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Review Article

Shifting stones: a review on rising tide of pediatric cholelithiasis in the modern age

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ABSTRACT

Once deemed a clinical rarity, paediatric cholelithiasis has now emerged as a common hepatobiliary disorder among children. The rising incidence is closely linked with the global propulsion in pediatric obesity, dietary transitions, sedentary behaviour, and improved imaging modalities, facilitating rapid and frequent diagnosis. This comprehensive literature review delineates the shifting epidemiology, explores pathophysiological mechanisms, and critically appraises diagnostic and management algorithms in the pediatric population.

Keywords: Pediatric gallstones, Pediatric cholelithiasis, Biliary stasis

INTRODUCTION

In recent years, the pediatric hepatobiliary diseases have witnessed a significant evolution— where gallstones, once a diagnostic curiosity in children, are assuming centre stage. While adult cholelithiasis is a well-charted territory, pediatric gallstone disease remains under-investigated and inconsistently managed with no standard management protocol. The need is to study the multifactorial etiology of pediatric gallstone disease. Non-hemolytic and cholesterol-based calculi are now more frequently observed, marking a demographic and clinical shift in the disease profile.

Examining the role of imaging, while addressing limitations in differentiating symptomatic from incidental findings can be a future research entity. Management remains controversial, especially in asymptomatic cases. The debate continues between conservative monitoring versus early cholecystectomy, with a focus on long-term outcomes, recurrence rates, and quality of life.

METHODS

Identification

A thorough search of electronic databases, including PubMed/MEDLINE, Research Gate, and Google Scholar was performed. The search encompassed keyword combinations like "Pediatric Gallstones," "cholelithiasis," "bile sludge," and "biliary stasis".

Inclusion criteria

Population

Studies involving patients aged 0–22 years with focus on pediatric data.

Study design

Original research articles, randomized controlled trials, observational studies, systematic reviews relevant to the topic.

Focus of study

Studies that addressed any aspect of gallstone disease in children.

Language and accessibility

Articles published in English with full-text availability.

Publication timeline

Articles published from inception to 2025, with no lower date limit due to the rarity and evolving understanding of pediatric cholelithiasis. Seminal works prior to 2000 were included when foundational or historically significant (e.g., Matos et al and Benjamin).

Exclusion criteria

Non-original literature including editorials, opinion pieces, letters to the editor, conference abstracts, non-English publications, incomplete access or data and irrelevant topic scope articles focused primarily on gallbladder cancer or non-gallstone hepatobiliary diseases without linkage to pediatric cholelithiasis.

EPIDEMIOLOGY

Global data reveals a slow yet steady rise in pediatric gallstone diagnoses. The most recent literature, reveals the prevalence of gallstones between 1.9% and 4% in the pediatric age group.[1] Intriguingly, even neonates and toddlers are no longer exempt.[2] The intersection of high-resolution ultrasonography and an obesogenic environment has rendered this once-exotic diagnosis, a quotidian finding in the pediatric clinics.[3] The research on neonatal cholestasis and disruption of bile acid homeostasis as a protective response to oxidative stress still remains relatively unexplored.[4]

ETIOLOGY

The traditional trifecta of pediatric gallstone risk—chronic haemolysis, total parenteral nutrition, and congenital biliary anomalies has expanded its territories. Today, clinicians must also reckon with the now most prominent phenotype: the obese, sedentary, fast-food-raised children with cholesterol-predominant stones early in life.[5] The prerequisites for paediatric cholelithiasis are- hemolytic anemias accounting for the most pigment stones. Bilirubin overproduction due to excess hemolysis can precipitate with calcium thereby forming pigment stones.[6] While hemolytic disorders take the limelight, iron deficiency anemia, in a nutritionally deprived country can also be a risk factor. Iron deficiency can potentially lead to bile super saturation due to alteration of hepatic enzyme metabolism followed by stone formation, suggesting a complex etiology.[7]

Management strategies are tailored to the patient's condition, and are usually surgical. Several studies reveal the indication for a combined splenectomy and cholecystectomy over a solitary splenectomy in cases of spherocytosis in the presence of gallstones as it confers a better survival rate.[8,9]

Obesity now fuels cholesterol super saturation, resembling adult metabolic syndromes with the advent of cholesterol stones in the pediatric population. The growing obesity epidemic is a catalyst to the disease due to impaired gall bladder motility, excessive hepatic secretion and cholesterol saturation of bile.[10,11] It acts potently on lithogenic mechanisms like gall bladder stasis, metabolic syndrome insulin resistance, hypertriglyceridemia thereby precipitating stones. With the significant burden of obesity, weight loss becomes goal for obese patients. Surprisingly the likelihood of cholelithiasis is also increased if the weight loss is too rapid, possible with a very low-calorie dietary intake.[12] Bariatric surgery's advent brings substantial weight loss with major improvements in portion and glycaemic control in severely obese patients. The Roux-en-Y gastric bypass (RYGB) technique is also a lithogenic risk factor.[12] Studies reveal the use of ursodeoxycholic acid prevents lithogenic changes in bile and could potentially eliminate the risk of cholelithiasis in obese patients undergoing rapid loss of weight.[12] However, a diet rich in fat, at least 7 g/day can improve gallbladder kinetics, and therefore decreases the risk of symptomatic gallstones.[13,14]

Ceftriaxone-induced biliary pseudolithiasis represents an iatrogenic twist in younger children. This miraculous drug, exploited due to wide spectrum of anti-microbial activity, excellent cerebrospinal fluid penetration and long plasma half-life allows developing significant complications.[15] In patients with adequate renal function, 60% of the drug is eliminated unchanged through the urine, and 40% through the bile.[16] Ceftriaxone, tends to accumulate in the bile at levels significantly higher than in the bloodstream. This high concentration enables it to easily combine with calcium, forming an insoluble compound that can crystallize into stones.[16] Experimental data indicate that this precipitation typically occurs with daily doses of 2 grams or more.[17] Additional factors that may increase the risk include elevated calcium levels, reduced bile movement due to fasting or reliance on total parenteral nutrition, impaired kidney function, prolonged or high-dose ceftriaxone therapy, and surgical procedures that result in gallbladder stasis.[18] For kidney stone formation linked to ceftriaxone, dehydration, restricted fluid intake, and use of medications harmful to the kidneys are known contributors. Fortunately, these bile-related precipitates usually resolve on their own once the drug is discontinued (Figure 1).[19,20,29]

Total parenteral nutrition (TPN) is evidently disrupting the enterohepatic circulation.[21] This leads to bile stasis and formation of pigment stones.[22] As per theory, humans are "meal eaters" and are ideally not adapted to the continuous

inflow of nutrients thereby altering the normal physiology of the body. Association with biliary sludge formation is more frequently observed in neonates, probably due to prematurity and immaturity of the enterohepatic cycle of bile acids. This sludge could lead to cholelithiasis which appears echogenic on ultrasound, lacking an acoustic shadow.[23] Investigators have successfully demonstrated reversal of hepatobiliary abnormalities on discontinuation of parenteral nutrition, though the mechanism is unclear. This could either be explained by the theory of removal of an offending agent, the parenteral solution concoction or the re-establishment of the normal alimentation.[23,24] Animal studies have revealed that a daily intravenous infusion of cholecystokinin-octapeptide has effectively prevented the development of gallbladder stasis in a cohort of prairie dogs with significantly unaltered lithogenic indices of gallbladder while administration of total parenteral nutrition.[25]

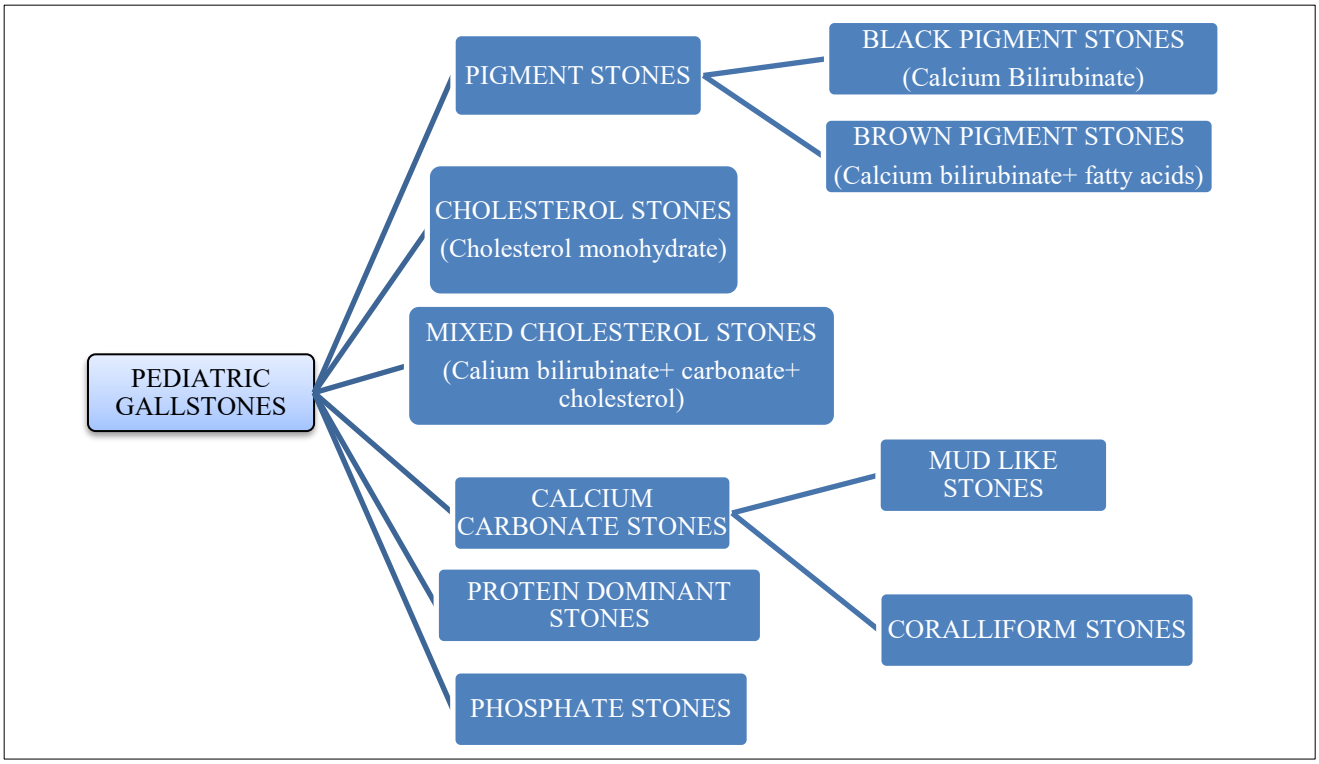

Figure 1: Gallstones of varying etiologies.

Genetic polymorphisms in genes such as ABCG5/8, CYP7A1, and UGT1A1 now hint at a heritable susceptibility, though pediatric genome-wide association studies remain scarce.[26]

PATHOPHYSIOLOGY

Gallstone formation is governed by the "three-legged stool": super saturation of bile with cholesterol or pigment, impaired gallbladder motility, and crystal nucleation.[27] However, paediatric physiology adds significant contribution to these pathways in subtle ways.

In neonates, the immature hepatobiliary transport systems and altered bile acid composition increase lithogenicity.[28,29]

In adolescents, hyperinsulinemia and estrogenic surges during puberty may act as additional lithogenic agents.[30]

Emerging work on gut microbiota suggests that dysbiosis may influence bile acid metabolism and gallstone risk, opening potential for probiotic and prebiotic interventions in the future.[31] "Microliths" refer to difficult to diagnose gallstones (<3 mm) with a clinical tendency to cause biliary colic and pancreatitis. 'Biliary sludge' a possible indicator for future pediatric cholelithiasis, is composed of precipitates of cholesterol monohydrate crystals, calcium bilirubinate, calcium phosphate along with calcium salts of fatty acids embedded in biliary mucin.[27,32]

CLINICAL MANIFESTATIONS

Pediatric cholelithiasis spans a broad clinical continuum from asymptomatic gallstones, which are a common incidental finding during abdominal imaging to biliary colic in older children mimicking adult presentations. With symptoms like postprandial epigastric pain, nausea, diarrhea, weight loss and vomiting some pediatric patients present with typical symptoms.[33-35] Murphy's sign, expiratory arrest on palpation of the right upper quadrant is still a pathognomonic to the disease. Since infants lack the ability to verbalize symptoms and discomfort, they may present with non-specific danger signs like poor

feeding, irritability, or failure to thrive. This diagnostic ambiguity often delays intervention. Elimination of the differential diagnosis like choledochal cyst, biliary disorders from cholelithiasis is important for initiating a management protocol.

DIAGNOSTICS

The workup of pediatric cholelithiasis is similar to that of the adult disease presentation with altered laboratory tests such as gamma–glutamyl transferase (GGT), amylase, urine analysis, indirect bilirubin, ALT, AST and alkaline phosphatase.[36] Ultrasound although only in cases of uncomplicated cholelithiasis, still remains the cornerstone of diagnosis. It offers high sensitivity for gallstones, sludge and gallbladder wall thickening. It highlights the importance of an accurate pre-operative evaluation of the condition. However, operator dependency and limitations in obese patients necessitate supplementary tools. Plain radiography is seldom of clinical importance as gallstones are radiolucent with the exception of calcium carbonate ones. MRCP is rated gold standard for suspected choledocholithiasis, especially preoperatively. ERCP, though technically demanding in children, is invaluable for managing choledocholithiasis and post-cholecystectomy syndromes.[37] The challenge lies in balancing surgical intervention with long-term risk mitigation and quality of life. ERCP in the pediatric population has been associated with the same frequency of success and complications as in adults.[38] Radionuclide scans can also be used to study the gall bladder filling and bile excretion especially as a response to CCK or lipid dense meal. However, the pediatric application of these diagnostic modalities still remains constrained by logistical and ethical concerns.

MANAGEMENT

There exists no universal consensus on how to treat pediatric cholelithiasis, especially in asymptomatic or minimally symptomatic children. The current knowledge regarding the disease has developed strategies as illustrated in Figure 2.

Watchful waiting

For asymptomatic stones, particularly in younger children. There is no indication to aggressively undertake a medical treatment in such cases with the simple exclusion of sickle cell disease cases. These children can undergo safe follow ups without progression to any complication, though the risk still prevails.[40,41]

Medical dissolution therapy

The symptomatic patients- simple or complicated gall stones require surgical intervention irrespectively. If the patient is asymptomatic or has minimal symptoms, it is reasonable to start an empirical treatment of UDCA/ antibiotics although not definitive cure.[42] Improvement in laboratory investigations and imaging within 2 weeks are signs of biliary disease resolution. Even for choledocholithiasis, watchful waiting for 1-2 weeks is recommended, allowing the possibility of spontaneous resolution.[43] The role of stone dissolution therapy is still under evaluation. Neonatal cholelithiasis is conferred as a temporary and a self-limiting phenomenon due to its spontaneous resolution in majority of asymptomatic cases.[36] Ursodeoxycholic acid (UDCA) has shown mixed results while its long-term safety and efficacy in children remain under debate. It suppresses hepatic cholesterol synthesis and secretion while inhibiting its intestinal absorption.[44] UDCA also protects cell membranes by decreasing the toxic effects of bile acids.[45] The overall effect is to increase the concentration level at which the saturation of cholesterol occurs by 40-60%. The patient shall be followed up even after stone dissolution due to the possibility of recurrence. Extracorporeal shock wave lithotripsy also proves beneficial for patients in a few studies. Its conjunction with UDCA shows promising results. Awaiting FDA approval for pediatric use, UDCA as an extemporaneous liquid formulation could be adapted.[46,47]

Surgical intervention

Cholecystectomy is an indication for symptomatic cholelithiasis as well as asymptomatic cholelithiasis persisting beyond a 12-month period of observation.[48] In these situations, ERCP appears promising, crediting its proven safety and efficacy in the pediatric population.[38] The possibility of pancreatitis, a well-known complication of the procedure, makes exploration of the common bile duct the superior approach.[49] Pediatric laparoscopic cholecystectomy is safe and effective and now a frequent procedure.[50] The data highlighting the incidence of post cholecystectomy syndrome in pediatric population, unlike the adult, is scarce. Researchers have proposed a grading system to evaluate gallbladders during surgery, taking into account factors such as the anatomical state of the gallbladder, the characteristics of the cystic pedicle, and the extent of surrounding adhesions.[51] Indocyanine green fluorescence imaging in difficult cases is used to enhance the Calot's triangle visualisation to minimize iatrogenic injury.[51,52] Subtotal cholecystectomy in cases where total cholecystectomy is contraindicated, is a safe and viable option.[54,55] Based on the limited research and the rarity of this condition in the past, the formulation of an accurate standardised general management algorithm is difficult.

Preventive imperatives

Prevention is a core tenet of pediatric hepatology. From combatting childhood obesity or prudent use of antibiotics, especially ceftriaxone, the need is critical and paramount. Establishing screening protocols for high-risk groups (e.g., hemolytic anemia) should be standardized throughout the country. In a developing country like India, nutrition-focused interventions, from the grass root levels can promise modulation in bile composition. Despite increasing recognition, pediatric cholelithiasis research

lags behind its adult counterpart to generate evidence-based guidelines. Ultimately, prevention hinges on upstream public health initiatives, not downstream clinical triage.

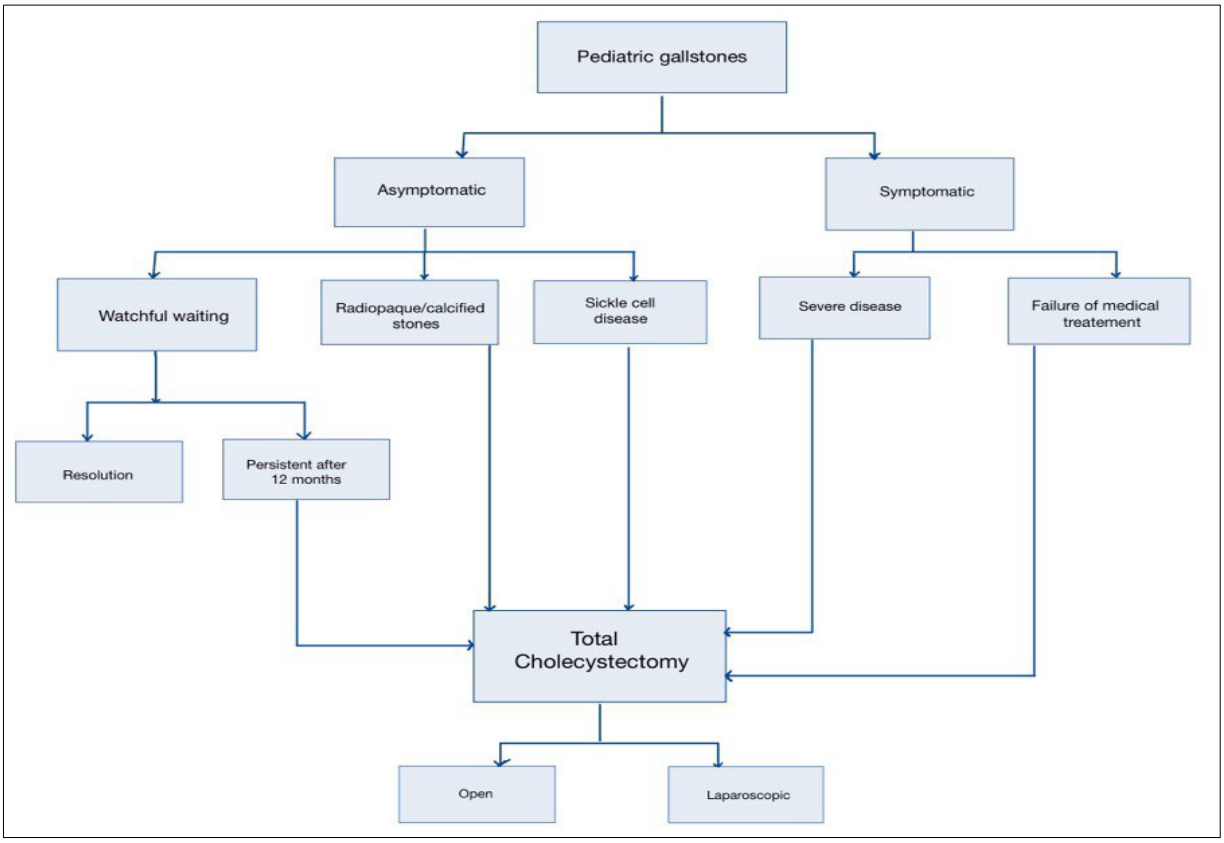

Figure 2: Represents suggested management algorithm of gallstones in children.

SUMMARY

Pediatric cholelithiasis, once rare, is now increasingly diagnosed due to rising obesity, sedentary lifestyles, and improved imaging. Current prevalence approaches all pediatric age groups, including neonates. Etiology extends beyond the classic triad. Pathogenesis follows the super saturation of bile, impaired gallbladder motility, and crystal nucleation, modified by pediatric-specific factors such as immature bile acid metabolism in infancy and hormonal influences in adolescence. Presentations range from incidental findings to acute biliary colic; infants often display nonspecific symptoms. Ultrasonography remains the diagnostic mainstay, with MRCP and ERCP reserved for complex cases. Management varies from observation in asymptomatic patients to cholecystectomy for symptomatic or persistent disease. Given the absence of standardized protocols, targeted guidelines are essential to address this increasingly prevalent, distinct pediatric hepatobiliary condition.

LIMITATIONS

The potential for publication bias cannot be overlooked, as studies reporting significant or favourable outcomes are more likely to be published and thus included. Furthermore, heterogeneity in study designs, populations, and diagnostic criteria across literature may limit the generalizability of findings. The scarcity of previous research constrains the robustness of the conclusions. Although comprehensive efforts were made to include recent and pertinent studies, it is possible that some emerging evidence and evolving clinical practices were not fully captured at the time of writing.

CONCLUSION

Pediatric cholelithiasis is no longer an esoteric diagnosis; it is a clinical reality which is constantly evolving in therapeutic complexity. As the lifestyle patterns shift and the diagnostic capabilities of medical science expand, clinicians must pivot towards a more nuanced understanding of this condition. The future of healthcare will depend on a triad of anticipatory screening, individualized therapy, and proactive prevention.

This is not merely a miniaturized version of adult cholelithiasis. It is a distinct entity, deserving of its own scientific and clinical identity.

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