Neonatal Intestinal obstruction

An essay

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Introduction

It is an arrest of downward flow of intestinal content in a baby of less than one month age. 99% of healthy full-term neonates pass their first bowel motion or meconium within 24 up to 48 hours. With preterm infants, the time can extend up to 9 days. Neonatal intestinal obstruction is not quite common, but creates serious and challenging problem for the surgeon and it occurs in 1/1500 live births, ***(Hernanz, 1999)***.

Developmental defects, abnormalities of peristalsis, abnormal intestinal contents, insults in utero after the formation of normal bowel are most of the causes sharing in the etiology of this problem, ***(YiSQ et al, 2004).***

Failure to pass meconium, progressive abdominal distention, refusal to feed and vomiting of bilious intestinal contents are the classic clinical signs of intestinal obstruction in neonates. Abdominal examination often reveals distended loops of bowel, which may be visible or palpable. Anal inspection is essential to exclude the presence of ano-rectal anomalies, ***(Kiely, 2006).***

The physiological characteristics of the neo-born infant, especially the premature, are such that optimal conditions and facilities required for survival. So early diagnosis and proper management of surgically treatable lesions are of great importance for there will be less stress upon the meagerness of metabolic and respiratory reserves, of such small patients, ***(Mason and Minkes,* 2002**).

The differential diagnosis for small bowel obstruction in neonates includes duodenal atresia, malrotation and volvulus,

jejunoileal atresia, meconium ileus and meconium peritonitis. Bilious vomiting, with or without abdominal distention, is usually the first sign of small bowel obstruction. The differential diagnosis for large bowel obstruction in neonates includes Hirschsprung's disease, ano-rectal malformations and meconium plug syndrome, ***(Kimura et al, 2000).***

In many cases of suspected neonatal intestinal obstruction, the clinical history and physical examination combined with plain abdominal radiographs and some time contrast enema radiograph, clench the diagnosis of emergency neonatal intestinal obstruction. Ano-rectal manometry and rectal biopsy eventually yield the definitive diagnosis of motility dysfunction in some special cases. The most difficult management decision is to decide between conservative management and emergency surgery. Ideally, all newborns suspected of having bowel obstruction should receive treatment at a center where an experienced surgeon minded by these some special problems, is available, ***(Hernanz, 1999)***.

Divisions of the Gut Tube

As a result of cephalocaudal and lateral folding of the embryo, a portion of the endoderm-lined yolk sac cavity is incorporated into the embryo to form the primitive gut. Two other portions of the endoderm-lined cavity, the yolk sac and the allantois, remain outside the embryo.

In the cephalic and caudal parts of the embryo, the primitive gut forms a blind-ending tube, the foregut and hindgut, respectively. The middle part, the midgut, remains temporally connected to the yolk sac by means of the vitelline duct, or yolk stalk ***(Skandalakis et al, 1994)***.

Development of the primitive gut and its derivatives is usually discussed in four sections: (a) The pharyngeal gut, or pharynx, extends from the buccopharyngeal membrane to the tracheobronchial diverticulum; since this section is particularly important for development of the head and neck. (b) The foregut lies caudal to the pharyngeal tube and extends as far caudally as the liver outgrowth. (c) The midgut begins caudal to the liver bud and extends to the junction of the right two-thirds and left third of the transverse colon in the adult. (d) The hindgut extends from the left third of the transverse colon to the cloacal membrane. Endoderm forms the epithelial lining of the digestive tract and gives rise to the parenchyma of glands, such as the liver and pancreas. Muscle, connective tissue, and peritoneal components of the wall of the gut are derived from splanchnic

mesoderm. Differentiation of the various regions of the gut and its derivatives depends on a reciprocal interaction between the endoderm (epithelium) of the gut tube and the surrounding splanchnic mesoderm. Mesoderm dictates the type of structure that will form, for example, lungs in the thoracic region and colon from the hindgut region, probably through a HOX code similar to the one that establishes the body axis. However, HOX expression requires induction by sonic hedgehog expression in the endoderm; thus the reciprocal interaction ***(Sadler, 2000)***.

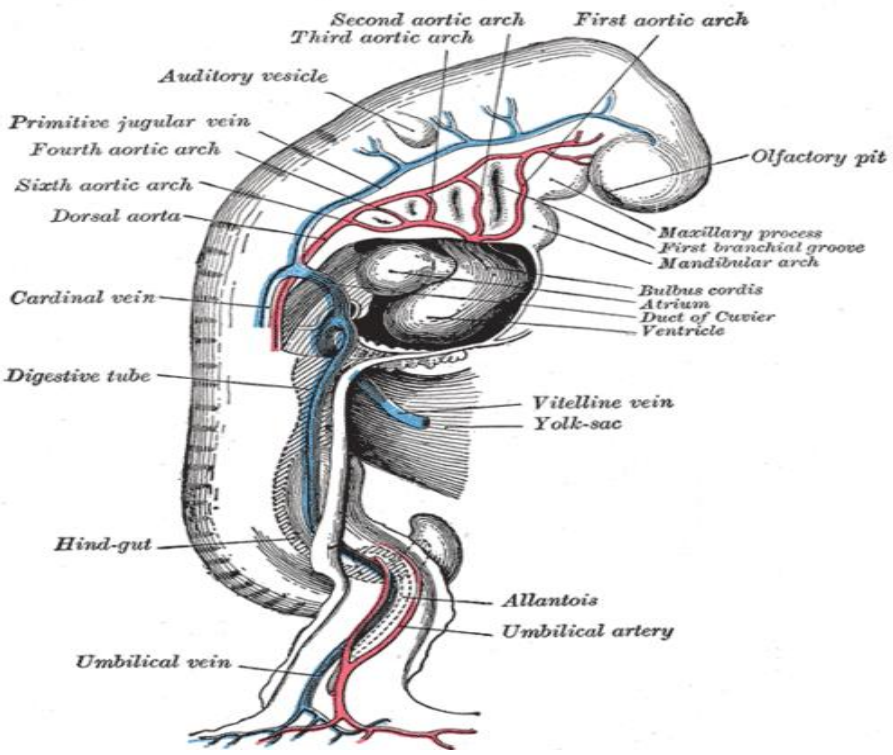

Figure 1: ***(Gray472. From Wikipedia, the free encyclopedia).*** **Profile view of a human embryo estimated at twenty or twenty-one days old. (Hindgut labeled at lower left.)**

Mesenteries

Portions of the gut tube and its derivatives are suspended from the dorsal and ventral body wall by mesenteries, double layers of peritoneum that enclose an organ and connect it to the body wall. Such organs are called intraperitoneal, whereas organs that lie against the posterior body wall and are covered by peritoneum on their anterior surface only (e.g., the kidneys) are considered retroperitoneal. Peritoneal ligaments are double layers of peritoneum (mesenteries) that pass from one organ to another or from an organ to the body wall. Mesenteries and ligaments provide pathways for vessels, nerves, and lymphatics to and from abdominal viscera ***(Kluth et al, 2000)***.

Initially the foregut, midgut, and hindgut are in broad contact with the mesenchyme of the posterior abdominal wall. By the fifth week, however, the connecting tissue bridge has narrowed, and the caudal part of the foregut, the midgut, and a major part of the hindgut are suspended from the abdominal wall by the dorsal mesentery, which extends from the lower end of the esophagus to the cloacal region of the hindgut. In the region of the stomach it forms the dorsal mesogastrium or greater ornentum; in the region of the duodenum it forms the dorsal mesoduodenum; and in the region of the colon it forms the dorsal mesocolon. Dorsal mesentery of the jejunal and ileal loops forms the mesentery proper ***(Skandalakis et al, 1994)***.

Ventral mesentery, which exists only in the region of the terminal part of the esophagus, the stomach, and the upper part of the duodenum, is derived from the septum transversum. Growth of the liver into the mesenchyme

of the septum transversum divides the ventral mesentery into *(a)* the lesser omentum, extending from the lower portion of the esophagus, the stomach, and the upper portion of the duodenum to the liver, and *(b)* the falciform ligament, extending from the liver to the ventral body wall ***(Kluth et al, 2000)***.

Foregut

Development of the duodenum:

The terminal part of the foregut and the cephalic part of the midgut form the duodenum. The junction of the two parts is directly distal to the origin of the liver bud. As the stomach rotates, the duodenum takes on the form of a C-shaped loop and rotates to the right. This rotation, together with rapid growth of the head of the pancreas, swings the duodenum from its initial midline position to the left side of the abdominal cavity. The duodenum and head of the pancreas press against the dorsal body wall, and the right surface of the dorsal mesoduodenum fuses with the adjacent peritoneum. Both layers subsequently disappear, and the duodenum and head of the pancreas become fixed in a **retroperitoneal position.** The entire pancreas thus obtains a retroperitoneal position. The dorsal mesoduodenum disappears entirely except in the region of the pylorus of the stomach, where a small portion of the duodenum **(duodenal cap)** retains its mesentery and remains intraperitoneal ***(Kanard et al, 2005)***.

During the second month the lumen of the duodenum is obliterated by proliferation of cells in its walls. However, the lumen is recanalized shortly thereafter. Since the **foregut** is

supplied by the **celiac artery** and the midgut is supplied by the **superior mesenteric artery,** the duodenum is supplied by branches of both arteries ***(Poki et al, 2005).***

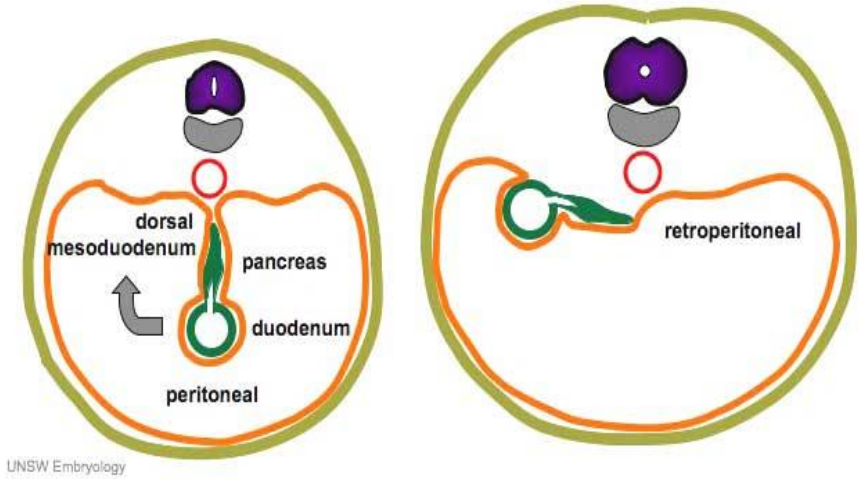

Figure 2: ***(Rubin. 2007)***. Rotation of the duodenum

Duodenal Stenosis :

Partial occlusion of the duodenal lumen, duodenal stenosis usually results from incomplete recanalization of the duodenum resulting from defective vacuolization. Most stenoses involve the horizontal (third) and/or ascending (forth) parts of the duodenum. Because of occlusion, the stomach´s contents (usually bile) are often vomited ***(Kimble et al, 2007)***.

Duodenal Atresia

Complete occlusion of the lumen of the duodenum "duodenal atresia" is not common. 20-30% of affected infants have Down syndrome and an additional 20% are premature. In about 20% of cases, the bile duct enters the duodenum just distal to the opening of the hepatopancreatic ampulla. During duodenal development, the lumen is completely occluded by epithelial cells. If re-formation of the lumen fails to occur, a short segment of the duodenum is occluded. Investigation of