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Evaluation of Partial Colonic Resection in Children with Intractable Constipation and Localized Rectosigmoid Dilatation

Thesis

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 $\mathcal{B}y$

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Tist of Abbreviations

Abb.	Full term
ARM	Anorectal manometry
BMP	Bowel management Program
CTT	Colonic transit time
FC	Functional constipation
HAPCs	High amplitude propagating contractions
IBS	Irritable bowel syndrome
IC	Intractable constipation
ICC	Interstitial cell of Cajal
NICE	National Institute of Clinical Excellence
NTC	Normal transit constipation
PAC -QOL	Patient-Assessment of Constipation - Quality of Life
STC	Slow transit constipation

Introduction

Constipation is referred to as 'idiopathic' if it cannot be explained by anatomical or physiological abnormalities. The exact cause of idiopathic constipation is not fully understood but factors that may contribute include pain, fever, dehydration, dietary and fluid intake, psychological issues, toilet training, medicines, and family history of constipation. Intractable constipation means that trial of medical treatment and bowel management program failed to treat the condition or improve the quality of life after at least 3 months of compliant treatment [1].

Estimates for the prevalence of idiopathic constipation vary from 5% to 30% of children and young people, depending on the criteria used for diagnosis, and are at their highest in toddlers. It is estimated that 1 in 100 children and young people aged between 11 and 18 years have idiopathic constipation ^[2].

Children and young people with Down's syndrome or autism are particularly prone to idiopathic constipation, as are those with physical disabilities, such as cerebral palsy. Continence problems (including soiling) can have a significant emotional impact on children and young people, and managing these problems can be stressful for parents. Many children and young people experience social, psychological, and educational consequences that need prolonged support [1].

AIM OF THE WORK

The aim of this study is to assess the role of localized colonic resection in patients with intractable constipation after failed bowel management program for at least one year.

REVIEW OF LITERATURE

Pathophysiology of Defecation and Related Anatomical Alteration

onstipation can be due to intrinsic problems of colon (primary) or secondary causes like organic disease, systemic disease, or medications.

Primary constipation is typically achieved with a colonic transit test and can be subclassified into NTC, STC, and Defecation disorders. Intrinsic defects in colonic function are considered when there is no obvious causes or alarm features are identified on history. These causes are typically considered after secondary causes of constipation are ruled out, often by history. If treatment is successful, no further workup is generally necessary.

Normal-transit constipation is the most common form of constipation seen. Stool transit is not delayed, and the stool frequency is often within the normal range ⁽³⁾. Most patients are treated empirically and respond well. If empirical therapy fails, further evaluation is suggested to rule out other primary causes of constipation.

Slow-transit constipation (STC) causes infrequent bowel movements (typically less than once per week) and common in young female ⁽⁴⁾. Patients may complain of associated bloating and abdominal discomfort and may not feel the urge to defecate. There is decreased frequency of HAPCs ⁽⁵⁾.

STC is believed to be a neuromuscular disorder of the colon which may be associated with decreased numbers of interstitial cells of Cajal (ICC) (6) and alterations in the number of plexus neurons expressing mventeric the excitatory neurotransmitter substance P (7) in the gut wall of patients with this disorder. The recent London classification of gastrointestinal neuromuscular diseases also lists hypoganglionosis, inflammatory neuropathy and degenerative leiomyopathy as other causes of STC ⁽⁸⁾. Treatment typically uses an aggressive laxative regimen. Patients with STC may respond well to subtotal colectomy and ileorectal anastomosis, provided that there is no significant pelvic floor dysfunction or dyssynergia ⁽⁹⁾.

Defecation disorders (DDs) are a group of functional and anatomical abnormalities of the anorectum that lead to obstructive defecation. Patients may experience severe straining and spend more time on the toilet. DDs can develop as a result of a longterm anxiety of defecation caused by anal fissure discomfort. They typical do manual rectal evacuation with a finger, change positions, or use an enema to defecate. Dyssynergia is the most common functional DD. Less common structural abnormalities are rectal intussusception, prolapse, rectocele and excessive perineal descent (descending perineum syndrome).

Dyssynergia is a result of poor toileting habits, painful defecation, obstetric or back injury, or brain-gut dysfunction (10). Patients with dyssynergia are unable to coordinate the abdominal, rectoanal and pelvic floor muscles during defecation, and may



also demonstrate rectal hyposensitivity (11). Different terms have floor dysfunction, been proposed like anismus, pelvic puborectalis spasm and outlet constipation.

To fulfill the formal diagnostic criteria for dyssynergic defecation, patients must follow Rome IV Diagnostic Criteria for Functional Evacuation Disorders [13]:

- > Patients must satisfy the diagnostic criteria for functional constipation and/or constipation-predominant IBS.
- ➤ Patients must demonstrate dyssynergic pattern during repeated attempts to defecate. A dyssynergic pattern of defecation (Types I-IV) is defined as a paradoxical increase in anal sphincter pressure (anal contraction), or less than 20% relaxation of the resting anal sphincter pressure, or inadequate propulsive forces observed with manometry, imaging or electromyographic recordings.



- ➤ Patients must satisfy one or more of the following criteria:*
 - Inability to expel an artificial stool (50 mL water-filled balloon) within 1-2 minutes.
 - Inability to evacuate or $\geq 50\%$ retention of barium during defecography.

*Some laboratories use a prolonged colonic transit time, ie, greater than 5 markers (≥ 20% marker retention) on a plain abdominal radiography taken 120 hours after ingestion of one radio-opaque marker capsule containing 24 radio-opaque markers.

Types of Dyssynergia:

Type I: The patient can generate an adequate pushing force (rise in intraabdominal pressure) along with a paradoxical increase in anal sphincter pressure.

Type II: The patient is unable to generate an adequate pushing force (no increase in intrarectal pressure) but exhibit a paradoxical anal sphincter contraction.

Type III: The patient can generate an adequate pushing force (increase in intrarectal pressure) but, either has absent or incomplete (< 20%) anal sphincter relaxation (ie, no decrease in anal sphincter pressure).

Type IV: The patient is unable to generate an adequate pushing force and demonstrates an absent or incomplete anal sphincter relaxation.

It should be noted that prolonged colonic transit time can be seen in patients with DD and in patients with STC. It should be noted that diagnosis of primary constipation subtypes often relies on more than a single functional test because of low specificity of used tests.

Delayed colonic transit is believed to be the characteristic of constipation, according to Metz et al., who discovered it in roughly half of his research patients with constipation symptoms that were resistant to fibre supplementation [11]. The causes of delayed transit are unknown, although they might include a decrease in the amount of high amplitude propagated colonic contractions [12] or anatomical changes in the colonic myenteric plexus. There was a significant correlation between increased rectal compliance and slower rectosigmoid transit suggesting combined afferent and efferent neural defect. The loss of parasympathetic innervation to the rectum and sigmoid colon caused by sacral plexus nerve section is well recognised for allowing unopposed sympathetic neural activity, which increases capacitance colonic rectal and decreases transit. Some constipation patients have an imbalance between sympathetic and parasympathetic effects, with sympathetic input dominating, leading in diminished propulsive motor activity and tone.