Incidental Finding of Gallbladder Cholesterolosis in an Infant with Rotavirus Infection and Transaminitis

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Abstract

Gall bladder diseases are relatively less prevalent in pediatrics compared to the adult population. With the use of ultrasound, more and more pediatric cases of gall bladder diseases are being reported. This is a case of an infant with Rotavirus gastroenteritis who was incidentally found to have cholesterolosis of gall bladder on abdominal ultrasound.

Keywords: Gall bladder; Cholesterolosis; Infant

Introduction

Gall bladder diseases continue to remain a less common condition reported in pediatric population compared to adults. Although many different studies have been reported, there is still insufficient information about the frequency of Cholesterolosis of gall bladder detection in children [1]. Here, we present a case of a 52 days old male, admitted with severe hypernatremic dehydration secondary to diarrhea and vomiting, who was reported to have cholesterolosis of gall bladder as an incidental finding on abdominal ultrasound.

Case Report

A 52 day old male presented to ED with 2 days of multiple episodes of non-bloody, non-bilious vomiting and watery diarrhea. Sick contacts included a brother with diarrhea. Denied any fever at home, runny nose or cough. The patient had a recent hospitalization to pediatric floor due to fever, with negative sepsis work up and treated with 4 days of IV antibiotics.

On presentation to ED, the patient was afebrile, tachpneic, tachycardeic with dry mucous membranes. Other than small reducible umbilical hernia, the rest of the physical examination was unremarkable. Labs done in ED were remarkable for Leukocytosis (WBC: 22.1), hypernatremia (Na: 155), Hyperkalemia K: 7.7, hyperchloremia (Cl: 134), metabolic alkalosis (CO₂: 6). Transaminitis with AST/ALT of 66/60 and total/direct bilirubin of 0.6/0.3. Urine and blood culture were drawn. Abdominal X-ray done in the ED was negative for any obstruction or gross morphologic abnormality.

Abdominal ultrasound done to rule out hypertrophic pyloric stenosis was negative but was remarkable for an incidental finding of cholesterolosis of gall bladder demonstrated by multiple small non-shadowing echogenic speckles in the mildly thickened gallbladder lumen (Figure 1).

An EKG was also done due to hyperkalemia, which was reported as unremarkable. The patient received two normal saline boluses in the ED and then was referred for admission to the pediatric intensive care unit due to severe hypernatremic dehydration secondary to acute gastroenteritis.

Figure 1: Non-shadowing, echogenic speckles in gall bladder lumen.

In the unit, the patient was started on continuous cardiorespiratory monitoring with Intravenous hydration and kept nil by mouth. Gastroenterology team was consulted to discuss the findings on abdominal ultrasound, which recommended cholesterolosis seems highly unlikely to be the underlying cause of the acute symptoms leading to dehydration.

Also, recommended to send stool for infectious work up including Rotavirus antigen studies. Repeat liver function tests in the unit revealed an increase in AST/ALT to 75/110 with bilirubin (total/direct) of 0.3/0.1. During the course of stay in the hospital, the patient's oral intake was gradually advanced and Intravenous fluids adjusted accordingly. Vomiting and diarrhea resolved over the course of time with almost complete resolution by 4th day of hospitalization.

Rotavirus antigen in the stool came back positive and the case was reported to state health department. The patient was discharged on 4th day of hospitalization after resolution of symptoms. A follow-up with the primary doctor and gastroenterology was arranged.
Discussion

Cholesterolosis of the gall bladder was first described by Moynihan and called "strawberry gall bladder" by MacCarty. Histologically, cholesterolosis is defined by the accumulation of lipids mainly cholesterol ester and triglycerides in the gall bladder mucosal wall [2].

The etiology has not been clearly defined. A positive correlation with high serum cholesterol level has been noted in some studies [3]. Multiple pathogenesis mechanisms have been proposed and studied including the biliary cholesterol supersaturation, motility defects and shorter cholesterol crystal nucleation time [4,5]. Similar to the other gall bladder disorders, most of the patients remain asymptomatic for long periods of time unless associated with other conditions like gall bladder stones. When symptomatic, the patients usually present with right upper quadrant pain, epigastric discomfort and vomiting.

Ultrasound continues to remain the imaging of choice for gall bladder diseases. Most of the times cholesterolosis continues to remain stable, however occasional cases of malignant transformation have been reported [4]. Due to potential for turning malignant, this condition needs a vigilant follow-up and surgery may be indicated if high suspicion for malignancy [6].

Conclusion

Gall bladder diseases are on a rising trend in pediatric population which in most part, seems to be related to the use of ultrasound. Most cases are asymptomatic and only reported as an incidental finding like in our patient. The increasing frequency of gall bladder diagnosis in pediatric population especially infancy brings to light, the need of rigorous study of pathogenesis for these conditions and also a close follow up after diagnosis, considering the potential for malignant transformation.

References

1. Kharitonova LA, Kosareva TM, Kochetova EA, Shakarian KA (2010) Clinical and morphological features of gall bladder cholesterolosis in children. Eksp Klin Gastroenterol 1: 20-24.
2. Wantanabe F, Hanai H, Kaneko E (1998) Increased acyl CoA-cholesterol ester acyl-transferase activity in gall bladder mucosa in patients with gall bladder cholesterolosis. Am J Gastroenterol 93: 9.
3. Khairy GA, Guraya SY, Murshid KR (2004) Cholesterolosis: Incidence, correlation with serum cholesterol level and the role of laparoscopic cholecystectomy. Saudi Med J 25: 9.
4. Satoh H, Koga A (1997) Fine Structure of cholesterolosis in the human gall bladder and the mechanism of lipid accumulation. Microsc Res Tech 39: 14-21.
5. Sandri L, Colecchia A, Larocca A, Vestito A, Capodicasa S, et al. (2003) Gall bladder cholesterol polyps and cholesterolosis. Minerva Gastroenterol Dietol 49: 217-224.
6. Sahlin S, Stahleberg D, Einarsson K (1995) Cholesterol metabolism in liver and gall bladder mucosa of patients with cholesterolosis. Hepatology 21: 1269-1275.