Functional gastrointestinal disorders: past and present

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Background: **Chronic abdominal pain is a common complaint in childhood and adolescence. Despite decades of clinical observations and research, it still poses a challenge to pediatric health care professionals. The aim of this review is to highlight the epidemiology of pediatric chronic abdominal pain and to describe the pathogenesis of this disorder, its clinical manifestations, evaluation and therapeutic options.**

Data sources: **Articles on chronic abdominal pain in the recent years from PubMed, MEDLINE, and reference textbooks were reviewed.**

Results: **Chronic abdominal pain, a functional gastrointestinal disorder (FGID), is a multifactorial condition that results from a complex interaction between psychosocial and physiologic factors via the brain-gut axis. A thorough history coupled with a complete physical examination and normal screening studies rule out an organic cause in 95% of the cases. It is highly important for the physician to establish a trusting relationship with the child and parents because successful treatment including modification of physical and psychological stress factors, dietary changes, and drug therapy depends greatly on education, reassurance and active psychological support.**

Conclusions: **FGIDs are a cause of great anxiety, distress and morbidity in children as well as adults. As our understanding of these conditions improves, our therapeutic interventions will progress not only to overcome them but also to intervene early in the disease course so as to limit long-term impact.**

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Introduction

Chronic abdominal pain is a common complaint in childhood and adolescence. Despite decades of clinical observations and research, it still poses a challenge to pediatric health care professionals. It may be the predominant clinical manifestation of a large number of organic disorders (Tables 1-4), but in the majority of cases, chronic abdominal pain is a functional disorder with no anatomic, metabolic, infectious, inflammatory or neoplastic cause to account for it.[1,2] Nevertheless, chronic abdominal pain generates a lot of anxiety and distress in patients and their families.

John Apley,[3-5] an English pediatrician in the 1950s, was the first to extensively study this disorder. He defined it as the occurrence of 3 or more episodes of abdominal pain of such severity as to interfere with the child's normal activity over a 3-month period without any clear etiology. Over the years since Apley's seminal paper, researchers have described various subgroups of abdominal pain. The effort began in Rome in 1988 when a group of experts attending the International Congress of Gastroenterology attempted to define criteria to more accurately diagnose irritable bowel syndrome using a symptom-based approach. The criteria for diagnosis which they proposed, referred to as the Rome criteria, made it possible to diagnose functional gastrointestinal disorders (FGIDs) without extensive exclusionary testing. The first Rome criteria, which was adult-oriented, was updated in 1999 (Rome II) when childhood FGIDs were included.[6] Following publication, the Rome II criteria was used by researchers to assess prevalence and estimate rates of FGIDs among children with abdominal pain.[7-10] The application of the criteria in the clinical setting, however, was limited.[11] Studies showed that 16%-35% of children who previously would have satisfied Apley's recurrent abdominal pain criteria did not fit any of the Rome II criteria.[8-10] In 2006 the Rome III criteria emerged as a new and improved document that addressed the previously encountered problems with the Rome II criteria.[12] The Rome III criteria now categorize abdominal pain into (1) functional dyspepsia (FD), (2) functional abdominal pain (FAP) and functional abdominal pain syndrome (FAPS), (3) irritable bowel syndrome (IBS), and (4) abdominal migraine.[12] A recent study examining the implications of these changes for patient classification found the

Table 1. Differential diagnosis of functional dyspepsia

Inflammation
Gastroesophageal reflux
Eosinophilic esophagitis or gastroenteritis
NSAID induced gastritis
Helicobacter pylori gastritis
Duodenal ulcer
Inflammatory bowel disease
Henoch-Schonlein purpura
Dysmotility
Gastroparesis
Biliary dyskinesia
Pseudo-obstruction
Extraintestinal disorders
Chronic hepatitis
Chronic pancreatitis
Chronic cholecystitis
Abdominal migraine
Psychiatric disorders
Ureteropelvic obstruction

Table 2. Differential diagnosis of functional abdominal pain

Fecal impaction (left lower quadrant mass or suprapubic fullness, dilated rectal vault, firm stool in rectal vault)
Infection
Parasitic (Giardia Lamblia)
Bacterial (Yersinia and tuberculosis can cause mucosal thickening of terminal ileum)
Partial small bowel obstruction
Crohns disease (RLQ tenderness, rash, joint pain, fever)
Malrotation with or without volvulus (bilious or non-bilious vomiting)
Intussusception
Postsurgical adhesions
Infections
Genitourinary disorders
Ureteropelvic junction obstruction (episodic vomiting)
Dysmenorrhea (dull pain, midline or generalized, starts at onset of bleeding)
Pelvic inflammatory disease
Ectopic pregnancy
Endometriosis
Musculoskeletal disorders (localized and sharp pain, triggered with change in position)
Vascular disorders
Polyarteritis nodosa (associated with renal involvement)
Systemic lupus erythematosus
Mesenteric thrombosis (history of oral contraceptive use)
Ischemic colitis (post surgery, hypotension)
Abdominal migraine (associated with cyclic vomiting)
Acute intermittent porphyria (associated with headaches, dizziness, weakness, syncope, hallucinations and seizures)
Psychiatric disorders (conversion disorder triggered by stress, history of hysterical personality)

revised criteria to be more inclusive, allowing the classification of 86.6% compared to 68% of pediatric patients with unexplained chronic abdominal pain when using the Rome II criteria.[13]

Table 3. Differential diagnosis of irritable bowel syndrome

Inflammation
Ulcerative colitis
Crohn disease
Infection
Parasitic (Giardia)
Bacterial (*Yersinia*, *Campylobacter*, *Tuberculosis*, *Clostridium difficile*)
Drug induced diarrhea
Celiac disease
Lactose intolerance
Neoplasia (lymphoma)

Table 4. Differential diagnosis of abdominal migraines

Gastrointestinal
Obstruction (volvulus)
Recurrent pancreatitis
Cholelithiasis
Ulcerative colitis
Crohn disease
Rheumatological
Familial Mediterranean fever
Systemic lupus syndrome
Behcet's disease
Metabolic
Acute intermittent porphyria
Genitourinary
Ureteropelvic obstruction
Ovarian torsion

Epidemiology

In early studies chronic abdominal pain was reported to affect 10%-15% of school-age children.[3,14,15] A study by Hyams et al[15] on 507 adolescents in a suburban town in the USA found that abdominal pain occurred at least weekly in 13%-17%, yet only half of them had seen a health care professional within the past year. Thus, the incidence of FGIDs is likely higher than what is seen in clinical practice. This is due in part to cultural, socioeconomic, cognitive, and familial factors that shape a child's response to pain and influence health care-seeking behavior. A systematic review of the published literature on the epidemiology of recurrent abdominal pain in childhood conducted by Chitkara et al[16] showed that prevalence in western countries ranges between 0.3% and 19%. The most common ages of onset are 4-6 years and early adolescence.[5,17] It is uncommon under the age of 4 years.[3,18] Gender differences manifest around puberty with a slight female predominance of 1.4:1.[17,19] While some studies suggest that children with certain familial environments, including a single parent household, a parent with gastrointestinal complaints, a mother with neuroticism, low parental academic attainment, or a low socioeconomic status, are more likely to develop

chronic abdominal pain;[20-22] these observations are not consistent with those reported in the literature.[23,24]

Morbidity

Morbidity associated with FGIDs is mainly psychosocial. Pain interferes with normal school attendance and performance, peer relationships and participation in family and personal activities.[25-28] One out of 10 children with functional abdominal pain attends school regularly while more than 28% of patients have absenteeism greater than one day in 10.[27] Data from adults have shown that patients who suffer from FGIDs may also endure sleep difficulties, headaches, dizziness and fatigue.[29] Similar studies, however, are lacking in pediatrics. Furthermore, psychological disorders such as anxiety and depression are common.[30,31] Children with FAP tend to have a lower quality of life than healthy children but similar quality of life as patients with demonstrable organic gastrointestinal disease.[32] There are also suggestions that siblings of children with FGIDs experience more emotional/behavioral symptoms than their peers but their symptoms are not readily identified by their parents.[33]

Children with chronic abdominal pain have a high utilization of the health care system as they, along with their parents, seek answers for the unexplained abdominal pain.[34,35] They have multiple visits to the healthcare providers and may even seek second and third opinions.[10,35] They report fear of having a serious condition that is missed by their provider.[36] As a result, pediatricians may feel compelled to subject patients to multiple tests, including blood tests, radiological studies and endoscopies to avoid missing a serious illness and to try to reach an organic diagnosis.[37] The FGIDs are, therefore, a potentially debilitating group of disorders.

Functional dyspepsia

The prevalence of dyspepsia varies between 3.5% and 27% depending on gender and country of origin.[12] The Rome III criteria define FD as persistent or recurrent pain or discomfort centered in the upper abdomen which is not relieved by defecation, not associated with a change in stool pattern and not explained by an organic disease. The pain should recur at least once a week for at least 2 months for a diagnosis to be made.[12] Dyspepsia is usually poly-symptomatic. Ninety-nine percent of adult patients report more than two symptoms, more than 80% report more than five symptoms and less than 0.1% report one symptom.[38] The pain or discomfort can be associated with vomiting, nausea, abdominal fullness, bloating or early satiety.[39] Hyams and colleagues[40] reported that 26% of 127 pediatric subjects had ulcer-like symptoms while 15% manifested dysmotility-like symptoms.

The etiologies of FD are not yet fully elucidated. Myoelectrical abnormalities including delayed gastric emptying, impaired gastric accommodation and visceral hypersensitivity have been proposed as pathophysiologic mechanisms. Visceral hypersensitivity is a conscious perception of visceral stimulation independent of the intensity of stimulation; adult patients with FD have lower discomfort thresholds to gastric distension compared with controls.[41] Also, studies have demonstrated that adolescents with FD have impaired gastric emptying and reduced gastric accommodation compared to controls.[42,43] Chitkara and collegues[44] reported that 40% of patients with FD had slow small bowel transit times and an increased likelihood of reporting bloating and abdominal pain. In adults, however, delayed gastric emptying has not been shown to correlate with impaired quality of life, suggesting that psychological factors may play a role in symptom expression.[45] German investigators reviewed several adult observational studies and found an association between FD, anxiety and depression;[46] however, although psychological and psychosocial factors can coexist with FD, a causative role has not been established. Additionally, although *Helicobacter pylori* (*H. pylori*) infection can cause ulcers and associated abdominal pain, in the United States *H. pylori* is an uncommon cause of dyspeptic symptoms in children.[47] As such the role of *H. pylori* in FD continues to be debated.

Because there are no symptoms or signs that distinguish functional dyspepsia from upper gastrointestinal inflammation, structural or motility disorders, careful history and complete physical examination should screen for red flags that suggest the need for further testing. These red flags would include nocturnal symptoms, weight loss exceeding 10% of body weight, growth retardation, blood in stool, extraintestinal manifestations, pain localized away from the umbilicus, significant vomiting, and family history of inflammatory

Table 5. Red flags

Involuntary weight loss
Growth retardation
Significant vomiting or diarrhea
Blood in stool
Pain localized away from the umbilicus
Pain interrupting sleep at night
Extraintestinal symptoms (fever, rash, joint pain, apthous ulcers, urinary symptoms)
Abnormal labs: anemia, elevated sedimentation rate
Family history of organic disease (e.g., peptic ulcer, inflammatory bowel disease)

bowel disease[48] (Table 5). An upper gastrointestinal endoscopy becomes warranted in the presence of pyrosis, when a diagnosis of *H. pylori*-associated disease is suspected in patients with persistent symptoms despite the use of acid blockers, or in patients whose symptoms recur upon stopping such medications.[12] Patients with significant vomiting require an upper gastrointestinal series with small bowel follow through to exclude mechanical obstruction, and gastric scintigraphy to exclude gastroparesis.[39]

Therapeutic approaches to FD are based on proposed pathophysiological mechanisms. Though controversial, diet modifications such as the avoidance of fatty foods which may delay gastric emptying or avoidance of gas-producing foods or drinks to prevent bloating are usually recommended and may be helpful in some patients.[49] Studies in adults suggest that proton pump inhibitors may improve symptoms in some individuals with FD even in the absence of gastroesophageal reflux.[50] In a double-blind, placebo-controlled trial of 25 children, famotidine was superior to placebo (68% vs. 12%) in treating abdominal pain and dyspepsia.[51] Although there is no convincing evidence of their effectiveness, prokinetic agents have also been used to treat FD. Erythromycin enhanced gastric emptying but was not associated with a beneficial effect on meal-related symptom severity in adult dyspeptic patients.[52] Itopride, a dopamine agonist with antiacetylcholinesterase activity, significantly improved dyspeptic symptoms in adults in one study but was not different from placebo in another study.[53,54] Other therapies that are reported in the literature include alternative therapies such as hypnotherapy which has been shown to produce long-term benefit,[55] and acupuncture which improved gastric emptying as well as dyspeptic symptoms in adult patients with FD.[56] Hyams et al[40] demonstrated that 70% of children with FD were either asymptomatic or much improved at 6 months to 2 years follow up with 85% of those children on no specific therapy.

Irritable bowel syndrome

IBS is defined by the Rome III criteria as abdominal discomfort or pain associated, at least 25% of the time, with two of the following: 1) improvement with defecation, 2) change in stool frequency or 3) change in stool form.[12] IBS is the most prevalent digestive disease in adults, representing 12% of visits to primary care physicians and 28% of referrals to gastroenterologists.[57] It is now widely accepted that the prevalence of IBS is 10%-20% of the US population.[58] Using the Rome II criteria, IBS was diagnosed in 22%-45% of children aged 4-18 years presenting to tertiary care clinics.[8,10] Female-to-male prevalence ratios vary from 1:1 to >2:1 across a variety of adult studies.[59] Although some of this gender-related bias has been attributed to differences in utilization of health care, there is considerable evidence to suggest that gender-related differences in sensory and autonomic responses to pain and stress exist.[60]

IBS is considered a biopsychosocial disorder that results from the interaction of psychosocial factors, altered motility, and increased sensitivity of the bowel.[59] Alterations along the brain-gut pain axis are thought to result in central nervous system (CNS) amplification of incoming visceral afferent signals resulting in hyper-responsiveness to physiologic as well as noxious stimuli.[11,61] Using functional magnetic resonance imaging that highlights activity in specific regions of the brain, adult studies have demonstrated that altered central nervous system processing of gut signals can play a major role in IBS.[62,63] Recent studies suggest that FAP and IBS are associated with rectal hypersensitivity;[64,65] however, in many cases the degree of hypersensitivity is not proportional to the severity of symptoms.[66] Hence, visceral hypersensitivity, while it is likely a mechanism of IBS, does not explain the entire symptom complex of the disorder.

Additional evidence suggesting involvement of physiologic stimuli, noxious stimuli, altered motility or psychological stress is the observation that in children, as in adults, IBS may develop following an enteric viral, bacterial or parasitic infection[67,68] which causes an altered interaction between gut flora and the enteric nervous system such that despite resolution of the infection, symptoms of IBS may persist for years.[68] Also some patients with IBS have demonstrated increased gut permeability and low grade gastrointestinal inflammation reflected by increased fecal calprotectin concentrations.[69] Children with FGIDs including IBS tend to use less effective coping strategies to handle stress compared to healthy controls, which explains the association between depressive symptoms and many FGIDs.[70,71]

Few environmental factors have been linked to IBS and the role of genetics remains controversial. However, IBS tends to run in families, and mothers of children with IBS are more likely to have a lifetime history of IBS compared with controls.[72,73] Also the concordance for IBS in monozygotic twins is greater than in dizygotic twins, suggesting a genetic predisposition.[74]

Treatment options vary and have targeted possible causes of the disorder. Peppermint oil which relaxes intestinal smooth muscles by decreasing calcium influx into the cells has been studied in a double-blind, controlled trial in the pediatric population and was found to be effective in reducing pain during the

acute phase of IBS.[75] Several probiotics have been evaluated as treatments for IBS. In a double-blind placebo-controlled trial, lactobacillus GG successfully reduced pain frequency but not pain severity particularly among children with IBS.[76] In adults the benefits from probiotics have been shown to be species specific.[77,78] Further studies with different strains are needed in children to determine if certain species are more effective than others. Of note, the oral antibiotic rifaximin was shown to be effective in improving IBS symptoms in adults.[79]

There might be a role for serotonin agonists and antagonists, cholinergic agonists and antagonists, and antidepressants. Alosetron, a serotonin receptor antagonist, showed significant beneficial effect in women with diarrhea-predominant IBS. It decreases visceral sensitivity, reduces postprandial motility and slows left colon transit. It was approved in 2000 only to be taken off the market due to its association with life-threatening gastrointestinal adverse effects such as ischemic colitis. It was reintroduced in 2002 with restricted use. Tegaserod, a serotonin receptor agonist, was previously approved for the treatment of constipation-predominant IBS but was withdrawn from the market in March 2002 due to its association with some serious cardiovascular adverse events. Citalopram, a selective serotonin reuptake inhibitor, was shown to improve IBS symptoms in children[80] and amitriptyline, a tricyclic antidepressant which has anticholinergic as well as analgesic effects, significantly improved symptoms and overall quality of life in adolescents with diarrhea-predominant IBS.[81] Anticholinergic agents, dicyclomine and hyoscyamine, block muscarinic effects of acetylcholine on the gastrointestinal tract, relaxing smooth muscles, slowing intestinal motility and decreasing diarrhea. There are, however, no randomized, double-blind, placebo-controlled trials conducted in the pediatric population investigating their efficacy. Lubiprostone, a chloride channel activator, has been studied in constipation-predominant IBS adult patients and has demonstrated significant improvement in gastrointestinal symptoms at 1, 2, and 3 months follow up.[82]

The role of fiber in the management of IBS patients remains controversial. It has proven beneficial in one double-blind, placebo-controlled trial in children;[83] however, a meta-analysis done by Huertas-Ceballos and colleagues[84] showed a lack of high quality evidence for the effectiveness of dietary interventions in pediatric IBS.

Gut-directed hypnotherapy has proved to be highly effective in treating children with long standing IBS and was superior to standard medical therapy which included education, dietary changes and pharmacological interventions in decreasing pain scores.[85] Cognitive behavioral therapy has also been shown to be beneficial.[86]

Functional abdominal pain

Functional abdominal pain per the Rome III criteria is characterized by episodic or continuous abdominal pain at least once a week for a minimum of 2 months.[12] It is distinguished from FD by the location of the pain and lack of association with food intake, and from IBS by the absence of associated bowel symptoms. FAP which interferes with daily functioning or is accompanied by somatic symptoms including headaches, limb pain or difficulty in sleeping is labeled functional abdominal pain syndrome.[12]

The pain is often described as frequently recurring, alternating with pain free periods of variable length. Less than 10% of the patients report constant pain and it lasts less than 1 hour in 50% of the patients.[87] It is typically periumbilical in location without radiation; however, Shulman et al[47] observed that children with FAP can present with pain remote from the umbilicus. The severity of the pain is variable with some parents describing the child during the episode as miserable, doubling over, crying and grimacing.

As with IBS, children with FAP may have an underlying visceral hypersensitivity. Van Ginkel et al[64] and Faure and Wieckowska[65] demonstrated that children with IBS and FAP have rectal hypersensitivity as opposed to controls or patients with FD. Additionally, children with FAP tend to have a lot of somatic complaints as well as depressive symptoms.[31,80]

A limited evaluation of the child with FAP that includes a complete blood cell count, complete metabolic profile, erythrocyte sedimentation rate or C-reactive protein measurement, urinalysis, urine culture, and stool for occult blood is considered reasonable.[12] Further diagnostic tests including stool culture, stool examination for ova and parasites, and breath hydrogen testing for carbohydrate malabsorption can be considered in a child with diarrhea. Additional tests should only be done when indicated, based on the history and physical examination.

Caring for these children can be difficult for both parents and physicians. Many parents indeed pay more attention to their child during the episodes which then provides an opportunity for secondary gain for the child. Using the water load test, where the child drinks a volume of water that causes less intense but representative abdominal pain, Walker et al[88] evaluated the effect of parental distraction on symptoms. Children with abdominal pain were randomized into either a group where the parents attended to the child's

symptoms with apologies, reassurance and sympathy or a group where the parents tried to distract the child. Compared with a control group where no specific therapy instruction was given, symptoms nearly doubled in the "attention" group, but were reduced by half in the "distraction" group.

In addition to the pharmacological treatments which are similar to those aimed at relieving abdominal pain in IBS, a biopsychosocial approach should be employed in the treatment of children with FAP. It is important to identify and, if possible, reverse physical and psychological stress factors that may play a role in the onset, severity, exacerbation or maintenance of pain. In some children, an acute or chronic physical illness may be the trigger for functional pain, while in others, simple physiological phenomena such as intestinal or gastric distention or psychological stressors such as death of a family member, parental separation or even physical illness in a parent or sibling may be the trigger. In a randomized controlled trial of cognitive-behavioral family intervention programs for pediatric recurrent abdominal pain, children and parents participating in a combined standard medical therapy and cognitive behavioral therapy reported significantly less abdominal pain immediately following intervention and for up to one year following intervention.[89] Cognitive behavioral therapy including guided imagery and progressive relaxation also improved social functioning and school attendance.[90]

When conventional therapies are not effective, some parents seek help from alternative sources. Almost 40% of parents of pediatric patients with a gastrointestinal disorder are turning to complimentary and alternative medicines for their children, even though evidence for the effectiveness of such treatment modalities in children is lacking.[91]

Abdominal migraines

Abdominal migraine is defined as paroxysmal episodes of intense, acute periumbilical pain that lasts for 1 hour to days separated by asymptomatic periods lasting weeks to months.[12] These episodes are often accompanied by nausea, vomiting, anorexia, photophobia, pallor or headaches. A history of two episodes occurring in the last 12 months is required to make the diagnosis.[12] The abdominal pain is often severe enough to awaken the child and interfere with normal activities while the vomiting can be intense (median of 6 times/hour at peak) and the nausea disabling.[92] Episodes are usually triggered by psychological or physical stress.

Abdominal migraine affects 1%-4% of children and is more common in girls than boys, with a mean age of onset at 7 years and a peak at 10-12 years.[12] It has been suggested that abdominal migraine, cyclic vomiting syndrome, and migraine headaches make up a continuum of a single disorder.[92] The etiology and pathogenesis remain unknown but postulated mechanisms include episodic dysautonomia, visual-evoked responses, mitochondrial DNA mutations that cause deficits in cellular energy production, and heightened hypothalamic stress response that activates the emetic response.[93-96] A family history of migraines is very common. A recent study confirmed that the mothers and grandmothers of patients with abdominal migraines have twice the prevalence of migraine headaches compared with controls.[97]

Healthcare providers experienced in managing patients with abdominal migraine may be confident-enough based on a classic history to treat without performing an extensive initial evaluation. It is, however, recommended that in all children with cyclic abdominal pain and vomiting a basic metabolic panel and an upper gastrointestinal radiography to exclude biochemical and anatomic abnormalities be performed.[98] Olson and Li[99] demonstrated that an upper gastrointestinal radiography along with empiric migraine therapy is the most cost-effective strategy to manage abdominal migraine.

Sixty-eight percent of patients with abdominal migraines can identify specific triggers for their symptoms. These are often infections (41%) or psychological stress (34%).[92] Advising patients to avoid precipitating events, therefore, is one way of managing these patients. Pharmacological therapy utilizes a dual approach of abortive and preventive therapy depending on the severity of the symptoms. Prophylactic therapy includes amitriptyline, cyproheptadine, phenobarbital or propranolol. These medications have been shown to reduce the frequency as well as severity of episodes in children.[100,101] Abortive therapy includes sumatriptan, a serotonin (5HT1) receptor agonist, which substantially decreases frequency, duration, and intensity of attacks in children[102] and ondansetron, a serotonin (5HT3) receptor antagonist, which has a 76% efficacy rate in reducing the severity of vomiting.[103] Abortive therapy is used when patients breakthrough prophylactic therapy or when attacks are not frequent enough to justify prophylactic therapy.

General counseling

Although there are different FGID subtypes with different pathophysiologies and pharmacological interventions, there are definitely some general considerations that are shared. FGIDs can be debilitating for the child as well as the entire family. It

is, therefore, imperative that the physician emphasizes the positive diagnosis of the FGIDs and acknowledges that the pain is genuine. The healthcare provider should explain the nature and the pathophysiology of the FGIDs to the parents and children and reassure them by reviewing available data such as normal physical examination, normal growth and development as well as normal screening studies. The patient and family should understand that the treatment should focus less on the relief of pain and more on return to full activity. It is often overwhelming to the child to return to school because of the amount of make-up school work that they need to cover. Hence, a plan should be formulated by the parents and the school to help the child tackle this in a manageable manner. Referral to a child psychiatrist or psychologist is indicated when there is concern about maladaptive family coping mechanisms or when all attempts fail to get the patient to return to a normalized lifestyle. It can be proposed to the family as part of a multidisciplinary approach.

Prognosis

Very little is known about the natural history of FGIDs. In adults, the prevalence of IBS and FD are stable over time, but with considerable turnover in symptom status.[104] Patients who are diagnosed with functional abdominal pain rarely end up having an organic disorder on subsequent follow-ups.[105] Perquin and his colleagues[106] reported that 30%-45% of their patients with chronic abdominal pain continued to experience pain after 2 years of follow-up but its impact on the child's behavior, social functioning or use of health care decreased. Hyams et al[40] showed that up to 70% of children with dyspepsia were asymptomatic or much improved within 2 years of diagnosis. Other studies, however, found that 30%-50% of children with chronic functional abdominal pain experienced pain as adults although the pain limited normal activity in only 30%.[107,108] Moreover, patients are more likely than controls to experience anxiety, somatization, hypochondriasis and social dysfunction as adults.[108] Studies have shown that severe symptoms at presentation as well as improvement in symptoms at 3 months were associated with better prognosis, while psychological factors and low pain tolerance were poor prognostic indicators.[109,110]

Conclusion

Chronic abdominal pain of childhood remains a very challenging clinical entity to the general pediatrician as well as the specialist. The differential diagnosis is extensive and there are no confirmatory laboratory markers. However with help from the Rome criteria, a detailed history and thorough physical examination in combination with limited laboratory and imaging studies when indicated should lead to the diagnosis.

FGIDs are a cause of great anxiety, distress and morbidity in children as well as adults. As our understanding of these conditions improves, our therapeutic interventions will progress to, not only attempt to overcome them, but also to intervene early in the disease course so as to limit long-term impact. Pediatricians should be aware of potentially modifiable childhood risk factors and should offer these children and their families a therapeutic option that takes into account the physiological as well as the psychological elements of the disease.

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