

32nd



INTERNATIONAL CONGRESS OF PEDIATRIC HEPATOLOGY GASTROENTEROLOGY & NUTRITION



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ABSTRACTS

16-19

SEPTEMBER 2026

Hurghada

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26 Tarablous street, Abbas El Akkad, Nasr City, Cairo, Egypt.

32nd
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Abstracts Day 1



32nd INTERNATIONAL CONGRESS OF PEDIATRIC HEPATOLOGY GASTROENTEROLOGY & NUTRITION

Professor George Yanni

Title: Hepatic Manifestations of Inborn Errors of Metabolism

Inborn errors of metabolism comprise a diverse group of inherited monogenic genetic disorders caused by pathogenic variants that impair enzymes, transporters, or cofactors required for normal metabolic pathways, leading to either toxic accumulation of upstream substrates or deficiency of essential downstream products. Since the human liver acts as the central processing hub and “warehouse manager” for nearly all digested nutrients, so determining how energy is distributed, stored or converted to sustain life. Any disruption result in toxic substrate accumulation, product deficiency, or impaired energy production, thereby affecting carbohydrate, amino acid, fatty acid, or complex-molecule metabolism. Clinical presentation varies widely, ranging from neonatal, sepsis-like metabolic crises after a symptom-free interval to later-onset, intermittent manifestations with vomiting, altered mental status, seizures, myopathy, cardiomyopathy, or progressive neurodegeneration.

Inborn errors of metabolism (IEMs) comprise a highly significant portion of pediatric liver disease. They account for up to 30 % of acute liver failure cases in children, 30% of chronic pediatric liver disease (excluding biliary atresia and autoimmune disorders), and roughly 10% to 20% of all pediatric liver transplants. The liver is the primary metabolic hub of the body, making it uniquely vulnerable to the accumulation of toxic metabolites or energy deficits caused by enzyme deficiencies. Hepatic presentations of IEMs vary widely in severity and age of onset.

Early recognition remains central to preventing irreversible morbidity and mortality, particularly during catabolic stress. Initial evaluation in acute settings prioritizes rapid identification of metabolic derangements using critical blood and urine samples, with subsequent biochemical and genetic testing to confirm diagnosis and refine long-term management.



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Background: Liver transplantation is a lifesaving, standard-of-care intervention for infants, children, and adolescents with irreversible liver disease, inborn errors of metabolism, and liver tumors. Advances in surgical techniques, organ allocation, and immunosuppressive protocols have improved 5-year patient survival to approximately 86%, with 97% survival in those who survive the first postoperative year.

Indications: The most common indication for pediatric liver transplantation is biliary atresia, accounting for 32–50% of cases. Metabolic and genetic conditions represent the second most common category (18–22%), including alpha-1-antitrypsin deficiency, urea cycle defects, Wilson disease, tyrosinemia, maple syrup urine disease, primary hyperoxaluria, Crigler-Najjar syndrome, and glycogen storage diseases. Acute liver failure accounts for approximately 11% of pediatric transplants and may require emergent listing. Primary hepatic malignancies, principally hepatoblastoma and hepatocellular carcinoma, comprise approximately 9% of cases; transplantation is indicated when complete surgical resection is not feasible. Additional indications include other cholestatic diseases (Alagille syndrome, progressive familial intrahepatic cholestasis, sclerosing cholangitis), autoimmune hepatitis with cirrhosis, cystic fibrosis-related liver disease, and graft failure requiring retransplantation. Clinical triggers for referral include hepatic decompensation (ascites, variceal hemorrhage, hepatic encephalopathy, coagulopathy), failure to thrive, growth failure, and a Pediatric End-Stage Liver Disease (PELD) score ≥ 12 .

Contraindications: Absolute contraindications include generalized extrahepatic malignancy, uncontrolled systemic infection (particularly fungal or viral), severe multisystem mitochondrial disease, Niemann-Pick disease type C, severe porto-pulmonary hypertension refractory to medical therapy, and evidence of brain herniation or brain death. Relative contraindications include hepatocellular carcinoma with venous invasion or rapid progression despite chemotherapy, hemophagocytic lymphohistiocytosis, high certainty of medication nonadherence despite multidisciplinary interventions, and critical psychosocial circumstances not amenable to intervention.

Conclusion: Careful candidate selection based on established indications and recognition of absolute and relative contraindications is essential to optimize outcomes in pediatric liver transplantation. Current guidelines from the AASLD, AST, and NASPGHAN (updated 2026) provide a comprehensive, evidence-based framework for the evaluation of transplant candidacy in infants, children, and adolescents.



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Professor Genie Beasley, MD

Associate Professor and Fellowship Program Director
Pediatric Gastroenterology, Hepatology and Nutrition, University of Florida

The Cholestatic Infant: What Has Changed in 2026?

Neonatal cholestasis is an important clinical finding that requires timely evaluation to identify conditions in which early diagnosis and treatment can significantly alter outcomes. Biliary atresia (BA) remains the most time-sensitive diagnosis, yet infants are frequently referred for evaluation and undergo Kasai portoenterostomy later than the optimal window. At the same time, advances in genetic testing have substantially expanded our ability to identify the underlying causes of cholestatic liver disease.

This presentation will review contemporary approaches to the evaluation of the cholestatic infant, with an emphasis on recent recommendations and areas of evolving practice. Initial recognition and interpretation of conjugated hyperbilirubinemia will be discussed, including the potential role of universal versus targeted bilirubin screening.

The presentation will then address the diagnostic evaluation of the infant with established cholestasis and consider how diagnostic algorithms can be adapted to available resources and expertise. Although diagnostic strategies may differ across healthcare systems, the overarching goal remains the same: to minimize diagnostic delay, rapidly identify infants with biliary atresia and other treatable conditions, and establish an etiologic diagnosis as efficiently as possible.

Ultimately, the challenge is not simply to recognize that an infant has cholestasis, but to identify its cause quickly enough to change the course of disease. The presentation will highlight practical considerations for clinicians caring for cholestatic infants in diverse healthcare settings.



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Professor Mohsen Elalfy

Pediatric Hem/Onc/BMT Ain Shams University/ESPHO Presiden

Standardization of outcome criteria in immune thrombocytopenia (ITP) in Children

Seventeen years following publication of the first International Working Group (IWG) consensus report on the standardization of terminology, definitions, and outcome criteria in immune thrombocytopenia (ITP). Given the emergence of multiple new classes of medications both licensed and in development as well as advances in the understanding of ITP pathophysiology and quality of life impact, the second IWG systematically evaluated all sections of the first IWG report and created this updated and more comprehensive second IWG report. This report provides the fit-for-purpose, updated terminology and definitions essential for proper design and interpretation of both prospective clinical trials and observational clinical studies in ITP. It also refines the basic classifications of primary versus secondary ITP, phases of disease, goals of treatment and response assessment. Several new definitions including durability of response, loss of response, exacerbation, remission, refractoriness to treatment and criteria for bleeding classification and patient reported outcomes are included. Like the first IWG report, the goal of this comprehensive update is for widespread adoption of the terminology, definitions, and outcome criteria in ITP clinical studies and amongst clinicians, investigators, and regulatory agencies worldwide, as well as in the harmonization of future ITP clinical practice guidelines. However it might take years to be applied in Egypt after being published internationally.



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Professor Omar Elalfy

Associate Professor Badr University & Researcher in NRC

Iron Metablism and therapy

Iron is an essential micronutrient involved in oxygen transport, energy metabolism, DNA synthesis, cell proliferation, and numerous enzymatic processes. Maintaining iron homeostasis is critical because iron deficiency impairs essential biological functions, while excess iron can be toxic. This presentation provides a practical overview of iron metabolism, focusing on intestinal absorption, systemic transport, recycling, and the central regulatory role of hepcidin. It highlights factors that enhance or inhibit dietary iron absorption and identifies pediatric groups at increased risk of iron deficiency. The presentation also discusses practical approaches to iron therapy and strategies to improve treatment outcomes. Finally, clinical cases illustrate the importance of recognizing alternative causes of hypochromic microcytic anemia, particularly-thalassemia trait and iron-refractory iron deficiency anemia (IRIDA), especially in children with poor or absent response to oral iron therapy.



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Professor Amira Abdelfatah

Nutrition during early childhood plays a critical role in shaping long-term growth, cognitive development, and overall health. The World Health Organization recommends exclusive breastfeeding during the first six months of life, and the gradual introduction of complementary foods while continuing breastfeeding from six months onward. Introducing complementary foods alongside breast milk helps meet the nutritional requirements of infants aged six months and older.

Appropriate complementary feeding practices can contribute significantly to preventing malnutrition, which remains a major public health problem in many low- and middle-income countries. Complementary feeding should be timely, adequate, safe, and appropriate, and should include a variety of food groups to ensure a balanced diet. Inadequate or inappropriate complementary feeding practices can negatively affect children's health during early childhood and may lead to malnutrition and its associated health complications.

Mothers' knowledge of complementary feeding and feeding practices is considered one of the key factors in improving child nutrition. This includes understanding the appropriate age for introducing different foods, meal frequency, and food diversity, in addition to the role of nutrition education in preventing malnutrition and promoting healthy growth. The aim of the present study was to assess the effectiveness of an educational intervention program on mothers' knowledge and practices regarding complementary feeding



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Prof. Ahmed El Hatw

Third-year Pediatric Resident at Rutgers Monmouth Medical Center, Long Branch, New Jersey

Authors: Ahmed Elhatw, Jonathan E. Teitelbaum

Department of Pediatrics, Monmouth Medical Center, New Jersey, USA

Title: Limiting Computerized Tomography in Ruling Out Appendicitis in the Pediatric Emergency Department Using Quality Improvement Methodology

Background:

Acute appendicitis is one of the most common causes of an acute abdomen among pediatric patients. The diagnosis of appendicitis is challenging due to the nonspecific presentation. Diagnosis is based on historical, physical, and serologic information as well as right lower quadrant ultrasound (RLQ US). In equivocal cases, or those with a high degree of suspicion, computed tomography (CT) of the abdomen and pelvis with intravenous (IV) contrast can be utilized to rule in appendicitis. However, optimizing diagnostic protocols to minimize ionizing radiation exposure while maintaining diagnostic accuracy is important.

Methods:

We performed a monthly retrospective analysis of CT usage among pediatric patients with suspected acute appendicitis presenting to the Pediatric Emergency Department (ED) from June 2023 to December 2023. We used quality improvement methodology to decrease CT use with monthly plan-do-study-act (PDSA) cycles with an aim to decrease CT use by 50%. The main intervention was coordination between the ED and surgical providers to require surgical consult before ordering a CT. We quantified the number of patients who received a surgical consult before CT, the number of RLQ US performed, and the number of appendectomies, specifically the number with perforation.

Results:

A total of 249 patients under 18 presented to the Pediatric ED with symptoms of acute appendicitis during the study period. The number of CTs performed decreased from a baseline of 14 in June to a nadir of four in September, representing a 71% decrease. There was a corresponding decrease in the percentage of patients receiving a CT after a RLQ US from 36.5% in June to 11.8% in September. There was an increase in the surgical consults rate from a baseline of 50% to 87%. There was no increase in appendiceal perforation.

Conclusion:

Multidisciplinary discussions between Pediatric ED physicians and Pediatric Surgeons reduced CT usage, and corresponding radiation exposure, in the evaluation of appendicitis.



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Prof. Ahmed El Hatw

Third-year Pediatric Resident at Rutgers Monmouth Medical Center, Long Branch, New Jersey

Authors: Ahmed Elhatw, MD, Ashwin Agrawal, DO, MA, MBA

Jonathan Teitelbaum, MD

Title: Pediatric gastroenterologists practice styles and adherence to international guidelines for pediatric celiac disease.

Introduction:

Celiac disease (CeD) is an autoimmune disease that affects 1-2% of the population. Given the high incidence and vague symptoms of pediatric CeD, there has been a concerted effort by organizations to create consensus and evidence-based guidelines for the diagnosis and management of pediatric CeD. In 2016, NASPGHAN published a clinical report on the diagnosis and treatment of CeD. In 2020, ESPGHAN published its update to its 2012 CeD guideline.

To date, there has been no study assessing pediatric gastroenterologists' adherence with national/international guidelines. This study aims to assess pediatric gastroenterologists' adherence to NASPGHAN/ESPGHAN guidelines to diagnose CeD, as well as elucidate clinical practices about diagnosis and treatment of CeD.

Methods:

An anonymous survey with 26 questions was designed to assess practice areas of CeD care and compare responses to guidelines by NASPGHAN/ESPGHAN. The survey instrument was designed in Rutgers University REDCap. This study was approved by Monmouth Medical Center's Institutional Review Board.

The survey instrument was distributed via the listserve for pediatric gastroenterology providers maintained by the University of Vermont (pedgi@list.uvm.edu). The survey was open for responses for 4 weeks between January 14, 2025 and February 11, 2025.

Results:

203 respondents completed the survey. Most responses were from the United States of America (USA) (72.9%); however, there were respondents from 27 unique countries. 78% of respondents were attending physicians, 9.9% were advanced practice providers, and 11.3% were fellows/trainees. 36.5% had more than 15 years of practice while 25.1% had 1-4 years of experience. 81.8% worked at tertiary or quaternary care centers.

Selected results of the survey are presented in Table 1. Almost all clinicians biopsied the second portion of the duodenum (99.0%) and the duodenal bulb (98.5%). When taking biopsies of the distal duodenum 93.6% took 3 or more biopsies. In the proximal duodenum, 72.4% of clinicians took 2 biopsies, 15.3% took 4 biopsies, 6.9% took 3 biopsies, 3.9% took one biopsy, and 1.5% took more than 4 biopsies.



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For a gluten challenge prior to testing for CeD, NASPGHAN recommends 3-6 grams of gluten, while ESPGHAN recommends 15 grams or more gluten. 47% of respondents recommended 15 or more grams daily, 36% recommended 10-14 grams, 31% recommended 5-9 grams, and 9.9% recommended 0-4 grams.

Pediatric gastroenterologists were asked if they use the NASPGHAN 2016, ESPGHAN 2020, no guideline, or other guideline for the diagnosis for pediatric celiac disease. 43.3% of respondents utilized the NASPGHAN 2016 guidelines while 47.3% of respondents utilized the ESPGHAN 2020 guidelines. 6.9% of respondents stated they do not follow either of the guidelines. Subgroup of respondents in the USA showed 54.7% used the NASPGHAN 2016 guidelines, 33.1% utilized the ESPGHAN 2020 guidelines, 9.5% did not use either guideline and 2.7% used a combination. For pediatric gastroenterologists practicing outside of the USA, 12.7% utilized NASPGHAN 2016 guidelines, 85.5% utilized ESPGHAN 2020 guidelines.

Discussion:

Our study highlights the adherence to guidelines and practices among pediatric gastroenterologists in managing celiac disease. There was good adherence to the use of TTG IgA testing for screening of CeD, however, disagreement in what age is considered a young child for additional testing. There was a diversity of opinions of what is considered IgA deficiency.

The use of non-biopsy diagnosis is endorsed by ESPGHAN in certain scenarios and generally utilized by non-USA pediatric gastroenterologists. In the USA, there are a growing number of pediatric gastroenterologists utilizing the non-biopsy diagnosis as postulated by ESPGHAN.

While most practitioners follow ESPGHAN or NASPGHAN recommendations, there is variability in specific practices, notably within the USA where a large portion utilized ESPGHAN guidelines despite not being endorsed by NASPGHAN. This may be because the ESPGHAN guidelines were more recently updated while NASPGHAN guidelines have not been updated in almost a decade. Caution must be taken utilizing ESPGHAN guidelines in North America as studies done in the United States do not show as strong of a positive predictive value of a TTG IgA > 10x ULN compared to that of European studies.

Conclusion:

Multidisciplinary discussions between Pediatric ED physicians and Pediatric Surgeons reduced CT usage, and corresponding radiation exposure, in the evaluation of appendicitis.



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Abstracts Day 2



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Professor Mostafa Yakoot

Autoinflammatory Syndromes

Autoinflammatory disease (AID) is a vast spectrum of disorders characterized by recurrent attacks of sterile inflammation caused by dysregulation of the innate immune system without involving antigen-directed autoimmunity. Clinically, AIDs typically present with multifaceted symptoms such as fever, joint pain, skin rash, abdominal pain, serositis, lymphadenopathy, and increased acute phase reactants, and thus may mimic infections clinically.

They may be divided into monogenic and polygenic types.

Monogenic autoinflammatory syndromes are those with identified genetic mutations, such as familial Mediterranean fever, tumor necrosis factor receptor-associated periodic fever syndrome (TRAPS), mevalonate kinase deficiency or hyperimmunoglobulin D syndrome, cryopyrin-associated periodic fever syndromes (CAPS), pyogenic arthritis pyoderma gangrenosum and acne (PAPA) syndrome, interleukin-10 and interleukin-10 receptor deficiencies, adenosine deaminase 2 deficiency and pediatric sarcoidosis.

Polygenic syndromes are those without an identified genetic mutation and include systemic-onset juvenile idiopathic arthritis, idiopathic recurrent acute pericarditis, Behçet syndrome, chronic recurrent multifocal osteomyelitis and inflammatory bowel disease and others.

Management has evolved to focus on targeted, steroid-sparing therapies, with specific agents selected based on the underlying molecular mechanisms of the disorder. These include colchicine, anti-IL-1 drugs (anakinra, canakinumab, rilonacept), anti-TNF agents (etanercept, infliximab), and JAK inhibitors (baricitinib, ruxolitinib), with ongoing research into novel targets and challenges remaining for patients without a genetic diagnosis.



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Professor Abd El Rahman El-Mashad

Skin of the Neonate: A Mirror of the Future

Background: The skin is the largest and one of the most informative organs in the newborn. Beyond serving as a physical barrier, neonatal skin reflects fetal development, genetic constitution, immune competence, and adaptation to extrauterine life. Careful skin examination can provide the earliest clues to disorders that extend far beyond the skin itself, influencing lifelong health and development.

Objective: To highlight the neonatal skin as a diagnostic and prognostic window into future health by reviewing its role in identifying systemic diseases, predicting long-term outcomes, and guiding early intervention.

Summary: Many neonatal cutaneous findings are benign and self-limiting; however, others represent the first manifestation of significant genetic, metabolic, infectious, vascular, endocrine, or neurocutaneous disorders. Recognition of characteristic skin lesions may facilitate early diagnosis of conditions associated with neurological impairment, developmental delay, immune dysfunction, or malignancy. In preterm infants, skin immaturity is closely linked to increased transepidermal water loss, infection, thermal instability, and impaired barrier function, making skin protection an essential component of neonatal care.

Recent advances have transformed our understanding of neonatal skin from a passive barrier to a dynamic immunological organ. The establishment of the skin microbiome, epidermal barrier maturation, and early inflammatory signaling are increasingly recognized as determinants of immune programming, allergic disease, and chronic inflammatory disorders later in life. Emerging technologies—including non-invasive skin imaging, biomarker analysis, genomics, and artificial intelligence-assisted pattern recognition—are opening new opportunities for precision neonatal medicine and earlier risk stratification.

Conclusion: The neonatal skin truly serves as a “mirror of the future.” A meticulous skin examination remains one of the most powerful, inexpensive, and non-invasive diagnostic tools available to pediatricians and neonatologists. Integrating traditional clinical skills with modern advances in dermatology, genomics, and microbiome science enables earlier diagnosis, personalized management, and improved long-term outcomes. Recognizing the messages written on the newborn’s skin today may profoundly influence the child’s health tomorrow.



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Prof. Heba Kamal

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Abstract:

The era of biologic and targeted small-molecule therapies in children with inflammatory bowel disease (IBD) has significantly improved disease outcomes. However, optimizing their use remains challenging in pediatric populations. Children exhibit unique pharmacokinetic and clinical characteristics, including faster drug clearance, higher inflammatory burden, growth related dosing challenges and body composition, all of which are crucial for long-term disease control. As a result, standardized dosing strategies are often inadequate, leading to treatment failure.

Treatment failure in pediatric IBD may also result from various factors, including different mechanisms of non-response to biologic therapies. Differentiating these mechanisms is essential for the selection of appropriate management, including dose escalation, interval shortening, switching within drug classes, or changing therapeutic mechanisms. Therapeutic drug monitoring (TDM), both proactive and reactive, is used to guide treatment decisions, prevent immunogenicity, and improve clinical and endoscopic outcomes. Higher early biologic trough concentrations have been associated with increased rates of steroid-free remission, while low drug levels are linked to antibody formation and loss of response.

Therapies beyond anti-TNF agents targeting IL-23, IL-12/23, JAK1, and integrin pathways provide additional options for individualized treatment selection based on disease phenotype, severity, and clinical urgency of symptom control. Rapid-onset agents may be preferred in sick patients with high inflammatory burden, while other biologics demonstrate efficacy in both, ileal and colonic disease.

This presentation will review mechanisms of biologic failure, clinical cases and practical interpretation of TDM, and evidence-based strategies for selecting and optimizing advanced therapies in pediatric IBD. The presentation will also focus on individualized treatment approaches in order to improve long-term outcomes and reduce disease-related complications.



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Professor Hala Agha

Frequently asked questions for Kawasaki disease spectrum

During the presentation, we will highlight on the definition of typical and atypical Kawasaki disease as well as multi-system inflammatory disease syndrome. We will present the indications for referral of pediatrician to pediatric cardiology and the value of transthoracic echocardiography and its frequency in Kawasaki disease as the features of high risk and severe Kawasaki disease. We will focus on the differential diagnosis of Kawasaki disease and the indications to perform multislices CT angiogram or cardiac MRI. We will present the initial management of Kawasaki disease and its duration as well the mechanism of IVIG and aspirin. We will focus on the treatment of high risk patients and coronary aneurysm and its duration. We will highlight on the contraindication of vaccination concerning the live attenuated vaccines. We will present the management and the diagnostic tools during long term follow-up of Kawasaki disease. We will give brief review on the status of Kawasaki disease spectrum in Egypt and give some examples of the publications.



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DR AMEER ELFEKY

Senior consultant of pediatrics Md pediatrics-
Cairo University

Medical nutrition therapy -National Nutrition Institute

Sports Nutrition in Children and Adolescent Athletes: Evidence-Based Updates for Pediatric Practice



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Prof. Yasser Elsayed, MD, PhD, FRCPC

Department of Pediatrics and Child Health, Section of Neonatology, University of Manitoba;
Children's Hospital Research Institute of Manitoba, Winnipeg, Manitoba, Canada

Gut Injury in Neonates

Neonatal gut injury spans a spectrum extending from self-limited food protein-induced enterocolitis to fulminant necrotizing enterocolitis, and the overlapping presentations of these entities, particularly bloody stools in a preterm infant, drive frequent misclassification. The consequences are asymmetric: stable infants are subjected to prolonged fasting and broad-spectrum antibiotics, while infants with evolving ischemic injury may reach the operating room late. This presentation offers an integrated triage framework that unifies clinical state, biochemical markers, radiology, and intestinal ultrasound into a single decision pathway. Five primary triggers are characterized, delayed feeding with perinatal antibiotic exposure, lymphocytosis-associated necrotizing enterocolitis, food protein-induced enterocolitis, hypoxic-ischemic injury, and lacto-bezoars, each mapped along an ischemia-progression axis that clarifies whether injury begins ischemic, ends ischemic, or remains confined to mucosal breach. Data are synthesized through the Manitoba Gut Injury Score-34, which aggregates clinical instability, abdominal signs, laboratory markers, abdominal radiography, and ten sonographic parameters scored as normal, localized, or generalized. Aggregate scores stratify severity and dictate escalation, with scores below 6 indicating non-specific presentations, 6 to 16 mild, 17 to 24 moderate, and 25 to 34 severe disease, moving management from observation through medical therapy to surgical intervention. Four clinical classes are defined, each with characteristic systemic signs, sonographic patterns, laboratory signatures, and trajectories ranging from complete recovery to stricture formation and mortality. Red flags mandating immediate surgical consultation include global wall thinning or thickening, loss of wall signature, complex peritoneal fluid, globally absent perfusion, clinical instability, worsening biochemistry, or perforation. Early and precise differentiation using this blueprint secures appropriate nutritional management for allergic enteropathy while accelerating life-saving intervention in severe injury.



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Prof. Yasser Elsayed, MD, PhD, FRCPC

Department of Pediatrics and Child Health, Section of Neonatology, University of Manitoba;
Children's Hospital Research Institute of Manitoba, Winnipeg, Manitoba, Canada

Bowel Ultrasound in Neonates

Abdominal radiography remains the conventional imaging standard for the neonatal abdomen, yet it captures only a static shadow and frequently misses the early, subtle, and dynamic changes that precede established bowel injury. Intestinal ultrasound offers a real-time, radiation-free window into tissue integrity and perfusion at the bedside. This presentation describes a reproducible point-of-care intestinal ultrasound protocol for the neonatal gut, based on a 9-zone, 34-clip scanning sequence in which every zone is imaged in two planes, 2D grayscale and color Doppler. Findings are organized into four diagnostic pillars, Air (portal venous gas, pneumatosis, pneumoperitoneum), Wall (thickness, intestinal signature, peristalsis), Fluid (simple, complex, dilated loops), and Color Doppler (normal, hyperemia, ischemia), yielding twelve reproducible sonographic markers across four phenotypes. Practical interpretive pearls are emphasized, including the separation of true pneumatosis, in which echogenic dots are locked within the bowel wall and peristalsis is lost, from intraluminal gas artifact with preserved peristalsis; the distinction between encysted air and free intraperitoneal air; recognition of the whirlpool sign of midgut volvulus; and dimensional criteria for hypertrophic pyloric stenosis. Reported diagnostic performance favors ultrasound substantially, with sensitivity of 81 to 100 percent and specificity of 80 to 100 percent, against 44 to 84 percent and 76 to 83 percent respectively for abdominal radiography, reflecting the capacity of ultrasound to detect perfusion compromise and mural injury before radiographic confirmation. The unifying clinical principle is that the sick bowel does not move. A systematic four-pillar approach therefore provides a more sensitive, specific, and physiologically informative assessment of the neonatal abdomen than radiography, which retains its primary value in the detection of free-air perforation. Mastery of this protocol is central to the early recognition of surgical emergencies in the NICU.



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Prof. Dalia Abdellatif Abdelrahman

Professor of pediatrics and neonatology/ESPEN diploma in clinical nutrition/ZU and AFCM-Egypt

Nutritional management remains a cornerstone of diabetes care in children and adolescents, playing a critical role in optimizing glycemic control, supporting normal growth and development, and reducing the risk of long-term complications. Recent advances in diabetes care have introduced innovative nutritional approaches that move beyond traditional carbohydrate counting toward more individualized, technology-assisted, and patient-centered strategies. This lecture will review emerging trends in the nutrition of children with diabetes, with a particular focus on the integration of continuous glucose monitoring (CGM), insulin pump therapy, and hybrid closed-loop systems into dietary planning. The role of precision nutrition, which considers individual glycemic responses, lifestyle factors, and metabolic characteristics, will be discussed as a promising approach to improving clinical outcomes. Current evidence regarding dietary patterns, including low-glycemic-index diets, healthy plant-based eating patterns, and optimized macronutrient distribution, will also be examined.

The presentation will highlight the growing understanding of the gut microbiome, meal timing, and family-centered nutritional interventions in pediatric diabetes care. Special attention will be given to challenges unique to children and adolescents, including growth requirements, food preferences, school environments, physical activity, and psychosocial factors that affect dietary adherence.

By exploring recent research and practical applications, this lecture aims to equip pediatricians, dietitians, diabetes educators, and healthcare professionals with evidence-based strategies to deliver personalized nutritional care that supports both metabolic control and healthy development in children with diabetes.

Keywords: Pediatric Diabetes, Type 1 Diabetes, Child Nutrition, Precision Nutrition, Continuous Glucose Monitoring, Carbohydrate Counting, Glycemic Control, Gut Microbiome, Diabetes Technology, Medical Nutrition Therapy



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Prof. Hala Atta

Total Parenteral Nutrition (TPN) in Neonates

Abstract

Total parenteral nutrition (TPN) is a critical form of nutritional support for neonates who cannot receive sufficient nutrients through enteral feeding. It is commonly used in premature infants, very low birth weight neonates, critically ill newborns, infants with necrotizing enterocolitis, congenital gastrointestinal anomalies, sepsis, short bowel syndrome, or those recovering from surgery. Because newborns, especially preterm babies, have limited nutrient reserves and high metabolic demands, early nutritional intervention is essential to support growth, brain development, immune function, and overall survival. TPN provides nutrients directly into the bloodstream, including fluids, glucose, amino acids, lipids, electrolytes, minerals, vitamins, and trace elements. The main goal is to maintain metabolic stability and provide adequate energy and protein until full oral or enteral feeding becomes safe and tolerated. Neonatal TPN must be prescribed individually according to gestational age, birth weight, postnatal age, clinical condition, fluid requirements, renal function, liver function, and biochemical results. Although TPN is life-saving, it carries important risks such as catheter-related infection, thrombosis, hyperglycemia, electrolyte imbalance, acidosis, cholestasis, liver dysfunction, and metabolic bone disease. Therefore, safe use requires strict aseptic technique, accurate calculation of nutritional components, careful catheter care, and regular monitoring of weight, serum electrolytes, glucose, triglycerides, blood urea nitrogen, liver function tests, calcium, phosphorus, and hemoglobin. Healthcare providers should also aim to introduce and advance breast milk or enteral feeding as soon as the neonate shows adequate tolerance, sucking, and swallowing ability. In conclusion, TPN plays a vital role in neonatal intensive care by preventing nutritional deficiency and supporting growth in vulnerable newborns. Its success depends on continuous monitoring and multidisciplinary cooperation among neonatologists, nurses, pharmacists, and dietitians to maximize benefits while reducing complications.

Keywords: Total parenteral nutrition, neonates, preterm infants, neonatal intensive care, nutritional support.

Prepared from the provided abstract and lecture content on neonatal parenteral nutrition



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Prof. Dalia Abdellatif Abdelrahman

Professor of pediatrics and neonatology/ESPEN diploma in clinical nutrition/ZU and AFCM-Egypt

Artificial Intelligence (AI) is rapidly emerging as a transformative tool in healthcare, with significant implications for paediatric nutrition. As childhood nutrition becomes increasingly complex due to the coexistence of undernutrition, micronutrient deficiencies, obesity, and chronic diseases, AI offers novel opportunities to enhance nutritional assessment, optimize dietary interventions, and support precision nutrition strategies tailored to individual needs. This lecture will explore the current and emerging applications of AI in pediatric nutrition, including automated dietary assessment, growth monitoring, prediction of nutritional risk, and personalized nutrition planning. Advances in machine learning, computer vision, wearable technologies, and digital health platforms are enabling more accurate analysis of dietary intake, early identification of growth and nutritional disorders, and real-time monitoring of nutritional outcomes. AI-driven tools can also integrate clinical, anthropometric, biochemical, genetic, and lifestyle data to provide individualized recommendations that support healthy growth and disease prevention.

The presentation will review recent evidence on the use of AI in the management of pediatric obesity, diabetes, malnutrition, food allergies, and specialized nutritional care. Opportunities and challenges related to data quality, algorithm bias, privacy, ethical considerations, and equitable access will also be discussed. Furthermore, the lecture will examine the evolving role of healthcare professionals in an AI-enhanced clinical environment and the importance of maintaining patient-centered care while leveraging technological innovation.

By highlighting current developments and future directions, this lecture aims to provide healthcare professionals, researchers, and nutrition specialists with a comprehensive overview of how AI is reshaping pediatric nutrition and advancing the goal of more precise, efficient, and accessible nutritional care for children worldwide.

Keywords: Artificial Intelligence, Pediatric Nutrition, Precision Nutrition, Machine Learning, Childhood Obesity, Malnutrition, Digital Health, Growth Monitoring, Personalized Nutrition, Pediatric Healthcare.



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Abstracts Day 3



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Prof. Naglaa M Kamal

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Genetic Cholestatic Liver Disease: Patient Journey and Emerging Hope in the Era of Ileal Bile Acid Transporter (IBAT) Inhibitors

Genetic cholestatic liver diseases, particularly Progressive Familial Intrahepatic Cholestasis (PFIC), are rare inherited disorders characterized by impaired bile acid transport, progressive cholestasis, debilitating pruritus, growth failure, fat-soluble vitamin deficiencies, and eventual liver fibrosis or failure. Delayed diagnosis remains common because of overlapping clinical presentations with other neonatal and pediatric liver disorders, resulting in prolonged patient suffering and irreversible hepatic injury. In regions with high consanguinity, such as Saudi Arabia, genetic etiologies account for a substantial proportion of infantile cholestasis, underscoring the importance of early recognition, genetic testing, and multidisciplinary management.

The patient journey often begins with persistent jaundice and intractable pruritus that profoundly impair sleep, physical functioning, psychosocial well-being, and family quality of life. Traditional management—including nutritional optimization, fat-soluble vitamin supplementation, ursodeoxycholic acid, rifampicin, antihistamines, and surgical biliary diversion—has largely focused on symptom control, with liver transplantation remaining the definitive option for progressive disease.

Recent advances in the understanding of enterohepatic bile acid circulation have transformed the therapeutic landscape. Ileal bile acid transporter (IBAT) inhibitors, particularly odeixibat, reduce intestinal bile acid reabsorption, decrease the circulating bile acid pool, and lower hepatic bile acid burden through medical interruption of the enterohepatic circulation. Clinical studies have demonstrated significant reductions in serum bile acids and pruritus, together with improvements in sleep, growth parameters, liver biochemistry, and long-term native liver survival in appropriately selected patients with PFIC.

Through representative PFIC1 and PFIC3 patient journeys, this review highlights the evolution from delayed diagnosis and conventional supportive care to precision medicine guided by molecular diagnosis. Careful patient selection, genotype characterization, and assessment of disease stage are essential to maximize therapeutic benefit, recognizing that treatment response varies according to the underlying molecular defect.

The emergence of IBAT inhibitors represents a paradigm shift in the management of genetic cholestatic liver diseases, offering an effective, non-surgical strategy to alleviate symptoms, improve quality of life, and potentially delay or reduce the need for biliary diversion and liver transplantation. Earlier diagnosis coupled with timely initiation of targeted therapy may substantially alter the natural history of these disorders.



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Prof. Carlos Lifschitz

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Title: Neonatal Diarrhea.

The etiology of neonatal diarrhea includes carbohydrate malabsorption, infectious agents (bacterial, viral, and parasitic), dietary factors, and congenital disorders. Common infectious causes include rotavirus, enteric bacteria like *Escherichia coli*, and, in specific settings, parasites such as *Giardia*. Risk factors include low birth weight, prematurity, poor feeding practices, and lack of access to clean water. The most serious ones are congenital diarrheas and enteropathies (CODEs), a heterogeneous group of rare inherited disorders characterized by chronic, severe diarrhea presenting in the neonatal period or early infancy. These conditions result from genetic defects affecting intestinal epithelial transport, digestion and absorption, epithelial polarity, enteroendocrine function, immune regulation, or lymphatic development. More than 60 disease-associated genes have been identified, resulting in improved molecular classification and precise diagnosis. Major categories include defects in nutrient and electrolyte transport (e.g., congenital chloride diarrhea, congenital sodium diarrhea, glucose-galactose malabsorption), epithelial trafficking and polarity disorders (e.g., microvillus inclusion disease), enteroendocrine dysfunction, immune-mediated enteropathies, and disorders of lipid transport and lymphatic function. Diagnostic evaluation requires careful clinical phenotyping, laboratory assessment of stool composition, endoscopic and histopathologic examination when indicated, and increasingly, comprehensive genetic testing using targeted gene panels or whole-exome sequencing. Management remains primarily supportive and includes meticulous fluid and electrolyte replacement, specialized nutritional therapy, parenteral nutrition for severe intestinal failure, and treatment of disease-specific complications. Advances in molecular diagnosis have facilitated genetic counseling, informed prognostic assessment, and identified opportunities for targeted therapies in selected disorders.



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Prof. Jose Spolidoro

Pediatric with specialization in Gastroenterology and nutrition at UCLA

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Prof. Richard H. Sandler, MD

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Over 20 % of all pediatric GI patients present with chronic abdominal pain symptomatology. A common and almost universally missed etiology of this is a condition called "abdominal cutaneous nerve entrapment syndrome," or ACNES. This presents as a sharp or dull (not cramping) very localized pain and tenderness, unaffected by eating or bowel movements, present day and night, often made worse by movement or coughing. The cause of ACNES is entrapment of one or more of the thoracic nerves as they pierce the abdominal wall in well-defined foramen locations. Treatment is properly placed local anesthetic which typically affords both immediate and long-term relief. Although previously described, in the author's experience with over 300 cases, ACNES remains the single most commonly missed abdominal pain condition by pediatric gastroenterologists, ER and referring primary care physicians. The diagnosis is usually easily made by history and physical examination alone. The condition meets the "so what" test, as it is common, causes significant pain, leads to extensive unneeded evaluations, and is usually promptly and easily treatable. Hence, ACNES is deserving of more widespread awareness by the pediatric GI community. This talk will review the presentation, pathophysiology, treatment and outcomes.



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Prof. Laila M Sherief

Professor of Pediatrics and Pediatric Hematology & Oncology

Food as Medicine: The Journey From Cancer Prevention to Recovery

Background:

Cancer prevention begins long before diagnosis, and nutrition represents one of the most accessible and potentially modifiable determinants of cancer risk. Increasing evidence suggests that dietary patterns, body composition, metabolic health, inflammation, and the gut microbiome interact to influence carcinogenesis. The concept of “food as medicine” therefore extends beyond supportive nutrition during cancer treatment to recognizing food as a potential tool for cancer prevention and lifelong health.

Objective:

To highlight the role of nutrition in cancer prevention and explore how the principles of healthy eating can be carried forward into cancer treatment, recovery, and survivorship.

Content:

Prevention provides the first and perhaps most powerful opportunity to use food as medicine. A healthy dietary pattern characterized by a high intake of vegetables, fruits, whole grains, legumes, nuts, and fiber, together with limited consumption of processed meat, excess red meat, highly processed foods, refined carbohydrates, and added sugars, may contribute to lowering cancer risk. Maintaining a healthy body weight is equally important, as excess adiposity is associated with chronic inflammation, insulin resistance, hormonal alterations, and increased risk of several cancers. Beyond individual nutrients, the overall dietary pattern and its interaction with the gut microbiome may influence immune regulation, inflammation, and metabolic pathways involved in carcinogenesis.

Importantly, cancer prevention should begin early in life. Establishing healthy eating habits during childhood and adolescence may help shape lifelong dietary behaviors and reduce future exposure to modifiable cancer risk factors. This emphasizes the need to move from a disease-centered approach to a life-course approach to cancer prevention, involving families, schools, healthcare professionals, and communities.

Following cancer diagnosis, the role of nutrition evolves from risk reduction to maintaining nutritional status, preserving muscle mass, supporting treatment tolerance, and promoting recovery. During survivorship, healthy dietary patterns remain essential for restoring health, managing treatment-related consequences, and reducing the burden of other chronic diseases.

Conclusion:

The journey of food as medicine begins before cancer begins. Nutrition should be viewed not simply as supportive care after diagnosis, but as an integral component of cancer prevention across the life course. Moving from “food on the plate” to “food as prevention, protection, and recovery” offers an opportunity to make cancer care more proactive, personalized, and holistic.



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Prof Elizabeth Tayler

Global Burden of Antimicrobial Resistance

Most deaths attributable to antimicrobial-resistant infections occur without the resistance being identified or recorded, creating substantial challenges in estimating the true scale of the problem. This presentation will examine how current estimates of the burden of resistant infections are generated, how they should be interpreted, and what subgroup analyses reveal about trends across age groups and infection syndromes.

The presentation will review current estimates of antimicrobial resistance burden and highlight the markedly different trends observed since 1990 among children under 5 years compared with older populations. In children, a decline of more than 50% has been driven largely by reductions in lower respiratory infections, including *Streptococcus pneumoniae*, and diarrhoeal diseases, reflecting improvements in immunisation and water, sanitation, and hygiene provision. At the same time, the proportion of deaths associated with resistant infections among children is increasing. The presentation will consider the implications of this burden for healthcare delivery in low- and middle-income countries, where high resistance rates can make interventional and intensive care substantially more hazardous.

Management of paediatric infections remains constrained by limited access to appropriate medicines and formulations. These gaps contribute to the overall burden of bacterial infection and increase the risk of resistance, underscoring the need for strengthened access, stewardship, and infection prevention strategies.



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Prof Elizabeth Tayler

Worldwide Policies and Procedures to Combat Antimicrobial Resistance

Antimicrobial resistance has received exceptional political attention among global health priorities. It was discussed for a second time at the United Nations General Assembly in 2024 and has featured in most G20 communiqués over the past decade.

Countries have committed to developing National Action Plans and to global targets, including a 10% reduction in antimicrobial resistance mortality and an increase in the proportion of Access antibiotics used to 70%. Despite these commitments, progress in implementing action at scale remains limited, and challenges in international and domestic health financing pose a major threat to continued progress.

A revised Global Action Plan for antimicrobial resistance was launched this year. Building on the 2015 Global Action Plan, it places an even stronger emphasis on One Health approaches, governance, and coordination as essential components of an effective response.



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Prof Gamal El Tagy

Evaluation of clinical outcomes in children with intrahepatic cholestasis post Partial External Biliary Diversion: a single Centre Experience

Purpose: Severe pruritus caused by progressive familial intrahepatic cholestasis (PFIC) and Alagille syndrome (AGS) is refractory to medical treatment. Surgical interruption of the enterohepatic circulation is considered the mainstay of alleviating distressing symptoms and delaying cirrhosis. This study aims to evaluate the short-term effect of partial external biliary diversion (PEBD) on pruritus, liver disease progression, patient's growth, and quality of life.

Methods: This prospective cohort study enrolled children with PFIC and AGS from July 2019 to July 2021, whose guardians consented to the PEBD procedure. A standard surgical approach was performed by a single surgeon. Outcomes were measured subjectively and objectively pre- and post-procedure using the pruritus 5-D itching score, Paediatric Quality of Life Inventory scale (PedsQL), growth parameters, bile acids level, and liver function tests. Patients' follow-up period ranged from 6 to 12 months.

Results: Seven patients had PEBD procedure; five with PFIC and two with AGS. A significant improvement was detected in the 5-D itching score (p-value < 0.001), PedsQL (p-value < 0.001), and bile acids level (p-value 0.013). The preexisting growth failure was ameliorated. The downward trend in the bilirubin level was not significant. No influential difference in the other liver function tests occurred. No intra-operative complications encountered. Only one case had a post-operative stoma prolapse which was managed surgically.

Conclusion: PEBD procedure could be considered as an effective and safe treatment options for intractable pruritus in patients with PFIC or AGS, providing preserved synthetic liver functions.



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Prof. Ahmed Elwakeel

The Half-Fixed Heart: A Guide to Diarrhoea in the Glenn Child

Children living with a bidirectional cavopulmonary (Glenn) anastomosis increasingly present first to the general paediatrician rather than to a cardiac centre. Their circulation is unfamiliar and counterintuitive: no ventricle propels blood to the lungs. Pulmonary blood flow is passive, driven by systemic venous pressure and permitted only by low pulmonary vascular resistance. This single fact converts an ordinary childhood illness into a circulatory threat.

Acute gastroenteritis in these children is not simply a gastrointestinal problem. Volume depletion reduces central venous pressure and, with it, pulmonary perfusion. Metabolic acidosis raises pulmonary vascular resistance, closing a vicious circle toward profound desaturation. Superimposed polycythaemia and thrombotic risk narrow the therapeutic window further – in both directions, since the single ventricle also can't tolerate volume overload.

This session equips the general practitioner to recognise the child, interpret the numbers, and act early. We define what baseline looks like – the expected saturation range, the polycythaemia, the venous congestion – and why deviation must be judged against the individual child rather than a population norm. We then work through a structured differential diagnosis that separates ordinary infectious causes from cardiac-specific ones: diuretic and drug effects, mesenteric venous congestion, bacterial overgrowth, and protein-losing enteropathy, whose triad of chronic diarrhoea, oedema and hypoalbuminaemia must never be missed.

Practical management is presented as a three-tier referral algorithm distinguishing the child who may be managed locally, the child who needs same-day cardiac consultation, and the child requiring immediate transfer. Emphasis is placed on early intravenous access, thoughtful diuretic adjustment, and the investigations that change decisions.



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JUVENILE AUTOIMMUNE DISORDERS

Juvenile autoimmune liver disorders (JAILD) are inflammatory liver diseases characterised histologically by a dense mononuclear cell infiltrate in the portal tract (interface hepatitis) and serologically by the presence of non-organ and liver specific autoantibodies and increased levels of immunoglobulin G (IgG), in the absence of a known etiology. JAILD usually respond to immunosuppressive treatment, which should be instituted promptly. Their onset is often ill-defined, frequently mimicking acute hepatitis. JAILDs include autoimmune hepatitis (AIH) and autoimmune sclerosing cholangitis (ASC).

AIH: There are two types of AIH: AIH-1 is seropositive for smooth muscle and/or antinuclear antibody (SMA/ANA); AIH-2 for liver kidney microsomal type 1 antibody (LKM1) and/or anti-liver cytosol type 1 (anti-LC1). In both 80% of patients are female. AIH-2 patients present more acutely, at a younger age and commonly have IgA deficiency, while duration of symptoms before diagnosis, clinical signs, family history of autoimmunity, presence of associated autoimmune disorders, response to treatment (prednisolone ± azathioprine) and long-term prognosis are similar in the two groups.

ASC is the most common form of juvenile sclerosing cholangitis. Clinical, biochemical, immunological and histological presentation is often indistinguishable from AIH-1. In both conditions, there are high IgG, ANA±SMA, and interface hepatitis. ASC responds to immunosuppressive treatment with prednisolone ± azathioprine satisfactorily and similarly to AIH-1 in respect to remission and relapse rates, times to normalization of biochemical parameters, and decreased inflammatory activity on follow up liver biopsies. However, cholangiopathy can progress in 30-50% of patients despite treatment, suggesting that current immunosuppression is effective in abating the parenchymal inflammatory damage, but not in controlling bile duct damage. Urso deoxycholic acid (UDCA) is usually added to treatment. AIH can evolve to ASC over the years, despite treatment, worsening the prognosis and transplant-free survival.



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TREATMENT OF VIRAL HEPATITIS B IN CHILDHOOD

In endemic areas hepatitis B is a disease of childhood, acquired either perinatally from HBsAg positive mothers or horizontally from infected mates/family members. Despite the availability of effective vaccination, hepatitis B virus (HBV) remains a major public health problem. The probability of becoming a chronic HBV carrier is correlated to age at infection and efficiency of the immune system, being highest in children infected within the first year of life, who tend to become 'tolerant' to the virus. Chronically infected children are usually asymptomatic, but their liver histology often shows progressive inflammation despite normal or minimally abnormal liver function. They spread the infection and, particularly if male, have a high risk of progressing to cirrhosis and hepatocellular carcinoma, these complications being correlated to the length of time to achieve anti-HBe seroconversion. Treatment should therefore aim at speeding up anti-HBe and ultimately anti-HBs seroconversion. Antiviral drugs for chronic HBV include interferons and nucleos(t)ide analogues. Importantly, compared to adults, children show a higher rate of cure after a finite-course of antiviral therapy, with ultimately 50% achieving HBsAg loss. Age at treatment initiation is an important factor affecting the efficacy of antiviral therapy, earlier initiation of antiviral therapy being linked to a greater ability to clear HBsAg.



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PATHOGENESIS OF AUTOIMMUNE LIVER DISEASE

An unknown but powerful stimulus promotes the formation of the massive inflammatory cell infiltrate present at diagnosis in AIH. The high number of activated inflammatory cells are likely to initiate and perpetuate liver damage. An autoimmune attack can follow different pathways to inflict damage on hepatocytes. Liver damage is likely to be orchestrated by CD4⁺ T lymphocytes recognizing a self-antigenic peptide on hepatocytes. To trigger an autoimmune response, the peptide must be embraced by an HLA class II molecule and presented to uncommitted (naïve) CD4⁺T helper (Th0) cells by professional antigen-presenting cells (APC), with the co-stimulation of ligand-ligand (CD28⁺ on Th0, CD80⁺ on APC) fostering interaction between the two cells. Th0 cells become activated, differentiate into functional phenotypes according to the cytokines prevailing in the microenvironment and the nature of the antigen, and initiate a cascade of immune reactions determined by the cytokines these activated T cells produce. Th1 cells, arising in the presence of the macrophage-produced interleukin 12 (IL-12), secrete mainly IL-2 and interferon-gamma, which activate macrophages, enhance expression of HLA class I (increasing hepatocyte vulnerability to CD8⁺ T cell cytotoxic attack) and induce expression of HLA class II molecules on hepatocytes. Th2 cells, which differentiate from Th0 if the microenvironment is rich in IL-4, produce mainly IL-4, IL-10 and IL-13 which favor autoantibody production by B lymphocytes. Physiologically Th1 and Th2 antagonize each other. Th17 cells arise in the presence of transforming growth factor beta (TGF-beta) and IL-6 and have an important effector role in inflammation and autoimmunity. The process of auto-antigen recognition is strictly controlled by regulatory mechanisms, such as those exerted by CD4⁺CD25⁺ regulatory T cells (T-regs), which are derived from Th0 in the presence of TGF-beta, but in the absence of IL-6. If regulatory mechanisms fail, the autoimmune attack is perpetuated. Defective T-regs number and function are present in AIH, particularly at diagnosis or during relapse.



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LABORATORY DIAGNOSIS OF AUTOIMMUNE LIVER DISEASE: FROM THE LAB TO THE CLINIC

Serology is key to the diagnosis of autoimmune hepatitis (AIH). Clinicians need to be aware of which tests to request, how to interpret the laboratory reports, and be familiar with the laboratory methodology. If correctly tested, more than 95% of AIH patients show some serological reactivity. Indirect immunofluorescence on triple rodent tissue is recommended as first screening step, since it allows the detection of all liver-relevant autoantibodies, except for anti-soluble liver antigen (SLA) antibody, which needs to be detected by molecular based assays. The threshold of immunofluorescence positivity is a titre equal or exceeding 1/40, but for patients younger than 18 years even lower titres are clinically significant. Anti-nuclear antibody (ANA) and/or anti-smooth muscle antibody (SMA) characterize type 1 AIH. ANA in AIH typically shows a homogeneous staining pattern on HEp2 cells, without any specific target antigen. Anti-SMA displays different staining patterns on indirect immunofluorescence: the vascular/glomerular (VG) and the vascular/glomerular/tubular (VGT) patterns are considered specific for AIH, whilst the V pattern can be found in a variety of diseases. Type 2 AIH, which is rare and affects mostly children/adolescents, is characterized by anti-liver kidney microsomal 1 (LKM1) and/or anti-liver cytosol 1 (LC1) antibodies. Presence of anti-neutrophil cytoplasmic antibody (ANCA), particularly atypical p-ANCA (pANNA), points to the diagnosis of AIH in absence of other autoantibodies. pANNA also associated with sclerosing cholangitis and inflammatory bowel disease, which to investigated. The only antibody specific for AIH is anti-SLA, which is associated with a more severe disease course.

