Gastric Volvulus

Gastric volvulus (GV) is a rare cause of regurgitation, retching or vomiting in children produced by an abnormal rotation of one part of the stomach around another. The volvulus can be either organoaxial if it develops along a line joining the hiatus and pylorus, mesenteroaxial if it occurs along the greater and lesser curvature axis (most common form seen in children), or a combination of both. GV may be idiopathic or secondary to other disorder and may be acute or chronic in nature. Most GV are secondary to deficient fixation of the gastric ligaments, acute in nature and associated to another underlying disorder, most commonly a diaphragmatic defect (eventration or hiatal hernias). Other associations include asplenia, pre-duodenal portal vein and previous Nissen fundoplication. The main symptoms of secondary GV are vomiting and respiratory difficulty whereas those of idiopathic GV are abdominal distension, pain and weight loss with or without failure to thrive. Borchardt’s triad (unproductive vomiting, epigastric distension, and inability to pass NG tube) is not always present in the pediatric patients. The diagnosis is suspected with plain abdominal films (large air-fluid level) and confirmed with upper contrast GI studies. A nasogastric tube will often relieve the acute situation. Surgical therapy is mandatory for the simultaneous correction of both the anatomical defects of gastric fixation and malposition and the diaphragmatic problem. Through an abdominal approach the stomach is decompressed, the volvulus is untwisted, the associated anomaly and cruras are repaired, and gastric fixation (gastropexy) is done. Anterior gastropexy in at least two places will prevent recurrent volvulus; gastrostomy alone may be followed by recurrence. Laparoscopy can accomplish this task.

References
Giant Cystic Meconium Peritonitis

Giant Cystic Meconium Peritonitis (GCMP) is an unusual form of intrauterine bowel perforation. The bowel perforation persists after birth and the peritoneal cavity becomes transformed into a huge meconium-filled giant pseudocyst with a thick membrane lining. The correct diagnosis can be reached either prenatally or soon after birth with the help of Ultrasonography or simple abdominal films respectively. Characteristically, a large cyst with dense calcifications or eggshell appearance is diagnostic. Intrabdominal calcifications once thought pathognomonic of meconium peritonitis can also be seen in other conditions such as hamartomas, adrenal hemorrhage or hemangiomas. Clinically the baby shows severe abdominal distension, bilious vomiting and failure to pass meconium. Delay in diagnosis may colonize and infect the pseudocyst creating a septic atmosphere. Associated cystic fibrosis should always be thought. The etiology of GCMP might be an intrauterine bowel atresia perforation, volvulus, intussusception or vascular accident. Remaining bowel is short. Management consists of decortication or removal of the cyst membrane with re-anastomosis of the GI tract if the surrounding inflammation permits. Other times temporary enterostomy may be necessary. The prognosis once dismal has improved in the last twenty years.

References
5- Neuhauser EBD: The roentgen diagnosis of fetal meconium peritonitis. AJR 51: 421-424, 1944

Congenital Mesoblastic Nephroma

Congenital Mesoblastic Nephroma (CMN), a fetal renal hamartoma, is the most common renal tumor in neonates. Usually presenting as an asymptomatic abdominal mass occupying most of the involved kidney, CMN is characterized by benign clinical behavior, gross appearance similar to uterine fibroids, and excellent prognosis after removal. Prenatally diagnosed CMN may be associated with polyhydramnios, low birth weight and premature labor. Large tumors might benefit from cesarean section. CT Scan shows a solid neoplasm with intrarenal distortion of the collecting system. Microscopically, typical CMN is composed of sheets of spindleshaped cells. Atypical CMN (cellular variant) is a potentially more aggressive tumor composed mainly of primitive mesenchymal cells, but
also usually contains varying numbers of differentiating fibroblasts and myofibroblasts. Management of CMN consists of simple nephrectomy with lymph node sampling to detect rare cases simulating malignancy.

References

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