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Intra-abdominal hypertension (IAH) in children has a clinically significant adverse effect on organ function and mortality despite its low incidence. The sparse reporting of IAH and abdominal compartment syndrome (ACS) in the pediatric literature may reflect inadequate awareness among providers. The most effective way of preventing ACS is by early recognition and intervention aimed at attenuating its progression. A high index of suspicion is of paramount importance.

Our patient was a 7 kg, 7 month-old male born at 33 weeks with a history of Trisomy 18, chronic lung disease, and left diaphragmatic agenesis. He underwent successful reduction of his diaphragmatic hernia with patch repair on day of life 14 but this patch became insufficient and his stomach herniated into the thorax. He was scheduled for exploratory laparotomy with diaphragmatic patch repair and jejunostomy tube placement.

The patient arrived in the OR with a Broviac catheter and endotracheal tube in situ and induction of general anesthesia was uneventful. Two peripheral IVs and an A-line were placed. Adequate ventilation and oxygenation were established with pressure control ventilation. The surgeon identified the diaphragmatic hernia and the patch was repaired. Bowel exploration revealed malrotation, an ileal stricture and Meckel's diverticulum, all of which were repaired.

Following closure the patient became difficult to ventilate necessitating a significant increase in inspiratory pressure and FiO₂. The end-tidal CO₂ rose sharply and ABG revealed respiratory acidosis. This was communicated to the surgeon who noted that the patient's abdomen remained soft and non-distended. However, the patient was oliguric and given the time course of events, increased abdominal pressure was suspected. The abdomen was re-opened, improving ventilation almost immediately. The fascia was bridged with mesh and skin closed. The patient returned to the NICU with ventilatory support only slightly increased from baseline.

This case illustrates the respiratory effects of elevated abdominal pressure which led to hypoxia and hypercarbia. Decompression of the abdominal cavity led to an immediate reversal of the respiratory changes. Of the many causes for respiratory compromise and cardiovascular collapse associated with abdominal surgery, increased abdominal pressure and ACS should be considered.

Although the typical patient has a distended abdomen and confirmation can be made by pressure measurements, in our case the decision was made to not measure IAP. The clinical signs were used as a guide for intervention to prevent further deterioration.

ACS may present insidiously and be life threatening. Optimal management is contingent on clinical suspicion and immediate treatment. This case serves as an example of the continued need for education and awareness of its occurrence so as to avoid potentially devastating effects in the children we care for.

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