Acute Confusional Migraine: Case series and brief review

Gascon GG[1], Coskun CJ[2], Brown WD[1]
Department of Clinical Neuroscience & Pediatrics, Brown University[1],
Neurology, Rhode Island Hospital[2], USA

ABSTRACT

Aim: *To review the clinical presentation and differential diagnosis of Acute Confusional Migraine and propose diagnostic criteria.* ***Methods And Materials:*** *The Pediatric Neurology Diagnostic Database of Hasbro Children's/Rhode Island Hospital was searched for diagnoses of migraine, complicated migraine syndromes, and Acute Confusional Migraine (ACM), for the 5 year period January 1, 1997 to December 31, 2001. Medical records of the ACM patients were then retrospectively reviewed for parameters of clinical presentation and ancillary diagnostic tests, and results compiled into tables. RESULTS: Total number of migraine patients seen during this time was 589. Total complicated migraine was 71 (12% of total). Acute Confusional Migraine numbered 13 (18% of complicated migraines); 6 males, 7 females. Ages ranged from 6 to 16 years, median 13. Follow-up ranged from 8.5 to 39, mean 20.7, months. All patients entered the hospital through the Emergency Room. ACM was the initial presentation (no preceding migraine headaches) in 8; 2 had recurrent episodes. EEGs recorded in 8 patients showed background delta slowing in various locations, usually posterior. Neuroimaging studies in 12 showed no obvious cause; 2 had incidental, unrelated findings. CSF studies in 8 were all normal.* ***Conclusion:*** *ACM occurs predominantly in late childhood and early adolescence, and is a diagnosis of exclusion. Attacks terminate with sleep, and are usually self-limited. No specific treatment is necessary. Inclusion and exclusion diagnostic criteria are proposed.* **(Int. J. Ch. Neuropsychiatry, 2005, 2(2): 189-194)**

INTRODUCTION

The first report of migraine presenting as an acute confusional state was published over 30 years ago[1]. In 1978 Ehyai and Fenichel[2] named this type of migraine "Acute Confusional Migrine" (ACM). Additional cases have since been reported by pediatric neurologists[2-10], and more recently by emergency room physicians[11-12]. However, it is still not widely recognized by pediatricians, family practitioners, psychiatrists and even neuropediatricians. We report a case series selected from 5 years experience in a university-affiliated academic teaching hospital, review the clinical presentation and short-term outcome, and propose diagnostic inclusion and exclusion criteria.

METHODS

From the computerized patient diagnosis database of the Division of Pediatric Neurology, Hasbro Children's/Rhode Island Hospital, we identified patients with a final diagnosis of acute confusional migraine seen between January 1, 1997 through December 31, 2001, and retrospectively reviewed their records. During this period two neurologists (GGG and WDB) saw all these patients. From the same database we identified patients with migraine with and without aura, and complicated migraine syndromes. The database included patients seen in resident clinics, faculty practice plan offices, satellite offices in surrounding communities, service inpatients, and inpatients seen as consultations on the pediatrics wards, the Pediatric Intensive Care Unit and the Emergency Room (ER).

RESULTS

We found 13 patients (Table 1), 6 males and 7 females, age range 6 to 16 years, median age 13 years, who had migraine presenting as an acute confusional state. Follow-up ranged from 8.5 to 39 months, mean 20.7 months. All presented initially to the Emergency Room, with the following initial ER diagnoses: change in mental status—8, encephalitis—2, question of drug overdose—2, and confusion—1. Five had previous migraine headaches, though not always diagnosed as such. For the other 8, ACM was the initial presentation of their migraine disorder. Over this 5 year period, 2 (15%) had recurrent episodes. No patients presenting with an initial ACM developed subsequent migraine, with or without aura, or other kinds of complicated migraine syndromes, such as basilar migraine, hemiplegia or ophthalmoplegic migraine, during the 2 to 3 year follow-up period.

The total number of migraine patients from 1997 to 2002 was 589; ACM comprised 2.2%. Total number of complicated migraine patients was 71, so ACM comprised 18% of all complicated migraine syndromes. The complicated migraine group (71) comprised 12% of the total migraine group.

Table (1) lists the qualitative factors during the confusional state. In 11 patients a complaint of some kind of headache could be elicited, often in retrospect after the attack was over, but was not the predominant presenting concern. In 4 patients headache did not occur until after the initial onset of disorientation and confusion. Vomiting occurred in 1 patient; aphasia was seen in 5.

EEGs were recorded on 8 patients during the ACM episode. The predominant abnormalities were background delta slowing in bilateral posterior quadrants in 4 patients and bilateral anterior and rhythmic slowing in 1. Delta slowing was lateralized to the left posterior quadrant in 2 and left temporal area in 1 (Table 2).

Neuroimaging was performed on 12 patients on presentation; 11 had noncontrast brain CT scans and 1 had a brain MRI. All neuroimaging studies were interpreted by a neuroradiologist. Brain CTs were normal, except one revealed a posterior fossa arachnoid cyst. The MRI revealed a small left parietal venous angioma (Table 2).

All 8 patients who had a lumbar puncture had normal CSF studies.

Table 1. Clinical parameters in 13 patients with Acute Confusional Migraine.

Patient Number	Sex	Age at Event (years)	History of Migraine	Initial Admitting Diagnosis	Recurrent episode	New Diagnosis of Migranous Disorder after ACM event	Follow-up (months)	Qualitative factors
1	M	16	N	Change in mental status	Y	N	27	Headache, disorientation, bizarre behavior
2	M	10	N	Overdose	N	N	29	Aphasia, lethargy, headache
3	F	6	Y	Encephalitis	N	N/A	39	Loss of consciousness, disorientation, aphasia, headache
4	F	13	Y	Change in mental status	N	N/A	32	Amnestic, lethargy, disorientation, headache 12 hrs later
5	F	13	Y	Change in mental status	N	N/A	23.5	Headache, disorientated, unsteady
6	M	16	Y	Change in mental status	N	N/A	21.5	Fluctuating hearing, light headed, lethargic
7	F	16	N	Change in mental status	N	N	21	Confusion, vomiting, headache 6hrs later
8	F	10	N	Encephalitis	N	N	17	Dizzy, lethargic, aphasic, headache 2hrs later
9	M	7	N	Change in mental status	N	Y	18	Disorientated, headache
10	M	12	N	Confusion	N	N	15	Aphasic, disorientated, headache 6hrs later, lethargic, repetitive
11	F	15	N	Change in mental status	N	N	8.5	Aphasia, disorientation, nausea, vomiting, headache, left body tingling
12	F	15	Y	Overdose	N	N/A	8.5	Slurred speech, lethargy, headache
13	M	11	N	Change in mental status	Y	N	9.5	Confusion, fluctuating arousal.

M= Male; F= Female; Y= Yes; N= No; N/A= Not applicable

Table 2. Ancillary neurodiagnostic tests in 13 patients with Acute Confusional Migraine.

Patient Number	EEG during attack	Imaging	Cerebrospinal Fluid
1	Bilateral slowing with lateralizing slowing of higher amplitude in right hemisphere.	MRI: left parietal venous angioma	W0, R378, P100, G72
2	Lateralized slowing in left posterior quadrant with occasional sharp forms in left occipital region.	CT NL	W0, R1, P34, G60
3	Left posterior quadrant slowing	CT NL	W0, R7, P26, G77
4	Occasional intermixed slow forms and possible sharp forms in both occipital quadrants	CT: posterior fossa arachnoid cyst	W1, R1, P25, G64
5	No	CT NL	NO LP
6	No	NO CT	NO LP
7	Left temp slowing and an occasional sharp forms in left temp region	CT NL	NO LP
8	No	CT NL	W0, R7, P16, G46
9	Bilateral posterior quadrant high voltage rhythmic delta activity	CT NL	W0, R0, P12, G63
10	No	CT NL	W0, R828, P49, G76
11	Bilateral anterior delta rhythmic slowing in wake/sleep	CT NL	NO CSF
12	Delta slowing, intermittent bilateral high voltage peaks delta waves, paradoxical arousal response	CT NL	W1, R693, P27, G70
13	No	CT NL	NO LP

W= White blood cells; R= Red blood cells; P= Protein; G= Glucose; NL= Normal

DISCUSSION

Headache and Acute Confusional Migraine. ACM occurs in either sex with an age of onset in late childhood to mid-adolescence[6,8,9,10]. It has been said that 50% of patients with ACM have a history of migraine[8], compared to 5 of 13, or 38%, in our series. The onset of headache in 12 of our 13 patients varied, from the beginning of the confusional episode to 12 hours after onset. The duration of attacks have been reported as short as 10 minutes, to many hours[2] and terminates with deep sleep. Upon awakening the patients have a normal mental status, but with spotty recall of events during the period of confusion.

Recurrent ACM. Two of our patients had recurrent episodes within 10 days of the first event. Ehyai and Fenichel[2] were the first to report that ACM can recur, usually within the first few weeks or months after the original event. The 8 patients in our study who had no previous history of headaches were followed for a mean of 18 months. None developed any other migraine disorders, but this was a relatively short-term follow-up.

EEG and ACM. Previous reports[1,2,4,6] have repeatedly confirmed EEG background slowing,

as seen in all 8 of our patients who had EEGs recorded. The high voltage delta slowing is usually prominent in the posterior quadrants. However, Pietrino et al[6] recorded frontal, intermittent, rhythmic delta activity (FIRDA) during attacks, a finding not previously reported. A bilateral high voltage, rhythmic delta arousal response was seen in 1 of the original 4 cases [1] and in 1 in our present series. This "paradoxical" response in an obtunded patient has been interpreted as signifying a functional disconnection between cortical and subcortical areas.

ACM and Transient Global Amnesia (TGA). In 1981 Caplan, et al[13] proposed that the syndrome of TGA in adults was caused by a migraine-vascular dysfunction. There is presumptive evidence that the pathophysiology of both ACM and TGA involves the vertebrobasilar circulation's end territories. Bilateral lesions in the amygdala and hippocampus produce a permanent amnesia with similar characteristics to TGA[14]. Brain SPECT studies during TGA have shown bilateral temporal lobe hypoperfusion[15]. In 1995 Sheth, et al[8] reiterated that a common pathogenic mechanism such as bitemporal hypoperfusion might exist in both syndromes. What is not clear is whether children with ACM are at risk for developing TGA in mid or late life.

Differential diagnosis. In our series the most common initial admitting diagnosis from the ER was "change in mental status", reflecting uncertainty about the etiology. Next most common was *encephalitis,* followed by suspected *toxic ingestion*. In other words the diagnosis was not evident even after emergency room management, and was usually made after inpatient admission, either at time of the EEG, or the next day, when the patient awoke with normal mental status.

Mild head injuries are known as a triggering factor for ACM[6], with 3 of Sheth, et al.'s patients[8] and 4 out of 13 of Shaabat's patients[9] having had preceding trivial head injury. However, this brings up *concussion* as a differential diagnostic possibility, at least on initial presentation.

Other conditions which may simulate ACM are *psychomotor or absence status epilepticus*, which may be difficult to differentiate without an EEG. Finally, adolescents may appear to be having an *acute psychosis*. However, careful mental status examination reveals no thought or affective disorder, but a picture of an acute "organic brain syndrome".

Proposal for diagnostic criteria.

Inclusion criteria.

1. Healthy prepubescent or adolescent patient presenting with acute disorientation, immediate and short-term memory difficulties, and disturbance in sensorium.
2. Neurologic examination of cranial nerves, motor and coordination, and sensory systems normal.
3. Duration of confusion, usually hours.
4. Termination of confusional episode by sleep.

Exclusion criteria.

1. Non-convulsive status epileptics (psychomotor or absence status)
2. Toxin/drug ingestion or withdrawal
3. Concussion
4. Encephalitis
5. Acute psychosis

Minimal diagnostic studies.

1. Toxic drug screen
2. Complete blood count
3. Brain CT scan
4. Electroencephalogram

Once the diagnosis is established, further invasive testing may be detrimental to the patient, especially given the low recurrence risk and no further migraines of any kind, at least during our short-term follow-up. No specific medical treatment is necessary.

It is not clear why ACM occurs around early adolescence. It raises the question of whether there are developmental vulnerabilities at different ages of the neurovascular instability that expresses itself as migraine equivalents or complicated

migraine, from cyclic vomiting of infancy, to benign paroxysmal vertigo and ataxia in childhood, to acute confusional in pre-pubescence and early adolescence, and then basilar migraine in adolescence.

REFERENCES

1. Gascon G, Barlow C. Juvenile migraine, presenting as an acute confusional state. Pediatrics 1970; 45: 628-635.
2. Ehyai A, Fenichel GM. The natural history of acute confusional migraine. Arch Neurol 1978; 35: 368-369.
3. Emery ES. Acute confusional state in children with migraine. Pediatrics 1977; 60: 110-114.
4. Tinuper P, Cortelli P, Sacquegna T, Lugaresi E. Classic migraine attack complicated by confusional state: EEG and CT study. Cephalalgia 1985; 5: 67-68.
5. Haan J. Acute confusional migraine. Case report and review of literature. J Clin Neurol Neurosurg 1988; 90(3): 275-278.
6. Pietrini V, Terzano MG, D'Andrea G, et al. Acute confusional migraine: Clinical and electroencephalographic aspects. Cephalalgia 1987; 7: 29-37.
7. D'druz OF, Walsh DJ. Acute confusional migraine: Case series and review of literature. Wisc Med J 1992; 92: 130-131.
8. Sheth RD, Riggs JE, Bodensteiner JB. Acute confusional migraine: Variant of transient global amnesia. Pediatr Neurol 1995;12:129-131.
9. Shaabat A. Confusional migraine in childhood. Pediatr Neurol 1996;15(1):23-25.
9. Al Twaijiri WA, Shevell MI. Pediatric migraine equivalents: Occurrence and clinical features in practice. Pediatr Neurol 2002; 26: 365-368.
10. Ferrera PC, Reicho PR. Acute confusional migraine and trauma-triggered migraine. Am J Emerg Med 1996;14(3):276-278.
11. Brown J. ED evaluation of transient global amnesia. Ann Emerg Med 1997; 30(4): 522-526.
12. Caplan L, Chedru F, L'Hermitte F, Mayman C. Transient global amnesia and migraine. Neurology 1981; 31: 1167-1170.
13. Caffarra P, Scagiolni A, Malvezzi L, Manzoni GM. Transient global amnesia and migraine. Ital J Neurol Sci 1988; 9: 287-289.
14. Goldenberg G, Podreka I, Pfaffelmeyer N, Wesely P, Deecke L. Thalamic ischemia in transient global amnesia: A SPECT study. Neurology 1991; 41: 1748-1752.