NEONATAL INTESTINAL OBSTRUCTION

Thesis

Submitted in Partial fulfilment of

Master Degree in Pediatrics

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1986

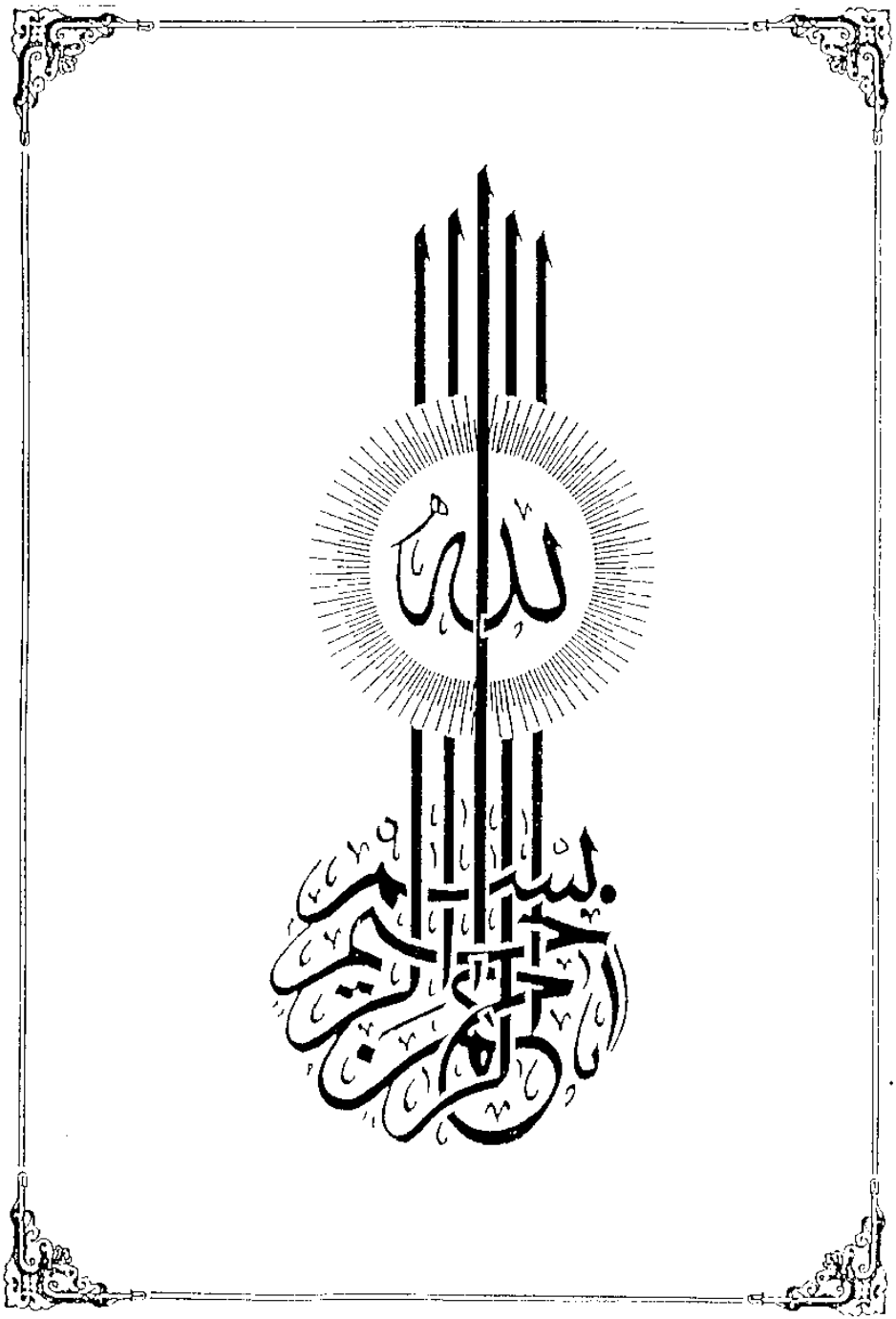
ACKNOWLEDGEMENT

I would like to express my profound gratitude to Professor Dr. Yehia El Gamal, Professor of Pediatrics, Ain-Shams University, for his generous help and valuable advice. His continuous stimulus made possible the achievement of this work.

I am particularly grateful to Dr. Iman Seoud, Assistant Prof. of Pediatrics, Cairo University for providing me the opportunity to accomplish this work.

I gain advantage to express my thanks to my colleagues in Poison Control Center for their endless help. I specially thank Dr. Sherif Sadek, Dr. Oussama Abou Zeid, and Dr. Samir Samy for sparing much time and effort to help me review this work.

Above all I'm deeply indebted to my mother but for her nothing would have come true.



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INTRODUCTION

When gastrointestinal luminal contents are pathologically prevented from passing distally, intestinal obstruction exists. intestinal obstruction may be caused by mechanical occlusion of the bowel or by paralysis of the intestinal muscles (Sabiston, D.C., 1981).

Intestinal obstruction in the newborn occurs from a variety of congenital diseases. These include duodenal atresia and stenosis, annular pancreas, malrotation with or without volvulus, meconium ileus, jejunal, ileal or colonic atresia, Hirschsprung's disease and imperforate anus. They are also susceptible to intestinal obstruction from internal hernias through mesenteric defects or congenital bonds from embryologic remnants such as Meckle's diverticulum.

Duplications of intestinal tract can cause obstruction at any age (Avery, G.B., 1981).

Intestinal obstruction is one of the most serious and alarming conditions that may face a surgeon, lest neonatal intestinal obstruction, this population of ever smaller size and weight put and add more challenge to the pediatrician and the pediatric surgeon alike. Intestinal obstruction is one of the surgical problems in neonates that continues to be an era that attracts considerable interest, also non-operative management is an integral part (Lobe & Schwartz, 1985). Current experience suggests that survival is now considerably

better and is likely to improve in the future, that improvement will rest in large part on a better understanding of that serious condition. Prompt recognition and diagnosis, followed by rapid operative and supportive therapy (Bell, M.J., 1985).

AIM OF THE WORK

The aim of this work is to emphasize the problem of neonatal intestinal obstruction, to explain its manifestation, and to introduce the possible aetiological factors, how to diagnose each one of them, the recent methods for diagnosis and how to treat it whether medically or surgically.

MANIFESTATIONS

MANIFESTATIONS OF NEONATAL INTESTINAL OBSTRUCTION

These will be categorized under the following items:

I) Vomiting

Intestinal obstruction is characterised by the early onset of vomiting, the higher the level of obstruction, the earlier will be the onset of vomiting (Thompson, T.R.,1983).

The causes of vomiting may be differentiated according to Sears 1981 by the presence or absence of bile staining. Non-bile stained vomitus is due to overfeeding, milk or formula intolerance, sepsis, central nervous system lesions, lesions above the ampulla of Vater, e.g., pyloric stenosis, upper duodenal stenosis or atresia and rarely annular pancreas. Bile-stained vomitus is due to duodenal atresia, or stenosis, intrinsic or secondary compression from an annular pancreas, peritoneal bands with or without malrotation, volvulus, aberrant superior mesenteric artery and preduodenal portal vein.

The presence of bilious vomiting in a neonate is always abnormal, although it may be caused by non-surgical disorders, such as sepsis, it must always be regarded as a surgical lesion until proved otherwise. A very early sign of obstruction that may even precede the onset of vomiting is the finding of gastric aspirate that is either bile stained or present in excess of the normal 15 ml. (Fanaroff & Martin, 1983).

Thompson 1983 stated that the stomach should contain less than 20 ml. of clear fluid at birth.

High intestinal obstruction is characteristic by vomiting which tends to be persistent even when feeding has been stopped. Distension may be absent, however, massive distension can be seen in high intestinal obstruction following perforation (Sunshine et al., 1983).

Low lesions causing gastrointestinal obstruction often will have delayed onset of vomiting, which is usually preceded by progressive abdominal distension. It has a rapid onset in the left upper quadrant when there is complete duodenal obstruction. Lower obstructions cause more generalized distension which varies with location (Sears, 1981). The distension may be massive even in the absence of perforation and is greatest in the more distal lesions (Fanaroff & Martin, 1983).

A distended abdomen is most oftenly the first sign of lower intestinal obstruction. A neonate suspected of having a duodenal obstruction would not have a distended abdomen, and the presence of distension in such a case suggests the presence of a volvulus or a more distal obstruction (Thompson, T.R., 1983).

II) Failure to Pass Meconium

In addition to abdominal distension and vomiting, newborns with distal intestinal obstruction often have little

or delayed passage of meconium, although delayed passage of meconium occasionally may be seen in non surgical disorders such as sepsis, prematurity, maternal narcotic administration hypothyroidism and hypermagnesemia (Sunshine et al., 1983).

Failure to pass meconium may also be the result of Hirschprung disease, meconium and mucous plug syndrome (Sears, 1981).

Obstipation is a late sign of an intestinal obstruction about 94% to 99% full term infants and 76% to 80% of premature infants pass stools in the first 24 hours of life, 100% of full term infants and 94% to 99% of premature, low birth weight infants pass stools about 36 hours after birth (Sherry & Kramer, 1955).

III) Peritonitis

Through the past decade, pediatric surgeons have been challenged by the improved survival of the neonatal population, primarily as a result of an increasingly skillful neonatal intensive care effort, this population of ever smaller size and weight has added considerably to the spectrum of neonatal disturbances presented to the pediatric surgeon, among these disorders has been that of the disaster of peritonitis (Bell M., 1985 a).

Bell M. at 1985 classified neonatal peritonitis into two main entities:

A. Primary

Which is the inflammation of the peritoneal surface unassociated with disruption of an abdominal viscous or the abdominal wall, this is rather not common in neonatal period.

B. Secondary

The secondary peritonitis is due to gastro-intestinal tract disruption. It is more important than the primary entity.

The burden of evidence suggests that the neonate, particularly the premature, does not have fully developed immune defense capability, while much of this evidence is controversial and study of this problem continues, it is probably appropriate to consider the neonate as a compromised host, much like individuals with cancer, malnutrition or transplant recipients receiving immuno-suppressive agents (Walker, 1976).

Both humoral and cellular mechanisms of immune defense have been shown to have limitations in the neonate, IgG is the only immunoglobulin that can cross the placental barrier. Maternally acquired IgG reaches its peak concentration at term and then is degraded at a rate of 2.5 per cent per day after birth, fetal production of IgG begins in the seventh gestational month, and after birth is at a much slower rate of production than the degradation rate of maternal IgG, therefore both the premature and the term neonate are likely to be deficient in IgG and the protection afforded by this

immunoglobulin. Similarly IgM, IgA are deficient in the preterm and term neonates (Allensmith et al., 1968).

Aberration in these aspects of immune status might contribute to susceptibility to perforation in the newborn. Several studies have shown abnormally increased serum IgA levels in neonates with gastrointestinal obstruction and necrotizing enterocolitis (Bell, M et al., 1985 b).

Functional activity of polymorphonuclear leukocytes (PNM's) include recognition of organisms, movement to the site of infection, adhesion to the involved organism and phagocytosis. It was found that PNM's from neonates with sepsis or other stress situations were less effective in bacterial killing, also they were more rigid possibly contributing to diminished movement and phagocytosis (Wright j. et al., 1975).

The adverse pathophysiologic effects of peritonitis have not been systematically evaluated in the human newborn. Certain factors that put the neonate at a disadvantage when dealing with perforation and peritonitis can be defined, localization of soilage from perforation is limited in the neonate by the normally diminutive size of the omentum, so that peritonitis tends to be generalized. The resultant, ileus and sequestration of electrolyte and protein-rich fluid in the peritoneal cavity and intestinal lumen is of relatively greater proportion than when perforation can be localized. The greater percentage of total body water that is

active in metabolism in the neonate implies that water loss due to peritonitis, ileus and emesis may contribute more significantly to hypervolaemia and diminished tissue perfusion and cell function. Since the newborn is heavily reliant on diaphragmatic ventilation, abdominal distension plays an important role in causing ventilatory embarrassment (Bell M., 1985 c).

Obturation of the lumen of the gut or even obstruction due to any cause may underlie perforation. Intestinal atresia, stenosis, meconium ileus, and Hirschsprung disease are relatively common obstructing lesion that may lead to perforation (Thompson T.R., 1983).

Meconium escapes into the peritoneal cavity and induces a severe chemical peritonitis followed by calcification of the meconium. Soon after birth the infant develops signs and symptoms of intestinal obstruction. Meconium is sterile during intrauterine life but rapidly becomes colonized with bacteria following birth. Continued intra-peritoneal leakage of meconium in the neonatal period through an unsealed perforation will result in a bacterial as well as chemical peritonitis (Fanaroff & Martin, 1983).

An abdomen tender to palpation, erythema of the abdominal wall, rapidly developing abdominal distension, uniformly tympanitic note on percussion with loss of the normally dull area of the liver, abdominal rigidity and induration usually signify peritonitis due to intestinal