

Acute cholecystitis in a child with scarlet fever: A rare association

Y Parvez, MBBS, MD, MRCPCH, MRCPS, DCH (UK); S Thomas, MBBS, MD

Department of Pediatrics, Dubai Hospital, United Arab Emirates

Corresponding author: Y Parvez (dryparvez@gmail.com)

Group A streptococcal infection is common in children; however, scarlet fever is now considered rare except for isolated outbreaks. One of the rarest complications of scarlet fever is acute cholecystitis – very few cases have been reported in the literature. A 5-year-old boy was admitted with scarlet fever complicated by acute cholecystitis. Clinical examination along with ultrasound of the abdomen confirmed the diagnosis of acute cholecystitis associated with scarlet fever. The patient was managed conservatively with broad-spectrum antibiotics and was discharged home successfully. Acute cholecystitis should be suspected as a rare complication of scarlet fever presenting with an acute abdomen.

S Afr J Child Health 2016;10(3):188-189. DOI:10.7196/SAJCH.2016.v10i3.1088

Scarlet fever is a syndrome characterised by exudative pharyngitis, fever and bright-red exanthem. It is caused by toxin-producing Group A β -haemolytic streptococcus. Otitis media, bronchopneumonia, meningitis, renal failure and hepatitis are some of the complications described in the literature. Acute cholecystitis is the rarest complication of scarlet fever – very few cases have been reported so far. We report a classic case to highlight this important complication and its management. A 5-year-old, previously healthy boy was admitted to our hospital with complaints of high-grade fever and scarlatiniform rash of 3 days' duration. On examination, the child was mildly icteric with a strawberry tongue, exudative pharyngitis and circumoral pallor. Besides the typical rash suggestive of scarlet fever, other systemic examination was unremarkable. His initial laboratory work-up showed: leukocytosis (14 300 cells/ μ L) with neutrophilia (76%); C-reactive protein (CRP) 57 mg/L; total bilirubin 3.8 mg/dL; direct bilirubin 1.6 mg/dL; alanine transaminase (ALT) 161 IU/L; aspartate transaminase (AST) 104 IU/L; gamma glutamyl transpeptidase (GGT) 24 IU/L; alkaline phosphatase (ALP) 355 IU/L; albumin 3.8 g/dL; prothombin time (PT) 12.9 s; activated partial thromboplastin time (APTT) 36.2 s; international normalised ratio (INR) 1.1; urine and stool examinations were normal. Serial antistreptolysin O titres showed a rising trend, eventually rising to 540 IU/ μ L from 220 IU/ μ L. Anti-DNase B blood test was not done owing to the unavailability of kit in the laboratory. The child's throat culture was positive for Group A β -haemolytic streptococcus. He was started on intravenous cefuroxime along with oral chlorpheniramine and emollients for local application. Two days after admission, the child developed abdominal pain in the right upper quadrant, associated with mild fever. On examination there was tenderness in the right upper quadrant of his abdomen. His amylase was 35 IU/L, lipase 22.6 IU/L, full blood count 16 800/ μ L (neutrophils 65%) and CRP 74 mg/L. Virology studies including Epstein-Barr virus (EBV, hepatitis A, B and C, cytomegalovirus (CMV) and HIV were normal. Urgent ultrasound of the abdomen was done, showing a mildly distended gallbladder with wall thickening and intraluminal sludge, likely representing acalculous cholecystitis (Fig. 1). The child was examined by a paediatric surgeon who advised management with intravenous cefuroxime, fluids and analgesics. His abdominal pain and fever gradually settled and he became asymptomatic after 10 days of treatment. The investigations that were repeated before discharge showed: white blood cell count 9 400 cells/ μ L (neutrophils 52%, lymphocytes 47%, Hb 10.4 g/dL); CRP 10 mg/L; total bilirubin 1.8 mg/dL; direct bilirubin 0.2 mg/dL;



Fig. 1. Ultrasound scan of the abdomen showing distended gallbladder with mucosal thickening with intraluminal biliary sludge.

ALT 32 IU/L; AST 28 IU/L; GGT 15 IU/L; ALP 355 IU/L; albumin 3.9 g/dL; PT 12.2 s, APTT 34.3 s; INR 1. His blood, urine and stool cultures were negative. The ultrasound was repeated 2 weeks after discharge and it was normal. The child is being followed up and he is doing fine with no complications.

Discussion

Group A streptococci are extracellular, Gram-positive pathogens responsible for pharyngitis, impetigo, scarlet fever, rheumatic fever and acute glomerulonephritis. Scarlet fever, which is rare nowadays owing to frequent antibiotic prescriptions in children, is caused by a toxin-producing Group A β -haemolytic streptococcus found in secretions from the nose, throat, ears and skin.^[1] The characteristic clinical features are exudative pharyngitis, fever and bright red exanthema. Otitis media, pneumonia, septicaemia, osteomyelitis, rheumatic fever and acute glomerulonephritis are the common complications associated with scarlet fever. However, hepatitis and vasculitis are other rare complications described in the literature.^[2-4] Acute cholecystitis as a complication of typhoid and various viral infections such as Epstein-Barr virus, CMV and hepatitis A and B has been well described in the existing literature; however, the association of scarlet fever with acute cholecystitis has rarely been

CASE REPORT

reported and its pathogenesis is not clear.^[5-7] It has been postulated that the streptococcus enters the gall bladder through the mucous membrane of the stomach by way of lymphatic channel producing toxin and lymphocytic infiltration responsible for the lesion in the gallbladder as well in the liver.^[8] Patients with acute cholecystitis due to scarlet fever usually present with fever, right upper abdominal pain, vomiting and jaundice. The diagnosis is based on clinical manifestation, laboratory investigations (leukocytosis, abnormal liver function tests) and ultrasonographic findings (gallbladder distension, gall bladder wall thickness (>3.5 mm), non-shadowing echogenic materials or sludge, and pericholecystic fluid collections); however, these may sometimes be ambiguous and confusing in children.^[8] The management is usually non-operative although cholecystectomy may be required.^[7,8]

Conclusion

Scarlet fever is usually a benign disease, but may develop serious complications. Acute cholecystitis should be suspected in a child presenting with an acute abdomen associated with scarlet fever. Urgent

ultrasound of the abdomen, along with laboratory investigations, is warranted to diagnose acute cholecystitis.

References

1. Cunningham MW. Pathogenesis of group A streptococcal infections. *Clin Microbiol Rev* 2000;13(3):470-511.
2. Gidaris D, Zafeiriou D, Mavridis P, Gombakis N. Scarlet fever and hepatitis: A case report. *Hippokratia* 2008;12(3):186-187.
3. Reddy UP, Albini TA, Banta JT, Davis JL. Post-streptococcal vasculitis. *Ocul Immunol Inflamm* 2008;16(1):35-36. DOI:10.1080/09273940701799163
4. Sandrini J, Beucher AB, Kouatchet A, Lavigne C. [Scarlet fever with multisystem organ failure and hypertrophic gastritis]. *Rev Med Interne* 2009;30(5):456-459. DOI:10.1016/j.revmed.2008.07.010
5. Gora-Gebka M, Liberek A, Bako W, et al. Acute acalculous cholecystitis of viral etiology—a rare condition in children? *J Pediatr Surg* 2008;43(1):e25-27. DOI:10.1016/j.jpedsurg.2007.10.073
6. Gnassingbé K, Katakao G, Kanassoua KK, et al. Acute cholecystitis from typhic origin in children. *Afr J Paediatr Surg* 2013;10(2):108-111. DOI:10.4103/0189-6725.115033
7. Yasuda H, Takada T, Kawarada Y, et al. Unusual cases of acute cholecystitis and cholangitis: Tokyo Guidelines. *J Hepatobiliary Pancreat Surg* 2007;14(1):98-113. DOI:10.1007/s00534-006-1162-9
8. Imamoğlu M, Sarihan H, Sari A, Ahmetoğlu A. Acute acalculous cholecystitis in children: Diagnosis and treatment. *J Pediatr Surg* 2002;37(1):36. DOI:10.1053/jpsu.2002.29423