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Review Article

Pediatric Migraine: Abortive Management in the Emergency Department

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Studies suggest that headache accounts for approximately 1% of pediatric emergency department (ED) visits. ED physicians must distinguish between primary headaches, such as a tension or migraine, and secondary headaches caused by systemic disease including neoplasm, infection, or intracranial hemorrhage. A recent study found that 40% of children presenting to the ED with headache were diagnosed with a primary headache, and 75% of these were migraine. Once the diagnosis of migraine has been made, the ED physician is faced with the challenge of determining appropriate abortive treatment. This review summarizes the most recent literature on pediatric migraine with an emphasis on diagnosis and abortive treatment in the ED.

Key words: migraine, pediatric, emergency department, abortive

Abbreviations: 5-HT 5-hydroxytryptamine (serotonin), AAN American Academy of Neurology, CGRP calcitonin gene-related peptide, CNS central nervous system, DHE dihydroergotamine, ED emergency department, FDA Food and Drug Administration, IHS International Headache Society, NSAIDs nonsteroidal anti-inflammatory agents

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Studies suggest that headache accounts for approximately 1% of pediatric emergency department (ED) visits.[1,2] ED physicians must distinguish between primary headaches, such as a tension or migraine, and secondary headaches caused by systemic disease including neoplasm, infection, or intracranial hemorrhage. A recent study found that 40% of children presenting to the ED with headache were diagnosed with a primary headache, and 75% of these were migraine.[3] Once the diagnosis of migraine has been made, the ED physician is faced with the challenge of determining appropriate abortive treatment. This review summarizes the most recent literature on pediatric migraine with an emphasis on diagnosis and abortive treatment in the ED.

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EPIDEMIOLOGY/PREVALENCE

Migraine headaches, though less common in children than adults, can begin in childhood, and increase in prevalence with age from as low as 3% in young children to 15% in adolescents.[4,5] The mean age of onset is earlier in boys than girls, starting at age 7 and 11, respectively. As a result, the incidence of migraine is higher in school-aged boys than girls, but becomes more common in adolescent girls than adolescent

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boys. The peak incidence of migraine in children occurs in adolescence around age 15.[6]

MIGRAINE PATHOPHYSIOLOGY

Migraine pathophysiology has mainly been researched in adults, but is thought to have similar mechanisms in the pediatric population, mechanisms which remain in question. Migraine is thought to have a neurovascular etiology and is associated with cranial vasodilation.[7] The primary dysfunction is believed to be related to centers in the brainstem that regulate vascular tone and pain sensation.[8] Vasoactive neuropeptides, including 5-hydroxytryptamine (serotonin) (5-HT) and calcitonin gene-related peptide (CGRP), are responsible for the vascular phenomena observed with migraine.[9-11] In particular, 5-HT has been shown to have an inhibitory role in the central nervous system (CNS) preventing the cascade of events that result in neurovascular vasodilation.[12] This explains the clinical efficacy of triptans as abortive migraine agents[13] because of their pharmacologic action as 5-HT agonists. In contrast to 5-HT, CGRP is a potent vasodilator in the CNS with an integral role in triggering migraine attacks. This has been demonstrated experimentally: migraine sufferers infused with human alpha-CGRP, as compared with those receiving placebo, developed a significant increase in frequency of acute migraine as well as pain.[14] Further evidence derives from studies demonstrating that antagonists of the CGRP peptide receptor are effective at aborting migraine headaches in adults.[15,16]

The pathophysiology of migraine is more complicated, however, than a neurovascular phenomena alone. Pain from migraine headaches is primarily carried by trigeminovascular pathways. The trigeminal sensory afferents that innervate meninges and large blood vessels become activated and undergo sensitization,[17] which then activates other neurons and centers of the brain associated with pain and meningeal inflammation. There appears to be a genetic component to migraines; dysregulation of cortical inhibition and excitation in migraine results in higher levels of excitation, causing pain.[18,19] This has been supported in mouse models of familial hemiplegic migraine that show increased glutamatergic neurotransmission, which activates nociception and causes of the characteristic premigrainous aura.[20-23]

Table 1.—Pediatric Migraine without Aura

	Criteria
I†	At least 5 attacks with features (II-IV) below
II	Headache between 1 and 48 hours
III	At least 2 of the following: • Bilateral or unilateral location‡ • Pulsating • Moderate-to-severe pain • Made worse with activity
IV	At least one associated symptom: • Nausea/Vomiting • Photophobia/Phonophobia

Adapted from Winner et al.[25]
†Recurrent attacks may not be required for ED diagnosis.[26]
‡Not to include posterior location.

DIAGNOSIS

The International Headache Society (IHS) has published diagnostic criteria for migraine headache.[24] However, these guidelines were developed for the adult population and migraine headache in children can present differently. For this reason, revised IHS diagnostic criteria have been proposed (Tables 1 and 2).[25] These revised criteria have significantly improved the diagnostic sensitivity of pediatric migraine and highlight the fact that migraines in children can be bilateral and the attack itself can be as short as 1 hour and as long as 48 hours.

The reliance on recurrent attacks as a diagnostic criterion using the IHS definition (Table 1) is limiting

Table 2.—Pediatric Migraine With Aura

	Criteria
I†	At least 2 attacks with features below
II	At least 3 of the following: • Gradual development of autonomic aura • Aura that is fully reversible • Aura is present less than 1 hour • Headache within 1 hour of aura

Adapted from Winner et al.[25]
†Recurrent attacks may not be required for ED diagnosis.[26]

in the emergency department (ED) setting as patients with new onset migraine headaches may present during their first attack. The Irma Criteria, which are the same as the IHS criteria, but eliminate the requirement for recurrent episodes, may be more sensitive in the ED, as suggested by a study by Trottier et al.[26] In this study, the authors found that 45% of pediatric patients clinically diagnosed with migraine were confirmed by a pediatric neurologist within 3 months to meet the IHS diagnostic criteria for migraine headache; however, when they applied new diagnostic criteria (the "Irma criteria"), 86% of this group would have fulfilled criteria in the ED.

The severity of headache pain may help to distinguish between migraine and tension headaches in children: one study suggests that severe headache is more characteristic of migraine whereas mild-to-moderate intensity is more likely related to tension-type headaches.[27] Descriptive characteristics such as pulsating quality may be difficult to elicit from young children, but become easier to describe in adolescents who can be asked directly. Other factors such as photophobia or phonophobia may be inferred from observation of patient behavior in response to light and noise; children who can't describe these symptoms often prefer a dark room and are visibly bothered by loud sounds.

Migraines can be divided into several subtypes. The two most common are migraine with and without aura. Children who experience migraine with aura may describe a variety of premigrainous phenomena, typically visual scotoma such as scintillating or shimmering lights, blind spots, or tunnel vision. Migraine without aura is by far the more common presentation accounting for 60-85% of all migraines.[4] Less common migraine syndromes include basilar-type migraine and familial hemiplegic migraine.

The diagnosis of pediatric migraine in the ED can be difficult, even with the IHS criteria described previously. Though recurrent symptoms over time are integral to the diagnostic criteria for migraine, children may present with their first attack to the ED, where it may be challenging to distinguish migraine from other primary or secondary headache disorders. Clues to secondary headaches include an occipital location of headache; morning emesis; focal neurologic deficits; papilledema; or other systemic symptoms or signs, such as fever or trauma.[28]

TREATMENT

The American Academy of Neurology (AAN) has endorsed clinical guidelines for abortive as well as prophylactic treatment of migraine headache.[29,30] A recent study done on pediatric headaches in the ED across the United States[31] showed a large amount of variability in diagnostics and treatment. One finding in particular was the high use of narcotics with almost a third of patients receiving these agents. Previous guidelines in children, as well as this review, advise against the use of these agents for management in the ED.[4,29-31] Our proposed pediatric migraine algorithm (Figure) in agreement with the literature review below highlights ED treatment and specifically omits the use of narcotics. Since the release of the AAN practice parameter in 2000, a number of individual trials, systematic reviews of specific therapeutic medications, and meta-analyses have been reported. The remainder of this review will focus on the pharmacologic treatment of migraine, with emphasis on abortive therapeutic options in the ED. A brief overview of prophylaxis is provided below, as patients may already be taking or have questions about these agents, and it is helpful for the ED provider to be familiar with these medications.

PROPHYLAXIS

The ED treatment is focused on abortive therapeutics. However, many patients present to the ED on prophylactic medications, and as such, it is useful for ED providers to know what these medications entail. The main goal of prophylactic therapy is to reduce the frequency of migraine attacks and improve the overall quality of life for recurrent migraine sufferers. Multiple classes of agents are available, including antiepileptics (topiramate, valproic acid, gabapentin), antidepressants (amitriptyline), antihistamines (cyproheptadine), and antihypertensive (nimodipine and propranolol).[32] In a study of 250 children with migraine headache in a single pediatric neurology practice, approximately 50% of patients were on prophylactic medication;[33] amitriptyline was the most

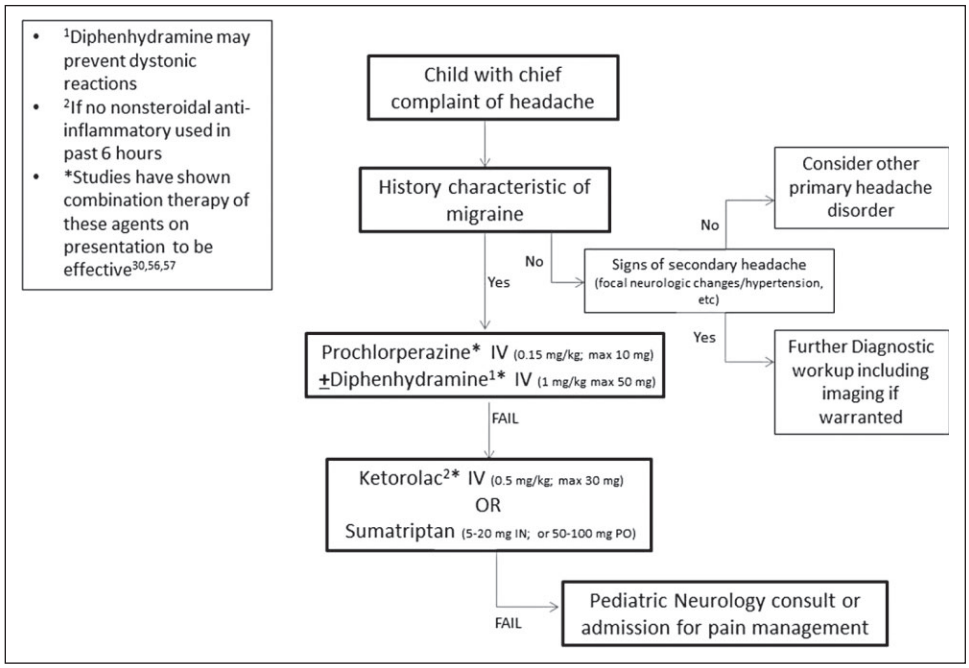

Figure.—Pediatric migraine emergency department (ED) abortive treatment algorithm. PO, oral; IV, intravenous; mg, milligram; kg, kilogram; IN, intranasal.

commonly prescribed agent, followed by cyproheptadine. In this study, 89% of patients taking amitriptyline and 83% of those taking cyproheptadine for migraine prophylaxis had a positive response during a 6-month follow-up period. Headache frequency was reduced by 62% for amitriptyline and 55% for cyproheptadine. This was a single practice and may not be representative of a national sample, but does highlight available options and the relative efficacy of these agents.

Nutraceuticals such as coenzyme Q10, riboflavin, magnesium, and Butterbur root have also been suggested as safe alternatives to pharmaceutical prophylaxis; however, high quality prospective trials are limited in children and adolescents, and have failed to demonstrate significant improvement beyond the high placebo response in controlled trials.[34-36]

ABORTIVE TREATMENT

Abortive therapy is the mainstay of treatment in the ED when a child presents with a moderate-to-severe migraine headache and is the focus of this review (Table 3). Despite the AAN clinical guidelines for migraine abortive therapy,[29,30] few robust clinical studies in the pediatric population exist, with even fewer based in the ED. The best-studied abortive medications fall into the following categories: nonsteroidal anti-inflammatory agents (NSAIDs), acetaminophen, 5-HT receptor agonists (triptans), dopamine receptor antagonists, and antihistamine agents. In addition, a few new therapies on the horizon warrant discussion.

NSAIDs/Acetaminophen (Table 4).—NSAIDs range from oral ibuprofen to parental ketorolac. A systematic review in 2005 evaluated the literature on oral ibuprofen and acetaminophen compared with placebo. This review concluded that there is moderate evidence that acetaminophen and ibuprofen are more effective at reducing headache symptoms at 1 and 2 hours than placebo; no increased adverse effects were noted. There was no evidence to suggest superiority of ibuprofen or acetaminophen individually.[37]

A more recent study conducted in an outpatient neurology clinic compared oral treatment with ibuprofen or zolmitriptan with placebo.[38] The primary outcome was a reduction in pain from moderate-to-severe to mild-or-none at 2 hours. Both medications were significantly better than placebo, and there was

Table 3.—Abortive Pediatric Migraine Medications

Medication	Dose: Route
NSAIDs	
Ibuprofen	➢ 10 mg/kg: PO (max: 800 mg/dose or 2400 mg/day)
Ketorolac	➢ 0.5 mg/kg: IV (max: 30 mg/dose)†
Dopamine Antagonist	
Prochlorperazine	➢ 0.15 mg/kg: IV (max: 10 mg/dose)
Metoclopramide	➢ 0.1 mg/kg: IV (max: 10 mg/dose)
Other Antiemetic	
Diphenhydramine	➢ 1 mg/kg: IV (max: 50 mg/dose)
Promethazine	➢ 0.25-1 mg/kg: IV (max: 25 mg/dose)
Triptans (†more available than listed below)	
Sumatriptan	➢ Multiple Routes: → 5-20 mg: IN → 50-100 mg: PO → 3-6 mg: SubQ
Rizatriptan	➢ 5-10 mg: PO
Sumatriptan/ Naproxen combination	➢ Multiple combinations → 85 mg/500 mg: PO (sumatriptan/naproxen) → 30 mg/180 mg: PO → 10 mg/60 mg: PO
Analgesics	
Acetaminophen	➢ 15 mg/kg: orally (max: 1 g/dose or 4 g/day)

†Can't be given within 6 hours of other NSAID use.
g = gram; IN = intranasal; IV = intravenous; kg = kilogram; mcg = microgram; mg = milligram; NSAID = nonsteroidal anti-inflammatory; PO = oral; SubQ = subcutaneous.

no significant difference between ibuprofen and zolmitriptan (69% vs 62% improved).

The parenteral NSAID ketorolac is frequently used to treat headache in the ED. Its efficacy has not been as well studied in the pediatric population as in adults. A recent systematic review of 8 adult trials found parenteral ketorolac safe and effective for the acute treatment of migraine in the ED.[39] The best study in the pediatric population was published in 2004, and compared parental ketorolac with prochlorperazine in a randomized, double-blind ED trial.[40] Sixty-six children received a normal saline bolus followed by randomization to ketorolac or prochlorperazine. *Efficacy* was defined as 50% or greater reduction in pain score at 1 hour. In this study, 84.8% of children who received prochlorperazine compared with 55.2% of children who received ketorolac demonstrated clinical improvement at 1 hour. Approximately 30% of children in each group reported recurrence of some headache symptoms at 48 hours phone follow up. The authors concluded that parental prochlorperazine was superior to parenteral ketorolac for acute pediatric migraine treatment in the ED.

Dopamine Antagonists (Table 5).—Dopamine antagonists are used frequently in the management of acute migraine with some central effect on migraine symptoms in addition to beneficial antiemetic effects, and are a mainstay of the ED treatment of adult migraine. A recent review of the adult literature emphasized the efficacy and safety of agents such as metoclopramide and prochlorperazine in this population.[41] Side effects of dopamine antagonists in children and adults include extrapyramidal symptoms such as akathisia and dystonic reactions, which can be treated with antihistamines, most commonly diphenhydramine. For this reason, many ED providers often combine dopamine antagonists with diphenhydramine when treating acute migraine.[42]

Fewer studies have evaluated the safety and efficacy of dopamine antagonists in the pediatric

Table 4.—Select NSAID Studies

Reference	Study Details	Pertinent Results
Damen et al, 2005[37]	Systematic review	• Ibuprofen and acetaminophen are more effective than placebo • Ibuprofen is comparable to acetaminophen
Evers et al, 2006[38]	Oral ibuprofen vs zolmitriptan vs placebo	• Ibuprofen and zolmitriptan are more effective than placebo • Ibuprofen is comparable to zolmitriptan
Brousseau et al, 2004[40]	Parental ketorolac vs parental prochlorperazine	• Prochlorperazine is more effective than ketorolac

Table 5.—Select Dopamine Antagonist Studies

Reference	Study Details	Pertinent Results
Brousseau et al, 2004[40]	Parental ketorolac vs parental prochlorperazine	• Prochlorperazine is more effective than ketorolac
Trottier et al, 2012[43]	Parental prochlorperazine and diphenhydramine effectiveness	• All patients had 50% reduction in their pain at time of discharge
Kabbouche et al, 2001[44]	Parental prochlorperazine effectiveness	• 90% of patients were free of pain within 24 hours of treatment

population. A 2012 study examined the effectiveness and side effects of prochlorperazine for migraine headache in children.[43] This prospective cohort study of pediatric ED patients used intravenous prochlorperazine in conjunction with intravenous diphenhydramine. Pain and the presence of akathisia were evaluated on presentation, 1 hour after treatment, and at the time of discharge. Outcomes of interest included a 50% reduction in pain at the time of discharge, percent of patients pain-free at discharge, and recurrence of pain on follow-up phone call. In this cohort, 100% of patients demonstrated a 50% reduction in pain at discharge, and half of the patients were completely pain-free at discharge. On follow-up phone call, approximately 65% of patients had a recurrence of their headache within 1 week of their ED visit. In addition, despite the use of diphenhydramine, 5% of patients were diagnosed with akathisia and approximately one third of patients were suspected of having possible akathisia based on review of notes, though not formally diagnosed or treated for symptoms.

A smaller pediatric study examined the efficacy of prochlorperazine in 20 children referred to an ED from a headache outpatient center.[44] This study assessed patients 1 and 3 hours after treatment with intravenous prochlorperazine (0.15 mg/kg) and intravenous fluids. At 1 hour after treatment, 90% of patients reported feeling better with a half of the patients reporting that they were pain-free. At 3 hours, 60% were pain-free. Patients were then contacted 24 hours after discharge, and 90% of patients reported no pain. However, of the patients who were not pain-free at discharge, 2 required admission and 1 required dihydroergotamine (DHE) therapy.

5-HT agonists (Triptans: Table 6).—Triptan agents have been shown to be effective in the treatment of acute migraine and are among the most studied agents for this purpose in the pediatric population. One benefit of the triptans is that they come in various preparations ranging from intranasal spray to oral tablets. Although they have shown effectiveness in outpatient clinics and the home setting, their use

Table 6.—Select Triptan Studies

Reference	Study Details	Pertinent Results
Eiland and Hunt, 2010[45]	Topic review	• Intranasal recommendations ➢ Sumatriptan and zolmitriptan • Oral recommendations ➢ Rizatriptan and almotriptan
Ho et al, 2012[47]	Oral rizatriptan vs placebo	• Rizatriptan is more effective than placebo
Hewitt et al, 2013[48]	Oral rizatriptan longitudinal study	• Rizatriptan is safe and effective ➢ 46% pain-free within 2 hours of medication
McDonald et al, 2011[49] Derosier et al, 2012[50]	Combination oral sumatriptan/naproxen vs placebo	• Sumatriptan/naproxen is more effective than placebo

may be limited in the ED if they have been used prior to presentation, as no more than 2 doses can be used in a 24-hour period. Although multiple triptans are now available, the best studied is sumatriptan. It should be noted that most triptan use in children is "off label" other than some Food and Drug Administration (FDA) approval in adolescents.

Intranasal Triptans.—Nasal formulations of sumatriptan and zolmitriptan are available. A recent review of triptans for abortive migraine treatment recommended these 2 therapies with data available on zolmitriptan in the pediatric population over 12 years of age and sumatriptan for patients 5 years and older.[45] The most common side effect reported with these nasal sprays is an unfavorable taste.

Oral Triptans.—Available oral triptan medications include sumatriptan, rizatriptan, zolmitriptan, naratriptan, almotriptan, eletriptan, and frovatriptan. Oral rizatriptan and almotriptan are recommended over all other available oral formulations.[45] Zolmitriptan has been studied in adolescents and has a shorter half-life in adolescents compared with adults.[46]

A recent study has compared oral rizatriptan with placebo among children 6-17 years of age.[47] The study used 5 mg (patients <40 kg) or 10 mg (patients >40 kg) of rizatriptan in a crossover design. The primary end-point was complete resolution of pain 2 hours after treatment. The end-point was achieved by 31% of patients who received rizatriptan compared with 22% of those who received placebo, which was statistically significant, but suggests limited clinical utility. The medication was well tolerated in the 6-17 age ranges in this study. A similar study in 2012 demonstrated the safety and tolerability of rizatriptan in a cohort of over 600 adolescents.[48] This study demonstrated safety for long-term acute treatment of migraine in adolescents with 46% of patients reporting complete pain relief 2 hours after their treatment.

Oral sumatriptan has recently been studied using a combination tablet with naproxen. One study in adolescents used a combination of 85 mg of sumatriptan with 500 mg of naproxen for abortive treatment of migraine at home, and examined subjects' headache diaries over a 6- to 12-month study period.[49] There were 602 subjects who recorded 8517 acute migraine attacks. Resolution of pain was reported by 24 hours in 71% of subjects who took the combination tablet, and 59% reported freedom from pain within 4 hours (42% within 2 hours). Most patients were satisfied with this treatment option and experienced improvement in quality of life.

A follow-up study compared a sumatriptan/naproxen combination tablet in 3 doses with placebo.[50] The formulations included 10/60 mg (sumatriptan/naproxen), 30/180 mg, and 85/500 mg. All patients' first migraine attack was treated with a placebo and those that continued to report pain 2 hours after placebo were enrolled in a 12-week trial of the combination tablet. The patient population consisted of 589 adolescents randomized to the various doses. The primary end-point of the study was percentage of patients pain-free after 2 hours. All doses of the combination tablet were significantly better at achieving the end-point compared with placebo. Interestingly, there was a trend toward decreasing effectiveness of higher doses with response rates of 29%, 27%, and 24% from the lowest to highest dose (vs 10% for placebo). However, better efficacy and lower recurrence was seen with the highest dose (85/500 mg) at later time points. All medications were well tolerated in this trial.

DHE.—Though widely used for the inpatient treatment of refractory pediatric migraine, data on the efficacy of DHE for acute abortive therapy in the ED are limited.[51] A single study of 12 children who had failed conventional treatment compared oral DHE (not available in the United States) with placebo using a crossover design.[52] Though 7 patients (60%) achieved at least a 2-point reduction in pain at 2 hours, using a 5-point scale compared with only 2 patients (16%) who received placebo, this result was not statistically significant.

Propofol.—Initial reports in the adult literature suggested that subanesthetic doses of propofol were effective for the abortive treatment of migraine headache in the clinic setting.[53-55] The pharmacokinetics of propofol, with its fast onset and offset of action, make it quite appealing for use in the ED. One case–control study in the pediatric ED examined subanesthetic doses of propofol to a "cocktail" of diphenhydramine, prochlorperazine, and ketorolac.[56] This study showed that, when used in subanesthetic doses (approxi-

mately 0.5 mg/kg given as a bolus and repeated every 15 minutes as needed), propofol was effective for abortive therapy of pediatric migraine. Patients treated with propofol had an 80% reduction in their pain from presentation to discharge compared with 60% in the case–control group, which was statistically significant. Furthermore, patients who received propofol were discharged from the ED almost 1.5 hours faster than controls (average length of stay after medication 122 minutes vs 203 minutes), although this did not reach statistical significance. No patients in this pediatric or previous adult studies experienced any serious adverse effects (respiratory depression or hypotension) at these doses of propofol. This pediatric study, however, was limited by a small patient population and nonrandomization. To date, there are approximately 150 patients in the combined adult studies that have been reported.[53-55] Although promising, larger trials are needed to either confirm or refute these findings.

Combination Therapy.—Many ED physicians utilize multiple medications administered as a "cocktail" for acute migraine treatment. A study of pediatric migraine treatment across 10 tertiary pediatric EDs in Canada found that many patients receive combination therapy with more than one agent as well as a large amount of variability in treatment of pediatric migraine across the 10 centers.[57] A similar study of practice variability in the United States confirmed variability in the ED management (as well as diagnostic evaluation) of pediatric migraine, with most patients receiving more than 1 abortive agent.[31]

The only pediatric study that examined a standardized "cocktail" treatment in the pediatric ED compared a combination of intravenous fluids, prochlorperazine, diphenhydramine, and ketorolac with "various migraine therapies based on attending practice preferences."[58] A retrospective chart review suggested that patients who were treated with the standardized combination therapy had significantly reduced headache pain scores, length of ED stay, and hospital admission rates.

HEADACHE RECURRENCE

The practical goal of abortive ED migraine treatment is to alleviate pain and facilitate discharge; however, headache recurrence can lead to further unscheduled medical visits. Preventing recurrence, therefore, is an important consideration for the treating physician.

In the adult population, steroids have been used to prevent headache recurrence, and a meta-analysis and systematic review of the efficacy of steroids for this purpose have been published. In the systematic review, 7 trials involving almost 750 patients who had received a single dose of dexamethasone in the ED to prevent recurrence were reviewed. The authors concluded that for every 100 patients treated with a single dose of dexamethasone in addition to standard migraine management in the ED, 10 would be expected to benefit by preventing recurrence of moderate to severe headache within 24-72 hours of ED treatment.[59] A second meta-analysis pooled data from 7 randomized trials that met their inclusion criteria.[60] The authors found that although dexamethasone offered no advantage over placebo for acute headache pain management, dexamethasone was more effective at reducing reported pain 72 hours after initial ED treatment, with a 26% relative reduction of recurrence and a number needed to treat of 9.

There are no randomized trials in pediatric ED of therapy to prevent recurrence. A single retrospective study of 187 children treated in the ED for status migrainosis, however, did attempt to characterize recurrence rates.[61] In this cohort, 11% of patients re-presented to the ED with migraine within a mean of 6.5 days from initial treatment. With the small number of recurrent visits, the authors could not identify any specific associations between the abortive or prophylactic medication administered in the ED and headache recurrence.

CONCLUSIONS

Though uncommon before age 7, migraine headaches become increasingly prevalent in adolescence, and acute headache is an important pediatric ED chief complaint. The diagnostic criteria for pediatric migraine differ from adult criteria in that children can present with bilateral headache of shorter duration than adults.

Recent advances in the understanding of the pathophysiology of migraine including the role of vascular and neuronal pain pathways has lead to the development of medications for both abortive and prophylactic therapy, and practice guidelines for the diagnosis and treatment of pediatric migraine have been published. NSAIDs and acetaminophen are well-established first-line abortive therapies. As opposed to adults, limited pediatric evidence and FDA indications support the potential effectiveness of some triptans and dopamine antagonists. Despite published guidelines, there is considerable variability across North America in the ED management of pediatric migraine. Emphasis should be placed on the recommendations against the use of narcotics for pediatric migraine, and ED physicians must take this into consideration. Important ED considerations include pharmacokinetic properties of potential abortive treatments in addition to safety and efficacy. New treatment options are promising but require validation through large prospective randomized trials.

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