Chapter 13

The Woman with a Change in Her "Migraine"

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Case History

A 39-year-old female university graduate, now working in a management position, presented with the complaint of alteration in her usual pattern of migraines and "tension headaches" that had started in her early twenties.

She had experienced the first altered episode 7 years before. This was severe and of abrupt onset, awaking her from a deep sleep, and characterized by altered consciousness with limited responsiveness, confusion, blindness in both visual fields, ptosis, vertigo, dysarthria and dysphagia, hemiplegia, ataxic gait, and paresthesias. It lasted 30 minutes, after which she was left weak and cold, with respirations 3 to 4 per min, blood pressure 80/50, and residual neurologic deficits that lasted for about a week. Subsequent attacks were similar and occurred at irregular intervals, often at rest or while sleeping. Almost all lasted for 15 to 60 minutes. Total resolution of all deficits was immediate thereafter in the mild episodes, but took up to 4 months if there had been a series of severe attacks rapidly succeeding one another in 1 day. All attacks were typically followed by a severe occipital headache with features typical of International Headache Society (IHS)-classified migraine.

The family history revealed that at least 1 in 4 of the females on both parents' sides had experienced regular headaches, with a high incidence of complicated migraine and basilar artery migraine (BAM). On her father's side, Prinzmetal angina and vertebrobasilar ischemia were common problems. Several females on her mother's side with headaches had also been found to have mitral valve prolapse (MVP).The genetic link goes back three generations, with BAM or "complicated migraine" having been diagnosed in every generation after the patient's great-grandfather.

Clinical and neurologic examinations were always normal between attacks, and electroencephalogram (EEG), electrocardiogram, computed tomography, and magnetic resonance imaging (MRI)/magnetic resonance angiography results were also negative. An electrocardiogram in her early twenties showed left ventricle enlargement that was considered to be related to MVP. Thyroid replacement therapy has also been advised.

A diagnosis of BAM was made 6 months after the onset of the new features.

Treatments have comprised over-the-counter preparations, ibuprofen up to 800 mg per day, behavior modification, stress-reduction techniques, and self-hypnosis. The attacks improved over the course of 2 years with treatment of temporomandibular joint disorder with a bite appliance, and subsequently became mild, but of longer duration over the next 10 years; medications were still needed. Depakote led to a weight gain of over 100 pounds over 6 years, causing other health problems. When its initial benefit decreased, the current regimen (480 mg verapamil, 400 mg topiramate, and 300 mg Wellbutrin daily, the latter to counter the depressive effects of frequent common migraines and weight gain) was substituted. The severity of the BAM attacks was reduced, but the patient still periodically experiences vertigo and other BAM symptoms, as well as frequent common migraines.

Other treatments found to be ineffective, intolerable, or both have included nortriptyline, aspirin 81 mg per day, propranolol, and Depakote up to 1,500 mg per day, with sublingual nifedipine 10 to 30 mg or sublingual nitroglycerine.

Questions on the Case

Please read the questions, try to answer them, and reflect on your answers before reading the author's discussion.

- What are tension headaches?

- Can one ascribe the clinical features of the attacks to a single location in the central nervous system?
- Is the MVP relevant?
- How are the forms of "complicated" migraine related?

History and Description

Gowers (1907), quoted by Pearce (1969), described a woman who had had right-sided migrainous headaches from the age of 18 years. After 10 years, the attacks altered, such that now she would lose the sight of both eyes ("a black curtain seemed to be dropped down, brilliant with thousands of golden points"), after which she experienced severe vertigo and dysesthesia in the arms, legs, and jaw for 10 minutes. She next became unconscious for 15 minutes, after which she recovered awareness but had severe bioccipital headache for 2 hours. It was impossible to prove a definite diagnosis.

Bickerstaff described 34 patients with a similar syndrome, almost all young women with a family history of migraine. As summarized by Blau, "Visual symptoms, teichopsia, or field detects were the first symptoms of an attack, followed by one or more other symptoms: vertigo, ataxia of gait, dysarthria, tinnitus, and sensory symptoms in the periphery of both upper and lower limbs and around the lips and tongue. These symptoms lasted from 2 to 45 minutes and were succeeded by throbbing occipital headache, vomiting, and finally by normal [but sometimes very deep] sleep. The attacks were infrequent and interspersed with more classical migrainous episodes." Again, to paraphrase Blau, Bickerstaff conceived that cerebral ischemia due to vasospasm in the internal carotid distribution caused the features of classic migraine, but that if the basilar artery were involved, then the clinical features would reflect the resulting ischemia of the brainstem, cerebellum, and occipital cortex bilaterally, even if asymmetrically.

It is strange that a disease as ubiquitous and pleomorphic as migraine still lacks a classification that is satisfactory to clinicians. That of the IHS recognizes many subtypes of migraine with aura, but there is considerable overlap in their clinical features:

1.2 Migraine with aura
1.2.1 Typical aura with migraine headache
1.2.2 Typical aura with nonmigraine headache
1.2.3 Typical aura without headache
1.2.4 Familial hemiplegic migraine
1.2.5 Sporadic hemiplegic migraine
1.2.6 Basilar-type migraine
1.3 Childhood periodic syndromes that are commonly precursors of migraine
1.3.1 Cyclical vomiting
1.3.2 Abdominal migraine
1.3.3 Benign paroxysmal; vertigo of childhood
1.4 Retinal migraine

It also lists symptoms that may accompany the headache or appear alone. Dysphrenic migraines are not included in the IHS classification, and so-called "ophthalmoplegic migraine" is now considered to be a variety of recurrent neuropathy.

Clinical Features of the Attack

In a study of 49 cases, the age of onset of BAM ranged from 10 to 63 years and about a third of the group was male. Most patients with BAM in youth report that their attacks gave way in time to other migraine headaches. Severe EEG abnormalities without seizures have been described during attacks in childhood, as have drop attacks in adults; they have been reviewed by Blau. A family history of some form of migraine is usual. Bickerstaff noted that most patients with BAM as adolescents find that these migraines diminish, but are replaced by other typical migraine headaches as they age, and other studies have agreed with this. The aura generally lasts less than 1 hour and is usually followed by a bioccipital headache, nausea, and vomiting. A typical hemianopic field disturbance can rapidly expand to involve all visual fields, leading at times to temporary blindness. In fact, a characteristic of basilar migraine is the bilateral nature of the associated symptoms, unlike the situation in typical migraine. The visual aura is usually followed by any of dysarthria, vertigo, tinnitus, decreased hearing, diplopia, ataxia, bilateral paresthesia, bilateral paresis, and impaired cognition. When the latter is severe, the condition is known as dysphrenic or confusional migraine.

The diagnostic criteria of the Headache Classification Committee of the International Headache Society require the presence of typical (IHS code 1.1) migraine attacks and two of the following features: diplopia, dysarthria, vertigo, tinnitus, hyperacusis, simultaneous bilateral visual symptoms, altered consciousness, ataxia, and simultaneous bilateral paresthesias. One of more of these should develop gradually over 5 minutes or occur in succession, and their duration should be between 5 and 60 minutes.

The first symptoms are usually positive visual phenomena such as scintillations, or negative ones such as graying of vision progressing to hemianopia or even complete visual loss affecting the full fields of both eyes. The typical hemianopic field disturbance can rapidly expand to involve all visual fields, leading at times to temporary blindness. A distinguishing characteristic of BAM is the bilateral nature of many of the associated neurologic events, which helps to differentiate it from typical migraine.

The initial symptoms usually last from 10 to 45 minutes before subsiding, giving way to the headache. Although

this is almost invariably bioccipital, the presence of a headache is not required for the attack to meet diagnostic criteria. This headache is at first bilateral and occipital, but may spread to the neck and the vertex. It lasts from seconds (usually in children) to days, and is commonly severe, throbbing, and associated with nausea, which may progress to intractable vomiting causing dehydration. Recovery is heralded by cessation of vomiting and the appearance of drowsiness, even approaching stupor, but with retained ability to be aroused at least to a drowsy state. Such drowsiness and some vertigo may persist for a day or so.

Aphasia with dysarthria and motor weakness are the most common features, after headache. Unsteadiness of gait usually occurs next, followed by dysarthria, mild vertigo, and bilateral tinnitus. Numbness and/or tingling begin peripherally, spreading proximally to the wrists and ankles, the mouth, and occasionally the tongue. Nystagmus and decreased hearing are uncommon. Parietal lobe symptoms such as disorders of body image and extrapersonal space, disorientation and metamorphopsia, and temporal lobe symptoms such as dysphasia, feelings of sudden intense fear, depersonalization, automatisms, and behavior disorder are also described.

Clues to the migrainous nature of the syndrome include the presence of positive visual symptoms, which like paresthesias, gradually build up and migrate or "march" and progress serially. Usually, their duration is less than 30 minutes. The occurrence of other more "typical" migrainous attacks over years, the absence of neurologic findings between attacks, the bioccipital headache, and the frequency of a family history of some form of headache are further diagnostic pointers.

Variants include forms with auditory-visual synesthesias and the occurrence of convulsions (usually in those with a positive family history of seizures). Parietal lobe syndromes (disordered body image and extrapersonal space, metamorphopsia, disorientation) and temporal lobe syndromes (dysphasia, sudden intense fear, depersonalization, automatisms, and behavior disorder) are also described.

Differential Diagnosis from Other Forms of Complicated Migraine or Other Conditions

Structural vascular disorders, such as thrombosis or dissection of the basilar or posterior cerebral arteries, the subclavian steal syndrome, vertebrobasilar insufficiency, epilepsy, posterior fossa, tumors, idiopathic intracranial hypertension, thrombocythemia, polycythemia, hyperviscosity, and lupus anticoagulant syndromes may present similarly and require exclusion. Cortical infarcts and basilar infarction with resulting locked-in, lateral medullary or other syndromes have been described as being due to BAM, but vertebrobasilar dissection is a more likely cause in such cases, with almost all being reported without benefit of modern imaging.

In "migraine with prolonged aura," one or more aura symptoms last more than an hour and less than a week.The overlap between this, BAM, and the following variants is obvious (Table 13-1).

"Familial hemiplegic migraine" is a rare, dominantly-inherited disorder characterized by sequentially progressive, transient hemiparesis and any of visual, sensory, and aphasic features, with subsequent headache. Two forms occur, linked respectively to chromosomes 19 and 1q 21–23.

"Sporadic hemiplegic migraine" is equally frequent. In this condition, the gradual progression and sequential appearance of aura symptoms are identical to those of familial hemiplegic migraine, the duration of each aura symptom is usually prolonged, and bilateral motor symptoms are more frequent, but there is no family history. The order of the aura symptoms is usually visual, followed by sensory, aphasic, motor, and lastly, basilar-type migraine symptoms. Most of these patients fulfill the criteria for basilar migraine. About three-quarters of the subjects surveyed by study authors fulfilled the criteria for basilar artery migraine.

Another variant is the combination of "basilar migraine, seizures, and severe epileptiform EEG abnormalities," a benign syndrome in adolescents characterized by the occurrence of rare focal or generalized seizures and rhythmic temporo-occipital spike wave complexes in association with the clinical features of basilar migraine. Whether or not this is the same condition as benign occipital epilepsy of childhood is not yet determined.

In the case of "occipital lobe seizures," migraine-like symptoms responsive to oral triptans can occur in association with spontaneous seizures, the ictal discharge arising from an occipital lobe.

The nature of "benign recurrent vertigo of childhood" is unexplained, although some subjects later recall co-incident features of basilar artery migraine, or go on to experience severe vertigo in their future attacks of that condition.

"Dysphrenic migraine," another rare variant, is characterized by episodes of confusion, dysmnesia, hallucinations, and a potential variety of temporary psychotic states which occasionally occur without headache in patients with a past history of migraines.

"Migraine without headache" is more common in older patients. Associated features sometimes occur without any succeeding headache, providing the phenotype of a transient ischemic attack. The "march" of symptoms, the presence of "positive" symptoms such as sparkles of light, and their persistence for hours are features suggesting BAM rather than embolic events.

Table 13-1. Features of Some "Complicated" Forms of Migraine

Factor	BAM	Ophthalmoplegic	Dysphrenic	Familial Hemiplegic	Sporadic Hemiplegic
Family history	+	?	?	+	–
Associated migraines	+	+	+	+	+
Altered consciousness	+	–	+	+	+
Confusion	+	–	+	+	–
Aphasia	+	–	+	+	–
EOM palsy	–	+	–	–	–
Uni/bilateral motor signs	+	–	–	+	+
Sensory symptoms	+	–	–	+	+/–
March of symptoms	+	+	+	+	+

BAM = basilar artery migraine; EOM = extraocular muscle.

"Cerebral autosomal dominant arteriopathy with subcortical infarcts and leukoencephalopathy" (CADASIL) is characterized by stroke, dementia, and migraine, but it may present as an acute reversible encephalopathy lasting up to 2 weeks with fever, delirium, seizures, and reduced consciousness. There is usually full recovery. All patients have a previous history of migraine with aura. MRI white matter changes, previous migraine with aura, and a family history of stroke and dementia are useful pointers to the diagnosis.

A form with "Lilliputian hallucinations," involving visual hallucinations of small beings, occurring at the peak of severe migraine attacks and lasting between 2 and 5 minutes, may also represent a migraine aura symptom. Their appearance in peduncular hallucinosis, due to mesencephalic and/or thalamic lesions, supports the notion that these may also represent aura symptoms arising from the brainstem in basilar migraine.

There is some evidence for the association of MVP or trauma with migraine-like headaches. If such associations are truly causal, then the mechanisms underlying them remain unclear, as does the occasional co-occurrence of cardiac arrhythmias.

In the rare syndrome of ophthalmoplegic migraine, the headache is associated with complete or partial paresis of the extraocular muscles, usually those innervated by the third nerve, and often outlasting the headache by weeks; indeed, complete recovery may not occur. Ptosis is an early sign and the pupil is dilated. Pain is maximal in the supraorbital region. Attacks occur irregularly and infrequently. The condition is considered to be a form of cranial neuropathy in the second edition of the IHS classification.

Pathophysiology

Bickerstaff attributed the syndrome to ischemia in the posterior circulation, considering that basilar artery constriction would cause brainstem or occipital cortex dysfunction. Indeed, many symptoms of BAM involving the occipital lobes (bilateral visual loss), posterior-medial temporal lobes (seizures, time distortion), cerebellum (ataxia), midbrain (gaze palsies and diplopia), pons (hearing loss, vertigo, and tinnitus), medulla (dysarthria), and overall brainstem (loss of consciousness, paresis, paresthesias) could thus be explained. However, while vasospasm and cerebral oligemia have been detected in patients with BAM and familial hemiplegic migraine, it is not possible to be certain that these findings are primary, rather than secondary to a state of cerebral hypometabolism. "Vasospasm" and cerebral oligemia have been detected by MRI in a patient with familial hemiplegic migraine, but this is less likely to be primary than secondary to cerebral hypometabolism; no diffusion-weighted MRI evidence of cerebral ischemia was found in a patient with familial hemiplegic migraine. Hypoperfusion was diffuse, but most obvious in the less symptomatic hemisphere. The persistent depression of cortical electrical activity suggests that neurogenic changes are more important than vascular changes in familial hemiplegic migraine.

The migraine aura is likely caused by a neurophysiologic phenomenon akin to Leao's cortical spreading depression (CSD), a wave of short-lasting neuronal excitation that travels forward over the cerebral cortex, followed by prolonged depression of cortical neuronal activity. Single-voxel ^{31}P magnetic resonance spectroscopy studies suggest that disordered energy metabolism or Mg^{++} deficiencies may underlie hyperexcitability of neuronal tissue in migraine patients. Patients suffering migraine with aura also have an increased expression of a dopamine *D2* receptor gene allele (*DRD2*). As antagonists to this may prevent both the headache and the aura, both may be evidence of *D2* receptor hypersensitivity. CSD may be incited by glutamate and/or by potassium-based mechanisms. Magnesium gates *N*-methyl-*D*-asparate receptors and inhibits glutamate- and potassium-induced spreading depression and Mg^{++}-deficient spreading depression in animal models. Anecdotally, prochlorperazine and magnesium have been claimed as effective in treating the migraine aura.

It is not surprising that the excitation followed by depression in cortical activity is manifest as positive and then negative cortical symptoms, but it is noteworthy that almost all of the "brainstem" symptoms in BAM (eg, diplopia, dysarthria, vertigo, tinnitus, hyperacusis, simultaneous bilateral visual symptoms, ataxia, and simultaneous bilateral paresthesias) are best interpreted as being due to excitation.

Management Strategies

Concern about the use of vasoconstricting agents in BAM has been due to the thought that ergotamine and the triptans would increase ischemia and worsen the attack, possibly even causing stroke. However, reduced blood flow might just as well be the result of the cerebral hypometabolism resulting from the intrinsic cerebral dysmetabolic process basic to migraine. Thus, although triptans are formally listed as contraindicated in patients with basilar or familial hemiplegic migraine, this recommendation should be reconsidered, since their use has been reported as being both effective and harmless.

Other agents employed with variable success have included simple analgesics, intravenous (IV) prochlorperazine, IV magnesium, and oral or IV verapamil (for sporadic and familial hemiplegic migraines). For prolonged migraine aura, inhaled 10% CO_2 in 90% O_2, amyl nitrite or isoproterenol, sublingual nifedipine, IV furosemide, oral sodium valproate or acetazolamide, and intranasal ketamine have been suggested. A recent report claimed excellent efficacy in prolonged migraine aura with IV prochlorperazine and $MgSO_4$.

The drugs most useful in the prophylaxis of basilar migraine include lamotrigine and topiramate. In the past, cyproheptadine, propranonol, tricyclics, phenytoin, and calcium channel blockers were used with moderate success. In the family of the patient described here, calcium channel blockers have been efficacious for both the migraines and the Prinzmetal angina. A 14-year-old female patient with BAM became headache free with the use of biofeedback techniques, specifically fingertip warming.

Commentary

Other clinical features never formally linked to BAM, but which (like mitral valve prolapse) occur frequently in patients with BAM, include hypotension, hypothyroidism, mild hypoglycemia, and allergy to wasp/bee venom, penicillin, and molds.

Since the publication of the Spectrum study, the diagnosis of "tension-type headaches" in patients with a history of migraines has been suspect, since these can equally well be considered to be diluted migraines, not fulfilling IHS criteria for migraine. No electromyographic study has ever demonstrated a secure pathophysiologic basis for such headaches, which respond well to triptan therapy.

The similarities between the various forms of "complicated" migraine reviewed above are notably greater than their differences (see Table 13-1), and clinically, there is substantial overlap between the various forms. Splitting is acceptable when recurrent clinical differences are perceived or when the pathophysiology of two conditions is demonstrably dissimilar. Because such is not the case here, it is most appropriate to suppose that until we achieve true understanding by the understanding of causes, most of the forms of migraine reviewed are but phenotypic variants upon a common, but yet undefined, genetic theme.

Case Summary

The complicated forms of migraine include

- Migraine with typical or prolonged aura
- Sporadic and familial hemiplegic migraines
- Basilar migraine
- Migraine aura without headache
- Ophthalmoplegic, retinal, and dysphrenic migraines

The presentations of migraine are pleomorphic, but because there is substantial overlap in the phenotypes encountered, it is most appropriate to regard these variants as having a common basis in intermittent primary excitation and subsequent depression of cortical dysfunction, followed by sensitization of the brainstem trigeminovascular complex, leading to overlapping syndromes of neurologic phenomena.

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Editorial Comments

Basilar artery migraine has been an area of intense clinical interest since the time of its major description in the classical paper by Bickerstaff. It is the bilateral nature of the symptoms that are most striking, and given the prominence of vascular mechanisms in the early considerations of the mechanisms of migraine, it is not surprising that BAM was felt to be somehow related to the basilar artery and its anatomic projections. However, it is possible that bilateral hemispheric dysfunction could also cause similar symptoms, and this is recognized in the new IHS classification for this disorder, which is now called basilar-type migraine. The mechanisms could also be neuronally based, rather than vascular, but are still unknown. Clinicians have to start rethinking their understanding of BAM and similar disorders, and this case goes a long way toward shaping that thinking in terms of diagnosis, differential diagnosis, and in particular, treatment. Dr. Pryse-Phillips has helped us all with this eloquent review and discussion, and this case and topic are worthy of more study.

Final diagnosis:

Basilar-type migraine