Abdominal Compartment Syndrome

Elevation of intra-abdominal pressure (IAP) may impair physiology and organ function producing what is known as Abdominal Compartment Syndrome (ACS). The physiological consequences of increased intraabdominal pressure consist of cardiac output reduction, pulmonary ventilation restriction (increasing peak inspiratory pressure and hypercapnia), renal function (oliguria) and visceral perfusion diminution (gut mucosal acidosis), and increased in cerebro-spinal pressure. ACS can be the result in abdominal wall defect closures (gastroschisis and omphalocele), inflammatory bowel conditions, trauma and intraabdominal infections (enterocolitis, appendicitis, bowel perforation). Vesical and inferior vena cava pressure recording have good correlation with IAP. Gastric, rectal, superior vena cava, femoral/brachial artery, and rectus compartment pressure are poor indicators of actual IAP. An elevated abdominal compartment pressure is considered as greater than 25 mm Hg. The bowel is the most sensitive organ to ACS and it develops evidence of end-organ damage before the development of classic renal, pulmonary and cardiovascular signs. Management consists of abdominal decompression. Reopening the abdominal wound is a lifesaving intervention prompted usually by cardiovascular deterioration. Use of delayed wound closure (staged celiotomy) may prevent development of this condition in high-risk surgical patients. Timely decompression of the ACS results in improvements in cardiopulmonary and renal function. Failure to recognize and treat ACS is inevitably fatal.

References:
Fetal Abdominal Wall Defects

Most common abdominal wall defects (AWD) are gastroschisis, omphalocele and hernia of the umbilical cord. Referral to tertiary centers with available neonatal intensive care is necessary in prenatally diagnosed cases. Changing the route of delivery does not affect outcome for either defect. Omphalocele has a high incidence of associated anomalies (cardiac, neurogenic, genitourinary, skeletal, chromosomal syndromes) that are the cornerstones of mortality. Detailed search for associated anomalies, fetal echocardiogram and karyotyping should be performed always. Cesarean section is justified in large omphalocoles (> 5 cm) to avoid liver damage, sac rupture and dystocia. Gastroschisis prenatal US appearance depends on gestational age and condition of extruded bowel. Fetal karyotyping testing is less important. Intestinal atresia complicates the defect, the result of an intrauterine vascular accident. Intestinal obstruction due to atresia or luminal constriction may cause polyhydramnios, fetal growth retardation and preterm labor, findings that can be monitored with serial US. No benefit has been found in recommending routine c-section for most cases of gastroschisis. Preterm deliveries by c-section have been found to prevent bowel damage in fetus with progressive bowel dilatation and thickening, a finding that has not been corroborated by others. Abnormal US appearance of fetal bowel is associated with more bowel edema, longer operative time and a higher incidence of postoperative complications.

References:
Cholecystokinin

Cholecystokinin (CCK) is a naturally occurring octapeptide hormone that has several utilities in children. Secreted in the proximal small bowel, increases bile flow, causes contraction of the gallbladder and promotes GI and colonic motility. As diagnostic source is used in determining the ejection fraction of the biliary tree. Therapeutically, CCK has been used to increase bile flow in cases of TPN cholestasis. CCK is associated with functional and histologic improvement in the periportal area of the liver as well as preservation of gallbladder emptying ability. CCK significantly diminishes direct bilirubin levels in infants with TPN cholestasis, effectively clears the biliary tree from sludge and stones. CCK use can be associated with cramping abdominal pain, feeding intolerance, flushing and rarely hypotension. Children with clinical liver failure have no response to CCK. If conjugated hyperbilirubinemia from TPN does not resolve after three weeks of full enteral feedings and stools remain acholic, CCK therapy should be considered. Evidence-based data that CCK prevents TPN cholestasis is not conclusive. Stopping TPN and resuming enteral feeding is the only effective management in TPN cholestasis.

References:

* Edited by: Humberto L. Lugo-Vicente, MD, FACS, FAAP
Associate Professor of Pediatric Surgery, University of Puerto Rico School of Medicine and University Pediatric Hospital, Rio Piedras, Puerto Rico.
Address - P.O. Box 10426, Caparra Heights Station, San Juan, Puerto Rico 00922-0426.
Tel (787)-786-3495 Fax (787)-720-6103 E-mail: titolugo@coqui.net
Internet: http://home.coqui.net/titolugo

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