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Irritable Bowel Syndrome or Just Inefficient Bowel-Emptying Syndrome - Explaining the Overlap of Gastro-Oesophageal Reflux Disease with Functional Gastrointestinal Disorders through the Effects of Colonic Distension

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Abstract

Colonic distension is not only the main trigger of functional colonic pain, but also activates multiple different reflexes affecting both upper and lower gastrointestinal (GI) motility. Irritable bowel syndrome (IBS) patients have been documented with increased colonic faecal loading on abdominal x-ray. Medications, reduced mobility and probably hereditary factors which promote colonic stasis along with poorly digested fermentable oligosaccharides, disaccharides, monosaccharides and polyols (FODMAPs) which increase colonic distension are known to trigger colonic symptoms. Colonic decompression has been shown to improve both functional colonic and upper gut symptoms as well as reducing typical gastro-oesophageal reflux disease (GORD) and laryngopharyngeal reflux (LPR) symptoms. The right colon appears to play an important role in regulating whole gut motility. GI smooth muscle functions according to the Frank-Starling principle where progressive distension initially promotes and later impairs motility and must be taken into account when selecting appropriate medical therapy. Colonic stasis is associated with the more benign form of acute ischaemic colitis. In mouse studies, colonic distension from a high FODMAP diet may well induce low-grade mucosal ischaemia, barrier dysfunction and ongoing low-grade inflammation causing IBS symptoms. Gut physiology suggests that occult constipation or proximal colonic faecal loading acts as a functional obstruction, which must be addressed by medical therapy to treat functional upper GI symptoms and refractory reflux optimally. Improvement in functional colonic symptoms with simple laxative therapy and other agents which promote colonic motility such as prucalopride correlates strongly with improvement in dyspeptic, refractory GORD and LPR symptoms.

Keywords: Gastro-oesophageal reflux disease • Functional dyspepsia • Irritable bowel syndrome • Ischaemia Functional bowel disorder

Introduction

Functional gastrointestinal disorders (FGID) are known to overlap with gastro-oesophageal reflux disease (GORD) without a definite pathophysiological link being identified. Multiple studies have assessed the effect of colonic distension on both gastrointestinal (GI) motility and symptoms using barostat balloons. Increasing luminal distension may initiate many functional gut symptoms or alter GI motility, which rapidly subside or reverse with decompression. Many studies suggest that colonic stasis or occult constipation is a common finding in patients with functional GI disorders. Colonic stasis may also play a role in organic gut disease such as ischaemic colitis and some GI cancers [1]. Fermentable oligosaccharides, disaccharides, monosaccharides and polyols (FODMAPs) are believed to cause gut symptoms by stimulating colonic mechanoreceptors in response to luminal distension. This occurs mainly through their osmotic effect increasing small intestinal water and the generation of gas from bacterial fermentation in the colon1.

Manipulating the intake of dietary FODMAPs thus provides an easy way to assess the clinical effects of colonic distension although other metabolically active substances such as short chain fatty acids (SCFAs) and changes in the gut microbiome may also need to be taken into account. Treatment with laxative therapy provides a simple safe alternative to reduce faecal loading and distension effectively and has been shown to improve functional GI symptoms in several small studies. The aim of this

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paper is to highlight the clinical and physiological evidence supporting the important role of colonic distension in GORD and FGID [2].

Materials and methods

Colonic Distension Thresholds

Both patients with IBS and other functional upper GI disorders (FUGID) are known to have enhanced visceral sensitivity to luminal distension causing discomfort**2-4**. IBS patients also have an enhanced colonic motility response to luminal distension compared with controls, but both the pain and motility reflexes are independent of each other and reversible with colonic decompression**2**. We know that FODMAPs also trigger colonic symptoms with colonic distension being the likely driver of this effect. This explains the rational of adopting a low FODMAP diet in irritable bowel syndrome (IBS) to help relieve symptoms**5** and may be one of the few IBS treatments which truly addresses the pathophysiology causing symptoms. However, colonic distension must be adequately reduced to effectively treat colonic symptoms. If the colon remains faecally loaded despite dietary changes then the treatment response is suboptimal [3].

Although IBS patients are generally more susceptible to colonic pain and hyper-contractility in response to distension than controls, there is still a wide range of distension thresholds across the group as a whole**2**. In other words, normal colonic transit with a low distension threshold may cause similar symptoms to slower colonic transit with a fuller colon and a higher distension threshold. Rectal hypersensitivity to distension is enhanced in females and decreases with age**6**. This explains why IBS patients almost invariably obtain relief of their abdominal discomfort with the bowel prep prior to colonoscopy albeit temporary for many. However, colorectal distension doesn't just trigger colonic pain and motility, but has also been shown in physiological studies to slow small intestinal transit**7**, delay gastric emptying**7**, **8**, impair gastric accommodation**9**, **10**, reduce oesophageal motility**11** and increase transient lower oesophageal sphincter relaxations (TLOSRs) and reflux events**12**. This is highly relevant to treating FUGID as well as refractory GORD and LPR, all of which are frequently associated with upper GI dysmotility. Not only may consumption of dietary FODMAPs bring on functional colonic symptoms, but both infusion of FODMAPs into the right colon in normals and a high FODMAP diet in GORD patients have been shown to increase TLOSRs and reflux events**13**, **14**. Although SCFAs generated from fermentation of FODMAPs by colonic bacteria have been shown to increase reflux events**13**, it is very likely that colonic distension plays an important role in triggering reflux [4].

Raahave et al demonstrated that IBS patients have increased colonic faecal loading on abdominal x-ray (AXR) compared with controls and this correlates with slower colonic transit times (although still in the normal range) and increased colonic redundancies. In other words, they simply have longer slower fuller colons, which don't clear as effectively. Bloating was shown to correlate with proximal or total colonic faecal loading and lower abdominal pain with distal colonic faecal loading15, which typically settles after defaecation as the colon decompresses. Patients with functional colonic symptoms are often found to have longer redundant colons at colonoscopy. Our GI histories often focus more on stool frequency and form. Although stool form and frequency generally correlate with whole gut or colonic transit time**16**, 75% patients with proven slow transit constipation (STC) by scintigraphy in one study had stool frequency within the accepted range and passed normal to loose stools, but often with associated straining or a sensation of incomplete emptying**17**.

Colonic transit time correlates with IBS subtypes**18**, **19**, but not with symptoms such as bloating or abdominal pain**18**, which are more related to the degree and location of colonic distension**15** and the individual patient's distension threshold which triggers symptoms**2**. Hence objective evidence of both the extent and distribution of colonic faecal loading with at least a plain AXR is essential to manage any patient presenting with functional GI, refractory GORD or LPR symptoms. Patients more sensitive to rectal balloon distension have been found to have higher indigestion, reflux, abdominal pain, constipation and IBS scores, which highlights the multiple different reflexes triggered by colonic distension generating both upper and lower GI symptoms**6** and hence explains the overlap between the different FGID, GORD and LPR. Most people move their bowels in the first half of the day and then consume regular meals throughout the day making their colon fullest in the evening or overnight, which is often relevant to the timing of gut symptoms. Bloating, dyspepsia, nausea and refractory reflux symptoms are often worse later in the day or sometimes in the early hours of the morning, which typically correlates with the timing of maximal colonic distension when reflexes are more likely to be triggered [5].

Like IBS, FUGIDs have a complex pathophysiology with other central neural mechanisms and associations with anxiety/depressive disorders, but they all share a universal sensitivity to luminal distension. Patients with functional heartburn are known to have increased sensitivity to oesophageal balloon distension**20** and are significantly more symptomatic when they have gas in their reflux**21**. Epigastric pain is more than likely caused by distension of the upper gut with retained food, fluid and/or gas, which is often easily seen on a plain AXR. Proximal colonic faecal loading, often associated with increased amounts of retained small intestinal (SI) gas consistent with SI dysmotility, is a frequent finding in patients presenting with FD**22** (refer figure 1). Recurrent belching is often attributed to the habit of excess swallowing of air or aerophagy, but in many cases it appears to be more the result of upper GI gas trapping above a full colon.

Decompressing the proximal colon with a one-off dose of polyethylene glycol (PEG) often rapidly settles both dyspepsia and belching. Many patients with STC present with fairly regular bowel habit sometimes associated with bloating or abdominal discomfort**17**, but have concurrent dyspeptic, refractory reflux or LPR symptoms. In this situation AXRs usually reveal extensive colonic faecal loading throughout, which is also common in those presenting with functional diarrhoea (refer figure 2)**23**. If colonic distension can trigger multiple different GI motility reflexes associated with a variety of both upper and lower gut symptoms, then logic tells us that our treatment of FGID and reflux must address this.

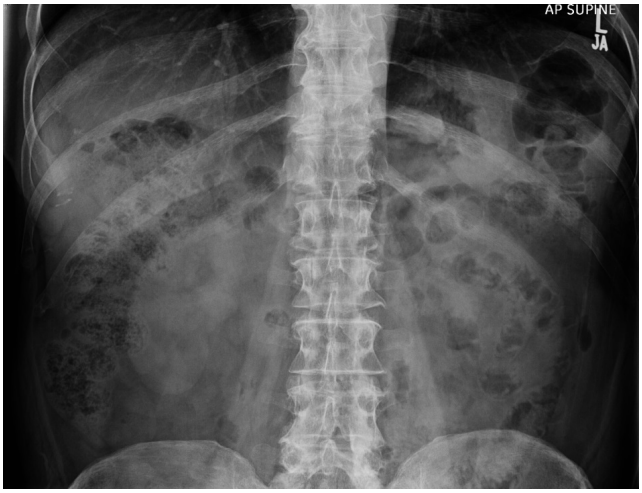

Figure 1: Patient with functional epigastric discomfort and excess belching, but normal bowel habit. Note proximal colonic faecal loading with prominent small intestinal gas trapping in the left upper quadrant.

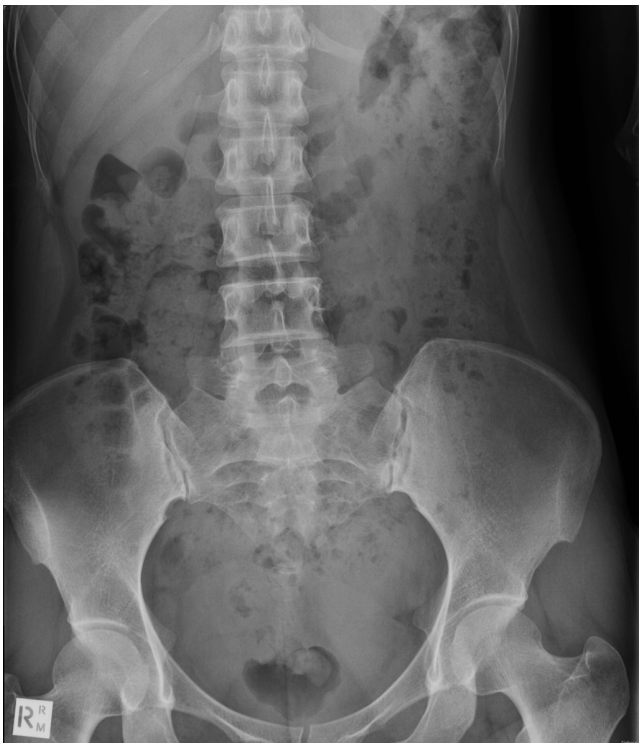

Figure 2: Patient with irritable bowel syndrome - diarrhoea. Note marked colonic faecal loading through to the rectum with no past history of constipation.

Results and discussion

Effect of Colonic Stasis

Most functional gut or reflux symptoms follow a fluctuating course, but colonic stasis appears to be a major trigger of symptoms. Studies have demonstrated that physical inactivity prolongs proximal colonic transit and moderate exercise accelerates colonic transit without affecting oro-caecal transit times24-26. Hence reduced activity contributes to colonic stasis and distension, which may then trigger colonic reflexes and gut symptoms. Taking an accurate medical history commonly reveals that any event or medication which promotes colonic stasis such as narcotic analgesia, anti-cholinergic medications, female hormones including progesterone and relaxin at the end of the menstrual cycle or in late pregnancy and prolonged physical stasis eg. hospital admissions or long-haul flights may cause a flare up of functional gut or reflux symptoms. It is likely that the increase in FGID in females occurring after menarche is related to the effect on gut motility of female hormones in the menstrual cycle. This is highly relevant to our current situation where recent lockdowns with Covid-19 have resulted in reduced physical activity or exercise. As a result, many patients have presented with new onset of gut symptoms even in those working from home without other significant stresses.

Clinical Response to Colonic Decompression

Multiple small clinical studies have confirmed that treating constipation (often occult and only confirmed on AXR) helps to resolve refractory GORD and recurrent vomiting**27**, functional dyspepsia (FD)**28**, functional abdominal pain (FAP)**29** and IBS symptoms**15**. Like Raahave, our recent prospective observational study also confirmed significant colonic faecal loading on AXR in most patients presenting with functional colonic symptoms with a high incidence of LPR symptoms affecting around half this group. More than 95% of patients with a combination of IBS and LPR symptoms had evidence of airway reflux on reflux scintigraphy. Effective treatment of functional colonic symptoms mainly with osmotic laxatives and occasional prucalopride correlated strongly ($p<0.005$) with improvement in dyspeptic and LPR symptoms at the other end of the gastrointestinal tract and resolved refractory reflux symptoms in all patients**30**. The key principle documented by this study is that more effective colonic clearance helps to improve all upstream symptoms extending right up to the airway including some patients whose rhinosinusitis resolved. Our other unpublished data with larger numbers confirms that refractory GORD rarely occurs if the colon is emptying effectively and that LPR without typical GORD symptoms generally cannot be treated effectively unless the colon (in particular the proximal colon) is emptying more efficiently.

Although it has been suggested that patients with STC suffer from a more generalised gut neuropathy with impaired whole gut motility**31**, subtotal colectomy in these patients has been shown to improve upper GI dyspeptic symptoms fairly promptly after surgery as well as improving gastric emptying in many but not all cases when reassessed a year later rather than immediately after surgery**32**, **33**. STC has been linked with impaired oesophageal, gastric, small intestinal and anorectal motility as well as delayed gallbladder emptying**34** consistent with a more generalised GI smooth muscle disorder, but the above studies suggest that even chronic smooth muscle dysfunction is potentially reversible with time if the colo-gastric brake is released and the functional obstruction treated before the affected smooth muscle becomes atonic. However, excessive distension of any smooth muscle organ may potentially cause irreversible dysfunction as we commonly see with distended atonic bladders requiring long-term catheters. Similar proximal colonic dilatation with associated smooth muscle hypertrophy has also been found in colonic specimens excised due to chronic colonic pseudo-obstruction**35**.

The potential reversibility of colonic distension motility reflexes can be easily assessed clinically by evaluating improvement in gut or reflux symptoms following bowel prep, which is often required as part of a patient's workup prior to colonoscopy. In addition, a one-off dose of 1 litre of PEG/sodium ascorbate solution has been shown to differentiate those with slow colonic transit with good specificity36. A longer response time > 3.8 hours indicates STC in 95% of cases. These patients also develop more prolonged diarrhoea following a one-off dose and are generally the ones with poor bowel preps at colonoscopy unless the situation is recognised beforehand. A recent colonoscopy performance measure study reported that symptomatic patients with IBS or bleeding have significantly worse bowel preps than those undergoing primary screening colonoscopies37, which may impact on adenoma detection rates (ADR)38. Many of the recognised risk factors for suboptimal bowel preps are linked with colonic stasis including constipation itself, inpatient bowel preps, diabetic, dementia and stroke patients and use of narcotic and anticholinergic medications such as tricyclic antidepressants39, 40. It appears very logical that the fuller the colon is, the harder it is to evacuate. As IBS patients have been documented with higher colonic faecal loading scores and slower colonic transit than controls15, they are also more prone to suboptimal bowel preps. Past studies have shown that colonic transit times > 30 hours predict poorer bowel prep41. As normal colonic transit times range from 20-72 hours, clearly the colon doesn't need to be excessively slow to contribute to poorer colonic clearance and predispose patients to functional colonic symptoms. With effective use of prior laxative therapy, good colonoscopic technique and adequate withdrawal times adenoma detection rates of 70% are then achievable (personal data), which translates to a significant reduction in the incidence of interval colorectal cancer.

Conclusion

In summary, reducing colonic distension below an individual's symptom threshold effectively reverses the physiological reflexes which influence gastrointestinal motility and reduces functional colonic, upper gut, refractory GORD and LPR symptoms. According to physiological principles, reducing colonic +/- secondary upper gut distension helps to preserve GI motility and function long-term. Preventing both SIBO and low-grade ischaemia should help to prevent the development of increased intestinal permeability, immune cell activation, low-grade gut inflammation, changes in microbiome and sensitivity to luminal distension as seen in FGID. Correlating GI symptoms with AXRs used to assess colonic faecal loading and gas retention throughout the GI tract is invaluable in planning medical therapy. Appropriate use of both high and low dose osmotic laxative therapy helps to evaluate colonic motility clinically and guides long-term therapy of FGID, GORD and LPR. If started prior to colonoscopy, it may also improve both the quality and tolerability of bowel prep with associated improvement in ADR and reduced incidence of interval colorectal cancer. There are many misconceptions about using long term laxatives, but they appear to be very safe and may help to prevent irreversible changes in gut motility.

In conclusion, there is abundant physiological and ample clinical evidence mainly from observational rather than RCTs to explain the link between LPR, GORD, FUGID and IBS. To control any gut disease the underlying pathophysiology causing gut symptoms must be addressed to remedy the problem. Although the physiological and experimental evidence has already been reported, the appropriate clinical RCTs just simply haven't been done yet. Our current approach is equivalent to treating peptic ulcers with acid suppression alone without eradicating Helicobacter. We all know from past experience that this approach is a quick fix and doesn't reverse the underlying pathophysiology. It's therefore no surprise that our treatments for FGID and GORD to date have been suboptimal.

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