Journal Articles Review

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Rinella ME, Lazarus JV, Ratziu V, et al. A multisociety Delphi consensus statement on new fatty liver disease nomenclature. Hepatology 2023;78(6):1966–1986. DOI: 10.1097/HEP.0000000000000520

Obesity or overweight is associated with liver injury in the form of hepatic steatosis, inflammation, and fibrosis known as nonalcoholic fatty liver disease (NAFLD). The term "nonalcoholic steatohepatitis" was first recognized in 1980 by Jurgen Ludwig. Later the term NAFLD was incorporated to imply the spectrum of hepatic steatosis to steatohepatitis. There was some disapproval of this term owing to the use of the word "fatty" which was considered to be stigmatizing, and to the fact that type 2 diabetics are at risk for NAFLD who consume alcohol more than the level which delineates being nonalcoholic. Also, some biological processes have been unsurfaced that contribute both to NAFLD and alcohol-related liver injuries. In 2020, it led to the proposal to use the term metabolic dysfunction-associated fatty liver disease (MAFLD). To change the nomenclature and definition a modified Delphi process was led by three large pan-national liver associations.

Proposed new nomenclature and definitions:

- Steatotic liver disease (SLD): Liver injury diagnosed histologically or by imaging.
- Metabolic dysfunction-associated steatotic liver disease (MASLD): Presence of hepatic steatosis in conjunction with one cardiometabolic risk factor (CMRF).
- Metabolic steatotic liver disease (MetSLD): No other discernible cause, alcoholic liver disease (ALD), and an overlap of the MASLD. There is the presence of one out of five CMRF and steatosis.
- Metabolic dysfunction-associated steatohepatitis (MASH): MASLD and steatohepatitis.
- Cryptogenic SLD: There is no identifiable cause, no CMRF, or steatosis.
- Other specific etiology SLD: There is no CMRF but steatosis is present, for example, drug-induced liver injury, monogenic, ALD, etc.

Following are the CMRFs in pediatric population:

- Body mass index (BMI) ≥85th percentile for age/sex (BMI *z*-score ≥+1) or waist circumference >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 glycated hemoglobin (HbA1c) ≥5.7% (39 mmol/L) or already diagnosed/treated type 2 diabetes or treatment for type 2 diabetes.
- Blood pressure (BP) age <13 years, BP ≥95th percentile or ≥130/80 mm Hg (whichever is lower): age ≥13 years, 130/85 mm Hg or specific antihypertensive drug treatment.
- Plasma triglycerides age <10 years, ≥ 100 mg/dL; age ≥10 years, ≥150 mg/dL or lipid lowering treatment.
- Plasma high-density lipoprotein (HDL) cholesterol <49 mg/dL or lipid-lowering treatment.

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Garcia-Tsao G, Abraldes JG, Rich NE, et al. AGA clinical practice update on the use of vasoactive drugs and intravenous albumin in cirrhosis: expert review. Gastroenterology 2024;166(1):202–210. DOI: 10.1053/j.gastro.2023.10.016

This expert review on the basis of published literature and expert opinions provides best practice advices on the use of vasoactive drugs and IV albumin in variceal hemorrhage, ascites and spontaneous bacterial peritonitis (SBP), and acute kidney injury (AKI) and hepatorenal syndrome (HRS).

Vasoactive drugs in variceal hemorrhage:

- It should be initiated as soon as the diagnosis of variceal hemorrhage is suspected or confirmed, preferably before diagnostic and/or therapeutic endoscopy.
- It should be continued for 2–5 days to prevent early rebleeding.
- Octreotide is the vasoactive drug of choice.

Intravenous (IV) albumin:

- It should be administered at the time of large-volume (>5 L) paracentesis.
- It may be considered in patients with SBP but not in patients with cirrhosis and uncomplicated ascites.

Acute kidney injury (AKI) and HRS:

- Intravenous (IV) albumin is the volume expander of choice in patients with cirrhosis and ascites presenting with AKI.
- Vasoactive drugs such as terlipressin (drug of choice), norepinephrine, and a combination of octreotide and midodrine should be used in the treatment of HRS-AKI.

- Terlipressin use is contraindicated in patients with hypoxemia and in patients with ongoing coronary, peripheral, or mesenteric ischemia.

Ananthakrishnan AN, Adler J, Chachu KA, et al. AGA clinical practice guideline on the role of biomarkers for the management of Crohn's disease. Gastroenterology 2023;165(6):1367–1399. DOI: 10.1053/j.gastro.2023.09.029

This guideline from the American Gastroenterological Association (AGA) analyses the performance of biomarkers such as fecal calprotectin, serum C-reactive protein (CRP), and Endoscopic Healing Index, for evaluation and monitoring of patients with established Crohn's disease (CD). These recommendations are as follows:

- Patients with CD in symptomatic remission: These patients can be monitored with a combination of biomarkers and symptoms, as symptoms alone may not be reliable. Biomarkers may be evaluated every 6–12 months.
- Patients with CD in symptomatic remission with the recent confirmation of endoscopic remission: Fecal calprotectin ≤150 µg/gm and CRP <5 mg/L can be utilized to rule out active inflammation which may avoid routine endoscopic assessment.
- Patients with CD in symptomatic remission without recent confirmation of endoscopic remission: In such patients, endoscopic evaluation should be done to rule out active inflammation and not rely solely on biomarkers. Radiological assessment may be a reasonable alternative to endoscopy.
- Patients with CD in symptomatic remission: If biomarkers are elevated (fecal calprotectin >150 µg/gm, CRP >5 mg/L), endoscopic assessment of disease activity should be done before treatment adjustment. If the remission is sustained but biomarkers are elevated, repeat measurement of biomarkers after 3–6 months, may be an alternative to endoscopy or radiological assessment.
- Patients with symptomatically active CD: Assessment should be done on the basis of biomarkers and treatment can be adjusted; symptoms alone should not be relied upon. Biomarkers can be assessed every 2–4 months and treatment can be adjusted in these patients. If the symptoms resolve and biomarkers normalize, endoscopic or radiological evaluation should be performed every 6–12 months to rule out active inflammation.
- Patients with CD with mild symptoms and elevated biomarkers of inflammation (fecal calprotectin >150 µg/gm, CRP >5 mg/L): Endoscopic assessment of disease activity should be done rather than empiric treatment adjustment. Persistently elevated biomarkers in patients with partially improved symptoms indicate active inflammation and may require treatment adjustment without endoscopic evaluation.
- Patients with CD with mild symptoms and normal biomarkers of inflammation (fecal calprotectin < 150 µg/gm and CRP <5mg/L): Endoscopic assessment of disease activity should be done before treatment adjustment.
- Patients with CD with moderate to severe symptoms: Fecal calprotectin >150 µg/gm or CRP >5 mg/L, suggest active inflammation which should warrant treatment adjustment and routine endoscopic assessment of disease activity should be avoided.
- Patients with CD with moderate to severe symptoms with normal biomarkers: In these patients, endoscopic assessment should be done before treatment.
- In asymptomatic patients who are in remission after surgery within the past 12 months, and are at low risk of recurrence or who have one or more risk factors for recurrence and are on pharmacological prophylaxis, can have fecal calprotectin assessment to avoid endoscopic assessment.
- In asymptomatic patients who are in remission after surgery within the past 12 months, but are at high risk for recurrence and are not receiving pharmacological prophylaxis, endoscopic evaluation should be done.

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