

CASE REPORT**Hepatology**

Cholecystitis due to gallbladder volvulus in a child

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Abstract

Gallbladder volvulus (GV) involves the rotation of the gallbladder along its axis, resulting in torsion. This pathology is rare, more commonly found in elderly females, but can occasionally occur in pediatric patients. Diagnosis is challenging due to often atypical symptoms, with imaging and laboratory findings typically nonspecific. Prompt surgical intervention is necessary when GV is suspected to prevent significant systemic illness. Laparoscopic cholecystectomy has proven to be safe and effective in pediatric cases. In this report, we present a pediatric case of GV, initially misdiagnosed as viral-induced acalculous cholecystitis, which was effectively managed using laparoscopic cholecystectomy.

KEYWORDS

gallbladder ischemia, laparoscopic cholecystectomy, pediatric cholecystitis, viral-induced acalculous cholecystitis

1 | INTRODUCTION

Gallbladder volvulus (GV) refers to the rotation of the gallbladder, including the cystic duct and artery, along its axis, resulting in torsion. While it primarily affects elderly females, cases have been reported in pediatric patients as well.^{1,2} Its prevalence rate is approximately 1:350,000 hospitalizations and is associated with anatomic factors such as a long, mobile mesentery, hepatic atrophy, and loss of visceral adiposity.^{1,3} The etiology in pediatric cases remains unclear. Diagnosis poses a challenge despite advanced imaging modalities, often necessitating operative intervention for confirmation. The differential diagnosis typically includes acute cholecystitis, acalculous cholecystitis, and hydrops of the gallbladder. In this report, we present the case of an 11-year-old with GV whose non-specific symptomatology created a diagnostic dilemma but ultimately necessitated laparoscopic cholecystectomy.

2 | CASE PRESENTATION

An 11-year-old female with mild intermittent asthma presented to the emergency room with acute, colicky abdominal pain. The pain started postprandially after a day of fasting in observance of a religious holiday. It was diffuse throughout the abdomen but most severe around the umbilicus. The pain was associated with nausea, nonbilious emesis, and nonbloody diarrhea. She was afebrile and hemodynamically stable. On physical examination, her abdomen was soft and nondistended, with mild diffuse tenderness, most pronounced in the epigastrium and right upper quadrant, accompanied by a positive Murphy's sign. Notably, she was anicteric, nonjaundiced, and did not exhibit any signs suggestive of Kawasaki disease. Laboratory studies revealed a viral panel positive for enterovirus, as well as leukocytosis ($15.8 \times 10^9/L$; reference range:

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4.5–13 × 10⁹/L) and elevated C-reactive protein (7.6 mg/dL; reference range: 0.0–0.9 mg/dL). Her hepatic transaminases, alkaline phosphatase, bilirubin, and gamma-glutamyl transpeptidase levels were all within normal ranges.

An abdominal sonogram was performed, which failed to visualize the neck of the gallbladder but depicted diffuse wall thickening and pericholecystic fluid, notably lacking gallstones or biliary dilatation (Figure 1). Subsequently, a computed tomography (CT) scan of the abdomen and pelvis with intravenous contrast was obtained, revealing a distended gallbladder without gallstones, biliary ductal dilatation, or pericholecystic fluid but with a small amount of hemorrhagic fluid in the pelvis.

Given the presence of a viral infection and absence of gallstones, the diagnosis of viral-induced acalculous cholecystitis (VIAC) was made. The patient was admitted to the hospital, and medical management was initiated, comprised of intravenous fluids, bowel rest, analgesia, and prophylactic intravenous antibiotics. Despite the suspected viral etiology, prophylactic antibiotics were administered to prevent further bacterial infection in the context of bile stasis and to mitigate the systemic effects of illness. Following admission, her pain began to improve, and she was gradually started on a diet. However, due to lingering uncertainty regarding the viral etiology of her cholecystitis, a magnetic resonance cholangiopancreatography (MRCP) with contrast was performed for further characterization of the biliary anatomy. The MRCP confirmed normal biliary anatomy alongside a hydroptic gallbladder without evidence of cholelithiasis or cystic duct stones (Figure 2).

Despite initial improvement, the patient's abdominal pain and tenderness worsened 4 days later, with no

progress observed with medical management. Laboratory studies repeated on hospital day four showed resolution of the leukocytosis with hepatic transaminases, alkaline phosphatase, and bilirubin levels remaining within normal ranges. Consequently, she underwent laparoscopic exploration and cholecystectomy.

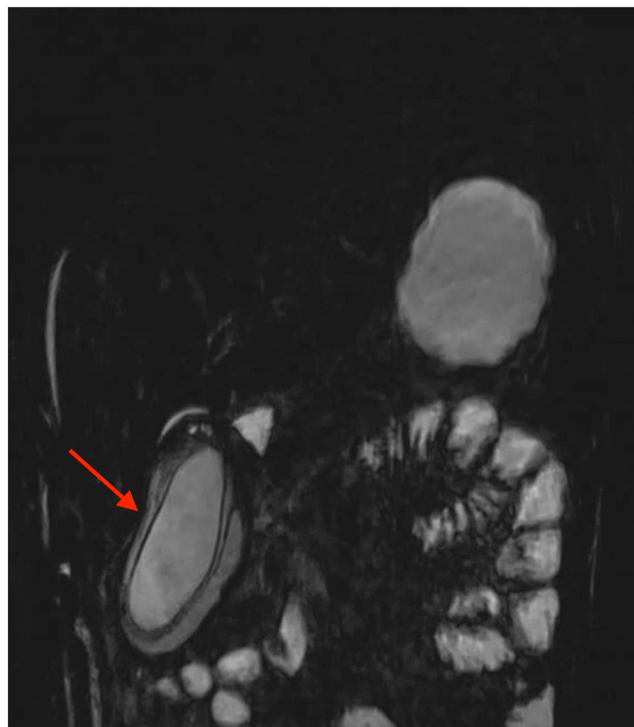


FIGURE 2 MRCP image demonstrating cholecystitis without cholelithiasis or discernable evidence of volvulus. MRCP, magnetic resonance cholangiopancreatography.

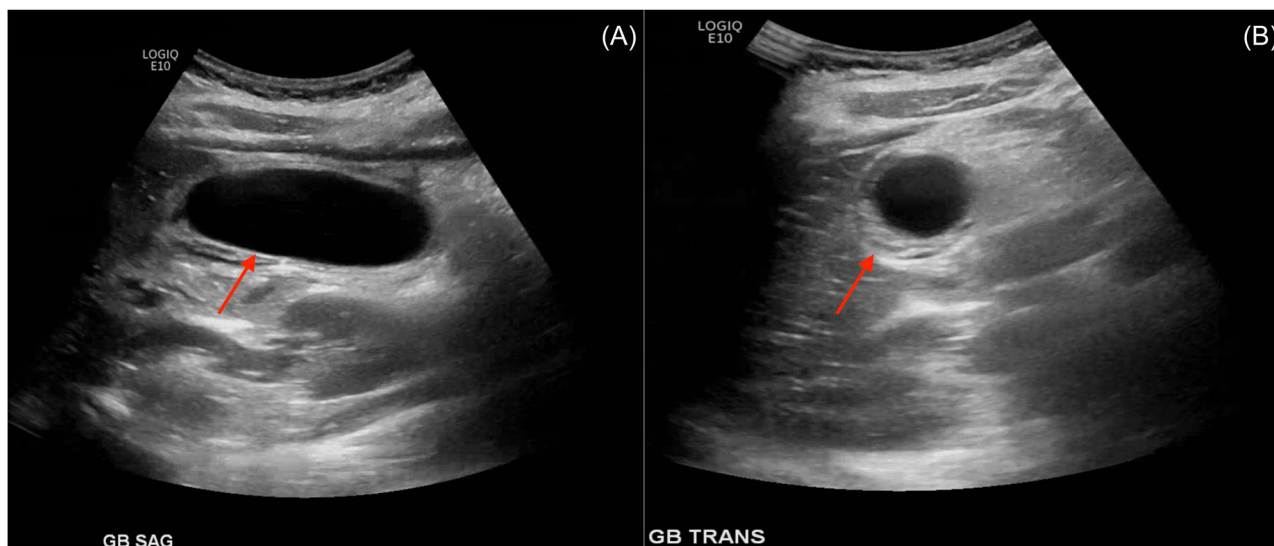


FIGURE 1 Sagittal and transverse sonographic images of the gallbladder. (A) Arrow emphasizes the absence of gallstones and shadowing. (B) Arrow highlights the edema and thickening of the gallbladder wall.

Intraoperatively, dense adhesive disease was encountered in the right upper quadrant, accompanied by considerable hemorrhagic ascites. The gallbladder appeared hemorrhagic, necrotic, and encased in dense inflammatory tissue. Upon dissection and mobilization, it was found to be volvulized, with the fundus enveloped by the liver. Detorsion revealed an area of demarcation at the neck of the gallbladder, with most of the tissue appearing necrotic except for a small portion proximal to the point of demarcation (Figure 3). Further examination revealed a clear absence of hepatic attachments, except for a few thin, fragile connections between the liver and cystic duct. A retrograde dissection of the cystic duct was performed to enable safe ligation and transection. The cystic artery was absent, having obliterated while torsing with the gallbladder. The patient recovered uneventfully postoperatively, and final pathology confirmed acute gangrenous cholecystitis (Figure 4).

3 | DISCUSSION

We present a rare case of GV in a child, a pathology scarcely reported in the pediatric literature. The volvulization was facilitated by a congenital lack of hepatic attachments and over-distention after fasting, possibly worsened by concurrent enterovirus. Diagnosis posed a unique challenge due to the nonspecific symptomatology, and diagnostic imaging that failed to depict any

evidence of volvulization. As often seen in such cases, laparoscopic cholecystectomy confirmed the diagnosis and provided definitive therapy.

GV can manifest as incomplete or complete volvulization, occurring due to clockwise or counterclockwise rotation.⁴ In pediatric cases, a congenital condition known as “floating gallbladder” has been described,

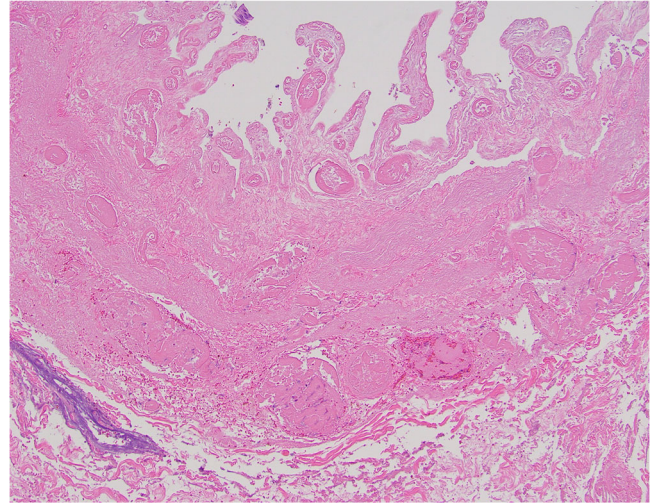


FIGURE 4 Representative histopathological image depicting gangrenous cholecystitis. In this image, the mucosal villi have become de-epithelialized with associated inflammatory infiltration.

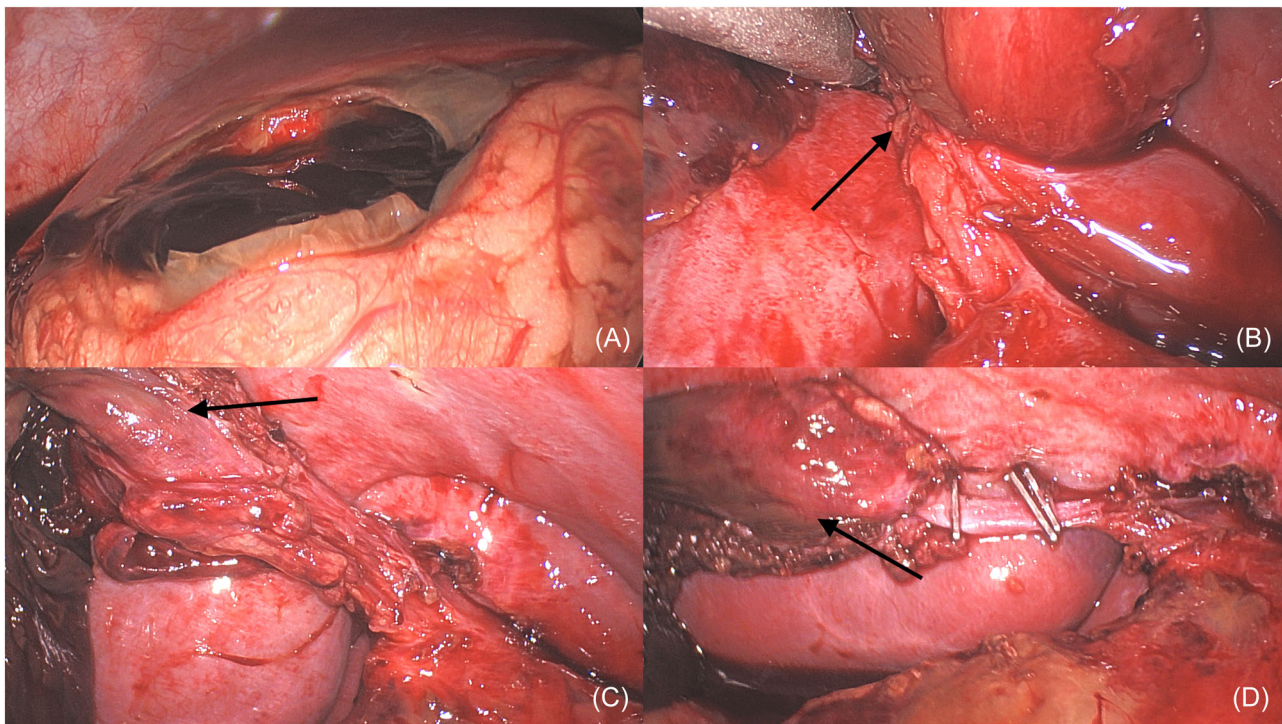


FIGURE 3 Intraoperative images during cholecystectomy. (A) Dense inflammatory tissue overlying necrotic gallbladder. (B) Arrow highlights area of gallbladder volvulization enveloped by liver. (C) Arrow identifies proximal extent of ischemia following devolvulization. (D) Successful ligation of the cystic duct, arrow emphasizing point of demarcation in the neck of the gallbladder.

characterized by limited attachments between the liver and proximal gallbladder.¹ In this instance, the complete lack of attachments represented a variant of this condition, predisposing the gallbladder to torsion. Excessive gallbladder distention following fasting likely further facilitated torsion around the cystic duct pedicle.

VIAC has been associated with Epstein–Barr, cytomegalovirus, and enterovirus infections in children.^{5,6} The pathophysiology of VIAC remains poorly understood but is thought to involve a complex mechanism of pro-inflammatory mediators, cholestasis, and reperfusion injury.⁵ Patients often present with nonspecific symptoms and lack specific clinical features, posing diagnostic challenges. In our case, the nonspecific presentation, absence of cholelithiasis, and concurrent viral illness led to the presumed diagnosis of VIAC. The patient's initial improvement with medical management further complicated the diagnosis. To our knowledge, there have been no previous reports of GV occurring in association with enterovirus infection.

Diagnosing GV is challenging due to the nonspecific clinical, imaging, and laboratory findings. The patient presented here had laboratory values and symptoms that were atypical for acute cholecystitis, notably diffuse abdominal pain and diarrhea, as well as a mild leukocytosis, elevated CRP, and normal liver function tests. The vague clinical symptoms of GV are attributable to gallbladder ischemia rather than gallbladder distention or focal peritoneal irritation from inflammation alone. In most cases, biochemical evidence of cholestasis or imaging demonstrating intrahepatic biliary dilatation will be lacking as torsion of the gallbladder results in bile being anatomically trapped within the gallbladder but its flow through the remainder of the biliary system unaffected. Sonogram findings are also typically nondiagnostic, as gallbladder wall edema and thickening also are found in acute cholecystitis.¹ CT imaging offers improved visualization and can demonstrate horizontal displacement of the gallbladder, a cystic duct located to the right of the gallbladder, or nonenhancement of the gallbladder wall.^{7,8} In some instances, “beak” and “whirl” signs have been identified as diagnostic findings which can be seen on sonogram or CT.⁸ An abrupt angulation of the gallbladder neck or “beak sign” is described as the tapering of the distended gallbladder lumen to the fulcrum point at the pedicle. Additionally, indrawing of the vascular pedicle with the surrounding fat at the gallbladder neck forms a “whirl sign” like that seen with intestinal volvulus. The use of doppler during sonographic evaluation can be used to further assess flow through the cystic artery when GV is suspected. Though, variability in artery size and sonographer ability to trace the artery throughout the length of the gallbladder infundibulum may limit the specificity of a finding of “no flow.” While magnetic resonance imaging (MRI) provides superior visualization of the biliary anatomy, its use remains

debated due to concerns about delaying definitive care.^{1–3,7} In a review by Moser et al., MRI was the least utilized modality but was the most successful diagnostically.³ In our case, none of the previously reported imaging signs consistent with GV were appreciated. However, our inability to visualize the neck of the gallbladder during sonographic evaluation represents a nonspecific finding that should prompt clinicians to consider GV. Unfortunately, despite high-quality imaging, definitive diagnosis is often made only at the time of surgery.

Prompt surgical intervention is crucial when GV is strongly suspected to prevent complications such as gangrenous cholecystitis, perforation, and resultant sepsis. Delayed diagnosis in adults has been associated with significant morbidity and mortality rates, 16% and 6% respectively.^{3,4} Before accurately diagnosing GV, patients may receive intravenous antibiotics, which may temporarily alleviate symptoms but ultimately fail to resolve them. This underscores the importance of considering alternative diagnoses, including GV. Operative intervention typically involves inspection, devolvulization, and cholecystectomy. Laparoscopic cholecystectomy has been shown to be safe and effective in children.² Alternative surgical approaches such as “top down” dissection may be utilized in these patients with such altered anatomy.

In conclusion, this report highlights a rare case of GV in a child with a congenital absence of gallbladder attachments. It underscores the importance of considering GV in children with atypical presentations, as preoperative diagnosis remains challenging despite advancements in imaging modalities. Surgery remains the definitive management, and urgent laparoscopic cholecystectomy should be performed when the diagnosis is strongly suspected or confirmed.

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CONFLICT OF INTEREST STATEMENT

The authors declare no conflict of interest.

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REFERENCES

1. Keeratibharat N, Chansangrat J. Gallbladder volvulus: a review. *Cureus*. 2022;14(3):e23362.
2. Kimura T, Yonekura T, Yamauchi K, Kosumi T, Sasaki T, Kamiyama M. Laparoscopic treatment of gallbladder volvulus: a pediatric case report and literature review. *J Laparoendosc Adv Surg Tech*. 2008;18(2):330-334.
3. Moser L, Joliat R, Tabrizian P, et al. Gallbladder volvulus. *Hepatobiliary Surg Nutr*. 2021;10(2):249-253.

4. Kerr L, White RZ, Au J. Gallbladder volvulus. *Surgery*. 2022;172(1):e1-e2.
5. Gora-Gebka M, Liberek A, Bako W, Szarszewski A, Kamińska B, Korzon M. Acute acalculous cholecystitis of viral etiology—a rare condition in children? *J Pediatr Surg*. 2008;43(1):e25-e27.
6. Simões AS, Marinhas A, Coelho P, Ferreira S. Acalculous acute cholecystitis during the course of an enteroviral infection. *BMJ Case Rep*. 2019;12(4):e228306.
7. Bergeron E, Désilets E, Do XV, McNamara D, Chergui S, Bensoussan M. A case of torsion of the gallbladder suspected with SPECT-CT: review and recommendations. *Case Rep Surg*. 2020;2020:8687141.
8. Hoshi R, Uehara S, Hosokawa T, Kaneda H, Koshinaga T. Gallbladder volvulus in two children: the importance of radiological features. *Pediatr Int*. 2022;64(1):e15260.

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