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ORIGINAL ARTICLE

Case Control Study

Effects of body mass index on gastric motility: Comparing children with functional abdominal pain disorders and healthy controls

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Abstract

BACKGROUND

Overweight children exhibit a higher prevalence of functional gastrointestinal disorders compared with their normal-weight peers, yet the underlying reasons remain unclear. Gastrointestinal motility, a key pathophysiological factor in functional gastrointestinal disorders, may be influenced by body mass index (BMI).

AIM

To evaluate the impact of BMI on gastric motility parameters in children with functional abdominal pain disorders (FAPDs).

METHODS

We assessed gastric motility in 176 children with FAPDs (61.4% females, mean age 7.94 years, SD 1.96 years) and 63 healthy controls (57.1% females, mean age 9.17 years, SD 1.90 years) at the Gastroenterology Research Laboratory, University of Kelaniya, Sri Lanka. FAPDs were diagnosed and subtyped using the Rome IV criteria: Functional abdominal pain 97 patients; irritable bowel syndrome 39 patients, functional dyspepsia (FD) 25 patients; and abdominal migraine 15 patients. Gastric motility was measured using a validated ultrasound method. Weight and height were measured using sensitive standard scales.

RESULTS
The BMIs of children with FAPDs and controls were 15.04 and 15.46 kg/m², respectively (P = 0.33). Fasting antral area (FAA) and antral area at 1 min (AA1) and 15 min (AA15) were significantly greater in patients with FAPD with a higher BMI (2.71 cm², 12.57 cm², and 7.19 cm², respectively) compared with those with a lower BMI (2.12 cm², 10.68 cm², and 6.13 cm², respectively) (P < 0.01). BMI positively correlated with FAA and AA15 (r = 0.18 and r = 0.19, respectively) (P < 0.01) in those with FAPDs. In controls, only AA1 was greater in the higher BMI group (12.51 cm² *vs* 9.93 cm²) and had a positive correlation (r = 0.33) ($P \leq 0.01$). Subgroup analysis revealed that in patients with FD, BMI negatively correlated with gastric emptying rate (GER) (r = -0.59) and antral motility index (MI) (r = -0.49), while in functional abdominal pain, MI positively correlated (r = 0.25) with BMI ($P \leq 0.01$).

CONCLUSION
In children with FAPDs, higher BMI was associated with increased gastric antral distention during fasting and postprandial periods (as indicated by FAA, AA1, and AA15) but not with contractility and transit (MI, GER). However, in the FD subgroup, high BMI correlated with reduced GER and MI. This indicates the possible role of BMI in gastric hypomotility and the pathophysiology of FD. These findings underscore the importance of lifestyle and dietary interventions aimed at optimizing BMI in the management of FAPDs, particularly FD.

Key Words: Body mass index; Gastric motility; Functional abdominal pain disorders; Functional gastrointestinal disorders; Sri Lanka

Core Tip: This study investigated the relationship between body mass index (BMI) and gastric motility in children with functional abdominal pain disorders (FAPDs). When considering FAPDs in general, higher BMI is associated with increased gastric antral distention but does not affect gastric contractility or transit. However, in the subgroup of children with functional dyspepsia, a higher BMI correlates with reduced gastric emptying rate and motility index, suggesting a role for BMI in gastric hypomotility. These findings offer new insights into how BMI influences gastrointestinal function in pediatric FAPDs, particularly in functional dyspepsia.

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INTRODUCTION

Both increased body weight (overweight and obesity) and functional abdominal pain disorders (FAPDs) are significant health problems among school-aged children worldwide, causing significant morbidity, poor quality of life, and high healthcare costs[1-3].

According to the World Health Organization (WHO), obesity is defined as body mass index (BMI) > 2 standard deviations (SD) above the WHO growth standard median and overweight as BMI > 1 SD above the WHO growth standard median[4]. It was reported that the overall prevalence of pediatric obesity, overweight, and excess weight is 8.5%, 14.8%, and 22.2%, respectively[3]. A higher prevalence of obesity among children and adolescents was reported in countries with Human Development Index scores of 0.8 or greater and high-income countries or regions compared with developing countries[3]. However, the prevalence of obesity and overweight is rapidly increasing in developing countries, too. The prevalence of obesity and overweight was reported to be 10.3% and 11.3%, respectively, among 5-18-year-olds in urban Sri Lanka[5].

Functional gastrointestinal disorders (FGIDs) are defined as chronic or recurrent gastrointestinal symptoms not explained by structural or biochemical abnormalities and are categorized into subtypes using the Rome IV criteria[6]. Irritable bowel syndrome (IBS), functional dyspepsia (FD), functional abdominal pain (FAP), and abdominal migraine (AM) are among the common FGIDs in children[7]. The global prevalence of FAPDs is estimated to be 13.5%[1]. In Sri Lanka, the prevalence of FAPDs is 12.5%[8].

The pathophysiology of FGIDs is a grey area in gastroenterology. Even with modern technologies, the pathophysiological mechanisms that explain the symptoms of FGIDs are poorly understood. The main pathophysiological factors related to FGIDs are visceral hypersensitivity, altered motility, increased mucosal permeability, immunological dysfunction, and psychosocial factors[9,10]. Gastric dysmotility is a known pathophysiological mechanism involved in the development of FAPDs[11-17]. Different gastrointestinal motility abnormalities have been observed in patients with FAPDs[18-20]. Delayed gastric emptying[18,21,22], the impaired initial distribution of a meal[15], impaired accommodation to a meal[23], and antral hypomotility[13,24] are among them. It is also noted that abnormal gastrointestinal contractions coincide with episodes of abdominal pain[25]. Furthermore, some studies have shown that gastric emptying rate (GER) had a negative correlation with scores obtained for the severity of symptoms[18].

Previous studies have shown a relationship between increased BMI and FGIDs. In one study, 50% of children who are overweight had at least one FGID[26]. Furthermore, obesity is reported to be associated with multiple FGIDs[27]. In addition, a long-term follow-up study conducted on children with FGIDs has shown that obesity is associated with poor outcomes and disability[28].

Several studies have demonstrated the complex indirect relationship between gastrointestinal motility and BMI abnormalities. Some peptides and hormones secreted in response to overeating, gastric distension, and high-body fat content (*e.g.*, leptin, insulin-like growth factor 1, peptide YY, cholecystokinin, glucagon-like peptide 1, glucose-dependent insulinotropic peptide, oxyntomodulin) are shown to reduce gastrointestinal motility. In addition, frequent food intake is associated with a reduction in gastrointestinal motility-inducing hormones secreted during the fasting period (*e.g.*, ghrelin, motilin), and this may contribute to reduced gastrointestinal motility[29-32].

A poorly understood pathophysiology is the main barrier to the management of FGIDs[33]. With increased urbanization, changes in dietary habits, and sedentary lifestyles, childhood obesity and overweight are becoming a global burden in both developed and developing countries around the globe. The main objective of this study was to assess the effects of BMI on gastric motility parameters in children with FAPDs.

MATERIALS AND METHODS

Study design

This was a comparative, cross-sectional study.

Recruitment of the patients

Children aged 5 to 12 years with FAPDs who met the inclusion criteria were consecutively recruited from the pediatric clinics at the North Colombo Teaching Hospital in Ragama, Sri Lanka. They were then assessed at the Gastroenterology Research Laboratory, Faculty of Medicine, University of Kelaniya, Sri Lanka. A comprehensive history was obtained from each child and their parents, after securing written informed consent. The diagnosis of FAPDs was made according to the Rome IV criteria[6].

Inclusion criteria

(1) Fulfill Rome IV criteria for at least one FAPD; and (2) Abdominal pain is severe enough to interrupt the activities of the child (*e.g.*, sleep, play, schooling, *etc.*).

Exclusion criteria

(1) Clinical or laboratory evidence suggesting organic pathology; (2) Chronic medical or surgical disease other than FAPDs; (3) Long-term medication for any illness other than FAPDs; and (4) Previous abdominal surgery.

All patients underwent a thorough screening for organic diseases. It included a detailed medical history, comprehensive physical examination (including assessment of growth parameters), stool and urine microscopy, urine culture, complete blood count, C-reactive protein, liver and renal function tests, and an abdominal ultrasound scan. Additional investigations, including upper and lower gastrointestinal endoscopy, serum amylase levels, and X-rays of the kidneys, ureters, and bladder, were performed when deemed necessary by the consultant pediatrician. Screening for celiac disease was not conducted due to its exceptional rarity in Sri Lanka.

Recruitment of controls

An age-matched and sex-matched group of healthy children from the same geographical area was recruited as controls after obtaining parental consent. None of the controls exhibited any acute or chronic gastrointestinal symptoms, FGIDs, or signs of other acute or chronic diseases.

Data collection

Assessment of gastric motility: Gastric motility was evaluated between 8:30 am and 9:30 am under thermoneutral conditions (26 °C). Females who had reached menarche underwent laboratory testing during the proliferative phase of their menstrual cycle. Any medications with prokinetic, adrenergic, or cholinergic properties were discontinued for a period of at least five times the half-life of the medication. Participants were instructed to avoid caffeine, nicotine, and alcohol-containing beverages for at least 8 h before testing.

Gastric motility was assessed in all subjects after a 6-h fast, using a validated ultrasound method previously reported in the literature[34]. A high-resolution real-time scanner (Siemens ACUSON X300™) equipped with 1.8 MHz to 6.4 MHz curved linear transducers, which allowed for recording and playback, was used for all measurements. After consuming a standard liquid meal heated to approximately 40 °C (200 mL of chicken broth, 54.8 kJ, 0.38 g protein, 0.25 g fat, 2.3 g sugar per serving, Ajinomoto Co., Tokyo, Japan) within 2 min, the subjects were seated in a chair leaning slightly backwards for the ultrasound examination. To eliminate inter-observer variability, all ultrasound measurements were conducted by the same investigator.

The primary gastric motility parameters evaluated and calculated included fasting antral area (FAA), antral area at 1 minute (AA1) and 15 minutes (AA15) post-meal, frequency of antral contractions, amplitude of antral contraction (AAC), antral motility index (MI), and GER[34].

Anthropometric measurements: The height and weight were measured by a trained investigator on participants who wore minimal clothing and were barefoot. Body weight was measured to the nearest 0.01 kg using a highly sensitive digital weighing scale. Body height was determined to the nearest 1 mm using a standard stadiometer. BMI was calculated as body weight (kg)/body height (m^2).

Study participants were categorized into two groups according to BMI based on Sri Lankan normal values: Lower BMI (BMI < 15 kg/m^2), and Higher BMI (BMI ≥ 15 kg/m^2).

Statistical analysis

At 80% power (1 - β = 0.80) and a 5% significance level (α = 0.05), the minimum sample required for this case-control study was 78 per group. Using a case-to-control ratio of approximately 2.8: 1, 176 patients with FAPDs and 63 controls were recruited.

Statistical analyses were conducted using PSPP version 2.0.1 (Free Software Foundation, Inc., http://fsf.org/). Continuous variables were expressed as mean ± SD, while categorical variables were presented as proportions. Differences in continuous variables were assessed using the independent samples *t*-test. The Pearson correlation coefficient (*r*) was used to measure a linear correlation between two variables. A two-tailed *P* value of less than 0.05 was considered statistically significant.

Ethical considerations

This study protocol was approved by the Ethics Review Committee, Faculty of Medicine, University of Kelaniya, Sri Lanka. Written informed consent was obtained from a parent or a legal guardian of the children recruited.

RESULTS

One hundred seventy-six patients with FAPDs were recruited for this study [108 (61.4%) females, mean age 8.06 years (SD 1.99 years)]. There were 97 patients with FAP, 39 patients with IBS, 25 patients with FD, and 15 patients with AM. The mean BMI of the females was 15.00 (SD 3.10) kg/m^2, and for males was 15.10 (SD 2.50) kg/m^2. According to the WHO definition[4], only 21 (11.9%) children with FAPDs were overweight, and none fulfilled the criteria for obesity.

Sixty-three healthy controls were recruited [36 (57.1%) females, mean age 8.97 years (SD 1.90 years)]. In the control group, the BMI of the females was 16.19 (SD 3.40) kg/m^2, and for the males was 14.49 (SD 2.30) kg/m^2.

Gastric motility parameters in patients with FAPDs and controls according to nutritional status

There were 140 children with lower BMI (< 15 kg/m^2) and 99 with higher BMI (≥ 15 kg/m^2). Table 1 compares the gastric motility parameters between lower BMI and higher BMI in patients with FAPDs and controls. FAA, AA1, and AA15 were significantly larger in patients with higher BMI than in those with lower BMI. In the control group, only AA1 was significantly larger in children with a higher BMI.

Correlation between BMI and gastrointestinal motility parameters in patients with FAPDs and controls

Table 2 describes the correlation between different gastric motility parameters and BMI in patients with FAPDs and controls. The only gastric mobility parameters that correlated with BMI were FAA and AA1 in the FAPD group and AA1 in the controls.

Correlation between BMI and gastrointestinal motility parameters according to the FAPD subtype

When subgroup analysis was performed (Table 3), the patients with FD, FAA, and AA15 had significant positive correlations with BMI, while GER, AAC, and MI had significant negative correlations. In the FAP subgroup, AAC and MI had positive correlations with BMI. The AM subgroup had shown a significant positive correlation between AA1 and BMI. The IBS group had no significant correlation between BMI and motility parameters.

DISCUSSION

To our knowledge, this is the first study to explore the relationship between BMI and gastric motility in children with FAPDs. In our cohort with FAPDs, those with higher BMI (≥ 15 kg/m^2) exhibited significantly greater gastric distension during fasting (FAA) and postprandial periods (AA1 and AA15) compared to their peers with lower BMI (< 15 kg/m^2). However, other key motility parameters, such as GER and antral MI, did not differ significantly between BMI groups. When examining the correlation between BMI and gastric motility in children with FAPDs, we found a positive relationship with FAA and AA1 but not with GER and antral MI.

However, when analyzing different FAPD subtypes, a distinct pattern emerged. In children with FD, BMI negatively correlated with key motility parameters, including GER and antral MI, in addition to parameters indicating gastric distension. Conversely, in children with FAP, we observed a weak but positive correlation between BMI and antral MI.

It is suggested that a higher BMI can influence gastric motility, potentially leading to delayed gastric emptying and other motility disturbances[35]. In contrast to this, when analyzing FAPDs in general, we did not observe the differences

Table 1 Comparison of gastric motility parameters in children with functional abdominal pain disorders and controls according to lower and higher body mass index

Motility parameter	FAPDs			Controls		
	BMI < 15 kg/m² (*n* = 109)	BMI ≥ 15 kg/m² (*n* = 67)	*P* value[1]	BMI < 15 kg/m² (*n* = 31)	BMI ≥ 15 kg/m² (*n* = 32)	*P* value[1]
Fasting antral area (cm^2)	2.12	2.71	0.008	1.65	2.16	0.058
Antral area in 1 min (cm^2)	10.68	12.57	0.004	9.93	12.61	0.001
Antral area in 15 min (cm^2)	6.13	7.19	0.009	4.03	4.95	0.088
Gastric emptying rate (%)	43.01	40.26	0.240	59.64	59.37	0.947
Frequency of antral contraction (3 min)	8.52	8.48	0.809	9.61	9.33	0.152
Amplitude of antral contraction (%)	43.21	46.28	0.117	61.93	61.25	0.868
Antral motility index	3.68	3.98	0.125	5.83	5.78	0.903

[1]Independent samples *t*-test. BMI: Body mass index; FAPDs: Functional abdominal pain disorders.

Table 2 Correlation of body mass index and gastric motility parameters in children with functional abdominal pain disorders and controls

Motility parameter	FAPDs (*n* = 176)		Controls (*n* = 63)	
	r	*P* value	*r*	*P* value
Fasting antral area	0.183	0.015	0.234	0.065
Antral area at 1 min	0.143	0.059	0.332	0.008
Antral area at 15 min	0.190	0.011	0.227	0.074
Gastric emptying rate	-0.137	0.071	-0.049	0.704
Frequency of antral contractions	0.014	0.858	-0.208	0.103
Amplitude of antral contractions	0.130	0.086	-0.016	0.900
Antral motility index	0.132	0.081	-0.076	0.552

r: Pearson correlation coefficient; FAPDs: Functional abdominal pain disorders.

Table 3 Correlation of body mass index and gastric motility parameters according to the functional abdominal pain disorders subtypes

Motility parameter	IBS (*n* = 39)		FAP (*n* = 97)		FD (*n* = 25)		AM (*n* = 15)	
	r	*P* value	*r*	*P* value	*r*	*P* value	*r*	*P* value
Fasting antral area	0.177	0.280	0.011	0.915	0.446	0.025	0.513	0.050
Antral area at 1 min	0.136	0.410	0.062	0.545	0.304	0.140	0.564	0.028
Antral area at 15 min	0.115	0.486	0.083	0.420	0.642	0.001	0.351	0.199
Gastric emptying rate	0.027	0.873	-0.082	0.427	-0.592	0.002	0.065	0.819
Frequency of antral contractions	0.137	0.404	0.040	0.698	-0.284	0.170	-0.172	0.539
Amplitude of antral contractions	0.144	0.381	0.263	0.009	-0.510	0.009	0.160	0.568
Antral motility index	0.204	0.213	0.254	0.012	-0.489	0.013	0.003	0.992

IBS: Irritable bowel syndrome; FAP: Functional abdominal pain not otherwise specified; FD: Functional dyspepsia; AM: Abdominal migraine; *r*: Pearson correlation coefficient.

in gastric emptying and antral MI with high BMI. However, in the patients with FAPDs, the gastric motility parameters indicating increased gastric antral distension were significantly higher in those with a high BMI. Similarly, when we assessed the correlation between BMI and gastric motility parameters, weak but positive correlations were found between BMI and FAA and AA15 after the test meal in the FAPD group and AA1 in controls. This pattern suggests that a higher BMI may contribute to more pronounced distension of the stomach during the fasting and postprandial periods in patients with FAPDs. This likely contributes to the development and/or aggravation of symptoms.

However, the subgroup analysis revealed that the relationship between BMI and gastric motility parameters varies across different FAPD subtypes. In children with FD, both FAA and AA15 were positively correlated with BMI, while GER, AAC, and MI were negatively correlated. This pattern suggests that in FD, in addition to increased distension of gastric antrum, a significant motility hypofunction is also present during both fasting and postprandial periods. This probably contributes to the symptoms such as early satiety, bloating, nausea, distension, and vomiting present in children with FD in addition to the epigastric pain[23,36,37].

The AM subgroup also exhibited a significant positive correlation between BMI and AA1 (which is an indirect indicator of gastric distension after a meal), suggesting that BMI might play some role in the pathophysiology of AM, especially causing symptoms such as nausea and vomiting. However, this association is less understood and requires additional research.

The absence of a significant correlation between BMI and motility parameters in the IBS subgroup could indicate that BMI has a less direct impact on gastric motility in IBS, which is primarily characterized by altered bowel habits than gastric dysmotility[21].

In contrast, in the FAP subgroup, BMI had weak but positive correlations with AAC and MI, indicating that a higher BMI might be associated with gastric hypermotility. This is a novel finding that warrants further investigation as it contrasts with the typically hypoactive motility patterns reported to be associated with high BMI in FGIDs[30].

The underlying pathophysiology for gastric antral distension and impaired gastric motility associated with higher BMI in our children with FAPDs, especially FD, needs further evaluation. The autonomic nervous system is considered one of the main regulators of gastrointestinal function including motility. Previous studies have suggested that functional extrinsic denervation and maladaptation in the parasympathetic division could partly contribute to the gastric dysmotility observed in children with FAPDs[20]. Although studies do not permit solid inferences regarding sympathetic activity in pediatric subjects who are obese, they do indicate lower vagal activity in children and adolescents who are obese compared with their normal-weight counterparts[38]. Dysfunction of the autonomic nervous system associated with high BMI is one possible underlying pathophysiological mechanism for the relationship between gastric motility dysfunction and high BMI in our patients with FD[39,40].

Furthermore, various hormones play a crucial role in modulating gastric motility, especially in individuals who are obese. The reduced levels of ghrelin, a hormone known to stimulate gastric motility, are often observed in subjects who are obese, leading to impaired gastric function and motility[29]. Cortisol, another key hormone, inhibits gastric motor function in the antrum and corpus through CRF-2 receptors[30]. Neuropeptide Y, a neurotransmitter secreted by visceral fat tissue promotes food intake and fat storage and inhibits gastric motility through its receptors found in the stomach [32]. Additionally, obesity is associated with increased endocannabinoid production in hypertrophic adipose tissue and the brain, which by acting on CB1 receptors stimulates appetite and perpetuates a cycle of weight gain. The activation of these receptors also delays gastric emptying and increases gastric acid secretion, further contributing to the motility disorders seen in obesity[31].

In addition, there is a direct relationship between energy intake, storage, expenditure, and gastrointestinal functions. The sensory and motor functions of the stomach, including gastric emptying and accommodation, play a crucial role in regulating energy consumption and appetite. Obesity, which is marked by an energy imbalance, often involves altered gastric functions. In particular, rapid gastric emptying and an increased fasting gastric volume in individuals who are obese can lead to greater food intake before reaching the sensation of fullness and heightened appetite[41]. Patients with FGIDs, including FD, usually have a hypersensitive gastrointestinal tract, and they probably have an exaggerated response, giving rise to this negative correlation[42].

These findings have important clinical implications for the management of FAPDs in children. The significant correlations between BMI and gastric motility parameters, particularly in FD, suggest that weight management could be a crucial component of therapeutic strategies for this condition. Interventions aimed at achieving a healthy BMI might help normalize gastric motility and reduce symptoms, especially in children with FD where a higher BMI is associated with more severe motility dysfunction. We used Sri Lankan BMI normal values to categorize children into different subgroups instead of international cutoffs. It is based on evidence that BMI cutoff values for overweight and obesity may not correspond to the same degree of fat content across different populations. The WHO technical report explicitly recognizes the need for population-specific rather than universal BMI cutoff values as ethnicity significantly influences body composition[4].

While the study provides valuable insights, it is important to acknowledge its limitations. The cross-sectional design limits the ability to infer causality between BMI and gastric motility abnormalities. Longitudinal studies are needed to determine whether changes in BMI over time directly influence the progression of gastric motility dysfunction in children with FAPDs. Additionally, the relatively small sample size in certain FAPD subgroups, particularly in AM, may limit the generalizability of the findings. Future research should aim to replicate these results in larger, more diverse cohorts to confirm the observed associations and explore the underlying mechanisms. Furthermore, the study focused solely on gastric motility, but other aspects of gastrointestinal function, such as intestinal transit and gut-brain axis interactions, may also be influenced by BMI and contribute to the symptoms of FAPDs. At the same time, other factors, such as dietary habits and psychological influences, may contribute to the observed gastric motility changes in children with FAPDs. These factors could modulate gastrointestinal function through mechanisms such as gut-brain axis interactions, stress-related hormonal changes, and altered feeding behaviors. Integrating assessments of these factors in future studies could provide a more comprehensive understanding of the relationship between BMI, gastrointestinal motility, and FGIDs in children.

CONCLUSION

Our study identified a significant relationship between elevated BMI and altered gastric motility dynamics characterized by increased gastric distension during both fasting (FAA) and postprandial (AA1, AA15) periods. Additionally, in children with FD, higher BMI was linked to reduced GER and a lower MI, suggesting an association between increased BMI and gastric hypomotility. These findings underscore the importance of lifestyle and dietary interventions aimed at optimizing BMI in the management of FAPD, particularly FD. Further investigation is needed to unravel the causal mechanisms connecting BMI and gastric dysmotility in pediatric FGIDs. Such insights could pave the way for more tailored and effective treatment strategies.

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FOOTNOTES

Author contributions: Karunanayake A contributed to the study design, data collection, analysis and interpretation of data, and wrote the initial draft; Rajindrajith S helped design the study and contributed to revisions to the final manuscript; Kumari MV contributed to the study design and data collection; Devanarayana NM conceptualized the study and contributed to the study design, data collection (by conducting motility studies), interpretation of data, and writing and revising the manuscript. All authors approved the final version to be published.

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