



TLI Quarterly

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INTRODUCING DR. HOPE BAKER

The Texas Liver Institute is thrilled to welcome Dr. Hope Baker to our team of providers. Dr. Baker earned her medical degree from Baylor College of Medicine in Houston, Texas. She then completed internal medicine residency and hepatology fellowship at Baylor College of Medicine, followed by gastroenterology fellowship at Ohio State University. Prior to joining the Texas Liver

Institute, Dr. Baker served as director of hepatology at the Robert B Green Clinic with the University Hospital System.

Dr. Baker is a member of the American Association for the Study of Liver Disease (AASLD), American College of Gastroenterology (ACG), and American Gastroenterological Association (AGA). She is interested in treating all forms of acute and chronic liver disease.



Hope Baker, MD
Associate Professor of Medicine

9th APP/Nursing & Primary Care Liver Conference

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FROM FATTY LIVER TO CANCER: UNDERSTANDING MASLD'S ROLE IN HCC DEVELOPMENT

Abdurrahman Kadayifci, MD (Transplant Hepatology Fellow)

Metabolic dysfunction–associated steatotic liver disease (MASLD) has emerged as one of the leading causes of chronic liver disease, with prevalence rising alongside obesity, type 2 diabetes, and sedentary lifestyles. Given the high prevalence of obesity in Texas, MASLD and its complications represent a substantial public health burden in the region. Recent studies indicate that MASLD contributes to a significant proportion of patients diagnosed with hepatocellular carcinoma (HCC). While the absolute risk of HCC for an individual with MASLD is relatively low, the population-level burden has led to a growing number of MASLD-related HCC cases.

The relationship between MASLD and HCC was a prominent theme at this year's AASLD conference. Presenters emphasized the rising prevalence of MASLD-related HCC and explored both genetic and lifestyle factors that increase risk. Among genetic factors, the PNPLA3 mutation,

which is more prevalent among individuals of Latino ancestry, was highlighted as the most significant determinant of HCC risk in MASLD patients. In addition, multiple cohort studies demonstrated that regular alcohol consumption remains one of the most preventable risk factors for HCC development in this population.

Most MASLD-associated HCC occurs in the setting of cirrhosis; however, a notable subset arises in non-cirrhotic patients. Salinas et al. from Baylor Scott & White in Fort Worth, Texas, reported that among 277 patients with MASLD-related HCC, 10% had no evidence of cirrhosis. Notably, none of the non-cirrhotic patients had undergone HCC surveillance prior to diagnosis. At presentation, tumors in these patients were larger, more frequently exceed Milan criteria, were associated with lower overall survival and were more often ineligible for liver transplantation.

Given the substantial number of non-cirrhotic individuals with MASLD,

identifying which patients should undergo HCC surveillance remains a critical challenge. To address this, Vutein et al. from the University of Washington analyzed data from 21,747 patients with non-cirrhotic MASLD who underwent FibroScan and serum FIB-4 testing. Ninety-one patients developed incident HCC during follow-up. HCC incidence was significantly higher among patients with high liver stiffness (>15 kPa) or moderate stiffness combined with high FIB-4 scores. These findings support considering HCC surveillance in non-cirrhotic MASLD patients with high-risk fibrosis profiles on noninvasive testing.

These presentations underscore the growing clinical and public health importance of MASLD as a driver of HCC and highlight the need for targeted surveillance strategies, particularly among high-risk non-cirrhotic patients, to enable earlier detection and improve outcomes.

SEMAGLUTIDE NOW APPROVED FOR MASH: WHAT PRIMARY CARE NEEDS TO KNOW

Krishna Venkata, MD (General Hepatology Fellow)

MASLD affects nearly one-third of US adults and has become the most common cause of chronic liver disease. While simple steatosis is often benign, progression to MASH with fibrosis determines long-term outcomes. Patients with F2–F3 fibrosis face significantly increased risks of cirrhosis, hepatic decompensation, hepatocellular carcinoma, and cardiovascular disease—the leading cause of death among individuals with MASLD. As a result, early recognition of patients with clinically significant fibrosis has become a central priority in primary care.

The FDA recently granted accelerated approval of **semaglutide (Wegovy 2.4 mg weekly)** for adults with **noncirrhotic MASH and F2–F3 fibrosis**, used alongside lifestyle modification. This represents a landmark shift: for the first time, an effective disease-modifying therapy for MASH can be initiated based on **noninvasive testing** rather than liver biopsy. For primary care

clinicians, this greatly expands the ability to intervene earlier in the disease course.

Semaglutide is particularly valuable for patients with obesity, type 2 diabetes, or high cardiometabolic risk—groups that frequently overlap with MASLD. Beyond improving steatohepatitis and fibrosis, semaglutide can result in substantial weight loss, improved glycemic control, and cardiovascular protection. Dosing follows the established titration schedule used for obesity: **0.25 mg weekly → 0.5 mg → 1 mg → 1.7 mg → 2.4 mg**, typically increasing every four weeks as tolerated. Gastrointestinal symptoms are the most common side effects and can be mitigated by slow titration. Clinicians should remain attentive to potential complications such as pancreatitis, gallbladder disease, dehydration-related acute kidney injury, and transient worsening of diabetic retinopathy with rapid glycemic improvement. Contraindications include pregnancy, a

personal or family history of medullary thyroid carcinoma or MEN2, and prior severe hypersensitivity.

Accurate fibrosis staging is essential before initiating therapy. The AASLD recommends a **two-step approach**:

1. Obtain **FIB-4** in all at-risk adults (obesity, T2DM, metabolic syndrome). A value ≥ 1.3 should prompt further assessment.
2. Perform **noninvasive fibrosis testing**—FibroScan (VCTE), MRE, or ELF. Thresholds supporting semaglutide eligibility include **VCTE 8–15 kPa, MRE 3.1–4.4 kPa, or ELF 9.2–10.5**, provided cirrhosis is not present.

TLI offers comprehensive noninvasive fibrosis staging and collaborative treatment planning. Early identification and timely referral ensure patients gain access to effective, disease-modifying therapy that can meaningfully improve long-term liver and cardiovascular outcomes.

DRUG-INDUCED LIVER INJURY: UPDATES IN 2025

Sidart Pradeep, MD (Transplant Hepatology Fellow)

Drug-induced liver injury (DILI) is an adverse reaction of the liver attributable to a medication, herbal product, or dietary supplement characterized by lab and clinical evidence of liver dysfunction. Symptoms include fatigue, nausea, abdominal discomfort, itching, dark urine, pale stools, and jaundice. Because symptoms can be nonspecific, diagnosis relies on identification of abnormal liver enzyme patterns and a careful review of all medications taken over **the past 1-2 years**. In addition, other causes of liver dysfunction must be assessed since DILI is typically a diagnosis of exclusion.

DILI manifests in three primary patterns. The first, is a liver cell injury pattern more commonly seen in younger individuals. The second, is cholestatic injury, in which there is impairment of bile metabolism and secretion; seen more often in patients >65 years of

age. Cholestatic injury often results in more pronounced jaundice and itching that may take longer to resolve. Lastly, patients can experience a mixed pattern with features of both liver cell injury and impaired bile flow. The primary forms of DILI can resolve with withdrawal of the offending agent.

DILI was a highlighted topic at this year's AASLD conference. Notably, during the primary topic session, presenters discussed the epidemiology of DILI and important considerations in diagnosis and prognosis. Non-prescription compounds such as **green tea extract and ashwagandha** were identified as potential causative agents. Incidence of DILI increases with age and the phenotype skews towards cholestatic, defined by evidence of impaired bile flow, in comparison to a direct liver cell injury pattern. Most importantly, DILI in older adults results in poorer

outcomes and increased mortality compared to younger individuals.

A commonly referenced study was that of the DILI network (DILIN). In a study of 899 patients with DILI, DILIN identified commonly associated medications and outcomes. **Amoxicillin-clavulanate** was the single most frequently implicated agent, including in individuals >40 years of age. Since this is one of the most widely prescribed medications in general practice, awareness regarding the association with DILI is paramount.

The presentations highlighted the shifting landscape of DILI, with over the counter and herbal substances playing a larger role. Age related differences in presentation and prognosis highlight the importance of minimizing medication exposure in older individuals when feasible.

PEARLS & PITFALLS: NONINVASIVE TESTING FOR STEATOSIS AND MASLD

Sarvanand Patel, MD (General Hepatology Fellow)

Steatosis affects roughly one-third of US adults, and progression to MASH is becoming a major cause of cirrhosis, hepatocellular carcinoma, and liver transplantation. Identifying patients with stage F2–F3 fibrosis is essential, as these individuals carry the highest long-term morbidity risk and may qualify for pharmacotherapy or clinical trials.

Steatosis vs MASLD

Pearls: Steatosis alone is often benign. Risk increases when steatosis occurs with metabolic dysfunction and chronic low-grade inflammation, typically seen as persistently high-normal or mildly elevated ALT. “True” abnormal ALT is lower than most lab reference ranges (>30 U/L in men and >20 U/L in women) and warrants evaluation. Historical ranges included individuals with undiagnosed liver disease.

Pitfalls: Normal ALT does not exclude ≥F2 fibrosis, especially in diabetes, obesity, or in the presence of multiple metabolic risk factors.

First-Line Blood Test: FIB-4

Pearls: AASLD endorses FIB-4 as the

primary care entry test. It is a calculated score using results from AST, ALT, platelets and patient age. Important cutoffs: <1.3 = low risk, >2.67 = high risk. In adults >65, <2.0 is considered low risk.

Pitfalls: Low accuracy in persons <35 (some studies suggest <40); young adults with metabolic risk or elevated ALT should proceed directly to ELF or imaging (VCTE/MRE). Unreliable in acute illness, heavy alcohol use, or nonhepatic thrombocytopenia.

Imaging: VCTE, SWE, MRE

Pearls: Vibration-controlled transient elastography (VCTE) is guideline-validated for MASLD staging. FibroScan® is most often used in the US, though other devices perform similarly when quality criteria are met such as shear-wave-elastography. Validated platforms show AUROCs ~0.85–0.90 for ≥F3 fibrosis. MRE with PDFF is the most accurate modality, especially in obesity or when other tests are discordant.

Pitfalls: Avoid ordering “ultrasound elastography” performed during routine

abdominal ultrasound—this is not guideline-validated and often overestimates fibrosis. VCTE overestimates stiffness in high BMI, central adiposity, active inflammation (ALT >5x ULN), cholestasis, active alcohol use, and heart failure.

Biopsy

Use for discordant results, uncertain etiology, or when confirmation of ≥F3/F4 is needed for treatment or trial eligibility. There is no BMI limitation and transjugular routes improve safety, when needed.

Follow-Up & Practical Pathway

- With steatosis and normal ALT plus ≤1–2 metabolic risks: repeat FIB-4 every 2–3 years. If diabetes or ≥2 risks: every 1–2 years.
- Choose VCTE/SWE first; use MRE if results are indeterminate or technically limited.
- Avoid non-validated ultrasound elastography.

MANAGING MASH THROUGH OUTREACH CLINICS

Erika Baillio, NP (2025 AASLD Emerging Liver APP Award Recipient)

This year's AASLD conference highlighted significant advancements in the treatment of Metabolic Dysfunction–Associated Steatohepatitis (MASH), a condition that is commonly diagnosed in our outreach clinics. Texas, with its diverse demographics, has a notably high prevalence of fatty liver disease. A significant factor contributing to this is a genetic predisposition among Hispanic/Latinos. The PNPLA3 gene is the strongest genetic risk factor for MASH and nearly 50% of US Hispanics carry a variant putting them at high risk. This variant is associated with an increased accumulation of fat in the liver, elevating the risk of developing MASLD/MASH, which can lead to advanced fibrosis and cirrhosis at a younger age. As clinicians, it is imperative to identify and manage these patients early to prevent severe outcomes. Recognizing this genetic predisposition allows us to extend screening to family members without incurring the high costs of genetic testing.

Early identification and referral to hepatology are vital for patients with



Erika Baillio, NP

MASH, since disease management can significantly improve long-term health outcomes the patient's long-term management of liver disease. Community providers, particularly in Laredo and Corpus Christi, play a crucial role in early detection. Key indicators for referral include:

- Elevated AST or ALT for >6 months, indicating ongoing liver injury.
- The use of the FIB-4 score to assess the likelihood of fibrosis, which requires only a CMP and CBC. A FIB-4

score below 1.3 suggests a lower risk of advanced fibrosis, while a score above 2.67 indicates a higher risk.

- Abnormal imaging results showing nodular liver contour or early morphological changes, as well as fatty liver in patients with metabolic risk factors such as diabetes, overweight/obesity, hypertension, and dyslipidemia.
- Platelets <150K, which may suggest portal hypertension, a potential consequence of advanced fibrosis or cirrhosis.
- Symptoms such as ascites, esophageal varices, coagulopathy (increased INR), hepatic encephalopathy, and jaundice, which are indicators of cirrhosis decompensation.

Referring patients to TLI allows us to collaborate with community providers to develop and implement effective treatment plans. Early identification and referral are essential strategies to improve long-term patient outcomes. When in doubt, referral is the best course of action.

To have a patient seen at one of our clinics, you can use the following link www.txliver.com/physician-referrals.

SA Central-607 Camden Street, San Antonio, TX 78215

SA Medical Center-4751 Hamilton Wolfe, Suite 200, San Antonio, TX 78229

SA North-4318 De Zavala Road, Suite 403, San Antonio, TX 78249

Austin-7940 Shoal Creek Boulevard, Suite 205, Austin, TX 78757

Corpus Christi-5022 Holly Road, Suite 106, Corpus Christi, TX 78411

Laredo-4151 Jaime Zapata Memorial Highway, Suite 101, Laredo, TX 78043