Acute Acalculous Cholecystitis in a Child with Primary EBV Infection

Lina Hadj Smaine, D.O., Viet Trinh, D.O., Gabrielle A. Balan, D.O.
Department of Family Medicine and Pediatrics, Riverside University Health System

INTRODUCTION

Acute acalculous cholecystitis (AAC) is rarely associated with Epstein Barr Virus (EBV) infection with majority of cases occurring in the adult population. AAC is typically managed conservatively in the pediatric population due to AAC not being the primary problem, but the consequence of an underlying disease etiology. Our report focuses on a 6-year-old female who presented with abdominal pain in the setting of URI symptoms and was diagnosed with EBV associated AAC.

CASE PRESENTATION

A 6-year-old female was admitted with a 4-day history of progressively worsening epigastric abdominal pain associated with 1-day history of fever (101 F). She also complained of 4 days history of sore throat, nasal congestion, itchy eyes and swollen eyelids. She had visited a local urgent care clinic prior to presentation and was prescribed with Amoxicillin-Clavulanate for bronchitis. She had no history of abdominal trauma, abdominal surgery, hepatitis or autoimmune conditions.

Physical Exam:
Vital Signs: BP 90/75, HR 100, RR 20, Temp. 98.3 F
General: + fatigued.
EENT: + swelling of eyelids, +rhinorrhea. - Scleral icterus. - Pharynx injection; - bilateral tonsillar hypertrophy
Cardiopulmonary: Regular rhythm, Clear to auscultation bilateral lungs
Abdomen: + epigastric abdominal tenderness to palpation. Liver and spleen were 3 and 2 fingerbreadths beneath the ribcage, respectively. Soft and non-distended. Murphy's sign was negative.

Significant Labs: Leukocytosis with >50% lymphocytic predominance and thrombocytopenia, elevated liver enzymes and total bilirubin. Mono Test +. Smooth muscle antibody + with 1:80 titer. EBV panel showed elevated viral capsid antigen (VCA) IgM > 160.0 U/mL and EBV DNA PCR 261,252 copies/mL. Of note, VCA-IgG and EBA IgG were negative (Table 1).

Pertinent Negative Labs: viral hepatitis panel, cytomegalovirus IgM, HSV IgM, HIV Ag/Ab, ANA, Anti liver-kidney IgG, and ferritin level. A urine drug screen and Acetaminophen toxicity level were both unremarkable.

Imaging: Abdominal CT and ultrasound showed gallbladder wall thickness 6.9 mm, common bile duct at 2.3 mm, pericholecystic fluid, and absent gallstones. Abdominal CT showed hepatosplenomegaly (Fig.1 and Fig.2).

LABORATORY RESULTS

Table 1.

Hospital Day	Day 1	Day 2	1 Week Post Discharge
Platelets	125	118	381
PT/INR	14.6/1.2	15.5/1.3	11.2/1.0
AST/ALT	189/347	17/289	56/80
Total bili/direct bili (mg/dL)	1.6/1.1	0.8	0.4
Gamma-glutamyltransferase (U/L)	263		75

IMAGING

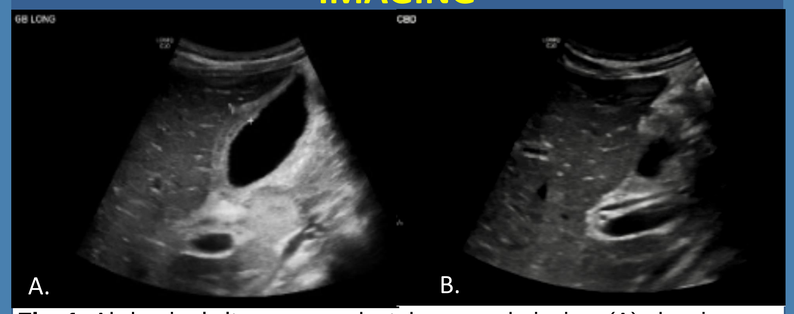

Fig. 1. Abdominal ultrasonography taken on admission. (A) showing gallbladder thickening of 6.9 mm without any definite echogenic stone and pericholecystic fluid, (B) showing common bile duct measures 2.3 mm in porta hepatis.

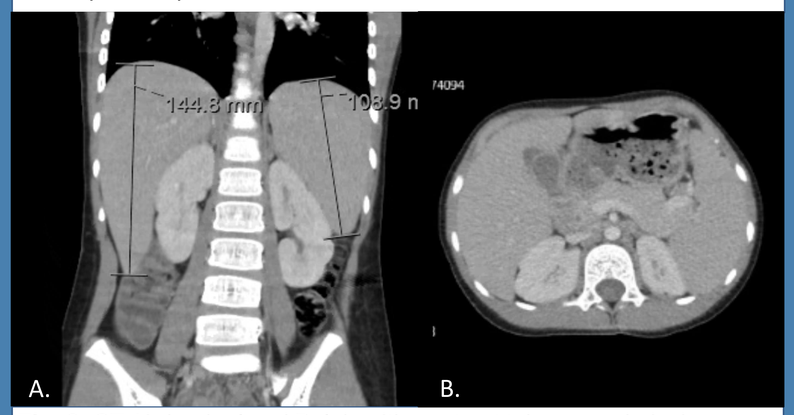

Fig. 2. CT Abdominal and Pelvis with contrast taken on admission. (A) shows hepatosplenomegaly; (B) shows pericholecystic fluid, gallbladder with no echogenic stones.

FINAL DIAGNOSIS/TREATMENT COURSE

Her abdominal pain was attributed to Acute Acalculous Cholecysitis (AAC) due to primary EBV infection.

- **Primary EBV infection:** elevated VCA-IgM and EBV DNA PCR.
- **AAC:** 2 major criteria on imaging (gallbladder wall thickness > 3.5mm and pericholecystic fluid) and absent gallstones.

She was managed conservatively, and her abdominal pain resolved 2nd day of admission at which point the patient was discharged home. She was given precautions not to participate in strenuous activities or contact sports given hepatosplenomegaly. Thrombocytopenia and elevated transaminases were monitored by her Pediatrician which had resolved 1-week post hospital discharge (Table 1).

DISCUSSION

To date, only 12 cases of EBV-associated AAC have been reported. AAC has also been attributed to infectious etiologies such as viral (EBV), bacterial (*Salmonella*) and parasitic (*Ascaris lumbricoides)* as well as systemic diseases including Kawasaki's, Hemophagocytic Lymphohystiocytosis, Leukemia and Autoimmune Diseases.

Patients often present with generalized symptoms (eg. fever, weight lost, night sweats) in addition to symptoms of cholecystitis.

LEARNING POINTS

- Acute Cholecystitis is a rare disease in the pediatric population. Its incidence in infancy, childhood and adolescence has been reported to be between 0.15% and 0.22%
- Physicians and allied health professionals who care for children should be aware that AAC attributable to EBV is a diagnosis of exclusion.
- Pediatric patients with incidental findings of AAC may benefit from additional workup to rule out other underlying causes, such as, but not limited to Kawasaki's, Hemophagocytic Lymphohystiocytosis, Macrophage Activating Syndrome, Malignancy, Autoimmune Disease and HIV.
- Unlike surgical management of AAC in the adult population, AAC is managed conservatively in the pediatric population.

DIFFERENTIAL DIAGNOSIS

Viral hepatitis (CMV, HSV, HIV, EBV)
Autoimmune hepatitis
Hemophagocytic Lymphohistiocytosis, Drug toxicity, Sepsis and Malignancy

REFERENCES

- Lobe T, Cholelithiasis and Cholecystitis in Children,Seminars in Pediatric Surgery, Volume 9, Issue 4, 2000; 170-176
- Poddighe D,, Sazonov V, Acute acalculous cholecystitis in children, World J Gastroenterol. Nov 21, 2018; 24(43): 4870-4879
- Tsakayannis DE, Kozakewich HP, Lillehei CW.,Acalculous cholecystitis in children. J Pediatr Surg. 1996 Jan;31(1):127-30; discussion 130-1.
- Kim A, Yang HR, Moon JS, Chang JY, Ko JS. Epstein-barr virus infection with acute acalculous cholecystitis. Pediatr Gastroenterol Hepatol Nutr. 2014;17(1):57–60. doi:10.5223/pghn.2014.17.1.57