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Migraine with visual aura in developing age: visual disorders

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Abstract. – Visual disorders are an important symptom in the migraine of developing age. Different kinds of visual disturbances can precede, accompany or follow a migraine attack. These visual disturbances can be grouped into negative (hemianopsia, quadrantopsia, scotoma) and positive (phosphene, teicopsia, metamorphopsia, macropsia, micropsia, teleopsia, diplopia, dischromatopsia, hallucination disturbances) disorders. The pathogenetic mechanism of the visual phenomena of migraine has not yet been clarified. Various hypotheses have been proposed: vasospasm with consequent ischemia of some cerebral areas, the opening of arteriovenous shunts between the intra and extra cerebral circulation, the formation of microthrombi in arterioles and dopaminergic hypersensivity of some nervous centers. We have studied 1787 children, affected by migraine with (13%) or without (87%) aura. Among the patients, 211 (12%) referred visual disorders, especially scotoma and phosphene. These data let us hypothesize that a relationship between migraine and visual disorders is present also in pediatric age. However this relationship is less important than in adults.

Key Words:

Visual disorders, Migraine.

Introduction

Visual disorders are considered as the most representative symptoms of migraine with aura and can accompany the pain and exceptionally persist after the end of the crisis[1]. Such disorders can last from a few minutes to one or two hours; their persistance can be the first expression of an involvement of the optical nerves and/or of the retina secondary to phlogistic or invasive processes[2]. These disorders can alter vision through a visus impairment (negative phase) or the appearance of excitatory phenomena (positive phase). These two phases can follow each other as in flittering scotoma or appear separately[3]. Negative visual disorders are amaurosis (a transient mono or bilateral total blindness) and hemianopsia (absence of the visual function in half of the visual field). Positive visual disorders are: phosphenes, teicopsias, metamorphopsias, macro or micropsia and teleopsias. When there are multiplied images we have diplopia or poliplopia. The aim of our study was to identify the possible association between migraine and visual disorders in evolutive age and to define which of the visual disturbances is more common in childhood.

Materials and Methods

We have examined 1787 children referred to our Unit between 1981 and 1995. The examined group consisted of 943 males and 844 females; age range was 3-15 years (mean age 6.6 years). Two hundred thirty patients (13%) were affected by migraine with aura and 1557 (87%) by migraine without aura. In order to rule out secondary cephalalgia, a series of laboratory and instrumental exams were requested; biohumoural and hematochemical tests, urinalysis, X-rays of the skull and of the paranasal sinuses, visual examination with visual field, EEG. All examinations resulted within normal limits. Moreover we have considered the relationship of any visual disorder with the following risk factors: headache index, familiar

trait, recurrent abdominal pain (RAP), limb pains, cyclic vomiting, kinetosis, sleep disorders, vertigo, hyperactivity. In the group of patients with aura, 211 (12% of the total of patients studied) referred visual disorders; of these patients 96 (14%) were males and 118 (56%) females.

Results

Among the visual disorders present in the migraine with aura group, we observed: phosphenes, scotomata, foggings, teichopsias, amaurosis, diplopias, photopsias, hemianospias, quadrantic hemianopsias, metamorphosias. There were no hallucinations and dyschromatopsias, which are more frequent in adults. We recordered the frequency of each single disorder: phosphene[4] were present in 74 patients (37,7%) and scotoma in 49 (25%), thus demonstrating a possible concurrence between positive and negative disorders. Furthermore, among the patients positive for visual disorders, 65% were positive for familiar trait, and 50% had a positivity of cephalalgic risk factors, such as hyperactivity. Finally, 70% of visual disorders accompanied the migraine attack while 30% were prodomic. Scotoma showed a peculiar characterist, since it appeared more frequently as a prodromic symptom (64%) than as an epiphenomenon (36%).

Discussion

No single etiopathogenic theory is able to explain the visual phenomenon that accompany migraine attacks. Many authors suggest that a vascular mechanism of ischemic type is at the origin of aura. The first studies in this field are due to Wolff et al[4] who demonstrated that visual disorders regressed with amyl nitrite and appeared with ergotamine. The evident reduction in the cerebral flow during aura has been shown by Skyhøj et al[5] and Olsen et al[6] by intra-arterial infusion of Xenon 133 under tomographic control and by Nattero et al by Doppler ultrasound[7]. Some authors[8] have shown that the opening of arterial-venous shunts between the intra-and-extracerebral circulation can determine a flow reduction at the level of cerebral cortex with consequent ischemia that could be secondary to the formation of arteriolar microthrombis[9]. In Leao's Child Depression Scale (C.D.S.)[10] visual disorders have been correlated to an intense initial neuronal excitation, provoked by different nociceptive stimuli, followed by a wave of extremely reduced electrical activity propagating in the postero-anterior sense[11,12]. C.D.S. has been experimentally reproduced both in animal models and in man[13,15]; the most employed experimental methods have been the electrical or mechanical stimulation and the injection of high KCl concentrantions. This latter technique shows the importance of the biochemical activity of some substances[16-18]. Different studies have evaluated the role of ATP, pH, and phosphocreatine intracellular concentrations[19]. N-methyl-d-aspartate (NMDA) has been identified as one of the most important receptors for C.S.D. triggering and propagation[20]. Recently, it has been observed that in patients affected by migraine with aura, plasmatic glutamate levels are high[21]. Some authors have correlated this finding to low levels of magnesium which facilitates the development of a vasoconstriction[22]. Anyhow, vasomotor activity is connected to hormones and neurotransmitters activity. According to Sicuteri[23], essential headache is the expression of a specific disorder of the nociceptive system on which the pain-producing functions depend[24]. Many substances act at this level: serotonin, catecholamines, bradykinin, angiotensin, endorphins, P-substance, etc[25]. Migraine symptoms including visual ones, could be related to a dopaminergic hypersensitivity of some nervous centres[19]. It is believed that visual disorders are due to an involvement of the optic pathways (retina, optic nerve, chiasm, optic tracts, cortex). When the disorder is monolateral (scotoma, amaurosis, hemianopsias) it originates from the retina or the optic nerve or the lateral portion of the chiasm. If the disorder is bilateral, its most likely origin will be in the central part of the chiasm; when the campimetric defect is bitemporal, or in the optic

tracts of the cortex, then it will be lateral and homonymous[26]. The common total amauroses, and rarely foggings, originate from an involvement of both the optic tracts or occipital areas. As far as excitation phenomena are concerned, phosphenes and teichopsia originate from the occipital cortex where they have also been experimentally reproduced by electric stimulation[27]. Perceptive distortions (both micro and macro-metamorphopsias and teleopsias) and visual hallucinations originate from the tempoparietal cortex.When other disorders such as vertigos, diplopia, ataxia, alternating paresis, mental confusion and nystagmus are associated to the visual symptomatology of cortical origin, probably all areas served by the vertebrobasilar arteries are involved[28]. Data emerging from our study suggest that in evolutive age visual disorders appear both as prodromic and accompanying phenomena of migraine, thus showing a diagnostic relevance. For this reason,their presence does not indicate an organic damage unless they persist for more hours. However, it should not be forgotten that migraine and aura in particular, often scare the small patient and his/her parents. When aura appears a prodromic sympton, it is experienced with anxiety and fear and visual disorders as accompanying factors worry both the patient and parents. We believe that a better and his/her deeper understanding of this symptomatology can prevent this syndrome in a great number of small patients who, otherwise, in the greater majority of cases, are likely be destined to develop severe forms of migraine over the years.

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