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Profile of vascular epilepsy in the Neurology Department of Conakry University Hospital

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Abstract

Introduction:*Vascular epilepsy (VE) is a frequent complication of stroke. The clinical presentation of VE is polymorphous, its treatment is not codified and its evolution is variable.*

Our aim was to report the epidemiological, electro-clinical, therapeutic and evolutionary characteristics in the Neurology Department of Conakry University Hospital.

Material and methods: *This was a prospective descriptive study, lasting one (1) year, from 01/01/2022 au 31/12/2022 carried out in the Neurology Department of Conakry University Hospital. Patients admitted for at least one unprovoked epileptic seizure after a cerebrovascular accident (CVA) within a period of more than one week were included in our study. All patients with history of epilepsy or head trauma were excluded.*

Results: *We recorded an incidence of 6.3%. The mean age was 58.2 ± 10.4 years, with a sex ratio of 1.31. The mean time to onset of VE was 33.42 ± 18.2 weeks, the inaugural mode of VE was serial seizures in 64.8%, and focal clonic seizures presented 40.5% of clinical manifestations. EEG abnormalities were lateralized (54.8%), focal (32.2%) and generalized (12.9%). These EEG abnormalities were mainly slow waves and spikes (70.2% and 29.7% respectively). The long-term antiepileptics used in this study were sodium valproate (54%), followed by lamictal (37.8%) and lamotrigine (8.1%). CE regression was observed in 78.3% of patients, although 21.6% progressed to MEE.*

Conclusion: *EV is frequent in men and young subjects in our context, and presents a good evolution.*

Keywords*: vascular epilepsy, epidemiology, Conakry*

Introduction

Vascular epilepsy (VE) is defined as the occurrence of at least one unprovoked seizure at least one week after a stroke [1]. The incidence of VE varies from 2% to 20%, depending on the type and study population [2]. Stroke and epilepsy are two (2) of the most common severe neurological disorders worldwide, and their association is increasingly documented [3]. After a stroke, patients with early-onset seizures have a 33% risk of recurrence of unprovoked epileptic seizures within 10 years. This risk is 72% within 10 years for late-onset seizures, which is why a single late-onset unprovoked seizure is sufficient for the diagnosis of VE [4]. Stroke is responsible for around 10% of all epilepsies and 55% of newly diagnosed seizures in the elderly [5]. The time to onset of VE is variable, with nearly 93% of VEs occurring within 2 years of a stroke, with a peak in 6 to 12 months [6]. The clinical presentation of VE is polymorphous, with a predominance of focal or generalized motor manifestations [7]. The electroencephalogram (EEG) is an important tool for assessing the risk of epileptic seizures following a stroke, and can also be used to establish a correlation between the epileptic seizure and the stroke. However, its normality does not exclude epilepsy [8]. Risk factors for VTE include young age, early-onset seizure, cortical topography and extent of ischemic or hemorrhagic stroke, and stroke severity. However, they do not represent an indication for antiepileptic treatment [9]. Today, there is no universal protocol for the management of VE, but the use of new-generation antiepileptics seems to have more advantages over old-

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generation antiepileptics in clinical practice [10]. In this study, we report epidemiological, clinical, paraclinical and therapeutic data on VE in the Neurology Department of Conakry University Hospital.

Material and methods

Our study was prospective and descriptive, carried out in the Neurology Department of Conakry University Hospital, over a period of one (1) year from November 1, 2022 to October 31, 2023. Patients admitted for vascular epilepsy as defined by the International League Against Epilepsy (ILAE) [4] were included in our study. That is, at least one unprovoked epileptic seizure after a cerebrovascular accident (CVA) within a period of more than one week. Patients with a history of epilepsy or head trauma were excluded. We collected the following data: age, divided into two age groups (< 65 years, 65 years), sex, time to onset of VE expressed in weeks, type of epileptic seizure according to the ILAE classification, inaugural mode of VE: isolated epileptic seizure, serial epileptic seizures or status epilepticus. Type of stroke, stroke severity distribution using the National Institute of Health Stroke Scale (NIHSS), number of stroke lesions on imaging. The type of antiepileptic drug administered and the evolution of the VE. We used SPSS version 7.2 to analyze our data. We determined the frequency of qualitative variables and the mean with standard deviation of quantitative variables.

Results (table 1)

During our study, out of 581 patients admitted for stroke, 37 patients presented with EV, with an incidence of 6.3%. The mean age of our patients was 58.2±10.4 years, with extremes of 48 and 73 years. The male sex was the most represented at 56.7%, with a sex ratio of 1.31. Early-onset attacks were observed in 21.6% of cases, and the mean time to onset was 33.42±18.2 weeks, with extremes of 7 and 64 weeks. The inaugural mode of VE was an isolated seizure in 32.4%, serial seizures in 64.8% and status epilepticus (SE) in 10.8%. The clinical presentation was mainly focal clonic seizures observed in 40.5%, followed by focal myoclonus and generalized tonic-clonic seizures with 24.3% and 18.9% of cases respectively. EEG abnormalities were lateralized (54.8%), focal (32.2%) and generalized (12.9%). These abnormalities were mainly slow waves and spikes (70.2% and 29.7% respectively); in 16.2% of cases, the EEG was normal.

Benzodiazepines were the first line of treatment, with clobazam used in 45.9% of cases and clonazepam in 21.6%. Long-term antiepileptics were dominated by sodium valproate (54%), followed by lamictal (37.8%) and lamotrigine (8.1%).We observed a regression of epileptic seizures in 78.3%, however 21.6% of patients progressed to status epilepticus.

Stroke was ischemic in 86.4% of cases and hemorrhagic in 13.5%. The distribution of stroke according to severity showed a predominance of moderate stroke (40.5%) and minor stroke (24.3%), with a mean NIHSS of 14.1±6.3 and extremes of 2 and 23. Brain imaging of our patients showed one brain lesion in 83.7%, two lesions in 10.8% and three lesions in 5.4%.

Discussion

Our incidence of VE is similar to those of Seshadri et al. and Nyassind et al., who found 6% and 5.2% respectively. But lower than the observations of Adoukonou et al, and Freiman et al, who report 9.3% and 15% VE respectively [11][12][13][14]. This variation in incidence could be explained by differences in diagnostic criteria, study population or study duration. As we have seen, a male predominance is generally reported [30][15].

In this study, the mean age of patients was 58.2±10.4 years, with a predominance of patients under 65 (67.5%). Several other studies have reported a high frequency of VE in young subjects, and many studies conclude that young age represents a risk factor for VE [15][16][17]. Age over 65 is a protective factor against seizure recurrence, due to a smaller cerebral cortex and reduced excitability induced by degenerative changes. This protection is cancelled out by the onset of stroke, which exposes this population to a high risk of epilepsy. Stroke is responsible for at least a third of epilepsies after the age of 60, making it the most frequent cause, ahead of degenerative pathologies, tumours and head trauma[18]. Regarding clinical semiology, epileptic seizures were exclusively motor and mainly focal during this study. This is consistent with the findings of our Senegalese colleagues (55.9% focal seizures) and those of Conrad et al. (57.1% focal seizures with bilateralization) [19][11][21]. The focal nature of clinical manifestations is more evidence of the symptomatic nature of epileptic seizures. However, it is important to point out that generalized seizures can also occur, as was the case in 5.4% of our patients. The time to onset of EV is variable, ranging from a few weeks to several years, with peak frequency between 6 and 11 months [25] [7] [38] . The average delay in our study was 33, 42 ±18.2 weeks.

This delay may be influenced by other factors such as cortical location or severity. VE risk factors can be used to identify high-risk individuals, but are not an indication for prophylactic treatment [35][37]. The main criterion for stroke severity is NIHSS, which is proportional to stroke severity. The NIHSS is considered an independent factor of VE, with a threshold of 4 according to Abrairal et al, and a threshold of 16 according to Hseih et al [32] [20][33]. We found a majority of minor strokes (40.5%) and a mean NIHSS of 14.1±6.3.

The intercritical EEG enabled us to assess the risk of subsequent seizures, and the link between epileptic manifestations and brain lesions. We observed abnormalities in almost all patients, mainly slow abnormalities (70.2%) and spikes (29.7%). These slow anomalies represent the sequelae of cerebral lesions secondary to stroke. A systematic review of the literature revealed critical and intercritical epileptiform activity on the EEG after stroke in 7% and 8% of patients respectively. These values may be even higher on long-term EEG [26]. Althou These values may be even higher in long-term EEGs [26]. Although EEG abnormalities, especially epileptiform ones, are predictive of seizure recurrence, a normal EEG does not rule out epileptogenicity [8]. Intercritical EEG was normal in 16.2% of our patients.

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We recorded 78.3% good evolution of epileptic seizures and 21.6% SE.

Nyassind et al recorded 17.6% SE, and proved that a history of two (2) strokes was predictive of VE ($p < 0.0000001$, $p = 0.007$) [34].

The majority of our patients (83.7%) were experiencing their first vascular event, and 16.2% had had two (2) or more strokes. Ischemic stroke was the most common type of stroke observed in our study, a finding similar to that of other studies [24]. This may be explained by the high frequency of ischemic stroke. During ischemic stroke, reperfusion reduces neuronal damage, which also appears to reduce the occurrence of VE [27]. Adequate statin therapy after stroke may reduce the risk of VE [28][36].

In terms of treatment, all our patients received an antiepileptic drug as monotherapy or in combination with a benzodiazepine. Sodium valproate (SV) was the most widely used molecule, accounting for 54% of all patients, a choice based on low cost, availability and efficacy. In terms of efficacy, a 12-month follow-up showed a seizure-free rate of 64.5% with VS, and 58.7% with levetiracetam (with levetiracetam (LEV) [23]. However, the newer antiepileptics, notably LEV and lamotrigine (LTG), are more widely used in Western countries, and appear to have fewer side-effects, as well as advantages in terms of tolerability and effects on cognitive function [22][40]. We administered LTG in 37.8% and LEV in 8.1%. There was no significant superiority between LEV and LTG [39]. Although there is no established treatment protocol for VE, Leung et al recommend the use of antiepileptic drugs for up to 4 years if the underlying etiology of the stroke cannot be completely treated [29]. VE are generally pharmacosensitive and pharmacodependent, although drug resistance is observed in around 20% of VE [31].

We did not observe any VE-related deaths during our study, however the results of a meta-analysis on VE suggest that it may be associated with a higher risk of all-cause mortality during long-term follow-up [41].

Conclusion

VE is a frequent complication of stroke, with a male predominance. This study reveals a predominance of VE in young subjects, with serial seizures the most frequent mode of onset and predominantly motor manifestations. In the majority of patients, a single stroke is sufficient to cause an VE, and ischemic stroke is the most common cause. Sodium valproate continues to play an important role in the treatment of VE in our context, due to its low cost, accessibility and efficacy. The prognosis of VE in our study was generally favourable.

A long-term study could determine the long-term prognosis of VE.

Table 1: Patient characteristics

Variables	**Effectifs**	**Proportion (%)**
Age		
< 65 years	24	67,5%
≥ 65 years	18	32,4%
Mean age (extrêmes) (48 years – 73 years)	58, 2 ± 10, 4 years	
Sex		
Male	21	56,7%
Female	16	43,2%
Sex ratio (M/F)	1,31	
History of early seizures		
Yes	8	21,6%
No	29	78,3%
Inaugural modes		
Isolated seizure	12	32,4%
Serial seizures	21	64,8%
EME	4	10,8%
Types of epileptic seizure		
CCTG	7	18,9%
Focal myoclonus	9	24,3%
Focal tonic seizures	4	10,8%
Focal clonic seizures	15	40,5%
Generalized clonic seizures	2	5,4%
Types of EEG abnormalities		
Slow waves	26	70,2 %
LPD plus	5	13,5%
Spikes	11	29,7%
Polypoints	8	21,6%
Normal	6	16,2%
Topographies of EEG anomalies		
Focal	10	32,2%
Lateralized	17	54,8%
Generalized	4	12,9%
Treatment		
Clonazepam	8	21,6%
Clobazam Clobazam	17	45,9%
Sodium valproate	20	54%
Lamotrigine	14	37,8%

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Levetircetam	3	8,1%
Evolution		
Regression of Epileptic seizure	29	78,3%
EME	8	21,6%
According to stroke severity		
Minor stroke 0-4	9	24,3%
Moderate stroke 5-15	15	40,5%
Severe stroke 16-20	8	21,6%
Severe stroke **≥ 20**	5	13,5%
Mean NIHSS (extremes) 14,1 ± 6,3 (2 – 23)		

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