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2022
ACG’S IBD SCHOOL & MIDWEST REGIONAL POSTGRADUATE COURSE
AUGUST 26–28, 2022 | JW MARRIOTT HOTEL INDIANAPOLIS, INDIANA

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The Deadline for Submission is Monday, June 20, 2022 at 11:59 pm ET

Abstract Categories:

- Biliary/Pancreas
- Colon
- Colorectal Cancer Prevention
- Endoscopy Video Forum
- Esophagus
- Functional Bowel Disease
- General Endoscopy
- GI Bleeding
- IBD
- Interventional Endoscopy
- Liver
- Obesity
- Pediatrics
- Practice Management
- Small Intestine
- Stomach
- Clinical Vignettes/Case Reports

Visit acgmeetings.gi.org to Submit!
Participating in the Webinar

All attendees will be muted and will remain in Listen Only Mode.

Type your questions here so that the moderator can see them. Not all questions will be answered but we will get to as many as possible.

How to Receive CME and MOC Points

LIVE VIRTUAL GRAND ROUNDS WEBINAR
ACG will send a link to a CME & MOC evaluation to all attendees on the live webinar.

ABIM Board Certified physicians need to complete their MOC activities by December 31, 2022 in order for the MOC points to count toward any MOC requirements that are due by the end of the year. No MOC credit may be awarded after March 1, 2023 for this activity.
MOC QUESTION

If you plan to claim MOC Points for this activity, you will be asked to: Please list specific changes you will make in your practice as a result of the information you received from this activity.

Include specific strategies or changes that you plan to implement. THESE ANSWERS WILL BE REVIEWED.

ACG Virtual Grand Rounds

Join us for upcoming Virtual Grand Rounds!

Week 23 – June 9, 2022
Overcoming the Challenges & Mitigating the Disparities in Our LGBTQI+ Patients: A Digestive Diseases Health Perspective
Sonali Paul, MD, MS
June 9, 2022 at Noon Eastern and 8pm Eastern!

Optimizing Mentorship for GI Mentees and Mentors
Loren Rabinowitz, MD, Christina M. Surawicz, MD, MACG, Lavanya Viswanathan, MD, MS, FACP, Renee L. Williams, MD, MHPE, FACP
June 15, 2022 at 8:30PM Eastern

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**Disclosures**

David E. Bernstein, MD, MACG  
AbbVie: Consultant, Research Grant  
Conatus: Research Grant  
CymaBay: Research Grant  
Gilead Sciences: Consultant, Research Grant  
Novartis: Research Grant  
NovoNordisk: Research Grant

Suthat Liangpunsakul, MD, MPH, FACG  
Direct Corporation: Consultant  
Surozoix: Consultant

Jorge L. Herrera MD, MACG  
Gilead Sciences: Speakers’ Bureau  
AbbVie: Speakers’ Bureau

*All of the relevant financial relationships listed for these individuals have been mitigated*
Objectives

- Epidemiology of ALD
- Impact of COVID-19 pandemic on alcohol use and ALD
- Non-invasive screening for excessive alcohol use and ALD
Spectrum of alcohol associated liver disease (ALD)


Normal

90%-95%

15%-30%

20%-40%

8%-20%

Alcoholic steatosis

Fibrosis

Cirrhosis

Alcoholic steatohepatitis

Prevalence of alcoholic fatty liver disease (AFLD) among US Adults from 2001-2016

ETOH >28 g/d in women and >42 g/d in men
AST/ALT >25 U/L in W and >35 U/L in men
Total bilirubin < 3mg/dl
No HBV or HCV infection
Excluding those with metabolic syndrome
Hepatic fibrosis: AST/Platelet >0.7 (stage≥2)
And Fib-4 score >2.67 (stage≥3)

Prevalence, %


JAMA. 2019;321(17):1723-1725
Mortality due to alcoholic cirrhosis is rising in the US, especially in young adults

Death certificate data from the Vital Statistics Cooperative, and population data US Census Bureau compiled by the CDC’s Wide-ranging Online Data for Epidemiologic Research (1999-2016)
Increasing trend of alcohol beverage sales in the US during the COVID-19 pandemic

<table>
<thead>
<tr>
<th>Annual Comparison</th>
<th>Jan</th>
<th>Feb</th>
<th>Mar</th>
<th>Apr</th>
<th>May</th>
<th>Jun</th>
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Legend

% 0 100 200 300 400 500 600 700 800 900 1000 1100

Heatmap showing the annual differences in monthly Beer, Wine and Liquor sales (BWLS) between consecutive years, U.S., 1992–2020

Percentage changes in monthly per capita sales of alcoholic beverages in 2020 or 2021 compared to the 2017-2019 (NIH/NIAAA)

[Link to NIH/NIAAA report](https://pubs.niaaa.nih.gov/publications/surveillance-covid-19/COVSALES.htm)
Cumulative projections for ALD morbidity and mortality for 3- and 20-year horizon

<table>
<thead>
<tr>
<th>Scenario</th>
<th>Results</th>
<th>2020–2023</th>
<th>2020–2040</th>
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<tbody>
<tr>
<td>COVID-19 consumption</td>
<td>ALD cirrhosis mortality</td>
<td>112,700 (107,800–117,700)</td>
<td>956,700 (916,500–996,900)</td>
</tr>
<tr>
<td></td>
<td>Decompensated cirrhosis incidence</td>
<td>118,000 (110,400–121,600)</td>
<td>1,128,800 (1,075,200–1,182,400)</td>
</tr>
<tr>
<td></td>
<td>HCC incidence</td>
<td>16,900 (15,900–18,000)</td>
<td>143,800 (136,200–151,500)</td>
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<tr>
<td>Counterfactual</td>
<td>ALD cirrhosis mortality</td>
<td>112,600 (107,700–117,500)</td>
<td>948,700 (909,000–988,300)</td>
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<td>Decompensated cirrhosis incidence</td>
<td>112,900 (107,400–118,400)</td>
<td>1,109,000 (1,056,600–1,161,400)</td>
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<tr>
<td></td>
<td>HCC incidence</td>
<td>17,000 (16,000–18,000)</td>
<td>142,800 (135,200–150,400)</td>
</tr>
<tr>
<td>Increase in outcomes because of COVID-19 drinking</td>
<td>ALD cirrhosis mortality</td>
<td>100 (100–200)</td>
<td>8000 (7500–8600)</td>
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<td>Decompensated cirrhosis incidence</td>
<td>3100 (3000–3300)</td>
<td>19,800 (18,600–21,000)</td>
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<tr>
<td></td>
<td>HCC incidence</td>
<td>0 (00–00)</td>
<td>1000 (1000–1100)</td>
</tr>
<tr>
<td></td>
<td>DALYs</td>
<td>531,200 (526,600–535,700)</td>
<td>8,902,000 (8,878,600–8,925,300)</td>
</tr>
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</table>

ALD mortality increased from 2017-2020 and accelerated during the COVID-19 pandemic
Summary – Part I

- No uniformity on how to define ALD in several epidemiological studies – affecting the prevalence estimate of ALD
- Estimated prevalence of Alcoholic steatosis ~ 4%-5% in the US population
- Among all hospitalized AH patients, we observed an increase in hospitalization rate notably among those 30-49 years old
- Mortality due to alcoholic cirrhosis is rising in the US, especially in young adults
- An increase in alcohol sales during COVID-19 pandemic affects ALD-related mortality

ALD is rarely detected at early stages compared with other types of chronic liver disease

Cross-sectional study of 3453 consecutive patients either early (N=1699) or advanced liver disease (N=1754) 17 tertiary care liver or gastrointestinal units worldwide from August 2015 through March 2017

Alcohol consumption 60 g/d men, 40 g/d women for at least 6 months Compatible clinical, analytical, imaging, or histologic findings BMI < 30

Early stage liver disease
Non-cirrhotic liver disease without history of liver failure or complications from portal HTN

Advanced stage liver disease
Those with compensated cirrhosis
Standard drink in the United States

A standard drink in the US = 14 grams of alcohol

The percentage of pure alcohol (alcohol by volume) varies within and across beverage types. Each beverage portrayed above represents one standard drink (or one alcoholic drink equivalent), defined in the United States as any beverage containing 0.6 fl oz (18 ml) or 14 grams of pure alcohol.

Alcohol drinking – How much is too much???

<table>
<thead>
<tr>
<th>grams of ethanol/day</th>
<th>Men</th>
<th>Women</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 10</td>
<td>1.0</td>
<td>1.0</td>
</tr>
<tr>
<td>10-20</td>
<td>1.1</td>
<td>2.9*</td>
</tr>
<tr>
<td>20-40</td>
<td>1.4</td>
<td>2.9*</td>
</tr>
<tr>
<td>40-60</td>
<td>3.8*</td>
<td>7.3*</td>
</tr>
<tr>
<td>60-100</td>
<td>5.9*</td>
<td>--</td>
</tr>
<tr>
<td>&gt; 100</td>
<td>9.1*</td>
<td>--</td>
</tr>
</tbody>
</table>

* Significant increased risk of alcoholic liver disease

Men: up to 2 drinks/day
Women: 1 drink/day

Moderate drinkers

Men: ≥4 drinks/day/ ≥14 drinks/wk and
Women: ≥3 drinks/day/ ≥7 drinks/wk

Excessive drinkers

http://pubs.niaaa.nih.gov
universe.gi.org
Non-invasive biomarkers for excessive alcohol use

<table>
<thead>
<tr>
<th><strong>Acute alcohol use biomarkers</strong></th>
<th><strong>Chronic alcohol use biomarkers</strong></th>
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<tbody>
<tr>
<td>Ethanol</td>
<td>AST/ALT</td>
</tr>
<tr>
<td>Most accurate determination of a patient’s alcohol level</td>
<td>Low sensitivity and specificity</td>
</tr>
<tr>
<td>1-12 hrs in blood or urine</td>
<td>GGT</td>
</tr>
<tr>
<td>Ethyl Glucuronide (EtG) and Ethyl Sulfate (EtS)</td>
<td>Inexpensive and lack of specificity</td>
</tr>
<tr>
<td>Direct minor metabolites of ethanol and are considered good markers of acute, short-term</td>
<td>MCV</td>
</tr>
<tr>
<td>Up to 36 hours in the blood, up to 5 days in urine) alcohol ingestion. Results do not accurately correlate with the amount or frequency of ethanol use</td>
<td>Low sensitivity and specificity</td>
</tr>
<tr>
<td>Ethyl Glucuronide (EtG) and Ethyl Sulfate (EtS)</td>
<td>Carbohydrate deficient transferrin</td>
</tr>
<tr>
<td>Direct minor metabolites of ethanol and are considered good markers of acute, short-term</td>
<td>An indirect metabolite of ethanol</td>
</tr>
<tr>
<td>Up to 36 hours in the blood, up to 5 days in urine) alcohol ingestion. Results do not accurately correlate with the amount or frequency of ethanol use</td>
<td>A marker of long-term, heavy alcohol use (≥40 g/day for up to 2 weeks) or relapse</td>
</tr>
<tr>
<td></td>
<td>Phosphatidylethanol (PEth)</td>
</tr>
<tr>
<td></td>
<td>Formed directly after alcohol intake via the enzyme phospholipase D from phosphatidylcholine (PC) in the presence of alcohol</td>
</tr>
<tr>
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<td>1-2 wks or longer</td>
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Non-invasive evaluation of steatosis in ALD

<table>
<thead>
<tr>
<th><strong>Liver ultrasound (US)</strong></th>
<th><strong>Magnetic resonance imaging</strong></th>
<th><strong>Controlled attenuation parameter</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>- Initial screening test</td>
<td>- Magnetic resonance spectroscopy (MRS) non-invasive studies into the molecular composition of tissues in vivo</td>
<td>- Part of transient elastography (Fibroscan)</td>
</tr>
<tr>
<td>- Non-invasive</td>
<td>- MRS quantifies the proton density fat fraction (PDFF), a standardized measure of liver tissue</td>
<td>- AUROC to diagnose mild (0.77), moderate (0.78), and severe (0.82)</td>
</tr>
<tr>
<td>- Widely available</td>
<td>- Sensitivity ~80-90%</td>
<td>- CAP &gt;290 dB/m – 88% specificity in diagnosing steatosis</td>
</tr>
<tr>
<td>- Sensitivity 60-90%</td>
<td>- Specificity ~90-95%</td>
<td>- non-invasive, can be done simultaneously with liver stiffness for both fibrosis and steatosis measurement</td>
</tr>
<tr>
<td>(depending on the severity or degree of steatosis)</td>
<td>- Operator dependent, patient’s body contour (obese), inaccuracy in determining fibrosis</td>
<td>- Optimal cutoff specifically for ALD steatosis</td>
</tr>
<tr>
<td>- Specificity ~90-95%</td>
<td>- High accuracy and reproducibility</td>
<td></td>
</tr>
<tr>
<td>- Operator dependent, patient’s body contour (obese), inaccuracy in determining fibrosis</td>
<td>- Cost and examination time</td>
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Non-invasive evaluation of fibrosis in ALD

<table>
<thead>
<tr>
<th>Non-invasive blood tests</th>
<th>Sensitivity</th>
<th>Specificity</th>
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<tbody>
<tr>
<td>APRI (cutoff 0.5)</td>
<td>49%</td>
<td>84%</td>
</tr>
<tr>
<td>FIB-4 (cut-off &lt;1.45)</td>
<td>42%</td>
<td>83%</td>
</tr>
<tr>
<td>FIB-4 (cut-pff &gt;3.25)</td>
<td>16%</td>
<td>99%</td>
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<tr>
<td>APRI &lt;0.5</td>
<td>48%</td>
<td>72%</td>
</tr>
<tr>
<td>APRI &gt;1.50</td>
<td>8%</td>
<td>98%</td>
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<tr>
<td>ELF moderate</td>
<td>83%</td>
<td>73%</td>
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<tr>
<td>ELF severe</td>
<td>78%</td>
<td>76%</td>
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<tr>
<td>FT (Fibrotest)</td>
<td>61%</td>
<td>80%</td>
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<tr>
<td>APRI (1.0-1.5)</td>
<td>54%</td>
<td>78%</td>
</tr>
<tr>
<td>APRI &gt; 2.0</td>
<td>0%</td>
<td>1%</td>
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<tr>
<td>APRI &lt; 1.0</td>
<td>35%</td>
<td>94%</td>
</tr>
<tr>
<td>ELF</td>
<td>80%</td>
<td>71%</td>
</tr>
<tr>
<td>FT</td>
<td>62%</td>
<td>91%</td>
</tr>
</tbody>
</table>

APRI = (AST/ULN of AST)/(platelet count X100). FIB-4 = (age XAST)/(platelet count X (ALT)1/2). ELF = Enhanced Liver Fibrosis score is calculated based on tissue inhibitor of metalloproteinases 1 (TIMP-1), amino-terminal propeptide of type III procollagen (PINCIP) and hyaluronic acid (HA) levels. FT = FibroTest consists of an algorithm of 5 fibrosis markers (alpha2-macroglobulin [g/L], apolipoproteinA1 [g/L], total bilirubin [mol/L], haptoglobin [g/L], Gamma glutamyltransferase [IU/L]).

Non-invasive evaluation of fibrosis in ALD - Transient elastography

MATRIX

Fibrosis stage F0-F4
Collagen deposition
Tissue matrix

PRESSURE

Water retention
Alterations in oncotic pressure
Inflammation
Alcohol consumption
Food intake
Inflow
Portal Pressure
Arterial Pressure
Sinusoidal pressure
Outflow
Venous Pressure
Cholestasis
LIVER STIFFNESS

J Hepatology 2019;70:273-283
universe.gi.org
Summary – Part II

- Several non-invasive biomarkers are available to screen for acute and chronic alcohol use

- Imaging-based diagnostic modalities for steatosis

- Markers (non-invasive blood tests or transient elastography) to diagnose underlying fibrosis and its severity (especially among excessive alcohol users without clinically apparent cirrhosis)

- K18 – diagnostic marker for AH
Thank You
Suthat Liangpunsakul MD, MPH

Alcohol-Associated Liver Disease – Clinical Presentation
Jorge L. Herrera MD, MACG
University of South Alabama College of Medicine, Mobile, AL
Alcohol is Not a Predictable Hepatotoxin

- Most people with AUD develop steatosis
- Only 10% - 20% will develop ALD
  - Inflammation
  - Alcoholic hepatitis
  - Fibrosis
  - Cirrhosis
  - HCC

Factors Increasing the Risk of ALD
- Alcohol dose
- Daily, fasting or binge drinking
- Smoking
- Female sex
- Increased BMI
- Genetics (PNPLA3, TM6SF2)
- Co-existing liver diseases

Alcoholic liver disease is a diagnosis of exclusion, even in people with alcohol use disorder

1. Binge drinking: ♂ 5 drinks, ♀ 4 drinks over 2hr

Disease Spectrum of Alcoholic Liver Disease


American College of Gastroenterology
Initial Approach to the Patient With AUD

- Elevated liver tests: AST > ALT, GGT, normal bilirubin
- Ultrasound to confirm steatosis
- Assessment of fibrosis: Elastrography, FIB-4
- LS < 8 kPa or FIB-4 < 1.3 excludes advanced fibrosis
- LS > 12 = advanced fibrosis
- Repeat after 1-2 wk abstinence if elevated AST

References:
- EASL, J Hepatology 2021;75:659-689

ALCOHOLIC HEPATITIS

Mild symptoms

- Initial presentation for many patients with ALD
- Often co-exists with cirrhosis (30%-50%)
  - Acute on chronic liver failure (ACLF)
- Typical biochemical presentation
  - Mild transaminase elevation, AST predominant
- Histologic features identical to NAFLD

Liver Failure

- Steatosis, ballooning, neutrophilic inflammation, Mallory-Denk bodies

References:
- American College of Gastroenterology
Clinical Diagnosis of Alcoholic Hepatitis

**CONSENSUS DEFINITION**
1. Onset of jaundice in the prior 8 weeks
2. Ongoing excessive consumption of alcohol with <60 days of abstinence before onset of jaundice
3. AST >50, AST/ALT >1.5 and both values <400 (often <200)
4. Serum total bilirubin >3.0 mg/dL

Liver Biopsy in Alcoholic Hepatitis

- **Definite AH**
  - Clinical diagnosis + biopsy proven
- **Probable AH**
  - Clinical diagnosis without confounding factors
- **Possible AH**
  - Clinical diagnosis with confounding factors
  - Biopsy needed for confirmation of AH
    - 10%-20% other diagnosis or no evidence of ASH

**Confounding factors:**
- Ischemic hepatitis
- Cocaine use
- Possible DILI
- Uncertain alcohol use history
- Atypical labs: AST <50 or >400, AST:ALT <1.5
- High titer autoimmune markers

**Role of liver biopsy: Resolve diagnostic dilemmas**

Crabb DW et al. Gastroenterology 2016;150:785-90
SIRS in Alcoholic Hepatitis (~70%)

- Difficult to differentiate from sepsis (~20% to 50%)
- Fever, tachycardia, leukemoid reaction common
- At admission
  - “Pan culture”: blood, urine, ascites, sputum
  - Chest X-Ray
  - Skin exam for cellulitis
  - Avoid antibiotics unless proven infection
- Careful monitoring of renal function
- Presence of SIRS predicts MOF

```
Crabb DW et al. Gastroenterology 2016;150:785-790
Singal AK, et al. Am J Gastroenterol 2018;113:175-194
Gustot T et al. J Hepatology 2017;67:1031-1050

Assessing Prognosis in Alcoholic Hepatitis

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<th>Clinical Use</th>
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<td>Start steroids</td>
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**Maddrey DF – PT/Bilirubin**

**Pro:** only score to determine who benefits from steroid use

**Con:** must be calculated on admission, may lead to excess CS use

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**MELD – Adds Scr**

**Pro:** Proven prognostic value, Δ MELD offers added prognostic information

**Con:** Uncertain threshold for starting CS (>20 suggested), falsely poor prognosis if renal dysfunction

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**MELD-Na – Adds Na**

**Con:** Adds no value to MELD

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**Lille – Adds SCr + Age + Albumin**

**Pro:** Allows for early cessation of steroids

**Con:** Uncertain decision making in partial response (0.46-0.56), 4 day score needs validation

---

Acute Kidney Injury in AH

- Common in severe alcoholic hepatitis
- SIRS or infection increases risk
- Avoid
  - Intravenous contrast
  - Aminoglycosides
  - NSAIDs
  - Careful with diuretics
- Use albumin if SBP
- Early treatment with vasopressors + albumin if rise in creatinine >0.3mg/dL
Assessing for Contraindications to CS Therapy

- **Uncontrolled infections**
  - CS allowed after 48 hours of antibiotics with response

- **GI Bleeding**
  - CS allowed >48 hours after control of bleeding

- **Acute kidney injury (creatinine >2.5mg/dL)**

- **Multiorgan failure**

- **Shock**

- **Comorbid conditions**: HBV, DILI, HCC, acute pancreatitis, HIV, TB

- **Severe AH**: MELD >30 or Maddrey DF >60

Crabb DW et al. Gastroenterology 2016;150:785-790

Approach to the Patient with ALD - Summary

**OUTPATIENT SETTING**

AUD + elevated liver tests

- Exclude other causes of elevated liver tests
- Assess fibrosis
- Recommend addiction medicine care

**INPATIENT SETTING**

Alcoholic Hepatitis + ACLF

- Establish a definite or probable diagnosis
  - History, typical labs, liver biopsy
- Assess severity/prognosis, determine need for therapy

- Abstinence and improved nutrition
- Corticosteroid therapy
- Liver transplantation
Thank You
Jorge L. Herrera MD, MACG

Treatment of Alcohol Related Liver Disease
David Bernstein, MD, MACG
Treatment of ALD

General:
- Avoid alcohol
- Adequate nutrition
- Treat complications as they develop

Specific:
- Alcoholic hepatitis
- Transplantation

Treatment of ETOH Hepatitis: AASLD Guidance Statements
- Lab-based prognostic scores should be used to determine prognosis in AH.
- The MDF (≥32) should be used to assess the need for treatment with corticosteroids or other medical therapies.
- A MELD score greater than 20 also should prompt consideration of steroid treatment.
- Abstinence from alcohol should be promoted to improve long-term prognosis in AH.

Treatment of ETOH Hepatitis

Abstinence  Nutrition  Corticosteroids

Pentoxifylline  NAC


Prednisolone/Prednisone
- Prednisolone preferred therapy
- 40 mg per day for 28 days

Pentoxifylline
- 400 mg TID
- Benefit in reducing risk of renal injury and death from hepatorenal syndrome
28-Day Survival in Alcoholic Hepatitis: Prednisolone vs. Placebo; DF>32

![Cumulative Survival Curve](image)

**Predictors of Survival**
- Age (p<0.0001)
- Creatinine (p<0.002)
- Prednisolone (p<0.002)

**Mathurin 2002**

Assessment of patients with alcoholic hepatitis likely to benefit from corticosteroids

Lille Score-Assessment of Response to Steroids for Alcoholic Hepatitis

- At day 7, discontinue in those with Lille score >0.45
  - Recent data shows day 4 assessment as accurate as day 7
  - 50-60% do not respond to steroids
- Patients non-responsive to corticosteroids, ineligible for early transplant, and with multiple organ failure may be considered for palliative therapy

<table>
<thead>
<tr>
<th>Age</th>
<th>years</th>
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<tbody>
<tr>
<td>Albumin</td>
<td>g/dL</td>
</tr>
<tr>
<td>Bilirubin (initial)</td>
<td>mg/dL</td>
</tr>
<tr>
<td>Bilirubin (day 7)</td>
<td>mg/dL</td>
</tr>
<tr>
<td>Creatinine</td>
<td>mg/dL</td>
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</table>

Louvet 2007, Singhal 2018

Treatment of Alcoholic Hepatitis

**Therapies with proven efficacy**
- Corticosteroids
- Nutritional Supplementation

**Therapies with potential efficacy**
- Pentoxifylline
- N-acetyl cysteine
- GCSF

**Therapies with no efficacy**
- Tumor necrosis factor
- Vitamin E
- Propylthiouracil
- Insulin, glucagon, anabolic steroids

Singhal et al. AJG 2018
Summary Treatment of ETOH Hepatitis: AASLD Guidance Statements

Prednisolone (40 mg/day) given orally should be considered to improve 28-day mortality in patients with severe AH (MDF ≥ 32) without contraindications to the use of corticosteroids (Fig. 3).

The addition of intravenous NAC to prednisolone (40 mg/day) may improve the 30-day survival of patients with severe AH.

The Lille score should be used to reassess prognosis, identify nonresponders, and guide treatment course after 7 days of corticosteroids.

Patients with AH should have malnutrition addressed and treated, preferably with enteral nutrition.

Abstinence is key to long-term survival; methods discussed previously for treatment of AUDs should be used to increase abstinence.

Pentoxifylline is no longer recommended in the treatment of AH.

Liver Transplantation for Alcoholic Hepatitis

May be considered for highly selected patients with severe alcoholic hepatitis

Selection remains controversial

- No standard criteria

COVID has led to increased hospital admissions for alcoholic hepatitis

- Especially amongst young adults

Few controlled clinical trials assessing selection criteria

Singhal et al. AJ 2018
Alcohol and Transplantation

Leading indication for liver transplantation in the USA

Major issues
- Appropriate patient selection
- Outcomes for patient and graft
- Impact of alcohol resumption
- Patient comorbidities
- Alcoholic hepatitis?

Patient and graft survival at 1 and 5 years similar to non-alcohol associated disease

Patient and graft survival at 10 years lower in alcohol related liver disease

Alcohol relapse occurs in 1-30% of patients post-transplant

Temporal Changes in the Frequency of LT in the US

Cotter. Liver Transplantation 2020; 26:141-159
Goals of Liver Transplantation in Alcoholic Liver Disease

Avoid

- Avoid transplants in those who will recover

Provide

- Provide transplantation to those who can achieve post-transplant survival benefit
  - At least 60-70% survival at 5 years

Avoid

- Avoid creation of new disparity in access to transplantation

Predictors of Alcohol Relapse after Transplantation

- Depression or other psychiatric disorder
- Lack of social support
- Tobacco use
- Family history of alcohol dependence
- Lack of insight
- Amount of alcohol consumed prior to evaluation
- Presence of hepatitis C
- Multi-variate model correctly predicted post-transplant alcohol recurrence in 89% of recipients

Kelly 2006
Factors not Predictive of Alcohol Recurrence following transplantation

- Length of abstinence
  - 6 months
  - 3 months
  - Other duration

Liver Transplantation for Alcoholic Hepatitis

Lee et al. Gastro 2018
Liver Transplantation for Alcoholic Hepatitis: Effects of Early Transplant (QuickTrans Study)

Relapse rates:
34% (AH) vs 25% (AC)

Survival:
90% (AH) vs 88% (AC)

AH Transplanted vs non transplant:
83% vs. 28%

Pros
- Short term mortality is high and patients should be offered LT
- Strict criteria has led to favorable outcomes with acceptable relapse rates
- 6-month rule not specific to identify relapse and may disadvantage those with favorable psychosocial profile
- AH not necessarily associated with higher relapse

Cons
- Abstinence for 6 months allows for recovery and patient may not need LT
- Need to show commitment for abstinence
- Negative public perception of self-inflicted condition
- Current predictive models not precise enough to predict lack of response
- Significant variation in practice and listing criteria
- Favors those with resources
Treatment paradigm for ALD and AUD

• Asrani et al. Hepatology 2021

Treatment of ETOH Use Disorders: AASLD Guidance Statements

• Referral to AUD treatment professionals is recommended for patients with advanced ALD and/or AUD, to ensure access to the full range of AUD treatment options.

• Multidisciplinary, integrated management of ALD and AUD is recommended and improves rates of alcohol abstinence among patients with ALD

Questions?

David E. Bernstein, MD, MACG

Suthat Liangpunsakul, MD, MPH

Jorge L. Herrera MD, MACG

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