Review of Abdominal Migraine in Children

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Abstract: Abdominal migraine is a type of functional abdominal pain disorder that affects 0.2% to 4.1% of children. It consists of paroxysmal, recurrent, and acute abdominal pain attacks with associated symptoms, including pallor, nausea, vomiting, anorexia, headache, and photophobia. In between episodes, patients return to their baseline health. Abdominal migraine is a clinical diagnosis. Its diagnostic criteria are outlined under the Rome IV criteria and the International Classification of Headache Disorders III criteria. Hypothesized contributors to its pathophysiology include a combination of visceral hypersensitivity, gut-brain enteric nervous system alterations, and psychological factors. Treatment is focused on preventive measures and mostly includes nonpharmacologic approaches. Possible pharmacologic treatments include abortive medications used for migraine headaches such as analgesics and antiemetics. Abdominal migraine is likely underdiagnosed and is poorly understood. Individuals who have abdominal migraine report a lower quality of life, rendering it an important diagnosis. The aim of this article is to review the epidemiology, clinical presentation, pathophysiology, diagnosis, and treatment of abdominal migraine in children.

In the early 1800s, abdominal migraine was thought to be part of cyclic vomiting syndrome, rather than its own entity.¹ It was not until 1922 that the term abdominal migraine was used by Brams, who initially described the condition in 3 adult patients.² He defined it as epigastric attacks lasting for a few days with associated nausea, vomiting, possible occasional diarrhea, and normal physical examination findings. The patient needed to have a personal or family history of migraine headaches. In 1933, Wyllie and Schlesinger reported children having a recurrent abdominal pain syndrome that was associated with migraine headaches.³ Currently, abdominal migraines are a subtype of functional abdominal pain disorders, conditions that are diagnosed clinically by their symptomatology.
2 peaks is that around the age of 5 years, children enter school, which may be a stressful experience for them. Stress has been noted to be a risk factor for abdominal migraine.10

Clinical Presentation

Abdominal migraine is characterized by paroxysmal, recurrent, acute-onset attacks of midline abdominal pain lasting an average of 17 hours.11 These attacks have been reported to range between 2 and 72 hours in duration.6 The pain is typically described as dull and periumbilical, but has also been reported as colicky and diffuse. An estimated 93% to 100% of patients have associated pallor, 91% have anorexia, 73% to 91% have nausea, and 35% to 50% experience vomiting.12 The end of the attack typically occurs abruptly.6 In patients who experience abdominal migraine, the average number of attacks per year is 14.11

Abdominal migraine attacks are typically sporadic, but a nonspecific prodrome may precede an attack. This can include changes in mood and behavior, anorexia, flushing, diarrhea, and auras involving flashing lights, visual changes, slurred speech, tingling in distal extremities, and numbness.6 The period between attacks can range from weeks to months, and patients typically do not have symptoms during this time.13

There are common triggers that provoke abdominal migraine attacks. These include stressors both in school and family life; poor sleep and irregular sleep habits; periods of prolonged fasting and food deprivation; dehydration; travel; exercise; high-amine foods; foods with additive flavoring, coloring, and monosodium glutamate (MSG); and flashing lights (Table 1).6,13 Examples of commonly ingested high-amine foods include citrus fruits, chocolate, cheese, vegetables such as eggplant and mushroom, and meats such as salami and ham.14 Sleep and rest have been reported to be relieving factors.10

Pathophysiology

There are numerous possible contributors to the pathogenesis of functional gastrointestinal disorders (FGIDs), but the cause of abdominal migraine remains unclear. The intricate brain-gut interaction seems to be disrupted in patients who have FGIDs.15 Although not specific to abdominal migraine, the following sections include brief overviews of various hypotheses that have been developed regarding the pathophysiology of functional abdominal pain disorders.

Visceral Hypersensitivity

The most accepted hypothesis is that FGIDs are a result of visceral hypersensitivity of primary sensory and central

Table 1. Common Triggers of Abdominal Migraine6,13

<table>
<thead>
<tr>
<th>Trigger</th>
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<tr>
<td>Stress</td>
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<tr>
<td>Sleep deprivation and irregular sleep habits</td>
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<tr>
<td>Food deprivation and prolonged fasting</td>
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<tr>
<td>Dehydration</td>
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<tr>
<td>Travel</td>
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<tr>
<td>Exercise</td>
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<tr>
<td>High-amine foods</td>
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<tr>
<td>Additive flavoring, coloring, and MSG in food</td>
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<td>Flashing lights</td>
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spinal neurons. Although not fully understood, it is thought that genetic, psychosocial, and environmental factors may all predispose an individual to visceral hyperalgesia. The brain-gut axis abnormally secretes excitatory neurotransmitters due to sensitization of these neurons. Serotonin, in particular, affects gastrointestinal sensation, which may contribute to abdominal pain attacks. Serotonin also affects motility and secretion of the gastrointestinal system. In patients who have FGIDs, there have been reported changes in visceral pain pathways on functional magnetic resonance imaging.2

Gut-Brain Enteric Nervous System Alterations

The pathogenesis of abdominal migraine may be associated with altered gut motility. In a study conducted in Sri Lanka between 2007 and 2012, 17 children between the ages of 4 and 12 years with the diagnosis of abdominal migraine were studied during periods of abdominal pain and were found to have significantly lower gastric emptying times and antral motility.19 Patients who had more severe symptoms were found to have lower-amplitude antral contractions, suggesting that slower gut motility may contribute to the pathogenesis of abdominal migraine. However, any painful stimuli, nausea of central origin, and several endocrine changes can all result in observed changes in gastric emptying and antral motility. Thus, it is unclear if the findings suggest a role in causation or signify a secondary effect due to another underlying etiology. Although not specific to the diagnosis of abdominal migraine, other studies have suggested that pediatric patients who have functional dyspepsia have abnormal gastric emptying.19,20 Because abdominal migraine is a clinical diagnosis based on symptoms following the Rome IV criteria, there is significant overlap between abdominal migraine and other FGIDs. In addition, although specific changes in the brain-gut enteric nervous system in patients with abdominal migraine have not yet been demonstrated, they have been shown to occur in patients experiencing abdominal pain due to other causes (eg, irritable bowel syndrome, inflammatory bowel disease). One such change involves alterations in levels of neuropeptides.21

Psychological Factors

Psychological factors may play a role in the pathophysiology of abdominal migraine. For example, risk factors for recurrent abdominal pain include abuse and stressful events.22 Additionally, children who have FGIDs have been shown to have mental illnesses such as anxiety and depression more frequently than children who do not have FGIDs.22 Although not clearly understood, increased stress has been reported to be correlated with more sensitive visceral perception.23 Increased stress induces central nervous system arousal, causing neurotransmitters and neuropeptides to be released, which then contribute to dysregulation of the gastrointestinal system. It may be that stress predisposes an individual to alterations in the hypothalamic-pituitary-adrenal axis.24

Diagnostic Evaluation

It is important to exclude the presence of organic disease causing a child’s symptoms while considering the possibility of the patient having a functional condition.7 A thorough history and physical examination are of particular importance when diagnosing a patient with abdominal migraine. Careful evaluation for any alarm signs and symptoms should also occur. Some of these include weight loss, changes in growth pattern, fever, right iliac fossa pain, hematemesis, bilious vomiting, chronic diarrhea, the presence of visible or occult blood in the stool, and dysphagia.7 If these alarm symptoms and/or signs are evident, additional imaging and testing should be performed.25

Under the Rome IV criteria (Table 2), the diagnosis of abdominal migraine can be made if at least 2 episodes of the following occur over a 6-month period: paroxysmal bouts of intense, acute periumbilical, midline, or diffuse abdominal pain occur for at least 1 hour, have a recurring pattern, are incapacitating, and interfere with normal activities. In between attacks, there should be weeks to months of no symptoms. At least 2 of the following associated symptoms must be included: anorexia, nausea, vomiting, headache, photophobia, and pallor. Finally, the symptoms cannot be attributed to another medical diagnosis.26 The ICHD III criteria classify abdominal migraine as 5 or more abdominal pain attacks that last between 2 and 72 hours (Table 2). Additionally, the pain must fulfill at least 2 of 3 of the following characteristics: dull or “just sore” quality; midline location, periumbilical, or poorly localized; and moderate or severe intensity. The patient should have at least 2 of these associated symptoms: anorexia, nausea, vomiting, and pallor.12

There are several key differences between these 2 criteria. The Rome IV criteria emphasize the need for attacks to have a stereotypical pattern and the pain to interfere with normal activities. These criteria also include a period of 6 months during which at least 2 abdominal pain attacks occur, whereas the ICHD III criteria include at least 5 attacks of abdominal pain without specifying the period of time during which these episodes need to occur. Under the Rome IV criteria, an abdominal migraine episode must last at least an hour, whereas under the ICHD III criteria, it must last at least 2 hours.

Overall, meeting the Rome IV or ICHD III criteria and lacking alarm signs and symptoms are sufficient to
make a provisional diagnosis of abdominal migraine. Therapy for abdominal migraine can be initiated at that time, rather than performing extensive testing to evaluate for the presence of other potential disorders.

**Relationship Between Abdominal Migraine and Migraine Headache**

An association between abdominal migraine and migraine headache has been reported. Abdominal migraine is considered a migraine equivalent because it constitutes a recurrent, paroxysmal syndrome that seems to have an associated etiology to that of migraine headache. Therefore, the diagnosis of abdominal migraine has been included under the ICHD III criteria. There seem to be similar triggers, signs and symptoms, relieving factors, and treatments as well as possibly a similar pathogenesis for abdominal migraine and migraine headache. In a study that reviewed 150 cases of abdominal migraine in children over a 10-year period, 90% of cases had a family history of migraine headache in a first-degree relative. In a retrospective study of children

<table>
<thead>
<tr>
<th>Rome IV Criteria</th>
<th>ICHD III Criteria</th>
<th>Rome IV Criteria</th>
<th>ICHD III Criteria for Migraine Without Aura</th>
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<tbody>
<tr>
<td>≥2 episodes over a 6-month period of all of the following:</td>
<td>A. ≥5 attacks of abdominal pain fulfilling criteria B-D</td>
<td>Rome IV Criteria</td>
<td>In infants and children ≤4 years, criteria must include:</td>
</tr>
<tr>
<td>• Paroxysmal episodes of intense, acute periumbilical, midline, or diffuse abdominal pain lasting ≥1 hour</td>
<td>B. Pain has ≥2 of the following 3 characteristics:</td>
<td>• ≥2 episodes over 6 months of unrelenting, paroxysmal vomiting lasting hours to days ± retching</td>
<td>A. ≥5 attacks fulfilling criteria B-D</td>
</tr>
<tr>
<td>• Periods of baseline health lasting weeks to months between episodes</td>
<td>• Midline location, periumbilical, or poorly localized</td>
<td>• Stereotypical pattern specific to infant or child</td>
<td>B. Headache attacks lasting 2-72 hours</td>
</tr>
<tr>
<td>• Pain is incapacitating and interferes with normal activities</td>
<td>• Dull or “just sore” quality</td>
<td>• Periods of baseline health lasting weeks to months between episodes</td>
<td>C. Headache has ≥2 of the following 4 characteristics:</td>
</tr>
<tr>
<td>• Stereotypical pattern and symptoms specific to child</td>
<td>• Moderate or severe intensity</td>
<td>• Insufficient criteria for IBS, functional dyspepsia, or abdominal migraine</td>
<td>• Unilateral location</td>
</tr>
<tr>
<td>• Pain associated with ≥2 of the following:</td>
<td>C. ≥2 of the following 4 associated symptoms or signs:</td>
<td>• After appropriate evaluation, the abdominal pain cannot be fully explained by another medical condition</td>
<td>• Pulsating quality</td>
</tr>
<tr>
<td>– Anorexia</td>
<td>• Anorexia</td>
<td>• Treatments as well as possibly a similar pathogenesis for abdominal migraine and migraine headache. In a study that reviewed 150 cases of abdominal migraine in children over a 10-year period, 90% of cases had a family history of migraine headache in a first-degree relative. In a retrospective study of children</td>
<td></td>
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<tr>
<td>– Nausea</td>
<td>• Nausea</td>
<td>• Aggravation by, or causing avoidance of, routine physical activity</td>
<td>• Vomiting</td>
</tr>
<tr>
<td>– Vomiting</td>
<td>• Vomiting</td>
<td>• Symptoms cannot be attributed to another cause after appropriate medical evaluation</td>
<td>• Photophobia</td>
</tr>
<tr>
<td>– Headache</td>
<td>• Pallor</td>
<td>E. Not better accounted for by another ICHD III diagnosis</td>
<td>• Photophobia and phonophobia</td>
</tr>
<tr>
<td>– Photophobia</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>– Pallor</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>• After appropriate investigation, symptoms cannot be fully explained by another medical condition</td>
<td>D. During headache, ≥1 of the following occurs:</td>
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<tr>
<td></td>
<td>• Nausea and/or vomiting</td>
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<tr>
<td></td>
<td>• Complete freedom from symptoms between attacks</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Not attributed to another disorder</td>
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IBS, irritable bowel syndrome; ICHD, International Classification of Headache Disorders; NOS, not otherwise specified.
in an ambulatory pediatric neurology practice who had either abdominal migraine or cyclic vomiting syndrome, 70% of patients had coexisting typical migraine headache and 65% of patients reported a family history of migraine headache.27

The features of abdominal migraine and migraine headache were compared by Abu-Arafeh and Russell in a study of 1754 children between the ages of 5 and 15 years.16 Inclusion criteria for the study were 2 or more episodes of severe abdominal pain of unknown etiology, or headaches that parents thought were migraine headaches. Of the study participants, 10.6% were diagnosed with migraine headache by the International Headache Society criteria, and 4.1% were diagnosed with abdominal migraine. These 2 groups were then compared in terms of clinical presentation and risk factors. The investigators found that common precipitants in both groups were stress, travel, and fatigue, whereas common relieving factors were eating and resting. Both groups of children had similar clinical presentations and associated symptoms, such as nausea, vomiting, and pallor. Additionally, 24% of patients who had abdominal migraine had migraine headache, as compared to a 10% prevalence of migraine headache in the general population. These findings suggest that there may be a similar pathogenesis for abdominal migraine and migraine headache.

The preceding findings may be explained by the bi-directional communication between neurons in the brain and gut.8 The autonomic nervous system, specifically the parasympathetic and sympathetic efferent neurons, is connected to the enteric nervous system.29 The enteric nervous system innervates visceral smooth muscle and end-organ structures that alter the secretory, endocrine, immune, and motor functions of the gastrointestinal tract. Signals from the gastrointestinal tract are sent through spinal and vagal afferent nerves to the brain stem and sensorimotor brain circuits. Enteroendocrine cells in the gut send various peptide hormones that contribute to gastrointestinal motility, sensitivity, and metabolism to the brain stem and hypothalamus via vagal afferent neurons. Thus, there is a complex, interconnected system between the brain and the gastrointestinal system.

Distinguishing Abdominal Migraine From Similar Diagnoses

As discussed, there is clinical overlap between abdominal migraine and migraine headache, and this is further complicated by overlap with cyclic vomiting syndrome and functional abdominal pain not otherwise specified. Although they are considered separate entities with their own diagnostic criteria, it is often difficult to distinguish them (Table 2). Abdominal migraine, cyclic vomiting syndrome, and migraine headache all share common associated symptoms, such as pallor, anorexia, nausea, and photophobia.30 Therefore, a detailed history is crucial in differentiating these diagnoses.

When comparing abdominal migraine and functional abdominal pain not otherwise specified, abdominal migraine has a notably stereotypical pattern, whereas functional abdominal pain not otherwise specified does not.5 Abdominal migraine also involves more associated symptoms, such as anorexia, nausea, vomiting, headache, photophobia, and pallor, as described by both the Rome IV and ICHD III criteria.12 Moreover, patients with abdominal migraine return to their baseline health between episodes. Finally, pain associated with abdominal migraine is typically more incapacitating than that associated with functional abdominal pain not otherwise specified, as described under the Rome IV criteria. It is important to recognize that the diagnosis of abdominal migraine does not exclude the presence of other functional abdominal pain disorders.

Although a patient with suspected cyclic vomiting syndrome may often have associated abdominal pain, the hallmark feature is typically rapid, persistent vomiting with nausea.12,30 Cyclic vomiting syndrome involves a stereotypical pattern of at least 2 episodes over 6 months of unremitting, paroxysmal vomiting lasting hours to days. As with abdominal migraine, patients with cyclic vomiting syndrome have periods in which they return to their baseline health between episodes, which can last weeks to months at a time. In addition to abdominal migraine being associated with migraine headache, cyclic vomiting syndrome has also been reported to be associated with migraine headache.

There have been attempts to further define these overlapping conditions. Abu-Arafeh and Russell suggest using exclusion criteria, which are specific symptoms that exclude abdominal migraine as a potential diagnosis.10 These include predominantly nonmidline abdominal pain, pain lasting less than 1 hour, burning discomfort, the presence of symptoms in between attacks, lack of interference with daily activities, and symptoms that are attributed to another gastrointestinal disease or a food allergy.14 The authors also recommend long-term follow-up to monitor for any other causes of recurrent abdominal pain.

Li and Balint suggest an emphasis on the core symptom to help dictate the diagnosis when comparing abdominal migraine, cyclic vomiting syndrome, and migraine headache.30 Of note, they state that the index symptom takes place in 100% of children who have the specific disorder. For instance, vomiting occurs in all children with cyclic vomiting syndrome. If both abdominal pain and vomiting have occurred, the authors recommend
using the predominant symptom to make the correct diagnosis. They have also offered 3 supportive criteria to further reinforce a diagnosis of abdominal migraine: negative screening diagnostic tests that exclude common gastrointestinal, hepatobiliary, and renal disorders; the development of migraine headache; and a response to antimigraine medications.

We propose that in order to distinguish these entities, a clinician should rely on a thorough history to capture a clear picture of the most distressing symptom and additional associated symptoms, the timing of symptoms, and the patient’s status in between episodes. We agree that identifying the core symptom is paramount to making the correct diagnosis, and the use of exclusion criteria when making the diagnosis of abdominal migraine is also useful.

**Table 3. Pharmacologic and Nonpharmacologic Treatment Approaches for Abdominal Migraine**

<table>
<thead>
<tr>
<th>Nonpharmacologic Treatment: STRESS Mnemonic</th>
<th>Preventive Pharmacologic Treatment</th>
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<tbody>
<tr>
<td>• S: stress management ± CBT</td>
<td>• Propranolol: 10-20 mg BID or TID</td>
</tr>
<tr>
<td>• T: travel tips</td>
<td>• Cyproheptadine: 0.25-0.50 mg/kg daily, syrup</td>
</tr>
<tr>
<td>• R: rest and adequate sleep hygiene</td>
<td>• Flunarizine: 5.0-7.5 mg/day</td>
</tr>
<tr>
<td>• E: look out for emergency symptoms</td>
<td>• Pizotifen: 0.25 mg BID, syrup</td>
</tr>
<tr>
<td>• S: avoid sparkling and flashing lights; rest in dark, quiet room</td>
<td><strong>Abortive Pharmacologic Treatment</strong></td>
</tr>
<tr>
<td>• S: snacking—avoid prolonged fasting and high-amine foods</td>
<td>• Analgesics: Ibuprofen 10 mg/kg, acetaminophen 15 mg/kg</td>
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<td></td>
<td>• Sumatriptan: 10 mg intranasal</td>
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</table>

**Treatment**

Only a few small studies have evaluated treatment approaches for abdominal migraine. Most treatment approaches are anecdotal and are adapted from approaches known to be beneficial for migraine headache. These include pharmacologic and nonpharmacologic approaches (Table 3). There are no medications approved by the US Food and Drug Administration for the treatment of abdominal migraine. The focus of treatment is typically on the prevention of attacks, which often involves nonpharmacologic intervention. An essential aspect of management is offering reassurance and educating family members about the diagnosis. It is important to attempt to identify triggers and encourage behavioral changes that may help resolve an attack or prevent one from occurring.

**Nonpharmacologic Interventions**

A helpful tool that physicians can use when caring for abdominal migraine patients is the STRESS mnemonic: stress management, travel tips, rest, emergency symptoms, sparkling lights, and snacks to avoid. Physicians should have open dialogue with patients and family members regarding coping mechanisms to deal with stress. In addition, for patients who have recurrent abdominal pain, cognitive behavioral therapy may be useful. Travel tips include finding ways to avoid motion sickness, such as limiting long motor-vehicle travel without adequate stops and avoiding high altitude. Adequate rest is essential for patients. Good sleep hygiene with a regular bedtime routine should be reviewed, as sleep disturbances can provoke symptoms. Alarm symptoms, as discussed earlier, should be reviewed with parents so that they are prepared to seek medical attention if patients develop any of these symptoms. Moreover, visual disturbances, such as sparkling and flashing lights, should be avoided. Finally, changes in diet may also help reduce symptoms. Parents should be encouraged to identify any foods that seem to trigger their child’s abdominal migraine attacks and remove these foods from the child’s diet. Patients should also avoid ingesting foods containing MSG and additive flavorings and colorings.

In a study conducted by Russell and colleagues, 22 patients with recurrent abdominal pain underwent a trial of an oligoantigenic diet, which is a diet involving the elimination of foods that may cause an allergic response. The diet typically consists of only a few foods, such as certain fruits, vegetables, rice, and meat. This was found to improve symptoms in 77% of patients. Probiotics have been shown to be effective in reducing pain frequency and pain intensity in patients with functional abdominal pain disorders, specifically functional dyspepsia and irritable bowel syndrome, but there have been no studies...
evaluating the efficacy of probiotics in the treatment of abdominal migraine patients. Finally, for abortive treatment of abdominal migraine attacks, encouraging the patient to rest in a dark and quiet environment until symptoms resolve is effective.

Preventive Pharmacologic Interventions
In one study, pizotifen syrup (0.25 mg twice daily), a serotonin and histamine antagonist, was given to 14 children with abdominal migraine. Seventy percent of the patients reported improved symptoms over a 4-month period. They had significantly fewer and less severe symptoms. In a retrospective review, both propranolol (10-20 mg 2 or 3 times daily) and cyproheptadine syrup (0.25-0.50 mg/kg daily) were found to improve symptoms. In another retrospective review, flunarizine (5.0-7.5 mg/day) use decreased the duration and frequency of abdominal migraine attacks.

Abortive Pharmacologic Interventions
For acute abdominal migraine attacks, analgesics, triptans, and antiemetics may be useful. It has been proposed that nasal sprays or suppositories may be better suited for patients who have nausea, vomiting, or anorexia. Ibuprofen (10 mg/kg) and acetaminophen (15 mg/kg) have been reported to resolve acute attacks. In a case report, intranasal sumatriptan (10 mg) improved abdominal migraine attacks in 5 of 6 pediatric patients. Intravenous sodium valproate (500 mg 3 times daily) has been reported to resolve episodes of abdominal migraine in refractory cases. A case series reported that the use of intravenous dihydroergotamine (0.5 mg with a mean total of 7.0-9.0 mg total over approximately 2-3 days) improved abdominal migraine attacks in 5 of 6 pediatric patients. The first dose was typically 0.5 mg, the second dose was 0.75 mg 8 hours later, and then 1 mg was given every 8 hours thereafter until the cumulative dose was approximately 9 mg. Although intravenous sodium valproate and dihydroergotamine have been used, they are typically only used in refractory cases because of their many potential negative side effects.

Potential Emerging Treatments
Newer approaches to treat migraine headache have not yet been evaluated for the management of abdominal migraine, but may hold promise. These may include non-invasive vagal nerve stimulation, which was shown to significantly reduce pain intensity in patients with migraine headache and serve as abortive therapy. Calcitonin gene-related peptide (CGRP) monoclonal antagonists have also been shown to be effective in the treatment of acute migraine. CGRP monoclonal antibodies targeting CGRP or its receptor have been developed and are currently being studied for the prevention of migraine headache. These may have potential in the treatment of abdominal migraine, but further research is needed.

Prognosis
There are limited studies regarding the long-term effects of abdominal migraine on children as they progress into adulthood. It has been proposed that perhaps abdominal migraine, cyclic vomiting syndrome, and migraine headache constitute a continuum of a single disorder, and that a patient may progress from one condition to another. In a study by Dignan and colleagues in which 54 patients with abdominal migraine were followed for 10 years, abdominal migraine continued into the late-teen years in 38%, but eventually resolved in 61% of patients. Of these patients, 70% developed migraine headache, whereas only 20% of patients in the control group developed migraine headache. Various authors have reported that children with abdominal migraine develop migraine headache in late childhood and adulthood. There have been reported cases of abdominal migraine in adults as well. These findings suggest that children with abdominal migraine may continue to experience it as they move into adulthood, and may develop migraine headaches. More longitudinal studies should be conducted.

Conclusion
Pediatric abdominal migraine is likely underdiagnosed. Community health resources are used both to treat symptoms and evaluate these patients. Seventy-two percent of patients state that their abdominal pain interferes with normal daily activities, making this diagnosis important to recognize and treat. Abdominal migraine often affects a child’s social and academic life, contributing to missed days of school. Chronic abdominal pain in children can be distressing and can result in a lower quality of life. Although abdominal migraine pain symptoms may resolve in adulthood, it has been reported that up to 70% of patients develop migraine headache as adults. Therefore, the effects of this diagnosis are not solely confined to the childhood period. Overall, there is still much to be understood regarding the diagnosis of abdominal migraine in children.

Disclosures
The authors have no relevant conflicts of interest to disclose.

References