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Paediatric abdominal compartment syndrome in a 4.6 kg infant

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SUMMARY

An infant with a history of intestinal atresia type IV repaired at birth presented to the emergency department with recurrent abdominal distension. She was admitted 1 month before for abdominal distension secondary to formula intolerance. Hypothermia and mild respiratory distress prompted an evaluation with imaging, laboratory investigations, and blood and urine cultures. She was admitted to the intensive care unit and management included immediate surgical consultation, nothing by mouth, nasogastric tube placement for decompression and initiation of intravenous fluids and antibiotics. Her clinical status deteriorated within hours, requiring intubation and initiation of pressors. She responded to resuscitation but developed signs of abdominal compartment syndrome (ACS), prompting surgical decompression. The patient had a prolonged hospital stay and was discharged with total parenteral nutrition and G-tube feeds. This case highlights the importance of prompt recognition of risk factors, symptoms and management of paediatric ACS facilitating a reduction in morbidity and mortality.

BACKGROUND

Abdominal compartment syndrome (ACS) is caused by a pathological elevation of intra-abdominal pressure (IAP), which can lead to multiple organ dysfunction or failure. The condition has a high mortality rate. Early detection and diagnosis are critical for survival.[1–4]

The term ACS was first mentioned in the literature in 1989 by Fietsam *et al* when describing findings in four adult patients after the repair of a ruptured abdominal aortic aneurysm.[5]

A recent study by Bozer *et al* reported the incidence of paediatric ACS as 0.17%.[6] That study reported the mortality rate at 48.87%.[6] It was a retrospective study spanning 13 years (2007–2013) and included patients across 49 children's hospitals. Increased mortality was seen in patients 0–30 days of age with ACS (58.61%) and mortality was higher in neonatal intensive care units (NICUs).[6]

The World Society of the Abdominal Compartment Syndrome (WSACS) has been a world leader in providing guidelines for diagnosing and managing ACS.[7] It was first founded in 2004 and introduced evidence-based guidelines in 2006, with a revision in 2013. This provided consensus and clinical practice guidelines for the diagnosis and management of IAP, intra-abdominal hypertension (IAH) and ACS.[7] The proposed paediatric definition of ACS was defined as sustained elevation of IAP >10 mm Hg associated with organ dysfunction. The reference standard in children involved bladder filling with a volume of 1 mL/kg of sterile saline (minimum 3 mL and maximum 25 mL).[3 7]

Radiology studies have provided some clinical imaging features of ACS. The most common risk factor was increased abdominal contents, particularly bowel dilatation.[8 9]

CT and MRI findings by Je *et al* showed the most common findings were ascites, basal lung atelectasis, inferior vena cava (IVC) compression, abnormal enhancement of bowel wall and compromised perfusion of kidneys. The ratio of maximal anteroposterior (AP) to transverse abdominal (AT) diameter which is significant in adult ACS was not found to be a significant finding in paediatric ACS. Epelman *et al* also noted a rounded appearance of the abdomen.[8 9]

Prompt recognition, monitoring, stabilisation and intervention improve survival in this condition known to have a high mortality rate.[10]

The female infant in this report had several risk factors including prior abdominal surgery in the neonatal period for intestinal atresia and prior admission for abdominal distension and ileus.

On the day of emergency department (ED) presentation, she exhibited abdominal distension, respiratory compromise, concomitant parainfluenza infection, hypothermia and acidosis. She deteriorated within hours of presentation despite the initiation of a nasogastric (NG) tube for decompression, bowel rest and broad-spectrum antibiotic coverage. She developed multiorgan failure requiring intubation for respiratory support, pressors, blood transfusion, and antibiotic and antifungal therapy.

This case highlights the importance of prompt recognition of paediatric ACS risk factors, presenting symptoms and management to reduce morbidity and mortality.

CASE PRESENTATION

A female infant with a history of intestinal atresia type IV repaired in the neonatal period presented to our ED with abdominal distension and hypothermia (figure 1).

The patient's mother stated the infant had decreased feedings, fussiness, spitting up and grunting for 1 day. On the day of presentation, her abdomen seemed more distended and firmer. She was refusing her feeds and had decreased wet diapers. She was recently transitioned from a low carbohydrate formula to breastmilk and was feeding 5 ounces of breastmilk every 4 hours. The patient's stools were normal and non-bloody. There was no history of a fever. The patient had

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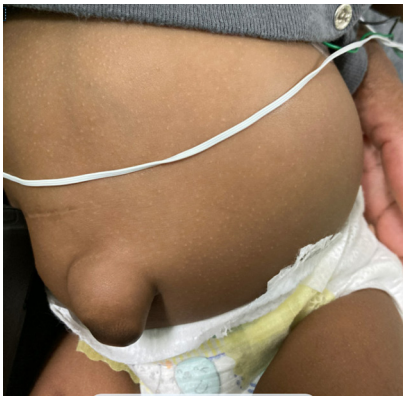

Figure 1 Abdominal distension with umbilical hernia.

recent contacts with respiratory illnesses including COVID-19 infection.

Medical history

The infant was born at 37 weeks 3 days gestation with the pregnancy complicated by maternal diabetes and hypertension. A prenatal ultrasound showed concern for dilated bowel and fetal growth restriction.

At birth, she was admitted to the NICU for suspected bowel obstruction. Her birth weight was 2.92 kg. An upper gastrointestinal series was suggestive of jejunal atresia. Intraoperatively, she was found to have multiple areas of intestinal atresia (classified as type IV intestinal atresia) which were repaired primarily. She had delayed return of bowel function and did not tolerate breastmilk during her hospital stay and was transitioned to a carbohydrate adjustable formula (3232A). She was discharged on the day of life 59 on formula feeds (3232A) with added dextrose at 110 mL every 4 hours. Her weight at time of discharge was 4.215 kg.

Two days after discharge, she presented to the ED with two episodes of bilious emesis. Abdominal X-rays showed gaseous distension of bowel loops in the right upper quadrant, as seen on previous X-rays. She was again admitted to the NICU.

Management included intravenous fluids, NG tube decompression and bowel rest. The distension resolved. It was determined her abdominal distention was secondary to incorrect mixing of the 3232A formula.

Subsequent X-rays of the abdomen showed decreased dilation of the right upper quadrant loops and mild residual distention. She resumed feeding and was discharged after 2 days of hospitalisation. Her weight at discharge was 4.168 kg.

Approximately 1 week prior to the ED presentation in this case report, she was transitioned off the 3232A formula to breastmilk.

On examination, the patient's vitals were as follows: blood pressure of 77/53 mm Hg, a heart rate of 174 beats/min, a body temperature of 35.7°C, respiratory rate of 50 breaths/min, oxygen saturation was 100% and she weighed 4.77 kg. The patient appeared well developed and well nourished. She was active, fussy and ill appearing with mild respiratory distress, nasal flaring, mild grunting with prolonged expiratory phase. Her breath sounds were normal. Her cardiovascular exam showed tachycardia with good pulses in her extremities. The patient's abdomen was distended and firm (figure 1). An umbilical hernia was present and easily reduced. She had a healed surgical scar in the right abdomen. There was no hepatosplenomegaly. There was no tenderness and bowel sounds were increased.

DIFFERENTIAL DIAGNOSIS

The differential diagnosis included abdominal obstruction due to adhesions from previous surgery, ileus, bowel perforation, necrotising enterocolitis (NEC), incarcerated umbilical hernia or pancreatitis.

Pneumonia, bronchiolitis or a viral illness including COVID-19 infection were considered because she presented with respiratory symptoms including grunting and nasal flaring.

Sepsis including a urinary tract infection or urosepsis was considered due to hypothermia and mild acidosis.

INVESTIGATIONS

To rule out obstruction, perforation or NEC, two-view abdominal X-rays were obtained. A chest X-ray and respiratory viral panel were performed given the patient's respiratory symptoms to evaluate her lungs for the presence of pneumonia or viral bronchiolitis. Additionally, point-of-care testing of electrolytes and blood gas (I-STAT), complete blood count, complete metabolic panel (CMP), C reactive protein (CRP), lipase, urinalysis (UA), a urine culture and blood cultures were ordered because of the patient's hypothermia and the possibilities of sepsis, acidosis, electrolyte derangements associated with poor feeding and emesis.

RESULTS

Initial I-stat showed pH 7.3, pCO2 32 mm Hg and HCO3 15.7 mmoL/L suggestive of mild metabolic acidosis.

CMP showed mild acidosis with a bicarb of 17 mmoL/L and mildly elevated glucose of 144 mg/dL which was likely stress-induced hyperglycaemia. The initial blood urea nitrogen (BUN) was mildly elevated at 18 mg/dL and creatinine was normal at 0.4 mg/dL. The white cell count was normal: 10.4 x 10 ^ 9/L with a haemoglobin of 83 g/L and haematocrit of 25% showing mild anaemia. The CRP was normal 0.5 mg/dL. The UA (catheterised specimen) showed small protein, red blood cells 0–2, white blood cells 10–25 and bacteria 3+ suggestive of possible urinary tract infection. The respiratory viral panel was positive for parainfluenza virus 3.

An abdominal X-ray (figure 2A) showed the abdomen was protuberant with marked dilatation of the bowel loops. There were mottled lucencies associated with these bowel loops, consistent with gas embedded within enteric contents. Air-fluid

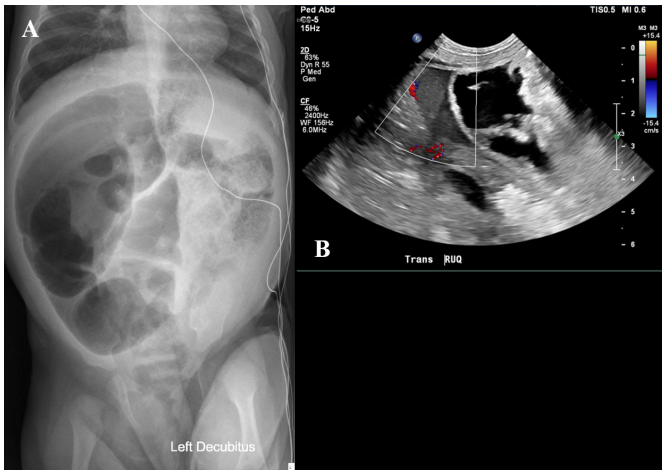

Figure 2 Radiographic findings. (A) Abdominal X-ray showing dilated bowel loops and air-fluid levels. (B) Ultrasound abdomen showing small complex fluid collections. RUQ, right upper quandrant

levels could be seen on the left lateral decubitus radiographs (figure 2A). There were no findings of free intraperitoneal air or pneumatosis intestinalis. Gas was noted in the colon. Again, noted was a bowel containing umbilical hernia. Chest X-ray was negative for pneumonia.

TREATMENT

A stat surgical consult was obtained. The patient was placed nothing by mouth (NPO), a normal saline (NS) bolus 20 mL/kg was given followed by maintenance intravenous fluids. Ceftriaxone and metronidazole were administered for presumed sepsis and possible intra-abdominal aetiology. An NG tube was placed for bowel decompression with improvement in her abdominal distention. Warming measures including a temperature management system and warm intravenous fluids were initiated for hypothermia. The patient was admitted to the intensive care unit (ICU) with a diagnosis of parainfluenza infection, abdominal distension and possible sepsis.

OUTCOME AND FOLLOW-UP

About 8 hours after admission, despite continued NPO status, NG tube for decompression, intravenous antibiotics, the patient developed progressively worsening tachycardia, hypotension, hypoxia with grunting, tachypnea and pallor. Repeat blood gas analysis showed worsening lactic acidosis (pH 7.19, lactate 5.5) and anaemia with an acute decrease of haemoglobin to 5.7 g/dL. The CRP increased to 7 mg/dL suggestive of an infectious or inflammatory process. The BUN increased to 25 and creatinine to 0.5 at that time consistent with acute kidney injury. Urine output was 1.3 cc/kg/hour.

She was emergently intubated using fentanyl and rocuronium due to respiratory failure with hypoxia and hypercapnia in the setting of sepsis. The patient required additional fluid resuscitation with NS (another 20 mL/kg given in the paediatric intensive care unit in addition to 20 mL/kg given in the ED), calcium chloride, dextrose, phenylephrine boluses and emergent blood transfusion (total 80 mL packed red blood cells (PRBC)). She improved haemodynamically and her lactic acidosis improved (lactate 1.3) with resuscitation. She was maintained on intermittent mandatory ventilation. Sedation was provided with dexmedetomidine and neuromuscular blockade with rocuronium. She received a phenylephrine infusion to maintain blood pressure and MAP. Norepinephrine infusion was added to maintain MAP 50–60 mm Hg. The NG tube was kept on low intermittent suction.

Her abdominal exam did not significantly change during resuscitation. Follow-up imaging demonstrated similar gaseous distention without free air or pneumatosis. An abdominal ultrasound showed small complex fluid collections (figure 2B).

Because the infant demonstrated rapid improvement in lactic acidosis and haemodynamics, she continued to be monitored closely. Bladder pressures were obtained every 4 hours and her urine output was monitored.

Approximately 9 hours after intubation, she had another change in her clinical exam. Despite making urine (1.7 cc/kg/hour, Cr 0.4, BUN 31), she was noted to have increased peaked inspiratory airway pressures from 20 to 30, increased bladder pressure from 10 to 15 mm Hg and diminished pulses in her lower extremities concerning ACS. This prompted emergent exploratory laparotomy, which revealed ischaemic bowel (figure 3A). Preoperatively, the diagnosis was ACS in a 4.6 kg infant. Postoperatively, the diagnosis was ACS with bowel obstruction from adhesions in a 4.6 kg infant. During the first surgery, on entry into the abdomen, the bowel appeared ischaemic. As the bowel was eviscerated there were numerous adhesions between the small bowel to the small bowel and the small bowel to the mesentery. There was an adhesive band causing a mechanical obstruction and exacerbated her initial jejunal atresia repair as demonstrated by the degree of size mismatch of the bowel (figure 3). This jejunal segment was resected. The distal small bowel was matted with adhesions and unable to separate without causing more damage. Throughout the case, perfusion to the bowel improved. The decision was made to keep her in discontinuity with an open abdomen using a negative pressure dressing with a planned second-look operation.

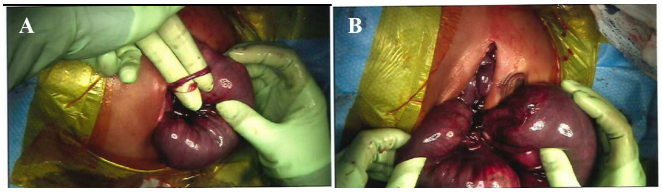

Figure 3 Operative findings during the first surgery. (A) Adhesive bandages. (B) Jejunal structure.

She returned to the OR the following day for a second look. Using indocyanine green (ICG) and a Stryker Spy-Phi portable handheld imager, bowel perfusion was assessed. During this operation, a complete adhesiolysis was performed and all the remaining small bowel and colon were evaluated (figure 4). Seven segments of small bowel that demonstrated no perfusion were resected. She remained in discontinuity with an open abdomen using a negative pressure dressing with a plan to return for another look. At the final washout and assessment, the ICG and Striker Spy-Phi portable handheld imager were used to assess bowel perfusion. Two preserved segments from the last procedure were not viable and required resection. The remaining bowel was perfused, and the decision was made to anastomose the distal segments and leave her in discontinuity to allow the distal segments to heal. The remaining small bowel, including the ileocecal valve, measured approximately 35 cm. The decision was made to place a gastrostomy tube during this operation as there was a high chance for intestinal failure given the degree of bowel loss. The viable, proximal jejunum was brought up as an ostomy and the abdomen was closed.

The patient was extubated on day 11 of the hospital stay with a resolution of multiorgan dysfunction. Her recovery was complicated by catheter-associated thrombus in the lower extremity and was treated with enoxaparin. She was placed back into continuity approximately 6 weeks later. She was admitted for 87 days and discharged with TPN, gastrostomy tube feeds, multivitamins and enoxaparin.

As of publication, the patient continues weekly follow-up with the multidisciplinary intestinal failure team to monitor TPN, trophic feeds and weight gain. Outpatient management includes occupational and physical therapy.

DISCUSSION

Clinicians should have a high index of suspicion for ACS. This patient had a history of abdominal distension and prior abdominal surgery. These are known risk factors for primary ACS. The abdominal X-rays were consistent with prior imaging showing a dilated bowel. The infant presented with abdominal distension, hypothermia, mild respiratory distress and metabolic acidosis. This prompted an evaluation

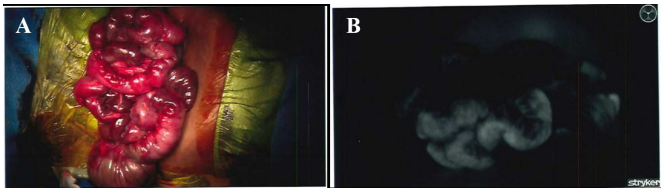

Figure 4 Operative findings during the second surgery. (A) Bowel following an extensive lysis of adhesions. (B) Fluorescent Imaging Technology (ICG SPY PHI) assessment of bowel perfusion. ICG, indocyanine green.

for suspected sepsis, administration of an NG tube for decompression, intravenous fluids, NPO and urgent surgical consultation. She was admitted to the critical care service and decompensated within 8 hours of admission as demonstrated by the development of hypotension, worsening acidosis and the requirement of intubation and pressors.

Despite the initiation of NPO, NG tube to suction, intravenous fluids and antibiotics, this patient further decompensated and required intubation, fluid boluses, a PRBC transfusion, expanded antibiotics coverage and exploratory laparotomy, which revealed necrotic bowel, requiring small bowel resection. Three exploratory laparotomies were required for removal of further necrotic bowel, wash out, appendectomy, jejunostomy, creation of mucous fistula, gastrostomy tube placement and reanastomosis.

ACS is caused by a pathological elevation of IAP leading to multiple organ dysfunction and failure. This condition has a high mortality rate, thus early detection and diagnosis are critical for survival.

ACS is defined as increased IAP >10 mm Hg in children and >20 mm Hg in adults, plus two of the following factors: oliguria or anuria, respiratory decompensation, hypotension, shock or metabolic acidosis. Increased abdominal contents, particularly bowel dilatation, is a common risk factor in paediatric ACS as shown in table 1.[3 7 11 12]

Paediatric ACS case reports in the literature include an 8-month-old patient with gastric volvulus[13] and another with a history of aerophagia, autism, developmental delay seizures and constipation.[14] Decompression of the stomach resulted in clinical improvement in the first case.[13] The second case involved a 19-year-old requiring decompressive laparotomy in the ED and then was transferred to the operating room for further management.[14] Three cases were described in another report, two of which were associated with trauma and the third associated with shock of unknown aetiology. The patients eventually required decompression and eventually recovered.[2]

A case of ACS associated with viral hepatitis also is described in the literature and the patient eventually recovered with medical management.[15] Another presentation of ACS occurred as a complication of posterior spinal fusion. Hypoxia, hypotension, respiratory acidosis and compromise developed near the end of the surgery in this 8-year-old with Duchenne muscular dystrophy. ACS was diagnosed with the patient in the supine position and treated with laparotomy, resulting in complete recovery within 5 days.[16]

These cases demonstrate the multifactorial risk factors in paediatric ACS and the importance of rapid recognition and treatment in producing a favourable outcome.

In a review of CT and MRI images in paediatric patients with ACS, common findings included ascites, atelectasis of the basal lungs, IVC compression and abnormal enhancement of the bowel wall.[8 9]

ACS occurs infrequently in children and there is often a delay in recognition.[10 11] Patients are usually critically ill with a high mortality rate (>48%).[6] Urgent decompression often results in clinical improvement.[10 17 18] Major mechanisms in the causation of paediatric ACS appear to be ischaemia and reperfusion injury.[10] It may occur at a lower IAP (10 mm Hg) than in adults (20 mm Hg).[4 18]

The WSACS provides clinical practice guidelines for the diagnosis and management of paediatric ACS, defined as sustained elevation of IAP >10 mm Hg associated with organ dysfunction.[3 7]

The non-operative management of ACS according to the paediatric guidelines subcommittee for the WSACS in 2013 includes the following therapeutic goals:

1. Measuring IAP in the presence of a known risk factor in a patient who is critically ill or injured.
2. The use of protocols to monitor and manage IAP in the care of critically ill or injured patients.
3. Decrease intra-abdominal volume by NG tube decompression, rectal tube decompression, bladder decompression with Foley catheter and evacuating ascites or intra-abdominal haematoma if possible. When technically possible, percutaneous catheter drainage should be used to remove fluid in patients with IAH/ACS versus no intervention or decompressive laparotomy
4. Proper position of the patient in the bed. The patient should be placed in the supine position as opposed to the head of bed >20° as often used to prevent the risk of pneumonia associated with ventilator use. The latter is associated with increased IAP.
5. Pharmacological paralysis may be necessary, especially in respiratory failure with severe hypercapnia. It decreases carbon dioxide production and relaxes the abdominal wall. Ventilatory support with oxygen and positive end-expiratory pressure improves hypoxaemia and reduces ventilation-prefusion (VP) mismatch.
6. Pain control and sedation. Sedation and proper pain control can improve the compliance of the abdominal wall and improve tissue perfusion.
7. Judicious fluid administration is recommended, and data suggest that large-volume crystalloid resuscitation for shock may be a factor in the development of ACS. In critically ill patients with or at risk of IAH, a protocol to optimise fluid balance is recommended.
8. In patients with obvious ACS, decompressive laparotomy should be performed.

Table 1 Risk factors for abdominal compartment syndrome

Decreased abdominal wall compliance	Abdominal surgery, major trauma, burns or prone positioning
Increased intraluminal contents	Gastroparesis, gastric distention, ileus, pseudo-obstruction of the colon, volvulus
Increased intraabdominal contents	Acute pancreatitis, distended abdomen, haemoperitoneum or pneumoperitoneum, Intra-abdominal infection/abscess, intraperitoneal or retroperitoneal tumours, laparoscopy with excess insufflation pressures, liver dysfunction with ascites, peritoneal dialysis
Capillary leak/fluid resuscitation	Acidosis, hypothermia, massive fluid resuscitation, polytransfusion
Miscellaneous associations	Acute gastroenteritis, bacteraemia, coagulopathy, massive inguinal hernia repair, mechanical ventilation, obesity, peritonitis, pneumonia, sepsis, shock, hypotension

Patient's perspective

The patient's mother commented as followed: There's so much I can say but blessed and thankful are the first words that come to mind when I think of this journey thus far. I found out at about 6 or 7 months of my pregnancy that she had a dilated bowel/colon, and I didn't know what that meant. Every week I had an ultrasound where the doctor realized it was increasing in size and wanted to reassure me that it could possibly be nothing at all, but I was still stressed the entire time after finding out something could be wrong. I tried to research as much as I could about similar situations and I came to terms that, after her birth, my baby would need surgery immediately and the healing process would be a short 3 weeks and we'd be home. Or so I thought.

When she was born, she was beautiful and perfect. She was assessed and it was determined she would have to be taken to the neonatal intensive care unit at the Children's Hospital right away. I got to see her before she left, and my pain started then: my baby left the hospital without me, and she had surgery the very next day. While I was unable to leave, her father was there with her until I was discharged the following day.

This experience was so surreal. Unexplainable nervousness and fear. You don't even know the NICU exists unless you have had a sick child. I didn't even know what a children's hospital was. It's just something you don't think about or expect to experience. The recovery was so slow even though she's 1 now. I remember being excited for rounds and hearing progress and other days crying because we'd be going backwards. Infection. Vomiting. Not pooping. Unable to eat. Celebrating holidays without her or without my older children who were at home. It was literally the roller coaster I had been reading about before she was born from other's experiences. The real help I had was joining support groups of other moms going through the NICU journey, which helped me become more grateful that it could be much worse and that I should be thankful no matter how hard. There are so many small details left out of course, but in the first months of her life I learned so much about her condition (at that time, intestinal atresia) that I felt like a nurse myself.

We went home from the NICU when she was 2 months old. We returned to the ED 1 week later and then again when she was 3 months old. During this ED visit, she was admitted and required emergency surgery. She caught parainfluenza from someone. I brought her to the ED because I felt like something wasn't right. Thankfully I was right, and she had several surgeries during which she lost most of her previously repaired intestines and became a short gut baby. During this time, we thought we'd lose her, and we were very distraught. We too became sick—I think we caught parainfluenza. While trying to maintain our home and children at home and being with her daily in the hospital, it was a struggle but never too much to get through because we had faith in God. We stayed in the hospital this time for 3 more months and I learned much more than I could imagine. I felt like I had gone to nursing school. From G-tubes to stomas and ostomy care, bolus feeds and how absorption played a role in her life. I was grateful to be her mom and her advocate. She grew during her 3 months of hospitalization, and we finally came home when she was about 6 months old. We have remained at home despite frequent visits to the hospital for weekly labs and appointments with our care team or whenever I think it's necessary to go to the ED. I never want to hesitate in getting her the care she needs. This journey has been a learning

Continued

Patient's perspective Continued

process and I'm fortunate to know my child is progressing and thriving in ways I never could have imagined.

Learning points

- Abdominal compartment syndrome (ACS) in adults and paediatrics has different parameters for definition.
- Providers should be aware of common risk factors for ACS.
- ACS requires prompt recognition and management to improve survival.

9. In patients with an open abdominal wound, wound therapy with negative pressure should be used to promote earlier closure of abdominal fascia.

Surgical decompression is the definitive treatment utilised if there is a worsening of the clinical status with failure of the previously mentioned measures.[2 8 17 19 20]

Prompt recognition, monitoring, stabilisation and surgical intervention ensured survival in this case of a condition with a high mortality rate.

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