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Analysis of clinical characteristics and risk factors for mortality in human status epilepticus

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Purpose: To analyse clinical data including aetiology, age, antecedents, classification and mortality in human status epilepticus (SE), and to assess prognostic factors for mortality.
Methods: A prospective study was performed, including detailed analysis of clinical and laboratorial data of SE in individuals of any age, except neonates.
Results: One hundred and eleven SE were included, with patients' age ranging from 3 months to 98 years. SE incidence peaked in the first year of life, and 59.4% of the individuals had previous epilepsy while 40.6% had not. The main underlying causes were noncompliance to treatment in the first group, and CNS infection, stroke and metabolic disturbances in the second group. Overall mortality was 19.8%, and deaths were correlated to aetiology and patient's age. Refractory SE affected 11.7% of the cases. Clinical types included focal, secondarily generalised and generalised SE. Clinical and clinicoelectrographic classifications were convergent, but EEG was essential for the diagnosis in 4.5% of the cases.
Conclusions: Epileptic patients are at greater risk to develop SE, however, individuals with no prior history of epilepsy and acute neurological problems can also present SE. Aetiology varies with patient's age, and mortality is high and related to age and underlying causes. Clinical and clinicoelectrographic classifications are usually convergent, but in some cases the diagnosis of SE would not be established without the EEG.

Key words: status epilepticus; refractory status epilepticus; epilepsy; mortality; EEG.

INTRODUCTION

Status epilepticus (SE) is well recognised as a medical emergency that requires prompt treatment, especially its convulsive types. Despite recent improvements in its diagnosis and treatment[1–7], SE is still associated with significant mortality[8–10].

The diagnosis of SE is not difficult when motor signs are overt. However, this happens in only part of the patients and other clinical types still pose serious diagnostic challenges. This is the case, for example, for the condition known as subtle SE[11] or for the complex partial SE that is more prevalent than previously considered[12–14]. More recently it has been additionally demonstrated that after the control of convulsive SE, 14% of the patients still persist in nonconvulsive SE[15].

Besides these diagnostic difficulties other points remain unclear, including the relative incidence of distinct clinical presentations[16,17] and the prognostic factors for outcome[18–20]. Mortality related to the prolonged seizure *per se* is low and in the range of 1–2%, but varies depending upon the underlying causes and the duration of the follow up[21]. In prospective population studies this rate reaches 22%[22], with increasing incidence and mortality in the last 30 years due to the occurrence of myoclonic SE after cardiac arrest[23].

Some investigators have observed that longer seizure duration[24], continuous seizure activity[25] or specific EEG patterns[26,27] correlate with increased mortality, while others found that patient's age and aetiology were more related to outcome than specific ictal patterns[28]. This article specifically addresses the main clinical aspects of SE and their role in the outcome.

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PATIENTS AND METHODS

Patients

In this study we included all cases of partial and generalised SE admitted to the Emergency Unit (EU) of the Ribeirão Preto University Hospital, São Paulo, Brazil, from 1989 to 1993. Neonatal SE was not included and patients with incomplete and/or insufficient information in their charts were also excluded from the analysis.

Methods

The following data were obtained from each patient: age, sex, previous history of epilepsy, aetiology, clinical and EEG presentation, treatment and mortality. The aetiologies were defined based on clinical and ancillary examinations. Noncompliance to antiepileptic drug (AED) treatment was defined based exclusively on anamnesis data. The following definitions were used in this study:

Clinical SE

SE was defined as an epileptic seizure whose duration was equal to or greater than 30 minutes or as recurrent seizures without recovery of consciousness in between.

Electrographic SE

Presence of an ictal EEG pattern in at least 80% of the recording time which lasted 30 minutes or longer.

Clinical classification

Classification based on the International Classification of Epileptic Seizures (ILAE, 1981). SE was then divided into three groups: (a) focal—consisting of focal seizures; (b) focal with secondary generalisation—consisting of generalised seizures and evidences of preceding clinical focal seizures (clinical history and/or ancillary examinations); and (c) generalised—generalised seizures without any evidence of prior focal seizures.

Statistical analysis

For statistical comparison of the data we calculated the confidence intervals and considered the differences as nonsignificant when intersection between intervals occurred, and as significant when there was no intersection between them. The level of significance was established at $P < 0.05$. For comparison of ages between the two groups of epileptic patients and individuals with no prior history of epilepsy we used the nonparametric Kruskal–Wallis test. To assess the clinical and electrographic classifications we applied the kappa (K) statistic in order to verify the degree of concordance between the clinical and EEG diagnosis. We considered a K index of $0 < K < 0.4$ as low concordance, $0.41 < K < 0.75$ as high concordance and $K > 0.75$ as very high concordance.

RESULTS

Between 1989 and 1993, 111 SE (102 patients) were diagnosed at the EU, including 9 recurrent SE in 7 patients that had two to three episodes of SE, indicating that 6.8% of the patients had recurrent SE. All cases had at least one ictal or immediate postictal EEG.

Age and sex

The distribution of patients by age and sex is shown in Fig. 1. There were 53.9% males (55 patients) and 46.1% females (47 patients). Their ages ranged from 3 months to 98 years, with a mean of 34.1 years and a median of 33.0 years. SE predominated in adults, however, when compared per year, there was an incidence peak in the first year of life.

Aetiology of SE

The patients were subdivided into two groups: group A consisting of epileptic patients (66 patients, 59.4%) and group B of individuals with no previous history of epilepsy (45 patients, 40.5%). The list of aetiologies included AED noncompliance, AED changes, metabolic disturbances, CNS infections, stroke, fever, CNS structural lesions, systemic infections, cardiac arrest, eclampsy, alcoholic intoxication, miscellaneous and undetermined aetiologies. In the group A the main causes were AED noncompliance (21 patients, 31.8%) and undetermined aetiology (26 cases, 39.3%), $P < 0.05$. In group B three aetiologies predominated: CNS infections (12 cases, 26.6%); stroke (11 cases, 24.4%); and metabolic disturbances (8 cases, 17.7%), $P < 0.05$.

Age-specific aetiology

For this analysis we subdivided the patients into four age groups: (a) infants (1–12 months): 8 cases, 7.2%; (b) children and adolescents (1–19 years): 29 cases,

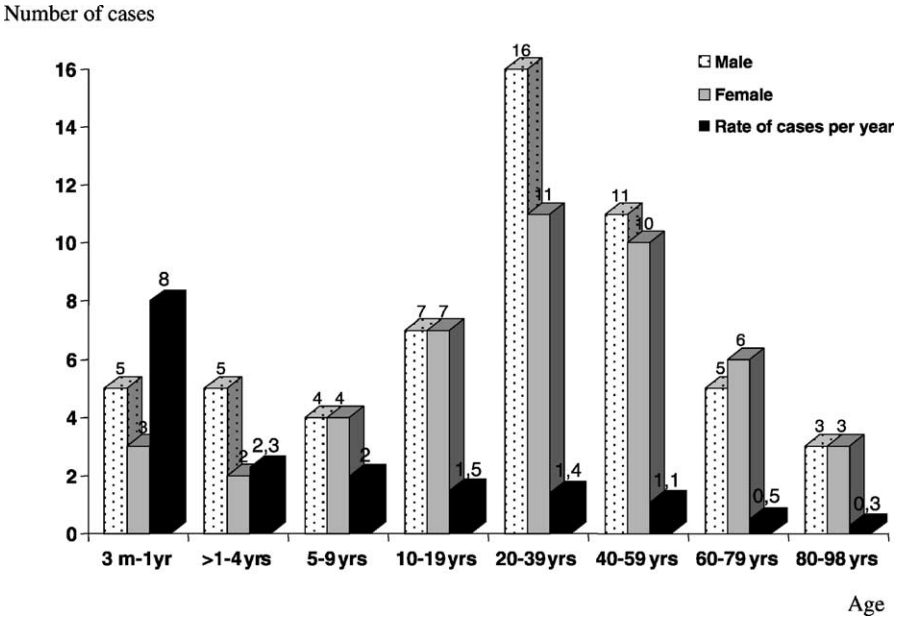

Fig. 1: Distribution of SE patients per age groups showing predominance of adult cases, but incidence peak in the first year of life.

Table 1: Distribution of aetiologies in different age groups.

Aetiology	Group A, previous history of epilepsy (%)	Group B, no previous history of epilepsy (%)	Total of cases (%)
Age group: 4 months to 1 year			
Metabolic	–	3 (37.5)	3 (37.5)
Hypoxia	–	1 (12.5)	1 (12.5)
CNS infection	–	3 (37.5)	3 (37.5)
Miscellaneous	–	1 (12.5)	1 (12.5)
Age group: 1–19 years			
	3 (10.3)	–	3 (10.3)
Undetermined	11 (37.9)*	1 (3.4)	12 (41.3)
AED noncompliance	1 (3.4)	–	1 (3.4)
Fever	5 (17.2)	1 (3.4)	6 (20.7)
Systemic infection	1 (3.4)	–	1 (3.4)
Cardiac arrest	2 (6.8)	–	2 (6.8)
CNS structural lesion	–	1 (3.4)	1 (3.4)
CNS infection	–	1 (3.4)	1 (3.4)
Eclampsy	–	1 (3.4)	1 (3.4)
Miscellaneous	–	1 (3.4)	1 (3.4)
Age group: 20–59 years			
AED noncompliance	18 (32.1)*	–	18 (32.1)
AED prescription changes	1 (1.8)	–	1 (1.8)
Metabolic	3 (5.3)	2 (3.5)	5 (8.9)
Stroke**	–	5 (8.9)	5 (8.9)
CNS infection**	–	6 (10.7)	6 (10.7)
CNS structural lesion	–	2 (3.5)	2 (3.5)
Systemic infection	1 (1.8)	–	1 (1.8)
Alcohol	–	1 (1.8)	1 (1.8)
Undetermined	13 (23.2)*	1 (1.8)	14 (25)
Miscellaneous	2 (3.5)	1 (1.8)	3 (5.3)
Age group: 60–98 years			
Stroke**	–	6 (33.3)	6 (33.3)
Metabolic	1 (5.5)	3 (16.4)	4 (22.2)
CNS infection	–	2 (11.1)	2 (11.1)
AED noncompliance	2 (11.1)	–	2 (11.1)
Undetermined	2 (11.1)	–	2 (11.1)
Miscellaneous	–	2 (11.1)	2 (11.1)

* $P < 0.05$ within the group. ** $P < 0.05$ between groups.

26.1%; (c) adults (20–59 years): 56 cases, 50.4%; and (d) elderly (60–98 years): 18 cases, 16.2% (Table 1).

In the first year of life all children belonged to group B. In the group of 29 children and adolescents, 23 (79.3%) were previously epileptic ($P < 0.05$) and 6 (20.6%) had no previous epilepsy. In this age range the main aetiologies in group A were undetermined aetiology ($P < 0.05$), followed by fever and AED changes. Among the group of 56 adult patients with SE, 38 (67.8%) had a previous diagnosis of epilepsy, and 18 (32.1%) had not ($P < 0.05$). The main aetiologies were irregular use of AED and undetermined cause in group A ($P < 0.05$), while in group B there was no predominant aetiology. In the group of older patients only 27.7% belonged to group A, and the remaining 72.2% to group B ($P < 0.05$), with stroke being the main aetiology in this last group of patients ($P < 0.05$).

SE clinical types

In this series, 48.6% of the cases had focal SE, 18.9% secondarily generalised SE and 19.8% generalised SE. It was impossible to classify based exclusively on clinical data in nine cases (8.1%), four of them with generalised motor signs and five nonconvulsive SE. The clinical history was not suggestive of SE in these last five cases (4.5%), and their diagnosis was only established after EEG recording. (Figs 2 and 3).

SE clinical types and EEG data

The EEG contributed to the clinical classification of nine cases previously considered as unclassified SE. Of these, four patients were initially thought to have generalised SE with bilateral motor manifestations, but their EEG showed ictal patterns regionalised or hemispheric, allowing their inclusion in the group of focal and secondarily generalised SE. Five patients had nonconvulsive SE and their EEG allowed to include three of them in the group of nonconvulsive generalised SE, and the remaining two in the nonconvulsive focal SE.

In order to compare the clinical and clinicoelectrographic classifications, we calculated the K index to assess the degree of concordance between the two classifications. There was a high degree of concordance between them, $0.41 < K < 0.75$, but the EEG was essential for the diagnosis in 4.5% of the cases.

Among the group of focal SE, the complex partial type predominated and was found in 52 (50%) cases ($P < 0.05$). The secondarily generalised SE was

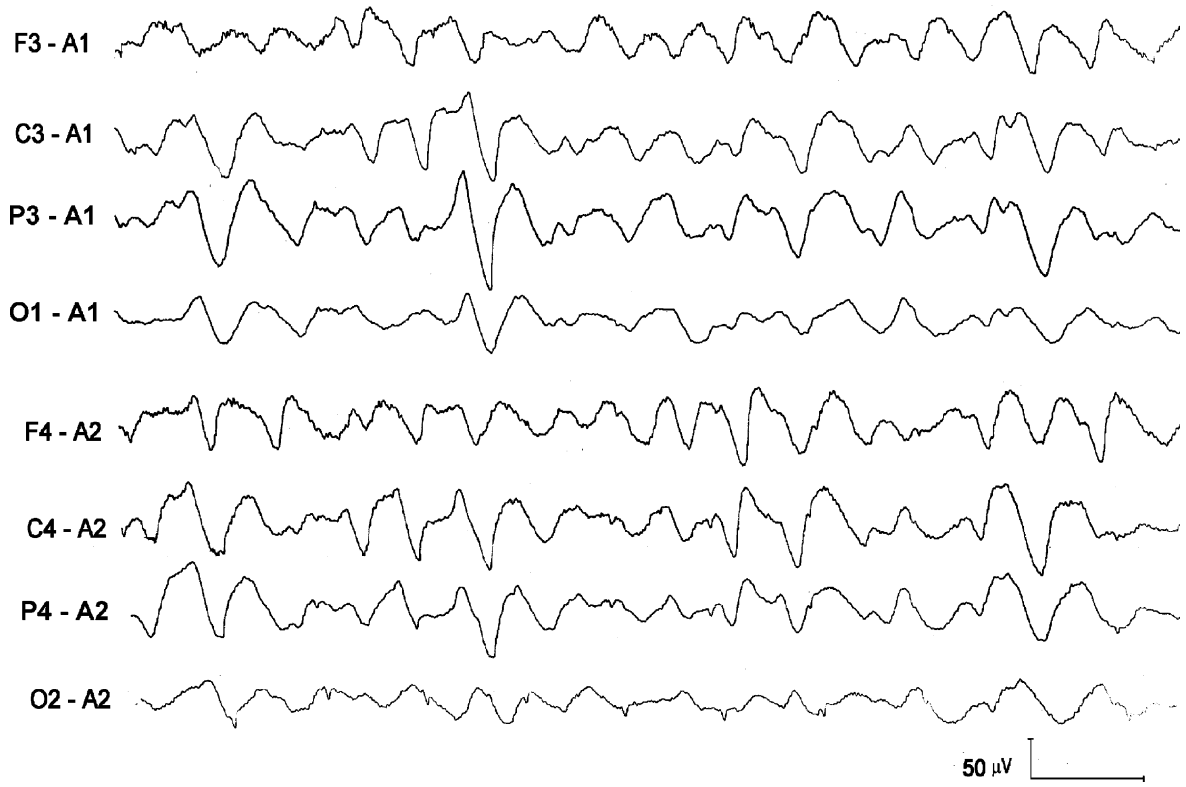

Fig. 2: Male, 52 years, 12 hours of nonconvulsive status only diagnosed after EEG showing intermixed rhythmic slow and sharp waves.

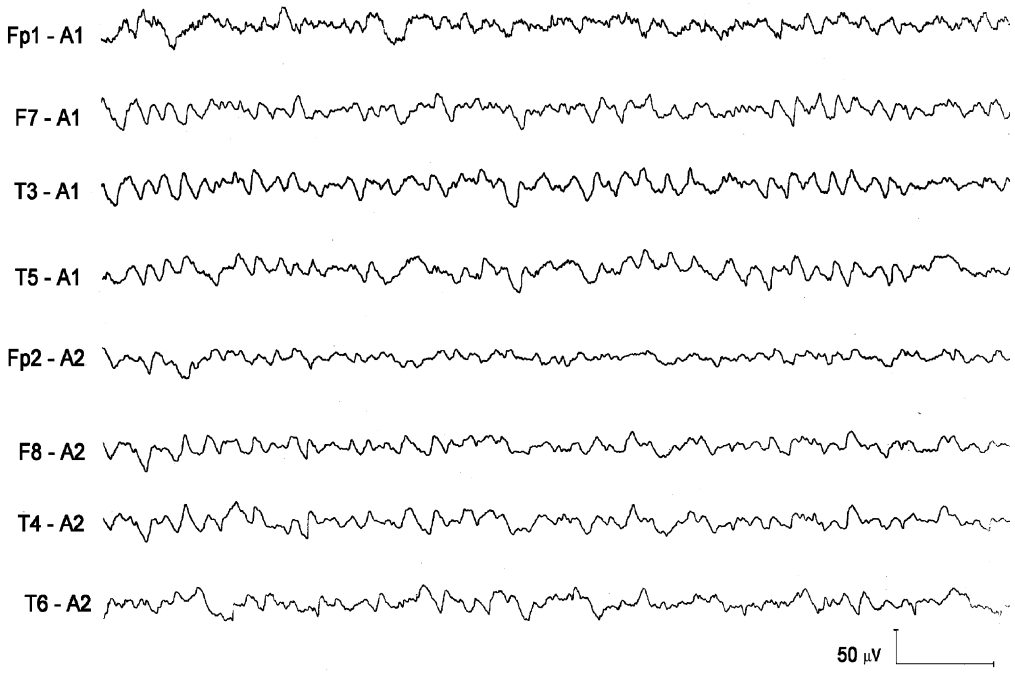

Fig. 3: Same patient, EEG 24 hours later, after diazepan and phenytoin injections. Patient already conscious, and EEG showing only residual background slow activity.

diagnosed in 29 cases (27.9%), all of them evolving from the complex partial type. Complex partial SE alone, with and without secondary generalisation reached a total of 77.9% of the cases.

Clinical features

The simple partial SE consisted of motor and visual phenomenology. The motor signs started in the face of one of the patients and further evolved to the ipsilateral arm, while in the other two patients, only the upper extremities were affected. The visual symptomatology was described as colourful balls, scintillate scotoma, scene hallucination and macropsia.

The complex partial SE group stood out not only for the high number of cases but also for its distinct clinical features. This group was additionally subdivided into two subgroups: (a) subgroup I consisting of 36 patients whose impairment of consciousness was exclusively attributed to the SE itself and (b) subgroup II consisting of 45 cases in which, in addition to the SE, the underlying aetiologies could also be playing a role in the determination of the impairment of consciousness. In these patients this overlap of factors further aggravated or even delayed the diagnosis of SE.

Out of the total of 81 cases of complex partial SE, 71 presented in addition to the impairment of consciousness, masticator automatisms and motor manifestations including eyelid myoclonia, eye and/or head deviation, and face and/or limb tonic or clonic contraction. These signs were observed in 30 patients of subgroup I and in 41 of subgroup II. The other 10 patients presented exclusively impairment of consciousness: 6 of subgroup I and 4 of subgroup II.

The group of generalised SE included absence seizures with unresponsiveness and eyelid myoclonia, multifocal or generalised myoclonic seizures, axial tonic seizures with eyes opening and turning up, hyperextension of the neck and eventually arms, and cases with generalised tonic–clonic seizures.

Age, aetiology and mortality

There were 22 deaths. The total mortality in relation to the number of episodes was 19.8% and in relation to the number of patients, 21.2%. Considering the different age groups, the number of deaths in relation to the number of patients in each group is shown in Fig. 4. The percentage of deaths among the elderly was greater than in any other age groups. The most frequent aetiologies leading to deaths were stroke, infection of CNS and miscellaneous factors (Fig. 5), all affecting the group with no previous history of epilepsy.

Refractory SE

In 13 cases (11.7%), the SE did not stop with first and second choice drugs (benzodiazepines, phenytoin and

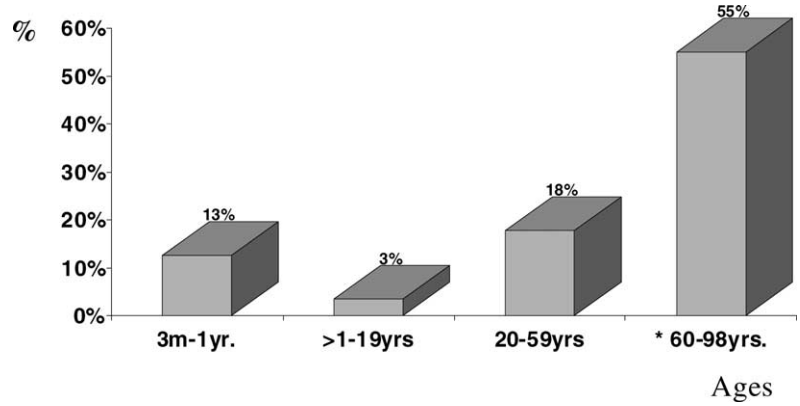

Fig. 4: Percentage of deaths per age groups ($^{*}P < 0.05$).

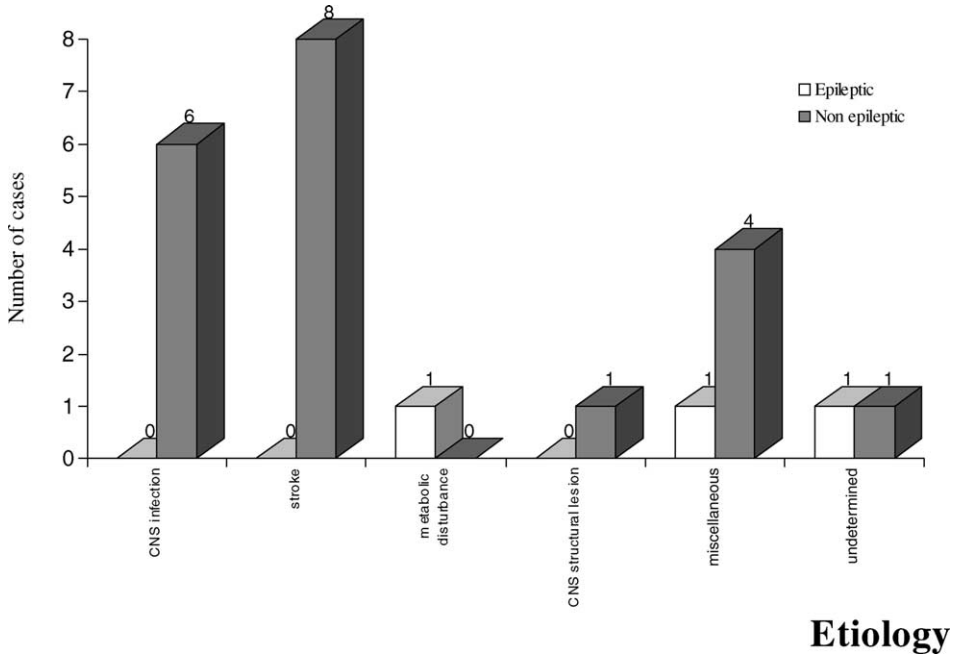

Fig. 5: Cause of deaths in relation to patient groups.

phenobarbital), thus, characterising the refractory SE. When we correlated aetiologies to the need of barbiturate coma induction, we found six cases of CNS infection, two of stroke, two of cardiac arrest, one of eclampsy, one of metabolic disturbance and one of undetermined cause. This correlation was more frequent among those patients with CNS infections.

DISCUSSION

The incidence of SE varies from 4 to 16% depending upon the population studied[21]. People with epilepsy represent an important risk group, but many SE also occur among individuals with no previous history of epilepsy, during systemic diseases and acute neurological problems. In more extended studies including epileptic as well as nonepileptic persons the incidence rates varied from 0.02 to 1.3%[29,30], but more recent population data demonstrated that SE is not as rare as previously supposed, with an overall incidence rate of 41 cases/100 000 inhabitants/year[8,22], and of 17.1/100 000/year when only adults are considered. This incidence was higher—54.5/100 000—in those aged 60 years and older[31].

This was an ample study that included different age groups and diverse clinical types of SE, excluding only the neonatal SE. There was no significant difference between sexes. Regarding age distribution, we observed only one peak of incidence in the first year of life, contradicting the previously proposed bimodal distribution with two peaks of incidence, one in the

first year of life and the other in patients over 60 years[8, 22].

Regarding the aetiology most patients had previous diagnosis of epilepsy (59.4%) and irregular use of AED represented the main cause of SE in the group of patients with previous epilepsy, while CNS infection, stroke and metabolic disturbances were the main aetiologies among the patients with no previous history of epilepsy. Overall, our data on aetiology and age groups are concordant with other series in the literature[32, 33]. In children CNS infection and metabolic disturbances represented the main causes of SE[34], while in individuals over 60 years cerebrovascular disease, head trauma, metabolic disturbances, brain tumours, CNS infection, undetermined aetiology and miscellaneous factors were the most common causes[35].

One aetiology that was probably underestimated in our study was compliance to treatment due to the fact that systematic checking of AED levels was not always possible in the EU. Some cases of noncompliance could then go undetected and be included in the group of undetermined cause, and this fact could probably explain the large number of cases of adult SE of undetermined aetiology.

The overall mortality rate was 19.8%, and the highest mortality was observed among the elderly, with death affecting 55.5% of the aged patients. Besides age itself, other factors probably contributed to mortality, including aetiologies that frequently aggravated the clinical condition of the patients. This possibility is somehow reinforced by the observation that SE mortality among epileptic patients in this age group was much lower when compared to aetiologies, such as stroke and CNS infection that were mostly prevalent in those patients over 60 years.

In the literature, the mortality rate is also variable depending on the aetiology of SE, with figures as high as 58%[36] and as low as 1–2% when considering deaths strictly related to the prolonged seizure[21]. In series including all age groups and all aetiologies the overall mortality reaches 22%, being lower in children (2.5%), moderate in adults (14%) and higher in the elderly (38%), as recently demonstrated[22].

Refractory SE was observed in 13 cases (11.7%) and 46.1% of them were related to CNS infection, again suggesting that aetiology is an important determinant of morbidity and mortality, independent of patient age.

In contrast to previous findings that considered generalised convulsive SE as the most prevalent type in adults[37], in our study we observed a predominance of focal SE. This is in good agreement with other series that reported generalised SE in patients with previous history of focal epilepsy and aetiologies compatible with focal SE, suggesting in reality secondarily generalised SE[38–41].

We should emphasise that since the 1970s some studies have already reported the predominance of focal SE in adults[36, 42], and this observation has also been confirmed in more recent studies[8, 22]. Most likely conceptual and terminology problems are involved, with authors possibly lumping together generalised and secondarily generalised SE in a single category.

Among the group of focal SE, the complex partial type predominated. We should stress, however, that distinction between simple and complex partial seizures is easier than simple and complex partial SE, since impairment of consciousness which is a fundamental criterion to differentiate simple from complex partial seizure is intrinsically part of the definition of SE. Moreover, many other factors contribute to impairment of consciousness, including different aetiologies and sedative medications frequently used to stop seizures. In our group of patients with complex partial SE, the impairment of consciousness could be exclusively due to the SE itself in only 36 patients. Most of these cases were related to previous epilepsy and noncompliance to AED treatment. In the remaining 45 patients aetiology could also be playing a role in causing impairment of consciousness, including metabolic disturbances, stroke and miscellaneous factors. This group of patients obviously requires greater attention since diagnosis of SE can be frequently delayed and only suspected after the appearance of convulsive motor signs.

Among the 81 cases of complex partial SE, 71 (29 secondarily generalised) presented discrete motor phenomena consisting of automatisms, eyelid or facial myoclonia, head or eye deviation or even limb hypertonia. Only 10 cases had isolated disturbance of consciousness. These cases have been called subtle convulsive generalised SE[11], a terminology proposed to classify cases with unusual rhythmic movements, sometimes continuously involving the eyelids, face, mouth, nistagmoid movements of the eyes, trunk or members jerks occurring in patients with stupor or deep coma. The EEG in these cases showed bilateral ictal discharges which led to their inclusion in the group of generalised SE. The same group later observed that in general there was an asymmetry in the EEG recordings which led them to reconsider these cases as secondarily generalised SE[43].

Subtle SE is generally considered as a late evolution of SE, or a situation that occurs associated with severe encephalopathies. In this study, we did not consider the diagnosis of subtle SE since we did not have late evolution of SE and it was not always found within the context of a serious encephalopathy. We, therefore, preferred the term complex partial SE to designate these cases.

In many instances seizure semiology alone is insufficient to define the case. A previous history of epilepsy,

EEG recording as well as imaging data are frequently necessary for syndromic and aetiologic classification. For example, a previously health patient with a focal lesion, continuous stupor and generalised EEG discharges, most likely can be better classified as having a nonconvulsive focal than generalised SE, considering the presence of the lesion and the low likelihood that an adult patient who never had generalised epilepsy would present a nonconvulsive generalised SE.

Complex partial SE is probably more frequent than previously thought. It is frequently misdiagnosed particularly when expressing isolated impairment of consciousness and when associated with aetiologies that could, by themselves, cause patient unresponsiveness (CNS infection, stroke, metabolic disturbances). These cases have probably been classified within other groups, causing underestimation of the real figure of this type of SE, as previously suggested[44–46].

Instead of three categories, i.e. generalised, focal and unilateral SE[47], the most recent proposal for classification indicates that SE should be classified into only two categories, generalised and focal SE[48]. The first group should be further subclassified into generalised tonic–clonic, clonic, absence, tonic and myoclonic SE, while the focal SE would encompass *epilepsia partialis continua* of Kojevnikov, *aura continua*, limbic SE (psychomotor status) and finally, hemiconvulsive status with hemiparesis. This new proposal appears to be more adequate, however, we should still consider the fact that not all focal nonconvulsive SE can be included in the classical limbic SE.

The diagnosis of generalised SE is usually not difficult, except for patients with epileptic encephalopathies and severe mental retardation whose impairment of consciousness related to cluster of atypical absences or absence status is frequently difficult to assess. A detailed clinical history carefully comparing baseline behaviour, presence of eyelid myoclonia and increase of EEG discharges is essential for the differentiation between interictal and ictal states.

In general, there was good agreement between clinical and clinicoelectrographic classifications. However, the inclusion of EEG additionally defined nine cases of unclassifiable SE, and was essential for the diagnosis of five cases (4.5%), similar to another study where EEG was fundamental for the diagnosis of SE in up to 8% of patients in coma[17].

EEG was also of fundamental importance to follow the evolution of all cases. Since the recovery of consciousness is improbable immediately after the initiation of treatment, and many other factors can contribute to stupor, confusion, somnolence and coma, continuous monitoring would predictably be useful to assess continuation or resolution of SE in a more sensitive way than on clinical grounds alone. This would certainly benefit patients avoiding unnecessary prolongation of aggressive treatment with sedative drugs in some cases, and more importantly, avoiding delayed diagnosis of nonconvulsive SE.

In conclusion, SE occurred in epileptic as well as in nonepileptic individuals. The main aetiologies were AED noncompliance in the first group, and CNS infections, stroke and metabolic disturbances in the group of individuals with no previous history of epilepsy which mainly included very young (first year of life) and elderly (above 60 years) patients. It predominated in adults, but the incidence peaked in the first year of life. Complex partial SE, with or without secondary generalisation, predominated. Mortality was related to underlying aetiology and age, and was higher in the elderly. Finally, the EEG was instrumental for the classification and evolution of SE, and most importantly, essential for the diagnosis under certain circumstances.

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