Review Article

AN OVERVIEW OF NEONATAL INTESTINAL OBSTRUCTION: A REVIEW OF LITERATURE.

Udefiagbon EO, Ngwu SN

Department of Surgery, Irrua Specialist Teaching Hospital, Irrua, Edo, Nigeria

Abstract

Aim: To highlight the common causes, pathophysiology, clinical features, diagnostic investigations and treatment of the common differentials of neonatal intestinal obstruction.

Method: Review of literature was done which encompassed common causes, pathophysiology, clinical features, diagnostic investigations and treatment of the common differentials of neonatal intestinal obstruction.

Result: Neonatal Intestinal Obstruction, the inhibition of flow of intestinal content, is mostly caused by congenital surgical conditions which result in mechanical or functional obstruction. Mechanical obstruction could be intraluminal e.g. Meconium ileus/plug; intramural e.g. Atresia, Stenosis and extramural e.g. Bands. Common examples of functional obstruction include Hirschsprung's disease and Neonatal sepsis.

The pathophysiology results in hypovolaemia and bacteria-translocation. The clinical features include abdominal distension, constipation, bilious vomiting, dehydration, hypothermia/fever.

Prenatal ultrasound finding of polyhydramnios is suggestive of pathologies like duodenal atresia. Postnatal, diagnosis is confirmed by abdominal radiograph (bowel dilatation and air-fluid levels) or contrast studies e.g. corkscrew appearance in malrotation.

Treatment includes fluid-electrolyte resuscitation, nasogastric decompression, antibiotics, vital signs/hourly urine monitoring and definitive surgical procedure.

Conclusion: Neonatal intestinal obstruction is a common surgical emergency amongst neonates for which early diagnosis and intervention, as well as support care facilities and trained staff of different specialties are crucial to having a good outcome.

Keywords: Neonate, Intestine, Colon, Anorectum, Bowel, Obstruction, Congenital, Mechanical, Functional, Resuscitation.

INTRODUCTION

Neonatal intestinal obstruction is a condition in which there is an impediment to the flow of intestinal content distal to the Ampulla of Vater in neonates (birth-28days olds). It is the commonest indication for emergency surgery and a major surgical determinant of morbidity and mortality among neonates.[1,2,3] The incidence of neonatal intestinal obstruction is 1 in 2,000 live births.[4]

Neonatal intestinal obstruction may be complete as in atresia or incomplete as in stenosis and fenestrated mucosa web. Neonatal intestinal obstruction is regarded as

All correspondence to Dr. Ernest O. Udefiagbon, Department of Surgery, Irrua Specialist Teaching Hospital, Irrua, Edo state, Nigeria.
E-mail: dokernie11@gmail.com
Affiliation: Paediatric Surgery

high obstruction when the level of obstruction is proximal to the ileum, and as low obstruction when the level of obstruction is at the ileum, colon or anorectum.

The cause is mostly congenital conditions requiring surgery most of the times, albeit medical conditions which would not require surgery e.g. neonatal sepsis, congenital hypothyroidism, children of diabetic mothers, etc might be the problem sometimes. Some forms of neonatal intestinal obstruction may be detected from prenatal ultrasound scan.

The cardinal features of neonatal intestinal obstruction often include bilious emesis, abdominal distention and failure to pass meconium in 24 hours of life.[4,5] Jejunal atresia and other forms of high obstruction may have maternal polyhydramnios.[4]

Before 1950, the morbidity and mortality of neonatal intestinal obstruction was very high but this has improved over the last few years from significant advances in neonatal surgery with early (sometimes antenatal) detection and intervention along with good neonatal intensive unit care. [5,6]

Usually, an accurate history, a thorough physical examination and a plain abdominal radiograph is all that is necessary to make a diagnosis. The radiographic bowel gas pattern is distinctive for different cases and often points to the site of obstruction. Gastrointestinal contrast study may be done when indicated.

Devastating consequences like aspiration of vomit, sepsis, midgut infarction or enterocolitis result from failure to recognize neonatal bowel obstruction early enough. The outcome is very much dependent on appropriate timely diagnosis and treatment, as delay in surgical intervention may result in loss of long bowel segment with consequent short bowel syndrome.[7] Prematurity, associated severe congenital anomalies and complications of surgery are other factors that contribute to high mortality. [3,8,9]

Combined efforts by the paediatric surgeon, neonatologist and paediatric anaesthetist/intensivist are crucial to achieve favourable outcomes for these patients.

METHODS

An overview of neonatal intestinal obstruction in terms of common aetiology, pathophysiology, clinical features, investigations and treatment was undertaken through a detailed literature review by Google search.

RESULT

The results from literature review for aetiology, pathophysiology, clinical features, investigations and treatment of neonatal intestinal obstruction is as follows.

Aetiology

The aetiology of neonatal intestinal obstruction is usually congenital developmental anomalies which may be due to genetic factors, in utero maternal environmental exposure, mesenteric vascular accident and so on.[10,11] On the basis of aetiology, Neonatal Intestinal obstruction can be classified as: Dynamic or Mechanical intestinal obstruction (e.g. atresia, volvulus) and as Adynamic or Functional intestinal obstruction (e.g. Hirschsprung's disease, Neonatal sepsis).[12,13]

The mechanism by which the obstruction occurs may be intraluminal e.g. meconium ileus and meconium plug, intramural e.g. atresia and stenosis as well as extramural compression by a band (malrotation, Meckel diverticulum) or by a duplication cyst, twisting by a volvulus, or kinking in an incarcerated hernia (inguinal or diaphragmatic).[12,13]

Common surgical conditions that are causes of neonatal intestinal obstruction include Hirschsprung's disease, Anorectal malformations, Jejuno-ileal atresia and stenosis, Duodenal obstruction—atresia, stenosis and annular pancreas, Meconium plug, Meconium ileus, Hernia—inguinal hernia, umbilical hernia, Malrotation with Ladd's bands, Mid-gut volvulus, Colonic stenosis and atresia.[8,10,12,13,14] Most of these conditions arise as developmental anomalies during the embryogenesis of the gastrointestinal tract (3-4weeks gestational age) while others occur in the foetal period, mostly from vascular accidents.[12,13]

Hirschsprung's disease (HD) is an enteric nervous system disorder characterized by absence of ganglion cells in the distal bowel beginning at the internal sphincter and extending proximally for varying distances. The aganglionosis is confined to rectosigmoid in most of the cases, 75% of patients.[15,16]

Anorectal malformations are congenital developmental abnormalities that affect the structures that are derived from the primitive cloaca. They are a spectrum of abnormalities which could present with abnormal rectal openings onto the perineum or into the genitourinary tract or as an imperforate anus. The abnormal anorectal development usually predisposes to neonatal intestinal obstruction especially when associated with an imperforate anus.[17,18]

Jejunoileal atresia and stenosis are congenital anomalies of the small intestine characterised by complete occlusion of the intestinal lumen (atresia) or partial intraluminal occlusion (stenosis) which usually result from intrauterine vascular accidents followed by varying degree of involution of the affected part.[19] Colonic atresia and stenosis which also result from in utero vascular insufficiency after organogenesis are rare causes of neonatal intestinal obstruction.[20] These conditions might be associated with small left sided colon, see figure 1.

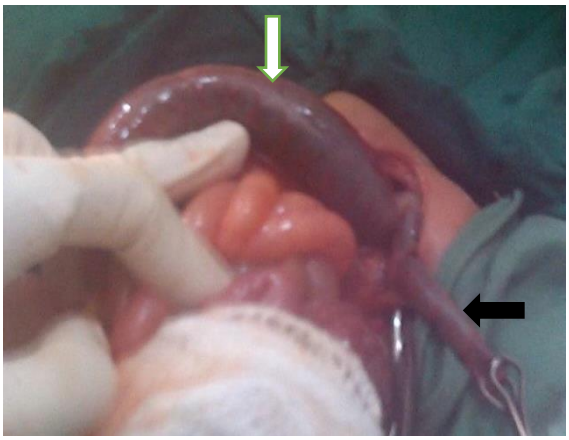


Figure 1: Shows small left sided colon, highlighted by the black arrow. Also, note the proximal dilated colon (white arrow).

Duodenal obstruction as a cause of neonatal intestinal obstruction could be due to duodenal atresia, duodenal stenosis, annular pancreas or Ladd's bands.[21] Duodenal atresia and stenosis result from errors in recanalization of the duodenum during embryogenesis while annular pancreas results from failed rotation of the ventral pancreatic bud which subsequently fuses with the dorsal bud to form a "pancreatic ring" around the second part of the duodenum.[21] Ladd's bands are formed from malrotation of the gut.[21]

Malrotation is a spectrum of anatomic abnormalities of incomplete rotation and fixation of the intestinal tract during foetal development. This may result in development of Ladd's bands (dense fibrous bands formed from peritoneal reflections) which extend from the caecum and right colon across the duodenum to the retroperitoneum of the right upper quadrant, thus obstructing the

duodenum by extrinsic compression. Midgut volvulus, the torsion of the midgut around a short mesentery, is a consequence of malrotation in extreme cases.[22]

Meconium ileus is neonatal intestinal obstruction due to abnormally thickened, inspissated and tenacious meconium within the gut (usually ileum). Commonly, the abnormality of the meconium results from pancreatic insufficiency, as well as abnormal mucus secreted by the intestine of patients with Cystic Fibrosis. In meconium plug syndrome, the inspissated meconium form plugs usually observed in the sigmoid colon which result in intestinal obstruction.[23,24] Meconium plugs are said to develop in the setting of bowel hypomotility e.g. prematurity, hypotonia, hypermagnesaemia, respiratory distress, sepsis, hypothyroidism, diabetes and Hirschsprung's disease.[24]

Neonatal intestinal obstruction might also result from obstructed hernias, especially inguinal and umbilical hernias, as well as from extrinsic compressing congenital tumours like sacrococcygeal teratoma.[12]

Pathophysiology

With obstruction to the flow of intestinal content, there will be accumulation of bowel content proximal to the point of obstruction. If the cause of obstruction is mechanical, there will be increased peristalsis in the proximal bowel which clinically manifests as colics. Also, in mechanical bowel obstruction the distal loop continues to undergo peristalsis which eventually empties the distal content leading to the clinical feature of absolute constipation (i.e. non passage of faeces or flatus) in cases of complete obstruction.[25]

The proximal accumulation is characterised by third space fluid loss, electrolyte loss and normal flora (bacteria) multiplication, as well as gaseous distension. When this proximal loop accumulation occurs in a high type intestinal obstruction, it presents clinically as early bilious vomiting because of proximity to the stomach whereas in the low type the presentation is abdominal distension with late bilious emesis.[25]

The third spacing of body fluid that occurs with intestinal obstruction predisposes to hypovolaemia and hypovolaemic shock. In addition, the progressive increase in the intraluminal pressure impedes the intramural microcirculation in a rapid succession of lymphatic obstruction, venous obstruction, arterial obstruction and consequent ischaemia. This bowel wall ischaemia will eventually lead to mucosa sloughing, bacteria translocation, bacteraemia, perforation, peritonitis and septicaemia.[25] This pathophysiology is outlined in figure 2.[26]

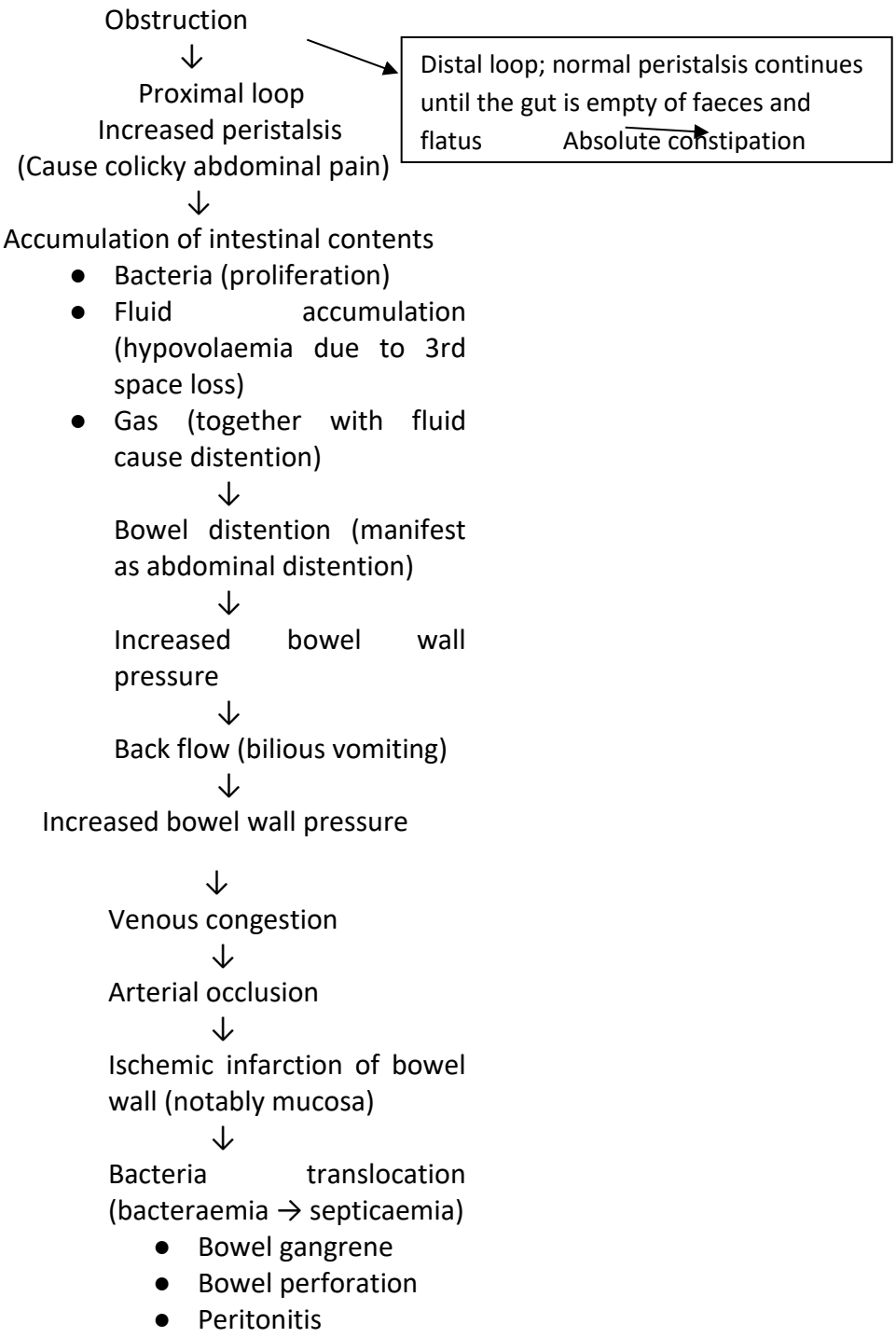
While the above description of pathophysiology is true for postnatal presentation of neonatal intestinal obstruction, there is some variation in the in-utero manifestation. Complete obstructions have dilated proximal loops and small (collapsed) distal loops as seen in atresia. The high intestinal obstruction manifests with maternal polyhydramnios. Increased intraluminal pressure with intramural ischaemia could present with meconium peritonitis as in severe meconium ileus. Functional obstruction especially in the distal (left sided) colon cause inspissation of the meconium leading to the meconium plug syndrome.[24]

Clinical Features

The clinical features manifested by these neonates are varied depending on the specific pathology. They present with delayed passage of meconium, abdominal distension and late bilious emesis when the pathology is

situated low down the gut e.g. Hirschsprung's disease,

Figure 2: An outline of the events that occur in the pathophysiology of neonatal intestinal obstruction



Anorectal malformations, Jejunoileal and Colonic stenosis and atresia, meconium ileus and meconium plug syndrome. When the intestinal obstruction is situated higher up, they tend to present with persistent bilious vomiting, upper abdominal fullness with the rest of the abdomen scaphoid (see figure 3) e.g. duodenal stenosis, annular pancreas, duodenal atresia, mal-rotation with Ladd's bands.[4] In all cases the neonates tend to be irritable.

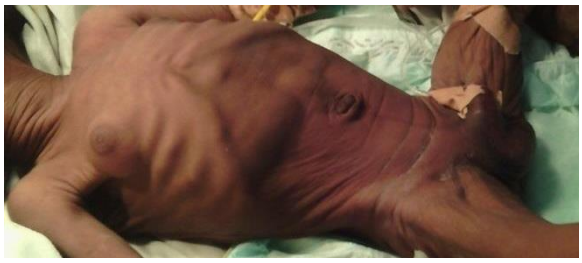


Figure 3: Scaphoid abdomen of duodenal obstruction

With the progression of the intestinal obstruction, there will be features of Hypovolaemia e.g. sunken fontanelle, sunken eyes, loss of skin tugor, altered sensorium or lethargy. There might be hypothermia or fever when sepsis sets in. Bowel sound is initially hyperactive and later becomes hypoactive or absent. On rectal examination, there may be meconium plug which is usually easy to dislodge but in most other cases the rectum is empty. In cases of anorectal malformation, the anus is observed to be absent in the case of imperforate anus or there is abnormal perineal opening which might be a fistula or an ectopic anus. [17,18]

In cases of malrotation with volvulus, the infant initially has a scaphoid abdomen but with delayed presentation there is progressive abdominal distention, abdominal wall erythema, haematemesis and/or melaena.[4]

Patients with Hirschsprung disease sometimes present with enterocolitis (25%–35% of patients), a common cause of mortality which is characterized by diarrhoea with abdominal distention, fever and other nonspecific symptoms including vomiting, rectal bleeding and lethargy.[27,28]

Diagnostic Investigations

The presence of polyhydramnios in the prenatal ultrasound scan points to intestinal obstruction, particularly the high or proximal type. Also the non-visualization of normally visible fluid-filled gut structures, echogenic bowel, abdominal calcifications and bowel wall thickness greater than 3mm in the third trimester ultrasound scan suggest intestinal obstruction.[29] Prenatally, conditions causing high intestinal obstruction are more readily diagnosed compared to distal obstruction because they are more likely to present with polyhydramnios.[30] Adequate preparation to ensure a good management outcome can be made with early prenatal diagnosis.

In the case of postnatal presentation, the initial diagnostic investigation is a plain abdominal radiograph, which is done in the supine and cross-table lateral (or erect) positions. Typically, the supine view shows dilated bowel loops while multiple air-fluid levels and absent or paucity of air in the pelvis are seen on the cross-table lateral (or erect) view, see figure 4. When four or more dilated bowel loops are observed, a distal obstruction is most likely whereas if they are less than four it is likely a high intestinal obstruction.[12,25,31] Pneumoperitoneum is observed on the cross-table lateral radiograph if bowel perforation has occurred, see figure 5.[12,25,31] When the obstruction is at

the level of the duodenum, the classical double bubble appearance (markedly dilated stomach and duodenal bulb) is seen on the plain abdominal radiograph, see figure 6.[21]

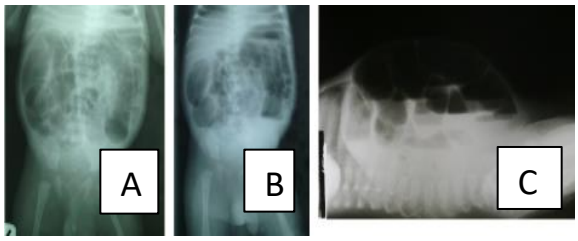


Figure 4: Plain abdominal radiographs for neonatal intestinal obstruction; A is the supine view, B is erect view and C is cross-table lateral view. A shows dilated bowel loops while both Band C show multiple air-fluid levels

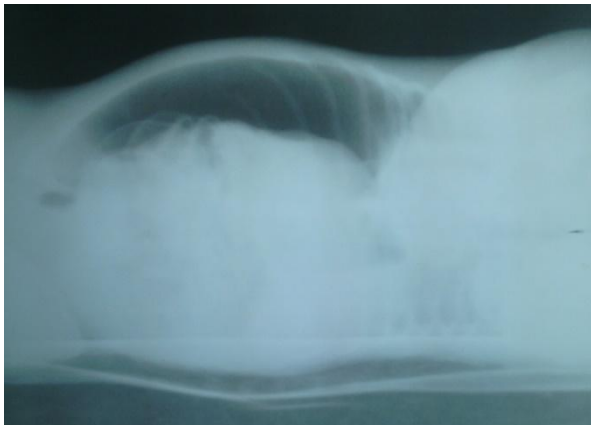


Figure 5: Cross-table lateral film showing pneumoperitoneum

However, if there is a normal-calibre duodenum with a distended stomach, the diagnosis is likely to be intestinal malrotation with Ladd's bands (which exert extrinsic compression on the duodenal bulb).[32] Meanwhile, the "triple bubble sign" (distended stomach, duodenum and proximal jejunum) when observed on the plain abdominal radiograph demonstrates jejunal atresia.[1]

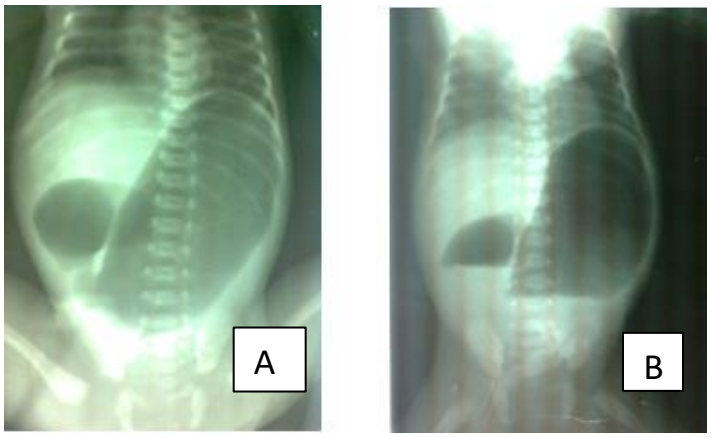


Figure 6: Plain abdominal radiographs showing the classical double bubble sign of duodenal atresia; A is supine view while B is erect view

In meconium ileus, the plain abdominal radiograph may show a bubbly gas pattern in the right abdomen with absence of air-fluid levels which is due to the thick and sticky nature of the meconium.[33] Also, if in utero perforation and meconium peritonitis occurred, features of calcifications may be seen on the plain abdominal radiograph, sometimes, with pseudocyst formation. [1,33]

A cross-table lateral abdominal radiograph in the prone Jack-knife position can help to differentiate anorectal malformations to high and low types by demonstrating the distance of the air-filled blind-ending rectal pouch to the expected location of the anus; if greater than 1cm it is a high type and if 1cm or less it is a low type (see figure 7).[17]

Contrast studies are indicated where plain abdominal radiographs findings are not conclusive. Barium sulphate (water insoluble) and Gastrografin (water soluble) are commonly used. An upper gastrointestinal

(UGI) contrast study is usually required when the plain abdominal radiograph suggests a high intestinal obstruction while a contrast enema is required for low intestinal obstruction.[4]

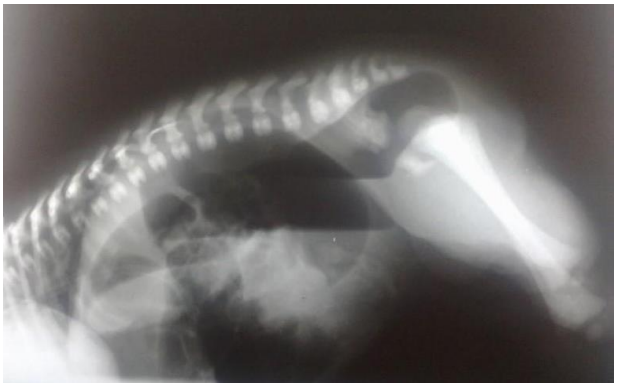


Figure 7: Cross-table lateral radiograph in prone Jack-knife position, shows feature in keeping with low anorectal malformation

UGI contrast study is the gold standard for diagnosis of intestinal malrotation.[4] For simple intestinal malrotation anomalies, the duodenujejunal junction is seen on the right side of the spine while for malrotation with volvulus, the contrast study shows a spiral corkscrew appearance. For malrotation with Ladd's bands, the duodenum appears Z-shaped on the contrast study.[1]

The contrast enema done for cases where the level of obstruction is proximal to the colon (e.g. jejunoileal atresia and meconium ileus) will show a small and unused microcolon. A contrast enema showing a small and unused microcolon with a rounded "cobra head" appearance is pathognomonic for colonic atresia.[29] In a case of meconium plug, a water-soluble contrast enema will demonstrate a normal rectal size and meconium plugs in a small left colon (which may extend from the sigmoid colon to the splenic flexure) with proximal colonic distension.[29] This procedure is usually followed by evacuation of the meconium plugs, hence it is both diagnostic and therapeutic; such is the case with meconium ileus in some instances.

Contrast enema for Hirschsprung disease may reveal abnormal recto-sigmoid ratio of less than 1, presence of a cone-shaped transition zone (between the ganglionic and aganlionic segments, see figure 8), irregular rectal contractions with a saw-tooth appearance and retained contrast material on delayed radiographs.[1]

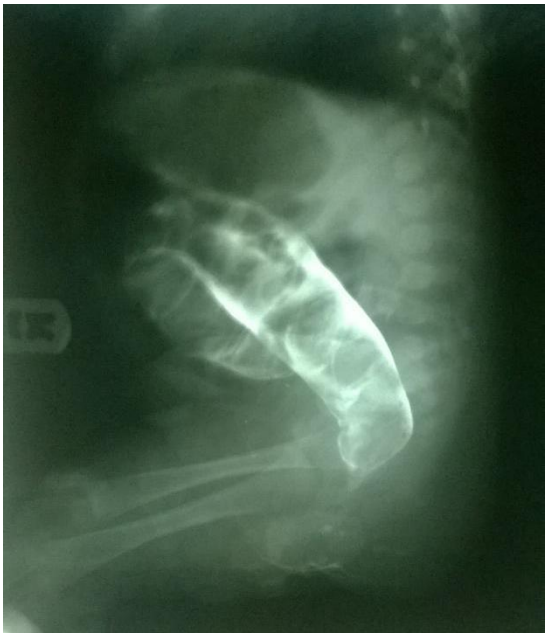


Figure 8: Contrast enema showing the transition zone in a patient with Hirschsprung's disease

In cases of intestinal atresias, it is sometimes necessary to do both an UGI contrast study and a contrast enema prior to surgery to rule out other concomitant atresias.[1]

Post-natal abdominal ultrasound scan is useful in certain cases of neonatal intestinal obstruction. In cases where duodenal obstruction is suggested on a plain

radiograph, it is useful in confirming an annular pancreas as a cause. Ultrasound evaluation may suggest the possibility of intestinal malrotation if the superior mesenteric vein (SMV) is observed to be on the left of the artery or rotated completely around the artery.[34] In intestinal malrotation with midgut volvulus, duodenal wall thickening and luminal dilation with distal tapering, a whirlpool sign on colour Doppler, fixed midline bowel, ascites and dilation of the distal SMV may be seen when abdominal ultrasound scan is done.[35,36]

Rectal biopsy with tissue samples for histological and histochemical analyses is usually required to confirm the diagnosis of Hirschsprung disease; absent ganglion cells and excessive acetylcholine staining are seen respectively.[33,37]

Laboratory work up should include full blood count, serum electrolytes, urea and creatinine. These may reveal findings like leukocytosis or leukopenia, hypokalemia, thrombocytopenia and metabolic acidosis.[4] Also blood grouping and cross-matching should be done in preparation for surgical intervention.

Investigations to detect associated anomalies should also be done. These include echocardiogram, renal/bladder ultrasound scan, babygram, voiding cystourethrogram and radiograph of the spine.[4,38]

Treatment

The general approach is to withhold oral intake in order not to worsen the increased intraluminal pressure and do nasogastric tube decompression to reduce the increased intraluminal pressure. Intravenous cannulation is done and blood specimens collected for full blood count, serum electrolytes, urea and creatinine and blood grouping and cross-matching. Then intravenous fluid resuscitation is commenced — Normal saline or Ringers' lactate is given as anti-shock (20mls/kg) if there is hypovolaemic shock and 8% dextrose in 0.18% saline is continued as maintenance fluid. Laboratory derangements in blood glucose, electrolytes and acid–base status should be corrected. Intravenous broad-spectrum antibiotics covering the target microflora are also commenced.[12,25]

Urethral catheterization is done for hourly urinary output monitoring while also ensuring adequate monitoring of vital signs. Mechanical respiratory support should be given when necessary. Surgical treatment is considered when the neonate has been optimized following adequate resuscitation (i.e. stable vital signs and optimal hourly urine). Explorative laparotomy is done when preoperative diagnosis is not certain. Resection and anastomosis or stoma creation could be done as indicated. The emergency surgery should be postponed until metabolic, cardiac and respiratory abnormalities have been diagnosed and treated. However, if surgical treatment is delayed over a long period of time, there are increased complications like aspiration pneumonitis from emesis, sepsis, bowel perforation and so on. Early parenteral nutrition should be part of the management protocol for ideal postoperative care to prevent negative nitrogen balance.[12]

The definitive surgical procedure done depends on the cause of the neonatal intestinal obstruction e.g. posterior sagittal anorectoplasty (PSARP) for anorectal malformations. For duodenal atresia, the procedure of choice is an end-to-side or side-to-side duodenoduodenostomy but duodenojejunostomy is also acceptable. In

cases of duodenal stenosis or atresia due to an intrinsic duodenal web, a vertical duodenotomy, web resection and transverse closure is ideal procedure. Resection of the atretic sections with end-to-end anastomosis is definitive surgical treatment for jejunoileal atresia.[38]

Surgery is reserved for meconium ileus cases that are refractory to therapeutic enemas or complicated by perforation.[39] For Hirschsprung's disease, high colonic washouts should be carried out routinely until definitive surgical treatment involving the resection of the aganglionic bowel segment and pull through of the normal ganglionic segment is done.[28,40]

Diverting colostomy as a temporizing measure is sometimes carried out with later definitive surgical treatment, especially for severe cases with sepsis refractory to resuscitation.[28]

Conclusion

Neonatal intestinal obstruction is a common surgical emergency amongst neonates commonly caused by intestinal atresias, meconium diseases, malrotation, anorectal malformations, Hirschsprung's disease, inguinal hernia and so on. The affected neonates commonly present with delay in passage of meconium, abdominal distension, bilious vomiting, irritability, dehydration and fever or hypothermia. Early diagnosis and intervention, as well as support care facilities and trained staff of different specialties are crucial to having a good outcome. Plain radiographs are usually sufficient to establish the cause but contrast studies may sometimes be required. Also, laboratory workup is important to identify leukocytosis or leukopenia, hypokalemia, thrombocytopenia and metabolic acidosis which must need to be corrected before treatment. The treatment starts with adequate resuscitation followed by the definitive procedure based on the specific cause of the neonatal intestinal obstruction; this may be non-operative procedures like colorectal washouts as in meconium plug or surgical procedures like resection of atretic parts and anastomosis.

References

1. Vinocur DN, Lee EY, Eisenberg RL. Neonatal intestinal obstruction. AJR Am J Roentgenol. 2012;198(1): W1–W10.
2. Mhando S, Young B, Lakhoo K. The scope of emergency paediatric surgery in Tanzania. Pediatr Surg Int 2008; 24(2):219–222.
3. Seth A, Chanchlani R, Rakhonde AK. Neonatal gastrointestinal emergencies in a tertiary care centre in Bhopal, India: A prospective Study. IJSS. 2016; 10(2): pp 10-13.
4. Juang D, Snyder CL. Neonatal bowel obstruction. Surg Clin North Am. 2012;92(3):685–711.
5. Hanif A, Hasina K, Hossain AZ, Alam MM, Huda SMS, Hassan MK et al. Neonatal surgery: demand and survival both are increasing: eight years experience in Dhaka Medical College Hospital, Bangladesh. Journal of Paediatric Surgeons of Bangladesh 2010;1 (1): 30-35.
6. Verma A, Rattan KN, Yadav R. Neonatal Intestinal Obstruction: A 15 Year Experience in a Tertiary Care Hospital. J Clin Diagn Res. 2016; 10(2): SC10–SC13.
7. Kimura K. Bilious vomiting in the newborn: rapid diagnosis of intestinal

obstruction. Am Fam Physician. 2000;61(9):2791–98.
8. Ameh EA, Chirdan LB. Neonatal intestinal obstruction in Zaria, Nigeria. East Afr Med J. 2000; 77:510–13.
9. Uba AF, Edino ST, Yakubu AA, Sheshe AA. Childhood intestinal obstruction in Northwestern Nigeria. West Afr J Med. 2004; 23:314–18.
10. Rowe MI. The newborn as a surgical patient. In: O'Neill JA, Rowe MI, Grosfeld JL, Fonkalsrud EW and Coran AG (eds.). Paedatric Surgery. 5th ed. Mosby year book Inc, Philadelphia. 1998, pp.43-55.
11. Osifo OD, Okolo JC. Neonatal intestinal obstruction in Benin, Nigeria. Afr J Paediatr Surg. 2009; 6:98-101.
12. Sidler D, Debrew M, Lakhoo K. Neonatal Intestinal Obstruction. In: Ameh EA, Bickler SW, Lakhoo K, Nwomeh BC and Poenaru D (eds.). Pediatric surgery, a comprehensive text for Africa. Global HELP Organization. Seattle. 2011, pp. 389–393.
13. Tandoh JFK. Neonatal Intestinal Obstruction; Acute Intestinal Obstruction. In: Badoe EA, Archampong EQ and da Rocha-Afodu JT (eds.). Principles and practice of surgery including pathology in the tropics. 4th ed. Ghana publishing co-operation, Accra. 2010, pp. 592–597.
14. Hajivassiliou CA. Intestinal obstruction in neonatal/pediatric surgery. j.sempedsurg. 2003.08.005; 241-253.
15. Moore SW, Tsifularo N, Nmadu PT, Gosche JR. Hirschsprung's Disease. In: Ameh EA, Bickler SW, Lakhoo K, Nwomeh BC and Poenaru D (eds.). Pediatric surgery, a comprehensive text for Africa.Global HELP Organization. Seattle. 2011, pp. 461–467.
16. Tandoh JFK. Aganglionic megacolon; Small and large intestine (including rectum and anus. In: Badoe EA, Archampong EQ and da Rocha-Afodu JT (eds.). Principles and practice of surgery including pathology in the tropics. 4th ed. Ghana publishing co-operation, Accra. 2010, pp. 671–674.
17. Meier DE, Hesse AAJ. Anorectal malformations. In: Ameh EA, Bickler SW, Lakhoo K, Nwomeh BC, and Poenaru D (eds.). Pediatric surgery, a comprehensive text for Africa. Global HELP Organization. Seattle. 2011, pp. 468–473.
18. Nwakor FA. Anorectal Anomalies; Small and large intestine (including rectum and anus. In: Badoe EA, Archampong EQ and da Rocha-Afodu JT (eds.). Principles and practice of surgery including pathology in the tropics. 4th ed. Ghana publishing co-operation, Accra. 2010, pp. 668–671.
19. Millar AJW, Gosche JR, Lakhoo K. Intestinal atresia and stenosis. In: Ameh EA, Bickler SW, Lakhoo K, Nwomeh BC and Poenaru D (eds.). Pediatric surgery, a comprehensive text for Africa. Global HELP Organization. Seattle. 2011, pp. 398–401.
20. Millar AJ, Cox S, Lakhoo K. Colonic atresia. In: Ameh EA, Bickler SW, Lakhoo K, Nwomeh BC and Poenaru D (eds.). Pediatric surgery, a comprehensive text for Africa. Global HELP Organization. Seattle. 2011, pp. 450–452.
21. Eckoldt-Wolke F, Hesse AAJ, Krishnaswami S. Duodenal atresia and stenosis. In: Ameh EA, Bickler SW, Lakhoo K, Nwomeh BC and Poenaru D

(eds.). Pediatric surgery, a comprehensive text for Africa. Global HELP Organization. Seattle. 2011, pp. 394–397.
22. Askegard-Giesmann JR, Amah CC, Kenney BD. Intestinal malrotation and midgut volvulus. In: Ameh EA, Bickler SW, Lakhoo K, Nwomeh BC and Poenaru D (eds.). Pediatric surgery, a comprehensive text for Africa. Global HELP Organization. Seattle. 2011, pp. 406–409.
23. Eckoldt-Wolke F, Abubakar AM. Meconium ileus. In: Ameh EA, Bickler SW, Lakhoo K, Nwomeh BC and Poenaru D (eds.). Pediatric surgery, a comprehensive text for Africa. Global HELP Organization. Seattle. 2011, pp. 414–416.
24. Ziegler MM. Meconium ileus. In: Coran AG, Adzick NS, Krummel TM, Laberge J-M, Shamberger RC and Caldamone AA (eds.) Pediatric surgery. 7th ed. Saunders, an imprint of Elsevier Inc. Philadelphia. 2012, pp. 1073–1083.
25. Naaeder SB, Tandoh JFK. Acute Intestinal Obstruction. In: Badoe EA, Archampong EQ and da Rocha-Afodu JT (eds.). Principles and practice of surgery including pathology in the tropics. 4th ed. Ghana publishing co-operation, Accra. 2010, pp. 572–598.
26. Udefiagbon OE. Clinical surgery tutorial manual. 2nd ed. Ambik press, Benin. 2016, pp. 68.
27. Gosain A. Established and emerging concepts in Hirschsprung's-associated enterocolitis. Pediatr Surg Int. 2016;32(4):313–320.
28. Burkardt DD, Graham JM Jr, Short SS, Frykman PK. Advances in Hirschsprung disease genetics and treatment strategies: an update for the primary care pediatrician. Clin Pediatr (Phila). 2014;53(1):71–81.
29. Desoky SM, Kylat RI, Udayasankar U, Gilbertson-Dahdal D. Managing neonatal bowel obstruction: clinical perspectives. Research and Reports in Neonatology. 2018, 8:19-32 .
30. Silva P, Reis F, Alves P, Farinha L, Gomes MS, Câmara P. "Fetal Bowel Dilatation: A Sonographic Sign of Uncertain Prognosis", Case Reports in Obstetrics and Gynecology. 2015:1-4.
31. Silva AC, Pimenta M, Guimaraes LS. Small Bowel Obstruction: What to Look For. RadioGraphics 2009;29(2):423–439.
32. Gilbertson-Dahdal DL, Dutta S, Varich LJ, Barth RA. Neonatal malrotation with midgut volvulus mimicking duodenal atresia. Am J Roentgenol. 2009;192(5):1269–1271.
33. Reid JR. Practical imaging approach to bowel obstruction in neonates: a review and update. Semin Roentgenol. 2012;47(1):21–31.
34. Askegard-Giesmann JR, Amah CC, Kenney BD. Intestinal malrotation and midgut volvulus. In: Ameh EA, Bickler SW, Lakhoo K, Nwomeh BC and Poenaru D (eds.). Pediatric surgery, a comprehensive text for Africa. Global HELP Organization. Seattle. 2011, pp. 406–409.
35. Marine MB, Karmazyn B. Imaging of malrotation in the neonate. Semin Ultrasound CT MR. 2014;35(6):555–570.
36. Raske ME, Dempsey ME, Dillman JR, et al. ACR appropriateness criteria vomiting in infants up to 3 months of age. J Am Coll Radiol. 2015;12(9):915–922.
37. Langer JC. Hirschsprung Disease. In: Coran AG, Adzick NS, Krummel TM,

Laberge J-M, Shamberger RC and Caldamone AA (eds.). Pediatric surgery. 7th ed. Saunders, an imprint of Elsevier Inc. Philadelphia. 2012, pp. 1267–1268.

38. Sidler D, Debrew M, Lakhoo K. Neonatal Intestinal Obstruction. In: Ameh EA, Bickler SW, Lakhoo K, Nwomeh BC and Poenaru D (eds.). Pediatric surgery, a comprehensive text for Africa.Global HELP Organization. Seattle. 2011, pp. 389–393.
39. Karimi A, Gorter RR, Sleeboom C, Kneepkens CM, Heij HA. Issues in the management of simple and complex meconium ileus. Pediatr Surg Int. 2011;27(9):963–968.
40. Sloan K, Wagener S. Updates in: Hirschsprung's disease and anorectal malformations. J Pediatr Surg Specialties. 2016;10(3):43–51.