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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:

1-DeCou JM, Abrams RS, Miller RS, Gauderer MW: Abdominal compartment syndrome in children: experience with three cases. J Pediatr Surg 35(6):840-2, 2000

2- Bendahan J, Coetzee CJ, Papagianopoulos C, Muller R: Abdominal compartment syndrome. J Trauma 38(1):152-3, 1995

3- Lacey SR, Bruce J, Brooks SP, Griswald J, Ferguson W, Allen JE, Jewett TC Jr, Karp MP, Cooney DR: The relative merits of various methods of indirect measurement of intraabdominal pressure as a guide to closure of abdominal wall defects. J Pediatr Surg. 22(12):1207-11, 1987

4- Burch JM, Moore EE, Moore FA, Franciose R: The abdominal compartment syndrome. Surg Clin North Am 76(4):833-42, 1996

5- Williams M, Simms HH: Abdominal compartment syndrome: case reports and implications for management in critically ill patients. Am Surg 63(6):555-8, 1997

6- Watson RA, Howdieshell TR: Abdominal compartment syndrome. South Med J 91(4):326-32, 1998

7- Neville HL, Lally KP, Cox CS Jr: Emergent abdominal decompression with patch abdominoplasty in the pediatric patient. J Pediatr Surg 35(5):705-8, 2000

8- Schein M, Wittman DH, Aprahamian CC, et al: The abdominal compartment syndrome: The physiological and clinical consequences of elevated intra-abdominal pressure. J Am Coll Surg 180: 745-753, 1995

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 omphaloceles (> 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:

1- Langer JC: Fetal Abdominal Wall Defects. Semin Pediatr Surg 2(2):121-128, 1996
2- Dykes EH: Prenatal diagnosis and management of abdominal wall defects. Semin Pediatr Surg. 5(2):90-4, 1996
3- Sipes SL, Weiner CP, Sipes DR 2d, Grant SS, Williamson RA: Gastroschisis and omphalocele: does either antenatal diagnosis or route of delivery make a difference in perinatal outcome? Obstet Gynecol 76(2):195-9, 1990
4- Lewis DF, Towers CV, Garite TJ, Jackson DN, Nageotte MP; Major CA: Fetal gastroschisis and omphalocele: is cesarean section the best mode of delivery? Am J Obstet Gynecol 163(3):773-5, 1990
5- Lurie S, Sherman D, Bukovsky I: Omphalocele delivery enigma: the best mode of delivery still remains dubious. Eur J Obstet Gynecol Reprod Biol 82(1):19-22, 1999
6- Raynor BD, Richards D: Growth retardation in fetuses with gastroschisis. J Ultrasound Med 16(1):13-6, 1997
7- Bethel CA, Seashore JH, Touloukian RJ: Cesarean section does not improve outcome in gastroschisis. J Pediatr Surg 24(1):1-3, 1989
8- Langer JC, Khanna J, Caco C, Dykes EH, Nicolaides KH: Prenatal diagnosis of gastroschisis: development of objective sonographic criteria for predicting outcome. Obstet Gynecol 81(1):53-6, 1993
9- Lenke RR, Persutte WH, Nemes J: Ultrasonographic assessment of intestinal damage in fetuses with gastroschisis: is it of clinical value? Am J Obstet Gynecol 163(3):995-8, 1990
10- Alsulyman OM, Monteiro H, Ouzounian JG, Barton L, Songster GS; Kovacs BW: Clinical significance of prenatal ultrasonographic intestinal dilatation in fetuses with gastroschisis. Am J Obstet Gynecol 175(4 Pt 1):982-4, 1996
11- Adra AM, Landy HJ, Nahmias J, Gomez-Marin O: The fetus with gastroschisis: impact of route of delivery and prenatal ultrasonography. Am J Obstet Gynecol 174(2):540-6, 1996

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:

1- Lugo-Vicente HL: Gallbladder Dyskinesia in Children. Journal of Society of Laparoendoscopic Surgeons 1(1):61-65, 1997

2- Curran TJ, Uzoaru I, Das JB, Ansari G, Raffensperger JG: The effect of cholecystokinin-octapeptide on the hepatobiliary dysfunction caused by total parenteral nutrition. J Pediatr Surg 30(2): 242-247, 1995

3- Teitelbaum DH, Han-Markey T, Schumacher RE: Treatment of parenteral nutrition-associated cholestasis with cholecystokinin-octapeptide. J Pediatr Surg 30(7): 1082-1085, 1995

4- Moss L, Amii LA: New Approaches to understanding the etiology and treatment of total parenteral nutrition-associated cholestasis. Sem Pediatr Surg 8(3): 140-147, 1999

5- Rintala RJ, Lindahl H, Pohjavuori M: Total parenteral nutrition-associated cholestasis in surgical neonates may be reversed by intravenous cholecystokinin: A preliminary report. J Pediatr Surg 30(6): 827-830, 1995

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