 60.3 | 55.1 | 53.8 | 75.6 | 75.6 | 73.2 | 62.2 | 76.9 |
| HCV | | | | | | | | | >100; >48 |
| Sensitivity | 69.6 | 51.3 | 23.5 | 11.3 | 76.2 | 71.4 | 60.3 | 49.2 | 74.8 |
| Specificity | 44.4 | 75.6 | 95.6 | 100 | 57.1 | 69.4 | 73.5 | 91.8 | 66.7 |
| DA | 62.5 | 58.1 | 43.8 | 36.3 | 70.9 | 70.9 | 64.0 | 61.1 | 72.5 |
| Non-Viral | | | | | | | | | >100; >70 |
| Sensitivity | 36.7 | 24.1 | 17.7 | 10.1 | 84.5 | 81.0 | 73.8 | 57.1 | 77.2 |
| Specificity | 94.1 | 100 | 100 | 100 | 56.8 | 59.5 | 75.7 | 83.8 | 73.5 |
| DA | 54.0 | 46.9 | 42.5 | 37.2 | 76.0 | 74.4 | 74.4 | 65.3 | 76.1 |