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EEG CHANGES IN MIGRAINE PATIENTS

TAPASWINI MISHRA*, DIPTI MOHAPATRA, MANASI BEHERA AND NIBEDITA PRIYADARSINI

Department of Physiology, IMS & SUM Hospital, Siksha O Anusandhan University, Bhubaneswar, Odisha, India.

ABSTRACT

Migraine is a common, debilitating disorder that imposes a large personal burden on sufferers and high economic costs on society. Sufferers have a significant level of migraine related disability in all aspects of their daily lives, including employment, household work and non-work activities. Since there is a wide range of disorders that cause headache, a systematic approach to classification and diagnosis is an essential prelude to clinical management and to useful research. The EEG is frequently used in the examination of patients with migraine now days.

KEY WORDS: **EEG, migraine, headache, hyperventilation, photic stimulation, FIRDA**

*Corresponding author

TAPASWINI MISHRA
Department of Physiology, IMS & SUM Hospital, Siksha O Anusandhan University, Bhubaneswar, Odisha, India.

INTRODUCTION

Migraine is associated with a variety of electroenphalographic (EEG) changes. EEG abnormalities in migraine have been reported by a number of authors. Prevalence varies considerably in the older literature. A number of specific rhythms related to drowsiness or hyperventilation have probably been counted as “abnormal “and the reported numbers of definitely abnormal EEG rhythms have been consistently low.In a few controlled and blinded studies, however, a slight excess of various EEG rhythms has been found in migraine patients. Similar prevalence of interictal EEG abnormalities has been found in patients with classic and common migraine, but the diagnostic classification may not have been precise enough in some studies.

HEADACHE AND MIGRAINE

Headache is one of the humanity’s most common afflictions. A headache or cephalalgia is pain anywhere in the region of the head or neck. It can be a symptom of a number of different conditions of the head and neck.There are more than 200 types of headaches. Some are harmless and some are life-threatening. Headaches are broadly classified as "primary" or "secondary" [1]. Primary headaches are benign, recurrent headaches not caused by underlying disease or structural problems. For example, migraine is a type of primary headache. While primary headaches may cause significant daily pain and disability, they are not dangerous. Secondary headaches are caused by an underlying disease, like an infection, head injury, vascular disorders, brain bleed or tumors. Secondary headaches can be harmless or dangerous. Certain "red flags" or warning signs indicate a secondary headache may be dangerous [2]. The International Classification of Headache Disorders (ICHD) is an in-depth hierarchical classification of headaches published by the International Headache Society. It contains explicit (operational) diagnostic criteria for headache disorders. The first version of the classification, ICHD-1, was published in 1988. The current revision, ICHD-2, was published in 2004[3]. The classification uses numeric codes. The top, one-digit diagnostic level includes 13 headache groups. The first four of these are classified as primary headaches, groups 5-12 as secondary headaches, cranial neuralgia, central and primary facial pain and other headaches for the last two groups [4]. The ICHD-2 classification defines migraines, tension-types headaches, cluster headache and other trigeminal autonomic cephalalgias as the main types of primary headaches [5]. Migraine is a chronic neurological disorder characterized by recurrent moderate to severe headaches often in association with a number of autonomic nervous system symptoms .Typically the headache affects one half of the head, is pulsating in nature, and lasts from 2 to 72 hours. Associated symptoms may include nausea, vomiting, and sensitivity to light, sound, or smell. The pain is generally made worse by physical activity. Up to one-third of people with migraine headaches perceive an aura: a transient visual, sensory, language, or motor disturbance which signals that the headache will soon occur. Occasionally an aura can occur with little or no headache following it[6]. Migraines are believed to be due to a mixture of environmental and genetic factors [7]. About two-thirds of cases run in families [8]. Changing hormone levels may also play a role, as migraines affect slightly more boys than girls before puberty, but about two to three times more women than men [9,10]. The risk of migraines usually decreases during pregnancy. The exact mechanisms of migraine are not known. It is, however, believed to be a neurovascular disorder [8]. The primary theory is related to increased excitability of the cerebral cortex and abnormal control of pain neurons in the trigeminal nucleus of the brainstem [11].Migraines typically present with self-limited, recurrent severe headache associated with autonomic symptoms [8,12]. About 15–30% of people with migraines experience migraines with an aura [13,14] and those who have

migraines with aura also frequently have migraines without aura. The severity of the pain, duration of the headache, and frequency of attacks is variable [10]. A migraine lasting longer than 72 hours is termed status migrainosus[15]. There are four possible phases to a migraine, although not all the phases are necessarily experienced [6]: 1.The prodrome, which occurs hours or days before the headache 2.The aura, which immediately precedes the headache 3.The pain phase, also known as headache phase 4.The postdrome, the effects experienced following the end of a migraine attack. Migraines are believed to be a neurovascular disorder[8] with evidence supporting its mechanisms starting within the brain and then spreading to the blood vessels . Some researchers feel neuronal mechanisms play a greater role [16], while others feel blood vessels play the key role[17]. Others feel both are likely important [18]. High levels of the neurotransmitter serotonin, also known as 5-hydroxytryptamine, are believed to be involved. Aura:Cortical spreading depression, or spreading depression according to Leão, is bursts of neuronal activity followed by a period of inactivity, which is seen in those with migraines with an aura. There are a number of explanations for its occurrence including activation of NMDA receptors leading to calcium entering the cell. After the burst of activity the blood flow to the cerebral cortex in the area affected is decreased for two to six hours. It is believed that when depolarization travels down the underside of the brain, nerves that sense pain in the head and neck are triggered. Pain:The exact mechanism of the head pain which occurs during a migraine is unknown [19]. Some evidence supports a primary role for central nervous system structures (such as the brainstem and diencephalon) [20] while other data support the role of peripheral activation (such as via the sensory nerves that surround blood vessels of the head and neck). The potential candidate vessels include dural arteries, pial arteries and extracranial arteries such as those of the scalp [19]. The role of vasodilatation of the extracranial arteries, in particular, is believed to be significant [21]. The utility of EEG in the diagnosis of headache has been controversial. EEG abnormalities in headache were originally described by Engel et al. During the past 50 years, a variety of electroenchalographic abnormalities have been reported in patients with migraine, with an incidence ranging from 11% to 74% [22].Although EEG is not useful in the routine evaluation of headaches including migraine, but may be benefit in those headaches which have an unusual symptoms, suggesting possible seizure disorder [23]. It is claimed that EEG is clearly indicated in cases with acute headache attacks, when epilepsy, basilar migraine, migraine with prolonged aura or alternating hemiplegic migraine is suspected [24], keeping in mind that the final judgment that the final diagnosis must mainly depend on clinical judgment however.

DISCUSSION

There are several conflicting reports about the EEG changes in migraine patients.Weil described paroxysmal high voltage abnormal slow wave activity in a number of patients and proposed the term dysrhythmic migraine for this group [23,24]. Dow and Whitty found that relatives of migrainous subjects also had a high incidence of EEG abnormalities providing circumstantial evidence for a constitutional basis for migraine. Since migraine is likely to be a constitutionally determined disorder, it is of interest to know if the EEG of such patients shows characteristic features. A few controlled and blinded studies have shown focal slow activity in 0-15% and spikes in 0.2-9% of patients with migraines generally not different from control group [25-28]. However spikes are reported more common in migraineurs than in headache free control subjects by other authors[29].Lauritzen et al and Westmoreland [30,31] reported showing, depression of background amplitude and also normal EEG during visual aura of migraineurs, others [32-34]showed definite unilateral or bilateral delta activity during attacks of basilar and hemiplegic migraines. De Carlo et al [35] reported usefulness of EEG particularly in children suffering from migraine with aura

during ictal phase, because they found abnormalities in 80% of cases. Gallo and Winter[36] described persistence of photic driving to 20 Hz flashes or above (H-responses) in nearly 90% of patients with idiopathic and post-traumatic migraine like headache and epilepsy, while 80% of headache free subjects lacked response above 14 Hz.Lack of alpha blocking during intermittent photic stimulation, a trend towards a greater driving response, and an increased alpha power asymmetry are also reported [37-41]. Appearance of slow wave and disturbance of consciousness have been reported in classic migraine with different clinical presentations (global amnesia, stupor, clouding of consciousness) [42-44]. Lauritzen demonstrated that reduction of cerebral blood flow is not of sufficient magnitude to explain the focal symptoms and also abnormal discharges in classic migraine, rather it may cause a neuronal dysfunction[45].Puca F et al, De Tommaso M et al[39] and Kramer U et al have reported slowing with an interhemispheric asymmetry of alpha rhythm in interictal EEGs of both MWO and MWOA.Schoermen and Francesco did not report slowing during interictalpahse of the migraineurs. Woman had more slowing in some reports [46,47].To differentiate attack of migraine from epilepsy, Panayiotopulos[48,49]concluded that if each characteristic of visual aura is identified, the diagnosis of migraine is easy. Brincicoth[50]showed that the presence of specific clinical features (amaurosis, scotoma and positive family history) together with bilateral EEG abnormity and no changes during IPS is related to migraine. In recent study[51] periodic lateralized epileptiform discharges (PLEDs) were reported.Several types abnormal EEG were reported in basilar migraine including 1) an excess beta activity in the ictal phase in children [52], 2) predominant delta activity during attack of headache and normal EEG during attack [53], 3) slowing in posterior region or slowing with spikes and sharp wave complexes [54-56] and 4) unusual association of acuteconfusional state with FIRDA (frontal intermittent rhythmic delta activity) during attack of migraine [56].Several authors applied EEG mapping to migraine patients who were assessed during the interictal period or in course of a spontaneous attack. The principal finding obtained between attacks was the increase in the power of the rapid activities, particularly in the range of the beta bands [57]and especially in the temporal regions, both in migraine patients with aura (70% of cases) and without aura [58]. Lia et al [41] demonstrated in 39.3% of the patients examined (17 with migraine without aura and 11 with migraine with aura) an increase in the relative power of theta and delta activity bilaterally in the parietal-occipital regions, with a reduction in the power of the alpha rhythm compared with control subjects. Similar results were obtained by Guidetti et al[59], but they were unilaterally located and consistent with the side of pain in the majority of attacks. In the study by Neufeld et al [60], significant differences emerged only between control subjects and patients with migraine with aura. These consisted of a decrease in the absolute power peak of the alpha band in the latter group. Analogous findings were obtained by Polich et al [61], who also found an increase in theta band power in the central-parietal regions. Other authors detected interhemispheric asymmetries concerning the alpha bands. In a study carried out by Jonkman and Lelieveld[62], almost 55% of patients showed the above asymmetries in the alpha band, compared with 5% of control subjects, in terms of absolute power (55% of cases) and peak frequency (20% of cases).The finding of an asymmetry in the alpha band is probably less constant in patients with migraine without aura than in those with migraine with aura. In fact, Facchetti et al [40] found an asymmetry in alpha absolute power in 41.9% of patients with migraine with aura, whereas in 54.8% there was an asymmetry of the peak frequency. The temporal relationship between attacks and asymmetry of bioelectrical activity is complex. In this regard, Nyrke et al [63]carried out several EEG recordings at different time intervals relative to the crises and found an increase in bioelectrical abnormalities mostly concerning the alpha band in the period immediately preceding the attacks compared to 10 days

before.Hemiplegic migraine has shown the most definitely abnormal EEGs [64,22]Pseudo periodic slow sharp waves over the hemisphere contralateral to the hemiplegia have also been recorded.9

CONCLUSION

EEG findings occur with other neurophysiological experiences indicating migraine is a condition characterized by altered neuronal excitability due to peculiar biochemical milieu favouring a greater susceptibility to depolarization. These alterations are also present in the interictal period and could reflect the bioelectrical threshold for migraine attack. The EEG patterns observed in migraine patients seem to suggest a possible physiological connection between sleep, hyperventilation and photic stimulation. Therefore the EEG studies on migraine patients may shed new light on its pathophysiology as well as management and prognosis.

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