

COMPLICATED MIGRAINE—A REVIEW OF TEN CASES

By T. G. Loh and J. C. Chawla

SYNOPSIS

Complicated migraine is not well known. It is associated with transient and protean sensory and motor neurological features although disturbance of mood, affect and psyche has also been described.

This paper is based on a study of ten patients suffering from complicated migraine and attempt is made to describe various neurological symptoms and signs.

It becomes apparent from this study that there are diverse manifestations of this interesting disorder. Furthermore complicated migraine seems to affect a younger age group and there may not be any family history of migraine. In most cases the neurological symptoms precede the attacks of headaches. The neurological deficit may persist after longer periods. Initially, the attacks are usually followed by complete recovery, with repeated attacks there may be residual neurological deficit. Though the exact aetiology of the migraine remains unknown, it has been suggested that it is due to spasm or oedema of cerebral vascular system. If the vessels involved are the carotid artery system, the symptoms and signs obviously would be due to involvement of cerebral hemisphere, whereas affection of basilar artery would produce signs and symptoms of cerebellar or brain stem deficiency.

At times it may be extremely difficult to differentiate between a typical attack of migraine and headache due to an underlying pathology e.g. intracranial angioma or space occupying lesions, which may require investigations such as angiography. The angiography may prove fruitless and cause deterioration in neurological signs. A careful follow-up should be planned for cases with complicated migraine to exclude any underlying pathology.

The major clinical features of migraine consisting of its paroxysms, its remissions, its relation to circumstances, its emotional and physical symptoms had been clearly recognised by the second century. Aretaeus of Cappodacia described it under the name Heterocrania. Classical and common migraine are the forms usually seen and readily recognisable. The associated family history of the more usual forms of migraine and the occasional occurrence of these in an individual usually helps to support a diagnosis of migraine.

Complicated migraine, another variant is not so well known, though it had been described as early as in 1714 by Piso and subsequently by others including Charcot in 1889. It is associated with transient and protean sensory and motor neurological features, although disturbances of mood, affect and complex psychic phenomena have also been described. The features in complicated migraine, are unlike those of classical and common migraine, and include visual field defects, metamorphosis, ophthalmoplegia, vertigo, tinnitus, paraesthesia, disturbances of sensation and speech,

and mental changes. It presents in such varieties like ophthalmoplegic, facioplegic, both sporadic and familial hemiplegic migraine.

This paper is based on a study of ten patients suffering from complicated migraine who were seen by us, and it is felt that by reporting them the diverse manifestations of this interesting disorder can be illustrated. All the cases have been summarized in the Table, but the following cases are reported in detail to illustrate the various neurological symptoms and signs.

SELECTED CASE REPORTS

Case 1

L.S.K. a 31 year old Chinese male had been having headache as frequently as once a week for as long as he could remember. The headaches were generalized over the vertex and were relieved by 'panadol'. At the age of 21 years, he felt a numbness and weakness of the left hand lasting for five to ten minutes. Five years later, he suddenly felt a momentary throbbing headache over the vertex followed immediately by numbness over the left face, which spread to the left arm and fingers. The left upper limb also felt weak. This symptom lasted for ten minutes. Three months before being seen by us he suffered a similar episode which was also associated with a slurred speech and difficulty in pronouncing words. He did not have difficulty in finding the

Departments of Medicine and Surgery, Faculty of Medicine, University of Malaya, Kuala Lumpur, Malaysia.

T. G. LOH, M.B., B.S. (Singapore), M.R.C.P., M.R.C.P.E., M.R.C.P.G.

J. C. CHAWLA, M.B., B.S., F.R.C.S.

TABLE I
SHOWING THE DIVERSITY OF THE NEUROLOGICAL MANIFESTATION IN
COMPLICATED MIGRAINE

Case number	1	2	3	4	5	6	7	8	9	10
Sex and when first seen	M 31	M 22	M 12	M 14	M 17	F 34	M 30	F 26	F 23	M 14
Age at first attack	21	22	12	14	18	13	30	8	12	7
Headache	+	+	+	+	+	+	+	+	+	+
Family history of headache	-	+	-	-	+	+	+	-	-	-
Vomiting or nausea	-	-	+	+	+	-	-	+	+	-
Teichopsia	-	-	-	+	+	-	+	+	-	-
Photophobia	-	+	-	-	-	-	-	-	-	-
Visual loss	-	-	-	+	+	+	+	-	-	-
Vertigo	-	-	-	-	+	+	+	+	-	-
Tinnitus	-	-	-	-	-	-	+	+	-	-
Hemiparesis & Monoparesis	+	-	+		+	-	+	-	-	-
Paraesthesia	+	+	+	-	+	-	+	-	-	-
Dysphasia	+	-	+	-	-	-	-	-	-	-
Sensory loss	+	-	+	-	-	-	-	-	-	-
Unconsciousness	-	-	+	-	+	-	-	+	-	-
Hemianopia	-	+	+	-	-	-	-		-	-
Cranial nerve palsy	+	-	-	+	+	-	+	-	+	+
Angiography	N	-	N	N	N	-	-	-	N	N
Electroencephalography	F.A.	N	F.A.	F.A.	D.A.	N	F.A.	D.A.	D.A.	F.A.
Other features				Ataxia						

F.A. = Focal Abnormality
D.A. = Diffuse Abnormality
N = Normal

proper words to say. This episode lasted an hour. Two months later he again had stereotype symptoms, but the symptoms instead of passing off like before, persisted for three days before any improvement. They were still present at the end of four weeks. There was no history of headache or similar illness in the family.

No carotid bruit was heard and B.P. was 120/70 mmHg. the fundi showed no abnormality. There was a left facial palsy of the upper motor neuron type. There was spastic weakness of the left upper limb. Wartenberg's and Hoffman's signs were positive. Plantar responses were flexor. He had impaired sensation over the face and the hand and impairment of two points discrimination on the left fingers. He had a mild slurring of speech. All other systems were within normal limits.

The investigations including skull X-rays and right carotid angiography, brain scan and C.S.F. were normal. The electroencephlogram (E.E.G.) recorded during wakefulness showed theta waves at 5-6 c/sec. and delta waves at 2-3 c/sec. localised to the right anterior and mild temporal regions indicating a focal lesion localised there. The patient gradually improved. The slurring of speech disappeared. Only minimal facial palsy and weakness with hyperreflexia of the upper limb persisted when seen two months later. A repeat electroencephlogram showed no evidence of focal delta waves on the right temporal region.

Case 3

Y.K.S. a twelve year old Chinese boy felt a momentary headache following a minor head injury. He did not lose consciousness. Two hours later he went to sleep. On waking up he felt slightly tired. He also vomitted several times. There was no headache. Six hours later he developed a severe left sided headache and also vomitted. He developed a right hemiparesis and became unable to speak, though he was able to understand and could indicate by signs the site of his headache. Seen three days later he still was unable to speak and was groaning in pain. There was no previous history of headache or visual disturbances. No members of the family had migraine.

He was right handed. He had a right hemianopia, an expressive dysphasia with a right hemiparesis and hemianaesthesia. There was a right Babinski's sign. The fundus was normal. No other abnormalities were found. The x-ray skull showed no fractures. A left carotid angiogram was normal and there was good filling of posterior cerebral artery. The E.E.G. showed focal slow waves at 1 c/sec. seen over the left parietal, central and mid-

temporal regions associated with diffuse theta waves at 5-6 c/sec. and delta waves at 2-3 c/sec. Other investigations including the CSF were normal. Most of the neurological signs had disappeared in three days.

The E.E.G. repeated a week later showed improvement. A month later he had a similar episode of left temporal headache with right hemiparesis and right homonymous hemianopia and had remained free of attacks when seen four months later.

Case 5

A.H.M. 17 year old Malay youth was admitted with one day history of sudden onset of blurring of vision, shimmering lights and diplopia associated with numbness and weakness of the left leg. Half an hour later, the numbness and weakness spread to the right leg and then upwards to both arms. Almost at the same time he had a throbbing bitemporal headache associated with nausea and vomiting. A few minutes later he began to see a bright spot with a dark background and the spot gradually enlarged. He was unsteady while walking. He felt drowsy and fell asleep. On waking up, the numbness persisted though of less intensity. There was no change in the other symptoms. Within another two to three hours the symptoms subsided. A year prior to admission he had an episode of numbness of the left lower limb which spread to the right lower limb then to both upper limbs associated with weakness. He started having paroxysmal headaches lasting only a few minutes, as well as bright zig-zag lights a month before admission. It was not associated with diplopia. His mother and a sister were said to have headache associated with numbness.

He was well orientated but drowsy. Cranial nerves were intact. No sensory or motor weakness were found. He had brisker reflex tendon jerks on the left side, with a left extensor plantar response. His B.P. was 130/80 mmHg. All pulses were felt and no bruit was heard over the carotids. On the following day he recovered fully.

Investigations consisting of routine blood, urine and CSF examinations were normal. A vertebral angiogram was done. No abnormalities were seen. E.E.G. was mildly abnormal with some diffuse theta and delta waves with no focal abnormality.

Following the angiogram there was depression of the level of the consciousness and he improved over the next three days. A transient up-going left plantar response and nystagmus on looking to the right were seen. He was discharged home well.

Case 6

R.Z. a 34 year old Malay female complained of recurrent episodes of blurring of vision, vertigo and unilateral headaches. Her symptoms dated back to the age of thirteen years. These episodes lasted from one to one and half hours. During these episodes she became unsteady in her gait and had to cling on to supports to prevent herself from falling. This was followed by a severe unilateral headache usually on the right side. She experienced about one attack every three to four months. She never experienced any techopsia or disturbances of consciousness. The mother and two other siblings also suffered from similar headaches. No abnormality was found on physical examination. A skull X-ray was normal. E.E.G. was normal except for a suspicious sharp transient on the left occipital region.

Case 7

M.S. a 30 year old Malay male was first seen in September 1970 with a history of recurrent episodes of numbness of the right side of the body, left face and flashes of light with black spots in his left eye, tinnitus with vertigo for three months. Each attack usually began with a feeling of numbness on the right hand which spread upwards to the shoulder, down to the chest, the right foot and then to both the legs and the thighs. Half an hour later, the numbness was felt on the tongue and subsequently on the throat and neck. The left face then also became numb. About this time he felt a throbbing headache on the left temporal region. Concurrently the left eye felt heavy. A similar feeling, though less intense was felt on the right eye. Bright flashes of light and black spots appeared in the visual field of the left eye. There was tinnitus, impairment of hearing and vertigo. Nausea with no vomiting was experienced. He had to lie down. Within three to four hours he would be perfectly well again. He had been having recurring episodes of these stereotype complaints sometimes as often as once a day. A sister also had headaches associated with vomiting and tendency to fall.

He had B.P. of 100/70 mmHg. All pulses were felt with no bruit heard. The tendon reflexes were slightly more brisk on the left side. Both plantars were down going. Two months later he was seen again with the similar stereotype symptoms. Again no gross neurological deficits were found except for the brisker reflex on the left side.

Investigations were all normal except the E.E.G. which showed occasional delta waves at 2-3 c/sec. over the left occipital region.

Case 8

A.B.Y. a 26 year old Malay female had experienced headache on the vertex and sometimes unilaterally since the age of 9 years. The headache occurred two to three times a week lasting two to three hours and was associated with nausea and vomiting. At the age of 22 years, she had an attack of giddiness, and vomiting, followed immediately by loss of consciousness. On regaining consciousness she had a severe headache which persisted for several hours. From then onwards, she began to have frequent episodes of vertigo, associated with diplopia, occasionally accompanied by bright spots before the eyes and vomiting. After ten to thirty minutes she would become unsteady in her gait. She would lie down as she felt drowsy being vaguely aware of what went on around her. About ten to thirty minutes later she would have her usual throbbing headache for a few hours. At the age of 25 she had a similar episode but there was also tinnitus and impairment of hearing. This attack lasted for ten to fifteen minutes. There was no history of migraine in the family. No abnormality was found on clinical examination. An E.E.G. showed some excessive diffuse theta waves at 5-6 c/sec. on both sides.

Case 9

R.M.N. a 23 year old Malay woman was admitted with history of severe right sided throbbing headaches associated with deep seated eye pain since the age of twelve years which started following a fall. There was no history of loss of consciousness though she was hospitalised. The headache came on a week after admission and lasted for 3 days. It was associated with vomiting. She also noticed ptosis of the right eye as well as inability to move her right eye properly. Ever since, she started to have these symptoms once a month which usually recovered within a day. There was no family history of similar illness. Examination showed she had a complete third right cranial nerve palsy with ptosis, external ophthalmoplegia and a dilated pupil, which had no response to light and accommodation. No other abnormalities were detected. Visual field charting was normal.

Investigations: A right carotid angiogram was normal and showed filling of the anterior, middle cerebral and posterior cerebral arteries. E.E.G. was mildly abnormal with diffuse theta waves at 4-5 c/sec. and delta waves at 2-3 cc/sec. Other routine blood, urine and CSF examinations were normal. Over a period of six to seven weeks she improved leaving only a slightly dilated pupil with mild ptosis.

Case 10

L.K.H. a 14 year old Chinese boy had been having recurrent episodes of right sided headaches for the past seven years before admission. The headache usually started on the supraorbital region and then radiated up to the vertex. He also noticed diplopia on looking to the left. Sometimes he woke up at night because of the headache. He had no nausea or vomiting. The attack disappeared within a week. He usually had one attack a year. He suffered one of these attacks six months prior to the latest one which brought him to hospital. There was no family history of similar illness. Clinically he was conscious and co-operative. There was a right oculomotor palsy with ptosis of the upper eye-lid with complete paralysis of the medial rectus, superior rectus and inferior oblique muscles. The pupils were equal and reactive to light and accommodation.

A right carotid angiogram showed good filling of the anterior, middle and posterior cerebral arteries. Other routine tests were normal. E.E.G. was moderately abnormal. Diffuse theta waves at 4-5 c/sec. and delta waves at 2-3 c/sec. were seen on both sides with definite preponderance over the left posterior quadrant suggesting the maximal disturbance was localised there.

Gradually the paralysis improved over the next three weeks. At the end of six weeks he had only mild ptosis with weakness of the extraocular muscles.

DISCUSSION

The clinicians are familiar with symptoms of classical migraine i.e. history of headaches, particularly associated with visual symptoms with a family history, although the diagnosis may be difficult to prove. Some of the patients may have underlying vascular abnormality. The diagnosis becomes more difficult when the headaches are associated with gross neurological deficit which persist for some time.

From our review of the ten cases it becomes evident that complicated migraine differs from classical migraine in many respects.

There is no consistent history of migraine among the families of patients with complicated migraine compared to classical migraine where the hereditary factor ranges between sixty to ninety per cent (Wolff, 1963). Only three out of ten had a family history of headaches. Complicated migraine usually occurs in younger age group (Foster, 1965). Of ten cases reported seven are below 30 years and only three between 30-34 years.

In most cases neurological symptoms precede the attacks of headaches. This is not however, invariably so. It may occur simultaneously or entirely dissociated from it (Bruyn, 1968). In Case 1, the headache precedes the neurological symptoms, while in the others headache occurs during or after the nervous symptoms.

In Cases 1, 2, 3, 4, the symptomatology suggested involvement of the cerebral cortex, secondary to involvement of the anterior, middle and posterior cerebral arteries or their branches. Such cases are similar to those described as hemiplegic migraine by Bradshaw and Parsons (1965). Their 77 patients had migrainous headaches associated with sensory or motor symptoms referred to the limbs on one side. The symptomatology range from transient paraesthesia or weakness to severe numbness and dense hemiplegia. Hemiplegic migraine is an unusual variant of migraine, often with a strong family tendency (Whitty, 1953, Blau and Whitty, 1955).

Although in Case 8, the history of the fall and knock suggested a head injury with the possibility of a subdural clot, but there have been reports of cases in which the motor weakness may be so precipitous that it caused falls (Clark, 1910). It is possible that the fall was due to motor weakness.

The history of Cases 5, 6, 7, 8 suggested affection of the basilar artery with production of features of brain stem involvement. Such cases apparently are not uncommon. Bickerstaff (1961a, 1961b) reported 34 cases among a personal series of 300 migrainous patients and discussed the syndrome. When unconsciousness occurs, the differential diagnosis of syncope, epilepsy and hysteria has to be considered. However, the long duration of the aura and the slowness in onset of unconsciousness frequently not profound, followed by a severe throbbing headache, helps in differentiating it from other conditions.

Ophthalmoplegic Migraine was seen in two patients (Cases 9, 10) who had repeated attacks with complete recovery initially. After repeated bouts there were residual ptosis, mydriasis and external ophthalmoplegia. This is a rare presentation of complicated migraine. The first case was reported as early as 1854 by Notta and followed subsequently by others including the classical descriptions by Charcot. Friedman *et al* (1962) saw only eight cases among a total of 5,000 migraine patients over a period of 30 years. Moebius disputed the concept of migraine as the cause of 'ophthalmoplegic migraine' proposed by Charcot maintaining that an organic basis produced such symptoms. Some of these cases of so called 'ophthalmoplegic migraine' were at post-mortem found to

result from organic lesions of oculomotor nerves such as tumours, fibrous thickenings and aneurysms of the posterior cerebral or posterior communicating arteries. Angiographic studies of true ophthalmoplegic migraine have consistently shown no abnormalities (Lincoff and Cogan, 1957). Presently ophthalmoplegic migraine has been suggested either to be the result of compression by certain major vessels (Walsh and O'Doherty, 1960) or brain swelling with tentorial herniation of the hippocampal gyrus (Harrington and Flock, 1953). Although the oculomotor nerve is chiefly involved, the trochlear and the abducens are also affected.

The electroencephalographies of eight out of these ten patients were abnormal. Three of these had diffused theta and delta waves while the other five showed focal delta waves. Smyth and Winter (1964) in 202 records of migraine patients found no consistent abnormality while resting. 87 (43%) are said to be abnormal. Dow and Whitty (1947) noted occipital high voltage theta activity during the scotoma phase of the migraine suggesting that abnormalities are seen if records are made during or soon after the attack.

Angiograms were performed in six of these patients and they were all normal. This is in agreement with the reports of Pearce and Foster (1965) and Bradshaw and Parsons (1965). In 33 angiograms on 40 migraine patients, Pearce and Foster found only two vascular malformations. They concluded that angiographic investigation would be fruitless in a majority of cases. Following the vertebral angiography, in Case 2, there was an aggravation of his symptoms. He remained unconscious for three days after this procedure with the recurrence of left up going plantar response. According to Hauge (1954) who investigated a large number of patients with vertebro-basilar angiograms, it was his impression that patients with migraine are more sensitive to contrast injection, as even small amounts can induce symptoms, of nausea, vomiting, visual disturbance, vertigo, dysathria, changes in pupil size, mental disturbance and in rare cases stupor and coma.

Initially, the attacks usually result in complete recovery. With repeated attacks, there may be residual paralysis. Permanent deficits are not uncommon. Connor (1962) reported 18 cases with residual neurological deficits following migraine of varying presentations. In Case 1, residual facial palsy and monoparesis with hyperreflexia persisted after two months. The two patients with recurrent ophthalmoplegia had residual mydriasis and mild ptosis with extraocular palsy. Rarely death has been known to occur during an attack (Clarke, 1910).

The mechanism of migraine has been under intensive research recently. Graham and Wolff (1938) drew attention to the role of extracranial vessels and the effect of ergotamine tartrate thereby suggesting the significant part played by vascular mechanism in migraine headache. Wolff (1963) stated the beginning of headache in migraine is heralded by vasodilatation, though the vasodilatation induced by heat, histamine or acetylcholine iontophoresis does not cause pain (Ostfeld, 1962). Chapman *et al* (1960) found increased amounts of neurokinins in the oedema fluid from the scalp of migraine patients. Bradykinin injected into the carotid artery can induce ipsilateral headache (Sicuteri *et al* 1963). Ostfeld *et al* (1957) found that 5-hydroxytryptamine can induce migraine. Lance, *et al* (1967) showed that plasma serotonin level drops at the onset of migraine. Histamine has been shown by Schamacher and Wolff (1941) from their studies of histamine headache to cause a headache resembling migraine. Other factors like fluid and electrolytes and certain hormones as well as the family history are said to play a role in producing the headaches.

The cause of the phenomenon of complicated migraine is even less well understood. Though the clinical presentations are well described in the literature, its pathogenesis remains a matter of controversy. It is believed that 'spasm' or 'oedema' of cerebral vessels have induced the multifaceted picture. The area of brain supplied by the involved vessels becomes ischaemic and leads to neurological symptoms. This theory suggests involvement of different portions of the cerebral vessels for the varying clinical syndromes. Bruyn and Weenik (1966) believe that the whole manifestation of complicated migraine is the result of an affection of the brain stem rather than of the cerebral cortex, but our observations suggest that it is not only the involvement of basilar system but also of the internal carotid artery system.

SUMMARY

Ten cases of complicated migraine of varying features are presented. The mechanism, the symptomatology of these forms of migraine are briefly reviewed. The diagnosis of complicated migraine may be impossible to prove but a history of episodic attacks of neurological deficit which may precede headaches and return to normality in a short time should help in arriving at a diagnosis. A careful follow-up and assessment of individual patients will help in discovering an underlying pathology. There may not be a definite family history. Symptoms and signs suggest involvement of branches of internal carotid and basilar arteries.

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