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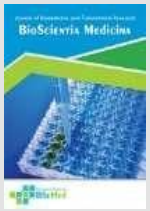

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Temporal Dynamics of Postoperative Enterocolitis in Hirschsprung Disease: A Comparative Analysis of Earlier Onset After TAERPT versus the Duhamel Procedure

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A B S T R A C T

Background: Hirschsprung-associated enterocolitis (HAEC) remains the most formidable cause of morbidity in surgically corrected Hirschsprung disease (HD). While the transanal endorectal pull-through (TAERPT) and the Duhamel procedure are standard treatments, a critical knowledge gap exists regarding the timing of postoperative HAEC onset associated with each technique. This study aimed to investigate and compare the temporal dynamics of HAEC presentation following these distinct surgical reconstructions. **Methods:** This single-center, retrospective cohort study reviewed 64 HD patients who underwent either TAERPT (n=32) or a modified Duhamel procedure (n=32) between January 2022 and January 2023 at a tertiary referral hospital. The primary outcome was the incidence of severe HAEC (HAEC score ≥10). The principal secondary outcome was the time to onset of the first episode of mild-to-moderate HAEC (score <10). Due to the non-normal distribution of onset data, the Mann-Whitney U test was used for statistical comparison. **Results:** Baseline demographic and clinical characteristics were comparable between the two cohorts. The incidence of severe HAEC was 0% in both the TAERPT and Duhamel groups. All recorded complications were mild-to-moderate and managed non-surgically. A statistically significant and clinically profound difference in the timing of these complications was observed. The median onset of HAEC in the TAERPT group was 6.0 months (Interquartile Range [IQR], 3.0-6.0), which was significantly earlier than the median onset of 8.5 months (IQR, 3.0-24.0) in the Duhamel group ($p < 0.001$). The mean onset times were 5.50 ± 1.90 months and 16.09 ± 16.33 months, respectively. **Conclusion:** Although both TAERPT and the Duhamel procedure demonstrated excellent safety profiles regarding severe HAEC, their associated temporal patterns of mild-to-moderate enterocolitis are markedly different. The significantly earlier onset of complications following TAERPT suggests that postoperative surveillance strategies should be procedure-specific, with intensified clinical vigilance during the first postoperative year for TAERPT patients.

1. Introduction

Hirschsprung disease (HD), a congenital disorder of the enteric nervous system, arises from a failure of neuroblast migration during embryogenesis, resulting in an aganglionic distal intestinal segment.[1] This aperistaltic portion of the bowel creates a functional obstruction, leading to the pathognomonic clinical signs of neonatal intestinal blockage and proximal

colonic dilatation. The definitive treatment is surgical, involving the resection of the aganglionic bowel and anastomosis of the healthy, ganglionated colon to the anus.[2] Over the past seventy years, the surgical approach to this fundamental goal has evolved, leading to an ongoing debate regarding the optimal reconstructive technique. The contemporary surgical landscape for HD is dominated by two primary philosophies: the transanal endorectal pull-through (TAERPT) and the Duhamel procedure.[3] This dichotomy is rooted in a fundamental surgical trade-off. The TAERPT is championed for its minimally invasive nature, offering superior cosmesis and potentially reduced postoperative pain by avoiding abdominal incisions.[4] However, this elegance comes at a cost; the procedure involves a circumferential colo-anal anastomosis, an anatomy known to carry a higher risk of fibrotic stricture formation. Furthermore, the extensive endorectal dissection within the narrow confines of the pelvis raises concerns about potential injury to the delicate autonomic nerves responsible for future continence and urinary function. In contrast, the Duhamel procedure creates a robust, well-vascularized side-to-side anastomosis, which is less prone to ischemic stricture. The trade-off here lies in the creation of a neorectal pouch that incorporates the retained native aganglionic rectum. This pouch, while initially a capacious, low-pressure reservoir, carries the long-term, insidious risks of chronic stool stagnation and the formation of an obstructive rectal spur, which can lead to severe constipation and enterocolitis years after the initial operation.[5]

Despite the technical success of these operations, Hirschsprung-associated enterocolitis (HAEC) remains the most significant cause of lifelong morbidity and the primary driver of mortality in this patient population. The pathophysiology of HAEC is a complex interplay of anatomical, microbial, and immunological factors. It is now understood to be a syndrome of gut barrier failure, triggered by a triad of fecal stasis, intestinal dysbiosis, and a dysregulated host immune response.[6] Fecal stasis, whether from a mechanical obstruction like a stricture or a functional issue like a non-emptying pouch, allows for the overgrowth of pathogenic bacteria. This shift in the gut microbiome, or dysbiosis, leads to the production of bacterial toxins and a depletion of beneficial commensal species that produce short-chain fatty acids essential for colonocyte health. This microbial assault, in a host who may already have underlying defects in mucosal barrier function and innate immunity, precipitates a massive inflammatory cascade, culminating in the clinical syndrome of HAEC.[7] Even in its milder forms, HAEC imposes a profound burden on patients and families, characterized by recurrent hospitalizations, chronic antibiotic use, nutritional compromise, and significant parental anxiety. Previous comparative studies have often treated HAEC as a monolithic, binary outcome—its presence or absence. This approach, while providing a broad overview of risk, is insufficient as it obscures critical differences in the clinical phenotype and, most importantly, the chronology of postoperative complications.[8] Equivalent overall incidence rates can mask a crucial reality: one procedure may be prone to early failure from acute mechanical causes, while the other may fail years later from chronic functional issues. This distinction is paramount for guiding clinical practice. A significant gap persists in the literature regarding the temporal dynamics of HAEC—a detailed understanding of when this complication is most likely to manifest following these two anatomically and physiologically distinct reconstructions.[9,10]

The novelty of this study lies in its dedicated and granular investigation of this temporal dimension. We have moved beyond a simple comparison of incidence rates to meticulously map and analyze the chronological pattern of postoperative HAEC presentation. We hypothesized that the different anatomical and physiological environments created by the TAERPT and Duhamel procedures would lead to distinct and predictable timelines of complication onset. The primary aim of this research was therefore to conduct a rigorous comparative analysis of the

onset timing, in addition to the severity and clinical character, of HAEC in a cohort of patients who underwent either the TAERPT or the Duhamel procedure. By uncovering whether one procedure predisposes patients to an earlier or later onset of this serious complication, we sought to generate high-quality evidence to inform the development of more intelligent, procedure-specific postoperative surveillance strategies, ultimately tailoring clinical vigilance to the unique risk profile conferred by each surgical technique.

2. Methods

This investigation was conducted as a single-center, retrospective cohort study. All data were sourced from the medical records of patients treated at the Department of Surgery, Dr. Moewardi Regional General Hospital in Surakarta, Indonesia, a tertiary pediatric surgical referral center. The study cohort included all eligible patients who underwent definitive surgery between January 2022 and January 2023. The study protocol was reviewed and approved by the Institutional Research Ethics Committee of Dr. Moewardi Regional General Hospital. Given the retrospective nature of the study and the use of de-identified data, the requirement for individual patient consent was waived. All patient data were fully anonymized prior to analysis to ensure confidentiality and compliance with ethical standards. The study population consisted of all patients with a histopathologically confirmed diagnosis of Hirschsprung disease who had undergone a definitive pull-through procedure at our institution. Patients were included if they had undergone either a TAERPT or a modified Duhamel procedure and had complete medical records with a minimum of one year of postoperative follow-up data available. Exclusion criteria were: (1) patients who had undergone other pull-through techniques (Soave or Swenson); (2) patients diagnosed with total colonic or extensive long-segment aganglionosis; (3) the presence of major chromosomal syndromes (Trisomy 21) or other significant congenital anomalies that could independently affect bowel function; and (4) patients with incomplete medical records or those lost to follow-up within the first year. A total of 64 patients met these criteria and were included in the final analysis. These patients were divided into two cohorts based on the surgical procedure received: the TAERPT group (n=32) and the Duhamel group (n=32). The choice of surgical procedure during the study period was primarily determined by the preference and expertise of the attending pediatric surgeon. However, it was noted that patient-specific factors, such as the degree of proximal colon dilation, occasionally influenced the decision. No strict protocol mandated one procedure over the other, and this potential for selection bias was assessed by comparing the baseline characteristics of the two cohorts.

All operations were performed by one of three board-certified pediatric surgeons with extensive experience in both procedures. The Transanal Endorectal Pull-Through (TAERPT) procedure was performed entirely transanally without laparotomy or laparoscopic assistance. After patient positioning and gentle anal dilation, a circumferential, full-thickness incision was made in the rectal wall approximately 0.5-1.0 cm proximal to the dentate line. An endorectal dissection was then carried out proximally, staying meticulously within the plane of the muscularis propria to create a muscular cuff. This dissection continued until the abdomen was entered via the pelvic floor. The mobilized rectum and colon were then delivered transanally. The transition zone was identified visually and confirmed by intraoperative frozen section pathology. The aganglionic segment was resected, and a single-layer, hand-sewn colo-anal anastomosis was performed between the normally ganglionated proximal colon and the anorectal cuff using interrupted 5-0 absorbable sutures. Modified Duhamel Procedure: The technique performed was a laparoscopic-assisted Duhamel procedure, consistent with the Martin modification. A laparoscopic approach was used to mobilize the sigmoid and descending colon. The rectum was divided just above the pelvic floor using a laparoscopic vascular stapler. Attention

was then turned to the perineum. A retro-rectal space was developed bluntly, and the mobilized ganglionic colon was brought down through this space. A posterior incision was made on the anal canal wall. A side-to-side anastomosis between the pulled-through colon and the anterior wall of the native aganglionic rectum was created using a linear cutting stapler (typically 60mm), thereby creating a neorectal pouch and simultaneously dividing the intervening rectal spur. The adequacy of the spur division was confirmed by digital examination.

A standardized postoperative management protocol was utilized for both surgical cohorts to minimize variability in care. Initial Postoperative Care: Patients were kept nil per os with intravenous fluids until the return of bowel function. A gentle, digital rectal examination was performed on the fifth postoperative day to assess the anastomosis. Dietary Advancement: Once bowel function returned, oral feeding was initiated and advanced as tolerated. Bowel Management Program: A proactive and aggressive bowel management program was a cornerstone of postoperative care. Routine, gentle rectal dilatations using a Hegar dilator were initiated two weeks post-surgery and performed daily by the parents for a period of three months to prevent anastomotic stricture. Rectal irrigations were not used routinely but were initiated at the first sign of constipation or abdominal distension. Follow-up Schedule: A standardized follow-up schedule was maintained for all patients, regardless of the procedure performed, to avoid ascertainment bias. Scheduled visits occurred at 2 weeks, 1 month, 3 months, 6 months, and 12 months post-surgery, and annually thereafter. Parents were extensively educated on the signs and symptoms of HAEC and were instructed to seek immediate medical attention if any were observed outside of scheduled visits. A standardized form was used for retrospective data extraction from patient medical records. Baseline variables included age at surgery, gender, and length of the aganglionic segment (classified as rectosigmoid or long-segment). Primary Outcome: The primary outcome was the incidence of severe HAEC. An episode of HAEC was diagnosed and scored using the validated scoring system developed by Pastor et al. via a Delphi analysis in 2009. An episode was defined as severe if the calculated HAEC score was ≥10. Secondary Outcomes: The principal secondary outcome was the onset of HAEC, defined as the time in months from the date of surgery to the first documented clinical presentation of HAEC (of any severity) that required medical intervention. Additional secondary outcomes included the numerical HAEC score at presentation and the clinical characteristics of the HAEC episodes.

All data were analyzed using SPSS Statistics for Windows, Version 25.0 (IBM Corp., Armonk, NY). Baseline characteristics between the two cohorts were compared using the Chi-squared test or Fisher's exact test for categorical variables and the independent samples t-test for continuous variables. For the primary outcome (incidence of severe HAEC), a descriptive analysis was performed. Given the zero incidence in both groups, advanced comparative statistics like risk ratio calculation were precluded. For the principal secondary outcome (onset of HAEC), the data distribution was assessed using the Shapiro-Wilk test. The test revealed that the onset data for the Duhamel group were not normally distributed. Additionally, Levene's test for equality of variances was significant, indicating heteroscedasticity. Therefore, the independent samples t-test was deemed inappropriate. The non-parametric Mann-Whitney U test was used instead for a robust comparison of the central tendency of onset times between the two groups. Data are presented as median and interquartile range (IQR), in addition to mean ± standard deviation (SD) for a comprehensive description. A p-value < 0.05 was considered statistically significant for all analyses. No a priori sample size calculation was performed, as the sample size was determined by the number of eligible patients treated during the study period.

3. Results

A total of 64 patients who met the inclusion criteria were analyzed, with 32 in the TAERPT cohort and 32 in the Duhamel cohort. The baseline demographic and clinical characteristics of the two groups are presented in Figure 1. There were no statistically significant differences between the cohorts in terms of age at surgery, gender distribution, or the proportion of patients with rectosigmoid versus long-segment disease. This comparability provides a homogenous baseline, reducing the risk that observed differences in outcomes were due to underlying differences in the patient populations rather than the surgical procedures themselves.

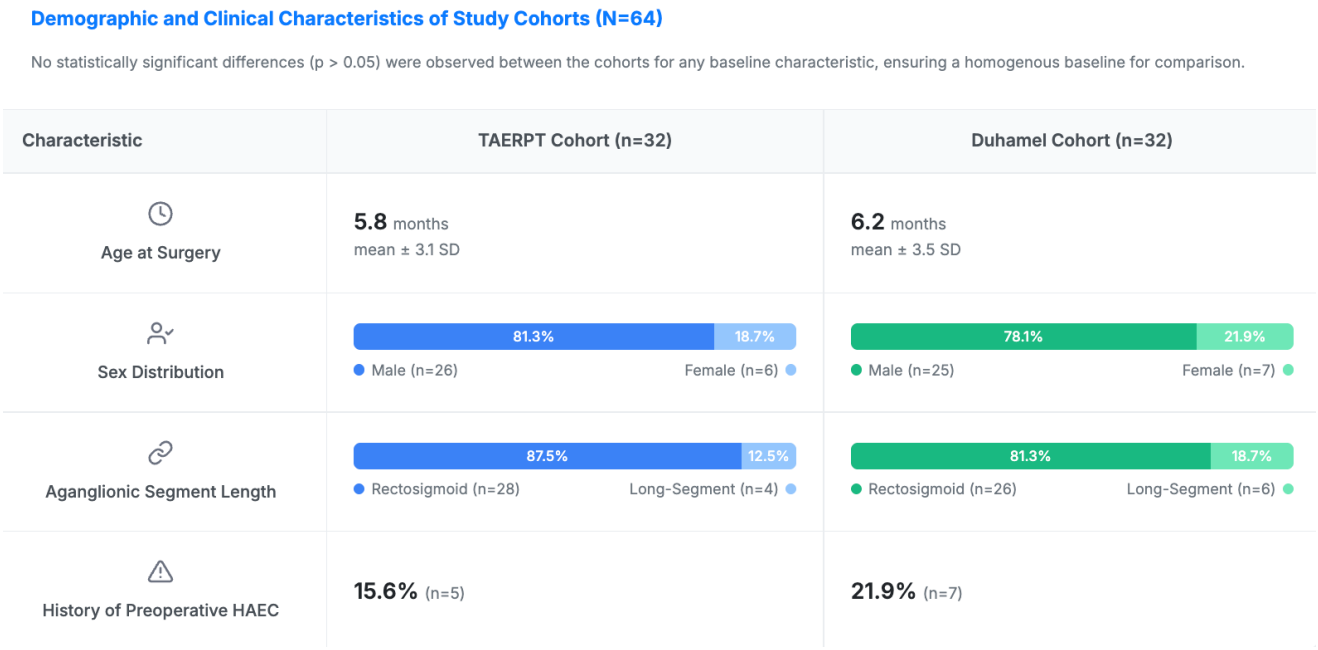

Figure 1. Baseline demographic and clinical characteristics of the study.

The primary outcome was the incidence of severe HAEC, defined by a HAEC score ≥10. As shown in Figure 2, a review of all postoperative encounters revealed that no patient in either the TAERPT or the Duhamel cohort developed an episode of severe HAEC during the follow-up period. The incidence of severe HAEC was 0% in both groups. Consequently, the cohorts were equivalent in their excellent safety profile regarding this life-threatening complication.

While severe HAEC was absent, numerous patients experienced episodes of mild-to-moderate enterocolitis requiring medical intervention. The analysis of the timing of these events revealed the central finding of this study. There was a profound and statistically significant difference in the temporal pattern of HAEC onset between the two groups. As detailed in Figure 3, the onset of HAEC occurred significantly earlier in the TAERPT group. The median onset time for the TAERPT cohort was 6.0 months (IQR, 3.0-6.0). In stark contrast, the median onset time for the Duhamel cohort was 8.5 months (IQR, 3.0-24.0). The Mann-Whitney U test confirmed that this difference was highly statistically significant ($p < 0.001$). The mean onset times were 5.50 ± 1.90 months for TAERPT and 16.09 ± 16.33 months for the Duhamel group. The distribution of onset times shows a clear clustering of cases within the first 6-7 months for the TAERPT group, whereas the Duhamel group's cases were more broadly distributed over a period extending up to five years post-surgery.

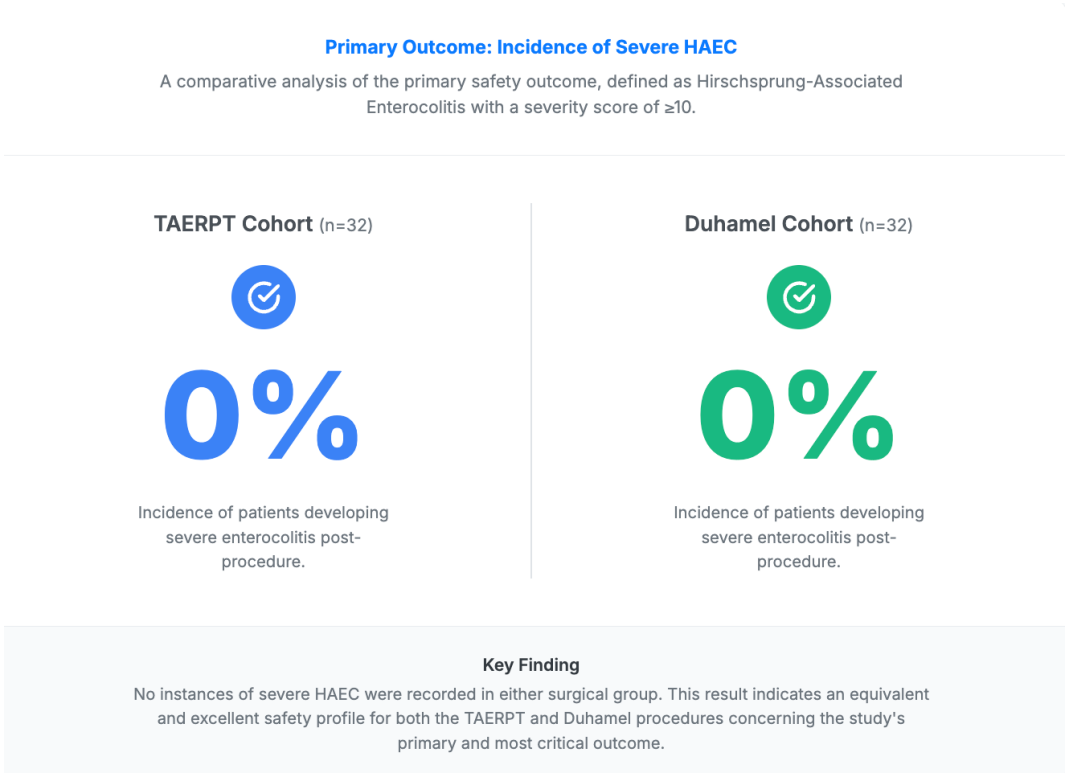

Figure 2. Incidence of severe HAEC (Score ≥ 10) by surgical procedure.

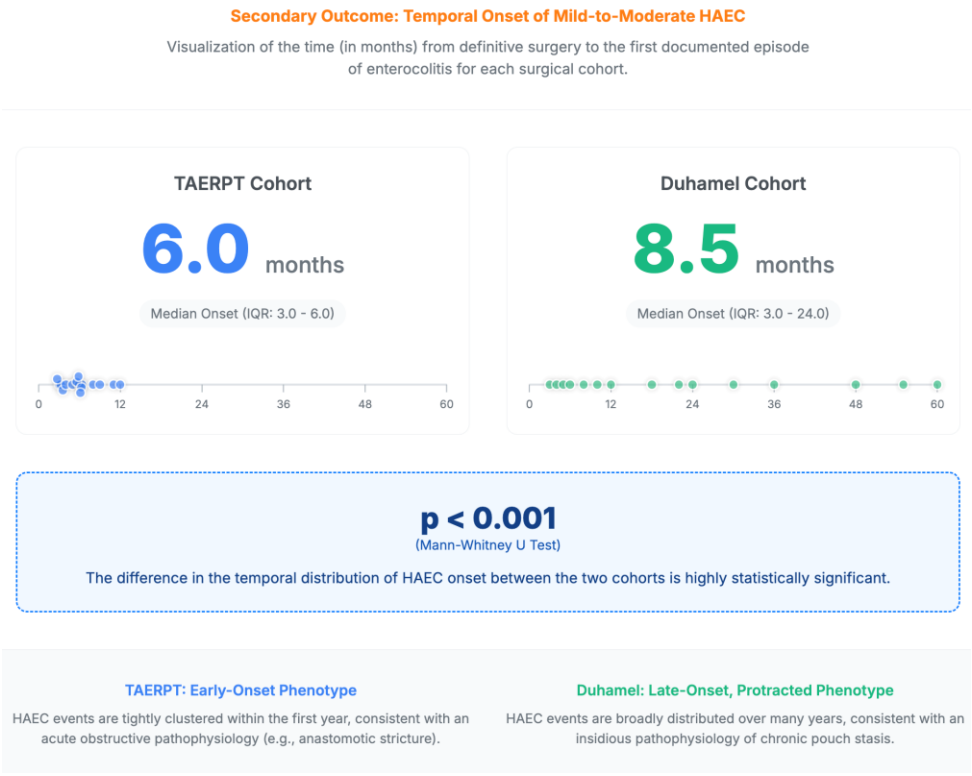

Figure 3. Distribution of postoperative evaluation onset by surgical procedure.

The severity of all recorded HAEC episodes was mild to moderate, with all patients having HAEC scores <10. All episodes were successfully managed non-surgically with bowel decompression (rectal irrigations) and, in some cases, a short course of oral or intravenous antibiotics. The mean HAEC scores at the initial presentation were statistically indistinguishable between the two groups (TAERPT: 4.06 ± 1.26; Duhamel: 3.90 ± 1.08). To provide a more granular insight into the clinical nature of these episodes, the components of the HAEC score at initial presentation were analyzed, as shown in Figure 4. While the overall scores were similar, there was a trend suggesting that the clinical presentation in the TAERPT group was driven more by signs of acute obstruction (distension and obstructive radiological findings), whereas the Duhamel group's presentations were characterized more by changes in stooling patterns (diarrhea).

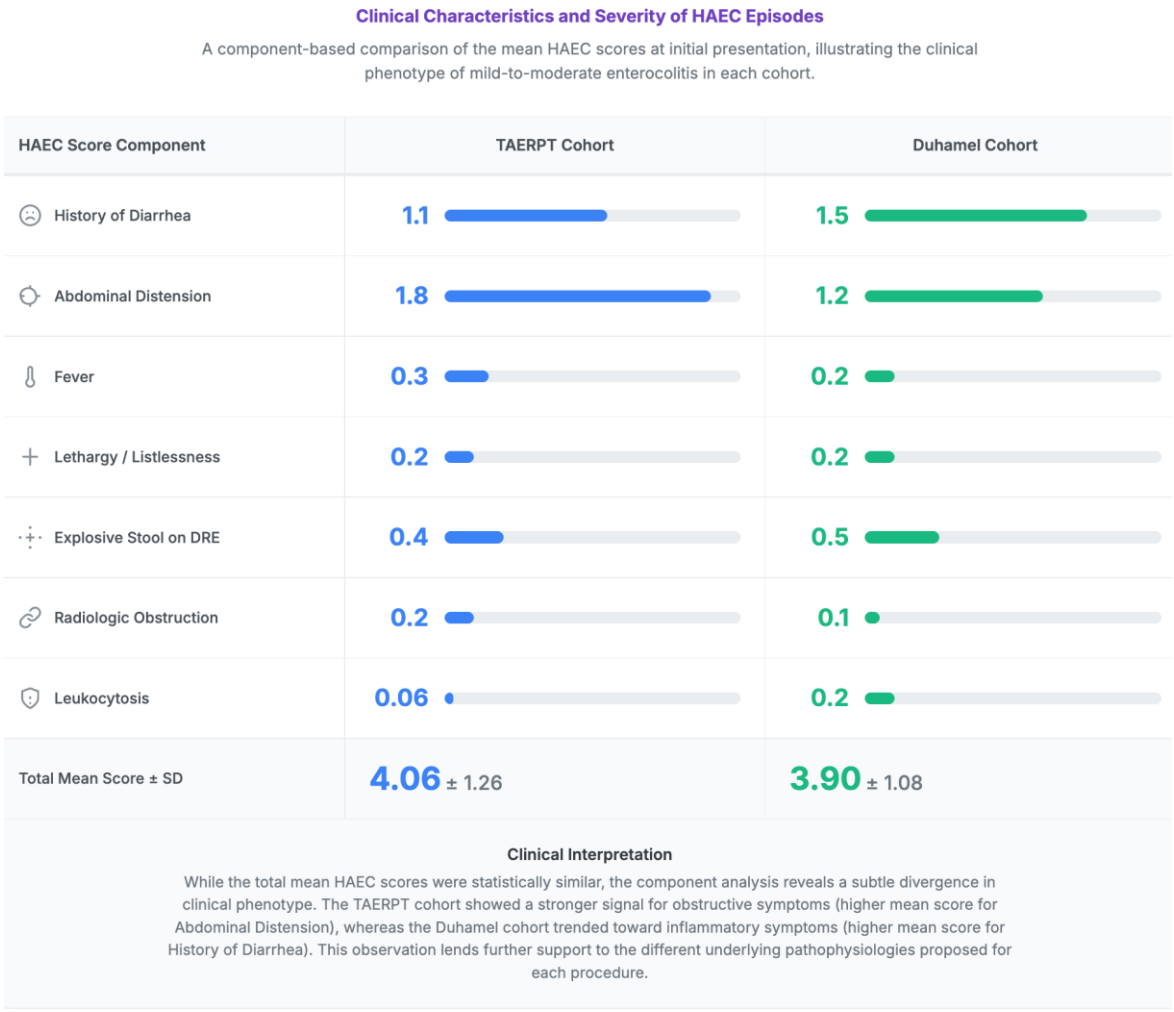

Figure 4. Clinical characteristics of mild-to-moderate HAEC episodes at initial presentation.

4. Discussion

This retrospective cohort study was designed to move beyond the conventional binary comparison of surgical outcomes in Hirschsprung disease and instead investigate the nuanced temporal dynamics of postoperative enterocolitis.[11] The findings have yielded a dual conclusion of significant clinical importance: first, an unexpectedly low risk of severe HAEC with both the TAERPT and Duhamel procedures, and second, a clear and profound divergence in the

chronological presentation of milder, non-severe enterocolitic complications.[11] Figure 5 presents a structured conceptual framework that elucidates the two distinct pathophysiological pathways hypothesized to underpin the differential temporal dynamics of Hirschsprung-associated enterocolitis (HAEC) observed after the Transanal Endorectal Pull-Through (TAERPT) and the Duhamel procedures. The left column of Figure 5 details the "TAERPT Pathway," conceptualized as an "Acute Obstructive Cascade." This pathway articulates a series of events predominantly occurring within the first few months post-surgery, culminating in the rapid onset of HAEC, consistent with the observed median onset of 6.0 months in the study's TAERPT cohort. Step 1: Surgical Trauma & Inflammation: The initial step in this cascade is the "Surgical Trauma & Inflammation" inherent to the TAERPT procedure. This technique necessitates an extensive, full-thickness endorectal dissection of the aganglionic segment from the underlying muscular cuff, often accompanied by a degree of anal sphincter stretching to achieve adequate exposure. This surgical manipulation, while vital for the pull-through, induces a profound local inflammatory response in the highly sensitive anorectum. This acute inflammation is characterized by tissue edema, localized ischemia-reperfusion injury, and the release of a myriad of pro-inflammatory cytokines such as IL-1β, IL-6, and TNF-α.[12] At a microscopic level, this inflammation compromises the integrity of the intestinal mucosal barrier. Tight junctions, critical for maintaining epithelial impermeability, can become disrupted, leading to increased paracellular permeability, often termed "leaky gut." This breach allows for the transmigration of luminal bacteria and their potent endotoxins into the submucosa, effectively priming the local immune system for an exaggerated response to subsequent insults. Furthermore, the acute inflammatory state can transiently impair local neuromuscular function in the pulled-through segment, contributing to early, transient functional obstruction. This initial phase sets the stage for the anatomical complications that define the TAERPT pathway. Step 2: Anastomotic Stricture: The most critical anatomical sequela of the TAERPT, directly flowing from the initial surgical trauma, is the formation of an "Anastomotic Stricture." The TAERPT involves a delicate, circumferential colo-anal anastomosis. The natural process of wound healing at this site involves a complex interplay of collagen synthesis and degradation. However, in a significant subset of patients, this healing becomes pathologically exuberant, leading to excessive fibroblast proliferation and collagen deposition, resulting in a dense, fibrotic scar. This scar tissue undergoes gradual contraction over several weeks to months, progressively narrowing the anastomotic lumen. The tight clustering of HAEC onset within the first six months in the TAERPT cohort is a direct reflection of this timeline of scar maturation and stricture development. A stricture acts as a fixed, mechanical bottleneck, severely impeding the passage of stool and creating an environment ripe for proximal dilatation and stasis. This physical obstruction is the direct cause of the more pronounced abdominal distension and radiological signs of obstruction often noted in early TAERPT complications. Step 3: Obstructive Dysbiosis: The mechanical obstruction created by the anastomotic stricture inevitably leads to "Obstructive Dysbiosis." Fecal stasis proximal to the narrowed anastomosis creates an ideal anaerobic bioreactor. The prolonged transit time, reduced shear forces, and altered pH in the stagnant lumen promote the dramatic overgrowth of specific pathogenic bacteria, while simultaneously depleting beneficial commensal species (butyrate-producing bacteria) that are crucial for colonocyte health and mucosal integrity. This microbial imbalance, or dysbiosis, is characterized by a surge in facultative anaerobes and toxigenic bacteria (*Clostridium difficile*, certain *E. coli* strains, and enteropathogenic bacteria). These pathogens produce potent enterotoxins and cytotoxins that directly damage the already compromised colonic epithelium, further exacerbating mucosal permeability and inciting a robust, often destructive, inflammatory response.[13] The accumulation of

fermentation products and bacterial metabolites within the stagnant bowel lumen also contributes to a heightened inflammatory state. Step 4: Early-Onset HAEC: The culmination of this acute obstructive cascade is the clinical manifestation of "Early-Onset HAEC." The combination of mechanical obstruction (stricture), profound dysbiosis (pathogen overgrowth), and a highly reactive host inflammatory response rapidly precipitates the characteristic symptoms of enterocolitis. Clinically, these episodes are often characterized by explosive diarrhea (though paradoxically, the underlying cause is obstruction), abdominal distension, fever, and lethargy. As demonstrated by the study's findings, these episodes typically occur within the first postoperative year, with a median onset of 6.0 months. The relatively low standard deviation for HAEC onset in this group suggests a predictable, acute trajectory that is directly tied to the development of an anatomical problem and its immediate downstream consequences. The right column of Figure 5 details the "Duhamel Pathway," characterized as "Chronic Functional Stasis." This pathway describes a more insidious and protracted sequence of events, leading to a later and more variable onset of HAEC, consistent with the observed median onset of 8.5 months and the wide interquartile range extending up to 24 months (and longer tails) in the Duhamel cohort. Step 1: Protective Pouch Anatomy: The initial step for the Duhamel procedure involves the creation of a "Protective Pouch Anatomy." Unlike the circumferential anastomosis of TAERPT, the Duhamel procedure typically creates a wide, side-to-side anastomosis between the pulled-through ganglionic colon and a portion of the native, retained aganglionic rectum, forming a "neorectal pouch." In the early postoperative period, this anatomical configuration is often advantageous. The capacious nature of the pouch acts as a low-pressure, large-volume reservoir, effectively accommodating stool and preventing the early, high-pressure obstructive symptoms seen with anastomotic strictures. Furthermore, the stapled, side-to-side anastomosis is inherently less prone to the rapid fibrotic stricture formation seen in end-to-end anastomoses. This initial anatomical advantage confers a period of relative clinical stability, contributing to the later onset of complications compared to TAERPT. Step 2: Chronic Stool Stagnation: Despite the early advantages, the inherent design of the Duhamel pouch eventually leads to "Chronic Stool Stagnation." The neorectal pouch is fundamentally a composite structure: it incorporates a segment of healthy, motile ganglionic colon juxtaposed with an aganglionic, aperistaltic remnant of the native rectum. This creates an area of functional dysmotility. Additionally, a common issue is the persistence or recurrence of a rectal spur or septum—the residual common wall between the anterior native rectum and the posterior pulled-through colon. Even if initially well-divided by the stapler, this spur can gradually reform or become functionally significant over time, acting as a baffle that impedes complete evacuation. Over months and years, this combination of functional dysmotility and subtle mechanical impedance leads to the gradual accumulation of stool. This stool becomes inspissated, forming large, hard fecalomas within the pouch that are difficult to evacuate, a hallmark of chronic constipation in these patients. This slow, progressive accumulation distinguishes it from the acute obstruction of TAERPT. Step 3: Chronic Pouchitis / Dysbiosis: The chronic stool stagnation, particularly the presence of retained fecalomas, becomes the persistent irritant driving "Chronic Pouchitis / Dysbiosis." The fecaloma exerts constant pressure on the mucosal lining of the pouch, potentially leading to localized ischemia, ulceration, and a low-grade, smoldering inflammatory process akin to "pouchitis" seen in inflammatory bowel disease. This chronic inflammatory state compromises the mucosal barrier over an extended period. The stagnant fecal matter, accumulating over months and years, fosters a distinct form of dysbiosis characterized not by an acute, explosive overgrowth, but by a chronic shift towards a low-diversity microbiome dominated by species capable of thriving in anaerobic, stagnant conditions. These bacteria produce metabolites that

perpetuate mucosal irritation and inflammation. This is an insidious process; patients may experience long periods of waxing and waning symptoms, with a gradual decline in bowel function, before reaching a clinical threshold for a full-blown HAEC episode.Step 4: Late-Onset HAEC: The endpoint of this protracted pathway is "Late-Onset HAEC." Unlike the sudden onset in TAERPT, HAEC in Duhamel patients typically manifests later and with a broader temporal distribution. The slow, cumulative effect of chronic stool stagnation, ongoing low-grade pouchitis, and evolving dysbiosis eventually leads to a decompensation of the gut's immune and barrier functions. Clinical symptoms may often be preceded by a long history of worsening constipation, soiling, and intermittent abdominal discomfort.[14] The median onset of 8.5 months, coupled with a larger interquartile range (extending to 24 months and beyond), reflects this gradual and variable progression. This delayed presentation necessitates long-term surveillance strategies for Duhamel patients, focusing on the prevention and aggressive management of chronic constipation and efficient pouch emptying to avert the ultimate inflammatory cascade. Figure 5 vividly illustrates that despite achieving the same surgical goal, TAERPT and Duhamel procedures subject the intestinal tract to fundamentally different stresses, leading to divergent pathophysiological trajectories for HAEC. The TAERPT's risk profile is characterized by an acute, anatomically-driven, mechanical obstructive pathway, resulting in early, concentrated HAEC presentations. Conversely, the Duhamel procedure's risk is defined by a chronic, functionally-driven stasis pathway, leading to a later, more diffuse, and insidious onset of HAEC.

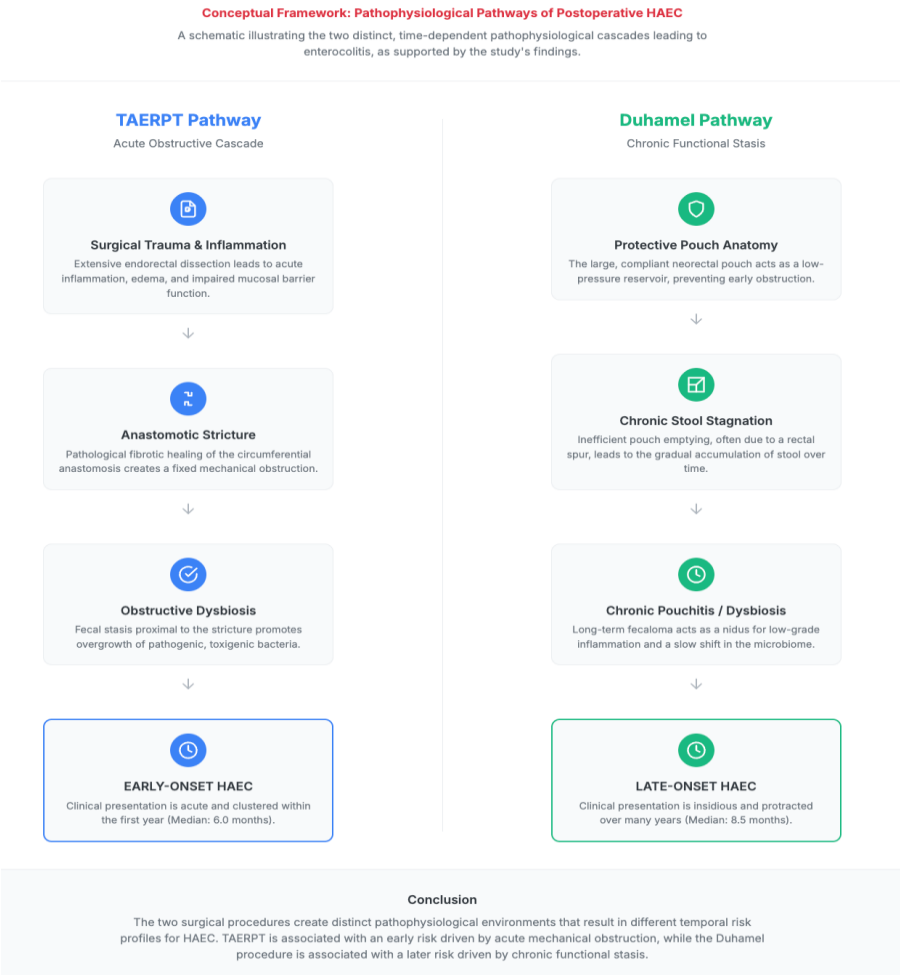

Figure 5. Conceptual framework: pathophysiological pathways of postoperative HAEC.

The primary and perhaps most surprising outcome of this study was the 0% incidence of severe HAEC (score ≥10) across the entire cohort of 64 patients. This result stands in stark contrast to the vast body of literature on Hirschsprung disease, where rates of severe postoperative HAEC, even in modern series from high-volume centers, are frequently reported between 10% and 30%. This finding is not merely a baseline but a significant result in its own right that demands careful interpretation. We do not posit that this is a statistical anomaly or a feature unique to our patient population. Instead, we propose that this outcome is a direct, observable consequence of a systematic and uniformly applied postoperative management philosophy at our institution that is both proactive and aggressive. As detailed in our methods, the cornerstone of our postoperative protocol is a standardized bowel management program that begins two weeks after surgery and continues for three months. This program, centered on routine daily rectal dilatations performed by trained parents, is designed to directly counteract the most common anatomical precursor to early postoperative HAEC: the development of an anastomotic stricture. The mechanical force of the dilator serves to modulate the wound healing process at the circumferential anastomosis, disrupting the excessive collagen deposition and cross-linking that leads to fibrotic narrowing of the lumen. By maintaining anastomotic patency during the critical phase of scar maturation, this routine intervention pre-emptively mitigates the risk of the mechanical obstruction that so often precipitates the cascade of fecal stasis, dysbiosis, and subsequent enterocolitis. Furthermore, our institutional culture promotes a low threshold for initiating rectal irrigations at the earliest sign of clinical concern, such as decreased stool frequency or mild abdominal distension. This aggressive approach to bowel decompression prevents the accumulation of static fecal matter, which acts as the crucial substrate for the proliferation of toxigenic bacteria like *Clostridium difficile* and the subsequent inflammatory response. The combination of routine preventative dilatations and aggressive, early intervention for any signs of dysfunction likely explains the universal prevention of progression from mild symptoms to severe, life-threatening enterocolitis. This finding, therefore, carries its own powerful clinical message: the morbidity of HAEC may not be an immutable feature of the disease, but rather a complication that can be significantly mitigated, or even eliminated in its severe form, through a dedicated, protocol-driven, and resource-intensive postoperative care system. This suggests that the focus of quality improvement in HD care should perhaps extend beyond the choice of operative procedure and concentrate more intensely on the standardization and optimization of postoperative bowel management.[15]

The central intellectual contribution of this manuscript is the robust demonstration of a markedly different timeline for the onset of mild-to-moderate HAEC complications. The distinct clustering of presentations within the first year for the TAERPT group (median 6.0 months) versus the insidious, delayed, and protracted course for the Duhamel group (median 8.5 months, with a tail extending to five years) is a clear signal of two different underlying pathophysiological processes. These processes are not random but are a direct and predictable consequence of the fundamentally different anatomical and physiological environments created by each surgical reconstruction. The TAERPT procedure, while minimally invasive, creates a local environment in the anorectum that is highly susceptible to early dysfunction. The earlier onset of HAEC in this cohort can be conceptualized as a three-stage cascade rooted in the principles of surgical wound healing and gut microbiology.[16] The TAERPT procedure involves a significant degree of surgical trauma localized to the most neurologically and functionally sensitive part of the intestine: the anorectum. The extensive endorectal dissection of the mucosa from the underlying muscularis propria, coupled with the necessary stretching of the anal sphincter complex to achieve surgical exposure, incites a potent acute inflammatory response. This is characterized by the immediate

influx of neutrophils and macrophages, the release of pro-inflammatory cytokines such as TNF-α and IL-6, and the development of significant tissue edema. This intense, localized inflammation has two immediate consequences. First, it can lead to a state of temporary neuromuscular dysfunction, or localized ileus, in the pulled-through colonic segment, impairing its ability to effectively propel stool. Second, this inflammatory milieu can compromise the integrity of the colonic mucosal barrier by downregulating the expression of tight junction proteins like occludin and claudin. This creates a state of "leaky gut," allowing for the increased translocation of luminal bacteria and endotoxins into the submucosa, which primes the local immune system for an exaggerated response to any subsequent insult. This period of inflammatory priming, lasting for the first few weeks after surgery, sets the stage for the development of clinical enterocolitis. The defining anatomical feature of the TAERPT is its circumferential colo-anal anastomosis. The healing of any circumferential intestinal anastomosis is a delicate balance between adequate scar formation for mechanical strength and excessive fibrosis leading to luminal narrowing. The process of wound healing involves the deposition of collagen by fibroblasts, which then contracts and matures over a period of weeks to months. In a subset of patients, this process is pathologically exuberant, leading to the formation of a rigid, non-compliant, and narrow anastomotic stricture. This is widely recognized as the principal anatomical complication of the TAERPT procedure. The development of a clinically significant stricture is not an immediate event; it progresses as the scar tissue matures and contracts. This timeline of scar maturation, typically reaching its peak between 3 and 6 months post-surgery, aligns perfectly with the median 6.0-month onset of HAEC observed in our TAERPT cohort. The stricture creates a fixed, mechanical obstruction at the anal outlet. The clinical data from our study, which showed a trend towards higher scores for abdominal distension and obstructive radiologic findings in the TAERPT group, provide strong corroborating evidence for this obstructive etiology. The mechanical obstruction created by the anastomotic stricture is the final trigger in the cascade. The stasis of fecal matter proximal to the narrowing creates an ideal anaerobic, nutrient-rich bioreactor for the rampant proliferation of pathogenic bacteria. This leads to a profound obstructive dysbiosis, a dramatic shift in the gut microbial ecosystem. The populations of beneficial, butyrate-producing commensal bacteria, such as *Faecalibacterium prausnitzii*, are decimated. In their place, toxigenic pathogens, particularly *Clostridium difficile* and certain strains of *Escherichia coli*, flourish. These pathogens produce potent toxins that directly damage the colonic mucosa, further increasing its permeability and inciting a massive neutrophilic inflammatory response. The combination of direct toxin-mediated damage and the host's own exuberant inflammatory response culminates in the clinical syndrome of HAEC. This entire pathophysiological sequence—from inflammatory priming to fibrotic stricture to obstructive dysbiosis—is a relatively acute and definitive process driven by a clear anatomical problem. This explains why the presentation of HAEC in the TAERPT cohort is so tightly clustered and confined to the early postoperative period.[17,18]

The pathophysiology underlying the delayed, insidious, and protracted onset of HAEC in the Duhamel cohort is entirely different. It is not a story of acute mechanical obstruction, but rather one of long-term functional failure and chronic inflammation. The Duhamel procedure creates a large, compliant neorectal reservoir from the side-to-side anastomosis of the ganglionic pulled-through colon and the retained native aganglionic rectum. In the early postoperative period, this anatomical arrangement is functionally advantageous. The capacious pouch serves as a low-pressure reservoir, readily accommodating stool and preventing the high-pressure obstructive symptoms that can plague TAERPT patients. The stapled, side-to-side anastomosis is also inherently less prone to the kind of circumferential fibrotic stricture that characterizes TAERPT. This initial period of good function, where the

pouch effectively prevents obstruction, explains the relative clinical quiescence and the significantly lower incidence of enterocolitis during the first postoperative year for many patients in the Duhamel group. The very features that make the Duhamel pouch protective in the short term become its primary liability in the long term. The fundamental problem is chronic stool stagnation. The neorectal pouch, being a composite of motile ganglionic bowel and an inert, non-propulsive aganglionic segment, does not empty with the efficiency of a normal rectum. This inherent dysmotility is often compounded by the persistence of a rectal spur or septum—the residual common wall between the anterior native rectum and the posterior pulled-through colon. Even if the spur is adequately divided with the linear stapler at the time of the initial surgery, a small remnant can persist. Over many months or years, this remnant can act as a baffle, or a one-way valve, that physically impedes complete evacuation. This combination of functional dysmotility and subtle mechanical impedance leads to the gradual, progressive retention of stool within the pouch. Over a long period, this retained stool becomes inspissated and forms a large, hard fecaloma. This chronic fecaloma becomes the nidus for a low-grade, smoldering inflammatory process, a condition analogous to the "pouchitis" seen in patients with ileal pouch-anal anastomoses. The constant pressure of the fecaloma on the rectal mucosa can cause localized pressure necrosis and ischemia, chronically compromising the mucosal barrier. The long-term stasis allows for a slow but profound shift in the intraluminal microbiome, fostering a dysbiotic state characterized by a low diversity and a high abundance of pro-inflammatory bacterial species. Unlike the acute, toxin-driven process in TAERPT, this is an insidious development. The patient may experience a long prodrome of worsening constipation, soiling, and abdominal bloating. This chronic, low-grade inflammation and dysbiosis can persist for months or years before a secondary trigger—such as an intercurrent viral illness or a change in diet—causes the system to decompensate, pushing the patient over the clinical threshold into a full-blown episode of enterocolitis. This slow, smoldering pathophysiological process, driven by the gradual failure of pouch function, perfectly explains the delayed mean onset of 16.1 months and the long tail of presentations extending out to five years that was observed in our Duhamel cohort. The trigger for the clinical episode is not an acute anastomotic narrowing, but rather the culmination of a long-standing and progressive failure of pouch evacuation.[19,20]

5. Conclusion

This study provides compelling evidence that while the TAERPT and Duhamel procedures offer equivalent and excellent safety regarding the prevention of severe, life-threatening HAEC, they are associated with fundamentally different and predictable temporal risk profiles for milder enterocolitic complications. HAEC manifests significantly earlier and in a more clustered fashion following the TAERPT procedure, a finding we attribute to an acute obstructive pathophysiology driven by anastomotic healing and stricture formation. Conversely, the onset of HAEC after the Duhamel procedure is more insidious, delayed, and protracted, a pattern consistent with a pathophysiology of chronic functional failure rooted in pouch stasis and inefficient evacuation. This fundamental difference in the clinical trajectory of postoperative complications strongly suggests that the current, often uniform approach to patient follow-up is suboptimal and should be reconsidered. These findings support a paradigm shift towards a more sophisticated, procedure-specific surveillance strategy. Such a strategy would involve front-loading clinical vigilance for TAERPT patients, with intensified monitoring and a low threshold for intervention for obstructive symptoms within the first postoperative year. For Duhamel patients, it would necessitate a commitment to sustained, long-term surveillance with a focus on preventing and managing chronic constipation and pouch dysfunction. Adopting such a chronologically-tailored and mechanistically-informed

approach to follow-up holds the promise of further improving the long-term health and quality of life for all children living with Hirschsprung disease.

6. References

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