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Abdominal compartment syndrome associated with Norovirus infection

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ABSTRACT

Norovirus infection is a leading cause of infectious gastroenteritis and is typically self-limited. Abdominal compartment syndrome is rare in the pediatric population. To date, there have been no reports of abdominal compartment syndrome secondary to Norovirus infection. This patient is a 7-year old female who presented with abdominal compartment syndrome and fulminant sepsis attributed to acute Norovirus infection. The patient was successfully treated with decompressive laparotomy, delayed abdominal closure, and supportive therapy. The patient's post-operative course was notable for acute hepatitis and pancreatitis, which resolved without further intervention. The patient was discharged home after a prolonged hospital stay in good condition.

Norovirus is a leading cause of infectious gastroenteritis and is usually a self-limited disease. Abdominal compartment syndrome in the pediatric population is uncommon, and there are no reports of abdominal compartment syndrome associated with Norovirus infection.

1. Case report

The patient is a 7 year-old female born at 28 weeks gestation. Her early medical history is significant for open Nissen fundoplication in the first year of life for refractory gastroesophageal reflux. The patient was feeling well until the morning of presentation, at which time she began to experience nausea, retching without vomiting, abdominal pain and distention. Over a period of hours she progressed to lethargy, acute mental status changes, and cyanosis. Of note, three household members had overcome recent flu-like illnesses characterized by nausea, emesis, and lethargy.

The patient initially presented to an outside hospital unresponsive and in respiratory distress. Examination revealed tachycardia, a severely distended and firm abdomen, and diffuse cyanosis with mottling of her lower extremities. Blood pressures could not be measured. The patient was emergently intubated due to respiratory distress. Attempts at gastric decompression with placement of a nasogastric tube were unsuccessful due to tube coiling in the esophagus. Serum laboratory studies were notable for a leukocytosis of 18,000 k/uL, glucose of 439 mg/dL, acute transaminitis with AST 152 U/L and ALT 116 U/L, hyperlipasemia of 434 U/L, lactic acidemia of 12.36 mmol/L, and an arterial blood gas pH of 6.9. Chest and abdominal x-rays demonstrated massive gastric and small bowel distention (Fig. 1). Computed tomography imaging demonstrated similar findings without a transition zone and gas distributed within the distal rectum; a nasogastric tube was looped in the esophagus (Fig. 2). The patient was subsequently transferred to our institution for further management.

Surgical evaluation confirmed the above findings as well as lack of palpable pulses in the lower extremities. Attempts at repositioning and replacing an orogastric and nasogastric tube were unsuccessful. Due to concern for abdominal compartment syndrome (ACS), the patient was taken emergently to the operating room. Esophagogastroduodenoscopy (EGD) revealed a normal appearing esophagus, and intact Nissen fundoplication. There was a significant amount of dark succus within the stomach and there was evidence of gastric mucosal sloughing. A decompressive orogastric tube was successfully placed under endoscopic guidance. Due to persistent abdominal distention, an exploratory laparotomy was performed. Laparotomy revealed a small amount of clear

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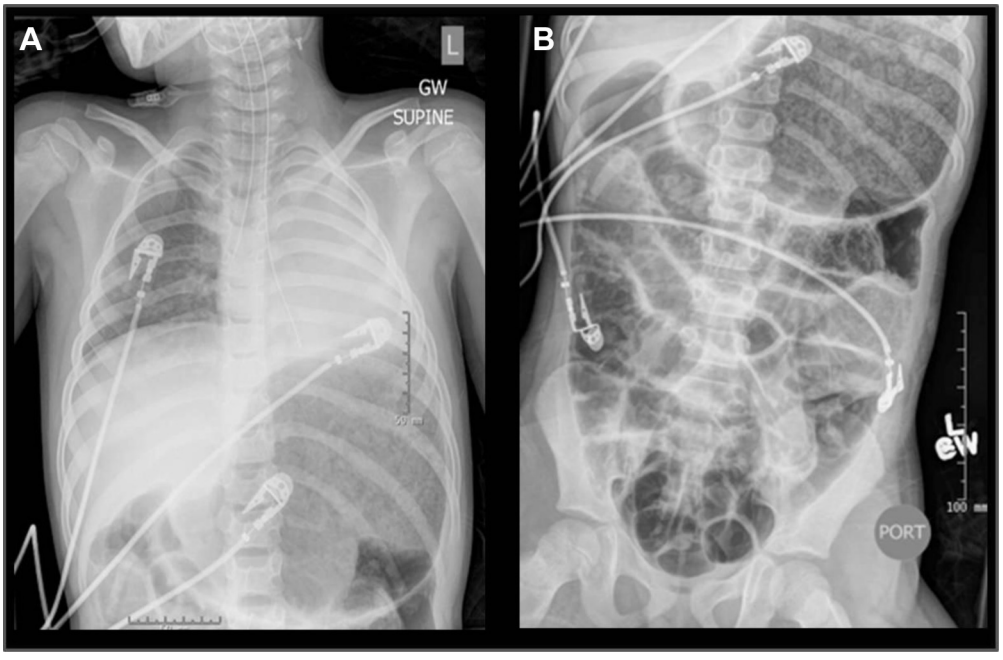

Fig. 1. Chest (A) and abdominal (B) radiographs demonstrate prominent gastric and bowel distention.

ascites without evidence of peritoneal contamination. The small bowel was markedly dilated and congested but without evidence of ischemia. Exploration of the abdomen revealed no additional abnormalities. The abdomen was temporarily closed with a modified negative pressure wound therapy dressing. The patient demonstrated immediate improvement in perfusion as evidenced by resolution of cyanosis and increased urine output.

The patient was maintained on mechanical ventilation post-operatively and admitted to the intensive care unit. She returned to the operating room on hospital day number two for a second look operation and repeat EGD. Endoscopy demonstrated congestion and erythema of the gastric mucosa with a large quantity of undigested food or tissue in the fundus. The gastric effluent was sent for cultures, and biopsies of the gastric mucosa and mass were obtained. Abdominal exploration revealed minimal serous ascites. The small bowel appeared viable but remained significantly dilated, precluding definitive closure. A liver biopsy was obtained due to worsening hepatitis and the abdomen was

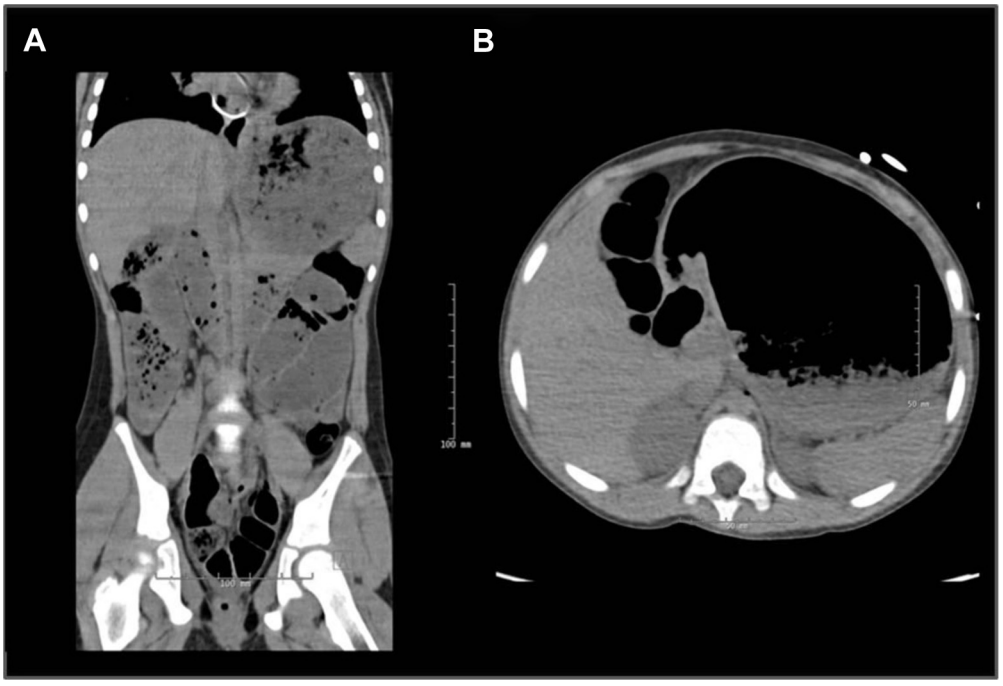

Fig. 2. Coronal (A) and axial (B) computed tomography images demonstrate prominent gastric and bowel distention. (A) also demonstrates intra-esophageal looping of the nasogastric tube.

again temporarily closed with a modified negative pressure device. Definitive abdominal closure was ultimately obtained on hospital day 6 during the patient's third operative exploration. The patient was subsequently extubated successfully.

The patient underwent extensive work-up to determine the cause of her severe enteritis. Blood testing did not find any culprit toxic agents. Infectious disease work-up, including shigella, salmonella, campylobacter, *Escherichia coli*, rotavirus, Hepatitis A and B, CMV, EBV, and adenovirus were negative. A stool sample obtained early in the patient's hospital course tested positive for Norovirus 2. Liver biopsy demonstrated non-specific portal inflammation without any infectious component. Stomach biopsy demonstrated reactive epithelial changes and lamina propria hemorrhage/edema, while the gastric effluent demonstrated Candida albicans. The patient developed acute hepatitis with elevation of AST and ALT to 4831 U/L and 3026 U/L, respectively, without evidence of elevated alkaline phosphatase or hyperbilirubinemia. She also developed pancreatitis, as evidenced by hyperlipasemia of 434 U/L. These abnormalities resolved during the patient's hospital course. The patient was ultimately discharged to home on hospital day number 41 in good condition. The patient's final diagnosis was acute Norovirus 2 infection causing abdominal compartment syndrome. The patient returned for outpatient follow-up at three and six months following her discharge. Gastric emptying studies performed at follow-up were normal. The patient had recovered fully and was without any identified sequelae from her disease process and interventions.

2. Discussion

Norovirus, an RNA virus, is amongst the most common causes of non-bacterial gastroenteritis. There are six recognized genogroups of Norovirus, three of which affect humans. Norovirus has been estimated to account for 47–96% of outbreaks of acute gastroenteritis and 5–36% of sporadic cases of gastroenteritis [1,2]. Among children less than 5 years of age, Norovirus is the second leading cause of gastroenteritis, eclipsed only by Rotavirus. In a 2009–2010 prospective study of US children less than 5-years of age for whom medical attention was sought due to gastroenteritis, Norovirus was detected in 21% of individuals with a rate of hospitalization of 7.2 per 1000 [2]. With improvements in Rotavirus vaccination, the prevalence of Norovirus as a cause for gastroenteritis appears to be increasing [3]. The highly contagious nature of Norovirus infection is in part due to its multiple modes of transmission, which include direct person-to-person fecal-oral route, ingestion of aerosolized vomit, and indirect transmission by means of contaminated surfaces, food, or water [4]. Additionally, the virus possesses a relatively low infectious dose, demonstrates prolonged stability on inanimate surfaces, and is relatively resistant to conventional cleaning agents.

Infection with Norovirus is more common during the winter months and commonly presents with symptoms that may include nausea, emesis, diarrhea, abdominal pain, malaise, anorexia, fevers, and chills [2]. Confirmation of infection is typically performed via real-time reverse transcription-polymerase chain reaction assay, which can be used on a variety of samples ranging from stool to vomitus, foods, water, and environmental specimens. The length of gastroenteritis is typically self-limited, lasting 1–3 days. Given the typical self-limited course of infection, treatment is non-specific. If treatment is instituted, it is directed at treating signs and symptoms of dehydration. Unlike Rotavirus, there is currently no vaccine for Norovirus.

Although infection with Norovirus is usually self-limited in healthy children, more severe disease courses have been reported. In two observational studies, Norovirus infection was found to be associated with benign seizures without any long-term neurologic sequelae [5–7]. Among premature infants infected with Norovirus, vomiting was less common and patients more frequently demonstrated symptoms of abdominal distention, apnea, or tachypnea requiring supplemental oxygen therapy; a sepsis-like appearance was also reported [8]. A more severe disease course has been observed in children with underlying medical conditions. In pediatric patients with inflammatory bowel disease (IBD), infection with Norovirus was associated with acute exacerbation of IBD, hematochezia, and prolonged viral shedding [9]. Similar results have been demonstrated in immunocompromised pediatric patients. In a prospective study of patients with inherited immune deficiencies, patients demonstrated prolonged viral shedding, with positive stool samples detected at a median of 9.5 months. Shedding was also associated with gastrointestinal symptoms and viremia in a significant proportion of subjects [10]. Interestingly, despite these reports of more severe disease courses following infection with Norovirus, there are no reports of associated abdominal compartment syndrome in the medical literature.

Abdominal compartment syndrome (ACS) is defined as a sustained elevation in intra-abdominal pressure (IAP) that results in end-organ damage, particularly to the cardiovascular, pulmonary and renal systems [11]. Untreated, mortality from ACS is almost certain. The intra-abdominal pressure is influenced by intraluminal abdominal volume and, in turn, determines abdominal perfusion pressure (APP, APP = mean arterial pressure − IAP). Recent updates issued by the World Society on Abdominal Compartment Syndrome recommend assessing IAP through the use of bladder catheters and advocate serial measurements in patients with risk factors for ACS and elevated intravesical pressures [12].

Similar definitions for ACS have been applied to pediatric patients (age < 18 years). However, ACS is poorly described in the pediatric population primarily due to its infrequency. An intra-abdominal hypertension threshold of 10 mm Hg has been suggested, but this number disregards the large variability in MAP and IAP within the pediatric population. Indeed, IAP as low as 4 mm Hg has been associated with ACS in critically ill children [12]. The physician must therefore demonstrate vigilance, relying on clinical judgment and on physical exam findings, in order to make an expedient diagnosis and to avoid delayed treatment. Findings suggestive of ACS include a tense abdomen, lack of distal pulses, and cyanotic extremities. The presentation may include other secondary effects, including wheezing, tachypnea, elevated ventilatory peak pressures, oliguria, low cardiac index, and other signs of hypovolemia. Further evaluation may be undertaken with laboratory analysis of lactate measurement, renal function tests, diagnostic imaging, and intra-abdominal pressure measurements.

Initial management should emphasize medical therapy to optimize fluid status, intra-viscus decompression, and increase of abdominal wall compliance. Although the incidence of ACS in pediatric intensive care unit patients has been reported to be between 0.6 and 4.9%, the observed mortality lies close to 60% [13]. Therefore, emergent decompression via laparotomy with the option of temporary abdominal wound closure is indicated. When possible, fascial closure should be attempted within five to seven days of laparotomy in order to reduce the associated mortality and morbidity. In a retrospective review of pediatric ICU patients, surgical decompression was performed within the first 24 h of admission with clinical improvement [14]. The authors encourage decompressive laparotomy for refractory oliguria, increased intra-abdominal pressures and ventilation pressures, lactic acidosis and vasopressor requirements. Similarly, an earlier prospective study confirmed that surgical intervention in pediatric ICU patients with IAP exceeding 15 mm Hg and accompanying kidney, lung or cardiovascular compromise yields immediate clinical benefit [15].

Although there is no identifiable cause for the severe course of Norovirus infection described herein, the authors suspect a role for the patient's previous Nissen fundoplication in exacerbation of the infection. Nissen fundoplication, a three hundred and sixty degree gastric fundus wrap around the gastroesophageal junction, serves to reinforce the function of the lower esophageal sphincter as a barrier to gastric reflux in the setting of GERD. Although Nissen fundoplication is widely regarded as safe and effective, it is associated with postoperative complications that include dysphagia, wrap disruption, paraesophageal hernia, and gas-bloat syndrome (GBS). GBS is a poorly defined condition characterized by symptoms that include nausea, early satiety, abdominal distention, and inability to belch and vomit. The ability of the stomach to vent itself may become impaired following fundoplication through a variety of proposed mechanisms including vagus nerve injury and a wrap that is too tight. This impairment is believed to result in GBS. In the adult population, the reported frequency of GBS following fundoplication varies widely from 1 to 85% [16]. In the pediatric population, GBS following fundoplication has been reported in several case series studies. However, its true frequency in this patient population is poorly defined. Although the patient described herein did not demonstrate any evidence of GBS, the authors theorize that her Nissen fundoplication may have impaired her gastric venting ability, resulting in her symptoms of retching without vomiting. This may have subsequently led to progressive gastric and bowel distention that ultimately resulted in her clinical presentation with ACS.

3. Conclusion

This case describes the first reported case of a pediatric patient with ACS associated with an acute Norovirus infection. Surgical decompression resulted in rapid normalization of physiologic parameters, with improved urine output, blood pressure and clinical perfusion. Persistent intra-abdominal edema and hypertension was managed with delayed abdominal closure. Associated hepatitis and pancreatitis were likely secondary to organ hypoperfusion and were managed expectantly with subsequent resolution. Ultimately, early surgical decompression with delayed definitive abdominal closure and supportive care resulted in a favorable hospital course for this patient. The authors suspect that the patient's history of Nissen fundoplication may have contributed to her severe Norovirus infection course by impairing gastric venting and thereby leading to progressive gastric and bowel distention that resulted in ACS.

Conflicts of interest statement

The authors have no conflict of interest to declare.

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