

Report

Neonatal intestinal obstruction

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SUMMARY We reviewed 36 cases of neonatal intestinal obstruction admitted to our surgical unit over a 10-year period, 1986–1996, for surgical intervention following the failure of conservative treatment. There were more males than females and the age range was 12 hours–26 days. Imperforate anus was the main cause of the obstruction (27.8%) followed by duodenal atresia (13.9%) and colonic atresia and meconium ileus (11.1% each). There were 8 deaths following surgery (22% mortality rate), the main causes being aspiration pneumonia, septicaemia and hypothermia.

Introduction

The neonatal period is defined as the first 28 days after birth [1]. The majority of infants admitted to surgical units within these first 28 days of life are suffering from neonatal obstruction [2]. These cases require surgical management by paediatric surgeons in medical centres with facilities for anaesthesia, radiology and the specialized paediatric and nursing care such cases require for successful survival [1,2].

The principal feature of neonatal intestinal obstruction is bile-stained vomiting. Early vomiting, in the first 24 hours of life, indicates a high obstruction (duodenal or jejunal) while the later onset of vomiting indicates a lower obstruction (ileal or colonic) [2–9]. Failure to pass meconium (48% of normal neonates pass meconium in the first 24 hours of life) is symptomatic of lower intestinal obstruction [2–8]. The degree of abdominal distension correlates

roughly with the height of the intestinal obstruction. In duodenal and high jejunal obstruction the distension is restricted to the upper abdomen, whereas in ileal and colonic obstruction there may be massive, unrestricted distension [2–8]. Waves of peristaltic movement may be visible during an abdominal examination of a neonate with intestinal obstruction [2].

In Basra General Hospital, all neonatal surgical emergencies are admitted to general surgical units. This is a review of the cases of neonatal intestinal obstruction admitted to one surgical unit during the 10-year period 1986–1996.

Patients and methods

Over the 10-year period, 36 neonates with neonatal intestinal obstruction were referred from paediatric units to this general surgical unit. All patients were aggres-

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Received: 03/03/98; accepted: 03/09/98

sively managed on admission. Their dehydration and electrolyte imbalance was corrected by administration of intravenous fluids and a nasogastric tube; prophylactic broad-spectrum antibiotics were given pre-operatively and blood was prepared prior to surgery. In all cases conservative treatment initially attempted in the paediatric units had failed and as a result all the patients underwent surgery at the general surgical unit.

Results

Of the 36 patients, there were 27 males and 9 females. Their ages ranged from 12 hours to 26 days. All patients had a plain X-ray of the abdomen in the erect position (Figures 1 and 2) and a few suspicious cases, espe-

cially those with a high obstruction, had a barium study (Figures 3–6) with the barium meal administered through a nasogastric tube. Despite these measures, the actual causes of obstruction were not determined until after exploratory laparotomies were conducted.

There were eight deaths following surgery, a mortality rate of 22%. The causes of death were aspiration pneumonia, septicaemia, hypothermia and cardiac arrest.

Discussion

Neonatal intestinal obstruction is the most common emergency condition in neonates [1–9] and atresia is the most common cause of obstruction [1–6]. In this study, duodenal atresia was found in 13.8% of the pa-

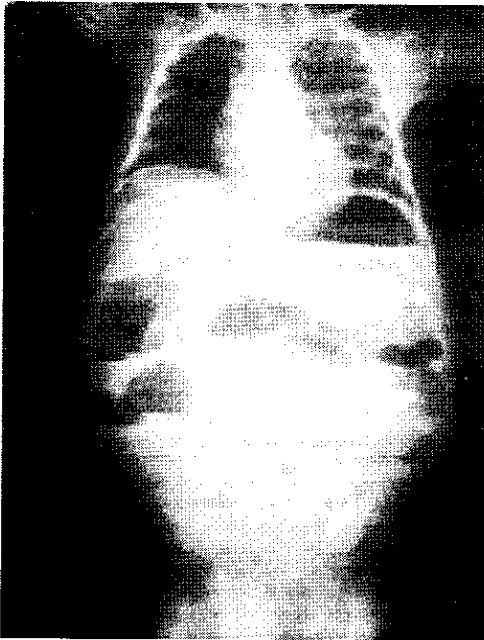


Figure 1 Plain abdominal X-ray (erect position) showing multiple fluid level

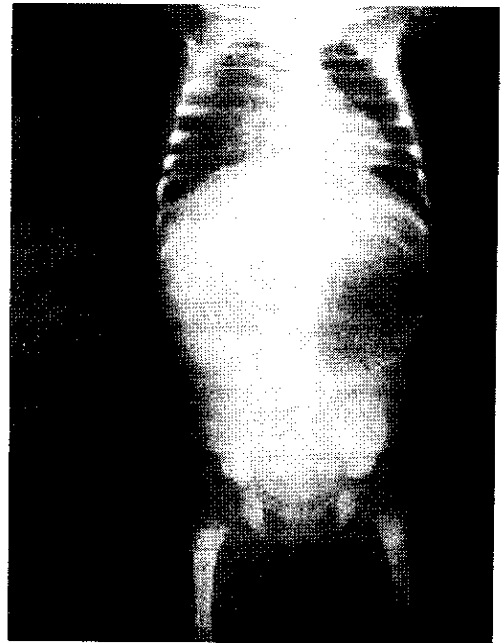


Figure 2 Plain abdominal X-ray (erect position) showing double bubble appearance

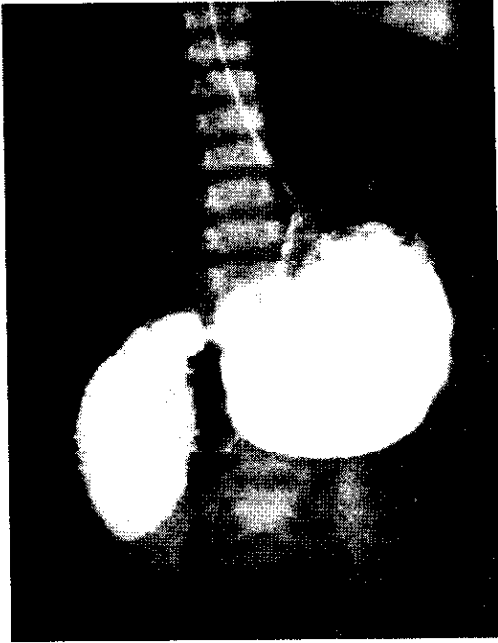


Figure 3 Barium abdominal X-ray showing duodenal atresia



Figure 4 Barium abdominal X-ray showing malrotation and Ladd band and pressure from outside across the second part of the duodenum

tients (5 cases) and the diagnoses were reached by radiological study. Plain X-rays of the abdomen in the erect position show double bubble appearance (as in Figure 2) and the barium meal results (Figure 3) show atresia [2-4]. Some cases of intestinal obstruction may be diagnosed in the uterus while using ultrasonography to check the fetus in cases of mothers with hydramnios. However, this type of investigation was not used in the cases we studied [10].

Duodenal atresia may be associated with annular pancreas but all the cases we studied were of the solid type with no association to annular pancreas [8]. Surgical management is by excision of the web where there is a web of mucosa or side-to-side duodenoduodenostomy where there is

complete segmental absence or solid cord (as in our cases) [1-6].

Malrotation of the bowel and Ladd band usually has the same symptoms as duodenal atresia and a barium meal (Figure 4) does not reveal the difference. However, a barium enema will show caecum in the left side of the abdomen or alternatively an explorative laparotomy may be required [1,2]. Two patients were found to have malrotation of the bowel and Ladd band (5.6%). Their diagnoses were only confirmed during laparotomy. In malrotation of the bowel, the Ladd band causes an obstruction to the duodenum in the second or third part, with or without adhesions of the remaining bowel [1-6]. Surgical treatment is by Ladd's procedure, which includes cutting



Figure 5 Barium follow-through X-ray showing jejunal atresia

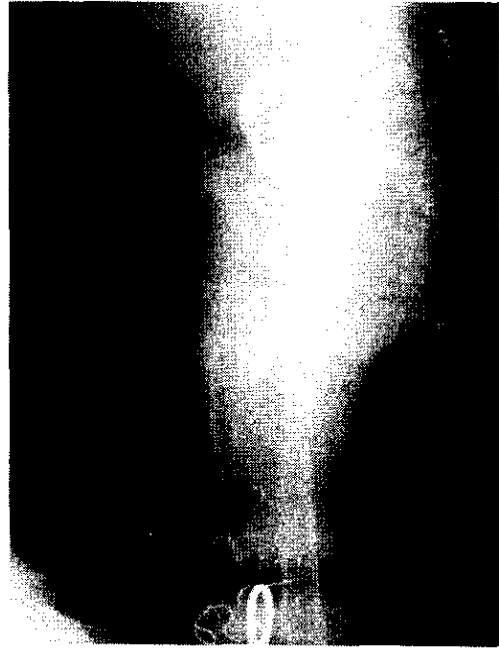


Figure 6 Barium follow-through X-ray showing anorectal anomalies, high type, and barium collected in the rectal pouch

the Ladd band, releasing the adhesion, cutting the ligament of Trietz and checking for intraluminal web [1,2,3,6]. In both patients, this procedure was successfully carried out.

Jejunal and ileal atresia both have symptoms of biliary vomiting and abdominal distension, although this is more prominent in ileal atresia. We diagnosed these radiologically with plain X-rays of the abdomen in the erect position showing the fluid level as in Figure 1 and the barium meal and follow-through showing the atresia as in Figure 5 (for jejunal atresia). Management is normally by resection of the atretic segment with end-to-end anastomosis, checking the whole bowel for another atresia [1,7,8]. Two patients with jejunal atresia and three with ileal atresia were suc-

cessfully treated. In colonic atresia there is a delay in biliary vomiting but a huge abdominal distension and failure to pass meconium [2,9]. With laparotomy it is possible to diagnose the atresia as either segmental colonic or whole colonic atresia. The management of segmental atresia of the colon is by resection of the atretic segment and end-to-end anastomosis; while in whole colonic atresia the management is by total colectomy and permanent ileostomy. The four cases in our study were of whole colonic atresia and required colectomy with permanent ileostomy [2,5,9]. The symptoms of meconium ileus are usually biliary vomiting and gross abdominal distension due to obstruction of the terminal ileum by thick, tenacious meconium and it

is difficult to differentiate it from ileal atresia. However, it can sometimes be differentiated by a plain X-ray of the abdomen revealing an absence of air/fluid level and ground-glass appearance due to the mixture of gas and meconium [1-4,12-17].

For uncomplicated cases of meconium ileus, a gastrographin enema may work as a therapeutic as well as diagnostic procedure. Gastrographin assists in liquifying the inspissated meconium which can then be evacuated. In complicated cases, such as those where there has been a perforation, treatment involves surgical resection of the obstructed or perforated segment, evacuation of the inspissated material, replacement to ensure bowel continuity and an ileostomy such as the Bishop-Koop operation, Santull operation or T-tube ileostomy; sometimes the appendicular stump is used for irrigation [1-4,12-17]. The four patients with meconium ileus were treated with the Bishop-Koop operation.

In cases of imperforate anus or anorectal agenesis, differentiation between the high and low types depends on the level of the levator ani muscle. A plain, lateral, inverted X-ray of the pelvis (24 hours after birth) may be helpful in establishing the level of agenesis. The three patients with low-level agenesis were managed by anoplasty and the seven patients with high-level agenesis were managed by initial defunctioning colostomy [1-4].

A further three patients with symptoms of intestinal obstruction (biliary vomiting and huge abdominal distension) had plain X-rays of the abdomen showing a fluid level but no pneumoperitonium (a feature of perforated viscus) and no pneumatosis intestinalis (a feature of necrotizing enterocolitis) [1-4]. Following exploratory laparotomies, one patient was found to have a jejunal perforation with meconium peritonitis which occurred 12 hours after a

Table 1 Distribution of patients by type of obstruction and etiological factors

Cause of obstruction	No.	%
Duodenal atresia	5	13.9
Malrotation and Ladd band	2	5.6
Jejunal atresia	2	5.6
Ileal atresia	3	8.3
Colonic atresia	4	11.1
Meconium ileus	4	11.1
Imperforate anus (3 low type, 7 high type)	10	27.8
Ileal obstruction (intussusception)	1	2.8
Splenic injury (with symptoms of intestinal obstruction)	1	2.8
Peritonitis (jejunal perforation due to birth trauma)	1	2.8
Peritonitis (necrotizing enterocolitis with ileal perforation)	2	5.6
Obstruction due to congenital diaphragmatic hernia	1	2.8

difficult home labour (birth trauma). The other two patients had full thickness necrosis with gangrene affecting the ileum and perforation near the ileocaecal valve causing peritonitis. Necrotizing enterocolitis was diagnosed. Cases of perforation and peritonitis were managed by anastomosis, resection of the affected segment and cleaning of the peritoneal cavity. The patient with jejunal perforation due to birth trauma survived while the patients with necrotizing enterocolitis and perforation of the terminal ileum and peritonitis died despite intensive care. Our mortality rate for patients with perforation and peritonitis was 66%, contrasting with findings in other studies of 33% [1,2,18-20].

Table 2 Mortality rate by etiological factor

Type of lesion	No. of cases	No. of deaths	Mortality rate (%)
Duodenal atresia	5	1	20
Ileal atresia	3	1	33
Colonic atresia	4	2	50
Meconium ileus	4	2	50
Necrotizing enterocolitis	2	2	100

Three other neonates had symptoms of intestinal obstruction and underwent exploratory laparotomies. The first patient was shown to have haemoperitonium and a splenic injury at its pedicle that required a splenectomy. Hocht has also reported two cases of ruptured spleens in neonates due to birth trauma which resulted in intestinal obstruction [21]. The second patient, who was 26 days old, had an ileocaecal intussusception and after milking the intussusception there was a mass at the terminal ileum requiring that segment to be resected with anastomosis. The histopathology of the resected segment revealed an ileal pancreas. Such intussusception in neonates is rare and indicates an underlying cause

Table 3 Distribution of the eight patients who died by cause of death

Cause of death	No.	%
Aspiration pneumonia	3	37.5
Septicaemia	2	25.0
Hypothermia	2	25.0
Cardiac arrest	1	12.5

[1,22]. The third patient had a diaphragmatic hernia with small bowel obstruction. The small bowel was reduced from the oesophageal hiatus and narrowing of the hiatus with Nissen's fundoplication was carried out [2,23].

Our mortality rate was 8 out of 38 neonates (22%). Tables 2 and 3 show the mortality level of each type of obstruction and the causes of death. As in other studies aspiration, pneumonia, sepsis and hypothermia were found to be the most significant causes of neonatal death [18,21]. Our experiences indicate that essential to improving success and survival in neonatal surgery and reducing morbidity and mortality is a fully equipped paediatric surgical unit with trained surgeons, anaesthetists and nursing staff.

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