MANAGEMET OF

Neonatal Intestinal Obstruction

Essay

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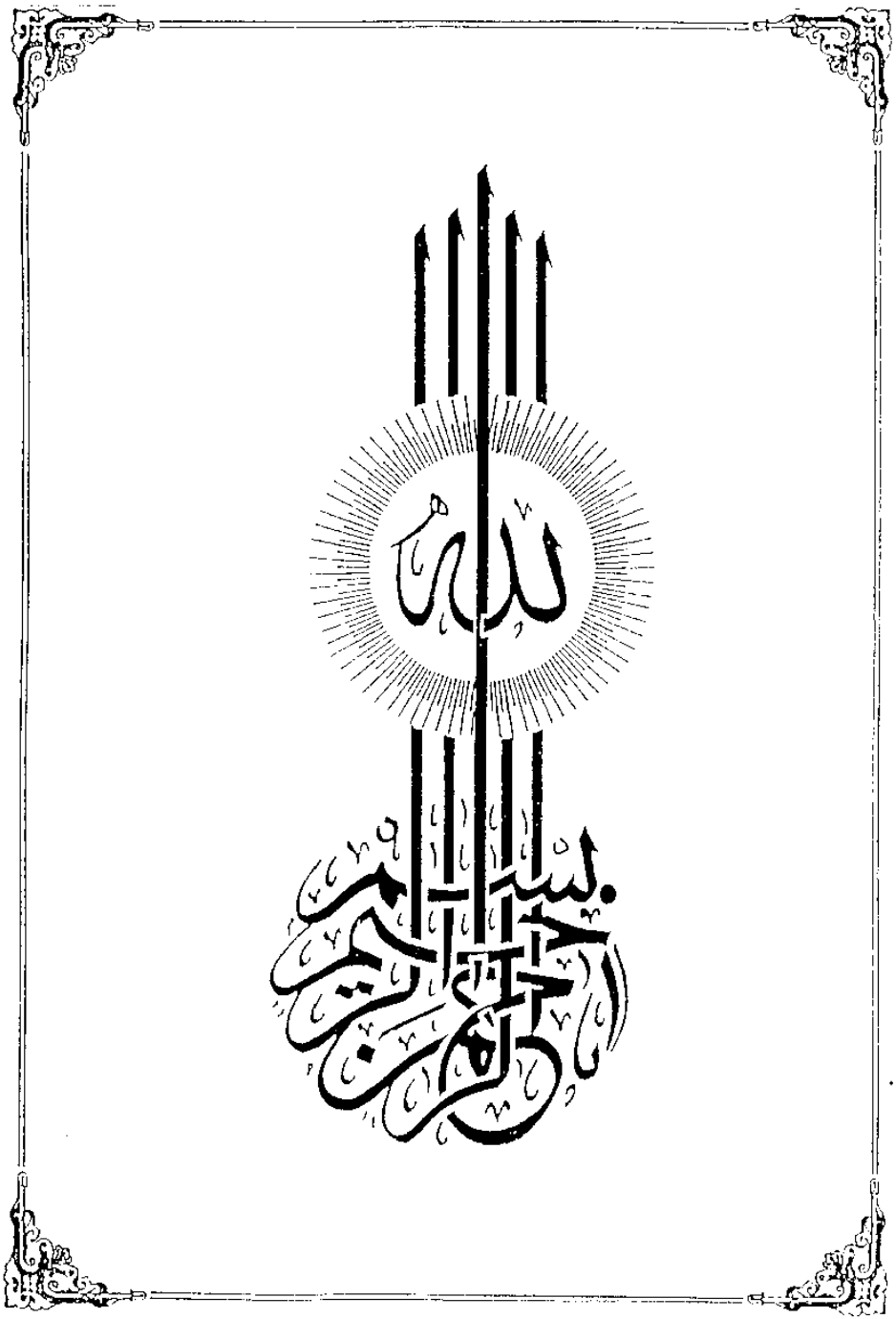

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NEONATAL INTESTINAL OBSTRUCTION
CLASSIFICATION & ETIOLOGY

For purpose of discussion, the neonatal period is considered to extend to the end of the fourth week of extrauterine life. A useful classification of the causes of obstruction is shown in the following outline, (Adapted from Fanaroff and Martin, 1983).

A) Mechanical: **(Occlusive)**

1. Congenital:

a) Intrinsic:

Atresia and stenosis

Meconium ileus

Hypertrophic pyloric stenosis

Meconium plug syndrome

b) Extrinsic:

Malrotation

Segmental volvulus

Peritoneal bands

Incarcerated hernia

Annular pancreas

Duplication

Meconium peritonitis

2. Acquired:

a) Intrinsic:

Intussusception

Milk curd obstruction

b) Extrinsic:

Adhesions

Bacterial peritonitis

After bowel perforation

Postoperative

B) Non - mechanical: (Non-occlusive)

1. Congenital:

a) Neurogenic:

Congenital aganglionosis

b) Prematurity

2. Acquired:

a) Neurogenic:

Paralytic ileus from cerebral injury

b) Functional:

Infection

Peritonitis - bowel perforation.

Respiratory distress syndrome.

Maternal drugs especially narcotic addiction.

C) Othe less common causes:

1. Mechanical:

Mesenteric cysts.

Vitelline duct remnant.

2. Functional:

Gastroenteritis.

Bowel hypoxia

Severe hypokalaemia

Adrenal insufficiency.

Hhypermagnesaemia.

Idiopathic obstruction.

Possible Etiology:

There are many different causes of various forms of neonatal bowel obstruction. They may be thought of as genetic, developmental and accidental. Accidental group refers to such acute intrauterine catastrophies as intra-uterine volvulus (Lyrenas, et al., 1982), Intussusception (Hayashida, et al., 1984), strangulation by omphelocele (Shegomoto et al., 1982), vascular accidents as thrombosis (Louro and Bernard, 1955), and

strangulation in gastroschisis defect (Grosfeld, 1970).

In many of the cases, genetic and acquired factors are combined. For example, the intestinal volvulus that may be associated with the meconium ileus in mucoviscoidosis. The history of maternal viral infections, Hormonal administration or toxic influences during the course of pregnancy may relate directly to the type of the lesion and to its management and prognosis. In addition to the previous causes, severe paralytic ileus may be due to a number of lesions e.g. cerebral injury, uraemia, enteric infections with pseudomonas and generalized sepsis of varied etiology. Occasionally an infant is encountered in whom segmental dilatation of the ileum or spasm in the terminal ileum has led to intestinal obstruction in the absence of any other obvious abnormality (Brown & Carty, 1984). There are some studies suggesting an increased incidence of gastrointestinal atresia in relation to the mother's occupation especially in women who work in laboratories during pregnancy (Ericson et al., 1982).

DUODENAL OBSTRUCTION

PATHOLOGY:

Obstruction of the lumen of the duodenum can be caused by several factors that may be either intrinsinc or extrinsinc and each of them may be partial or complete. The extrinsinc causes compress the lumen from outside. Among the famous causes of extrinsinc obstruction are: peritoneal bands, e.g. Ladd's bands, Preduodenal portal vein (due to mal rotation of the duodenum), Abberant pancreatic tissue in the duodenal wall, and Annular pancreas. The extrinsinc obstruction may occur at any level of the duodenum, but is mostly seen in the vicinity of the ampulla of Vater. In about 40% of cases it is pre-ampullary and in 60% of cases it is post ampullary (Rickham 1970). It may take the form of a mucosal web or a diaphragm with or without opening or may be due to atresia. Duodenal atresia as described by Gray and Skandalakis is classified into three groups:

In the first group there is an intact diaphragm or membrane formed of mucosa and submucosa, in the second group there are two blind ends of the duodenum connected by a fibrous cord along the intact end of the mesentery, in the third group the two blind ends of the duodenum are seperate and the mesentery shows a V shaped defect. In a few cases there is a T-shaped obstruction at the junction of the bile duct with the stenosed duodenum (Rickham 1970).

In annular pancreas, there is a thin flat segment of the pancreatic tissue surrounding the duodenum either completely or partially causing either partial or complete obstruction (Hope 1954 and Kiesewetter & koop 1954). This ring of pancreatic tissue is thought to be due to persistent ventral premordium of the pancreas which normally rotates around the duodenum to join the dorsal bud to become the head of the pancreas. Commonly the annular pancreas is associated with either stenosis or atresia of the duodenum at the same level (Elliot et al.,1968).

Annular pancreas is often classified as a cause of extrinsinc obstruction of the duodenum (Lynn,1963), but strictly speaking, this is not true as the lumen of the dudenum within the collar of the annular pancreas is not simply compressed by the pancreatic tissue, but on cross section the annular pancreas surrounds a stenosed segment of the duodenum (Rickham 1954).

Over half the patients with duodenal atresia have other congenital anomalies. Down's syndrome is present in up to 30% of cases and requires accurate preoperative assesment and full parental consultation prior to surgical correction. The frequency of associated anomalies is: Down's syndrome 30%, Incomplete rotation of the midgut 30%, Congenital cardiac disease 20%, Genitourinary anomalies 10%, Oesophageal atresia 8%, Anorectal malformations 7%, and Skeletal anomalies 6%. (Lewis Spitz 1985).

EMBRYOLOGY:

In 1900, Tandler propsed his theory of mucosal proliferation according to which, during the fifth and sixth weeks intrauterine life the mucosal cells of the duodenum proliferates at such a rate that the lumen of the intestine is completely obliterated, this is followed by a process of recanalization which recanalizes the gut and is completed by the eighth to tenth weeks of intrauterine life. Failure of complete recanalization would produce atresia or stenosis of the intestine. In 1913, Forssner produced histological proof of this theory by showing epithelial proliferation and occlusion of the duodenum in a 2.9mm. long embryo. More recently, however, it has been discovered that in only one third of the cases does the duodenum pass through the solid stage (Louw, 1952). A vascular catastrophe was suggested as a responsible cause (Lynn & Espinar, 1959). Such prenatal incidents as intussusception or volvulus could cause ischaemia and hence disappearance of part of the intestine (Louw, 1959).

A number of theories have evolved to explain the formation of annular pancreas on an embryological basis. The ventral pancreatic anlage with the

duct of Wirsung normally rotates with the duodenum untill it fuses with the dorsal pancreatic anlage and its duct, the duct of Santorini, to form the head of the pancreas. An anastomosis then occurs between the ducts of Wirsung and Santorini. Lecco in 1917 postulated that if the free end of the ventral anlage is fixed, it will be drawn around the right side of the duodenum to fuse with the head of the pancreas, thus encircling the duodenum. According to Tieken the condition is caused by two ventral anlages fusing with the dorsal anlage to form a ring round the pancreas. Baldwin, on the other hand, believed that persistence of the left ventral anlage, which normally atrophies, is responsible.

INCIDENCE:

It is a rare condition occuring once in 20.000-40.000 (Lynn,1963). Sixty percent of infants with duodenal obstruction were born premature (Rickham,1970).

CLINICAL PICTURE:

History of maternal poly-hydramnios is evident in about 50% of cases (Flowers,1983) as the amniotic fluid swallowed by the fetus is absorbed in the distal small intestine, so that an obstruction high up in the duodenum results in accumulation of amniotic fluid in an abnormal manner (Lloyd,1958).

Vomiting of bile stained material, often within a few hours of birth, is the most common and often the earliest sign in infants with duodenal obstruction. Vomiting is copious and forceful but rarely projectile. It may be delayed for 12 hours or longer. The vomitus may be colorless if the obstruction is above the level of the ampulla of Vater. In this case the diagnosis is difficult as vomiting of some gastric contents is frequently seen in newborn infants.

Abdominal distention is not the common finding in these infants. A fullness in the epigastrium due to dilated stomach is seen in 25% of cases. Visible gastric peristalsis is of little diagnostic value as it may be occa-

sionally seen in normal infants. The abdomen may be scaphoid because of the absence of the gas in the intestine.

Theoratically speaking, absence of meconium stools is absolute in cases of atresia. In fact, however, the passage of one or two small and dry meconium stools is not uncommon but this is not always the case (Gross,1953).

Rarely, obstructive jaundice may occur specially in annular pancreas (Mackenzie et al.,1960).

Later signs such as dehydration, weight loss, and change in blood chemistry result from profuse and continous vomiting and soon become apparent.

DIAGNOSIS:

Diagnosis is made by a simple upright film of the abdomen. In cases of duodenal atresia, the classical picture is that of (double bubble) sign. A large air distended stomach with a fluid level and a markedly distended first portion of the duodenum with a fluid level and absence of air from the remaining G.I.T. This may be obscured if there is considerable fluid in the stomach and duodenum. If the stomach is aspirated and 60 cc. of air are instilled by a nasogastric tube, a spectacular double bubble sign is seen. In duodenal stenosis, the proximal duodenum is less distended and some air will be observed in the lower intestine.

Annular pancreas may show itself in the X ray plain films as two areas of gas, one above the other, seperated by a narrow opaque band. However, this is not the common picture and the commonest is the picture of duodenal atresia described above (Koop 1953). It is to be noted that barium should not be given for fear of vomiting and aspiration of the vomitus.

Prenatal diagnosis of duodenal atresia has become common with the routine use of maternal ultrasonography (Hayden et al. 1983) for complications such as polyhydramnios in the third trimester of pregnancy. The obstructed duodenum of the fetus can easily be seen as a large, fluid filled,

cystic masses. The ultrasonographic appearance of normal fetal stomach may be confused with duodenal atresia when the incisura angularis is prominent. Such an erroneous diagnosis can be avoided by noting the absence of polyhydramnios and by assessing the fetal stomach in planes which traverse its long axis with real time scanning (Gross & Filly,1982).

In the past, microscopical examination of the meconium for cornified epithelial cells swallowed by the infant along with the amniotic fluid in utero, the so called Farber's test, used to be carried out in order to differentiate complete and incomplete intestinal obstruction. This test is no longer used nowadays as cornified epithelial cells are frequently present in the meconium of infants suffering from intestinal atresia.

*** Pre operative Management:**

The patient is usually admitted within the first two days of life. If the child is in a good condition and has no other life threatening anomalies, surgical correction may be under-taken immediately. If volvulus has been excluded, operation may be postponed as long as necessary to evaluate an infant with a severe cardiac anomaly, severe metabolic imbalance, or respiratory distress syndrome.

Nasogastric tube is placed with intermittent suction. Replacement of water and electrolytes is initiated. lmg. vit. K, as well as penicillin and streptomycin are given parenterally. The body temprature is controlled by foments.

Observation of the skin, muscle tone, moisture of the lips and tongue, infant's cry and noting the wet diaper is also of great importance.

Once the infant has reached the operating room, a slow drip of blood is the best supportive measure.

*** Operation:**

The area of obstruction of the duodenum is easily approached through a supra umbilical right transverse abdominal incision running from a point 1 cm. to the left of the midline into the right flank. The ascending colon

and hepatic flexure of the colon are mobilized and the duodenum is exposed. The entire length of the duodenum is inspected and the point of obstruction is determined. The choice of the operation depends on the type of anomaly encountered.

A simple duodenal web or diaphragm is excised through a longitudinal incision across the area of obstruction. Care must be taken in excising the web as the opening of the ampulla of Vater is frequently in the web itself. Cautery or suturing of the mucosa of the web is contraindicated because of the possible damage of the entrance of the bile duct. The vertical incision is closed transversely, to enlarge the lumen and prevent stricture, with 5/0 or 6/0 silk sutures. If a wind-sock anomaly is suspected in which the elongated web may produce distention of the duodenum several centimeters beyond the attachment of the diaphragm to the duodenal wall, an intra luminal nasogastric tube may be used to find out this point. Pressure on the tube at the bottom of the web produces an indentation in the duodenal wall indicating the point of attachment, and the incision should be placed at that point.

Previously, the standard procedure for duodenal atresia and stenosis was duodenoejejunostomy. A retrocolic and isoperistaltic anastomosis was the procedure of choice. (Koop,1953). A similar procedure had been used for annular pancreas. (Gross and Chisholm 1944). A loop of jejunum was pulled through an opening in the right transverse mesocolon and anastomosed to the most dependent portion of the dilated duodenum using two layer anastomosis of 5/0 chromic catgut in the mucosal layer and 5/0 black silk suture in the seromuscular layer. The anastomosis was then pulled through the opening in the mesocolon and the duodenal wall sutured to the mesocolon to avoid constriction of jejunal limbs by the mesocolic ring. The temptation to divide the pancreatic ring should be resisted because there is often an intrinsic duodenal obstruction, also some of the pancreatic tissue may be intramural and pancreatic fistula or pancreatitis may result.

Duodenoduodenostomy being a more physiological bypass procedure is considered now the operation of choice in intrinsic duodenal obstruction. A transverse incision is made in the lowest portion of the dilated proximal duodenum and a vertical incision in the smaller distal segment.

Multiple atresias or webs are uncommon, but, before completing any of these anastomoses, a small catheter should be threaded into the distal small intestine and saline injected until it reaches the colon, thus ensuring patency.

Gastro-jejunostomy as a means of bypassing the duodenal obstruction should never be done because of long term problems with marginal ulceration, bleeding, and stricture of the anastomosis.

Excision of the diaphragm only is not satisfactory as fibrous annulous is still there always, but with shunt operation, the diaphragm must be excised, otherwise wind sock will occur and obstruct the stoma even many years after the operation. Establishing gastrostomy and trans anastomotic feeding tube is wise particularly in tiny infants.

*** Post-operative care:**

The patient should return to the incubator with suction either from a nasogastric tube or a gastrostomy tube. Characteristically, there is a prolonged period of duodenal ileus due to impaired peristalsis of the hypertrophied and dilated proximal duodenum and to anastomotic oedema. During this phase, when normal nutrition cannot be tolerated, feeds may be introduced into the distal intestine via the trans anastomotic tube. Gentle digital rectal examination several times a day is stimulating and is quite rewarding by the second day.

Fluid and elecrolytes homeostasis is maintained by maintainance intravenous fluid administration supplemented by replacement of nasogastric or gastrostomy aspirate. Also, parentral antibiotics and vit. are given.

Nasogastric tube can be removed after 36 hours but no oral feeding is yet allowed. A frequent reinsertion of the tube must ensure patency of the stoma to saliva and gastric juice for another 36 hours. After decrease in suction, oral 5% glucose can be started and followed by protein hydrolysate and feeding by small meals.