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IDENTIFICATION OF UNDERLYING CAUSES OF CHRONIC UNSPECIFIC UPPER GASTROINTESTINAL-RELATED SYMPTOMS IN CHILDREN, A PILOT STUDY

By

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Abstract

Upper gastrointestinal tract (GIT) symptoms are not disease specific and of limited value in the differentiation of GIT disorders. The present study aimed to determine the etiology of chronic unspecific symptoms in children and to test the need for upper endoscopy in diagnosis.

This is a prospective study for 30 Egyptian children presented with chronic upper GIT symptoms for at least 2 months. History regarding severity and frequency of GIT symptoms were asked for. Children with known disorder that explains presenting symptoms were excluded. Upper GIT endoscopy was performed and 5 biopsies were obtained for pathological examination and for *H pylori* testing.

The results showed that children age ranged between 2.5-18 years with mean ± SD of 13.6 ± 3.4 and 63.3% were females. The main complaints were epigastric pain in 43.3%, hematemesis in 30% and vomiting in 26.7%. Motility disorders were diagnosed in 66.7% children; in the form of GERD in 63.3% and achalasia in one. Complication of GERD in the form of erosive esophagitis was present in 15.8% children, while Barrett's esophagus was not observed. *H. pylori* infection was diagnosed in 80% histologically. Eosinophilic esophagitis was not detected.

Key words: Children, Egypt, Endoscopy, Erosive esophagitis, GERD, *H pylori*, Pathology.

Introduction

Gastrointestinal tract (GIT) symptoms can be attributed to many causes. In children gastrointestinal dysmotility are among the most common causes of GIT complaints, including: gastroesophageal reflux disease (GERD), esophageal achalasia and gastroparesis (Gariepy and Mousa, 2009; Ambartsumyan and Rodriguez, 2014). *Helicobacter pylori* (*H pylori*) gastritis and eosinophilic esophagitis (EE) are other probable etiologies (El-Mouzan *et al.* 2004). Gastroesophageal reflux is the retrograde movement of gastric contents across the lower esophageal sphincter into the esophagus. Reflux in older children is considered pathologic and consistent with GERD (El-Serag *et al*, 2004; Orenstein *et al*, 2006), while reflux is considered physiologic in infants and usually resolves at 2 years of age spontaneously (Campanozzi *et al,* 2009). GERD may be caused by mechanical factors, such as the increased frequency of transient lower esophageal sphincter relaxations, the presence of hiatal hernia, delayed gastric emptying, increased gastric acid secretion or overeating (Ambartsumyan and Rodriguez, 2014; Blanchard and Czinn, 2016). GERD is the most common GIT motility disorder affecting children, its symptoms occur in 2-8% of children aged 3–18 years (Nelson *et al*, 2000). In GERD, reflux causes symptoms (frequent heartburn, regurgitation and/or vomiting) and complications (erosive esophagitis, strictures, Barrett's esophagus and/or extra-intestinal manifestations). The prevalence of complications in children is unknown (Gilger *et al*, 2008). GERD is diagnosed clinically and treated initially with acid secretion blockade. Endoscopy is typically reserved for patients whose symptoms do not respond after 2 weeks therapeutic trial, to assess associated complications and to exclude other etiologies (Ambartsumyan and

Rodriguez, 2014). The prognosis of GERD in healthy children is good after treatment (Boccia *et al*, 2007).

Esophageal achalasia and disorders occurring after repair of congenital anomalies of upper GIT are common causes of GIT symptoms in children (Gariepy and Mousa, 2009). Symptoms were the same as that of the GERD (Lee *et al*, 2010).

H pylori colonize over 50% of people worldwide (Mitchel, 2001). It is one of the most common chronic bacterial infections world-wide. The prevalence rate is high in developing countries and the risk factors for acquiring infection are related to socioeconomic status, overcrowding, migration from high prevalence areas and other infected family members (Mitchel, 2001; Correa and Piazuelo, 2008). Colonization is usually acquired during childhood and is the most common cause of chronic gastritis, duodenal ulcer, gastric ulcer, gastric adenocarcinoma and mucosa-associated lymphoid tissue lymphoma. Manifestations are non-specific and similar to many GIT diseases (Pacifico *et al,* 2010). Children with *H pylori* have at least a fivefold increased risk of developing stomach neoplasia later life. Treatment of H. pylori infection in childhood is crucial to prevent premalignant changes in the future (Moayyedi, 2005).

EE is defined as a clinicopathologic condition characterized by esophageal symptoms and a dense esophageal eosinophilic infiltrates which is characteristically absent in the rest of the GIT. It is increasingly recognized in both children and adults (Dellon *et al*, 2008; Dalby *et al*, 2010). Symptoms are similar to those seen in GERD, with no response to acid suppression. They are responsive to anti-allergic treatments including dietary restriction and corticosteroids (Hirano, 2011).

Upper GIT endoscopy is essential to get knowledge about pattern of GIT disorders and to obtain a small bowel biopsy as well (Canan *et al.* 2011). Endoscopy carries a low risk of complications. Adverse event rates reported ranged from 1 in 200 to 1 in 10,000 and mortality rates ranging from 0- 1 in 2000 (Heuss *et al*, 2005).

The present study aimed to determine the etiology of unspecific upper GIT symptoms in children and to test the utility of upper endoscopy and histopathology in diagnosis.

Patients, Materials and Methods

A prospective cohort study was carried out for 30 children who presented to pediatric clinic at National Hepatology and Tropical Medicine Research Institute (NHTMRI) with chronic/recurrent upper GIT-related symptoms for at least 2 months duration prior to presentation. The study protocol was approved by institutional ethical committee and children were enrolled after obtaining an informed consent from their parents.

Inclusion criteria were children 2-18 years, of both sexes. Exclusion criteria were known disorder that explain the presenting symptoms such as; cholicystitis, gall bladder stones, urinary tract infections, renal stones, parasitic infestations, significant medical or neuropsychiatric illnesses, chronic use of non-steroidal anti-inflammatory drugs/ steroids or history of upper abdominal surgery.

All children were subjected to detailed medical history including duration, frequency and severity of the presenting symptoms. History of common dyspeptic symptoms was asked for (e.g. nausea, vomiting, regurgitation, hematemesis, heartburn/ retrosternal pain, epigastric/abdominal pain, anoexia/polyphagia, dyspepsia; post-feeding discomfort; dysphagia or food impaction with or without poor weight gain). They underwent full clinical examination and anthropometric measurements assessment including; weight in Kg, height in cm and body mass index in kg/m^2. All were plotted on Egyptian growth charts (Egyptian Growth Curves, 2008).

Laboratory investigations included urine analysis, stool analysis, complete blood count, creatinine, alanine aminotransferase (ALT) and aspartate aminotransferase (AST).

Abdominal ultrasound examination was performed by a single sonographer using an FFsonic; Fukuda Denshi Co, Tokyo, Japan.

Upper GIT endoscopy and biopsies: The use of H_2 blocker, proton pump inhibitor (PPI) or antibiotics was forbidden for 4 weeks prior to endoscopic examination. The procedure was performed under intravenous sedation by a single endoscopist after at least 6-hour fasting. It was performed after pharyngeal anesthesia with lidocaine spray (Xylocaine, AstraZeneca, Södertälje, Sweden). Sedation with 2-3 mg intravenously administered midazolam (Dormicum, Roche AB, Stockholm, Sweden) was provided on demand. Ulceration was defined endoscopically as a mucosal break with unequivocal depth and a diameter of at least 3 mm. The presence of hiatal hernia, nodularity and ulceration in the esophagus were also recorded.

The mucosal biopsies were obtained from the proximal, mid and distal esophagus, stomach and duodenum for histology. By an Olumpus XQ 260 Gastro-videoscope and standard jaw biopsy forceps were used for obtaining biopsies.

Histologically *H pylori*: biopsy specimens were examined by a single pathologist. Specimens were fixed in 10% buffered formalin processed and embedded in paraffin wax. From each block, several 5u thick sections were cut on slides and dried overnight at 37^0 C. Before staining, the sections were dewaxed in xylene and rehydrated thought graded concentrations of ethanol to distilled water. Sections were stained with hematoxylin and eosin for morphological observation and Giemsa for the detection of *H. pylori*. As regard gastric biopsy, the following parameters were scored according to Sydney classification system: lymphocytes and plasma cell infiltration denote chronic inflammation of *H. pylori* while polymorphonuclear leucocyte infiltration stands for activity of *H. pylori* infection. Inflammation score is based on the density of inflammatory cells in both lamina propria and glandular epithelium. The histological glandular atrophy is identified when the gastric glands were correspondingly decreased in amount and/or widely separated. In accordance with the update Sydney system, the degree of mucosal inflammation, activity of *H. pylori* infection, glandular atrophy and intestinal metaplasia were classified into four grades as follows: 0, none;1, mild; 2, moderate and 3, marked (Dixon *et al*, 1996).

The presence of eosinophils was tested in all biopsy specimens. Esophageal eosinophilic count >15 per high power field (HPF) is defined as dense esophageal eosinophilia, which if present in the absence of eosinophilic inflammation in the rest of digestive tract correlates with EE (Dellon *et al*, 2008; Dalby *et al*, 2010).

Statistical analysis: Data were analyzed by the Statistical Package for Social Science (SPSS) program version 17.0 was used. Mean and standard deviation (SD) or median and interquartile range (IQR) were estimates of quantitative data while frequency and percentage were estimates of qualitative data. Differences in clinical and biochemical characteristics were tested by Student's t test for quantitative data and by chi-square test for non-parametric (qualitative) data. A two-sided P value <0.05 was considered statistically significant. Odds ratio were used to measure the strength of associations.

Results

Patients were 11 (36.7%) males and 19 (63.3%) females with a mean age of 13.6 $\pm$ 3.4 years (2.5-18). Seventy percent (21 children) of them lived in urban areas while 30% lived in rural areas and 25 (83.3%) children were educated. The main complaints were; epigastric pain in 13 (43.3%), hematemesis in 9 (30%) and vomiting with or without nausea in 8 (26.7%). The median (IQR) duration of complaint was 12 (6.5) months ranged between 2-24 months. The frequency of recurrence of symptoms had a median of 3.5 (5) times per week, ranged from 1-30 times/week. Precipitating factor for symptoms was food in 23.3%, fasting in

16.7% and 60% of children have no observed cause. Reliving factors were fasting in 16.7%, medications in 10%, vomiting in 6.7%, food in 6.7% while 60% had no reliving factor. Associated respiratory symptoms were present in 43.3% (Tab. 1).

The mean weight was 46±15.3 kg, (range 13-81) and 7 (23.3%) of them were below the 5th weight for age percentile. The mean height of children was 150±18 cm, (range 87-180) and 23% were below the 5th percentile. The mean BMI was 19.8±3.9 kg/m², (range 14.4-32.9) and it was below 5th in 20% and above 85th in 6.7% of children. Abdominal examination revealed epigastric tenderness in 18 (60%) of children with no palpable organs. Ultrasound examination showed echogenic liver in 2 (6.7%) children. Increased echogenicity was statistically significant among children with BMI ≥85th percentile than in those <85th percentile (50% vs 3.6%, P: 0.01*). Complete blood count, creatinine, ALT, AST were within normal. HCV antibody was positive in 4 (13.3%) children with a detectable HCV RNA in blood by PCR (data not shown).

GERD was diagnosed in 20 children, mainly grade A, except one who was grade B, one had lower small ulcer and one with diffuse esophagitis showed ulcerations with annular pattern. All gastrodoudenitis were associated with diffuse esophagitis (Tab. 2).

The achalasia case was 16 years old boy, presented with vomiting, dysphagia to solids, anorexia, epigastric pain and weight loss. He was frequently admitted at chest hospital in the year prior to presentation with recurrent chest infection. Upper endoscopy revealed a dilated esophagus with retained food products. Histological findings revealed lower esophagitis and gastrodoudenitis. Barium swallow was performed showing the classic "bird beak" appearance of the distal esophagus, with proximal dilation and air-fluid levels.

Pathology: *H pylori* were detected in 24 (80%) specimens (Fig. 1), in the antrum in 19 cases (97.2%) and in both antrum and duodenum in 5 (20.8%) cases. None had eosinophilia > 15/ HPF. Histological examination was normal in 21.1% with GERD. Among cases with normal endoscopic esophageal appearance, one had lower esophagitis and another one had diffused chronic esophagitis (Tabs. 3 & 4).

Comparison between children diagnosed with GERD and other group given (Tab. 5). Vomiting with or without nausea was significantly associated with GERD (*P*=0.002*). Cases with GERD had more regurgitation (26.3% vs 0%) and increased food intake (26.3% vs 0%) than those without GERD reaching near statistical significant. Cases with GERD were more frequently associated with upper respiratory manifestation (laryngitis, sinusitis, otitis media) than cases without GERD (*P*=0.003*). There was no significant difference when comparing other factors. Pathologically, esophagitis was detected in 78.9% of cases with GERD; erosions were observed in 3/19 children (15.8%). *H pylori* was diagnosed in 78.9% of cases with GERD. Female sex was a risk factor for GERD with odds ratio 6.6 (95% Confidence Interval 1.3-34.2). Other factors showed no increased risk for GERD (data not shown).

H pylori were mainly females and significantly older than children without *H pylori.* Epigastric pain was significantly more frequent among them. Twenty-five percent were below 5th percentile for weight, height and BMI. Two *H. pylori* positive had BMI >85th percentile with increased liver echogenicity by ultrasound. Histologically chronic antral gastritis was in all but doudenitis in 95.8% (Tab. 6). Risk factors were female gender, age > 10 years, living in urban area, histological gastritis and doudenitis (Tab. 7).

Table 1: Characteristics of studied group (n=30)

	Number	Percent
- *Male*	11	36.7
- *Female*	19	63.3
Main presenting complaint		
• *Epigastric pain*	13	43.3
• *Hematemesis*	9	30
• *Vomiting with or without nausea*	8	26.7
Dyspeptic Symptoms		
• *Epigastric pain*	25	83.3
• *Vomiting with or without nausea*	23	76.7
• *Hematemesis*	14	46.7
• *Dyspepsia*	13	43.3
• *Anorexia/early satiety*	10	33.3
• *Dysphagia*	6	20
• *Regurge*	5	16.7
• *Neck contortion*	5	16.7
• *Increased food intake*	5	16.7
• *Chocking*	1	3.3
Course; - *Progressive*	17	56.7
- *Stationary*	13	43.3
Precipitating factors		
• *Non*	18	60
• *Food*	7	23.3
• *Fasting*	5	16.7
Relieving factors		
• *Non*	18	60
• *Fasting*	5	16.7
• *Treatment*	3	10
• *Food*	2	6.7
• *Vomiting*	2	6.7
Respiratory symptoms		
• *Non*	17	41.5
• *Laryngitis*	9	22
• *Sinusitis*	8	19.5
• *Otitis media*	5	12.2
• *Asthma*	2	4.9
Past history of abdominal pain	6	20
Previous treatment for dyspepsia	27	90
Positive consanguinity	10	33.3
Positive family history of gastrointestinal disease	2	6.7
Positive family history of allergy	10	33.3

Table 2: Endoscopic findings of study group (n=30)

Endoscopic Findings	Number	Percent
Esophagus		
• *Normal*	7	23.3
• *GERD A/B*	19	63.3
• *Cardiac achalasia*	1	3.3
• *Diffuse esophagitis, with or without ulceration*	3	10
Stomach & Duodenum		
• *Antral gastritis*	7	23.3
• *Doudenitis*	2	6.7
• *Gastrodoudenitis with or without erosions*	21	70

Table 3: Endoscopic and pathological finding of esophagus

Pathological Findings N (%)	Normal N=7	GERD N=19	Cardiac achalasia N=1	Diffuse esophagitis N=3	P value
• *Normal*	5 (71.4)	4 (21.1)	0	0	
• *Lower esophagitis with/without hyperplasia*	1 (14.3)	8 (42.1)	1 (100)	2 (66.6)	
• *Diffuse chronic esophagitis with/without erosions*	1 (14.3)	7 (36.8)	0	1 (33.3)	0.1

* *P*<0.05 significant. GERD: gastroesophageal reflux disease

Table 4: Endoscopic and pathological finding of stomach and duodenum

Endoscopic findings / Pathological Findings	Antral gastritis N=7	Doudenitis N=2	Gastrodoudenitis N=21	P value
Stomach				
• *Normal*	0	1 (50)	0	<0.001*
• *Chronic gastritis with/ without erosions*	7 (100)	1 (50)	21 (100)	
Duodenum				
• *Normal*	3 (42.9)	0	0	0.02*
• *Acute doudenitis*	0	0	1 (4.8)	
• *Chronic doudenitis*	4 (57.1)	2 (100)	20 (95.2)	

* *P*<0.05 significant

Table 5: Comparisons of children with and without GERD

	GERD (N=19)	Non GERD (N=11)	P value
Age;			
• *Mean ± SD*	13.3 ± 3.8	14 ± 2.6	0.6
• *> 10 years; N (%)*	16 (84.2)	10 (90.9)	
• *Male*	4 (21.1)	7 (63.6)	
• *Female*	15 (78.9)	4 (36.4)	0.04*
Dyspeptic Symptoms; *N (%)*			
• *Epigastric pain*	14 (73.7)	11 (100)	0.06
• *Vomiting with or without nausea*	18 (94.7)	5 (45.5)	0.002*
• *Hematemesis*	9 (47.4)	5 (45.5)	0.9
• *Dyspepsia*	9 (47.4)	4 (36.4)	0.6
• *Anorexia/early satiety*	5 (26.3)	5 (45.5)	0.3
• *Dysphagia*	4 (21.1)	2 (18.2)	0.8
• *Regurgitation*	5 (26.3)	0	0.06
• *Neck contortion*	3 (15.8)	2 (18.2)	0.8
• *Increased food intake*	5 (26.3)	0	0.06
• *Chocking*	1 (5.3)	0	0.4
Respiratory symptoms; *N (%)*			
• *Non*	8 (42.1)	9 (81.8)	0.003*
• *Laryngitis*	9 (47.4)	0	
• *Sinusitis*	7 (36.8)	1 (9.1)	
• *Otitis media*	5 (26.3)	0	
• *Asthma*	0	2 (18.2)	
Epigastric tenderness; *N (%)*	12 (63.2)	6 (54.5)	0.7
Endoscopic gastritis; *N (%)*	19 (100)	9 (81.8)	0.1
Endoscopic doudenitis; *N (%)*	12 (63.2)	11 (100)	0.03*
Histology of esophagus; *N (%)*			
• *Normal*	4 (21.1)	5 (45.5)	0.2
• *Esophagitis*	15 (78.9)	6 (54.5)	
H pylori; N (%)	15 (78.9)	9 (81.8)	0.9

* *P*<0.05 significant. GERD: gastroesophageal reflux disease.

Table 6: Comparison between cases with and without *H pylori*

	Positive (N=24)	Negative (N=6)	P value
Age			
• *Mean ± SD*	14.4 ± 2.4	10.1 ± 4.7	0.003*
• ≥ 10 years; N (%)	23 (95.8)	3 (50)	0.003*
• *Male*	6 (25)	5 (83.3)	
• *Female*	18 (75)	1 (16.7)	0.02*
Main complaint			
• *Epigastric pain*	13 (54.2)	0	0.03*
• *Hematemesis*	5 (20.8)	4 (66.7)	
• *Vomiting with or without nausea*	6 (25)	2 (33.3)	
Dyspeptic Symptoms			
• *Epigastric pain*	22 (91.7)	3 (50)	0.01*
• *Vomiting with or without nausea*	19 (79.2)	4 (66.7)	0.5
• *Hematemesis*	10 (41.7)	4 (66.7)	0.3
• *Dyspepsia*	12 (50)	1 (16.7)	0.1
• *Anorexia/early satiety*	9 (37.5)	1 (16.7)	0.3
• *Dysphagia*	5 (20.8)	1 (16.7)	0.8
• *Regurgitation*	5 (20.8)	0	0.2
• *Neck contortion*	5 (20.8)	0	0.2
• *Increased food intake*	4 (16.7)	1 (16.7)	0.4
• *Chocking*	1 (4.2)	0	0.6
Positive HCV Ab (n=15)	4 (40)	0	0.09
Epigastric tenderness	16 (66.7)	2 (33.3)	0.2
Endoscopic GERD	15 (62.5)	4 (66.7)	0.9
Endoscopic gastritis	23 (95.8)	5 (83.3)	0.4
Endoscopic doudenitis	19 (79.2)	4 (66.7)	0.6
Pathology			
Esophagus			
• *Normal*	6 (25)	3 (50)	0.2
• *Chronic esophagitis*	18 (75)	3 (50)	
Stomach			
• *Normal*	0	1 (16.7)	
• *Chronic antral gastritis*	24 (100)	5 (83.3)	0.04*
Duodenum			
• *Normal*	1 (4.2)	2 (33.3)	0.03*
• *Chronic doudenitis*	23 (95.8)	4 (66.7)	

* $P<0.05$ significant. GERD: gastroesophageal reflux disease.

Table 7: Risk factors for *H. pylori*

	positive N=24	Negative N=6	P value	Odds ratio	95% Confidence Interval	
					Lower	Upper
• *Male*	6 (25)	5 (83.3)				
• *Female*	18 (75)	1 (16.7)	0.02*	15	1.4	155.3
Age ≥ 10 years	23 (95.8)	3 (50)	0.2*	23	1.8	298.4
Urban area	18 (75)	3 (50)	0.3	3	0.5	19
Histological gastritis	24 (100)	5 (83.3)	0.04*	5.8	2.6	12.9
Histological doudenitis	23 (95.8)	4 (66.7)	0.01*	11.5	0.8	158.7

Discussion

Upper GIT symptoms are not disease specific and remain of limited value in the differentiation of GIT disorders. Among studied children with chronic upper GIT symptoms, both endoscopy and histopathological examination were used to reach diagnosis. Motility disorders were diagnosed in 20 (66.7%) children; GERD in 19 (63.3%) and achalasia in one. GERD was associated with

complications in the form of erosive esophagitis in 3 (15.8%) children. *H pylori* was diagnosed histologically in 24 (80%).

Among the study group, GERD was diagnosed in 19 (63.3%) children by endoscopy. GERD is commonly encountered in pediatric clinics among otherwise healthy older children/adolescents (Ambartsumyan and Rodriguez, 2014). Although endoscopy has low sensitivity (41%) and specificity (77%) as a diagnostic tool of GERD, it has proven to be a valuable procedure in children with persistent symptoms (Kwon *et al*, 2008).

In the study group, females were commonly diagnosed with GERD than males (78.9% vs 21.1%, *P*=0.04). This is in contrary to other studies that observed preponderance of male children with GERD (Tolaymat and Chapman, 1998; Gilger *et al*, 2008; Kwon *et al*, 2008). Vomiting was the commonest complaint in cases with GERD followed by epigastric pain. This was in agreement with Lee *et al*. (2011).

In the present study, laryngitis, sinusitis and otitis media were more common in children with GERD while asthma was not. GERD in older children is more frequently associated with airway manifestations related to asthma or to otolaryngologic disease such as laryngitis or sinusitis (Khan and Orenstein, 2016).

Among the children, esophagitis was detected in 78.9% of cases with GERD; erosive esophagitis were observed in 3 out of the 19 children (15.8%). Erosive esophagitis, stricture and Barrett's esophagus have been listed among complications associated with GERD (Ambartsumyan and Rodriguez, 2014). The prevalence of erosive esophagitis in our GERD children is similar to that reported in other studies conducted in neurologically normal children with GERD who underwent endoscopy; it ranged between 12-30% (El-Serag *et al*, 2002; Gilger *et al*, 2008; Kwon *et al*, 2008). Endoscopy use is useful to delineate an erosive esophagitis from a non-erosive reflux disease (Park and Chang, 2012), as both present very similar symptoms in children (Boccia *et al*, 2007). The lack of Barrett's esophagus in our studied specimens is in agreement with other reports (El-Serag *et al*, 2006; Nguyen *et al*, 2011). They suggested that most likely the duration of reflux is the major risk factor for Barrett's esophagus. On the other hand, an older study had reported Barrett's esophagus in 2.7% of the studied children with GERD (Tolaymat and Chapman, 1998).

The present case of achalasia was 16 years old boy, presented with vomiting, dysphagia to solids, anorexia, epigastric pain, weight loss and history of recurrent chest infections. He lost 13 kg in one year prior to presentation with provisional diagnosis of chest infections. Other reports had stated the higher incidence of esophageal achalasia in adolescents (Upadhyaya *et al*, 2008; Marlais *et al*, 2011). Däbritz *et al.* (2010) proposed that weight loss due to dysphagia may be attributed to other causes leading to a delay in diagnosis and treatment.

Among the studied children, the prevalence of *H. pylori* was 80%. Other studies in children from developing countries had reported prevalence that ranged from 10% to over 90% (Hunt *et al*, 2011). In Egypt, previous studies reported prevalence rates ranged between 50-70% in children (Omar *et al*, 2001; Mohammad *et al*, 2008). There are significant differences in the prevalence of *H. pylori* infection worldwide and even in various parts of specific country. Its prevalence rate is high in developing countries (Torres *et al*, 2000).

From our results, *H. pylori* infection was significantly more common in children older than 10 years (95.8% vs 50%, *P*=0003). This was agreed with previous pediatric studies that showed cumulative infection rate with increasing age (Malaty *et al*, 2002; Jafar *et al*, 2013). On the other hand, in other studies *H. pylori* was acquired early in life, before age of 10 years and persisted for life in absence of antibiotic therapy (Kato and Sherman, 2005; Hobsley *et al*, 2007; Correa and Piazuelo, 2008; Hestvik *et al*, 2010).

Females were a risk factor for *H pylori* in the present study. Others found no statistical difference between the rates of prevalence in male and female populations (Malaty *et al*, 2002; Jafar *et al*, 2013). Other studies reported male preponderance (Hestvik *et al*, 2010). The small sample size of our study may contribute to this finding.

Epigastric pain was observed in 91.7% of *H. pylori* positive children. *H. pylori* role in children with recurrent abdominal pain was controversial (Zeyrek *et al*, 2008; Hestvik *et al*, 2010; Badr *et al*, 2012).

Among studied *H pylori*-positive children, 25% of them were below 5th percentile for weight, height and BMI. Many reports in children observed similar association between *H. pylori* and growth impairment. They concluded that dyspepsia caused by *H. pylori* might be considered to cause malnutrition secondary to decreased caloric intake (Mera *et al*, 2006; Süoglu *et al*, 2007; Soylu and Ozturk, 2008). Others suggested that growth suppression reported in children with *H. pylori* infection could be due to socioeconomic class and not related to infection (Sood *et al*, 2005). In positive children, endoscopic gastritis was present in 95.8%, while it was present in all cases histologically. Doudenitis was detected by endoscopy in 79.2% and histologically in 95.8%. No doubt, during childhood, *H. pylori* is associated with predominant antral gastritis and duodenal ulcers in rates much less common than in adults (Sherman *et al*, 2002; Kato *et al*, 2004). In developing countries in which the rates of ulcer or gastric cancer are high, initial endoscopy is an appropriate initial approach than starting empirical treatment with a PPI (Hunt *et al*, 2011).

In the present children with *H. pylori*, 62.5% had GERD and among cases with GERD, 78.9% were positive for *H. pylori*. A potential role of *H. pylori* infection in GERD has been suggested recently (Pellicano *et al*, 2009).

None of children showed isolated esophageal dense eosinophilia >15/HPF and hence none was diagnosed with EE. But, increased rate of EE in both children and adults was repotted (Dellon *et al*, 2008; Dalby *et al*, 2010).

Conclusion

The motility disorders were common among our studied children; mainly in the form of GERD and only one case was diagnosed with achalasia. Complication of GERD in the form of erosive esophagitis was present in 15.8%, while none showed Barrett's esophagus. *H. pylori* infection was very common among study group, causing growth impairment in 25% of cases. EE was not detected in the study group. Upper GIT symptoms are not disease specific and remain of limited value in the differentiation of GIT disorders. Endoscopy is essential to get knowledge about pattern of GIT disorders and to obtain a small bowel biopsy for pathological examination.

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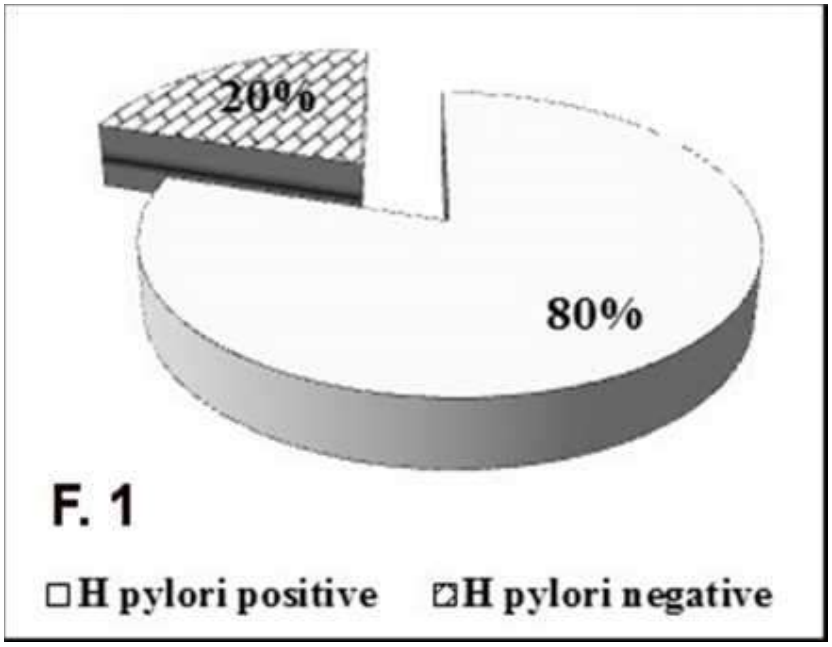

Fig. 1: Prevalence of *H pylori* in study group