

Fatty Liver In Ultrasound

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INTRODUCTION

The global obesity epidemic of the present century is increasing the burden of several non-communicable diseases (NCD), including nonalcoholic fatty liver disease (NAFLD), which is now recognized as the most frequent cause of chronic liver disease in children and adolescents.

EPIDEMIOLOGY

It is currently estimated that NAFLD affects ~3–10% of children worldwide. A recent school based cross-sectional study from North India has reported that 22.4% of normal weight and overweight children aged 5 -10 years had fatty liver on ultrasound imaging. Further, a high proportion of normal weight children with fatty liver (18.8%) indicates the silent burden of this NCD among our children which calls for urgent action from a public health perspective.

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PATHOGENESIS OF PEDIATRIC NAFLD

The pathophysiological mechanisms involved in the onset and progression of liver damage in paediatric NAFLD remain unclear. It is conceivable that *genetic predisposition* like PNPLA3 acts in conjunction with *epigenetic factors* in multiple ways to precipitate the development of liver disease. On this background, an unhealthy lifestyle, mainly characterized by high intake of certain fats and/or carbohydrates coupled with sedentary behaviours, seems to be the main trigger of NAFLD in children.

NONALCOHOLIC FATTY LIVER DISEASE DEFINITIONS AND PHENOTYPES

NAFLD - umbrella term referring to the full spectrum of disease. Indicates fatty infiltration of the liver in the absence of significant alcohol, genetic diseases, or medications that cause steatosis. Fatty infiltration is typically defined as fat >5% of the liver by imaging, direct quantification, or histologic estimation. *NAFL* - Steatosis without specific changes to suggest steatohepatitis, with or without fibrosis

Pediatric NASH - Hepatic steatosis with inflammation, with or without ballooning injury to hepatocytes and fibrosis

NAFLD with fibrosis - NAFL or NASH with periportal, portal, or sinusoidal or bridging fibrosis

NAFLD with cirrhosis - Cirrhosis in the setting of NAFLD

When to screen for pediatric NAFLD ?

- Screening should be considered beginning between ages 9 and 11 years for all obese children (BMI more than

or equal to 95th percentile) and for overweight children (BMI more than or equal to 85th and less than 94th percentile) with additional risk factors (central adiposity, insulin resistance, prediabetes or diabetes, dyslipidemia, sleep apnea, or family history of NAFLD/NASH).

- Earlier screening can be considered in younger patients with risk factors such as severe obesity, family history of NAFLD/NASH, or hypopituitarism.
- Consider screening of siblings and parents of children with NAFLD if they have known risk factors for NAFLD

When to consider alternative aetiology for pediatric fatty liver?

- Early onset steatosis (<5 years of age)
- No risk factors for NAFLD
- Clinical phenotypes - acute liver failure, neonatal / infantile conjugated hyperbilirubinemia, organomegaly and psychomotor retardation

How to screen?

Currently, the best screening test for NAFLD in children is alanine amino transferase (ALT) despite its limitations (sensitivity of 88% and a specificity of 26%).

Interpretation of ALT should be based upon sex-specific upper limits of normal in children (22 U/L for girls and 26 U/L for boys) and not individual laboratory upper limits of normal.

Persistently (>3 months) elevated ALT more than twice the upper limit of normal should be evaluated for NAFLD or other causes of chronic hepatitis. ALT of >80 U/L warrants

increased clinical concern and timely evaluation, as the likelihood of significant liver disease is higher.

When evaluating a child suspected to have NAFLD, it is recommended to exclude alternative etiologies for elevated ALT and/or hepatic steatosis and investigate the presence of coexisting chronic liver diseases like chronic viral hepatitis B and C, Wilson disease and pediatric autoimmune liver disease.

ROLE OF IMAGING AS A SCREENING TOOL FOR NAFLD.

Liver ultrasonography is one of the most frequently used imaging modalities that is often used as an initial screening test in those with suspected NAFLD. On these images, hepatic steatosis appears as *diffuse increased liver echogenicity*. Liver ultrasonography is an attractive screening test because of its wide availability, low cost, and absence of radiation. However, ultrasonography is unreliable in detecting mild steatosis (approximately <30% fat infiltration); normal findings do not definitively rule out NAFLD. Overall the sensitivity of ultrasound in NAFLD ranges from 60% to 94%, with specificity from 84% to 100%.

LIVER BIOPSY

Liver biopsy remains a key method for the diagnosis of NAFLD and for grading and staging disease severity. The four key components of liver histology involved in the diagnosis and staging of NAFLD are steatosis, hepatocyte ballooning, inflammation and fibrosis.

Liver biopsy should be considered for the assessment of NAFLD in children who have increased risk of NASH and/or advanced fibrosis.

IMAGING TO ASSESS FIBROSIS

Transient elastography (TE) is a non-invasive ultrasound-based method that uses shear wave velocity to measure liver stiffness. TE has also been validated as a method to predict the presence of moderate-to-severe fibrosis in paediatric NAFLD.

TREATMENT OPTIONS IN PEDIATRIC NAFLD

(a). *Non pharmacologic therapy*

Lifestyle modifications to improve diet and increase physical activity are recommended as the first-line treatment for all children with NAFLD. Avoidance of sugar-sweetened beverages is recommended as a strategy to decrease adiposity. Increasing moderate- to high-intensity physical

activity and limiting screen time activities to <2 hours per day is recommended for all children including those with NAFLD.

Weight loss is the only proven treatment for pediatric NAFLD. Multiple studies have shown that weight loss can normalize ALT and that substantial long-term benefit may be seen with as little as a 5% to 10% reduction in body weight. Weight loss, however, should be gradual because too rapid of a loss may acutely worsen liver disease.

(b.) *Pharmacologic therapy*

The 2018 American Association for the Study of Liver Diseases (AASLD) practice guidance supports the use of *vitamin E* (400 IU twice a day of the natural form) but not metformin in children with biopsy-proven NASH. This dose and frequency has been shown to improve hepatic histology (ballooning) and have greater resolution of NASH.

(c.) *Bariatric surgery*

It may be considered for selected adolescents with BMI more than or equal to 35 kg/m², who have noncirrhotic NAFLD and other serious comorbidities (eg, T2DM, severe sleep apnea, idiopathic intracranial hypertension) that are likely to improve with weight loss surgery.

(d.) *Screening for co-morbidities*

Children with NAFLD should be screened for *dyslipidemia* at diagnosis and periodically as indicated by current lipid guidelines for children. It is recommended to *monitor blood pressure* in children with NAFLD and to screen children with NAFLD for *diabetes* at diagnosis and annually (or sooner if clinical suspicion arises) using either a fasting serum glucose level or an HbA1c level.

(e.) *Vaccination*

Children with NAFLD should be vaccinated routinely against hepatitis A and should have prior receipt of hepatitis B vaccine be verified and be immunized if no prior vaccination was received.

CONCLUSION

With the increasing burden of obesity, escalation of the incidence of NAFLD seems to be alarming in children. Multidisciplinary approach to care in children with NAFLD/NASH should include specific support involving nutritional services for successful weight loss and improvement in hepatic steatosis. As paediatricians, we have a unique opportunity and responsibility to help identify the children most at risk from the worst manifestations of this epidemic and to provide interventions at an early age to help prevent serious life-altering consequences from chronic liver disease in adulthood.