EVALUATION OF PATIENTS WITH HEMIPLEGIC MIGRAINE IN MACKENZIE HEADACHE CLINIC


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ABSTRACT

Background: The sporadic type of Hemiplegic Migraine (HM) is sometimes observed among migrainous patients (MP) and mimics ischemic strokes.

Methods: In an evaluation of two-hundred consecutive adult MP in the Mackenzie headache clinic, Canada during 2004, 9% of the patients met the criteria established by the International Headache Society for sporadic HM. Female to male sex ratio, family history of migraine, frequency of epileptic seizures, migraine status and migraine aura without headache were investigated in the HM group and compared with other MP. The relationship between side of hemiplegia and distribution of head pain was also evaluated.

Results: All of these clinical dimensions were significantly more common in the HM group than in the other MP. All of the patients with HM had other types of aura. Hemiplegia was often ipsilateral to the side of the headache. These results highlight the importance of documenting the side of hemiplegia in relation to the headache side, as well as determining the history of seizures in HM patients.

Conclusion: This study supports the argument that migraine is a disease which may appear with different or mixed presentations in each episode.


Keywords: Hemiplegic, Migraine, Sporadic.

INTRODUCTION

The sporadic type of Hemiplegic Migraine (HM) is a subtype of migraine with hemiplegia in the prodromal phase of the headache. Raskin¹ and Moskowitz² believed that in HM, the hemiplegia is generally contralateral to the headache side and the side affected by the hemiplegia can vary from attack to attack. Raskin¹ described a more profound form of HM in which the hemiplegia often affects the same side as the migraine headache and persists for days after remission of the headache. Sporadic HM is defined as migraine attacks associated with some degree of motor weakness or hemiparesis during the aura phase and where no first degree relative (parent, sibling or child) has identical attacks.¹ The degree of motor deficit is highly variable, ranging from mild clumsiness to total hemiplegia. The affected limbs will feel both heavy and dead during the attack. Attacks of hemiplegia may alternatively be right or left sided or always involve the same side.¹ The hemiparesis is often accompanied by hemihypesthesia. Dysarthria and dysphasia may also be seen in some HM patients.¹ Presentation and order of aura symptoms in sporadic HM is similar to those in typical migraine aura. Until recently, the literature described about 215 sporadic HM cases worldwide.¹ In this paper clinical characteristics of patients with HM were evaluated and compared with other migrainous patients (MP).

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PATIENTS AND METHODS

Two-hundred consecutive adult MP aged more than 18 years enrolled in a prospective study in Mackenzie headache clinic, Canada during 2004. Diagnosis of migraine and sporadic HM were made by a neurologist based on the criteria developed by the International Headache Society.3,5 Patients with mixed headache, e.g. migraine and tension headache, were excluded. In this group sex ratio, family history of migraine, frequency of epileptic seizures, migraine status and migraine aura without headache were evaluated and compared with other MP. A diagnosis of epilepsy was given when clinical manifestations of at least two unprovoked seizures were observed but in many of the patients the EEG showed typical epileptic discharges (spike, sharp, spike and wave, sharp and wave).6 In our migrainous patients, those experiencing continuous migraine with a unilateral, throbbing and disabling pain, lasting more than 72 hours,7 were considered as status migrainous. Other subtypes of aura including visual, sensory and dysphasic auras were investigated in the HM group. Patients with sporadic HM were classified into four groups: (1) hemiplegia contralateral to headache side, (2) hemiplegia ipsilateral to headache side, (3) relationship of hemiplegic location to headache side varies across headache episodes, and (4) hemiplegia presents with bilateral headache.

RESULTS

In our two-hundred MP (154 females, 46 males), 18 patients (15 females, 3 males) or 9% had sporadic HM. Gender ratios in the HM and other MP were comparable with a ratio of 83% females to 17% males in the HM group and 71% females to 29% males in the other MP. Table I shows a comparison of family history of migraine, frequency of epileptic seizures, status migrainous and migraine aura without headache between the two study groups. All of the clinical dimensions examined were more common in the HM group than in the other MP. All of the patients with HM experienced some form of visual aura such as dazzling zigzag lines, glistening points of light and blurred vision that occurred at some point in the course of each headache either alone or accompanied by hemiparesis. Sensory aura, e.g. hemihypesthesia or hemiparesthesia, was present in 61% of the HM group (73% females, 27% males). Dysarthria or dysphasia were present in 27% of the patients (60% females, 40% males). The auras in our HM patients spreaded with a slow march of symptoms, usually starting with visual, then sensory and finally motor and aphasic symptoms. In our HM group, 5.5% of patients suffered of basilar migraine (100% females). According to the relationship between hemiplegic location and headache side, the patients were divided into four groups as shown in Figure I. In the first group, which was made up of 27.7% of HM patients (75% females, 25% males), hemiplegia was contralateral to the headache side. In the second group consisting of 50% of HM patients (88% females, 12% males), hemiplegia was ipsilateral to the headache side. In the third group made up of 5.5% of HM patients (100% females) hemiplegia presented either contralaterally or ipsilaterally to the headache side. In the fourth group consisting of 16.6% of HM patients (75% females, 25% males), hemiplegia was present with bilateral headache.

DISCUSSION

The higher female to male ratio of our HM patients in comparison to other MP is similar to female/male ratio; 4.5/1 of sporadic HM patients in Denmark.7 Family history of migraine was more common in our HM group than the other MP. In the Danish population, first degree relatives of all sporadic HM probands had an increased risk of migraine with or without aura.7 Epileptic seizures were approximately ten times more frequent in the HM group than the other MP. This significant co-occurrence has been suggested to be caused by a dys-

Table I. Comparison of clinical data between patients with sporadic hemiplegic migraine (HM) and other migrainous patients (MP).

<table>
<thead>
<tr>
<th>Clinical Data</th>
<th>Patients with sporadic HM (n=18)</th>
<th>Other MP (n=182)</th>
</tr>
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<tbody>
<tr>
<td>Family history of migraine</td>
<td>55%</td>
<td>43.5%</td>
</tr>
<tr>
<td>Epileptic seizures</td>
<td>12%</td>
<td>2.4%</td>
</tr>
<tr>
<td>Migraine status</td>
<td>55%</td>
<td>47%</td>
</tr>
<tr>
<td>Migraine aura without headache</td>
<td>61%</td>
<td>30%</td>
</tr>
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</table>
function of the motor cortex in patients which may manifest as convulsions or as a motor aura. However, no mechanism common to both epilepsy and migraine has been established to date. In our HM group, the slow spread of the aura symptoms and their temporal succession clearly indicates a phenomenon spreading contiguously in the cerebral cortex. The only known phenomenon that can spread in this fashion is a cortical spreading depression of Leao, the mechanism thought to underlie typical migraine with aura. The described clinical findings support that sporadic HM symptoms are caused by a cortical spreading depression. There was always more than one aura symptom and sometimes all typical auras were present in our HM patients. Thus the cortical spread of the symptoms is more extensive and larger area of brain are affected by the aura in HM patients than other MP. In support of this data, Olsen's study with Single Photon Emission Computed Tomography showed that wave of spreading oligemia in the phase of aura does not respect arterial boundaries. It can be concluded that the oligemia is secondary to cortical hypometabolism and may spread from occipital to motor cortex. We often observed hemiplegia ipsilateral to the headache side. This data is in opposition to the hypothesis linking the aura to the painful phase of the migraine. Our results are congruent with the cross-over of corticospinal tracts because the headache side is not often the side of cortical dysfunction. Thomsen's review of sporadic HM revealed that the pain may be unilateral or bilateral, and if one sided it may be ipsilateral or contralateral to the weak limbs. Frequently, the pain will switch sides or become bilateral during the attack. Whenever headache follows on the side opposite of the aura symptoms, it suggests that intracerebral and extracerebral disturbances take place on the same side of the head. However, ipsilateral headache is surprisingly common, particularly in people referred to specialist clinics, presumably because they are considered to be unusual by the referring physician. Ipsilateral headache is even found in patients who collect information about their attacks in a prospective basis. Therefore, it is improbable that a direct connection exists between the aura symptoms and the headache side. It is more likely that both aura and headache reflect a common pathogenetic mechanism which produces each symptom on one or other side of the head.

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