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Unusual Cause of Gall Stone and Cholecystitis in An Adolescent

Sujatha Chinnappan*

Department of Pediatrics, Kovai Medical Centre and Hospital, India

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***Corresponding author:** Sujatha Chinnappan, Department of Pediatrics, Kovai Medical Centre and Hospital, Coimbatore, Tamil Nadu, India, E-mail: drsujathachinnappan@kmchhospitals.com

ABSTRACT

Fascioliasis is caused by Fasciola hepatica or Fasciola gigantica which are transmitted by ingesting raw contaminated freshwater plants. Sheep and cattle are definite hosts with snails being intermediate and humans are incidental hosts1. The clinical manifestations are divided into two phases. Acute hepatic phase occurring 6 to 12 weeks of ingestion with abdominal pain, fever, anorexia, loss of appetite and weight, myalgia and marked eosinophilia. Chronic hepatic phase starts from about 6 months to up to 10 years or more. Most are asymptomatic but can develop common bile duct obstruction, gall bladder stones, cholangitis, biliary cirrhosis and sclerosing cholangitis.

Keywords: Gall stone, Cholecystitis, Adolescent, Abdominal pain

1. Case Study

A 13-year-old boy presented with abdominal pain, low grade fever and non- bilious vomiting. Examination showed right upper quadrant tenderness, hemodynamic stability with other systemic examinations being normal. Investigations: WBC 19,000 cells/cu mm (polymorphs 74%, eosinophils 8%); CRP 100mg/dl; normal liver function test (LFT) and negative hepatitis serology (A, B and E). Ultrasound abdomen showed partially distended gall bladder with diffuse thickening of the wall, calculus of 1.5 cm and significant peri portal lymphadenopathy. Clinical features were fitting with diagnosis of cholecystitis, hence underwent laparoscopic cholecystectomy. Nodules over the right lobe of liver were noted.

Histopathology of the nodules showed features of Fascioliasis. Stool complete analysis showed eggs of both Fasciola hepatica and Ascaris lumbricoides. He was commenced on Nitazoxanide and Albendazole. After 2 days of treatment, developed jaundice, upper abdominal pain, recurrent non-bilious vomiting and tender hepatomegaly was present. Investigations showed increased inflammatory markers (WBC counts 34000 cells/cu mm, CRP 89 mg/dl, increased total bilirubin 5.73mg/dl with predominant conjugated hyperbilirubinemia (5.35mg/dl) and elevated liver enzymes (AST 91U/L, ALT 219U/L). In view of history fitting with cholangitis, was started on broad spectrum antibiotics. MRI with MRCP showed abrupt narrowing at proximal common hepatic duct and features of cholangitis. ERCP showed biliary stricture and common bile duct stenting was done. Gradually improved clinically and biochemically with hyperbilirubinemia/transaminases resolving. On follow up, he gained weight with normalizing LFT.

Fascioliasis is caused by Fasciola hepatica or Fasciola gigantica which are transmitted by ingesting raw contaminated

freshwater plants. Sheep and cattle are definite hosts with snails being intermediate and humans are incidental hosts[1]. The clinical manifestations are divided into two phases. Acute hepatic phase occurring 6 to 12 weeks of ingestion with abdominal pain, fever, anorexia, loss of appetite and weight, myalgia and marked eosinophilia. Chronic hepatic phase starts from about 6 months to up to 10 years or more. Most are asymptomatic but can develop common bile duct obstruction, gall bladder stones, cholangitis, biliary cirrhosis and sclerosing cholangitis[2].

Diagnosis is based on the clinical picture, detection of eosinophilia, abnormal LFT, elevated ESR, typical findings on ultrasound or computed tomography scans and identification of Fasciola eggs in stool and duodenal or biliary aspirates[3]. It is treated with Triclabendazole or Nitazoxanide. Our patient presentation fits with chronic hepatic phase and symptoms due to cholecystitis followed by cholangitis improving with treatment.

2. Conclusion

Fascioliasis is treated with Triclabendazole or Nitazoxanide. Our patient presentation fits with chronic hepatic phase and symptoms due to cholecystitis followed by cholangitis improving with treatment.

3. References

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