Steatotic Liver Disease

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Overview of Metabolic Dysfunction-Associated Steatotic Liver Disease (MASLD) and the Clinical Evaluation of Metabolic Dysfunction-Associated Steatohepatitis (MASH)

Disclosure

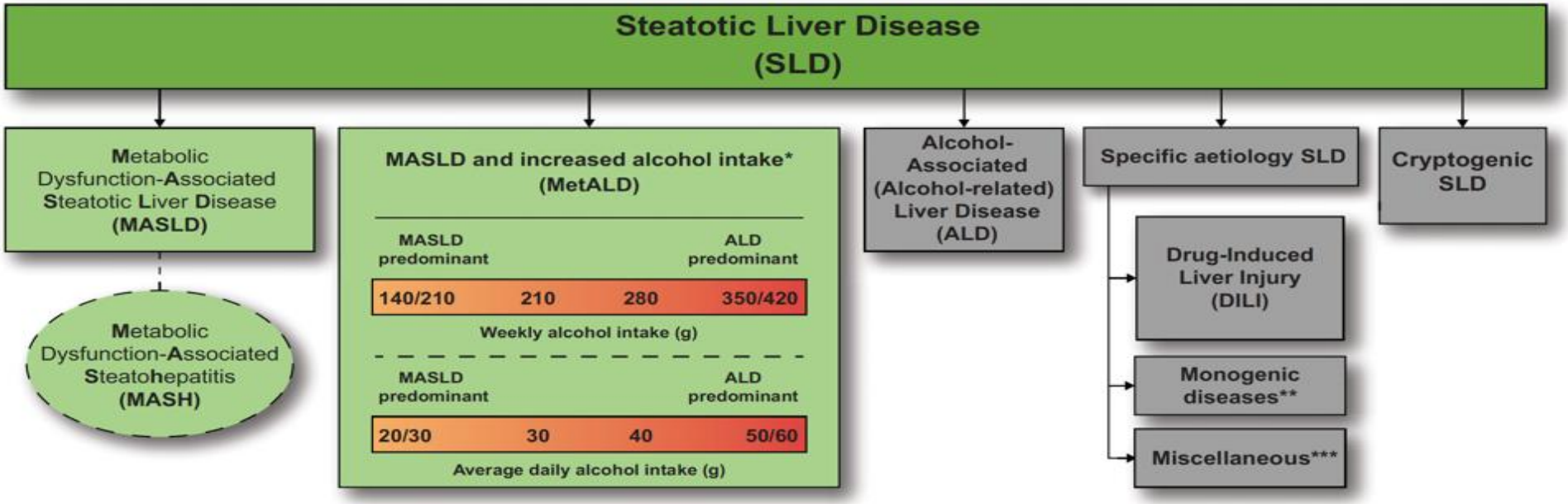
I have no financial disclosure

Learning Objectives

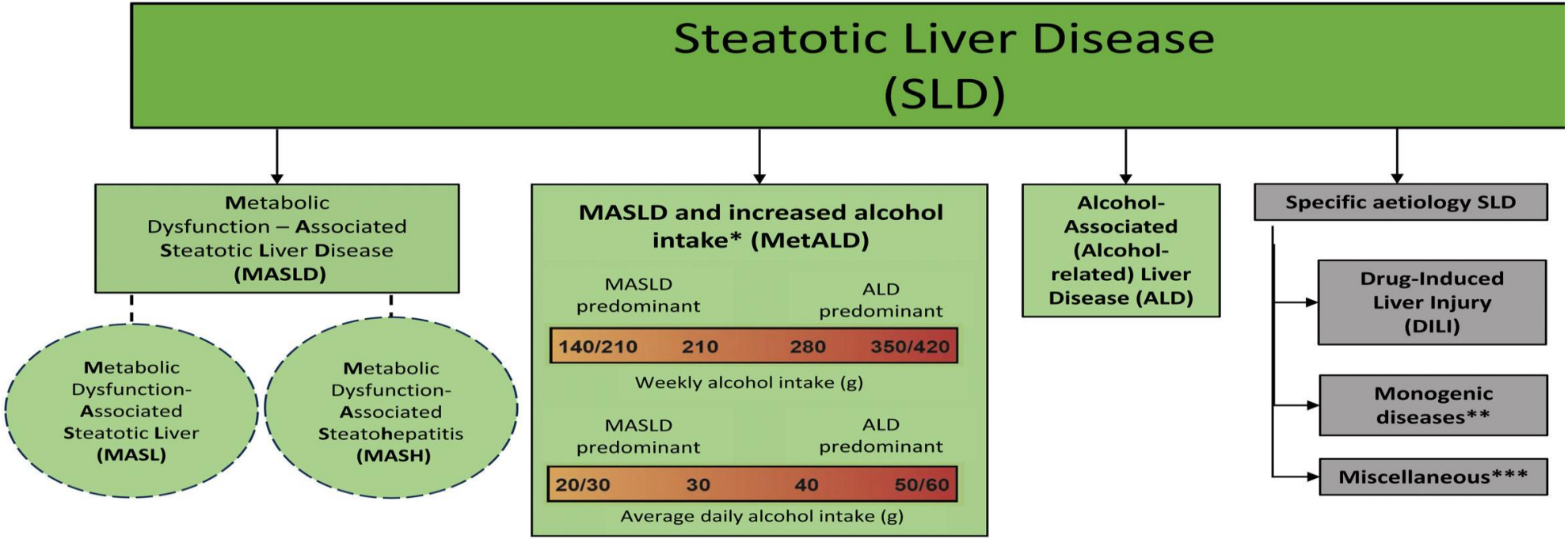
- Understanding MASLD/MASH terminology
- Screening and monitoring High-Risk Patients
- Use noninvasive test as FIB-4 for risk stratification
- Identifying at risk patients for MASH
- Initiate treatment and referral to specialist

Endorsements – more than 70 societies





*Weekly intake 140-350g female, 210-420g male (average daily 20-50g female, 30-60g male)
**e.g. Lysosomal Acid Lipase Deficiency (LALD), Wilson disease, hypobetalipoproteinemia, inborn errors of metabolism
***e.g. Hepatitis C virus (HCV), malnutrition, celiac disease, human immunodeficiency virus (HIV)



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Standard drink sizes are shown in the pictures below.

12 fl oz of **regular beer**	=	8-9 fl oz of **malt liquor** (shown in a 12-oz glass)	=	5 fl oz of **table wine**	=	3-4 oz of **fortified wine** (such as sherry or port; 3.5 oz shown)	=	2-3 oz of **cordial, liqueur, or aperitif** (2.5 oz shown)	=	1.5 oz of **brandy** (a single jigger or shot)	=	1.5 fl oz shot of **80-proof spirits** ("hard liquor")
												
about 5% alcohol		about 7% alcohol		about 12% alcohol		about 17% alcohol		about 24% alcohol		about 40% alcohol		about 40% alcohol

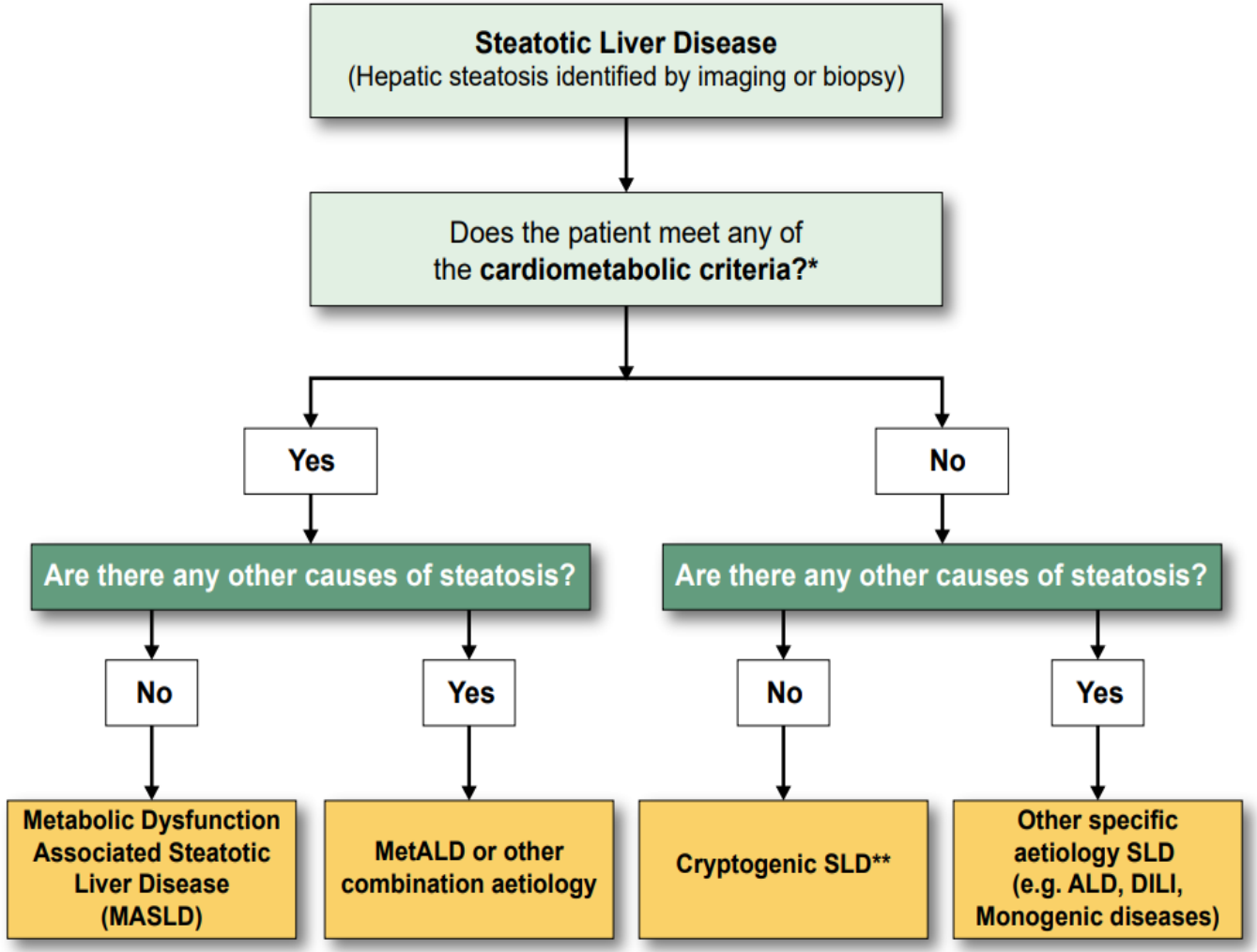
Why the Name Change?

- NAFLD→MASLD
- NASH→MASH

Why the Name Change?

- ~~NAFLD~~ → MASLD
- ~~NASH~~ → MASH
- Non Alcoholic defines disease negatively
- Recognize metabolic dysfunction
- Reduce stigma
- Steatohepatitis is main driver of the disease

Decision Support Tool



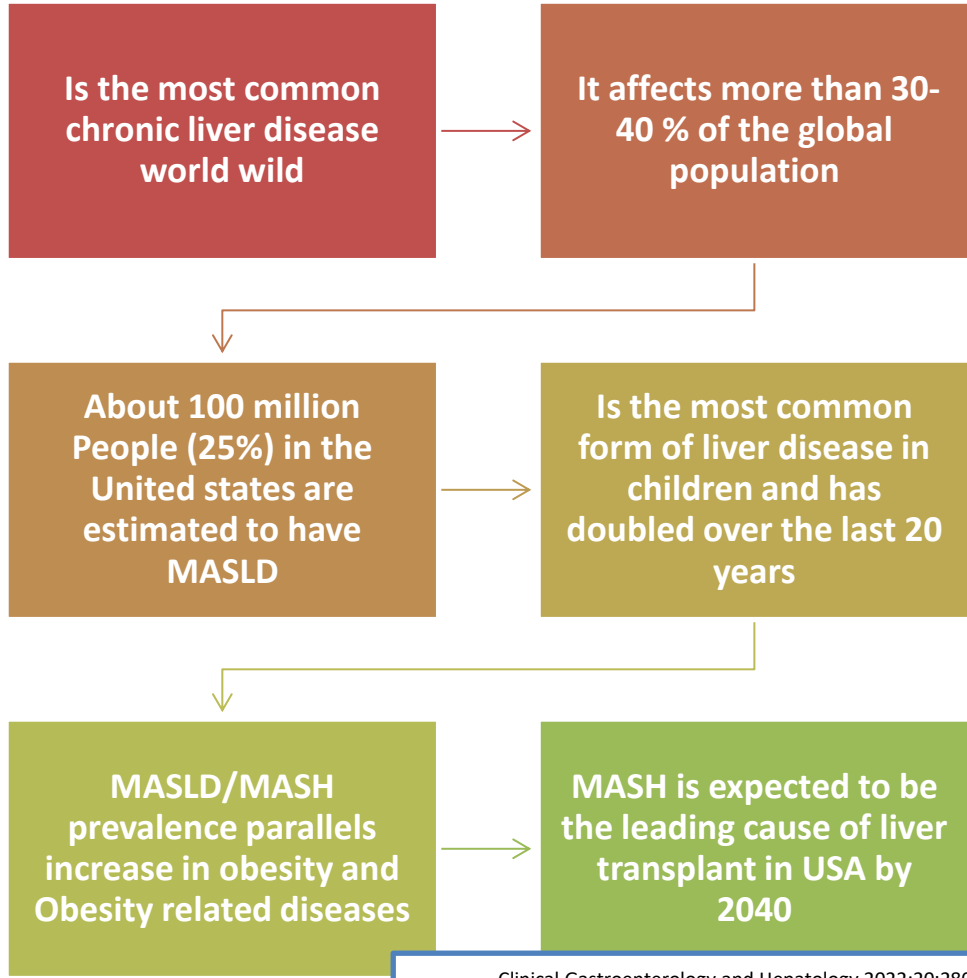
MASLD

Formerly Known as NAFLD

MASLD

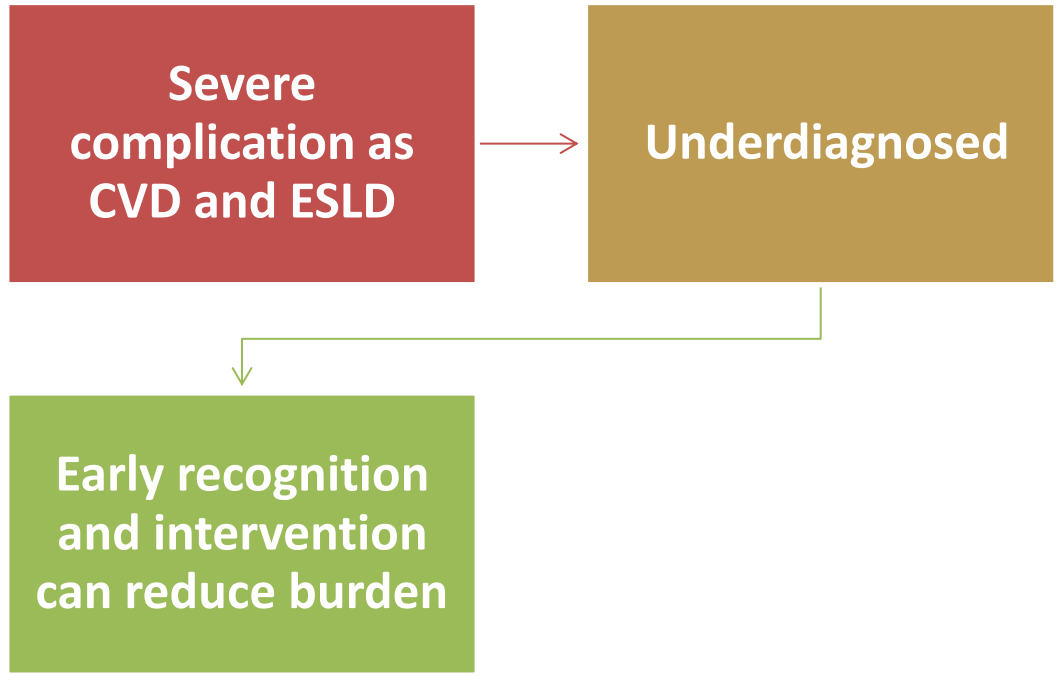
Why It Matters

MASLD



Clinical Gastroenterology and Hepatology 2022;20:2809–2817

Clinical Burden



Chan Wk,et al. J Obes Metab Synd. 2023;32(3):197-213. Shieh C,et al. JGH Open.2020;4(5):1000-1006

MASLD

Hepatic Steatosis is the hallmark

One or more CMRFs

no other cause of steatosis

MetLAD

Hepatic Steatosis is the hallmark

CMRFs

Alcohol consumption above MASLD threshold but below ALD ranges

*Cardiometabolic criteria

Adult Criteria

At least 1 out of 5:

- ☐ BMI ≥ 25 kg/m² [23 Asia] **OR** WC > 94 cm (M) 80 cm (F) **OR** ethnicity adjusted equivalent
- ☐ Fasting serum glucose ≥ 5.6 mmol/L [100 mg/dL] **OR** 2-hour post-load glucose levels ≥ 7.8 mmol/L [≥140 mg/dL] **OR** HbA1c ≥ 5.7% [39 mmol/L] **OR** type 2 diabetes **OR** treatment for type 2 diabetes
- ☐ Blood pressure ≥ 130/85 mmHg **OR** specific antihypertensive drug treatment
- ☐ Plasma triglycerides ≥ 1.70 mmol/L [150 mg/dL] **OR** lipid lowering treatment
- ☐ Plasma HDL-cholesterol ≤ 1.0 mmol/L [40 mg/dL] (M) and ≤ 1.3 mmol/L [50 mg/dL] (F) **OR** lipid lowering treatment

Pediatric Criteria

At least 1 out of 5:

- ☐ BMI ≥ 85th percentile for age/sex [BMI z score ≥ +1] **OR** WC > 95th percentile **OR** ethnicity adjusted equivalent
- ☐ Fasting serum glucose ≥ 5.6 mmol/L [≥ 100 mg/dL] **OR** serum glucose ≥ 11.1 mmol/L [≥ 200 mg/dL] **OR** 2-hour post-load glucose levels ≥ 7.8 mmol [140 mg/dL] **OR** HbA1c ≥ 5.7% [39 mmol/L] **OR** already diagnosed/treated type 2 diabetes **OR** treatment for type 2 diabetes
- ☐ Blood pressure age < 13y, BP ≥ 95th percentile **OR** ≥ 130/80 mmHg (whichever is lower); age ≥ 13y, 130/85 mmHg **OR** specific antihypertensive drug treatment
- ☐ Plasma triglycerides < 10y, ≥ 1.15 mmol/L [≥ 100 mg/dL]; age ≥ 10y, ≥ 1.70 mmol/L [≥ 150 mg/dL] **OR** lipid lowering treatment
- ☐ Plasma HDL-cholesterol ≤ 1.0 mmol/L [≤ 40 mg/dL] **OR** lipid lowering treatment

Diagnostic criteria for MASLD

MASLD (Rinella *et al.*, 2023)[12]	
Hepatic steatosis	Evidence of hepatic steatosis in adults (By imaging techniques or histological techniques)
Cardiometabolic factor	At least 1 of the 5 cardiometabolic criteria: • BMI ≥ 25 kg/m^2 (≥ 23 kg/m^2 in Asians) or waist circumference ≥ 94/80 cm in men and women or ethnicity-adjusted thresholds • Fasting serum glucose ≥ 5.6 mmol/L (100 mg/dL) or 2-h post-load glucose ≥ 7.8 mmol/L (≥ 140 mg/dL) or HbA1c ≥ 5.7% (39 mmol/L) or type 2 diabetes or treatment for type 2 diabetes • Blood pressure > 130/85 mmHg or specific antihypertensive drug treatment • Plasma triglycerides ≥ 1.70 mmol/L (150 mg/dL) or lipid lowering treatment • Plasma HDL-cholesterol ≤ 1.0 mmol/L (40 mg/dL) in men and ≤ 1.3 mmol/L (50 mg/dL) in women or lipid lowering treatment
Alcohol	< 140 g/week in women, < 210 g/week in men, exceeding these thresholds classifies as MetALD or ALD

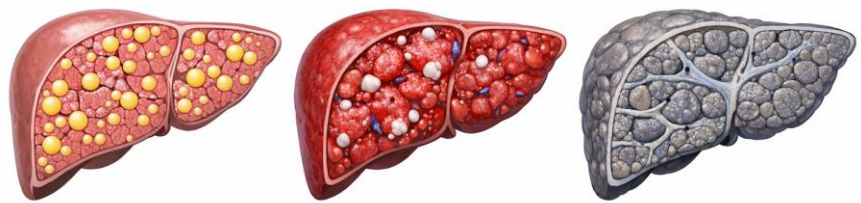
MASLD: Metabolic dysfunction-associated steatotic liver disease; MetALD: metabolic dysfunction-associated alcohol-related liver disease; ALD: alcohol-related liver disease; BMI: body mass index; HbA1c: glycated hemoglobin; HDL: high-density lipoprotein.

Estimated Prevalence in High-Risk groups



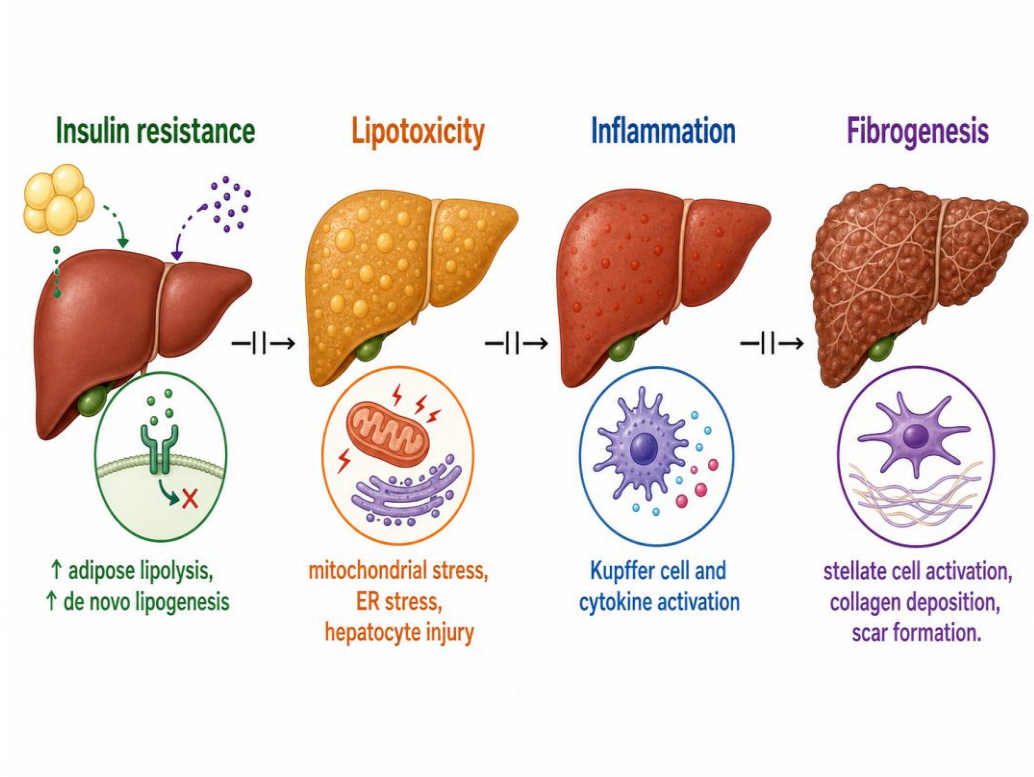
Hepatic steatosis to steatohepatitis

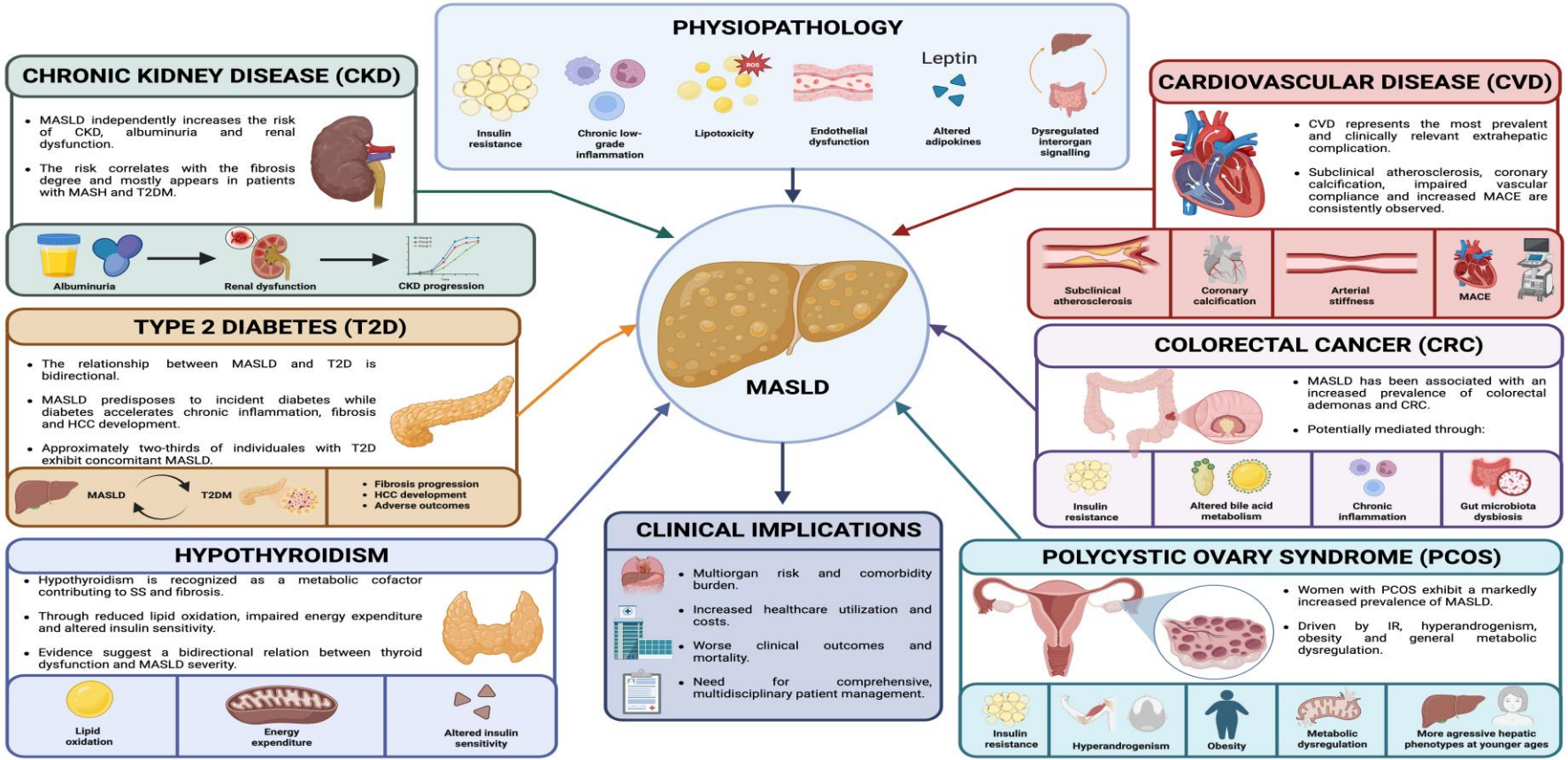
- Steatosis: Accumulation of fat in liver cells.
- Steatohepatitis: Inflammation and liver cell damage.
- Fibrosis: Scar tissue formation due to chronic inflammation.
- Risk factors: Obesity, insulin resistance, and dysglycemia.



MASLD/MASH Pathophysiology

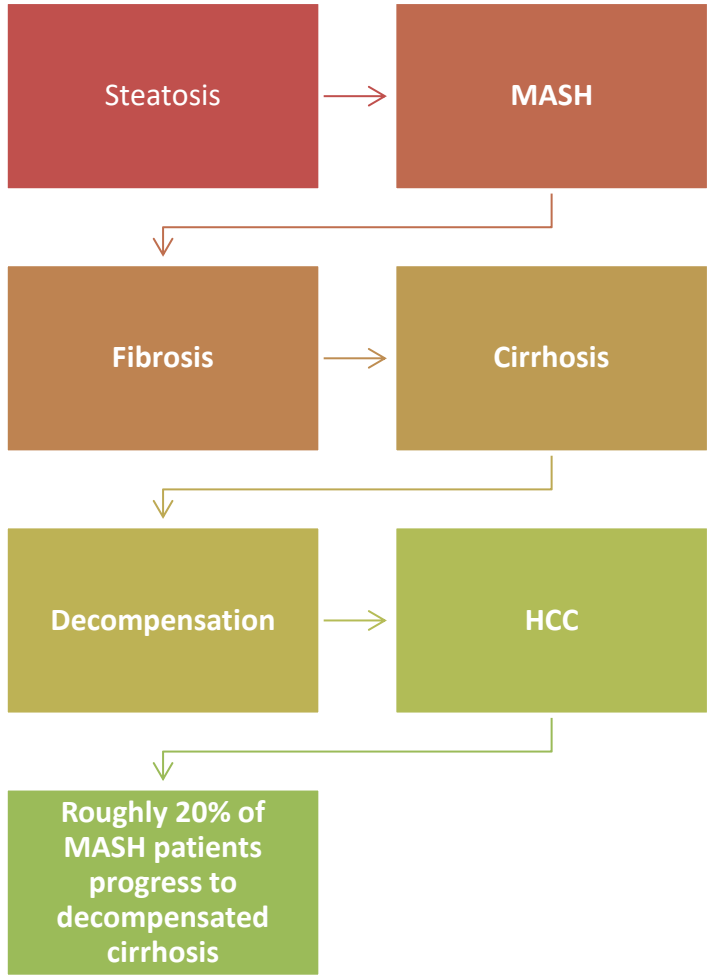
- Insulin resistance increases adipose lipolysis and hepatic de novo lipogenesis, driving triglyceride accumulation.
- Excess free fatty acids and toxic lipid cause mitochondrial stress, and hepatocyte injury.
- Injured hepatocytes activate Kupffer cells and cytokine pathways, sustaining inflammation and cell death.
- Hepatic stellate cell activation promotes collagen deposition, scar formation, and progressive fibrogenesis.

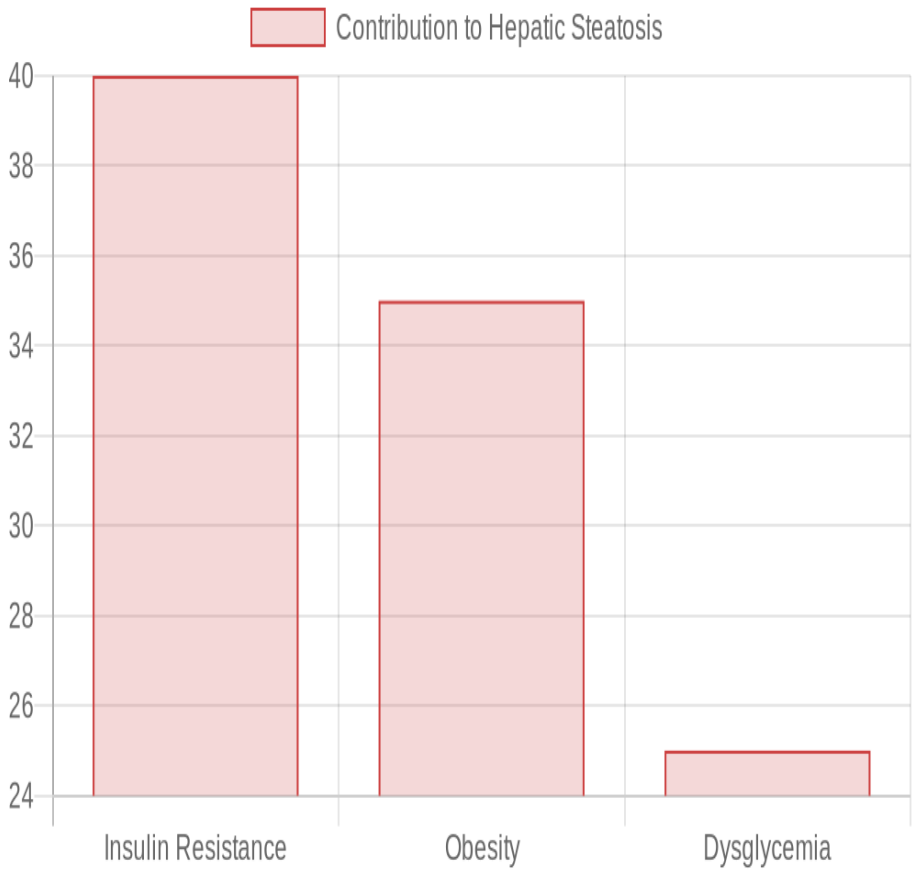




- Extrahepatic manifestations of MASLD. Created in BioRender. Ramírez, M. (2026) https://BioRender.com/ce9bge2. MASLD: Metabolic dysfunction-associated steatotic liver disease; CKD: chronic kidney disease; CVD: cardiovascular disease; MACE: major adverse cardiovascular events; T2D: type 2 diabetes; CRC: colorectal cancer; MAFLD: metabolic-associated fatty liver disease; HCC: hepatocellular carcinoma; PCOS: polycystic ovary syndrome; SS: simple steatosis; IR: insulin resistance.

Natural History





Pathophysiology linking insulin resistance, obesity, and dysglycemia to hepatic steatosis

- Insulin resistance, obesity, and dysglycemia are key contributors to hepatic steatosis. Insulin resistance leads to increased lipolysis and free fatty acid flux to the liver. Obesity exacerbates this by promoting adipose tissue dysfunction. Dysglycemia further impairs hepatic lipid metabolism, collectively driving steatosis.

Genetic Drives

PNPLA3, TM6SF2, MBOAT7, HSD17B13 influence steatosis and fibrosis risk



Environmental Factors

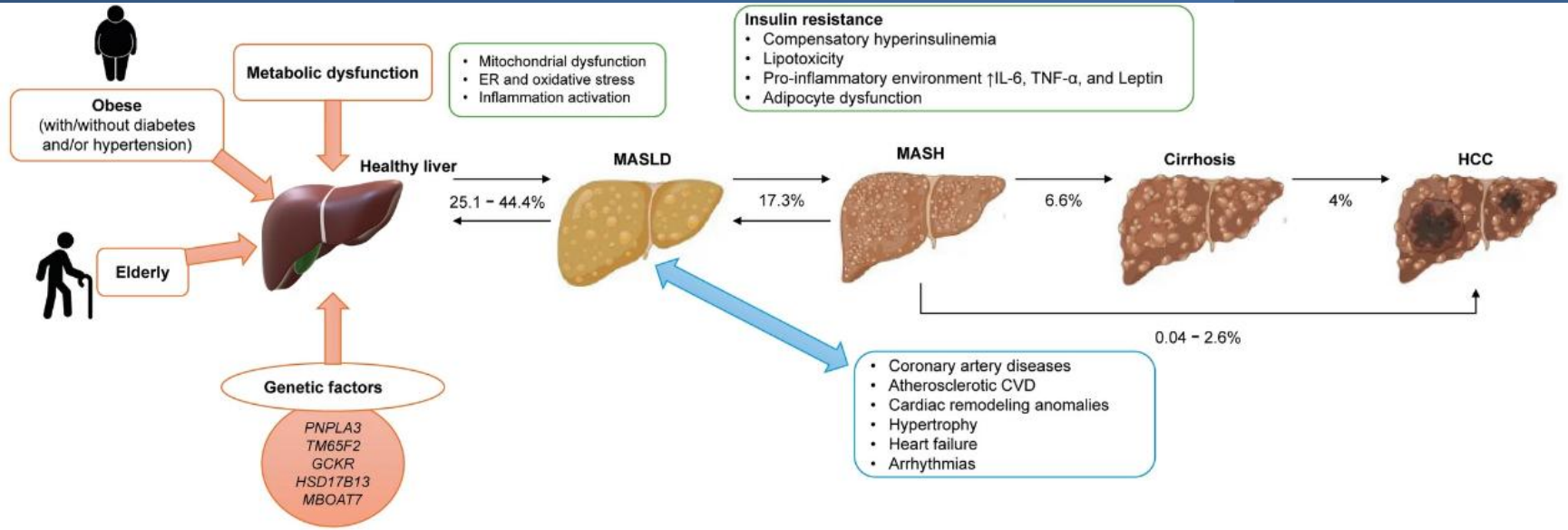
- Fructose intake
- Cholesterol intake
- Alcohol intake

Nagai K, et al. Frontiers of Collabration between primary care and specialist in the management of metabolic Dysfunction-Associated steatotic liver disease. A Review. Life(Basel).2023:13(11):2144.

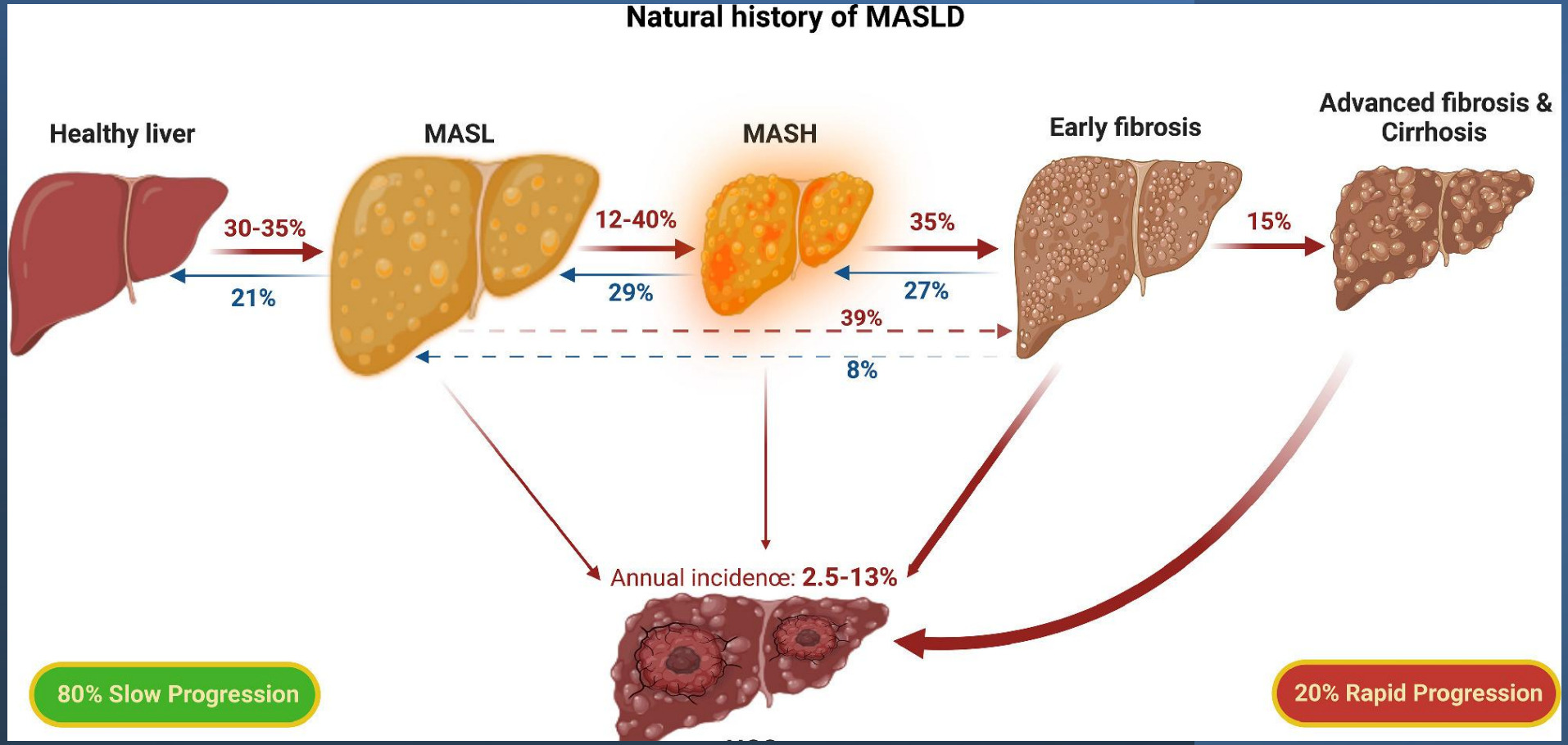
Liver Fibrosis and MASH Outcomes

- The degree of liver fibrosis is a critical predictor of outcomes in MASLD and MASH. Patients with advanced fibrosis or cirrhosis face higher risks of liver-related and all-cause mortality.

- MASH-related liver disease is now the second leading cause of liver transplantation in the U.S., and the leading cause in women.



Pathogenesis and progression of MASLD/MASH-HCC. *CVD* cardiovascular disease, *ER* endoplasmic reticulum, GCKR glucose kinase regulator, *HCC* hepatocellular carcinoma, *HSD17B13* hydroxysteroid 17-beta dehydrogenase 13, *IL* interleukin, *MASH* metabolic dysfunction-associated steatohepatitis, *MASLD* metabolic dysfunction-associated steatotic liver disease, *MBOAT7* membrane-bound O-acyltransferase domain-containing 7, *PNPLA3* patatin-like phospholipase domain-containing protein 3, *TM6SF2* transmembrane 6 superfamily member 2, *TNF* tumor necrosis factor


Natural history of MASLD
Healthy liver
MASL
MASH
Early fibrosis
Advanced fibrosis & Cirrhosis
30-35%
21%
12-40%
29%
35%
27%
15%
39%
8%
Annual incidence: 2.5-13%
80% Slow Progression
20% Rapid Progression

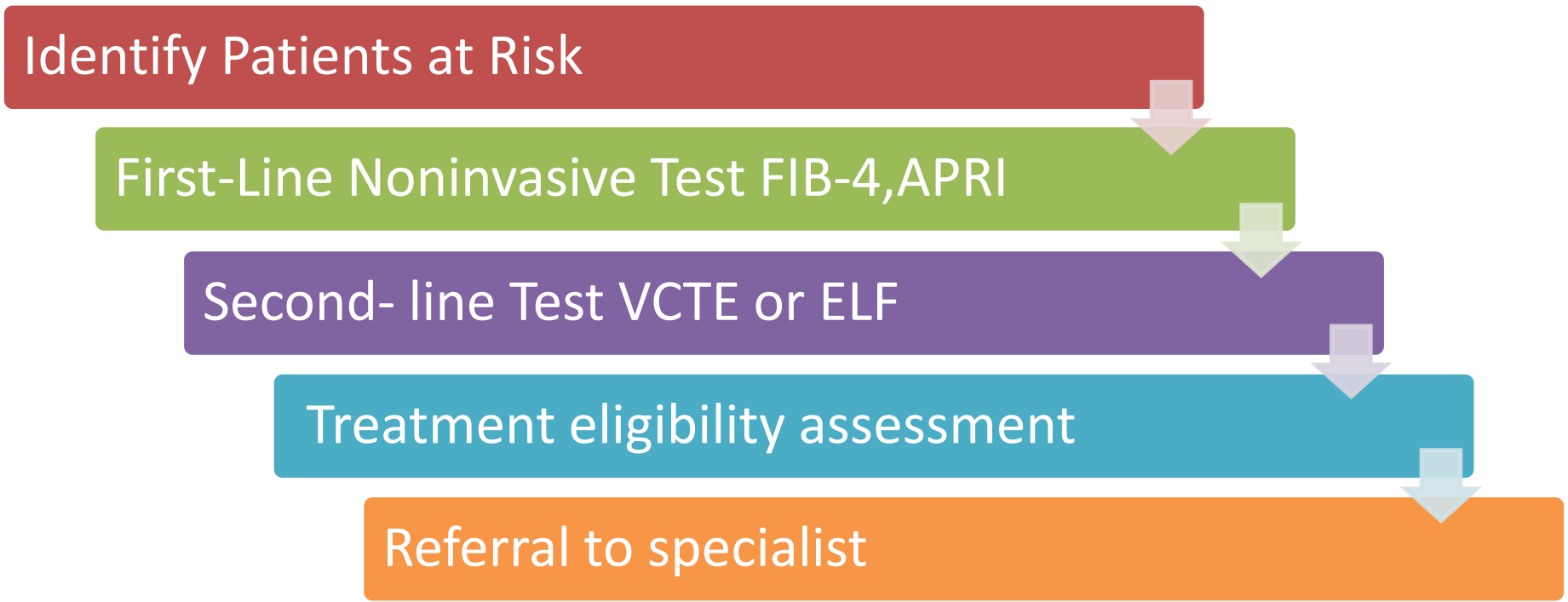
MASH

Hepatic Steatohepatitis is the hallmark

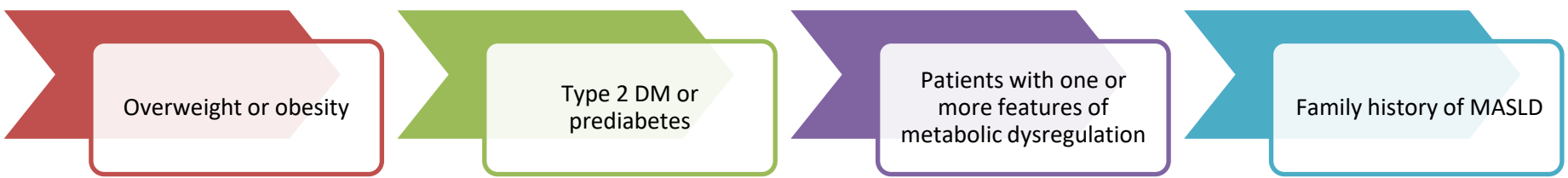
15-40% of patient of MASLD develop MASH

25-30 %of MASH progress to advanced fibrosis in 9-10 years

Clinical Algorithm Overview



Patients at Risk



Non-Invasive Liver Disease Assessment (NILDA)

- Blood based test
- Imaging based
- Combination

Common NILDA

Blood based

Fib-4	**ASL, ALT,Age,PLT**
APRI	AST, PLT
NAFLD score	AST,ALT,AGE,PLT,BMI,Alb
ELF	TIMPI, PIIINP,HA
Fibrosure	A2macroglobulin, apoliporotein1, haptoglobin , Bilirubin, GGT

Imaging Based

VCTE (Fibroscan)	Liver stiffness
Shear wave Elastography	Liver stiffness
MRE	Liver stiffness

Blood Based NILDA

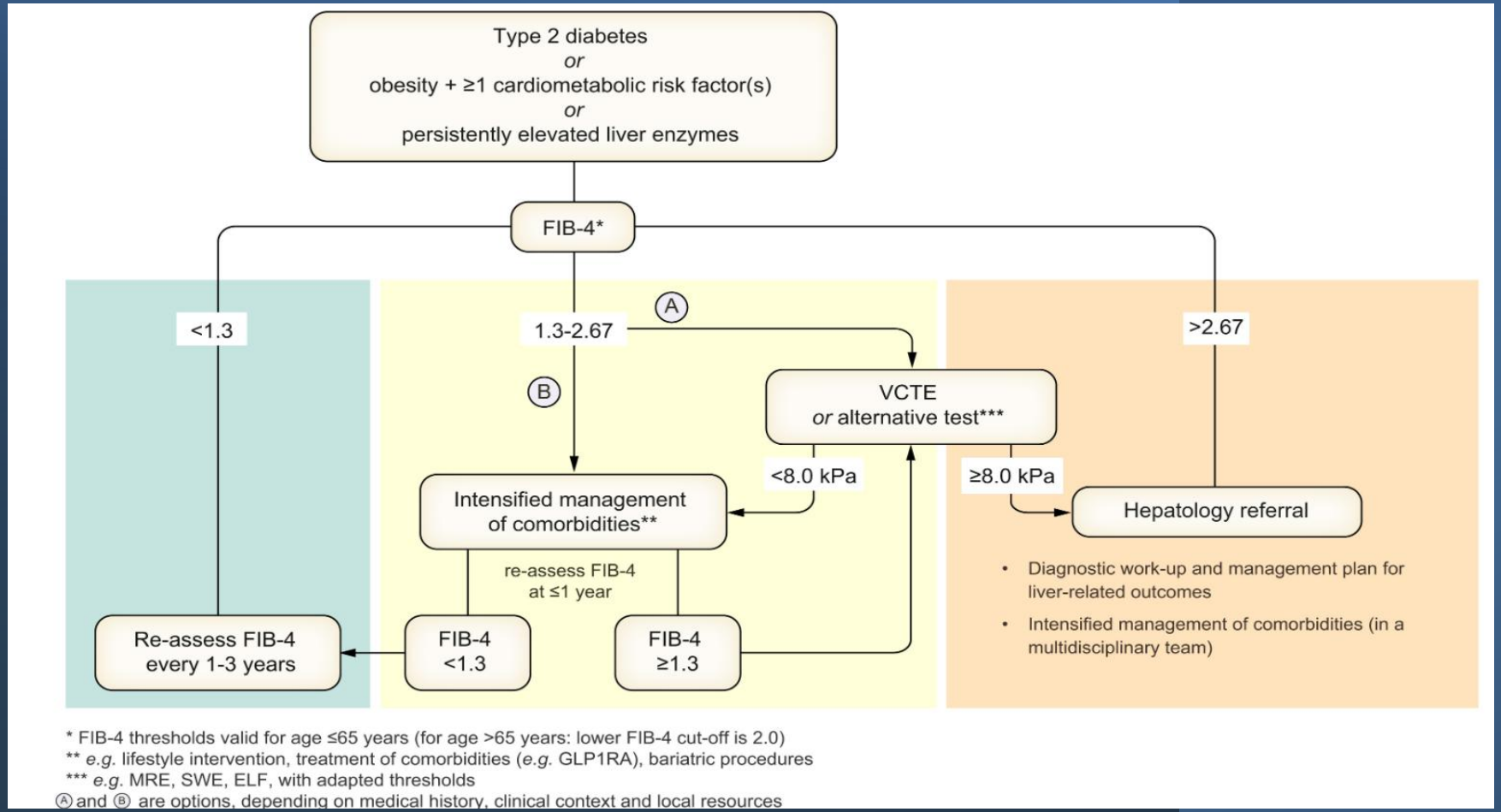
Clinical condition	Tools affected	
Age	Fib-4 /ELF	age extremes test cannot be performed
Splenectomy	FIB-4/APRI	these tools use platelets as a biomarker of portal hypertension, attenuated thrombocytopenia from splenectomy gives a falsely lower estimation.
Thrombocytopenia	APRI /FIB-4	thrombocytopenia from other conditions gives a falsely higher estimation.
CKD	APRI/ FIB-4	Elevated urea levels can result in falsely lower estimation. Hemodialysis patients tend to have lower ALT and AST levels, resulting in falsely lower estimation.
Malnutrition	NAFLD score	Albumin reduction that is disproportionate to liver dysfunction results in falsely elevated estimation.

What is Fib-4(Fibrosis-4 Index)

- Clinical marker to evaluate the degree of fibrosis
- Simple and easy to use
- Use with caution in patients <35 years and > 65 years old

FIB-4 (Fibrosis-4 index) = age x AST/(platelet count x √(ALT))
(age in years, ALT and AST in U/L, and platelet count in 10^9/L)

AASLD Algorithm for screening



Noninvasive Test for Risk Stratification

Noninvasive tests (NITs) like the FIB-4 score are essential for identifying patients at risk of advanced fibrosis. FIB-4 is a simple calculation using age, AST, ALT, and platelet count. Patients with low scores can often be ruled out for advanced fibrosis, while those with high scores may require further evaluation with imaging techniques like transient elastography or serum-based tests

Screening and Referral guidelines

- Primary care providers are encouraged to use FIB-4 as a first-line screening tool for patients with risk factors. Patients with indeterminate or elevated FIB-4 scores should undergo secondary testing, such as transient elastography, and may require referral to specialists for further management. This approach helps identify the 20-25% of patients with significant fibrosis who are at the highest risk.



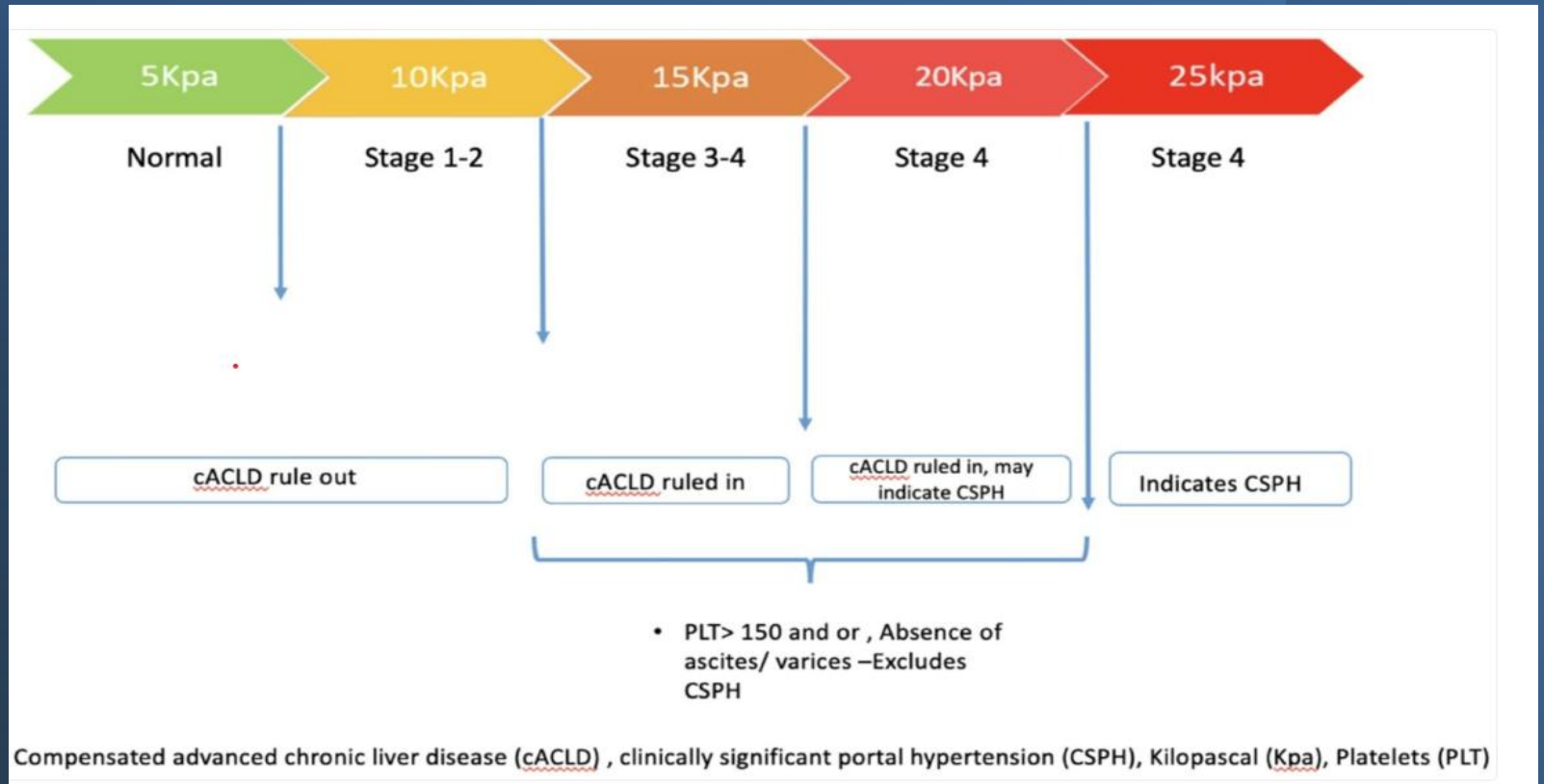
Vibration-Controlled Transient Elastography

- FDA approved in US 2013
- Fasting for 3 hours
- Very fast and easy test do (5 minutes)
- Less expensive than liver biopsy

Limitation of VCTE

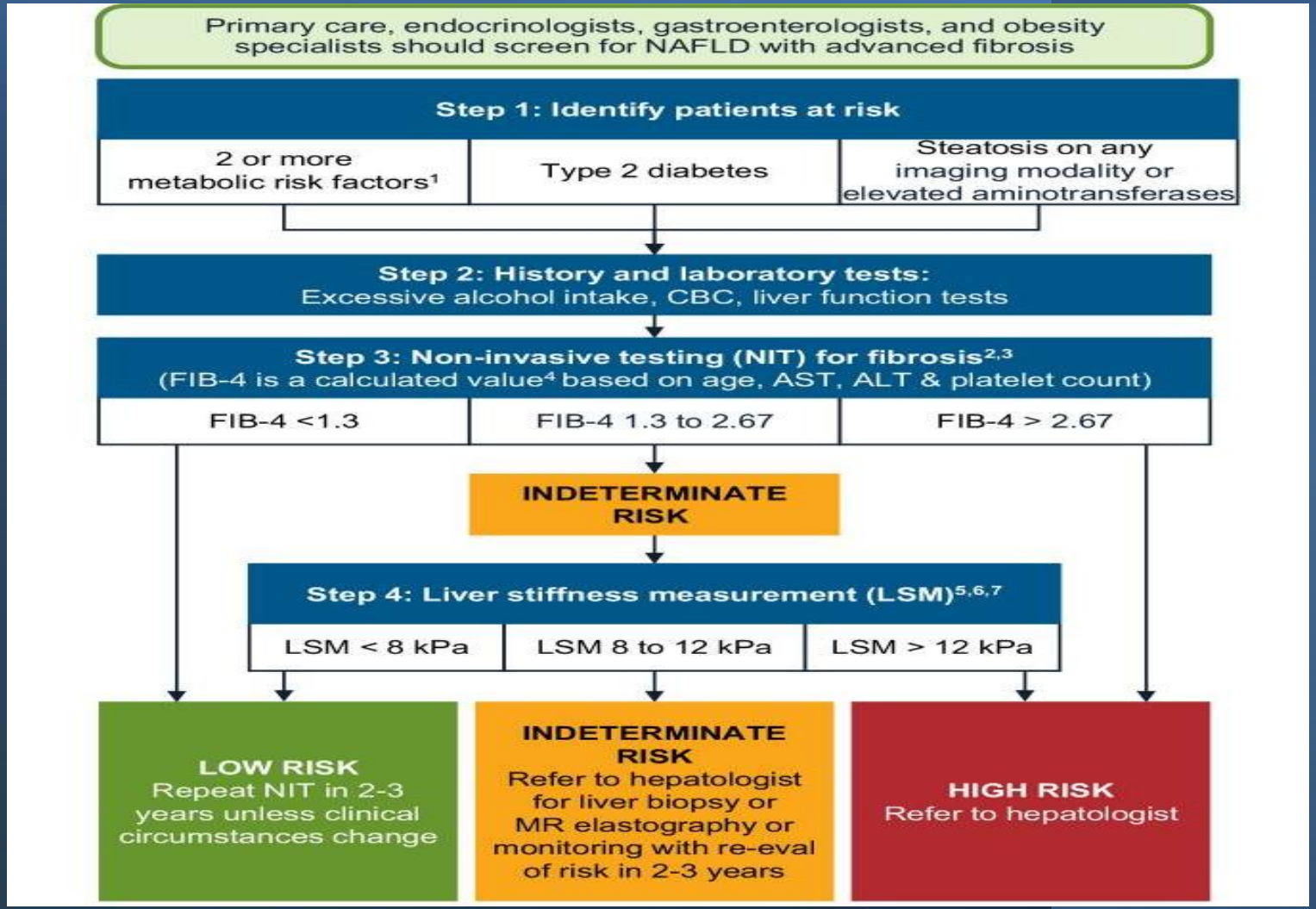
- Recent Food Intake
- High liver inflammation
- Severe obesity
- Right Side Heart Failure

Liver Stiffness Measurement and Liver Disease



Liver Stiffness-Based Risk Stratification for Advanced Fibrosis and Portal Hypertension Using the "Rule of 5"

Curr Hepatol Rep. 2025 Nov 27;24(1):10.1007/s11901-025-00713-7


Primary care, endocrinologists, gastroenterologists, and obesity specialists should screen for NAFLD with advanced fibrosis
Step 1: Identify patients at risk
2 or more metabolic risk factors[1]
Type 2 diabetes
Steatosis on any imaging modality or elevated aminotransferases
Step 2: History and laboratory tests:
Excessive alcohol intake, CBC, liver function tests
Step 3: Non-invasive testing (NIT) for fibrosis[2,3]
(FIB-4 is a calculated value[4] based on age, AST, ALT & platelet count)
FIB-4 <1.3
FIB-4 1.3 to 2.67
FIB-4 > 2.67
INDETERMINATE RISK
Step 4: Liver stiffness measurement (LSM)[5,6,7]
LSM < 8 kPa
LSM 8 to 12 kPa
LSM > 12 kPa
LOW RISK
Repeat NIT in 2-3 years unless clinical circumstances change
INDETERMINATE RISK
Refer to hepatologist for liver biopsy or MR elastography or monitoring with re-eval of risk in 2-3 years
HIGH RISK
Refer to hepatologist

Enhanced liver Fibrosis(ELF)

High cost and limited Accessibility

ELF endorsed when VCTE unavailable or unreliable

ELF <7.7= low risk; ELF ≥9.8 referral threshold; ELF ≥11.3 suggests cirrhosis.

Enhanced liver Fibrosis(ELF)

Low risk group	Intermediate Risk group	High risk Group
< 7.7	7.7-9.8	>9.8

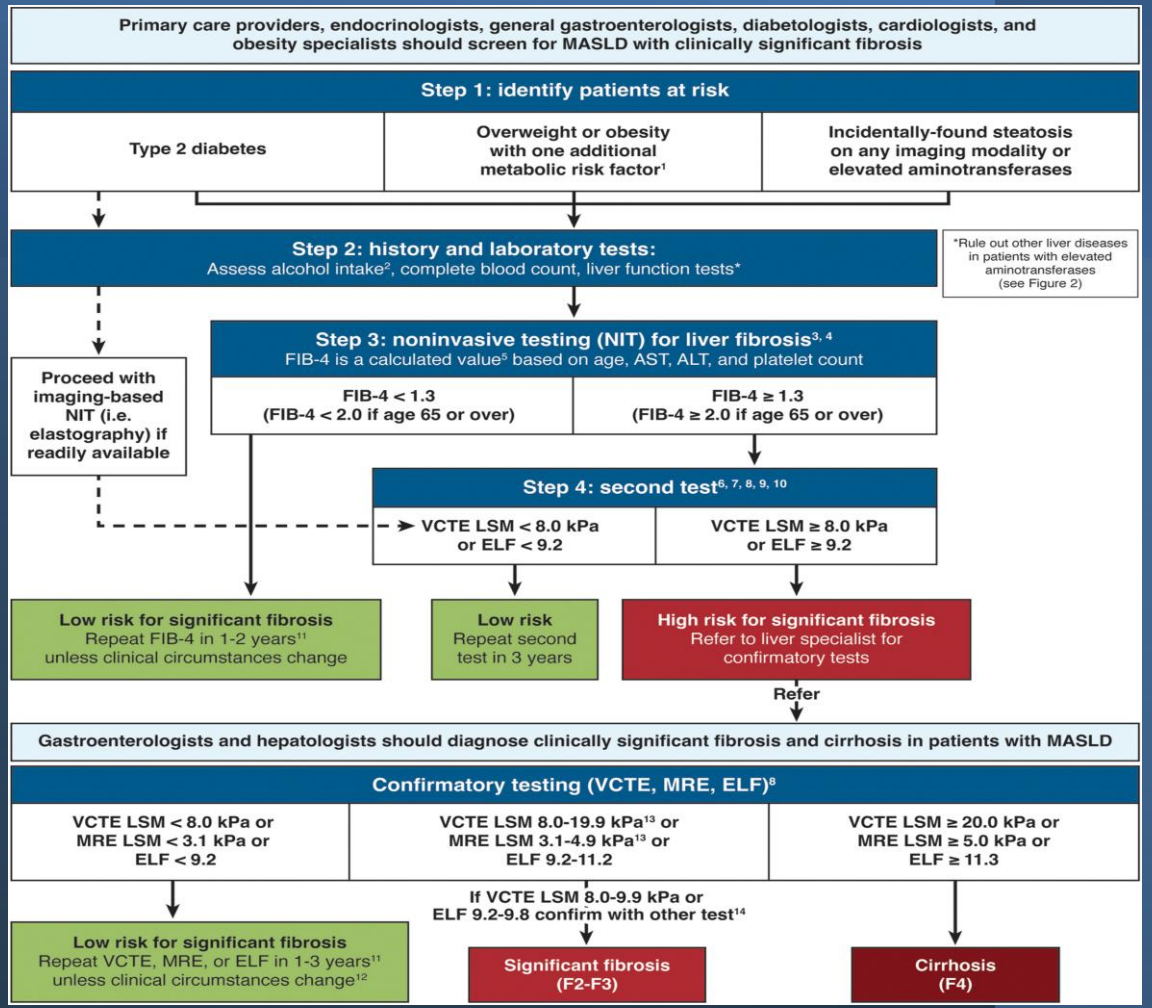
Magnetic Resonance Elastography

- Outperforms VTCE ruling in earlier stage of Fibrosis F2 or greater
- Similar accuracy in ruling in advanced fibrosis F3 and F4
- Superior performance particularly in obese patients
- High cost and limited availability

Magnetic Resonance Elastography

MRE SCORE (kilopascals/Kpa)	Corresponding METAVIR Stage	Fibrosis Severity
<2.5 KPa	**F0 - F1**	Normal/Normal variant
2.5 to 2.9 KPa	**F1**	Borderline/mild Fibrosis
3.0 to 3.5 KPa	**F2**	Significant/Moderate fibrosis
3.6 to 4.9 KPa	**F3**	Advanced/Severe Fibrosis
>5.0 KPa	**F4**	Cirrhosis

AGA Guidelines



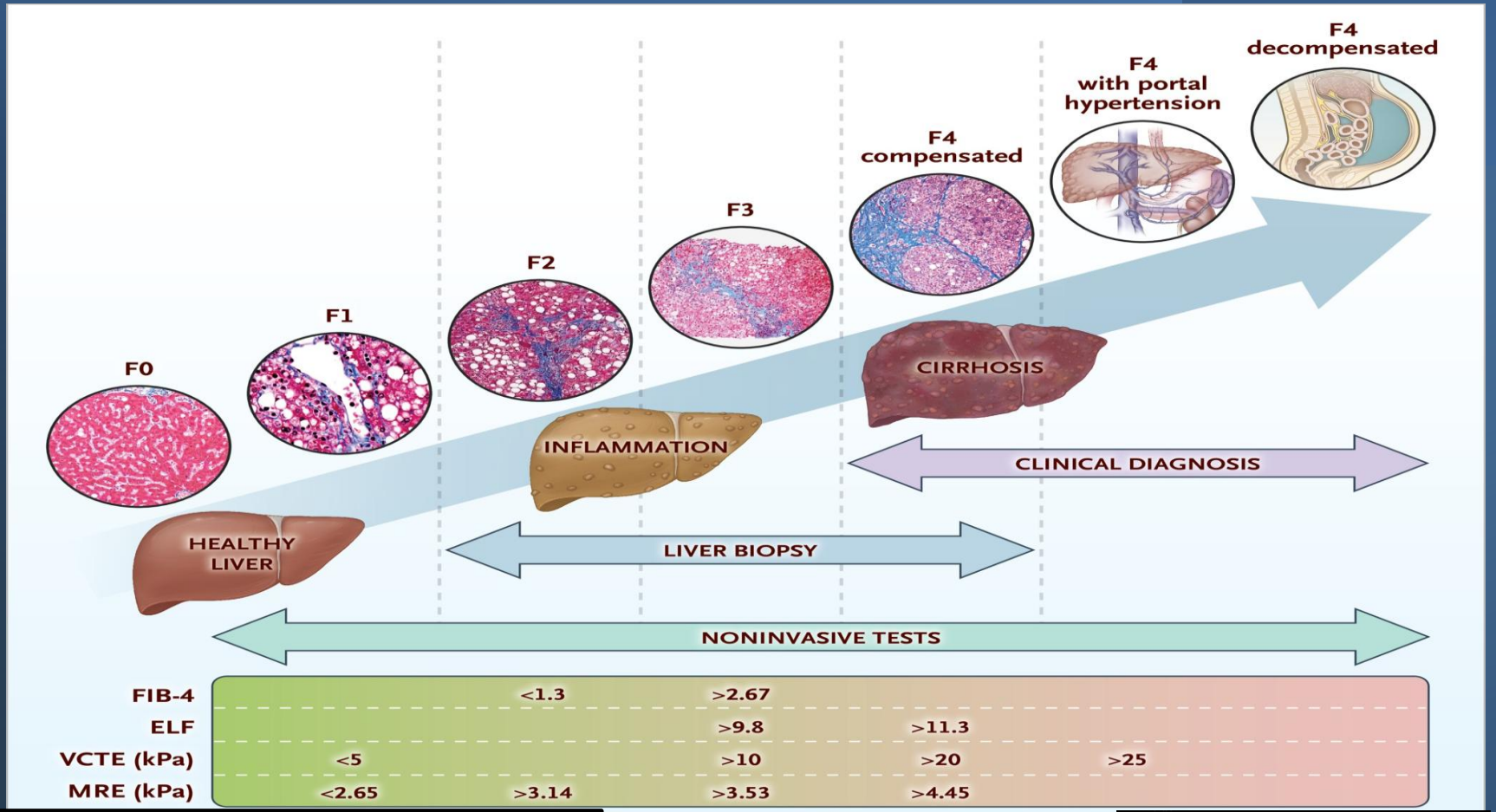
METAVIR Fibrosis Stages

METAVIR Stage	Clinical meaning	
F0	**No fibrosis**	Healthy liver
F1	**Mild Fibrosis/scaring**	Portal tract fibrosis without septa formation/ minimal scaring
F2	**Moderate/significant fibrosis**	Portal tract fibrosis with infrequent septal formation
F3	**Advanced/Severe Fibrosis**	Numerous septa/ bridging fibrosis
F4	**Cirrhosis**	Advanced scarring

Liver biopsy

- Gold standard for diagnosis of MASH and for hepatic fibrosis staging
- Discordance between NITs and imaging, or indeterminate results that leave fibrosis risk unresolved
- Diagnostic uncertainty or suspected competing/overlapping etiology(as viral hepatitis , Drug induced injury or genetic liver disease)
- Confirmation of MASH activity
- Patients with Persistently elevated ALT and AST > 6 months

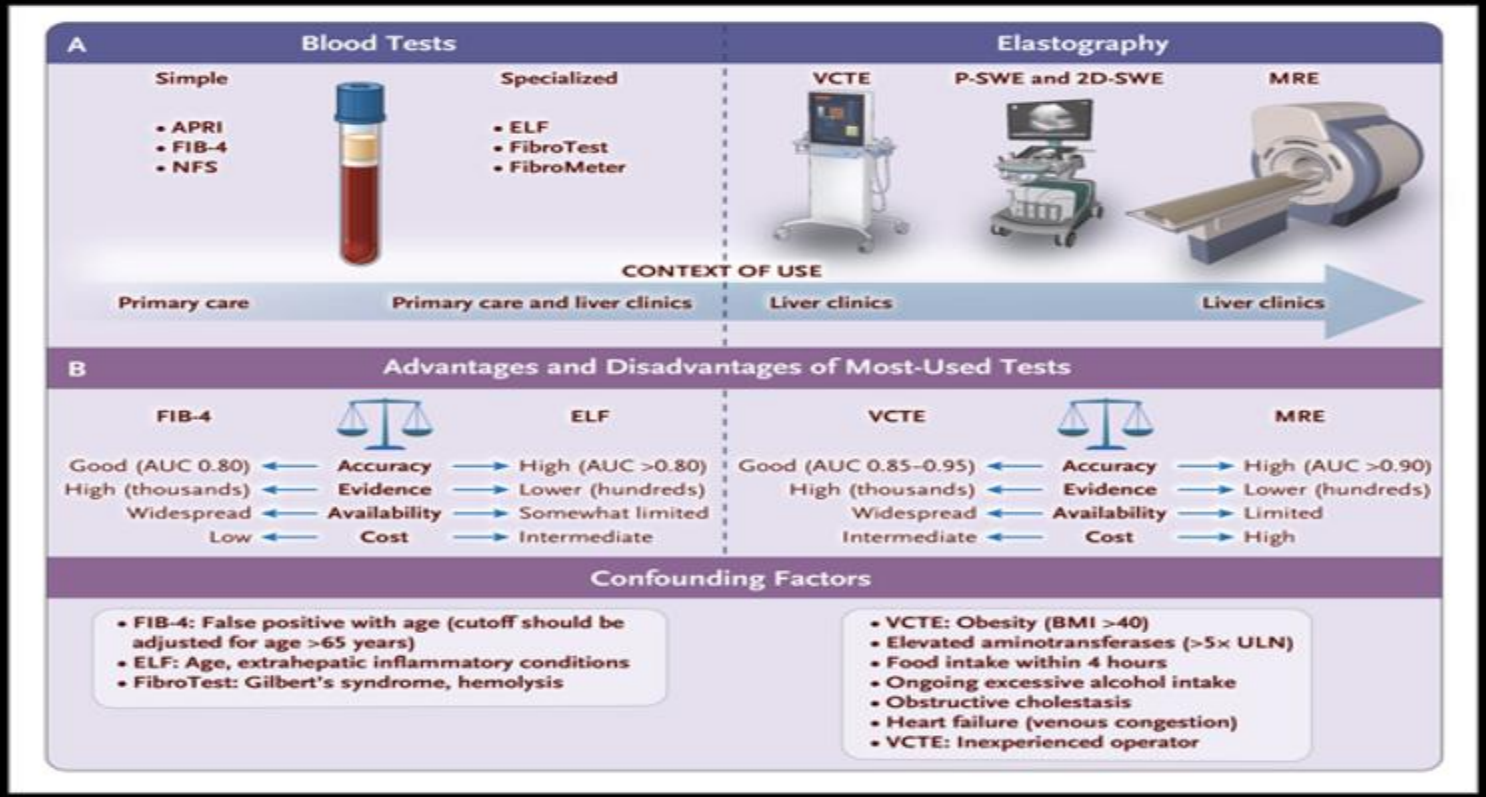
Non-Invasive Liver Disease Assessment



Castera l et al. N Engl J Med2025;393:1715-1729

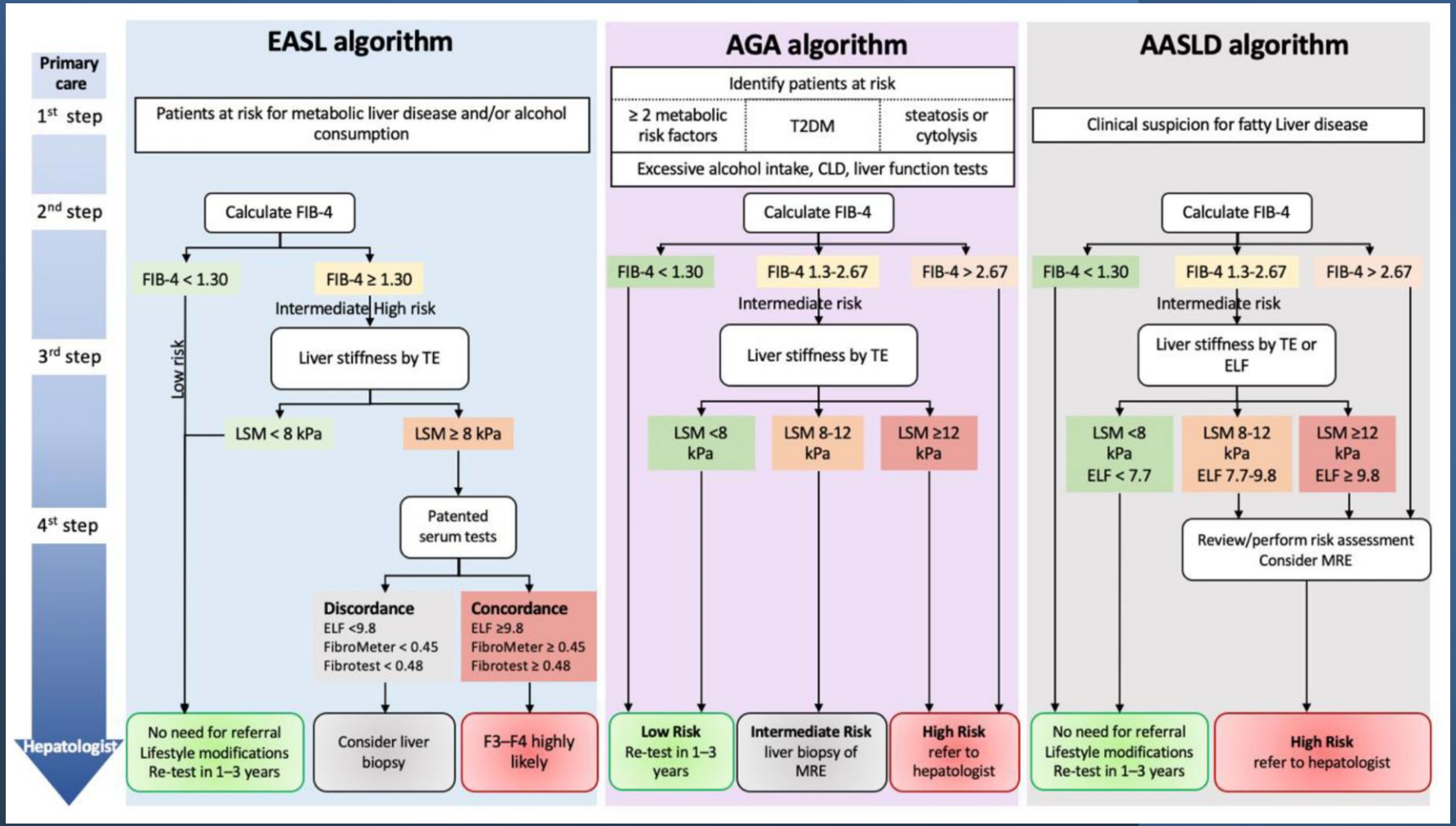
The NEW ENGLAND JOURNAL of MEDICINE

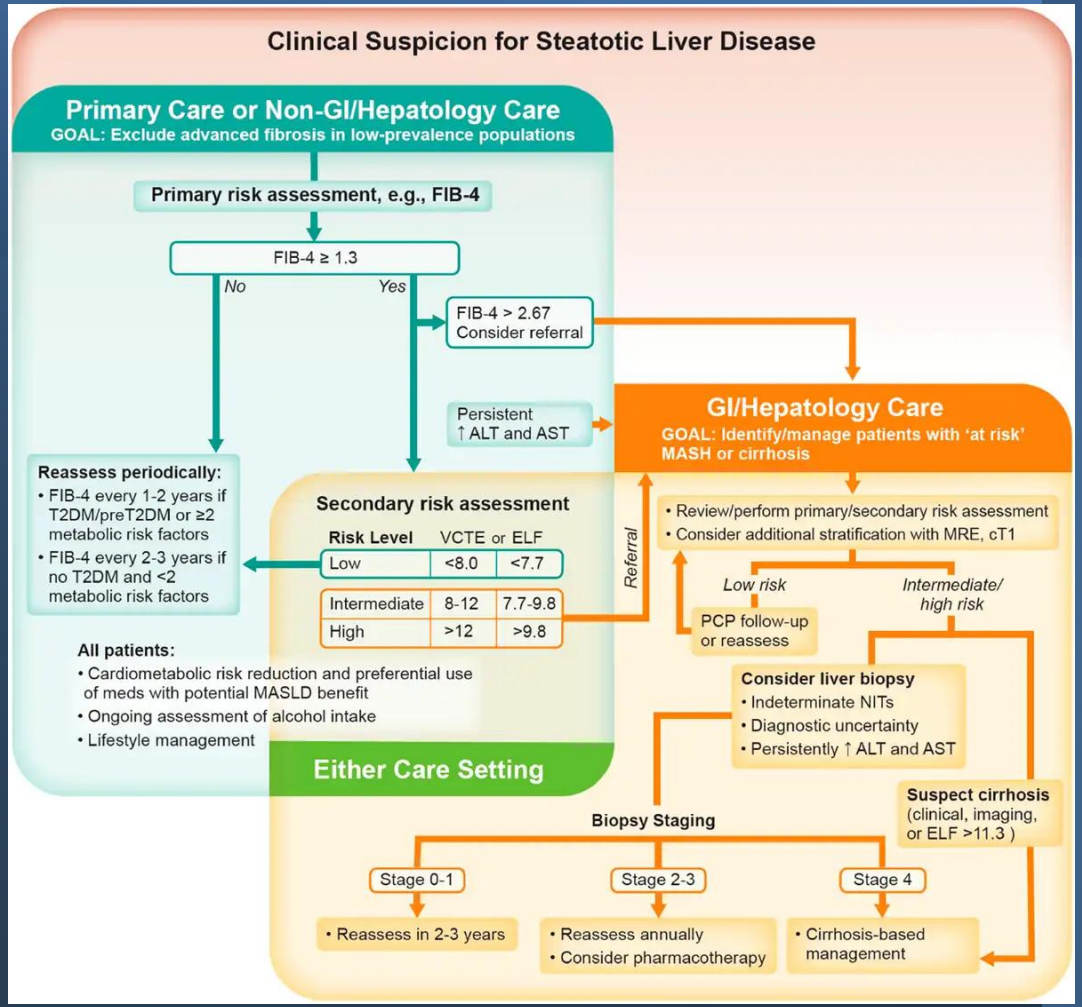
Noninvasive Tests for Live Fibrosis Assessment



Castera L et al. N Engl J Med2025;393:1715-1729

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Primary care
1st step
2nd step
3rd step
4st step
Hepatologist
EASL algorithm
Patients at risk for metabolic liver disease and/or alcohol consumption
Calculate FIB-4
FIB-4 < 1.30
FIB-4 ≥ 1.30
Intermediate/High risk
Low risk
Liver stiffness by TE
LSM < 8 kPa
LSM ≥ 8 kPa
Patented serum tests
Discordance
ELF <9.8
FibroMeter < 0.45
Fibrotest < 0.48
Concordance
ELF ≥9.8
FibroMeter ≥ 0.45
Fibrotest ≥ 0.48
No need for referral
Lifestyle modifications
Re-test in 1–3 years
Consider liver biopsy
F3–F4 highly likely
AGA algorithm
Identify patients at risk
≥ 2 metabolic risk factors
T2DM
steatosis or cytolysis
Excessive alcohol intake, CLD, liver function tests
Calculate FIB-4
FIB-4 < 1.30
FIB-4 1.3-2.67
FIB-4 > 2.67
Intermediate risk
Liver stiffness by TE
LSM <8 kPa
LSM 8-12 kPa
LSM ≥12 kPa
Low Risk
Re-test in 1–3 years
Intermediate Risk
liver biopsy of MRE
High Risk
refer to hepatologist
AASLD algorithm
Clinical suspicion for fatty Liver disease
Calculate FIB-4
FIB-4 < 1.30
FIB-4 1.3-2.67
FIB-4 > 2.67
Intermediate risk
Liver stiffness by TE or ELF
LSM <8 kPa ELF < 7.7
LSM 8-12 kPa ELF 7.7-9.8
LSM ≥12 kPa ELF ≥ 9.8
Review/perform risk assessment
Consider MRE
No need for referral
Lifestyle modifications
Re-test in 1–3 years
High Risk
refer to hepatologist


Clinical Suspicion for Steatotic Liver Disease
Primary Care or Non-GI/Hepatology Care
GOAL: Exclude advanced fibrosis in low-prevalence populations
Primary risk assessment, e.g., FIB-4
FIB-4 ≥ 1.3
No
Yes
FIB-4 > 2.67
Consider referral
Persistent
↑ ALT and AST
GI/Hepatology Care
GOAL: Identify/manage patients with 'at risk' MASH or cirrhosis
Reassess periodically:
• FIB-4 every 1-2 years if T2DM/preT2DM or ≥2 metabolic risk factors
• FIB-4 every 2-3 years if no T2DM and <2 metabolic risk factors
Secondary risk assessment
Risk Level
VCTE or ELF
Low
<8.0
<7.7
Intermediate
8-12
7.7-9.8
High
>12
>9.8
Referral
• Review/perform primary/secondary risk assessment
• Consider additional stratification with MRE, cT1
Low risk
Intermediate/ high risk
PCP follow-up or reassess
Consider liver biopsy
• Indeterminate NITs
• Diagnostic uncertainty
• Persistently ↑ ALT and AST
Suspect cirrhosis
(clinical, imaging, or ELF >11.3)
All patients:
• Cardiometabolic risk reduction and preferential use of meds with potential MASLD benefit
• Ongoing assessment of alcohol intake
• Lifestyle management
Either Care Setting
Biopsy Staging
Stage 0-1
Stage 2-3
Stage 4
• Reassess in 2-3 years
• Reassess annually
• Consider pharmacotherapy
• Cirrhosis-based management

Treatment of MASLD, MASH in Primacy Care

Target population: noncirrhotic MASLD/MASH with F2-F3 fibrosis identified by NILDA



Lifestyle interventions for MASLD

- A healthy lifestyle is the foundation of treatment
- Weight loss of 7-10% improves liver histology and is considered the most effective intervention
- Mediterranean diet reduces liver fat and inflammation. It has a strongest evidence
- Reduced ultra processed food and sugary beverages.
- Calorie deficit diet 500 Kcal/day
- Limiting alcohol intake



Lifestyle interventions for MASLD

Weight loss remains the most effective intervention

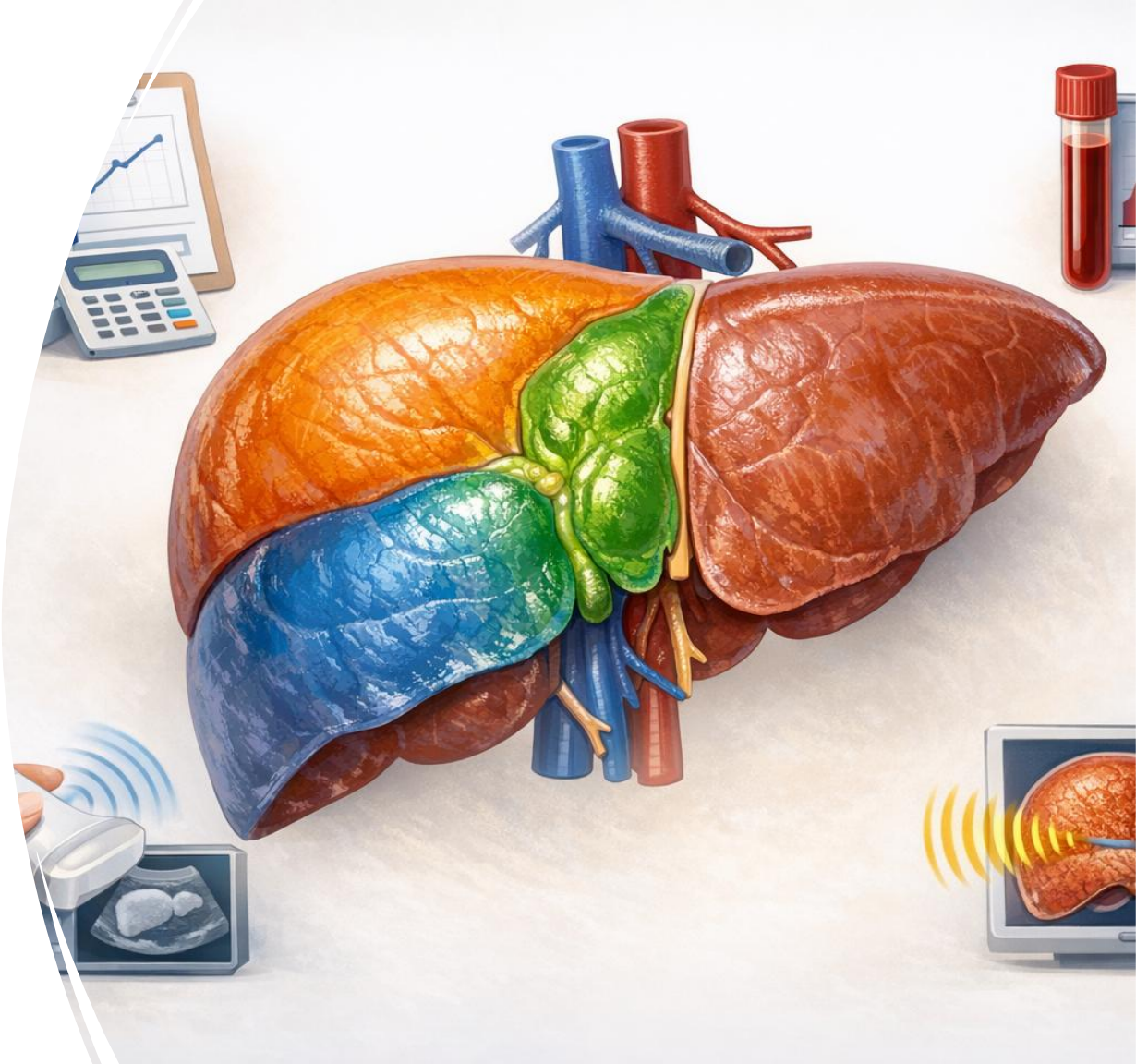
- **≥5% body weight loss** improves steatosis
- **≥7-10% body weight loss** improves steatohepatitis
- **≥10% body weight loss** associated with fibrosis regression



Lifestyle interventions for MASLD

- Regular physical activity enhances insulin sensitivity.
- 30 minutes per day of moderate intensity for at least 150 minutes per week
- 20 minutes per day of vigorous intensity intensity for 75 minutes per week
- Resistance training
- Behavioral therapy supports sustainable lifestyle changes.

Pharmacological Treatment

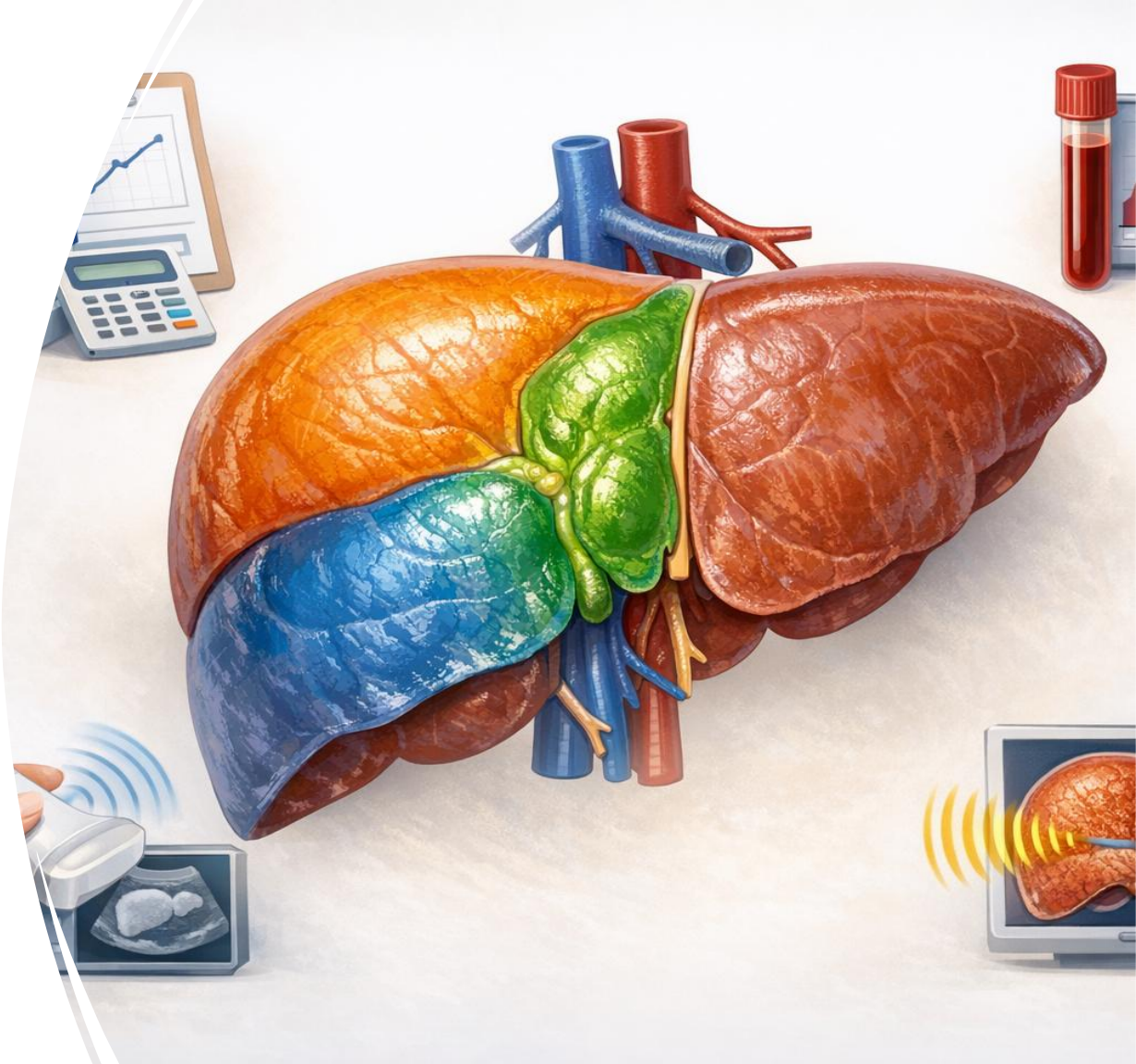


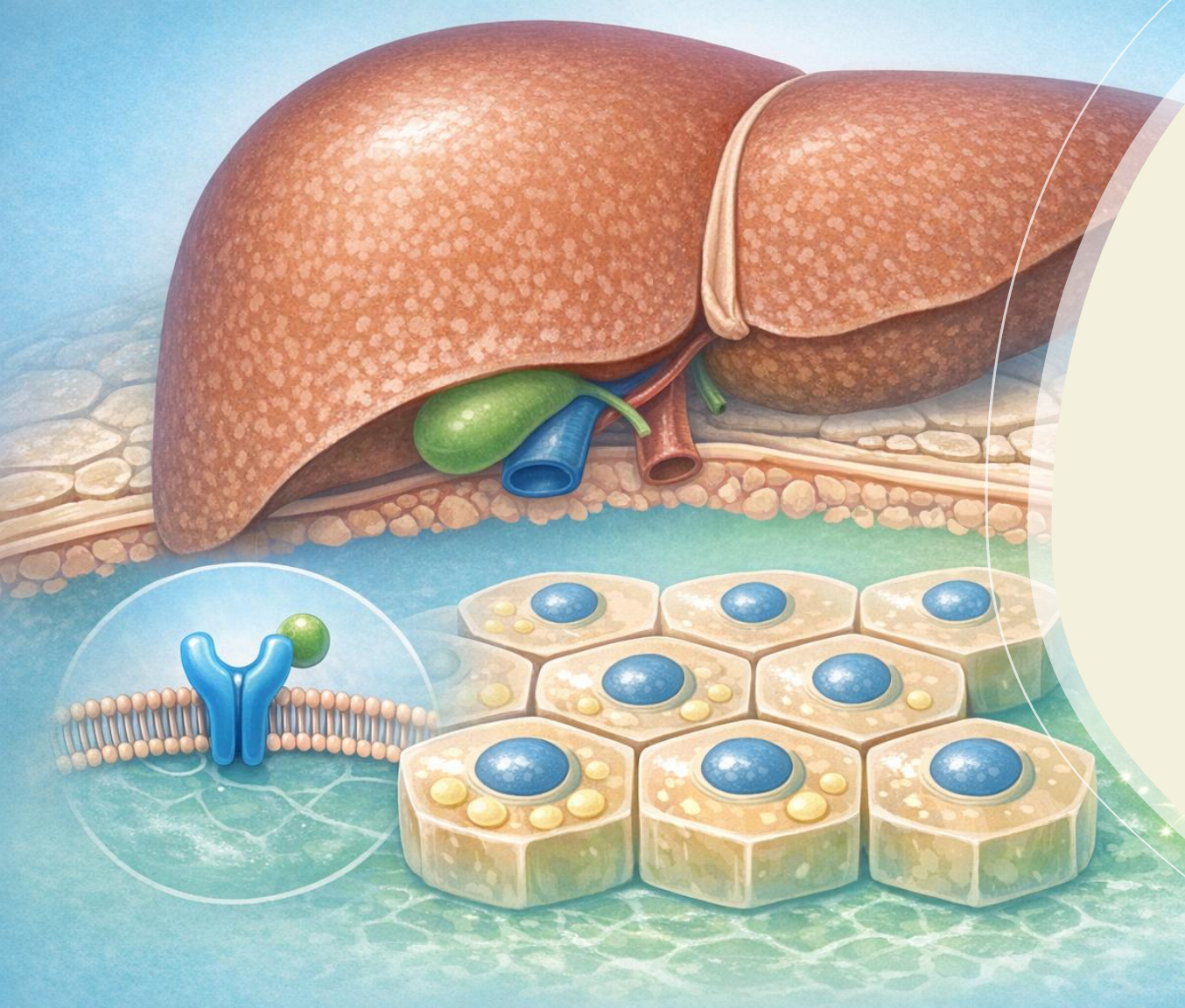
Liver-beneficial antidiabetics for MASLD

- Pioglitazone: Improves liver histology in MASLD
- Metformin: Reduces liver fat content modestly.
- SGLT2 inhibitors: Decrease liver fat and fibrosis risk.
- DPP-4 inhibitors: Potential liver protective effects.
- These medication are not per se approved for MASH or MASLD



New Therapies and Future Direction



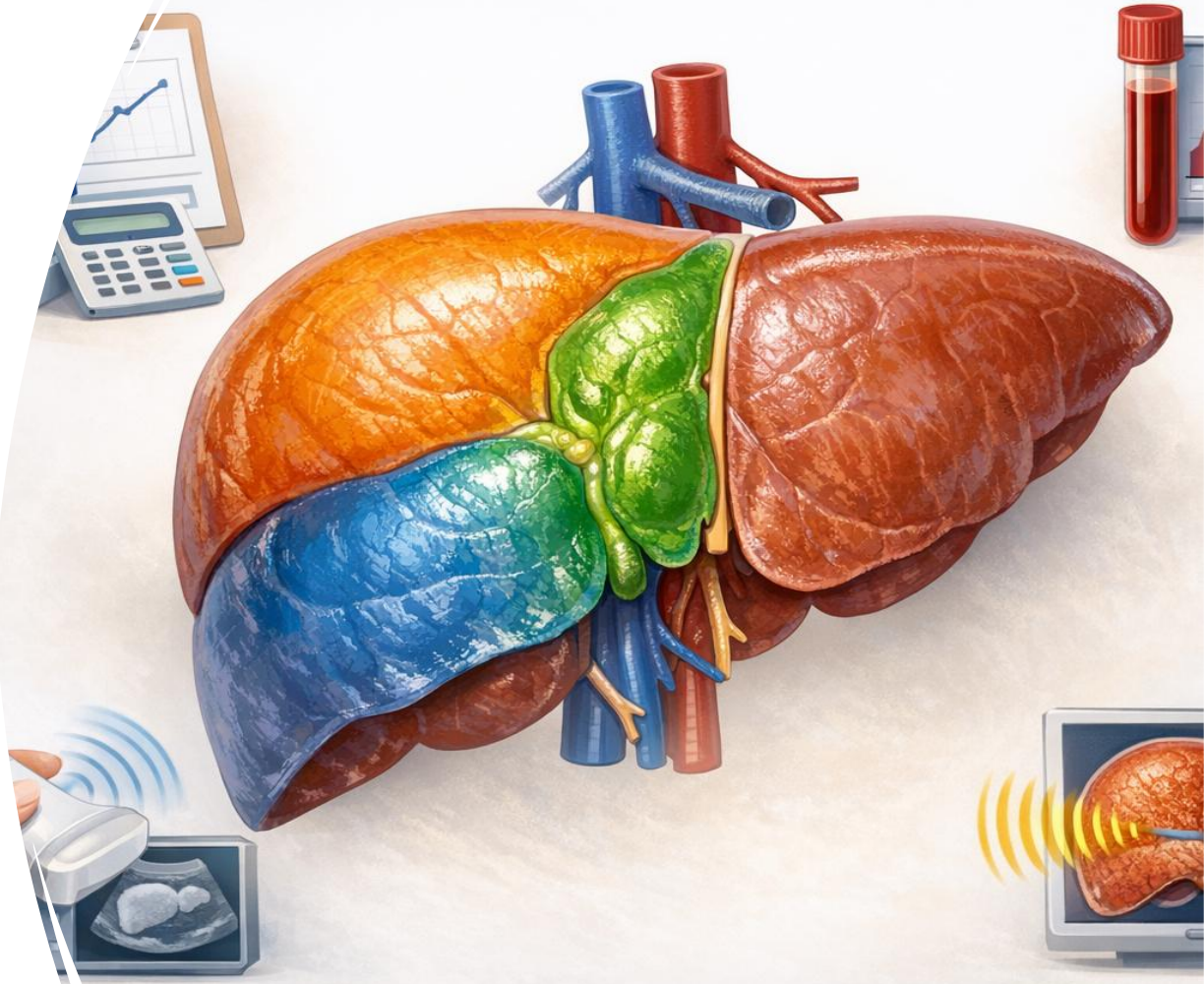


GLP-1 agonists in MASH management

- GLP-1 receptor agonists improve liver enzymes and reduce hepatic fat.
- Aid in weight loss, crucial for MASLD management.
- Studies show reduced liver inflammation and fibrosis markers.
- improve insulin sensitivity and glycemic control.

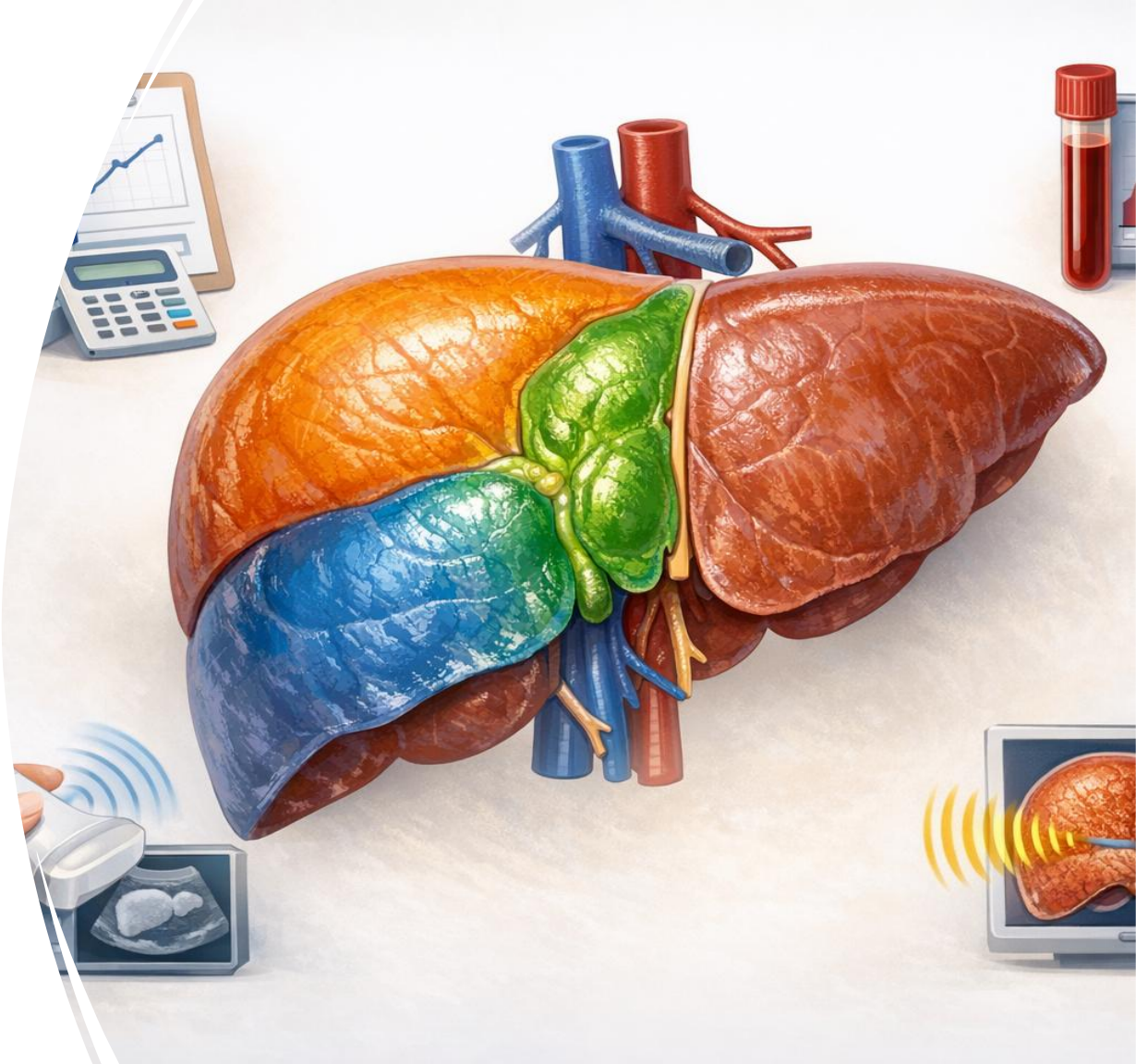
New Therapies and Future Direction

- New Therapies and Future Directions
- Resmetirom(Rezdiffra), a thyroid hormone receptor beta agonist, is the first FDA-approved drug for MASH with moderate to advanced fibrosis (F2-F3)
- improves LDL cholesterol, triglycerides, and lipoprotein(a)
- It is indicated with diet and exercise
- reduced circulating free T4 by about 15–20%, with no effect on serum TSH or free T3



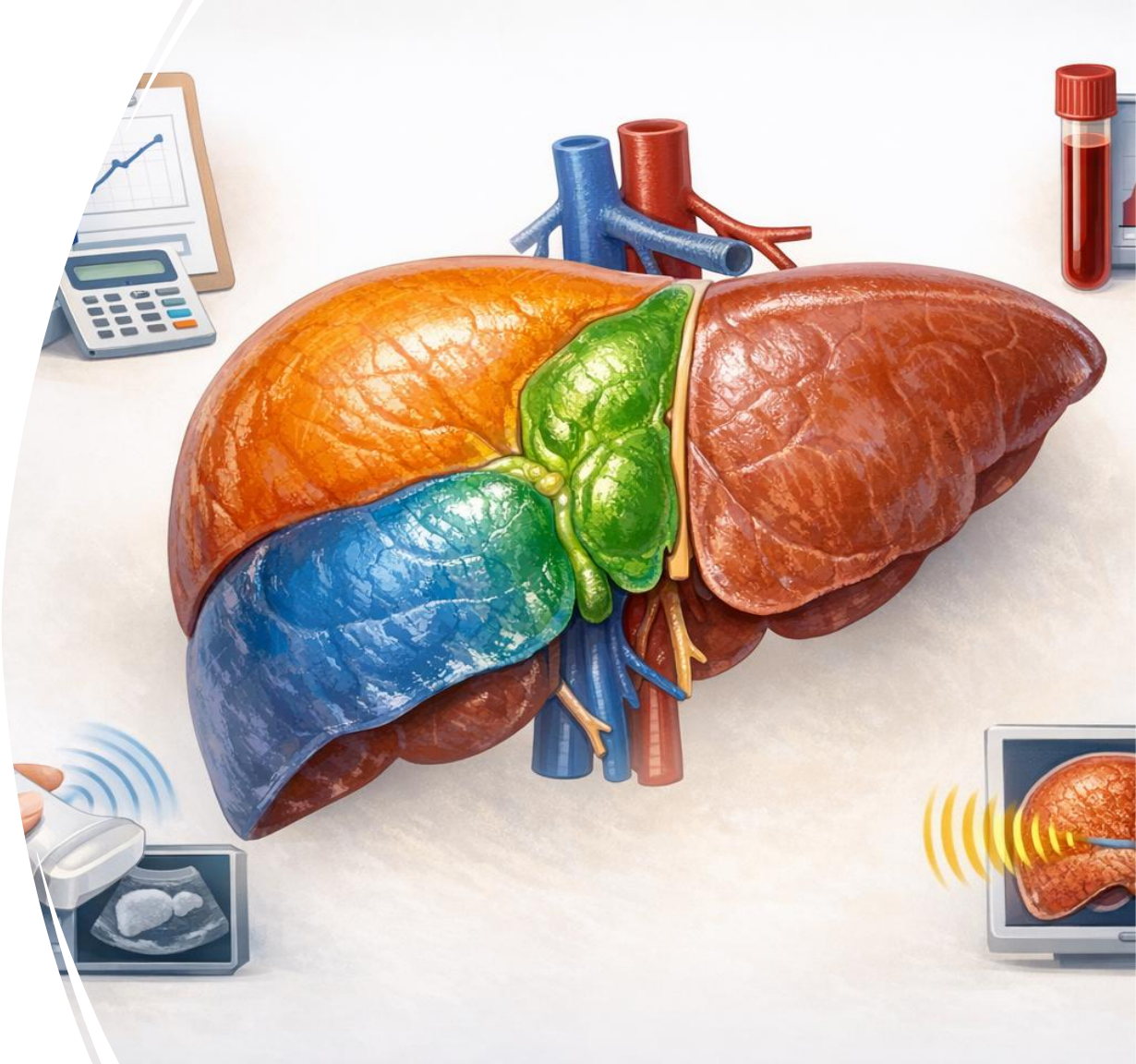
Resmetirom Eligibility

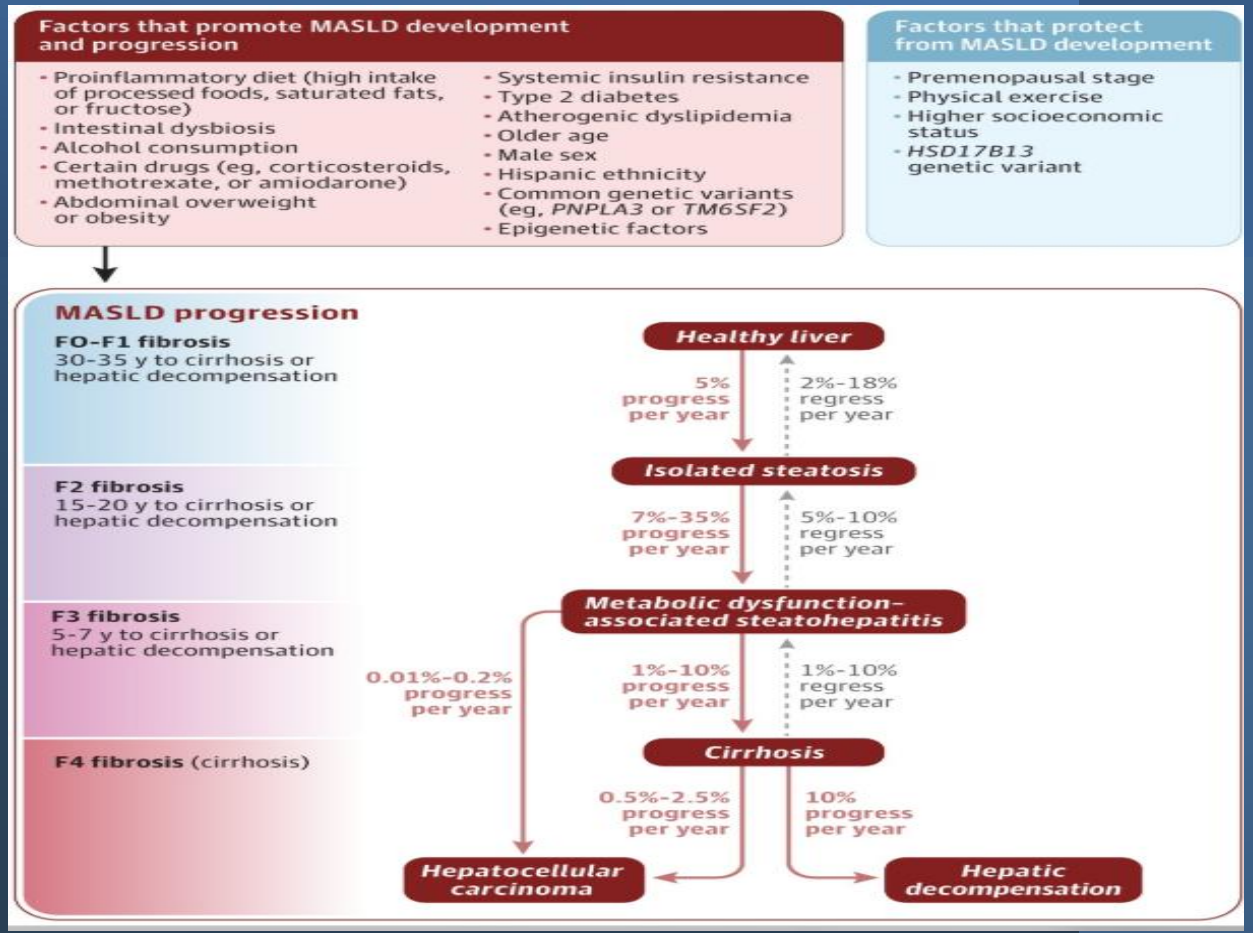
- Adults with MASH with moderate to advanced fibrosis
- Non cirrhotic
- Evidence of MASH by Biopsy or combination of noninvasive test
- Not a substitute for weight loss



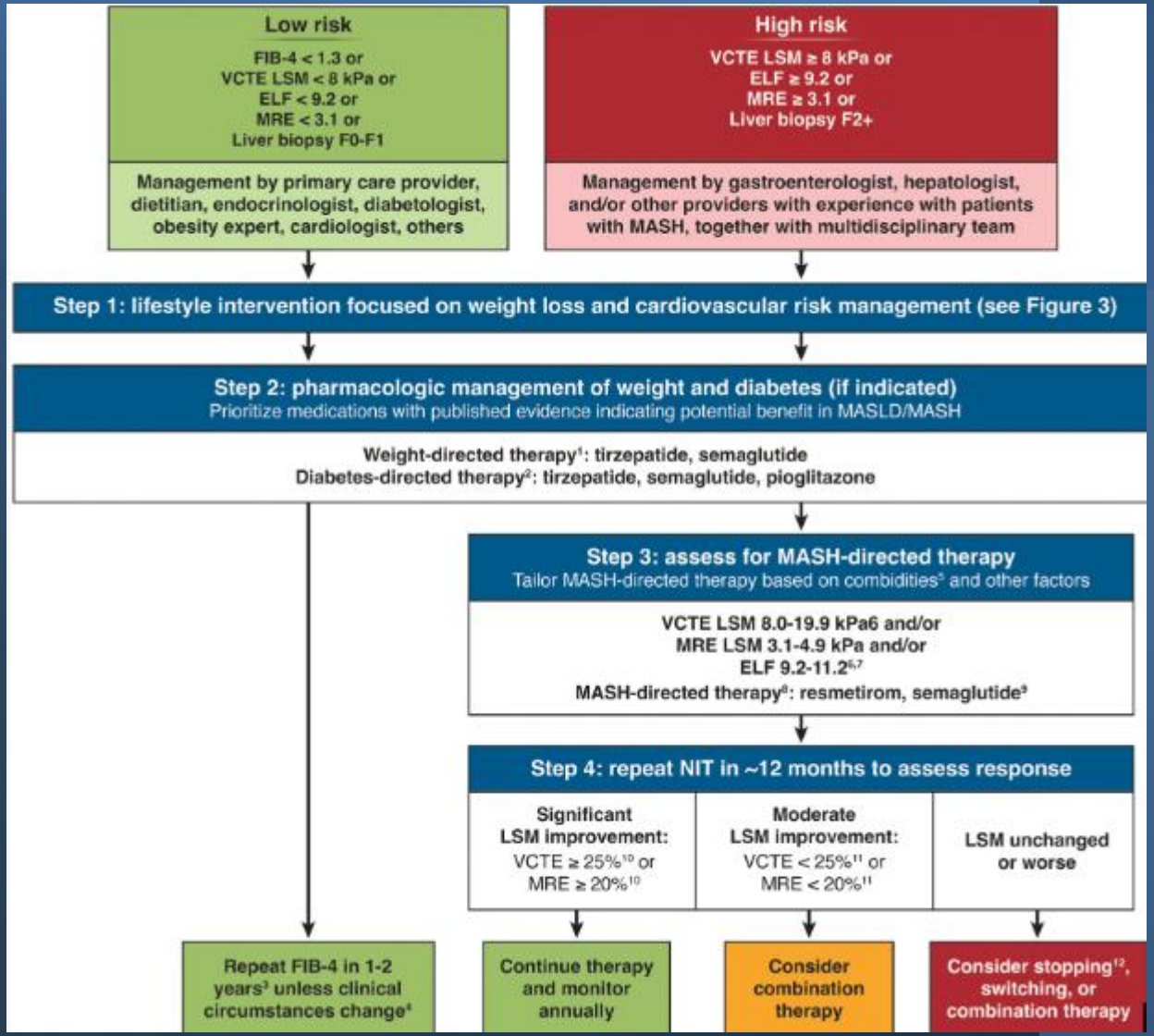
Bariatric Surgery

- Significant weight loss
- Resolution of MASH in 70-85%
- 70 % fewer cardiovascular events over 10 years




Factors that promote MASLD development and progression
- Proinflammatory diet (high intake of processed foods, saturated fats, or fructose)
- Intestinal dysbiosis
- Alcohol consumption
- Certain drugs (eg, corticosteroids, methotrexate, or amiodarone)
- Abdominal overweight or obesity
- Systemic insulin resistance
- Type 2 diabetes
- Atherogenic dyslipidemia
- Older age
- Male sex
- Hispanic ethnicity
- Common genetic variants (eg, PNPLA3 or TM6SF2)
- Epigenetic factors
Factors that protect from MASLD development
- Premenopausal stage
- Physical exercise
- Higher socioeconomic status
- HSD17B13 genetic variant
MASLD progression
F0-F1 fibrosis
30-35 y to cirrhosis or hepatic decompensation
F2 fibrosis
15-20 y to cirrhosis or hepatic decompensation
F3 fibrosis
5-7 y to cirrhosis or hepatic decompensation
F4 fibrosis (cirrhosis)
Healthy liver
5% progress per year
2%-18% regress per year
Isolated steatosis
7%-35% progress per year
5%-10% regress per year
Metabolic dysfunction-associated steatohepatitis
0.01%-0.2% progress per year
1%-10% progress per year
1%-10% regress per year
Cirrhosis
0.5%-2.5% progress per year
10% progress per year
Hepatocellular carcinoma
Hepatic decompensation

AGA Management guidelines



Integrate MASLD/MASH assessment into daily clinical workflows



Identify High-Risk Patients

Screen patients with diabetes for MASLD risk factors such as obesity, insulin resistance, and elevated liver enzymes. Use noninvasive scores to stratify risk.

Implement Diagnostic Plans

Incorporate routine liver function tests and imaging modalities like ultrasound in patient care. Establish criteria for referral to hepatology.

Coordinate Multidisciplinary

Collaborate with hepatologists, endocrinologists, and dietitians to optimize management plans. Regularly review patient progress and adjust treatments as needed.

Coordination of care with hepatology for MASLD patients

1

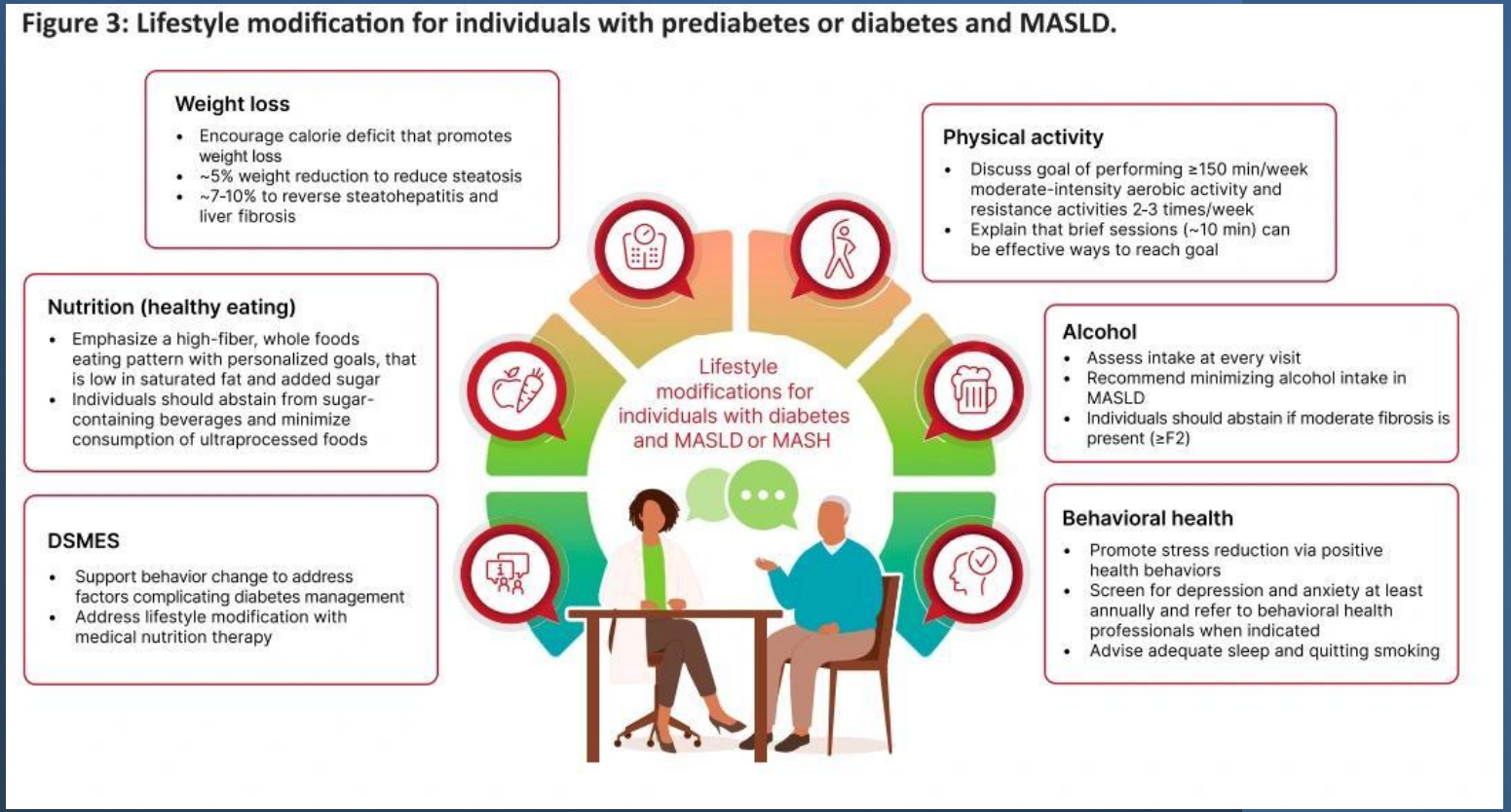
Effective coordination with hepatology is crucial for MASLD/MASH management. It involves timely referrals for patients with advanced fibrosis or cirrhosis, ensuring comprehensive liver assessment and monitoring. Collaborative care improves patient outcomes by integrating hepatology expertise in managing liver-related complications

2

Key steps include establishing clear referral pathways, regular communication between diabetes care teams and hepatologists, and shared decision-making. Multidisciplinary teams enhance treatment plans, addressing both metabolic and hepatic aspects of MASLD. This ensures holistic patient care and optimizes therapeutic strategies

Lifestyle modification for individuals with prediabetes or diabetes and MASLD

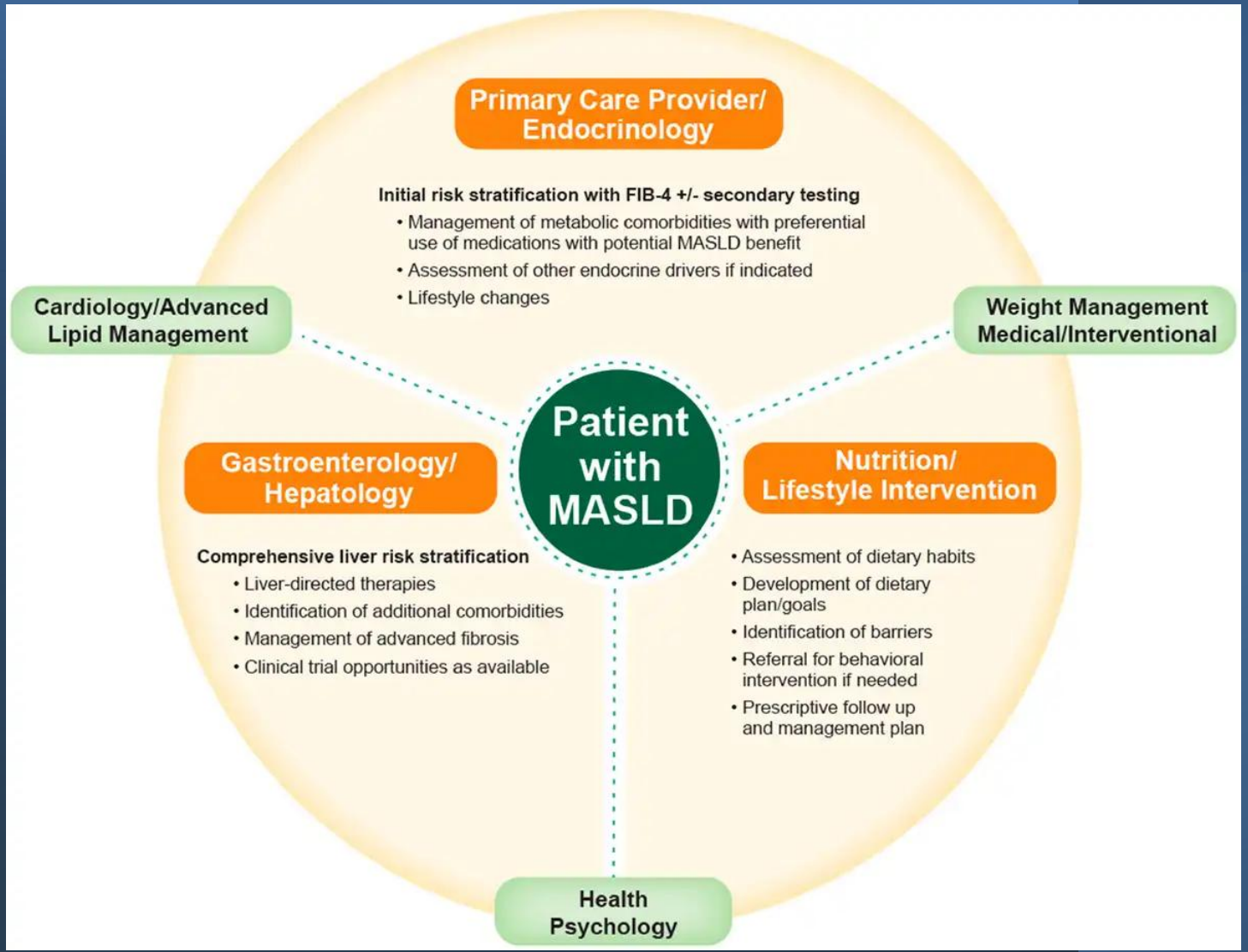
Figure 3: Lifestyle modification for individuals with prediabetes or diabetes and MASLD.



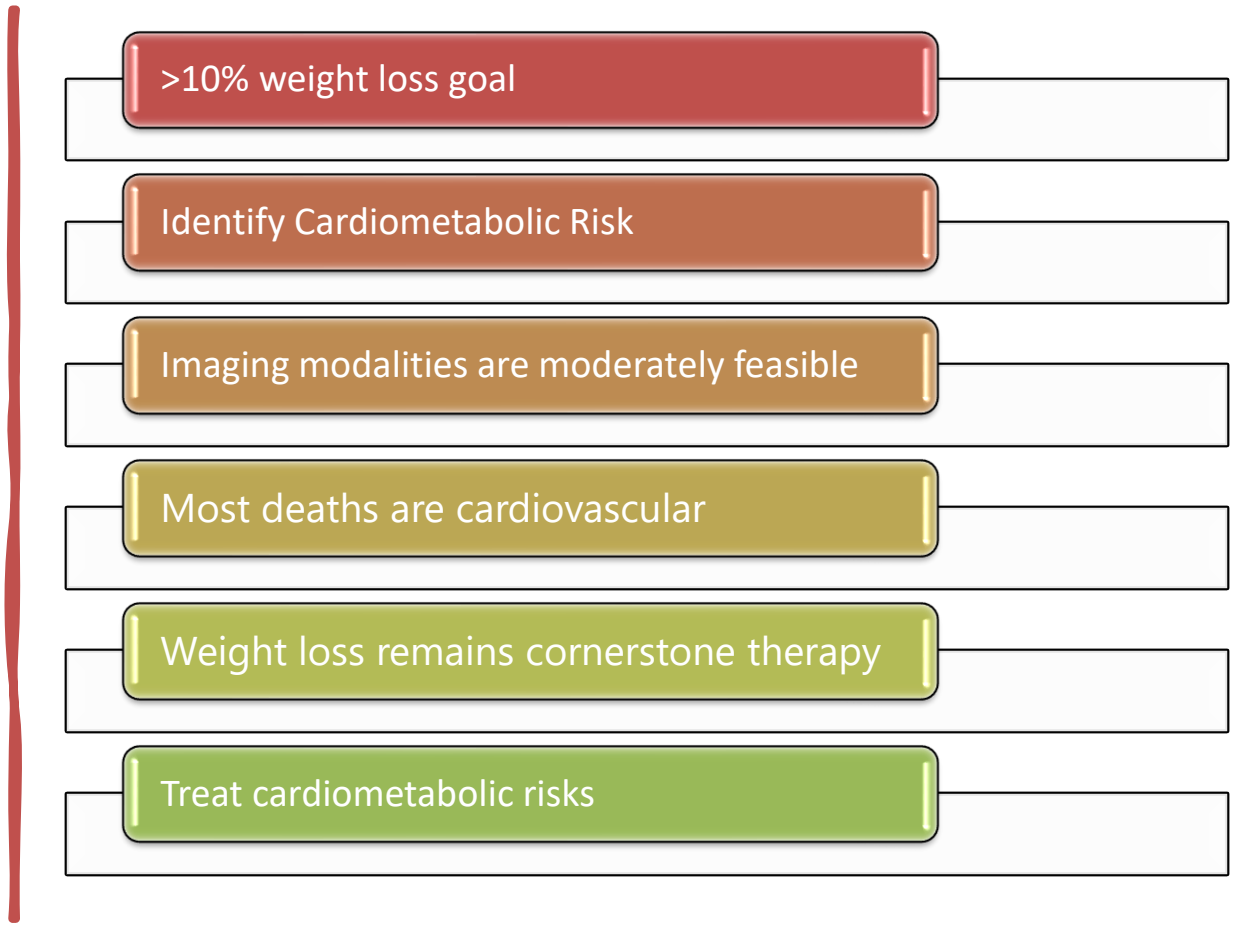
Commonalities and differences among current MASLD/MASH screening pathways

	ADA 2025	AGA 2026	EASL-EASD-EASO 2024	Chinese guideline 2024	AASLD 2023
Target population	T2DM, prediabetes, obesity, or cardiometabolic risk	High-risk metabolic populations, including T2DM and obesity	T2DM, obesity, abnormal liver enzymes, or imaging-detected steatosis	Individuals with MASLD risk factors; emphasis on metabolic comorbidities	Patients with steatosis or metabolic risk factors, especially T2DM
First-line test	FIB-4 Age, AST, ALT, platelet count	FIB-4 Age, AST, ALT, platelet count	FIB-4 Age, AST, ALT, platelet count	FIB-4 Age, AST, ALT, platelet count	FIB-4 Age, AST, ALT, platelet count
Second-line test	VCTE or ELF	VCTE or ELF; MRE in selected cases	VCTE or other validated non-invasive tests	VCTE/FibroScan; serum-based fibrosis tests when appropriate	VCTE, ELF, or MRE in selected cases
Referral criteria	High-risk FIB-4 or abnormal second-line test	FIB-4 >2.67 or elevated VCTE/ELF	Elevated FIB-4 and/or increased liver stiffness	Suspected advanced fibrosis/cirrhosis or diagnostic uncertainty	FIB-4 >2.67 or high-risk secondary assessment
Follow-up interval	Periodic reassessment in T2DM	Low risk: repeat every 1–2 years in high-risk patients	Risk-adapted reassessment	Dynamic risk-based follow-up	Every 1–2 years in T2DM or multiple metabolic risks
Treatment focus	Lifestyle, weight loss, cardiometabolic risk reduction, glucose-lowering agents with metabolic benefit	Lifestyle, weight management, metabolic control, referral for advanced disease	Weight loss, cardiometabolic management, multidisciplinary care	Lifestyle intervention, metabolic risk control, individualized pharmacotherapy	Lifestyle, weight loss, cardiometabolic risk reduction, liver-directed therapy when eligible

FIGURE 2
Commonalities and differences among current MASLD/MASH screening pathways. The image summarizes major recommendations from ADA 2025, AGA 2026, EASL-EASD-EASO 2024, the Chinese 2024 guideline and AASLD 2023 guidance. Across guidance documents, T2DM and metabolic risk factors define high-priority populations, FIB-4 is used as the first-line fibrosis triage tool, and VCTE/FibroScan, ELF or MRE are used for second-line assessment in selected cases. Differences remain in target populations, referral thresholds, reassessment intervals and the emphasis on metabolic, multidisciplinary and liver-directed care.


Primary Care Provider/ Endocrinology
Initial risk stratification with FIB-4 +/- secondary testing
• Management of metabolic comorbidities with preferential use of medications with potential MASLD benefit
• Assessment of other endocrine drivers if indicated
• Lifestyle changes
Cardiology/Advanced Lipid Management
Weight Management Medical/Interventional
Patient with MASLD
Gastroenterology/ Hepatology
Nutrition/ Lifestyle Intervention
Comprehensive liver risk stratification
• Liver-directed therapies
• Identification of additional comorbidities
• Management of advanced fibrosis
• Clinical trial opportunities as available
• Assessment of dietary habits
• Development of dietary plan/goals
• Identification of barriers
• Referral for behavioral intervention if needed
• Prescriptive follow up and management plan
Health Psychology

Take Home Message



Thank you

QUESTIONS