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The "Ascending Cascade" of Colorectal Motility Disorders: A Narrative Review of Hindgut-Driven Global Gut Dysfunction and Foregut–Airway Sequelae

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Abstract

Objectives: To synthesise emerging evidence that colorectal motility disorders, traditionally viewed as isolated hindgut conditions, may initiate a progressive, system-wide sequence of gastrointestinal and extra-intestinal dysfunction. This review proposes an "ascending cascade" model in which partial functional obstruction of the hindgut triggers upstream effects on the midgut, foregut, biliary-pancreatic axis, and respiratory and ENT systems.

Design: Narrative review.

Data Sources: PubMed, Embase, Scopus, and Google Scholar were searched for literature relating to colorectal motility disorders, partial obstruction, dysbiosis, small bowel bacterial overgrowth, duodenal compression syndromes, biliary disease, reflux, and gut–airway interactions. Additional anatomical and physiological sources were reviewed to support mechanistic interpretation.

Eligibility Criteria: Studies, case series, anatomical analyses, and mechanistic reports describing hindgut obstruction, colonic stasis, vascular watershed vulnerability, ileocecal reflux, midgut dysfunction, duodenal compression, biliary sequelae, and foregut or respiratory manifestations were included. Both paediatric and adult populations were considered.

Results: Evidence suggests that hindgut partial functional obstruction generates intraluminal hypertension, segmental distention, and low-grade ischemia at antimesenteric and marginal arcade watershed zones. These changes promote mucosal barrier disruption, bacterial translocation, dysbiosis, appendiceal stasis, ileocecal reflux, backwash ileitis, Peyer's patch activation, and mesenteric adenitis. Proximal propagation of pressure and stasis contributes to small bowel bacterial overgrowth and ileal dysfunction. Mesocolonic traction at the duodenojejunal flexure may produce functional variants of midgut malrotation or non-rotation, while high DJ insertion can mimic superior mesenteric artery syndrome, leading to duodenitis, pancreatitis, cholecystitis, cholelithiasis, and biliary gastritis. These midgut and biliary disturbances subsequently impair gastric emptying and oesophageal barrier function, resulting in gastro-oesophageal reflux, reflux oesophagitis, and laryngopharyngeal reflux. Chronic refluxate exposure is associated with recurrent respiratory infections, tonsillitis, sinusitis, otitis media, and periodontal disease.

Conclusions: Colorectal motility disorders may represent the initiating event in a broader, ascending continuum of gastrointestinal and extra-intestinal pathology. Recognising this cascade may improve diagnostic accuracy, reduce fragmented care, and support earlier, more holistic intervention. Further research is needed to validate anatomical and mechanistic pathways and to determine whether targeted treatment of hindgut dysfunction can prevent upstream complications.

Keywords: Colorectal Motility Disorders; Hindgut Obstruction; Intraluminal Hypertension; Watershed Ischemia; Dysbiosis; Ileocecal Reflux; Small Bowel Bacterial Overgrowth (SIBO); Duodenojejunal Flexure; Mesocolonic Traction; Midgut Malrotation Variants;

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Superior Mesenteric Artery (SMA) Syndrome; Duodenitis; Pancreatitis; Biliary Stasis; Gastro-Oesophageal Reflux Disease (GERD); Laryngopharyngeal Reflux; Respiratory and ENT Infections; Global Gut Dysfunction; Ascending Cascade Model

Summary Box

What is already known on this topic

- Colorectal motility disorders can cause distal bowel stasis, intraluminal hypertension, and functional partial obstruction.
- Colonic distention may impair mucosal perfusion at antimesenteric and marginal arcade watershed zones, predisposing to low-grade ischemia and barrier dysfunction.
- Dysbiosis, small bowel bacterial overgrowth, appendiceal stasis, and ileocecal reflux are recognised complications of chronic hindgut dysfunction.
- Duodenal compression syndromes (e.g., SMA syndrome) and biliary stasis can arise from anatomical variants or altered mesenteric tension.
- Gastro-oesophageal reflux and laryngopharyngeal reflux are known contributors to recurrent ENT and respiratory infections.

What this study adds

- Proposes a unified "Ascending Cascade" model in which hindgut partial obstruction initiates a progressive chain of dysfunction extending through the midgut, foregut, and airway systems.
- Identifies mesocolonic traction at the duodenojejunal flexure as an under-recognised mechanical pathway linking hindgut distention to functional midgut malrotation, duodenal kinking, and SMA-like compression.
- Highlights how hindgut-driven pressure dynamics and dysbiosis can propagate proximally to cause duodenitis, pancreatitis, cholecystitis, cholelithiasis, and biliary gastritis.
- Demonstrates that foregut dysfunction arising from midgut compromise can lead to GERD, reflux oesophagitis, and laryngopharyngeal reflux, with downstream ENT and respiratory sequelae.
- Reframes colorectal motility disorders as global gut disorders with multisystem implications, supporting a shift toward holistic, axis-wide assessment and management.

Strengths and Limitations of This Study

- Proposes a novel, anatomically coherent "Ascending Cascade" framework linking hindgut partial obstruction to midgut, foregut, biliary–pancreatic, and airway manifestations.
- Integrates mechanical, vascular, microbial, and neuro-immune mechanisms into a unified explanatory model, offering a systems-level perspective rarely applied to colorectal motility disorders.
- Highlights under-recognised pathways such as mesocolonic traction at the duodenojejunal flexure and functional SMA-like duodenal compression, which may explain complex multisite symptom clusters.
- Synthesises evidence across paediatric and adult populations, supporting broad clinical relevance.
- Limited by reliance on heterogeneous literature and absence of large prospective or mechanistic studies directly validating the full cascade.
- Conceptual model requires empirical testing to determine causality, quantify risk, and evaluate whether early treatment of hindgut dysfunction prevents upstream complications.

Introduction

Colorectal motility disorders - including functional constipation, colonic inertia, segmental dysmotility, and Hirschsprung spectrum variants - are traditionally conceptualised as isolated hindgut conditions [1-4]. Yet patients frequently present with symptoms extending far beyond the distal colon, including small bowel dysmotility, biliary disease, duodenitis, pancreatitis, gastro-oesophageal reflux, and recurrent ENT or respiratory infections [5-11].

These associations are often dismissed as coincidental or attributed to functional overlay. However, emerging evidence suggests that chronic hindgut dysfunction generates intraluminal hypertension, segmental distention, and low-grade ischemia at antimesenteric and marginal arcade watershed zones [12-14], promoting dysbiosis, bacterial translocation, appendiceal stasis, ileocecal reflux, backwash ileitis, and mesenteric immune activation [15-21]. Proximal propagation of pressure and altered motility may contribute to small bowel bacterial overgrowth [8,15], ileal dysfunction, and duodenal or biliary complications [22-26].

Anatomical continuity between the mesocolon and the Duodenojejunal (DJ) flexure provides a plausible mechanical pathway for hindgut-derived tension to influence midgut configuration, potentially producing functional malrotation variants or SMA-like duodenal compression [22,27-31]. These midgut disturbances can impair gastric emptying and oesophageal barrier function, leading to GERD, reflux oesophagitis, and laryngopharyngeal reflux [5-7], with downstream respiratory and ENT sequelae [9-11].

This review proposes a unified "Ascending Cascade" model to explain how hindgut dysfunction can propagate upstream effects throughout the gastrointestinal tract and beyond. The model integrates mechanical, vascular, microbial, and neuro-immune mechanisms [32-34], reframing colorectal motility disorders as global gut disorders with multisystem implications.

Methods

Study Design

Narrative review following BMJ Open guidance for conceptual and mechanistic synthesis.

Search Strategy

Databases searched**:** PubMed, Embase, Scopus, Google Scholar.

Keywords included: colorectal motility disorders, hindgut obstruction, dysbiosis, SIBO, ileocecal reflux, duodenal compression, SMA syndrome, biliary stasis, GERD, laryngopharyngeal reflux, ENT infections.

Inclusion Criteria

- Human studies (paediatric and adult).

- Mechanistic, anatomical, or physiological analyses.
- Case reports/series describing upstream effects of hindgut dysfunction.
- Studies on duodenal compression, biliary disease, or reflux linked to distal obstruction

Exclusion Criteria

- Non-gastrointestinal primary disorders.
- Studies lacking mechanistic relevance.
- Animal studies unless directly applicable.

Synthesis Approach

Findings were grouped into four mechanistic domains:

1. Hindgut obstruction
2. Midgut consequences
3. Foregut dysfunction
4. Extra-intestinal manifestations

Results/Synthesis

Hindgut Partial Functional Obstruction: The Primary Driver

Mechanisms of Partial Obstruction: Partial functional obstruction may arise from: Colonic inertia or neuropathy, Pelvic floor dyssynergia, Transition zone dysmotility, Redundant or elongated sigmoid colon, Chronic fecal loading and megarectum, Subclinical Hirschsprung variants or hypoganglionosis.

Even without complete obstruction, impaired propulsion creates segmental stasis, intraluminal hypertension, and progressive bowel distention.

Hindgut Pathophysiology: Ischemia and Translocation

Partial functional obstruction leads to chronic bowel distention, specifically affecting the antimesenteric border. Because the large intestine relies on a circumferential blood supply from the marginal arcade (Drummond's), distention increases intramural pressure, compromising microvascular flow in "watershed" zones [1]. This localized ischemia weakens the mucosal barrier, facilitating:

- Bacterial Translocation: Pathogens bypass the compromised barrier, entering the portal and lymphatic systems.
- Dysbiosis: Luminal stasis alters the microbiome, favoring pro-inflammatory species.

Intraluminal Hypertension and Distention: Chronic stasis increases colonic pressure, causing segmental distention and impaired propulsion.

Vascular Watershed Vulnerability: The antimesenteric border and marginal artery arcade create linear "grey zones" prone to low-grade ischemia, mucosal injury, and bacterial translocation. Distention compromises perfusion at the antimesenteric border, where circumferential blood supply is weakest. The marginal artery arcade creates linear "grey zones" susceptible to: Low-grade ischemia, Mucosal barrier disruption, Translocation of luminal bacteria, Localised inflammation. These micro-ischemic events may be clinically silent yet pathophysiologically significant.

Stasis, Dysbiosis and Immune Activation: Stasis promotes dysbiosis, methane production, appendiceal stasis, ileocecal reflux, backwash ileitis, Peyer's patch activation, and mesenteric adenitis. Colonic stasis fosters: Dysbiosis with overgrowth of gas-producing and proteolytic species, Increased fermentation and methane production (slowing motility further), Mucosal immune activation, Appendiceal stasis → appendicitis, Ileocecal reflux → backwash ileitis. The ileocecal valve becomes incompetent under pressure, allowing retrograde flow of colonic contents into the terminal ileum, exposing Peyer's patches to abnormal antigenic loads and triggering mesenteric adenitis.

Midgut Consequences: The Secondary Cascade

Midgut Sequelae: Reflux and Inflammatory Responses

As distal pressure rises, the "back-pressure" effect breaches the ileocecal valve, leading to ileocecal reflux and backwash ileitis.

- SIBO and Adenitis: Stasis in the terminal ileum triggers Small Intestinal Bacterial Overgrowth (SIBO). This chronic antigenic stimulation involves Peyer's patches, frequently manifesting as mesenteric adenitis or secondary appendicitis.
- Anatomical Shifts: The mesocolonic attachment at the Duodenojejunal (DJ) flexure acts as a mechanical pivot. Chronic hindgut distention can exert "traction-like" effects:
    - Low DJ Flexure: May mimic variants of midgut malrotation or non-rotation.
    - High DJ Flexure: Increases the acute angle of the mesenteric root, leading to functional Superior Mesenteric Artery (SMA) Syndrome or partial obstruction of the duodenal C-loop leading to ischemia, peptic ulceration, diverticulum, bleeding, perforation, etc.

Luminal Back-Pressure and SIBO: Proximal pressure propagation contributes to small bowel bacterial overgrowth and ileal dysfunction. Sustained hindgut pressure transmits proximally, causing: Small Bowel Bacterial Overgrowth (SIBO), Ileal dysmotility, Functional partial obstruction, Distention-induced mucosal injury.

Mesocolonic Traction at the DJ Flexure; The mesocolon's continuity with the DJ flexure creates mechanical effects rarely considered clinically. The mesocolon's anatomical continuity with the DJ flexure creates a mechanical interface rarely considered in clinical practice.

Two key variants emerge:

Low-lying DJ flexure

- Increased mesocolonic drag
- Apparent or functional midgut malrotation or non-rotation variants
- Kinking or compression of the proximal jejunum

High insertion of the DJ flexure

- Compression of the duodenal C-loop between the SMA and aorta
- A functional mimic of Superior Mesenteric Artery (SMA) syndrome
- Low DJ insertion → functional malrotation/non-rotation
- High DJ insertion → SMA-like duodenal compression

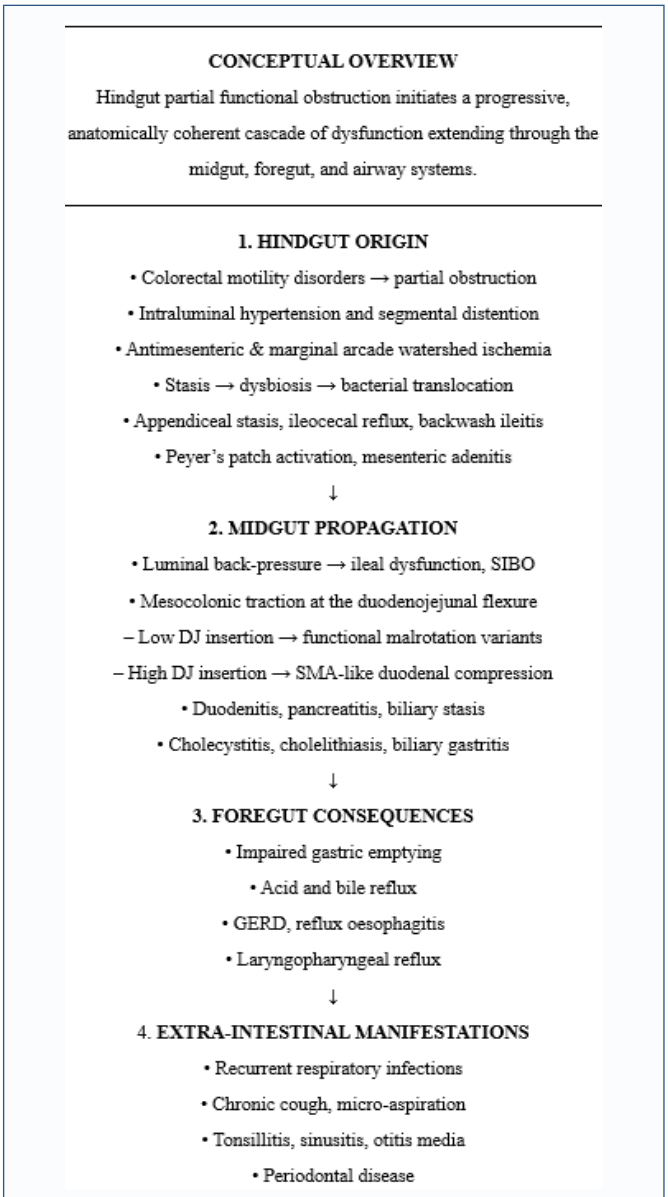

Figure 1: Conceptual graphical abstract illustrating the "Ascending Cascade" model of colorectal motility disorders. This schematic summarises the proposed pathophysiological continuum beginning with hindgut partial functional obstruction and progressing through midgut compromise, foregut dysfunction, and extra-intestinal manifestations. Key mechanisms include intraluminal hypertension, watershed ischemia, dysbiosis, ileocecal reflux, mesocolonic traction at the duodenojejunal flexure, SMA-like duodenal compression, biliary stasis, and reflux-mediated airway disease. This review reframes colorectal motility disorders as "global gut disorders" with upstream effects driven by mechanical, vascular, microbial, and neuro-immune pathways. The proposed "Ascending Cascade" model offers a unifying explanation for complex, multisite symptom clusters frequently seen but rarely connected in clinical practice.

Duodenal and Pancreatic Sequelae: Functional obstruction predisposes to duodenitis, pancreatitis, cholecystitis, cholelithiasis, and biliary gastritis. These midgut and biliary complications are often misattributed to primary foregut disease.

Midgut derivatives Hepatobiliary pancreatic involvement; Duodenal hypertension resulting from the obstructed C-loop compromises biliary and pancreatic drainage. This stasis is a precursor to duodenitis, pancreatitis, cholecystitis cholangitis, and cholelithiasis.

Foregut Dysfunction: The Tertiary Cascade

Gastric and Oesophageal Effects: Duodenal partial functional obstruction disrupts gastric emptying and lowers oesophageal sphincter tone, causing GERD, oesophagitis, and laryngopharyngeal reflux. 4.1 Gastric and Oesophageal Effects.

Duodenal obstruction and biliary reflux disrupt gastric emptying and lower oesophageal sphincter tone, leading to:

- Gastro-oesophageal Reflux Disease (GERD)
- Reflux oesophagitis
- Laryngopharyngeal reflux

Tertiary Foregut Involvement

Biliary Gastritis: Duodenogastric reflux of bile salts irritates the gastric mucosa, contributing to Gastroesophageal Reflux Disease (GERD) and reflux esophagitis.

Airway and ENT Manifestations- Foregut Derivatives- Quaternary Extra-Digestive Effects

The "Ascending Cascade" concludes with extra-esophageal manifestations driven by micro-aspiration and systemic inflammation. Chronic refluxate exposure contributes to:

- Respiratory & ENT: Recurrent upper/lower respiratory infections, chronic cough, chronic tonsillitis, sinusitis, and ENT complications (otitis media) are often linked to nocturnal reflux of gut-derived pathogens and acid.
- Periodontal Health: Chronic exposure to gastric contents and altered oral microbiome leads to persistent periodontal disease.

These extra-intestinal manifestations are rarely linked back to hindgut pathology, yet the mechanistic chain is anatomically and physiologically coherent 4. Extra-Intestinal Manifestations at the top end of the gut.

Airway and ENT Sequelae: Chronic refluxate exposure contributes to recurrent respiratory infections, chronic cough, tonsillitis, sinusitis, otitis media, and periodontal disease.

Extra-Intestinal Manifestation at Hindgut Derivatives- Proctodeum and Cloaca

Perianal pathology-fissure, fistula, abscess, external piles, etc.

Genital tract effects-vulvovaginitis, synechia vulva, vaginitis, pelvic inflammatory disease.

Urinary tract involvement - Balanitis, balanoposthitis, idiopathic scortal edema, Fournier's gangrene, Necrotising fasciitis, urethritis, cystitis, pyelonephritis.

Discussion

This narrative review introduces a unified "Ascending Cascade" model linking hindgut partial functional obstruction to a progressive sequence of midgut, foregut, and extra-intestinal dysfunction. Existing literature has largely treated colorectal motility disorders as regionally confined conditions [1-4], with limited attention to their upstream consequences. Our synthesis demonstrates that intraluminal hypertension, segmental distention, and watershed ischemia [12-14] create a permissive environment for dysbiosis,

bacterial translocation, ileocecal reflux, and immune activation [15-21], all of which may propagate dysfunction proximally.

The model aligns with established evidence on SIBO and ileal dysmotility [8, 15], but extends current understanding by highlighting the role of mesocolonic traction at the DJ flexure as a mechanical transducer of hindgut pressure. While malrotation and SMA syndrome are traditionally viewed as congenital or anatomical disorders [22, 27-31], our framework suggests that functional or acquired variants may arise in the context of chronic hindgut distention.

Midgut compromise - manifesting as duodenitis, pancreatitis, biliary stasis, cholecystitis, or cholelithiasis - has typically been attributed to primary biliary or pancreatic pathology [23-26]. By contrast, this review positions these conditions as potential secondary phenomena within a hindgut-driven cascade. Similarly, foregut disorders such as GERD, reflux oesophagitis, and laryngopharyngeal reflux [5-7] may represent tertiary manifestations of upstream mechanical and microbial disturbances rather than isolated foregut disease.

The downstream respiratory and ENT sequelae associated with reflux - chronic cough, sinusitis, otitis media, tonsillitis, and recurrent respiratory infections - are well documented [9-11], yet rarely linked to distal gut pathology. Integrating these observations within a single mechanistic continuum offers a more coherent explanation for complex multisite symptom clusters frequently encountered in clinical practice.

Finally, the model is consistent with broader systems-level frameworks describing gut-brain-immune interactions [32-34], reinforcing the concept that regional motility disorders can exert global physiological effects. While the evidence base remains heterogeneous, the anatomical and mechanistic plausibility of the Ascending Cascade model warrants further empirical investigation through imaging studies, motility mapping, microbiome profiling, and interventional trials targeting hindgut dysfunction.

By reframing colorectal motility disorders as global gut disorders, this review offers a clinically meaningful perspective that may reduce diagnostic fragmentation, improve recognition of multisite symptom clusters, and support earlier, holistic intervention.

This review proposes a unified "Ascending Cascade" model linking hindgut obstruction to multisystem dysfunction. The model integrates mechanical, vascular, microbial, and neuro-immune pathways, offering a systems-level perspective on conditions traditionally viewed as isolated to the distal colon.

Clinically, this model may explain complex symptom clusters, reduce diagnostic fragmentation, and support earlier intervention. It also highlights the need for imaging and assessment across the entire gut axis, not solely the symptomatic region.

Future research should include prospective studies, imaging correlates of mesocolonic traction, microbiome profiling, and interventional trials targeting hindgut motility.

This narrative review introduces a novel, anatomically coherent framework that reconceptualises colorectal motility disorders as the initiating event in a progressive, system-wide cascade of gastrointestinal and extra-intestinal dysfunction. While hindgut obstruction is well recognised, its upstream consequences - spanning the midgut, foregut, biliary - pancreatic axis, and even the respiratory and ENT systems - remain under-appreciated and poorly integrated in current clinical practice.

Our proposed "Ascending Cascade" model synthesises mechanical, vascular, microbial, and neuro-immune mechanisms into a unified explanatory pathway. The review highlights several under-recognised phenomena, including:

- Watershed ischemia at antimesenteric and marginal arcade zones
- Ileocecal reflux, backwash ileitis, and Peyer's patch activation
- Mesocolonic traction at the duodenojejunal flexure producing functional malrotation variants
- SMA-like duodenal compression arising from high DJ insertion
- Secondary duodenitis, pancreatitis, cholecystitis, and biliary gastritis
- Tertiary GERD, laryngopharyngeal reflux, and recurrent respiratory/ENT infections

Principal Findings

- This narrative review identifies hindgut partial functional obstruction - arising from colorectal motility disorders - as the initiating event in a progressive, anatomically coherent cascade of gastrointestinal dysfunction extending proximally through the midgut and foregut and ultimately into the airway system.
- Hindgut obstruction generates intraluminal hypertension, segmental distention, and watershed ischemia at antimesenteric and marginal arcade zones. These changes promote dysbiosis, bacterial translocation, appendiceal stasis, ileocecal reflux, backwash ileitis, and mesenteric immune activation.
- Proximal propagation of pressure and stasis contributes to small bowel bacterial overgrowth, ileal dysmotility, and immune stimulation. Anatomical continuity of the mesocolon with the duodenojejunal flexure creates mechanical transmission of tension, producing functional malrotation variants or SMA-like duodenal compression depending on DJ flexure position.
- Midgut compromise leads to duodenitis, pancreatitis, biliary stasis, cholecystitis, cholelithiasis, and biliary gastritis, establishing a mechanistic bridge between hindgut dysfunction and foregut pathology.
- Foregut consequences include impaired gastric emptying, acid and bile reflux, GERD, reflux oesophagitis, and laryngopharyngeal reflux, which in turn contribute to recurrent respiratory infections, chronic cough, tonsillitis, sinusitis, otitis media, and periodontal disease.
- Collectively, these findings support a unified "Ascending Cascade" model in which colorectal motility disorders function as global gut disorders with multisystem implications rather than isolated hindgut conditions.

Comparison with Existing Literature

Existing literature on colorectal motility disorders largely treats

them as segmental or regionally confined conditions, focusing on stool frequency, transit time, and local structural or neuromuscular abnormalities. Most studies describe downstream consequences such as faecal loading, megarectum, or megacolon, but rarely extend their analysis to systematic upstream effects on the midgut, foregut, or extra-intestinal systems.

Work on dysbiosis, Small bowel Bacterial Overgrowth (SIBO), and ileocecal reflux has shown that chronic stasis and altered motility can reshape microbial communities and promote mucosal immune activation, yet these phenomena are typically discussed in isolation rather than as components of a continuous, pressure-driven axis originating in the hindgut. Similarly, reports of backwash ileitis, mesenteric adenitis, and Peyer's patch activation are often framed within inflammatory bowel disease or infectious contexts, rather than as potential sequelae of functional colorectal obstruction.

The literature on duodenal compression syndromes, including Superior Mesenteric Artery (SMA) syndrome, and on midgut malrotation variants has traditionally emphasised congenital or anatomical predisposition. The potential contribution of mesocolonic traction and hindgut distention to functional or acquired variants of these conditions is rarely explored. Our model extends this body of work by proposing that mesenteric attachments and DJ flexure position can act as mechanical transducers of hindgut pressure and tension.

Similarly, biliary and pancreatic complications - such as cholecystitis, cholelithiasis, biliary gastritis, and pancreatitis - are usually attributed to primary biliary pathology, metabolic risk factors, or sphincter of Oddi dysfunction. The possibility that these may, in some patients, represent secondary phenomena within a hindgut-driven cascade is not systematically addressed in existing reviews.

The association between Gastro-oesophageal Reflux Disease (GERD), laryngopharyngeal reflux, and recurrent ENT or respiratory infections is well documented, but current literature typically positions reflux as a primary foregut disorder. Our model reframes reflux, in selected patients, as a tertiary manifestation of upstream midgut and hindgut dysfunction, thereby linking established gut-airway interactions to a more distal origin.

In contrast to the predominantly compartmentalised approach in existing literature, this review offers a systems-level synthesis that integrates mechanical, vascular, microbial, and neuro-immune mechanisms into a single "Ascending Cascade" framework. Rather than contradicting prior work, it re-orders and connects disparate observations - colonic stasis, dysbiosis, SIBO, duodenal compression, biliary disease, and reflux-related airway pathology - into a coherent, anatomically grounded continuum. This integrative perspective is largely absent from current reviews and represents the principal conceptual advance of this work.

Integrating the Ascending Cascade Model

A Systems-Level Perspective

The gastrointestinal tract is not a series of isolated compartments but a continuous, pressure-sensitive, microbially dense, neuro-immune organ. Hindgut dysfunction can propagate upstream through:

- Mechanical pathways (pressure, traction, obstruction)
- Vascular pathways (watershed ischemia, mucosal injury)
- Microbial pathways (dysbiosis, translocation, SIBO)
- Neuro-immune pathways (*viscero-visceral* reflexes, cytokine signalling)
- Biliary-pancreatic pathways (duodenal obstruction, reflux)

Clinical Implications

Recognising this cascade may:

- Prevent misdiagnosis of foregut symptoms as isolated disorders
- Reduce unnecessary investigations
- Support earlier intervention in hindgut motility disorders
- Encourage multidisciplinary management
- Highlight the need for imaging of the entire gut axis, not just the symptomatic region

Future Directions

- Prospective studies mapping symptom progression from hindgut to foregut
- Imaging correlates of mesocolonic traction and DJ flexure variants
- Microbiome profiling across the cascade
- Biomarkers of low-grade ischemia in colonic watershed zones
- Interventional trials targeting hindgut motility to prevent upstream complications

Summary

This narrative review proposes a unified "Ascending Cascade" model in which colorectal motility disorders - traditionally viewed as isolated hindgut conditions - initiate a progressive, anatomically coherent sequence of dysfunction extending through the midgut, foregut, and airway systems. Hindgut partial functional obstruction generates intraluminal hypertension, segmental distention, and watershed ischemia, promoting dysbiosis, bacterial translocation, ileocecal reflux, and immune activation. Proximal propagation of pressure and mesocolonic traction at the duodenojejunal flexure contribute to small bowel bacterial overgrowth, ileal dysmotility, functional malrotation variants, and SMA-like duodenal compression, leading to duodenitis, pancreatitis, and biliary stasis. These midgut disturbances impair gastric emptying and oesophageal barrier function, resulting in GERD, reflux oesophagitis, and laryngopharyngeal reflux, with downstream respiratory and ENT sequelae. Collectively, the model reframes colorectal motility disorders as global gut disorders with multisystem implications, highlighting the need for holistic assessment and early intervention.

Conclusion

Colorectal motility disorders may represent the first step in a broader, ascending cascade of gastrointestinal and systemic dysfunction. Hindgut partial obstruction initiates a chain reaction involving vascular compromise, dysbiosis, immune activation, midgut mechanical distortion, biliary and pancreatic involvement, and ultimately foregut and airway disease. Recognising this continuum reframes colorectal motility disorders as global gut disorders with multisystem implications. A holistic, anatomically grounded approach may improve outcomes and reduce diagnostic

fragmentation. Recognizing colorectal motility disorders as global gut pathologies is essential. Management should transition from local symptomatic relief to addressing the entire "Ascending Cascade," acknowledging that foregut and ENT symptoms may be rooted in hindgut dysfunction.

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