JOURNAL WATCH

1. NORTH AMERICAN SOCIETY FOR PEDIATRIC GASTROENTEROLOGY, HEPATOLOGY, AND NUTRITION (NASPGHAN) POSITION PAPER ON THE DIAGNOSIS AND MANAGEMENT OF PEDIATRIC ACUTE LIVER FAILURE

Squires JE, Alonso EM et al. North American Society for Pediatric Gastroenterology, Hepatology, and Nutrition Position Paper on the Diagnosis and Management of Pediatric Acute Liver Failure. J Pediatr Gastroenterol Nutr. 2021 Aug 3.doi: 10.1097/MPG.0000000000003268.

Pediatric Acute Liver Failure Study Group (PALFSG) defines Pediatric Acute Liver Failure (PALF) as acute onset of liver disease without evidence of chronic liver disease and biochemical evidence of severe liver injury along with coagulopathy not corrected by vitamin K, where Prothrombin time (PT) ≥ 15 seconds or International Normalized Ratio (INR) ≥ 1.5 with evidence of hepatic encephalopathy, or PT ≥ 20 seconds or INR > 2 with or without encephalopathy. The incidence is around 500-600 cases per year in USA and may be more in the developing nations which can be attributed to the high incidence of infective hepatitis. The clinical presentation of PALF depends on the age and underlying etiology. In infective cases prodromal symptoms as fever, fatigue, malaise, nausea or abdominal pain can be reported before the onset of jaundice. Early detection of hepatic encephalopathy (HE) should be done by identifying change in behaviour or attention. History should include questions pertaining to etiology as infections, exposure to toxins as over the counter medications or herbal preparations, family history of autoimmune disease or pre-existing metabolic disease. A detailed physical examination should be done to detect any underlying etiology and hepatic encephalopathy. The natural course of PAFL is very dynamic and dubious. So, establishing a diagnosis for timely intervention is a must. Specific laboratory investigations should be done to assess inflammation and function of the liver. Also, investigations to identify etiology, systemic comorbidities and complications should be sought. In absence of cardiovascular instability 90% of maintenance fluid should be administered using hypertonic glucose (D10) one-half normal saline supplemented with 15mEq of potassium /L. Lactated Ringer should be avoided. Ammonia levels of >75-100 μmol/L on admission represent an independent risk factor for the development of intracranial hypertension and high-grade HE, which can be reduced using lactulose or Rifaximin. A level of ammonia > 200 μmol/L is a known risk factor for mortality. Coagulopathy can be corrected using subcutaneous vitamin K. Correction of coagulopathy with fresh frozen plasma or pro-coagulation products should be avoided. Apart from this, acute kidney injury, infection, cardiovascular failure and nutrition should be given due attention. Various liver support therapies as, albumin dialysis, plasma exchange, bioartificial liver support systems, extracorporeal liver assist device and molecular absorbent recirculating system have failed to show any major impact on the outcome and are not routinely recommended. Prompt liver transplantation when indicated can be lifesaving therapeutic option.

2. EASL CLINICAL PRACTICE GUIDELINES ON NON-INVASIVE TESTS FOR EVALUATION OF LIVER DISEASE SEVERITY AND PROGNOSIS – 2021 UPDATE.

European Association for the Study of the Liver. EASL Clinical Practice Guidelines on non-invasive tests for evaluation of liver disease severity and prognosis – 2021 update. Journal of Hepatology. Volume 75, Issue 3, September 2021, Pages 659-689. Doi: https://doi.org/10.1016/j.jhep.2021.05.025

Compensated advanced chronic liver disease(cACLD) is a term used in those asymptomatic patients with chronic liver disease who are at risk of developing clinically significant portal hypertension, severe fibrosis and cirrhosis. EASL recommends the use of non-invasive tests (NITs) for monitoring of liver fibrosis and its complications. Liver fibrosis can be measured using transient elastography [TE]; ultrasound devices based: point-shear wave elastography (pSWE), bidimensional shear wave elastography (2D-SWE); and Magnetic Resonance Elastography (MRE). Out of these TE is the most widely validated and available. Main NITs are as follows:

1) Serum Markers: blood tests as aspartate aminotransferase (AST), together with alanine aminotransferase (ALT) and platelet count can be utilised. These tests are cheap, easily available and has high applicability. Though they are non-liver specific and performance is not equivalent to TE, they are well validated and are useful in some chronic liver disease.
2) TE: It is widely available and well validated in compensated cirrhosis. This can be done bedside and has good reproducibility. False positives can be seen in acute hepatitis, Liver congestion, extra hepatic cholestasis etc.
3) pSWE: It is done along with regular ultrasound and has higher applicability than TE. It can give false positive results as in TE but has good prognostic value in cirrhosis.
4) MRE: It has high performance for cirrhosis and and higher applicability than TE. But is costly and does not have any prognostic value.

Following conditions in pediatric population can be monitored using NITs:

1) NASH/NAFLD: Conventional ultrasound is used as the first line investigation for screening of steatosis. TE can be used to diagnose advanced fibrosis.Liver biopsy remains the reference standard for diagnosis.
2) Cholestatic and autoimmune liver disease: For primary biliary cirrhosis (PBC) TE is the best

surrogate marker for ruling in severe fibrosis/cACLD. For primary biliary cholangitis serum markers along with TE is recommended for baseline screening for fibrosis.

3. GUT MICROBIOME IN ACUTE PANCREATITIS: A REVIEW BASED ON CURRENT LITERATURE

Patel BK, Patel KH, Bhatia M, Iyer SG, Madhavan K, Moochhala SM. Gut microbiome in acute pancreatitis: A review based on current literature. World J Gastroenterol 2021; 27(30): 5019-5036.DOI: 10.3748/wjg.v27.i30.5019

Alteration in gut microbiota has been implicated in many diseases, not only pertaining to gastrointestinal system but also other systems as well. A healthy pancreas does not harbour microbes, translocation of bacteria due to loss of gut barrier function is now said to be the cause for acute pancreatitis (AP) related infection. This is a review which summarises the human and animal studies where gut microbiome has been associated with pathogenesis of AP, and the treatment which would target the gut microbiome. It has been shown the hypovolemia and microcirculation due to AP leads to loss of gut barrier functions leading to translocation of gut bacteria. This results in pancreatic and systemic infection. Along with bacteria, the translocation of inflammatory compounds and gut's toxic products also contribute to SIRS and pancreatic injury. It has been shown that in AP the population of beneficial bacteria of Bacteroides phylum decrease and that of pro-inflammatory bacteria Proteobacteria increase. Also, there is an increase in opportunistic Escherichia and Shigella sp. amongst other. It has been observed that the extent of dysbiosis predicts the severity and complications of pancreatitis. Also, restoration of the gut microbiota leads to decrease in the inflammatory compounds as cytokines and tumour necrosis factors. The role of administration of Lactobacillus and Bifidobacterium sp. in animal models seems promising. Avoidance of routine antibiotic use in AP and early enteral nutrition may help attain the homeostasis earlier.

4. PERCUTANEOUS ENDOSCOPIC GASTROSTOMY IN CHILDREN: AN UPDATE TO THE ESPGHAN POSITION PAPER

Homan M, Hauser B, Romano C et al.Percutaneous Endoscopic Gastrostomy in Children: An Update to the ESPGHAN Position Paper. J Pediatr Gastroenterol Nutr. 2021 Sep 1;73(3):415-426. doi: 10.1097/MPG.0000000000003207. PMID: 34155150.

Percutaneous Endoscopic Gastrostomy (PEG) in Children is a safe and effective intervention for nutrition where oral intake is not feasible. The indications for PEG are: neurological impairment where oral intake may be unsafe with respect to aspiration, inadequate or time consuming; conditions as cystic fibrosis, metabolic disease or inflammatory bowel disease where increased energy requirement is the need; conditions that hamper adequate oral intake as cardiomyopathy with dyspnoea, oral malformations, food aversions or eating disorders; metabolic disorders where nocturnal feeding is required; condition where naso-gastric/jejunal feed is required more than -36 weeks or unsafe as long gap esophageal atresia or severe esophageal strictures etc. The absolute contraindications for PEG include: active gastritis or peptic ulcer; coagulation disorder; previous abdominal surgery; gastric varices; ascites; spinal deformity; hiatus hernia etc. A multidisciplinary team approach should be there for the decision of putting PEG and for further management. Antibiotics should be administered prior to the procedure to prevent PEG site infection.

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