Internalizing and externalizing symptoms in preschool and school-aged children with epilepsy: Focus on clinical and EEG features

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1. Introduction

Children with epilepsy have an increased risk for emotional and behavioral problems [1,2]. The International League Against Epilepsy (ILAE) estimates that psychiatric comorbidities in children with epilepsy occur in around 35–50% of cases, and that these rates are even higher, i.e., over 50%, in children with additional intellectual disability (ID) [3]. Moreover, psychiatric disorders occur in children with epilepsy more often than in children with other chronic disabling illnesses, such as diabetes and asthma [4,5]. The most common psychiatric comorbidities in children with epilepsy are attention-deficit/hyperactivity disorder (ADHD), depressive disorders, and anxiety disorders [6]. Such conditions may be present in the early phase of the illness [7], or even precede the epilepsy onset [8].

Mechanisms and risk factors for psychopathology in epilepsy have been long studied [9] leading to several hypotheses that, in part, explain the high prevalence of those comorbid conditions. Firstly, the distressing and unpredictable nature of seizures and the social stigma could influence the psychosocial development, and negatively affect quality of life, fostering psychopathology [10,11]. Moreover, coexisting neurological comorbidities, frequently occurring in children with epilepsy, may contribute to the development of emotional and behavioral disturbances. In particular, population-based prevalence studies have reported the ID as the most common neurological comorbidity in children with epilepsy [30–40%] [12]. Cognition, indeed, is an intrinsic component of children’s personality development, and mild-to-moderate cognitive deficits have been associated with higher rates of emotional and behavioral problems [9]. Antiepileptic treatments could also play a role in developing psychiatric comorbidities, since they may produce emotional, behavioral, and cognitive adverse effects [13]. Furthermore, epileptogenic networks may be associated with enduring changes in brain function and structure, possibly resulting, through many different mechanisms, in emotional and behavioral alterations [14]. Although the available knowledge on the role of the epileptic focus in developing psychiatric comorbidities is only partial, some evidence suggests that its location may influence the psychopathological outcome [15]. Individual genetic and neurobiological underpinnings should be, finally,
considered as additional independent factors possibly predisposing to epilepsy-psychopathology comorbidity. Indeed, the association between behavioral problems and epilepsy may be bidirectional. Epidemiological studies suggest that anxiety and depression may precede the onset of epilepsy and act as independent risk factors for the development of uncomplicated epilepsy and behavioral disorders [18]. Furthermore, animal models of anxiety and depression, and humans suffering from these disorders, may share several mechanisms facilitating the occurrence of seizures or the actual kindling process in animals [19].

Emotional and behavioral disorders in patients with childhood-onset epilepsy have a profound impact on health-related quality of life [20], but often they go untreated or even unrecognized [21]. In addition, studies evaluating the clinical and electroencephalographic (EEG) risk factors for psychiatric comorbidities in children with epilepsy are scarce, therefore hindering an early diagnosis and prevention.

In the present study, we aimed at: (i) assessing the emotional and behavioral problems in children with epilepsy using a specific questionnaire for psychopathology in children and adolescents, the Child Behavior Checklist (CBCL) [22,23]; and (ii) evaluating the association between emotional and behavioral disorders, as characterized by the CBCL, with specific clinical and EEG variables.

2. Methods

2.1. Participants

One hundred fifty-nine patients consecutively referred at the third-level epilepsy center of the IRCCS Stella Maris were evaluated. Patients' enrollment was based on the following criteria: diagnosis of epilepsy according to the clinical–EEG criteria of the ILAE (1989), age between 1.5 and 18 years. Individuals with severe ID (IQ < 35) were excluded, in order to increase the reliability of the psychopathological assessment. The study was approved by the Local Ethics Committee. All caregivers signed an informed consent form.

2.2. Measures

Parents completed the CBCL. The CBCL is a questionnaire that assesses emotional and behavioral problems in children and adolescents. This is a valid assessment tool in children with epilepsy [24], widely used in research and clinical practice. The CBCL generates a broad-band (i.e., internalizing problems, externalizing problems, total problems), and narrow-band emotional and behavioral scales (e.g., withdrawn, attention problems, Anxious/Depressed, Aggressive Behavior), with a standardized T-score (mean = 50, SD = 10). To identify the subjects at risk for psychopathology, we defined the cutoff points on the CBCL T-score scales. Following Achenbach's suggestions [22,23], we defined borderline groups (scores 65 to 70 in narrow-band scales and 60 to 63 in broad-band scales) and clinical groups (scores greater than 70 in narrow-band scales and greater than 63 in broad-band scales). Two different CBCL versions were used, according to the age of the child: the preschool version (CBCL 1 1/2–5) and the school-aged version (CBCL 6–18). The results from the two versions were analyzed separately (preschool-aged children group and school-aged children group).

2.3. EEG recording and evaluation

We evaluated the wake/sleep interictal scalp EEG of 158 patients (for 1 individual the EEG was not available). Electroencephalographic findings were analyzed for type, distribution, and lateralization of discharges. Two types of interictal EEG abnormalities were defined: (i) paroxysms (spikes, sharp waves, spike, and wave complexes) and (ii) focal slowing [25]; based on their distribution they could be focal, multifocal, or diffuse. Focal abnormalities (paroxysms or slowing) were also classified according to their site predominance in anterior (Fp2, F4, C4, Fp1, F3, C3, Fz, Cz), posterior (P4, O2, P3, O1, Pz, Oz), and temporal (F8, T4, T6, F7, T3, T5) brain regions, and as left-sided, right-sided, or bilateral. Electroencephalographic abnormalities were also classified according to their occurrence on awake or sleep states only, or in both conditions.

2.4. Clinical and EEG variables

The clinical features of the sample were obtained from medical records. The associations between emotional and behavioral symptoms and clinical and electroencephalographic variables were investigated. Categorical variables (presence of ID and level of cognitive development, etiology of epilepsy, type of anticonvulsant therapy, seizure control, and site of interictal EEG abnormalities) and continuous variables (age at epilepsy onset, duration of illness) were considered. According to the International Statistical Classification of Diseases and Related Health Problems (ICD-10) and the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV-TR), children with ID were defined as having both reduced intellectual functioning (full-scale IQ < 70) and impaired adaptive abilities to cope with daily demands of the social environment [26,27]. Mild ID was defined as IQ = 70–50 and moderate ID as IQ = 49–35. The illness was defined as “controlled” in children with a seizure-free period longer than 2 years.

2.5. Data analyses

Statistical analyses were performed using the IBM® SPSS® Statistics software version 20 (Armonk, New York, NY). Descriptive statistics were used to evaluate the features of the sample. We examined the distribution of all CBCL domains and found that the broad-band CBCL scales (Internalizing, Externalizing, and Total problems scales) were normally distributed. The narrow-band CBCL scales did not reach instead the criteria for normal distribution. To analyze the mean differences between the categorical variables on broad-band CBCL scores, we performed t-test, analysis of variance (ANOVA) and post hoc multiple comparisons (Bonferroni correction or Dunnet test in accordance with the Levine test on variance homogeneity). Nonparametric methods (Mann–Whitney U test and Kruskal–Wallis test) were instead performed on narrow-band CBCL scales. We also divided the sample in two subgroups, based on the presence of normal vs borderline/clinical scores for each CBCL scale, and used the Pearson’s chi-squared test to evaluate their association with the categorical clinical and EEG variables under study, and the t-test for continuous variables. Correspondence analyses were used to decompose significant chi-square and capture the associations between variables. Relationships were considered statistically significant for p-values < 0.05.

3. Results

3.1. Subject characteristics

Subjects (n = 159; boys n = 82, 52%; girls n = 77, 48%) were aged between 1.5 and 18 years. The preschool-aged group, who received the CBCL 1 1/2–5, consisted of 39 children (mean age: 4.1 yrs.; SD: 1.3); the school-aged group, investigated through the CBCL 6–18, was composed by 120 individuals (mean age: 11.6 yrs.; SD: 3.3). The characteristics of the two study groups are described in Table 1. Since the sample was recruited from a third-level epilepsy center, the proportion of patients with nonidiopathic forms (n = 114; 72%), and with neurologi- cal comorbidities such as ID (n = 59, 37%), was relatively high. In our sample, 33 children (50% of them suffering from idiopathic epilepsy) did not take anticonvulsant therapy. Among them, 14 were in remission from at least 1 year, 10 had rare seizures, mainly in the context of reflex or idiopathic epilepsies, 7 had a recent onset of epilepsy and were in a diagnostic phase, and 2 patients were noncompliant.
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