• Hepatitis C is an infectious, blood–borne RNA virus
  • Up to $10^{12}$ viral particles ("virions") produced in an infected individual each day
  • HCV RNA PCR can detect infection with as few as 12 virions (IU/mL)
• Primarily affects the liver with the immune response causing hepatocyte death and fibrosis
  • ALT fluctuates month–to–month but progression to cirrhosis is variable (avge. 30 yrs; range 5–50 yrs)
• Infection persists in approximately 75–85% of those infected ("chronic Hep C")
  • HCV IgG identifies “exposure” but only HCV RNA PCR (i.e. test for viraemia) identifies “infection”
• Up to 7 major “genotypes” (genetic subtypes of virus with slightly differing characteristics)
  • In Australia: GT1 (50%) > GT3 (40%) > GT2 (5%) > GT4 (3%) > other/mixed (2%)

From Holmes and Thompson. Hepatic Medicine: Evidence and Research 20
• Combinations of drugs types to
  • Increase efficacy
  • Minimise resistance

• Analagous to HIV treatment
  • But MUCH easier

• Recent efforts:
  • Improve efficacy further
  • Minimise side-effects
  • Optimise pill burden/compliance

---

### Table B (A). Treatment protocols after liver transplantation for hepatitis C virus (HCV) infection in people with compensated liver disease

<table>
<thead>
<tr>
<th>HCV Geno</th>
<th>Treatment regimen</th>
<th>Duration</th>
<th>PBS listing</th>
</tr>
</thead>
<tbody>
<tr>
<td>1-6</td>
<td>Sofosbuvir 400 mg, orally, daily + Velpatasvir 100 mg, orally, daily</td>
<td>12 weeks</td>
<td>The combination of sofosbuvir + velpatasvir is PBS-listed for the treatment of GT 1-6 HCV</td>
</tr>
<tr>
<td>1</td>
<td>Sofosbuvir 400 mg, orally, daily + Ledipasvir 90 mg, orally, daily + Ribavirin 1000/1200 mg, orally, daily (weight-based)</td>
<td>12 weeks (24 weeks if ribavirin intolerant)</td>
<td>The combination of sofosbuvir + ledipasvir is PBS-listed for the treatment of GT 1 HCV. Ribavirin is not PBS-listed for use in combination with sofosbuvir + ledipasvir</td>
</tr>
<tr>
<td>1, 3</td>
<td>Sofosbuvir 400 mg, orally, daily + Dasabuvir 60 mg, orally, daily + Ribavirin 1000/1200 mg, orally, daily (weight-based)</td>
<td>12 weeks (24 weeks if ribavirin intolerant)</td>
<td>The combination of sofosbuvir + dasabuvir is PBS-listed for the treatment of GT 1 and 3 HCV. Ribavirin is PBS-listed for use in combination with sofosbuvir + dasabuvir</td>
</tr>
<tr>
<td>1a, 1b plus prior non-response to pegIFN plus ribavirin</td>
<td>Paritaprevir–ritonavir (150 mg/100 mg), orally, daily + Ombrabibavir 25 mg, orally, daily + Dasabuvir 250 mg, orally, twice daily + Ribavirin 600-800 mg, orally, daily</td>
<td>24 weeks</td>
<td>For GT 1a HCV. PegIFN + ribavirin is PBS-listed for 24 weeks' treatment duration only for people with cirrhosis and prior null response to pegIFN plus ribavirin. PBS listing for other situations is for 12 weeks' treatment duration</td>
</tr>
<tr>
<td>1a, 1b plus treatment-naive</td>
<td>Paritaprevir–ritonavir (150 mg/100 mg), orally, daily + Ombrabibavir 25 mg, orally, daily + Dasabuvir 250 mg, orally, twice daily + Ribavirin 600-800 mg, orally, daily</td>
<td>24 weeks</td>
<td>For GT 1b HCV. PegIFN + ribavirin is PBS-listed for 12 weeks' treatment duration only</td>
</tr>
</tbody>
</table>

*GT = genotype; PBS = Pharmaceutical Benefits Scheme; pegIFN = peginterferon-alfa; PKOCO = paritaprevir–ritonavir – ombrabibavir – dasabuvir; mODI = maximal dose of ribavirin.
* Addition of ribavirin may be considered for all patients in the post-transplant setting.
* Ribavirin dosing to weight based: recommended dose is 1000 mg for people weighing > 75 kg and 1000 mg for people weighing > 75 kg.
* PKOCO is associated with drug–drug interactions that require dose modification of ribavirin. Use in combination with mODI interferons is not recommended.
CASE STUDIES
CASE STUDY #1

• 26 year-old man
• History of iv speed use from age 17 to 23
  • Recent split with his girlfriend of 4 years
• Complaining of fatigue, affecting his work as a spray painter
• Otherwise well, no regular medications
• Blood tests: Hb 145, Plts 300, Bili 10, ALP 95, **GGT 75**, ALT 85, AST 45
WHAT IS YOUR NEXT STEP?

(a) Reassure him – his blood tests are mostly normal and the fatigue should improve
(b) Prescribe an anti-depressant
(c) Check viral serology (EBV, CMV, Hep B, Hep C)
(d) Do a full CLD screen (including Fe studies, alpha1–AT and Cu studies)
CASE STUDY #1 – INITIAL RESULTS

• EBV IgG and IgM negative; CMV IgG positive, IgM negative

• HBV sAg negative; HCV IgG positive
  • (NB. HBV DNA level is required if HBV sAg positive)

• The patient asks: “Should my Hep C be treated?”
WHAT DO YOU TELL YOUR PATIENT?

(a) Yes – but the treatment has lots of side-effects; his fatigue should improve by itself

(b) Yes – but Hep C is never completely cured so he should wait for a while

(c) No – because the Hep C hasn’t caused enough liver damage to warrant it

(d) Yes – treatment is easy and if he gets rid of it now, it will be like he never had it
DIRECT-ACTING ANTIVIRALS (DAAs)

- Estimated 227,000 people with Hepatitis C in Australia
  - Approx. 80–85% of these have been diagnosed (i.e. positive Hep C serology result)
  - Many do not know they are infected or have not been referred for treatment
- Only increased drug efficacy and treatment uptake will impact on patient outcomes
  - Can reduce projected liver-related deaths universal Hep C clearance
- Highly effective (approx. 95% of patients will eradicate Hep C with DAA treatment)
  - Mostly require 12 weeks of treatment only; few side-effects (if any) and rarely severe
- PBS–listed regimens for all genotypes and unrestricted access to all patient types

Australian Consensus Statement 2017
WHAT SHOULD YOU DO NEXT?

(a) Refer him to a Specialist Clinic – no further testing is needed
(b) Check HCV genotype and RNA PCR level and then refer to a Specialist Clinic
(c) Start him on 12 wks of SOF/VEL – you’ve used it before and it’s “pan-genotypic”
(d) Do a full CLD screen (including HCV GT & PCR) then ask a Specialist for advice
(e) Send him to a nearby Sexual Health Clinic where they do Hep C treatment
MODELS OF HEP C TREATMENT

• Specialist clinics
  • Easy for GPs and very efficient – provided patients are appropriately-referred
  • Patients typically start DAAs at their 1st or 2nd appointment (mostly limited by not knowing genotype)
  • Major downside is waiting list and capacity (variable across the MNHHS)

• Specialist-led community-based care
  • Can be Nurse-led model of care, usually in consultation with specialists
  • PBS now allows for experienced NP-prescribing in addition to GP-prescribing
  • Advantages in certain patient populations (e.g. PWID, opiate-substitution therapy, incarcerated)

• Novel models of care (e.g. CURE-IT – “Queensland’s Project ECHO”)
  • Hepatologist/CNC/Psychologist/Pharmacist MDT
  • 2 x Telehealth sessions per week to verify plan with GP
  • Have commenced 460 patients on treatment to date (NGO referrals, Metro and regional GPs)
MODELS OF HEP C TREATMENT

• GP-led community-based care
  • Requires “experienced prescriber” or “consultation with an experienced (specialist)”
  • “Experienced” is not defined and can be time-consuming for GPs to complete formally
  • Enormous advantages for patients (especially vulnerable or marginalized patients)
  • Obstacle is accurate fibrosis assessment/identifying cirrhosis ± access to specialist advice/support

• Anticipated that GP-led treatment will become the predominant model of Hep C care
  • Treatment uptake must increase to reduce the burden of cirrhosis and its complications

• This can be achieved with education, innovation and determination
CASE STUDY #2

• 57 year-old businessman, history of IVDU (1979–1982)
  • Diagnosed with Hepatitis C in 2005 (routine GP check–up; treatment–naïve)

• Other PMHx of:
  • NSTEMI with 2 x stents to LAD in 2014
  • Hypertension, Dyslipidaemia, Ex–smoker (since 2014, 60 PYH)
  • GORD complicated by short–segment Barrett’s Oesophagus
  • Alcohol misuse (average of ½ carton of XXXX Gold plus 4 bottles of wine per week)

• Meds include:
  • Diltiazem SR 240mg mane, Esomeprazole 40mg nocte, Atorvastatin 40mg nocte,
    Aspirin 100mg mane, Rivaroxaban 5mg bd

• No known drug allergies
WHAT ELSE WOULD YOU LIKE TO KNOW?

• Examination findings: HR 75 regular (SR), BP 140/75, BMI 31, central obesity, palmar erythema, hepatomegaly

• Baseline blood tests: Hb 140, Plts 170, Cr 88, Alb 38, Bili 14, INR 1.1, ALP 90, GT 140, ALT 54, AST 56

• Hep C genotype: GT 3a (+ HBVsAg neg, HBVcAb neg, HIV neg)

• Ultrasound findings: Hepatomegaly, increased echogenicity, coarse echotexture, no space-occupying lesions; Spleen 12cm, PV 14mm diameter with normal flow
IS THIS 57 YEAR-OLD MAN CIRRHOTIC?

(a) No
(b) Yes
(c) Probably
(d) Possibly but very unlikely
(e) Can’t decide on the information given
ANYTHING ELSE YOU NEED TO KNOW?

- Any OTC or complimentary/herbal meds?  
  No
- Details of coronary stenting in 2014?  
  Difficult anatomy, needs *some* antiplatelet (i.e. at least aspirin)
- Are there any DDIs of significance?  
  Yes (discussed later…)
- Risks of interrupting/reducing/changing meds?  
- What does he know about Hep C and treatment?  
  Nothing but very interested
WHAT IS THE MOST APPROPRIATE WAY TO ASSESS THIS PATIENT’S FIBROSIS LEVEL?

(a) Liver elastography
(b) APRI or FIB–4 Scores
(c) Abdominal CT Scan or MRI Liver
(d) Ultrasound elastometry (ARFI)
(e) Liver biopsy
WHAT IS THE MOST APPROPRIATE WAY TO ASSESS THIS PATIENT’S FIBROSIS LEVEL?

(a) Liver elastography
   Median TE 13.8 kPa
(b) APRI or FIB-4 Scores
   APRI 0.824, FIB-4 2.56
(c) Abdominal CT Scan or MRI Liver
   No HCC or overt features of PHT
(d) Ultrasound elastometry (ARFI)
   1.75 m/s
(e) Liver biopsy
   “Suggestive of early cirrhosis”
   “At least F3 (of 4)”
NON–INVASIVE ASSESSMENT OF LIVER FIBROSIS

• Most tests are measured according to their “accuracy” measured by area under ROC curve
  • AUROC Curve > 80% is “good”; AUROC > 90% is “excellent”
  • Measured against liver biopsy (or expert opinion) – imperfect gold standard
  • Clinical practice: Use “cut-offs” to exclude or diagnose cirrhosis (based on Sens and Spec)

• US–based liver stiffness preferred but limited access (often involves Specialist referral anyway)
  • Med stiffness measure < 12.5kPa “excludes cirrhosis (Sens 84%) and is recommended cut–off in clinical practice

• Serum biomarkers have reasonable accuracy (AUROC > 80%) and are much more accessible
  • Generally measure markers of advanced fibrosis/cirrhosis (i.e. AST:ALT ratio, platelet count, age)
  • Excluding cirrhosis: APRI < 1.0 has a Sens of 76%; FIB–4 < 1.45 has a Sens of 74%
  • ELF Test (Age, TIMP–1, P3NP, hyaluronic acid) is commercial (S&N $195) but slightly better (AUROC 88%)

• NB. “Post–test probability” is affected by test performance and “pre–test probability”
  • Prevalence of cirrhosis likely to vary across practices (i.e. General Practice vs. Hospital Clinic)
WHAT DAA OPTIONS ARE THERE FOR HIM?

(a) Sofosbuvir + Velpatasvir for 12 weeks
(b) Sofosbuvir + Daclatasvir for 12 weeks
(c) Sofosbuvir + Daclatasvir for 24 weeks
(d) Sofosbuvir + Daclatasvir + Ribavirin for 12 weeks
(e) Pr-O-D + Ribavirin for 12 weeks
(f) Sofosbuvir + PegIFN/Ribavirin for 12 weeks
**WHAT DAA OPTIONS ARE THERE FOR HIM?**

(a) Sofosbuvir + Velpatasvir for 12 weeks  
**YES**

(b) Sofosbuvir + Daclatasvir for 12 weeks  
**NOT IF CIRRHOTIC**

(c) Sofosbuvir + Daclatasvir for 24 weeks  
**YES**

(d) Sofosbuvir + Daclatasvir + Ribavirin for 12 weeks  
**YES**

(e) Pr-O-D + Ribavirin for 12 weeks  
**NO – GT1 ONLY**

(f) Sofosbuvir + PegIFN/Ribavirin for 12 weeks  
**YES – BUT TOXICITY**
GENOTYPE 3 – THE “PROBLEM CHILD” IN THE ERA OF UNIVERSAL ACCESS HEPATITIS C TREATMENT
HOW EFFECTIVE ARE DAA REGIMENS IN PATIENTS WITH GENOTYPE 3 CIRRHOSIS?

- 12 weeks of Sofosbuvir + NS5A-inhibitor (DCV) not good enough in cirrhosis
- Extended to 24 weeks or addition of RBV for more acceptable SVR rates
- Now have option of SOF/VEL but SVR drops if previous PegIFN/RBV treatment failure
  - Consider adding RBV in these patients

<table>
<thead>
<tr>
<th>DAA Regimen</th>
<th>SVR Rate (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>GT3 Cirrhosis, Treatment naïve</td>
<td>68%</td>
</tr>
<tr>
<td>GT3 Cirrhosis, PegIFN failure</td>
<td>85%</td>
</tr>
</tbody>
</table>

WHAT DRUG–DRUG INTERACTION/S SHOULD YOU BE WORRIED ABOUT?

• Diltiazem?
• Esomeprazole?
• Atorvastatin?
• Aspirin?
• Rivaroxaban?
• All of them?

Adapted from Dick et al. Hepatology 2016.
<table>
<thead>
<tr>
<th>Drug Class</th>
<th>LDV↓</th>
<th>Concomitant Drug↑</th>
<th>SOF↓</th>
<th>Concomitant Drug↑</th>
</tr>
</thead>
<tbody>
<tr>
<td>Azole antifungals</td>
<td></td>
<td>ND</td>
<td></td>
<td>ND</td>
</tr>
<tr>
<td>Calcium channel blockers</td>
<td></td>
<td>ND</td>
<td></td>
<td>ND</td>
</tr>
<tr>
<td>Erythromycin</td>
<td></td>
<td>ND</td>
<td></td>
<td>ND</td>
</tr>
<tr>
<td>HMG-CoA reductase inhibitors</td>
<td></td>
<td>ND↑</td>
<td></td>
<td>ND</td>
</tr>
<tr>
<td>Methadone</td>
<td></td>
<td>ND</td>
<td></td>
<td>ND</td>
</tr>
<tr>
<td>Phosphodiesterase inhibitors</td>
<td></td>
<td>ND</td>
<td></td>
<td>ND</td>
</tr>
<tr>
<td>Proton pump inhibitors†</td>
<td></td>
<td>ND</td>
<td></td>
<td>ND</td>
</tr>
<tr>
<td>St. John’s wort</td>
<td></td>
<td>ND</td>
<td></td>
<td>ND</td>
</tr>
<tr>
<td>Warfarin</td>
<td></td>
<td>ND</td>
<td></td>
<td>ND</td>
</tr>
</tbody>
</table>

* Denotes using caution and/or additional monitoring may be necessary when administering the respective medications together.


Liverpool University HEP Drug Interactions Website
www.hep-druginteractions.org/checker
• Not very hard and, in reality, few clinically significant DDIs BUT

• Mandatory in each case + work out a plan prior to starting!
WHAT WILL YOU DO IN HIS CASE IF YOU PRESCRIBE 12 WEEKS OF SOF/VEL?

• Diltiazem?
  Consider a mild dose reduction and/or observe BP

• Esomeprazole?
  Reduce dose to 20mg nocte or cease and use PRN
  Need to warn about timing of PPI dose with DAA dose

• Atorvastatin?
  Confirm safety of reducing dose (i.e. plaque stabilisation)

• Aspirin?
  Stay on aspirin unchanged

• Rivaroxaban?
  Confirm safety of only taking aspirin with Cardiologist
  If he needs DAPT then warn and monitor for bleeding

• Others?
  Tell patient to notify you if given any other meds
  (and check the Liverpool University Website!)
CASE STUDY #2 – PROGRESS ON TREATMENT

<table>
<thead>
<tr>
<th>Symptoms</th>
<th>Week 2</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mild insomnia</td>
<td></td>
</tr>
<tr>
<td>Feels “fuzzy”</td>
<td></td>
</tr>
<tr>
<td>BP 140/80</td>
<td></td>
</tr>
<tr>
<td>Missed 1 x tab</td>
<td></td>
</tr>
<tr>
<td>Symptoms</td>
<td></td>
</tr>
<tr>
<td>Bloods</td>
<td>ALT 58, AST 60 Hb 140, Bili 16</td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td>Action</td>
<td>Reassure and continue</td>
</tr>
<tr>
<td></td>
<td>Review in 1-2 weeks</td>
</tr>
</tbody>
</table>

**What do you do?**

(a) Stop treatment
(b) Refer to a specialist
(c) Continue but review in 1–2 wks
(d) Check HCV PCR, stop if RNA pos
(e) Reassure patient and continue
CASE STUDY #2 – PROGRESS ON TREATMENT

• What do you do?
  (a) Stop treatment
  (b) Refer to a specialist
  (c) Continue but review in 1–2 wks
  (d) Check HCV PCR, stop if RNA pos
  (e) Reassure patient and continue

<table>
<thead>
<tr>
<th>Symptoms</th>
<th>Week 2</th>
<th>Week 4</th>
</tr>
</thead>
<tbody>
<tr>
<td>Symptoms</td>
<td>Mild insomnia</td>
<td>Mod fatigue</td>
</tr>
<tr>
<td></td>
<td>Feels “fuzzy”</td>
<td>O’wise well</td>
</tr>
<tr>
<td></td>
<td>BP 140/80</td>
<td>100% adherence</td>
</tr>
<tr>
<td></td>
<td>Missed 1 x tab</td>
<td></td>
</tr>
<tr>
<td>Bloods</td>
<td>ALT 58, AST 60 Hb 140, Bili 16</td>
<td>ALT 50, AST 65 Hb 135, Bili 22 Lipase 80</td>
</tr>
<tr>
<td>Action</td>
<td>Reassure and continue</td>
<td></td>
</tr>
</tbody>
</table>

Mild symptoms
Missed 1 x tab
BP 140/80
ALT 58, AST 60 Hb 140, Bili 16
ALT 50, AST 65 Hb 135, Bili 22 Lipase 80

Reassure and continue
## CASE STUDY #2 – PROGRESS ON TREATMENT

<table>
<thead>
<tr>
<th>Symptoms</th>
<th>Week 2</th>
<th>Week 4</th>
<th>Week 6</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mild insomnia</td>
<td>Mod fatigue</td>
<td>Feels well</td>
</tr>
<tr>
<td></td>
<td>Feels “fuzzy”</td>
<td>O’wise well</td>
<td>100% adherence</td>
</tr>
<tr>
<td></td>
<td>BP 140/80</td>
<td>100% adherence</td>
<td>100% adherence</td>
</tr>
<tr>
<td></td>
<td>Missed 1 x tab</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bloods</td>
<td>ALT 58, AST 60 Hb 140, Bili 16</td>
<td>ALT 50, AST 65 Hb 135, Bili 22 Lipase 80</td>
<td>ALT 24, AST 28 Hb 135, Bili 12 Lipase 40</td>
</tr>
<tr>
<td>Action</td>
<td>Reassure and continue</td>
<td>Review in 1–2 weeks</td>
<td></td>
</tr>
</tbody>
</table>
CASE STUDY #2 – OUTCOME

- EOT Bloods:
  - ALT 20, AST 22, Bili12, Lipase 20
  - HCV RNA not detected
  - Meds returned to pre-treatment doses

- 12 wks post-EOT Bloods:
  - HCV RNA not detected (SVR!!)
  - Repeat US showed no HCC or ascites

- Ongoing management:
  - 6-monthly review for surveillance
  - Remains well, energy levels improved
  - Referred to local Liver Clinic
OTHER PATIENT TYPES?

• Genotype 2, 4, (5), 6?
  
  SOF/VEL is very effective (SVR > 95%)

• Genotype 1?
  
  Multiple regimens available (SVR ≥ 95%)

• Genotype 1 and **terrible** GORD?
  
  EBR/GZR has no DDIs with PPIs

• Cirrhotic patients?
  
  Generally recommend specialist treatment

• Decompensated cirrhosis?
  
  Specialist referral mandatory

• DAA–experienced patients?
  
  Specialist referral mandatory ("RASs")

• Coinfected individuals?
  
  Suggest referral to experienced specialist
WHY SHOULD I TREAT HEPATITIS C?
WHY SHOULD I TREAT HEPATITIS C?

• SVR confers all-cause mortality benefit

Cirrhotic HCV with SVR vs. no SVR

All-comer HCV with SVR vs. untreated

Terrault and Hassanein Hepatology 2016
WHY SHOULD I TREAT HEPATITIS C?

• SVR confers all-cause mortality benefit

• Reduced incidence of HCC post-SVR

Adapted from El-Serag et al. Hepatology 2016
WHY SHOULD I TREAT HEPATITIS C?

• SVR confers all-cause mortality benefit

• Reduced incidence of HCC post-SVR

• Patients *feel* much better, better QOL, less stigma
WHY SHOULD I TREAT HEPATITIS C?

• SVR confers all-cause mortality benefit
• Reduced incidence of HCC post-SVR
• Patients feel much better, less stigma, better QOL
• Gov’t has invested $3 billion in clearing HCV
WHY SHOULD I TREAT HEPATITIS C?

- SVR confers all-cause mortality benefit
- Reduced incidence of HCC post-SVR
- Patients feel much better, less stigma, better QOL
- Gov’t has invested $3 billion in clearing HCV
- Reduced healthcare burden, increased contribution to society

Connolly et al. J Med Econ 2017
WHY SHOULD I TREAT HEPATITIS C?

• SVR confers all-cause mortality benefit
• Reduced incidence of HCC post-SVR
• Patients feel much better, less stigma, better QOL
• Gov’t has invested $3 billion in clearing HCV
• Reduced healthcare burden, increased contribution to society
• Enormous job satisfaction to cure a chronic illness
TAKE HOME MESSAGES

• Look for Hep C in your patient (abnormal LFTs, risk factors)
  • HCV IgG serology (+ HCV RNA PCR & Genotype if positive to confirm current infection)

• Should you refer to a specialist for treatment?
  • Unwilling or uncomfortable with GP–led treatment (not “experienced”)
  • Cirrhotic or “complicated” patient (coinfection, co–morbidities that will impact treatment)
  • Previous exposure to Hep C treatment

• Advice is only a phone call away
  • Lots of online resources as well (www.gesa.org.au/resources/hepatitis-c-treatment/)

• GP–led treatment in consultation with a specialist is VERY do–able
TAKE HOME MESSAGES

• DAA regimen traditionally determined by:
  • Genotype
  • Fibrosis severity (i.e. cirrhosis vs. no cirrhosis)
  • Previous treatment exposure

• All DDIs must be checked prior to treatment (and contingency plans in place)

• Check Hepatitis B status prior to commencing DAA treatment (re-activation is rare)
  • Co-infected patients should be managed in a Specialist Clinic

• Establish presence of cirrhosis prior to DAA treatment
  • Baseline US scan of liver is mandatory prior to DAA treatment (exclude HCC)
  • Appropriate management (surveillance for complications) thereafter
TAKE HOME MESSAGES

• Offer appropriate support while on-treatment to optimize compliance
  • Often just requires reassurance and common-sense ± investigation/specialist review (rarely)

• Ensure treatment success with appropriate testing during treatment
  • FBC and E/LFTs on treatment (usually week 4 ± as indicated; E/LFTs at week 8 if on EBR/GZR)
  • HBV DNA (around week 4–6 if HBVsAg positive pre-treatment)
  • HCV RNA PCR at EOT and 12 weeks after EOT (to ensure SVR-12)

• Persistently abnormal LFTs need further assessment (2nd cause of CLD)
  • + Mandatory surveillance of cirrhotics (despite significant reduction in HCC/mortality)

• Treating Hep C has many benefits – for patient, society … and doctor!
QUESTIONS AND DISCUSSION