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Editorial

Body Mass Index and Gastrointestinal Symptomatology: Possible Pathophysiology

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Overweight and obesity represent a major healthcare problem worldwide and has increased rapidly in recent decades. Obesity has been considered as a risk factor for many gastrointestinal disorders. Regular weight monitoring has become a key element in all populations. Body Mass Index (BMI) is a clinical screening tool that estimates body weight in relation to height and is commonly used as an indirect measure to assess the amount of body fat. It is the most widely used parameter to judge whether a person has a healthy weight or, alternatively, to classify the degree of overweight or obesity. Currently, there are few studies that explore in detail how BMI may alter the dynamics and physiology of the digestive tract. It is well known that the gastrointestinal (GI) tract plays key roles in food breakdown, nutrients absorption and waste discharge. However, changes in body weight, specifically weight excess, can negatively affect GI motility, alter digestive hormone secretion and increase intra-abdominal pressure. Gastrointestinal hormones that have been demonstrated to be affected by overweight are ghrelin and leptin. These alterations may contribute to the development of symptoms such as Gastroesophageal Reflux Disease (GERD), abdominal distention, functional dyspepsia and constipation. In this editorial, we explore how BMI affects physiology of the GI tract and discuss the possible association between development and severity of gastrointestinal symptoms. Special attention is paid on possible pathophysiology [1].

The association between obesity and specific gastrointestinal symptoms has been demonstrated by clinical investigations. A meta-analysis performed in 2012, concluded that obesity is significantly associated with symptoms such as diarrhea, vomiting, and GERD [2]. More recent evidence strengthens these findings. Increased odds ratio for upper abdominal pain, GERD and incomplete excretion, among obese individuals, has recently been reported [3]. Stool structure and its correlation with BMI has also been studied. Watery stools are more frequently reported among individuals with a higher BMI. While a lower BMI is associated with more formed stools; which points-out towards constipation [4].

Obesity is now recognized as a systemic metabolic condition and as a modulator of intestinal microbiota and immune homeostasis. Actually, obesity-induced gut dysbiosis has recently been described [5]. The imbalance in gut microbiota is characterized by an altered microbial diversity and short chain fatty acid (SCFA) production [6]. Therefore, dysbiosis may lead to an intestinal barrier disruption. This phenomenon triggers a low-grade inflammation and develops an environment prone to chronic GI diseases. On the other hand, reduced levels of useful SCFAs, such as butyrate and propionate, have been associated with an impaired control of anti-inflammatory mediators. Some of these mediators involve Forkhead Box P3 (Foxp3) and IL-6, IL-1β and TNF-α. These mechanisms can modify epithelial tight junctions which increase intestinal permeability and favors bacterial translocation. All these phenomena further perpetuate GI inflammation. Moreover, in visceral adiposity, especially within the mesenteric region, the fat works as an active endocrine organ that contributes to systemic and local inflammation. Leptin, resistin and other proinflammatory adipokines are responsible for this condition. This inflammatory state may affect all: GI motility, hormone signaling (e.g., ghrelin and leptin) and immune responses within the gut mucosa [5,6]. On the other hand, recent evidence shows that micronutrient synthesis is compromised in human beings with disrupted gut microbiota and higher BMI. Gut microbes such as Bacteroides and Bifidobacterium are involved in the production of essential vitamins like B12 and K. A lower prevalence of these bacteria may restrict their biosynthesis, further contributing to GI dysfunction. Additionally, nearly half of daily vitamin K requirement originates in microbes. This highlights the key role of gut microbiota in GI metabolism [5,6].

Scientific studies have revealed that there is a significant association between people with obesity and a higher prevalence of functional bowel disease and GI symptomatology. Actually, there is a proportional relationship between BMI and the severity of gastrointestinal symptoms. Individuals with overweight and obesity show greater abdominal discomfort, distention and

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diarrhea. Accordingly, bowel health significantly worsens as body weight increases. These observations are consistent with the hypothesis that obesity-related gastrointestinal dysfunction is mediated by low-grade inflammation and adipokine activity. The release of pro-tumoral factors and pro-inflammatory cytokines, from visceral fat, has been associated to reduced gastrointestinal motility. This phenomenon is probably due to increased abdominal pressure and altered neuromuscular coordination. Although certain patterns are regularly observed, the relationship between obesity and GI physiology disorders lacks consistency across populations. Although ongoing uncertainty is observed in the literature, variability is often attributed to symptom type, gender and other co-morbidities [7].

A higher BMI has also been associated with increased prevalence of functional GI disorders (FGIDs) such as; gastroesophageal reflux (GER), postprandial distress and "abdominal balloon" feeling, especially among women. From a physiological perspective, retrograde flow across the gastroesophageal junction is forced up in conditions of increased intra-abdominal pressure; that is obesity. This basic mechanism is responsible for the development of both GERD and Hiatal Hernia [8]. Furthermore, adipocyte-derived proinflammatory cytokines play a key role in low-grade chronic inflammation, which underlies both metabolic and GI pathophysiology. The imbalance in the leptin/adiponectin ratio, observed in obesity, weakens insulin sensitivity. It also triggers intestinal permeability, T-cell infiltration and immune dysregulation. All these features are commonly observed in Inflammatory Bowel Disease (IBD). In fact, obesity has been shown to worsen the course of IBD and is associated with poor clinical outcomes, particularly in Crohn's disease. Moreover, visceral adiposity has been shown to correlate with disease activity and increased surgical risk [9].

From a scientific point of view, body mass index should not be regarded just as a number, but as a marker of complex biological interplay. These include, but are not limited to: endocrine activity, microbial balance and immune system regulation. They all together help to understand GI symptomatology, in those with overweight or obesity; while showing opportunities for individualized therapeutic strategies. A healthy diet, physical activity, and targeted microbiota restoration through prebiotics, probiotics or even fecal microbiota transplantation are promising strategies. Future studies should focus on better understanding causality, optimizing timely interventions and identifying biomarkers that anticipate symptom development and therapeutic response in patients with increased BMI and gastrointestinal symptomatology.

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