WHAT’S HOT AT THE LIVER MEETING

Edward Gane
New Zealand Liver Transplant Unit
Auckland, New Zealand
Eradicating Hep C
GLE/PIB is highly effective in the Real World

Real-world Experience of 8-week G/P in 571 DAA-naïve, HCV GT1 or GT2, non-cirrhotic patients from 33 institutions in Japan

8-week G/P was highly effective with a favorable safety profile in DAA-naïve non-cirrhotic patients with HCV in a real-world settings in Japan

Ehira et al., AASLD 2019; #1580
Real-World Outcomes with GLE/PIB in historically difficult to treat marginalised populations

Pooled analysis of real-world studies from 9 countries (Nov 2017–Sep 2019) to evaluate the efficacy, safety and PROs in patients aged ≥18 years with HCV GT1–6 treated with GLE/PIB.

- SVR12 (% Patients)
  - Overall: 98.6 (1456/1477)
  - PWID: 98.0 (98/100)
  - Psychiatric Disorders: 99.3 (140/141)
  - Alcohol > 2 drinks/day: 96.5 (195/202)
  - Adherence < 90%: 90.9 (10/11)
  - Unemployed: 98.3 (354/360)
  - No secondary education: 98.2 (322/328)

- Patient-related outcomes (PROs) using SF-36
  - Mental functioning improved in 48%
  - Physical functioning improved in 42%

Lampertico et al., AASLD 2019; #1583
Assessment of adherence to GLE/PIB for 8 or 12 weeks across 2086 treatment-naïve patients treated in 10 Phase 3 trials

Predictors of non-adherence were psychiatric disorder and longer treatment duration

<table>
<thead>
<tr>
<th>Treatment interval:</th>
<th>Overall</th>
<th>G/P 8 weeks</th>
<th>G/P 12 weeks</th>
<th>G/P 8 weeks</th>
<th>G/P 12 weeks</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 (Wks 0–4)</td>
<td>100</td>
<td>98.4</td>
<td>97.1</td>
<td>100</td>
<td>98.4</td>
</tr>
<tr>
<td>2 (Wks 5–8)</td>
<td>100</td>
<td>97.3</td>
<td></td>
<td>100</td>
<td></td>
</tr>
<tr>
<td>3 (Wks 9–12)</td>
<td>100</td>
<td></td>
<td>97.2</td>
<td>100</td>
<td>99.4</td>
</tr>
</tbody>
</table>

Without cirrhosis

With compensated cirrhosis

Jacobson et al., AASLD 2019; #1515
8 weeks GP for HCV cirrhosis (EXPEDITION 8)

Objective: To evaluate the safety and efficacy of 8-week G/P in treatment-naïve adults with HCV GT1-6 infection and compensated cirrhosis.

Phase 3b, single arm, open-label, multicenter study

Treatment-naïve GT1-6 n = 343

Inclusion Criteria
- Aged ≥18 years
- Chronic HCV GT1-6 infection
- HCV treatment naïve
- With compensated cirrhosis (Confirmed by liver biopsy with a METAVIR score of 4 (or equivalent), a FibroScan score of ≥14.6 kPa, or a FibroTest score of ≥0.75 and an APRI >2)
- Absence of hepatocellular carcinoma

Exclusion Criteria
- Coinfection with HBV or HIV
- Platelets <50 x 10⁹ cells/L
- Albumin <2.8 mg/dL
- Total bilirubin >3 mg/dL
- Past or current evidence of decompensated cirrhosis

APRI, AST (aspartate aminotransferase) to Platelet Ratio Index; GT, genotype; HBV, hepatitis B virus.

8 weeks GP for HCV cirrhosis (EXPEDITION 8)

<table>
<thead>
<tr>
<th>SVR12 Rates for 8-week G/P in Patients with GT1–6</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
</tr>
<tr>
<td>---------</td>
</tr>
<tr>
<td>GT1-6</td>
</tr>
<tr>
<td>GT1</td>
</tr>
<tr>
<td>GT2</td>
</tr>
<tr>
<td>GT3</td>
</tr>
<tr>
<td>GT4</td>
</tr>
<tr>
<td>GT5</td>
</tr>
<tr>
<td>GT6</td>
</tr>
<tr>
<td>Non-VF*</td>
</tr>
<tr>
<td>1</td>
</tr>
<tr>
<td>2</td>
</tr>
</tbody>
</table>

- The overall SVR12 rates for GT1–6 were 99.7% in the PP population and 97.7% in the ITT population.

FDA and EMA label changed: MAVIRET for 8 weeks for ALL compensated treatment-naïve HCV

- All GT 3 patients with baseline NS5A resistance (A30K, Y93H) cured.
### 8 weeks GP for HCV cirrhosis (EXPEDITION 8)

<table>
<thead>
<tr>
<th>AE, n (%)</th>
<th>Total (N = 343)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Any AE</td>
<td>158 (46)</td>
</tr>
<tr>
<td>Any serious AE*</td>
<td>6 (2)</td>
</tr>
<tr>
<td>Any drug-related serious AE</td>
<td>0</td>
</tr>
<tr>
<td>Any AE leading to discontinuation of study drug</td>
<td>0</td>
</tr>
<tr>
<td>AEs occurring in ≥5% of patients</td>
<td></td>
</tr>
<tr>
<td>Fatigue</td>
<td>30 (9)</td>
</tr>
<tr>
<td>Pruritus</td>
<td>29 (8)</td>
</tr>
<tr>
<td>Headache</td>
<td>28 (8)</td>
</tr>
<tr>
<td>Nausea</td>
<td>19 (6)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Laboratory abnormalities (grade ≥3), n/N (%)*</th>
<th>Total (N = 343)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Alanine aminotransferase (&gt;5 × ULN)</td>
<td>1¹/342 (&lt;1)</td>
</tr>
<tr>
<td>Aspartate aminotransferase (&gt;5 × ULN)</td>
<td>0/342</td>
</tr>
<tr>
<td>Total bilirubin (&gt;3 × ULN)</td>
<td>0/342</td>
</tr>
</tbody>
</table>
Study on the common reasons for GLE/PIB treatment interruption and its impact on efficacy from data pooled from 13 Phase 3 clinical trials

Total interruptions: 33/2,839 (1.2%)
Interrupted days: 2 (1-62) days. median (range)

<table>
<thead>
<tr>
<th>Reason for interruption(s)*</th>
<th>8-week GLE/PIB (N=17)</th>
<th>12-week GLE/PIB (N=16)</th>
<th>All (N=33)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Adverse event</td>
<td>6 (35)</td>
<td>10 (63)</td>
<td>16 (48)</td>
</tr>
<tr>
<td>Missed dose†</td>
<td>9 (53)</td>
<td>5 (31)</td>
<td>14 (42)</td>
</tr>
<tr>
<td>Other</td>
<td>3 (18)</td>
<td>3 (19)</td>
<td>6 (18)</td>
</tr>
</tbody>
</table>

*Patients could have >1 reason for interrupting treatment and/or more than 1 treatment interruption. †Including: forgot to take dose (n = 10), misplaced dose (n = 3), and missed study visit refill (n = 1).

No patients who interrupted GLE/PIB had virologic failure

GLE/PIB is a very forgiving regimen!

Zamor al., AASLD 2019; #1553
GLE/PIB is safe and effective in young children

- 1200 HCV+ children infected through vertical transmission
  - 300 will be <18 years old and **200 <12 years old**
- GLE/PIB approved for all adolescents at adult dose
- Only IFN approved for < 12 yrs but learning and growth AEs

**Zamor al., AASLD 2019; #1553**

<table>
<thead>
<tr>
<th>Cohort</th>
<th>Age Range</th>
<th>Dose</th>
<th>Follow-up</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cohort Two</td>
<td>9 - 12 yrs</td>
<td>250 mg GLE + 100 mg PIB</td>
<td>12-week</td>
</tr>
<tr>
<td>Cohort Three</td>
<td>6 - 9 yrs</td>
<td>200 mg GLE + 80 mg PIB</td>
<td>12-week</td>
</tr>
<tr>
<td>Cohort Four</td>
<td>3 - 6 yrs</td>
<td>150 mg GLE + 60 mg PIB</td>
<td>4-week</td>
</tr>
</tbody>
</table>

**N=16**
GLE/PIB is safe and effective in young children

- 9 yr old relapsed with Y93H NS5A RASs
- 3 yr old refused medicine after D1
- PK equivalent to adult dosing
- No safety issues

Test all children of HCV+ women and treat all who are HCV RNA+
Preventing HCC
Is the risk of developing HBV-HCC lower with Tenofovir than with Entecavir?

ANRS CO22 Cohort: 1960 (all races) HBeAg+/patients who received tenofovir (1075) or entecavir (885) followed-up for a mean of in 45 months

PAGE-B Cohort: 1935 Caucasian adults HBeAg+/with or without compensated cirrhosis on ETV (n=772) or TDF (n=1163)

No difference in HCC risk between tenofovir and entecavir
Association Between Anti-Platelet Therapy and HCC Risk

Retrospective cohort study in patients receiving entecavir or tenofovir for ≥6 months

- All adult subjects with CHB prescribed with ETV and/or TDF, N = 55,119
  - 24,966 subjects excluded
  - Entecavir/TDF-treated CHB patients included in final analysis, N = 30,153
    - No HCC, N = 28,419
    - HCC, N = 1,734

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Multivariable analysis^</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Adjusted HR</td>
<td>95% CI</td>
<td>P value</td>
</tr>
<tr>
<td>Antiplatelet#</td>
<td>0.83</td>
<td>0.72 – 0.95</td>
<td>0.007</td>
</tr>
<tr>
<td>Aspirin monotherapy#</td>
<td>Referent</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Non-user#</td>
<td>1.12</td>
<td>0.96 – 1.30</td>
<td>0.152</td>
</tr>
<tr>
<td>DAPT#</td>
<td>0.72</td>
<td>0.54 – 0.97</td>
<td>0.029</td>
</tr>
</tbody>
</table>

Suggests long-term protection against HCC

Yip et al #094
Curing Hep B
HBV CURE Targets

Reduce Viral Burden

- Capsid allosteric modulator
  - NVR-3778
  - JNJ-3979
  - JNJ-440
  - ABI-40731
  - ABI-H2158
  - ABI-H3733
  - AT-130
  - BAY41-4109
  - HAP-12
  - GLS4JHS
  - HAP-R01
  - SBA-R01
  - AB-506
  - RG-7907
  - EP-027367
  - EDP-614
  - ALG-001024
  - GLP-26

- RNA destabilizers
  - AB-492
  - sRNA
  - AS0LNA
  - RO7052931
  - ETV
  - GS3228386
  - GS3389404
  - ISIS 5053583

- DNA editing
  - CRISPR Cas9
  - ZNF nuclease
  - ARC nuclease
  - TALENS

- Myrcludex B antibodies

- Entry inhibitors

- NAPs
  - REP2055
  - REP2139

- RNA helicase inhibitors

- PPAD5/7 inhibitor

- HB X-inhibitors

- FXR Agonists

- HDAC inhibitors

- Epigenetics

- siRNA

- CD8+

Activate Host Immunity

- RIG-I inhibitor

- TLR8 agonist
  - 1.GS-9668

- TLR7 agonist
  - 1.GS-9620
  - RO6864018
  - RO7020531
  - JNJ-54794964

- Vaccines
  - GS-4774
  - TG-1050
  - T-101
  - SCI-B-VAC

- CTL

- Antibodies

- IDO1

- Arginase

- Checkpoint inhibition

- Metabolic regulation

- Cytokines

- Cy Inhibitor

- TLR7 agonist

- Anti-PD-1/L1

- PD1 LNA

- Oral PDL1sm
CAMs have Dual Mechanisms of Action

‘Primary’ mechanism (‘empty capsid’ CAM)
Interference with capsid assembly kinetics, preventing encapsidation of pgRNA and blocking HBV replication
JNJ-0440 median EC$_{50}$/EC$_{90}$ = 22 nM/103 nM

‘Secondary’ mechanism
Inhibition of the de-novo formation of cccDNA by interfering with disassembly of the capsid
JNJ-0440 median EC$_{50}$/EC$_{90}$ = 136 nM/373 nM

1. Berke JM et al. AASLD, San Francisco, Nov 9–13, 2018; Poster 0402

HBV = hepatitis B virus; pgRNA = pregenomic RNA; HBsAg = hepatitis B surface antigen; NA = nucleos(t)ide analogue; CAM = capsid assembly modulator; EC$_{50}$/EC$_{90}$ = 50%/90% effective concentration; cccDNA = covalently closed circular DNA
Oral HBV capsid assembly modulator (CAMs)

- **Antiviral effect during 28 days dosing**

1. **NVR3-778**

   - NO change in HBsAg levels after 28 days

2. **JNJ-379**

3. **RO7049389**

4. **ABI-H0731**

   - 1200mg $\Rightarrow$ 2 log reduction
   - No effect on HBsAg
   - Skin rash

   - 250mg $\Rightarrow$ 2.9 log reduction
   - No effect on HBsAg
   - Occ ALT elevation

   - 200mg $\Rightarrow$ 3.2 log reduction
   - No effect on HBsAg
   - ALT elevation in 20%

   - 400mg $\Rightarrow$ 3.9 log reduction
   - No effect on HBsAg
   - Skin rash

*Will increased potency/duration achieve this 2’ MoA?*
Increased potency of 2nd Gen CAMs

AASLD 2019, Abstract #89:

(1) HBV DNA Levels

- Placebo
- 750 mg QD
- 750 mg BID

Mean (±SE) HBV DNA change from baseline (log10IU/mL)

(2) HBV RNA Levels

- Placebo
- 750 mg QD
- 750 mg BID

Mean (±SE) HBV RNA change from BL (log10 copies/mL)

Decline of HBV pgRNA likely reflects reduction in cccDNA

Gane EJ, et al. AASLD 2019, Boston, USA. #89
Oral HBV capsid assembly inhibitor (CpAMs)

AASLD 2019, Abstract #LP1: Longer Duration of 1st Gen CAMs

- Mean HBV DNA decline 6.1 $\log_{10}$
- Mean HBV RNA decline 3.0 $\log_{10}$
- Mean HBsAg decline 0.4 $\log_{10}$ (3 pts ≥1.0 log)

pgRNA and HBsAg declines support secondary MoA of CAMs (i.e. reduction in cccDNA)

Suggests all oral CAM + NUC may achieve functional cure but how long?
Inhibition of virion and SVP production
- Inhibition of HBV antigen expression could stimulate endogenous immune responses AND increase effectiveness of immunotherapies

Mechanism of Translation Inhibitors

- **Viral RNA**
  - **siRNA**
  - **cccDNA**
  - **DNA**
  - **Proteins**

### ASO/LNAs
1. RO7062931
2. GSK3228836
3. GSK3389404
4. ISIS 505358

### siRNAs
- ARC-520
- ARC-521
- ALN-HBV
- ARB-1467
- ARB-1740
- AB-729
- ARO-B/JNJ-3989
- GalXC-HBVS/DCR-HBVS
- ALN HBV02/VIR-2218

1. Gane E, et al. Poster #698
2. Yuen M-F, et al. Poster #695
4. Gane E, et al. Poster #696
5. Yuen M-F, et al. Poster #LP4
Translation Inhibitors in CHB Patients at AASLD

- siRNA in Phase 1b
  - 25-400mg monthly x3 subcut
  - Doses < 100mg less effective
  - No ALT elevations

- ASO in Phase 1b
  - 150–300 mg twice weekly x6 subcut
  - High rate of ALT elevations which presume immune-mediated (good)

Gane E, et al. AASLD 2019, #696

Yuen M-F, et al. AASLD 2019, #700
Translation Inhibition: other approaches at AASLD

2. mRNA destabilisers

- Small molecules target host poly-A polymerases PAPD5/7 (TENT4A/4B) which destabilise HBV transcripts from both integrated and cccDNA. Mueller Hepatol 2019; 69: 1398
  - Initial compounds associated with preclinical toxicity

- Gal-NAC LNA ASOs targeting host PAPD5/7
  - POC study in AAV-HBV mouse model (Poster #704)
    - decrease HBsAg in all animals - mean 2.3 log_{10}
    - 4/8 mice had sustained HBsAg loss with anti-HBs, i.e. achieved functional cure

Currently in Phase II in CHB patients at ACS

Mueller H, et al. AASLD 2019; #704
PD1/L1 blockage

- CHB characterised by immune exhaustion
- PDL1 blockage should restore effective intra-hepatic HBV-specific T-cell responses

- Single dose IV nivolumab 0.3mg/kg in CHB
  - 20/22 had reduction in HBsAg
  - One functional cure
  - Overall effect was small

- Dose will be limited by IR-AEs which can be prolonged and life-threatening
  - ACTG study exploring repeated doses
  - Need new approaches to PD1/L1 blockade

Inhibition of PD-L1 synthesis by LNA (Abstract #691)

- GalNAc-conjugated LNA ASO directed against PD-L1
  - Mice received 5 weekly subcut doses 5 mg/kg
    - 50% reduction in PD-L1 maintained for 8 weeks
    - 40-fold increase in liver HBV specific IFN-γ cells
    - 2.4 log reduction in HBsAg which was sustained
  
  Currently in Phase II in CHB patients at ACS
  Luangsay S et al. #691

Inactivation of PD-L1 by small molecule inhibitors

- Several small molecules can bind to and dimerise PD-L1 and inactivate the receptor
  - short lived PD effect improving safety if IR-AEs develop

Currently in Phase II in CHB patients at ACS

HBV CURE Combination Studies

Replication inhibition ± Antigen reduction ± Immune stimulation

- RNAi
- RNA destabiliser
- CAM
- PD1/L1 inhibitor
- RNAi
- Th. vaccine
- RNAi
- TLR Agonist
- RNAi
- PD1/L1 inhibitor
- RNA destabiliser
- PD1/L1 inhibitor
- RNA destabiliser
- TLR Agonist
HBV CURE Combination Studies

Triple therapy: siRNA plus CAM plus NUC for 12 weeks
- 12 eAg+/eAg- CHB patients
- Well tolerated,
- Robust antiviral activity after 3 months
  - HBV DNA decline up to 8 logs
  - HBV RNA decline up to 7 logs
  - HBsAg decline mean 1.8 logs

Next studies will be 48 weeks
How can we manage Fatty Liver Disease?
Weight loss in NASH

1. **Very Low Energy Diet (VLED)**
   - 113 patients with NAFLD cf. 100 without NAFLD
   - Primary endpoint was 5% weight loss after 3 & 6 months
     - In 67% non-NAFLD patients and 47% non-NAFLD (p<0.04).
     - Does NAFLD interfere with weight homeostasis?
       - Still worth trying in the most motivated NAFLD patients
       - Farrell A, et al. #65

2. **Bariatric surgery**
   - 868 morbidly obese patients underwent bariatric surgery with sleeve gastrectomy (SG) vs. Roux-en-Y gastric bypass (RYGB)
   - Paired liver bx at surgery and 6 years post-op
     - 181 F3/F4 at baseline
     - F3/F4 persisted in 45% despite 24 kg weight loss
     - RYGB better than SG for NASH fibrosis regression
     - Pais R, et al. #62
Weight loss in NASH

3. Duodenal mucosal resurfacing (DMR)
   • Endoscopic ablation of duodenal mucosa alters metabolic signalling ⇒ improved glycaemic control in T2DM in pigs and Chileans
   • 108 patients with T2DM and NASH randomised to DMR vs. SHAM
     – Endpoints assessed at 24 weeks post-procedure
     – No SAEs, transient epigastric pain in 15% DMR (vs. 6% SHAM)

<table>
<thead>
<tr>
<th></th>
<th>DMR</th>
<th>SHAM</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>(\Delta) MRI  PDFF</td>
<td>5.4%</td>
<td>2.4%</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>PDFF &gt;30%</td>
<td>54%</td>
<td>22%</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>(\Delta) HBA1C</td>
<td>-0.6%</td>
<td>-0.3%</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>(\Delta) Weight</td>
<td>-2.4 kg</td>
<td>-1.2 kg</td>
<td>&lt;0.05</td>
</tr>
</tbody>
</table>

Safe and well tolerated

Improvements still evident at 2 years post-DMR

Time to start considering this as treatment option?

Bergman J et al. AASLD 2019, #L02
### Is there a pill for NASH yet?

<table>
<thead>
<tr>
<th>Therapeutic Agent</th>
<th>Mechanism of Action</th>
<th>Duration</th>
<th>↓ fat &gt;30%</th>
<th>ΔWeight</th>
<th>ΔHBA1C</th>
<th>ΔLipids</th>
<th>AEs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cotadultide</td>
<td>GLP-1/glucagonR dual agonist</td>
<td>26 weeks</td>
<td>↓ CAP</td>
<td>↓</td>
<td>↓</td>
<td>↓</td>
<td>GI +</td>
</tr>
<tr>
<td>Licogliflozin</td>
<td>SGLT1/2 inhibitor</td>
<td>12 weeks</td>
<td>↓ 67%</td>
<td>↓</td>
<td>↓</td>
<td>-</td>
<td>GI ++</td>
</tr>
<tr>
<td>Tropefexor</td>
<td>FXR agonist</td>
<td>12 weeks</td>
<td>↓ 65%</td>
<td>↓</td>
<td>↓</td>
<td>↑ LDL</td>
<td>itch</td>
</tr>
<tr>
<td>Saroglitazar</td>
<td>PPARα/γ agonist</td>
<td>16 weeks</td>
<td>↓ 41%</td>
<td>-</td>
<td>↓</td>
<td>↓</td>
<td>nil</td>
</tr>
<tr>
<td>PF-05221304</td>
<td>Acetyl-CoA carboxylase inhibitor</td>
<td>16 weeks</td>
<td>↓ 90%</td>
<td>-</td>
<td>↓</td>
<td>↑ TAG</td>
<td>nil</td>
</tr>
</tbody>
</table>

Combination therapies will be needed that are synergistic, safe and affordable.
Last one for John
G-CSF in acute on chronic liver failure

• G-CSF induces of haemopoietic stem cells which could lead to hepatocyte regeneration and improved liver function
  – In single centre Indian RCT study G-CSF improved 60 day survival in 24 patients
    Garg, Gastro 2018

• Larger German RCT performed to validate results
  – 163 patients recruited at 18 centres 2016-2019
  – 70% had alcohol hepatitis
  – All fulfilled EF-CLIF criteria for ACLF
G-CSF in acute on chronic liver failure

Engelmann C, et al. LP-017
G-CSF in acute on chronic liver failure

No improvement in MELD

<table>
<thead>
<tr>
<th></th>
<th>G-CSF+SMT</th>
<th>SMT</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bacterial sepsis</td>
<td>32</td>
<td>34</td>
</tr>
<tr>
<td>Death from sepsis</td>
<td>3</td>
<td>2</td>
</tr>
</tbody>
</table>

Increased adverse reactions

<table>
<thead>
<tr>
<th></th>
<th>G-CSF+SMT</th>
<th>SMT</th>
</tr>
</thead>
<tbody>
<tr>
<td>SAEs - total</td>
<td>87</td>
<td>85</td>
</tr>
<tr>
<td>Multi-organ failure</td>
<td>2</td>
<td>0</td>
</tr>
<tr>
<td>Fulminant SBP</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Acute Liver failure</td>
<td>2</td>
<td>0</td>
</tr>
<tr>
<td>Acute renal failure</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Acute resp failure</td>
<td>1</td>
<td>0</td>
</tr>
</tbody>
</table>

- NO benefit form G-CSF in ACLF in any subgroup and possible harm from increased adverse drug reactions
- Ongoing Indian study shows benefit of 5 days bid G-CSF q12 weeks in 29 patients with decompensated cirrhosis

Singh V, et al. #101
Thank you!