Although a link between epilepsy and migraine has been claimed since 1907 because their clinical symptoms may overlap and a genetic susceptibility could explain this complex link [1], the hypothesis of co-morbidity of epilepsy and migraine in adults is mainly based on a single study that reported a 2.4-fold increased risk of migraine in a cohort of nearly 2000 patients with epilepsy compared with their relatives without epilepsy [2]. In this issue of the Journal, Duchaczek and associates investigate the frequency, clinical characteristics and treatment of pericentral headache in patients with epilepsy, and they assess epidemiologically the concept of co-morbidity of epilepsy and interictal headache/migraine [3]. The authors raise some doubts on the widely held concept of co-morbidity of the two conditions, as in their study pericentral headache and, in particular migraine, is not encountered more often in patients with epilepsy than expected in the general population [1]. Furthermore, they found that pericentral headache occurs in more than one-third (35%) of patients with epilepsy [3].

Interestingly, there are recent conflicting findings concerning this topic, which can be explained by the co-occurrence of confounding variables used according to the different sampling methods and study designs [4]. Noteworthy, while epilepsy and migraine share overlapping physiopathological mechanisms [1], the prevalence and incidence of these disorders in the general population, including all stages of life, differ [5]. There is no conclusive evidence of a real relationship between the two disorders; however, migraine and epilepsy co-morbidity, beyond any reasonable doubt, clearly differs in children when compared with adults. Children are more likely to have long-lasting ictal autonomic manifestations both in epilepsy and in migraine attacks, while ictal autonomic manifestations (both in epilepsy and in headache) in adults are usually associated, simultaneously or sequentially, with other motor or sensory ictal signs [1,6].

In patients with epileptic seizures, headache can occur as an interictal, preictal, ictal or postictal symptom, making differentiation of migraine from epilepsy difficult in some patients [6]. Misdiagnosis of occipital seizures with migraine and vice versa is common, especially in the pediatric age [6]. For these reasons it is difficult to obtain firm conclusions on this hot topic deriving only by epidemiological data [4].

Of note, temporal association between migraine and epilepsy symptoms differs. Preictal, pericentral and, especially, postictal headache may have the clinical characteristics of migraine without aura as well as of tension-like headache. In particular, among these three forms of epilepsy-related headaches, ictal headache is the rarest and also the least understood. Over the last two decades, the medical literature has been flooded with single case reports of ictal headache as the sole clinical manifestation and, recently, the term ‘ictal epileptic headache’ has been proposed to depict an electroencephalogram (EEG)-recorded ictal seizure with migraine/headache-like features [1]. The second edition of the International Classification of Headache Disorders (ICHD-II, 2004) [7] distinguishes three disorders, namely, migraine-triggered seizures (migralepsy), hemicrania epileptica (HE) and postictal headaches. Although headache with migraine features is commonly a postictal phenomenon, occurring in about 50% of the epileptic patients, only a few case reports of migralepsy and HE have been published, despite the fact that migraine and epilepsy are among the more common brain diseases [8]. In their study, Duchaczek and associates [3] found that pericentral headache was quite frequent (35% of the patients), but underestimated. We agree that recognition of headache as an epileptic manifestation per se still represents a challenge, and preictal and ictal headaches are often neglected because the seizure overshadows the headache for both the patient and the physician.

The occipital lobe is deemed to be the brain structure most responsible for both the development of migraine and occipital lobe epilepsies [8]. Of note, ictal discharges originating in the occipital cortex may remain localized or spread to adjacent areas. The patients’ ability to recall visual symptoms points to initial localization of the ictal discharge near the calcarine fissure, followed by slow propagation to an adjacent area. When the discharge spreads outside the occipital cortex, the most frequent ictal pattern is a sequence of epigastric discomfort, unresponsiveness and vomiting. Moreover, the symptom cluster of visual aura, abdominal discomfort, vomiting and headache can make clinical differentiation between occipital seizures and migraine particularly difficult. In this sense, the nature of pericentral or ictal headache as an epileptic
manifestation could be diagnosed only by ictal EEG recordings [1,8].

In view of the above uncertainties, further epidemiologically studies are warranted to better delineate the complex link between epilepsy and migraine. The clinical depiction and the EEG recording of the temporal sequence between headache and epilepsy symptoms [8,9] are imperative to achieve clarity and consensus in this area of research. According to the ICHD-II classification criteria [7], the concepts of migralepsy and HE are currently challenged, thus a revision of terminology and classification has been proposed by our group [8] in order to better define the most common ‘epilepsy–migraine sequence’ than a ‘migraine–epilepsy sequence’ (i.e. migralepsy) [6].

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