We report a 19-year-old man presenting to the department of Psychiatry for the evaluation of prominent behavioral symptoms associated with episodic headaches, with normal inter-episodic periods. A diagnosis of classic migraine with hypomanic aura was made. Other possible co-morbid or causative illnesses were excluded and preventive therapy with valproate was started due to the prominent affective symptoms as a part of the migranous aura. With this the frequency of headaches gradually decreased over the next four months. He was followed up for 2 years when he was found to be symptom-free. Recent research into the mechanisms of migraine has identified that the cortical hyperexcitability and an imbalance between neuronal inhibition and excitement mediated by gamma-aminobutyric acid and excitatory amino acids respectively may be the underlying mechanism. The high rate of affective disorders in patients with migraine, association of migraine with an aura comprising of mood symptoms and good response to treatment with mood-stabilisers might give newer insights into the pathophysiology of mood disorder as well.

Key words: Migraine, hypomania, aura, sodium valproate.

Introduction

The diagnosis of migraine with aura, requires at least two migraine attacks with any three of the following four features: [a] One or more fully reversible aura symptoms (which could be visual, sensory or speech disturbance); [b] aura developing over a course of more than four minutes; or [c] aura lasting less than sixty minutes; and [d] headache following aura within sixty minutes. Here we present a case of migraine with aura with atypical features, who presented to the department of Psychiatry for the prominent behavioral symptoms as part of the aura of classic migraine. He would fall in the subtype of probable migraine with aura with atypical features (lasting longer than 60 minutes). His clinical course, response to treatment and condition at follow up is presented. Overlap of mood disorders and migraine might have implications for common biological etiology, life style factors, reactive factors to the transiently disabling and painful neurological illness and common treatment factors.
and serum electrolytes, blood biochemistry were all within normal limits. A normal Magnetic Resonance imaging of brain and Electroencephalogram (EEG) ruled out any structural pathology and seizure activity. The differential diagnoses considered were temporal lobe epilepsy, migraine without aura associated to hypomanic symptomatology and migraine with aura. Since the patient had no other symptoms suggestive of seizure equivalents, had full recall of events that occurred in the entire episode, had other associated symptoms of migraine with aura and had a normal EEG tracing, the diagnosis of epilepsy was not considered. Hypomanic symptoms occurred in close association with migraine attacks, preceding the headache onset by 15-20 minutes and lasting during most of headache period. He had no hypomanic symptoms without the occurrence of headache.

Initially patient was started on Tab propranolol 40 mg once daily, with which he had symptomatic postural hypotension. Propranolol was then substituted with Tab sodium valproate (VPT), as this could help both the migraine and mood symptoms. VPT was started at a dose of 200 mg once daily and was gradually increased to 200 mg thrice daily according to the clinical response and serum level of valproate, optimizing between response and toxicity. On a daily dosage of 600 mg of valproate, he maintained a serum level of 90 µg/ml and had a good clinical response.

Follow up at 2 years: This patient had responded well to treatment. The frequency of headache reduced slowly over the four months following the initiation of therapy. He did not have a single episode of headache in the last one-year.

**Discussion**

There is a strong cross-sectional relation between affective disorders and migraine. Mood change is also one of the commonest prodromal (premonitory) symptoms among migraineurs. Psychiatric factors and migraine may interact in three general ways, etiologically, psychophysio logically or biobehaviorally and comorbidly (the two disorders coexist). The relation between the two disorders may be a result of chance or one disorder can cause another disorder (cause-effect relationship). Yet others have speculated that there might be shared environmental risks between migraine and mood disorders. Last, but not the least, there may be environmental or genetic risk factors that produce a brain state giving rise to both conditions, that is, there may be some common biology underlying both conditions. Depression and anxiety are more common with migraine as comorbid illnesses, as compared to mania or hypomania. In our current case, mood symptoms occurred as a part of the migraine headache more specifically the aura. It is worth mentioning that the patient had no mood symptoms inter-morbidly. In our opinion, mood symptoms occurring as part of migraine aura are rare. Another possibility to be considered in this patient would be premonitory symptoms. These symptoms that warn of an impending migraine headache have been recognized for many years. However, the ability of premonitory symptoms to accurately predict the migraine attack is doubtful. In the above study, the most common premonitory symptoms noted were feeling tired and weary (72% of attacks with warning features), having difficulty concentrating (51%) and a stiff neck (50%). Subjects who functioned poorly in the premonitory phase were the most likely to correctly predict headache. Another study reported the occurrence of prodromal symptoms in about a third of migraine patients that lasted on an average of 7-9 hours.

The most common symptoms were tiredness, mood change and gastrointestinal symptoms. Although it is difficult to ascertain whether the hypomanic symptoms in our patient were prodromal symptoms or occurred as part of aura; the possibility of them being symptoms of aura is higher as all those episodes were followed by occurrence of migraine headache.

The patient had complete remission of symptoms after treatment with a potent mood stabiliser (VPT). Currently the role of VPT in migraine prophylaxis is well documented.

In conclusion, mood changes such as hypomanic features may occur as part of migranous aura and should be clinically recognized as such. Sodium valproate is effective in these cases and leads to remission of symptoms. This observation may imply common pathophysiologic mechanisms underlying migraine and mood disorders. However, it is emphasized that the association of hypomania and migraine and their improvement with VPT could be a co-incidence and this finding needs to be corroborated with more case reports.

**References**