

“A LITERARY REVIEW ON PAEDIATRIC CONSTIPATION W.S.R TO ORGANIC PATHOLOGY, ITS DIAGNOSIS AND MANAGEMENT.”**¹*Dr. Bhagyashree Manohar Kachare and ²Dr. Deepnarayan V. Shukla**

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ABSTRACT

Constipation is a severe concern in day today outpatient and indoor admission in infant and young children. The evaluation of constipation involves first determining whether the change in the frequency or consistency of stools is secondary to functional constipation or is related to an organic disorder. A detailed history of bowel pattern becomes necessary to identify the causes. Thus proper case taking becomes essential to reveal the medical history for chronic diseases, such as metabolic, neurological, physiological or anatomical abnormalities. A proper physical examination of patient helps to rule out anatomical disorder /defects which lead to paediatric constipation. These may be examination of spine and sacral area, the location of the anus, and digital rectal examination. This determination is based on

identifying historical features and examination findings that suggest an underlying disorder and prompt further investigation. This alerts physician and parents for the need for further evaluation, diagnosis and management necessary.

KEYWORDS: Paediatric Constipation, Hardstools, Feecal continence, Meconium ileus, bowel emptying.

INTRODUCTION

The word constipation is derived from the latin word Constipare i.e. to press together.^[1] The child suffering from constipation strains during defeacation or gives pressure on abopelvic cavity.

Thus to understand the organic pathology in constipation, it becomes essential to know anatomy and physiology of colon.

Anatomy of colon in relation to constipation.

Large intestine extends from ileocaecal junction to anus. It is 1.5m in length.^[2] It consists of following organs.

1. Caecum: It is 6m long and 7.5 cm broad. Caecum is the commencement of large intestine. Pouch like caecum, widest part of large intestine. It is completely intraperitoneal organ and has no mesentery therefore it is freely mobile in right iliac fossa. Ileocecal valve prevents reflux from caecum to ileum and regulates passage of ileal contents.^[3]

2. Ascending colon: The ascending colon is superior, secondarily retroperitoneal continuation of the ileocecal valve and right colic flexure. It is 12.5cm in length.

3. Transverse colon: Transverse colon is 50 cm long. The transverse colon is suspended by the transverse mesocolon between the right and left flexures, is the longest and most mobile part of the large intestine.

4. Descending colon: It is 25 cm long. The descending colon occupies a secondarily retroperitoneal position between the left colic flexure and left iliac fossa, where it is continuous with sigmoid colon.

5. Sigmoid colon: The S-shaped sigmoid colon, suspended by the sigmoid mesocolon, is highly variable in length. Extends from pelvic brim to third piece of sacrum. It is 37.5cm in length.

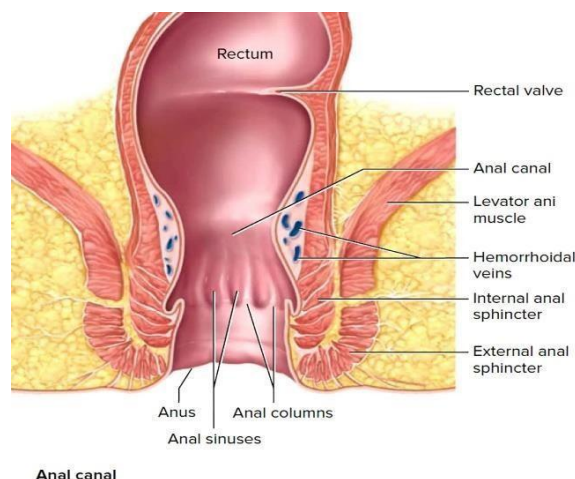
6. Rectum: Lies between sigmoid colon and anal canal below. It is 12cm in length. In posterior part of lesser pelvis in the front of three pieces of sacrum and coccyx.

Course

Rectum runs first downwards and backwards then downwards and finally downwards and forwards.

Functions

Functionally sigmoid colon is fecal reservoir and whole of the rectum is empty in normal individual, being sensitive to distension.



Passage of faeces into the rectum therefore causes the desire to defecate. Fig below describes anatomy of rectum and anal canal.(fig 1.a).^[4]

Anal canal: It is 3.8 cm long. Terminal part of large intestine. It is situated below the level of pelvic diaphragm. It lies in anal triangle of perineum in between rt. and lt. ischioanal fossae, which allows its expansion during passage of the faeces.

Anorectal ring: Muscular ring present at anorectal junction. By fusion of puborectalis, uppermost fibres of external sphincter and internal sphincter.

Functional anatomy: Due to weak musculature of rectum and sparing of tone of external sphincter by transverse lesion of cord, rectal disturbances tend to cause constipation.

Physiology of defecation

Passage of small hard, dry stools that contain mainly solids and minimal water. It is important to understand that all pelvic and abdominal muscles play an important role in defecation. Three muscular sphincters surrounding anus and rectum are important viz- 1)

- 1) Internal anal sphincter
- 2) External anal sphincter
- 3) Puborectalis muscle

Factors contributing to constipation

1) Defects in emptying rectum

May be due to interference with defecation reflex. It may be due to pressure initiated by pressure receptor in rectal muscles.

-also lesion of rectal muscles, lesions in sacral region of spinal cord (afferent fibres) and

lesions of pelvic floor and abdominal muscles are responsible for defects in emptying rectum.

2) Due to poor gut peristalsis.

Feacal continence and physiologic defecation are dependent on the anal inhibitory reflex, which in turn dependent on the proper structure and function of internal and external anal sphincter and pelvic floor.

Internal anal sphincter is an involuntary muscle, it is contracted at rest. When bolus of stools distend the rectum, the internal anal sphincter relaxes.

This process generally results in the child sensing the need to defecate. The external anal sphincter and puborectalis muscle of pelvic floor under voluntary control contract upon rectal distension, respectively closing the anus and decreasing anorectal angle, thus allowing the child to hold the stool until it is socially convenient to defecate. Voluntary relaxation of puborectalis muscle and external anal sphincter straightens the anorectal angle and allows the child to defecate.

Constipation

Constipation may be defined as decrease in frequency of bowel movements and difficult and painful passage of hard stools.^[5]

Constipation is defined symptomatically as the infrequent passage of hard stools, straining while passing a stool, or pain associated with passage of hard stool.

Sign and Symptoms seen in acute constipation

- Delayed passage of meconium(after 48 hrs of life.)
- Fever, vomiting, or diarrhoea, rectal bleeding, severe abdominal distension.

Sign and Symptoms seen in chronic constipation

- Constipation present from birth or early infancy -Ribbon stool.
- Urinary incontinence or bladder disorders.
- Weight loss or poor weight gain.

Physical findings in organic etiologies of constipation

- Severe abdominal distension
- Pelvic mass

- lumbosacral dimple, hair tuft or lipoma or deviation of gluteal cleft
- Anal scars
- Anteriorly displaced anus
- Perianal fistula
- High anal canal tone with empty rectum.
- Explosive expulsion of stools after digital examination.



Fig below demonstrates the appearance of stools in various conditions according to Bristol stool chart(fig 1.b).^[6]

The range of normal defecation pattern in children is widely variable, though in general formula fed infants may have 4-5 episodes per day in 1st week of life, while breastfed infants usually pass softer and more frequent stools.

Stool frequency in both gradually decrease to 1-2 per day by 1 year of age.

Most children aged 1-4 years have 1 or 2 daily bowel movements to 1 bowel movement every other day.

Constipation is classified broadly as functional or secondary to underlying conditions like anatomical abnormalities, medication or metabolic disorder etc.

Changes in diet such as formula changes or the addition of solid foods may lead to constipation in infant. Minor illness including infectious diarrhoea, can subsequently result in episodic constipation.

Causes of constipation during neonatal period(table 1.1).^[7]

Causes	Organic	Endocrine	Metabolic/Genetic	Neurological	Medications
Types	-Meconium plug. -Meconium ileus -Hirschprung disease -Anteriorly displaced anus -Ectopic anus -Anal stenosis -Intestinal pseudoobstruction -Imperforate anus -Spina bifida	Hypothyroidism	Hypercalcemia	Neuronal intestinal dysplasia types A and B	-Opioids - Paralytic agents -Magnesium

Above are mentioned causes of constipation in neonates, which necessary to be ruled out if neonate doesn't pass meconium beyond 48 hrs of birth.

Similarly we shall thereby mention**Causes of constipation in infants and children's(table1.2).^[8]**

Types of constipation	Causes
Functional	Dietary (Inadequate nutrition, poor fibre intake, excessive cows milk) -Situational.
Anatomical defects/Organic pathology	-Anterior anal displacement -Ectopic anus -Anal stenosis -Malrotation Colonic anomalies(rectocele) - Stricture(postsurgical, sequel of inflammatory disorders) -Painful anorectal lesions(fissures,dermatitis,abscess) -Abnormal abdominal musculature(prune belly,gastroschisis) Intestinal neoplasm,extraintestinal pelvic mass.
Endocrine	-Hypothyroidism -Panhypopituitarism -Diabetes mellitus
Genetic/Metabolic	-Hypercalcemia -Metal intoxication -Dehydration -Hypokalemia -Acute intermittent porphyria -Rubinstein-Taybi syndrome -Williams syndrome(hypercalcemia)
Abnormal innervation/neurological	-Aganglionosis -Hirschsprung disease -Neural dysgenesis(pseudo obstruction syndromes -Hyperganglionosis -Myotonic dystrophy -Cerebral palsy
Spinal cord lesions	-Spina bifida and spina bifida occulta -Spinalcord tumors

Infectious diseases	-Typhoid -Infant botulism -Chagas disease
Medications	-Anticonvulsants -Antacids -Barium -Opioids -Antidepressants -Anticholinergics -Phenothiazides -Calcium channel blockers -Bismuth -Antihistamines -Diuretics
Psychological	-Anorexia nervosa - Depression.
Other	Protein induced anal inflammation and fissure formation

Thereby after overlooking the causes of constipation in neonates, infants and children we shall focus on anatomical causes or organic pathology in relation to paediatric constipation.

DISCUSSION

Here various organic pathology related to constipation, its diagnosis and further evaluation and management shall be discussed.

Hirschsprung disease

Congenital absence of parasympathetic ganglion cells from intramural plexus of a portion of intestinal tract, usually the distal end of colon is responsible for the entity described here. The involved section of gut is narrowed and devoid of peristaltic activity, and is thus responsible for accumulation of fecal material above it. As a result the uninvolved portion of intestine becomes greatly dilated and hypertrophied and is responsible for abdominal distension, which with the obstinate constipation creates the typical clinical pattern.^[9]

Congenital aganglionic megacolon or Hirschsprung disease is common cause of neonatal intestinal obstruction, occurring approximately 1:5000-1:15000 live births. Disease is rare in premature and many a times associated with trisomy 21. The male female ratio of the disease is 4:1. **Pathology of Hirschsprung disease.**

The absence of ganglionic cells in the Meissner (submucosal) plexus and Auerbach (myenteric plexus) result in inability of the involved segment of bowel to relax in the response to distension from the presence of stool.

Narrowing of portion of rectum or rectosigmoid is usually demonstrated. Proximal to narrowed segment the colon is tremendously dilated and filled with faeces and gas and muscular coat is hypertrophied.

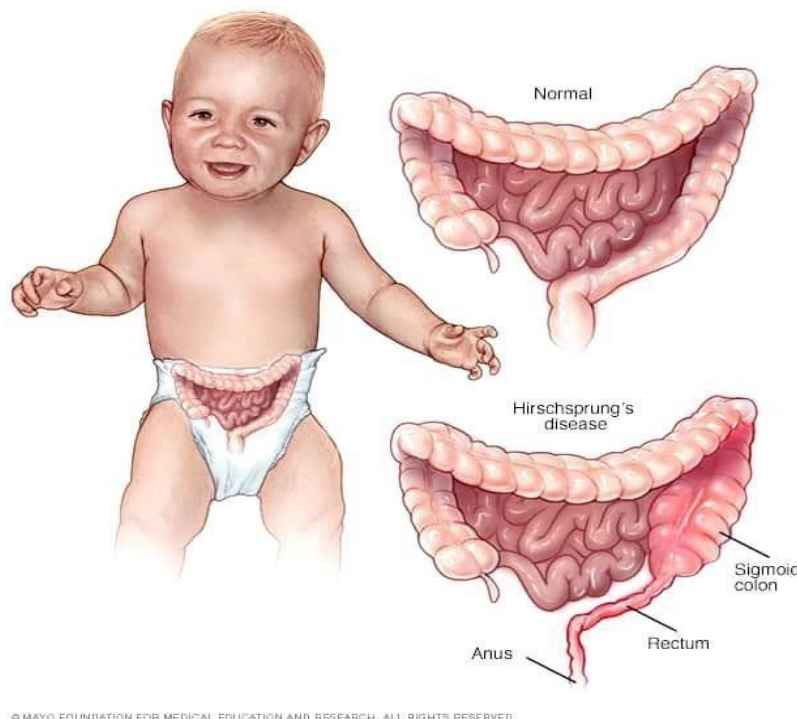


Fig given below illustrates the organic pathology (fig 1.c).^[10]

Most affected patients are diagnosed in infancy. 50% are diagnosed in 1st month of life, 15% by 3 months, 80% by the end of 1st year.

Diagnosis.

Normal internal anal sphincter relaxation with transient rectal distension rules out hirschsprung disease.

Definitive diagnosis of hirschsprung disease requires histologic confirmation of absence of ganglion cells.

Treatment

- Retention enemas of 3 to 4 ounces of olive oil followed by repeated colonic irrigation with isotonic saline solution.
- Rectal catheters can be inserted beyond the constricted segment.

Surgical treatment

- Surgical resection of the affected segment of bowel and various strategies for an ileal or colonic, rectal pull through procedure.
- If pull through technique doesn't prove to be effective then colostomy should be preferred.

Complications

Toxic megacolon or infectious enterocolitis.

Treatment of complications- 1. Broad spectrum parenteral antibiotics

Bowel, rest

Rectal tube placement

Correct electrolyte abnormalities specially (hypokalemia).

Caecostomy /colectomy if in emergency.

Imperforate anus**Incidence**

1:4500 live birth. Common in female child.^[15]

Imperforate anus is usually diagnosed in the neonatal nursery. Passage of meconium is delayed or is noted to take place through an abnormal location as a result of the presence of fistula (e.g. rectovaginal fistula, rectovesicular, or rectoperineal).

Cutaneous collection of meconium in the perineal region, presence of an anal fissure in place of opening and usually a fistulous opening in perineum into vulva or urethra.



Fig below illustrates imperforate anus (fig 1.d)^[12]

Infants often have urological findings. Upto 40% patient have associated lumbosacral lesion such as spina bifida occulta or tethered cord. Bladder of these patient are at risk for voiding dysfunction.

Types of imperforate anus

Low anamolies: Termination of bowel below anorectal bundle. 1. Covered anus –Anal orifice is covered by tag of skin.

Membranous anus: Covered with thin membrane. 3. Anterior ectopic anus: Anus is situated anteriorly. 4. Stenosed Anus: Anal orifice is microscopic.

High Anamolies: They are supralelevator; bowel terminates above the anorectal bundle.

Diagnosis

Wangensteen's Invertogram – 12 hrs after birth, child is held upside down(12 hrs is the time for gas shadow to reach the distal end of gut).

Metal coin is strapped to site of anus and x-ray is taken. If gas shadow is above the pubococcygeal line, it is high anamoly. If the distance between the colon and gas shadow more than 2.5 cm, it is high anamoly.

If the gas shadow is below the pubococcygeal line, it is a low anamoly.

Treatment

Patient require intermittent catheterization to achieve continence.

Low anamoly

- Easy to treat.
- Division of membrane or skin followed by dilatation with plastic reconstruction i.e.anoplasty.

High anamoly

Repaired by three procedure 1st stage-Preliminary transverse colostomy to relieve intestinal obstruction. 2nd stage-When child becomes nearly of 8-10 kg of weight a 'pull through operation' is done with division of fistula. 3rd stage-Colostomy is prefferd by the age of 2 months.

Anal stenosis

The diagnosis of anal stenosis may be delayed beyond the neonatal period, especially if the degree of stenosis is not severe. Any portion of the anal canal or the entire canal may be involved. Constipation is caused by fecal retention secondary to outlet obstruction.

Diagnosis: Digital examination or endoscopy is helpful for the same.

There may be history of passage of stools resembling expelled toothpaste.^[13]

Treatment

Anal dilatation or anorectal myomectomy.

Anterior anal displacement

There are two forms of displacement of the anus.

In anterior ectopic anus the anal canal and internal anal sphincter are displaced anteriorly in the perineum as a unit and are separated from the external anal sphincter, which remains posterior in its usual position.

Symptoms of constipation often begin in neonatal period and are related to the difficulty in expelling stool through a canal that is angled anteriorly.^[14]

Diagnosis

Rectal examination often reveals a sharp posterior angulation in the anal canal. In anterior anal displacement, the entire normal anal unit is located in the anterior perineum. Both entities are commonly found in females.

Treatment

Surgical correction to relocate the anus and relieve the obstruction.

Meconium ileus

Meconium ileus is common presentation in cystic fibrosis of pancreas.

Feeding difficulty, abdominal distension, bilious vomiting between 34 to 48 hrs of birth and fecal pellets. Meconium ileus refers to an intraluminal intestinal obstruction produced by thick meconium. This meconium formed is dry and contains higher than usual concentration of proteins, including albumin. The abnormal meconium adheres firmly to mucosal surface of

the distal small bowel, creating an intraluminal obstruction.^[15]



Figure illustrates appearance of meconium in meconium ileus.(fig.1.e).^[16]

Diagnosis

Physical examination reveals firm palpable masses throughout the abdomen that are freely movable in any direction.

Per abdomen reveals palpable lump.

- Barium meal shows micro colon.
- X-ray shows uneven loop of bowel air, fluid levels, bubbly granular density.

The abdominal film may show in addition to gas filled loops, intraabdominal calcification indicative of intrauterine perforation and meconium peritonitis.

Treatment

In simple meconium ileus, approximately 60% of infants have their obstructions successfully relieved by diagnostic contrast enema using water soluble contrast agent.

Failure of contrast to dislodge the meconium after two attempts is an indication for surgical intervention.

Surgical treatment

Enterotomy with acetylcysteine irrigation and immediate closure.

OR

Resection of ileum with or without construction of various defunctioning ileostomies are rarely indicated.

Prune belley syndrome

Severe hypotonia is seen in prune belley syndrome. Which is elicited by congenital absence of abdominal muscles. Constipation is seen due to the hypotonia.

Classically it shows abdominal wall laxity, bilateral undescended testes and massively dilated upper urinary tract and bladder.



Below is the image showing baby of prune belley syndrome.(fig 1.f).^[17]

Treatment

Surgical reconstructive procedures play an important role in treatment. Thus abdominal wall plication plays important role in the line of treatment.

LATER INFANCY AND CHILDHOOD Anal Fissure

Anal fissure cause pain during defecation and thus may lead to withholding of stools. Later it is difficult to empty such a heavily loaded rectum. Followed by dilation of proximal colon to it.

Fig below illustrates the anal

Treatment

- Encourage fibre diet -Mild laxatives.
- Encourage child not to postpone defeacation.
- Surface anaesthetics like lignocaine jelly -Metronidazole and antibiotics.
- Agents which reduce sphincter pressure should be used e.g. Glyceryl Trinitrate-Reduces spasm and increases vascular perfusion.
- Sitz bath.

Surgical treatment

- Lateral anal sphincterotomy
- Lord's dilatation
- Fissurectomy and local advancement flap.^[18]

Spina bifida and spina bifida occulta

Defecation disturbances, most frequently constipation, are common in patient with spina bifida occulta, especially if the defect involves the lumbosacral spine. The spinal and nerve root defect lead to poor functioning of the terminal bowel.

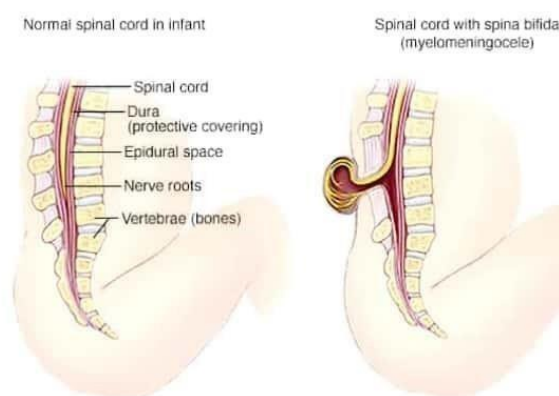


Fig below shows spina bifida(fig 1.f)^[19]

Voluntary external sphincter control and rectoanal sensation are most often diminished or absent. The degree of difficulty in defecation is related to degree and extent of the injury.

Treatment

Conservative treatment becomes essential here.

- Dietary fibres -Stool softners
- Suppositories
- Enema continence catheters can be used for the same.
- Pudendal nerve stimulation are successful in some patient.

CONCLUSION

Since paediatric constipation is a major alarming issue amongst parents and physicians, along with functional, metabolic and endocrinological causes organic pathology too needs to be taken into consideration. This would be quiet helpful for proper diagnosis and management of constipation in paediatric patient.

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