PATIENT SAFETY INCIDENT REPORTING DATA TRENDS OF A REGIONAL NEONATAL TRANSFER SERVICE

INTRODUCTION Neonatal transfers take place in a high-risk environment and patient safety must be a key priority.

METHODS Risk reporting for this regional transfer service has evolved over the last five years. The patient safety incident form has been adapted over the last 3 years, providing a clear structure of what is to be reported, ensuring the significance of the adverse event is considered and the response to the incident recorded. This study reviewed the patient safety incident forms completed over the last three years.

RESULTS Form completion significantly improved from 61% in 2011 to 96% in 2013. Comparing trends between 2012 and 2013: significant improvements were seen in delayed dispatch to critical transfers, time delays during stabilisation, equipment problems, vascular incidents and unintended hypothermia. How- ever, a significant increase in hypocarbia and endotracheal tube repositioning.

CONCLUSION We report improved risk reporting and an improvement in many of the incident categories. Future work needs to focus on sustaining and improving other categories.

FOOD PROTEIN INDUCED ENTEROCOLITIS SYNDROME (FPIES) IS AN IMPORTANT DIFFERENTIAL DIAGNOSIS OF NECROTIZING ENTEROCOLITIS IN PRETERM INFANTS

Aims GI dysmotility is common in preterm infants. Necrotizing enterocolitis (NEC) may develop when dysmotility, luminal contents and gut bacteria drive inflammation. Breast feeding is notably protective. Food Protein Induced Enterocolitis Syndrome (FPIES) may mimic NEC, including distension and intramural gas. In contrast to NEC, FPIES manifests thrombocytosis and neutrophilia. We have examined whether cases initially diagnosed as NEC were due to FPIES induced by cow’s milk (CM) protein.

Methods 9 preterm infants from one tertiary centre (23–36 weeks, BW 535–1700 g) were identified as possible FPIES because of temporal link between introduction of CM and onset of acute GI symptoms. A timeline was obtained of symptoms, feeding and blood parameters.

Results 3/9 showed gastro-oesophageal reflux ± constipation, improving on breast milk exclusion and worsening on reintroduction. NEC was queried but not diagnosed. All gained weight poorly, leading to CM formula introduction, which substantially worsened symptoms. All settled on exclusion and relapsed on CM challenge, remitting only with hypoallergenic formulae. Investigations showed thrombocytosis (>400) and reduced albumin after CM introduction. 6/9 were diagnosed with NEC, two twice. All showed similar dysmotility and poor weight gain on breast milk, and in all cases NEC symptoms began within 48 h of introduction of CM formula ± fortifier or thickener. In 1/ 6 thrombocytopenia and neutropenia was consistent with classic NEC, while in 5/6 platelets increased (mean 422, range 310–550) as did wcc (mean 17, range 16–25). All settled only on hydrolysate or amino acid formula.

Conclusion The NEC-like episodes in these infants concord with classic reports of FPIES in LBW infants. Most documented cases of NEC develop thrombocytopenia – indeed thrombocytosis is unreported. All showed dysmotility, poor weight gain and thrombocytosis on breast milk: a pattern characteristic of non-IgE-mediated allergy. CM introduction because of poor weight gain was uniformly deleterious. Recognition that non-IgE-mediated CM allergy may cause dysmotility and impaired growth in preterm infants should promote consideration of maternal milk exclusion diets while breastfeeding, or introduction of hypoallergenic rather than CM formulae. Thrombocytosis in a preterm infant with dysmotility should be a red-flag sign for non-IgE-mediated food allergy and risk of FPIES on formula introduction.
Aims Clear guidelines exist for the management of major haemorrhage in adults. These include a strong emphasis on early transfusion of Fresh Frozen Plasma (FFP) in conjunction with packed red cells (PRC) in order to avoid/treat possible accompanying coagulopathy. Current practice within the local neonatal population is to await clotting profiles and administer FFP if coagulation is found to be abnormal. This practice may lead to a significant delay in treatment. The aim of this service evaluation was to identify whether term neonates with major haemorrhage around the time of delivery required support with coagulation factors. This may support the use of prophylactic FFP alongside RBC transfusion prior to results becoming available helping to stabilise the infant sooner.

Methods Data was requested from our local blood transfusion laboratory for all requests for blood products made for patients on the neonatal unit between April 2011 to December 2013. Further details regarding antenatal and neonatal course were then taken from the Badger database on all babies that were identified to have received blood products on their date of birth or following day. Babies born at less than 37 weeks were excluded.

Results We identified 7 term babies with a clear history of major perinatal blood loss. 5/7 (71%) required transfusion of FFP in addition to PRC on the basis of abnormal clotting profile. All babies receiving FFP needed extensive resuscitation at birth followed by intensive care support, whereas the 2 babies that were not transfused were clinically stable. One of the babies not receiving FFP did not have their clotting profile checked. All babies received Vitamin K post delivery as standard.

Conclusion Term infants with a clear history of perinatal blood loss who are compromised at delivery and require packed red cell transfusion should be considered for early transfusion of FFP without awaiting coagulation profiles.