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Short Communication

Primary Chronic Constipation with Secondary Abdominal Compartment Syndrome

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ABOUT THE STUDY

A high Intraabdominal Pressure (IAP) and signs of organ failure are known as Abdominal Compartment Syndrome (ACS). As well as in neonates with abnormalities in the abdominal wall, but it is seldom described in children. We go over the peculiar case of an 11-year-old child who had a lengthy history of chronic constipation and who had acute ACS, necessitating resuscitative measures and emergent disimpaction. He had experienced escalating stomach pain, nausea, decreased appetite, and protracted encopresis for the previous two weeks. This was found to be severely underweight and to have an enormously swollen belly with a palpable fecaloma. These findings were supported by abdominal XR. He developed orthostatic hypotension, tachycardia, and oliguric within 24 hours of presentation. He rapidly deteriorated after two enemas, exhibiting severe hypotension, pronounced tachycardia, acute respiratory distress, and a deteriorating mental state. After starting endotracheal intubation, fluid boluses, and vasopressors, urgent surgical faecal disimpaction was performed. Vital signs quickly improved as a result of this. He has undergone a complete examination, and the only condition found is functional constipation. This example highlights the necessity to actively manage constipation and what can happen if it is not, even though ACS secondary to constipation is exceedingly uncommon [1].

A High Intraabdominal Pressure (IAP) with cardiovascular, renal, and pulmonary failure is known as Abdominal Compartment Syndrome (ACS), and abdominal decompression can help. Adults experience the effects of ACS at IAPs greater than 35 mmHg, whereas children are believed to have a lower threshold for IAP. Patients with elevated IAP should be continuously watched for deterioration since the condition has the potential to develop into ACS with multiorgan failure. Medical techniques to lower IAP can be tried if ACS develops, but recalcitrant cases necessitate surgical decompression [2,3].

Clinical symptoms such as a tensely inflated abdomen, impalpable femoral pulses, cyanosis of the low extremities, and increasing oliguria and hypoxia are sufficient to warrant decompression when there is no opportunity to assess IAP. If left untreated, ACS is linked to a death rate of 80–100%. Mortality rates are still high with early decompression, ranging from 40% to 60%. Organ failure is a direct mechanical result of ACS. As shown in our patient during his acute decline, respiratory derangements happen when the raised diaphragm reduces functional residual capacity and raises airway pressures. Reduced venous return due to compression of the heart and inferior vena cava causes cardiovascular impairment. Our patient's hypotension and tachycardia were the first clinical symptoms of this derangement, followed by his raised diaphragm and compressed heart on imaging tests. As it got worse, it led to venous hypertension, limited venous return, and decreased limb perfusion, which resulted in priapism [4].

The dopamine need, which disappeared right away after disimpaction, serves as a good example of the effects of his elevated IAP. Kidney function is hampered in ACS by renal vein compression. Renal impairment is also suggested by oliguric increasing creatinine (40 to 70 Umol/L after 25 hours of presentation). Lactic acidosis and an increased INR are signs of persistent tissue hypoxia. The fragile baseline of a grossly inflated colon was challenged by the amount of two enemas, resulting in a condition of acute decompensation, and our patient's ACS likely stemmed from acute and chronic alterations. There was a significant improvement on the first day following disimpaction, and there was no clinical sign of perforation caused by the enemas or disimpaction. This was most likely caused by a recto sigmoid junction reperfusion injury after healing [5].

Only two cases of ACS in children have been recorded to date, and the progression of the disease from constipation is inadequately described in the pediatric literature. A boy with congenital megacolon who developed ACS as a result of extensive colonic dilatation is described by Birkhahn and Gaeta. Surgical decompression caused reperfusion damage, coagulopathy, and the patient's death. It is important to pay attention to the underlying chronic constipation that caused ACS in our patient. He required routine manual disimpaction and frequent nasogastric electrolyte solution because his severe

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constipation was resistant to multiple stool softeners and enemas. It was so bad that it made going to school difficult. The severity of the chronic malnutrition our patient experienced is more startling, as evidenced by his low albumin level (19 g/L), anaemia that required numerous transfusions, and the onset of refeeding syndrome [6].

CONCLUSION

An extensive examination was conducted in an effort to pinpoint the fundamental cause. Biopsies taken from the colon and the rectal were free of Hirshsprung's disease. An MRI of his spine ruled out the potential of a tethered chord after repeat thyroid and celiac screenings came up negative. An intestinal motility study was uneventful, a Shapes study was consistent with a diffuse colonic dysmotility, and anorectal manometry showed no signs of outlet dysfunction. An accurate diagnosis of functional constipation remains despite comprehensive testing. The treatment team has also been effective in reintroducing a healthy bowel routine after his reanastomosis with careful monitoring.

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