Introduction Neonatal resuscitation guidelines routinely recommend delayed cord clamping.\(^1\) This recommendation is applicable to preterm neonates, provided they are born vigorous and crying. It is associated with increased haemoglobin, preventing neonatal anaemia.\(^2\) Failure to delay cord clamping may necessitate recurrent neonatal blood transfusions.\(^3\) Delayed cord clamping reduces the risk of parenteral nutrition associated liver disease, necrotising enterocolitis, and intra-ventricular haemorrhage.\(^4\) Case Series Two extremely premature neonates were born by spontaneous vaginal delivery, at 23+4 and 26 weeks respectively. Prior to delivery, the neonatal team requested delayed cord clamping. Both infants cried spontaneously and were vigorous at birth. At 1 minute, APGAR scores were 6 and 8 respectively. However both umbilical cords were immediately clamped. The first infant required five red cell transfusions during his neonatal admission. He developed necrotising enterocolitis, managed medically. He had two episodes of suspected late-onset sepsis. Cranial ultrasound revealed a left-sided Grade 3 intraventricular haemorrhage. The second neonate required three red cell transfusions during her NICU admission. She developed parenteral nutrition associated liver disease. This was characterized by an elevated direct bilirubin. She also developed Klebsiella pneumonia.

Discussion Non-adherence to delayed cord clamping may cause chronic anaemia in preterm neonates. Multiple transfusions compound the risk of acute transfusion-related reactions. These include fever, haemolysis and anaphylaxis. Transfusions are an independent risk factor for parenteral nutrition associated liver disease. Transfusions also have an immunosuppressive effect, increasing the risk of pneumonia and late onset neonatal sepsis. Transfusion-related alloantibodies increase the survival benefit, increasing the risk of pneumonia and late onset neonatal sepsis. Transfusion-related alloantibodies increase the risk of pneumonia and late onset neonatal sepsis. Transfusion-related alloantibodies increase the risk of pneumonia and late onset neonatal sepsis. Transfusion-related alloantibodies increase the risk of pneumonia and late onset neonatal sepsis. Transfusion-related alloantibodies increase the risk of pneumonia and late onset neonatal sepsis. Transfusion-related alloantibodies increase the risk of pneumonia and late onset neonatal sepsis. Transfusion-related alloantibodies increase the risk of pneumonia and late onset neonatal sepsis. Transfusion-related alloantibodies increase the risk of pneumonia and late onset neonatal sepsis. Transfusion-related alloantibodies increase the risk of pneumonia and late onset neonatal sepsis. Transfusion-related alloantibodies increase the risk of pneumonia and late onset neonatal sepsis. Transfusion-related alloantibodies increase the risk of pneumonia and late onset neonatal sepsis. Transfusion-related alloantibodies increase the risk of pneumonia and late onset neonatal sepsis. Transfusion-related alloantibodies increase the risk of pneumonia and late onset neonatal sepsis. Transfusion-related alloantibodies increase the risk of pneumonia and late onset neonatal sepsis. Transfusion-related alloantibodies increase the risk of pneumonia and late onset neonatal sepsis. Transfusion-related alloantibodies increase the risk of pneumonia and late onset neonatal sepsis. Transfusion-related alloantibodies increase the risk of pneumonia and late onset neonatal sepsis. Transfusion-related alloantibodies increase the risk of pneumonia and late onset neonatal sepsis. Transfusion-related alloantibodies increase the risk of pneumonia and late onset neonatal sepsis. Transfusion-related alloantibodies increase the risk of pneumonia and late onset neonatal sepsis. Transfusion-related alloantibodies increase the risk of pneumonia and late onset neonatal sepsis. Transfusion-related alloantibodies increase the risk of pneumonia and late onset neonatal sepsis.

Conclusion Blood transfusions in preterm neonates should be avoided where possible.

Delayed cord clamping provides a practical, non-invasive method to prevent neonatal anaemia and transfusions. Clinical knowledge and communication between neonatal and obstetric teams is fundamental to optimising patient care.

REFERENCES

129 INTRA-ABDOMINAL COMPARTMENT SYNDROME COMPLICATING POSTOPERATIVE GASTROSCHISIS NEONATE – A LEARNING LESSON
Abhidhamma Kaninde*, A Battiwala, S Taha, S Mittal. Leicester Royal Infirmary
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Introduction Abdominal compartment syndrome (ACS) is a known complication of abdominal wall defect repair which requires vigilant monitoring. Here we emphasise bladder pressure monitoring system as one of the tools for early detection of ACS.

Case Summary This was a term baby with an antenatal diagnosis of gastrochisis born by elective caesarean section. The baby was born in good condition and the abdominal contents were covered with a plastic bag to prevent fluid and heat loss, and a nasogastric tube was passed. The baby was taken to theatre within a few hours of birth for surgical repair. Intraoperative findings included the stomach, small and large bowel being outside of the abdominal cavity.

In view of the repair being under moderate tension, bladder pressure monitoring for ACS was instituted using an indwelling catheter and standard pressure transducer. Over the following 12 hours, the bladder pressure gradually increased from a baseline of 8-10 mmHg to 24 mmHg. This was accompanied by oliguria, elevated lactate, a wide toe-core temperature gap and high ventilator pressure requirement to achieve the target tidal volume.

Interestingly, the abdomen remained non-tender for a significant time period, which delayed early suspicion of ACS. But with worsening metabolic acidosis and increasing bladder pressure reading decision was made to take baby to theatre for laparotomy. Following decompressive laparotomy, the baby’s clinical condition improved and observation parameters stabilised.

Discussion Gastrochisis is defined as a defect in the abdominal wall resulting in evisceration of abdominal contents. Its incidence is reported as around 1.35 per 10,000 total birth in England and Wales as per Tan et. Risk factors include young maternal age and maternal cigarette smoking, alongside certain medications and maternal genitourinary infections.

ACS can occur as a rare complication of surgical repair of gastrochisis, and can result in intestinal and renal ischaemia. As per Akhobadze et al sustained intra-abdominal pressure (IAP) of greater than 20 mm of Hg is defined as abdominal compartment syndrome.

Intravesical pressure monitoring is recommended by the World Society for Abdominal Compartment Syndrome (WSACS) as the most reliable technique for IAP measurement.

Clinical staff working in surgical neonatal unit should be aware of sensitivity of Intrabdominal pressure monitoring system over clinical signs of ACS.

Conclusion Abdominal signs may not appear in early abdominal compartment syndrome.

Intraabdominal pressure monitoring is important for early diagnosis of abdominal compartment syndrome.