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Research Article

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Vascular Epilepsy

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Abstract

Seizures can be a presenting feature of acute stroke. They may complicate its early clinical course or be a late complication or sequel. The incidence of combined "early" and "late" seizures after stroke has been reported to be approximately 10% and the prevalence is of 2-4%. Mechanisms leading to post stroke seizures and chronic epilepsy, in both ischemic and hemorrhagic strokes, are diverse. Focal slowing or diffuse slowing activities, rhythmic slow waves are associated with a low risk of early onset seizures whereas focal spikes, sharp waves, periodic lateralized, or periodic bilateral discharges are associated with a higher risk.

There are no randomised controlled trials performed in stroke patients evaluating management strategies for poststroke seizures and epilepsy. Treatment options should be extrapolated from those of epilepsy in general, taking into consideration hepatic and renal function, tolerability, costs, interactions between antiepileptic drugs (AED), interactions with antithrombotic and other treatments. However, randomised controlled trials of AED prophylaxis for acute and remote seizures are essential to improve the evidence level of current guidelines and recommendations for the management of post-stroke seizures

Keywords: Seizure, Epilepsy, vascular, EEG, MRI

Introduction

Seizures can be a presenting feature of acute stroke. They may complicate its early clinical course or be a late complication or sequel. Stroke and other vascular lesions of the brain, including subcortical vascular encephalopathy, are the main cause of epilepsy in old age [1-3].

We define post stroke seizure as ''single or multiple convulsive episode/s (fit/s) after stroke and thought to be related to reversible or irreversible cerebral damage due to stroke regardless of time of onset following the stroke'' and post-stroke epilepsy (PSE) as ''recurrent seizures following stroke with confirmed diagnosis of epilepsy'' [1, 2].

Incidence and Prevalence of Post Stroke Seizures

The frequency of seizures and epilepsy in stroke. Post stroke seizures and post stroke epilepsy constitute important complications in patients surviving a stroke, and stroke has been long recognized to be a common cause of epileptic seizures. Indeed stroke represent the most common cause of new-onset seizures and epilepsy in adults aged ≥60 accounting for 25-50% of new onset symptomatic epilepsy. The incidence of combined "early" and "late" seizures after stroke has been reported to be approximately 10% and the prevalence is of 2-4%. The 5-years risk of post stroke seizures ranges from 7% to 11%. In addition, stroke is one of the most frequent aetiologies of status epilepticus (SE), although this condition occurs in only 1.5% of strokes [1-3].

The relationship between early and late seizures

The occurrence of early seizures increases the risk of subsequent epilepsy, but epilepsy develops in a minority of cases only (about one third). In addition, if a late seizure occurs, the risk of subsequent epilepsy is much higher [1, 4-5].

Table 1: Classification of vascular seizures and epilepsy.

Type of seizure	Clinical characteristics
Acute seizure	• Epileptic seizure occurring within the first 24 h after onset of stroke; 40% of the seizures after ischaemic stroke and 57% of the seizures after haemorrhagic stroke, occurred within 24 hours of onset. • Other causes and precipitants of seizures, either in the brain or systemically (e.g. hypoglycaemia, hypoxia, hypocalcaemia, meningitis and the proconvulsant effect of treatments) should be excluded. • Patients with a first acute provoked post-stroke seizure have a risk of a subsequent recurrent seizure over the following 10 years of around 30%, whereas patients with a first unprovoked seizure have a risk of 70%. • They can be either partial or generalised. Partial seizures are more frequent, and are usually motor seizures, sometimes with secondary generalisation
Early post stroke seizure	• One or more seizures within 1 week or 2 weeks (1-15 days) after the stroke and most are single seizures. • Approximately a third of post stroke seizures occur in the early phase • Occurred in 4.8% of ischaemic stroke patients and in 7.9% of haemorrhagic stroke patients • Usually present with a focal onset, arising from areas in close proximity to the area of infarcted tissue • After the first early post-stroke seizure, the risk of recurrence is estimated to be 10–20%
Late post stroke seizure	• One unprovoked epileptic seizure at least 2 weeks after the stroke and have a peak within 6 to 12 months after the stroke and have a higher recurrence rate of up to 90% in both ischaemic and haemorrhagic stroke. • Occurred in 3.8% of ischaemic and 2.6% of haemorrhagic stroke patients. • Are often generalised tonic-clonic seizures. • Late seizures may be associated with a higher likelihood of epilepsy
Post stroke epilepsy/recurrent seizures	• Occurred in 2.5% of the patients. • Initially defined as two or more unprovoked epileptic seizures occurring at least 2 weeks after the stroke (ILAE). Actually, according to the latest revised classification of the International League Against Epilepsy (ILAE), a late-onset seizure in the context of stroke defines vascular epilepsy, making it no longer necessary to wait for a second seizure. Thus, it is no longer necessary to wait for seizure relapse before starting antiepileptic drug treatment.
Status Epilepticus	• Occurs in only 0.14–1.1% of strokes • May the first epileptic symptom of stroke in 0.12% of cases or it can follow one or more single seizures • Stroke is the most common aetiology for status epilepticus in the elderly (22% of new-onset status epilepticus are related to stroke) and occurs more often among patients with disabling strokes: the type of stroke, topographic findings, size of the lesion, or electroencephalographic (EEG) pattern do not predict the progression to status epilepticus • Early-onset status is more often nonconvulsive than convulsive.

Pathogenesis

Conventionally, epilepsy is regarded as arising from cortical regions. However, subcortical changes as seen in cerebrovascular small vessel disease, may contribute to epileptogenesis.

Mechanisms leading to post stroke seizures and chronic epilepsy, in both ischemic and hemorrhagic strokes, are diverse:

The mechanism of early seizures associated with stroke include the changes in the penumbra zone, brain edema, the effects of ischaemia (global hypoperfusion can also cause seizure activity, particularly in sensitive areas such as the hippocampus, a well known epileptogenic area.), metabolic changes (breakdown of membrane phospholipids, and release of free fatty acids), alterations in neurotransmission (ischaemia released excitatory amino acids, mainly glutamate, producing a cascade of toxic events; down-regulation in the efficacy of GABAergic intracortical inhibition with subsequent NMDA-receptor-mediated excitation), and finally, the alter-

ations in intracellular ions (reduction of the blood flow leads to subsequent cytotoxic effects with an accumulation of intracellular calcium and sodium, resulting in depolarisation of the cellular membrane and lowering of seizure threshold) [1, 3, 5-6].

The mechanisms of for late-onset seizures and post stroke epilepsy are associated with later changes in the structure and function of cerebral tissue, the formation of abnormal scar with reorganisation of axonal connections, loss of GABA-mediated inhibitory pathways and changes in the properties or composition of the glutamate receptor subunits. The presence of haemosiderin and iron in cortical neurons (iron inhibits glutamate reuptake into astrocytes), free radical formation and membrane peroxidation, selective neuronal cell death and apoptosis, changes in membrane properties, mitochondrial changes, receptor changes (e.g. loss of GABAergic receptors), deafferentation, and collateral sprouting (both at the site of ischemia as well as in remote areas) [3,4,7].

Table 2: Risk Factors of post stroke seizures and epilepsy.

Risk factors patient related (demographic variables, vascular risk factors)	Risk factors stroke related, both clinical (symptoms/signs, severity and dependency) and radiological (stroke type, location and size)	Risk factors related to comorbid conditions	Risk factors related to seizures
-Demographic variables: younger patients, men	- Clinic: *Loss of consciousness, confusional syndrome and persistent paresis	Myocardial infarction, peripheral, vascular disease, large-artery atherosclerosis, cerebrovascular disease and left ventricular hypertrophy	- Clinic: Early seizure (≤7 days)
-Risk factors for vascular disease: hypertension, smoking, diabetes, raised serum total cholesterol, heavy alcohol abuse and lack of exercise.	*Severe and disabling strokes (NIHSS score >4 at the stroke presentation) independently of the size of the ischaemic lesion on CT scanning, perhaps because the ischaemic penumbra, not visualized on CT, is epileptogenic. *Neurological and medical complications *Stroke recurrence * The severity of persistent disability after the stroke *Seizures were more often associated with cardioembolic than with large-vessel atheroma or small-vessel disease.	APOE e4 allele, dementia	Clinic: Early seizure (≤7 days)
-All these factors, have been associated with the development of seizures or epilepsy, even in the absence of overt stroke.	-Imaging: *Lobar haemorrhage, haematoma size, hemorrhagic transformation of ischaemic lesions, total anterior circulation infarcts, infarcts larger than one-half of the hemisphere, large infarcts, infarcts involving more than one lobe, and multifocal infarcts. *Cortical involvement, especially if the infarct has an anterior hemispheric location; 'watershed' topography; irregular borders and areas of incomplete destruction. *Large combined cortical and subcortical infarct, hippocampus involvement * involvement of the middle cerebral artery (MCA) territory, involvement of the parieto-temporal cortex, supramarginal gyrus, and superior temporal gyrus NB: Lacunar, subcortical, and posterior circulation infarcts are infrequently associated with the development of epilepsy	Global and regional brain atrophy	

PLEDs=periodic lateralized epileptiform discharges, FIRDAs=Frontal intermittent rhythmic delta activities

Etiologies of Vascular Seizures and Epilepsy

Subarachnoid Haemorrhage (SAH)

Patients with SAH or those with cerebral venous thrombosis, have a higher rate of seizures than ischaemic stroke patients [3, 5].

Early seizures after admission to hospital complicate the clinical course of 4–10% of patients admitted for SAH. Hemiparesis, cerebral infarct, the clinical and imaging severity of the bleeding, intraparenchymal haematoma and an aneurysm as the cause of bleeding predict the likelihood of post-stroke seizures. Their occurrence is thought to be related to the amount of blood in the basal citerns, which are in direct contact with the frontal and temporal lobes [1, 4, 8].

Intracerebral Haemorrhage (ICH)

ICH is associated with the highest incidence of post-stroke seizures (10.6%-15.4% of them develop the condition).

Factors which are predictive of early and/or late-onset seizures include younger age, the severity of neurological deficits, lobar (cortical) hemorrhage, hemorrhage location within the cerebrum, greater volume of hematoma, subarachnoid hemorrhage (SAH), subdural hematomas and surgical hematoma evacuation (an open surgery can be expected to add neuronal injury to an already damaged area, increasing the risk of seizure). These criteria are indicative of prophylactic antiepileptic drugs (AEDs) [1, 2, 5, 7].

The epileptogenesis process in early seizures is explained by a space-occupying lesion with a resulting mass effect, focal ischemia and direct cellular dysfunction from blood products, while in late seizures, it is caused by gliotic scarring, hemosiderin deposition, neuronal reorganization, and neurodegeneration, with the hemorrhage no longer present. In ICH, there is no evidence that either the amount of blood present in the brain parenchyma, or the amount of blood in the cisternal and ventricular spaces seems to influence the occurrence of seizures [3-5].

Cerebral Arteriovenous Malformations (AVMs)

Epilepsy is the second most frequent presentation for all AVM patients, but its importance is often overlooked in favor of intracranial hemorrhage, which is the most frequent and feared component of an AVM's natural history. Various patient and nidus characteristics are linked to AVM-associated epilepsy, including male gender, younger age, larger unruptured AVM size, cortically based frontal or temporal nidus location, superficial AVM topography, AVM location at an arterial borderzone, absence of intranidal aneurysms, presence of a venous varix, nidus supply by the middle cerebral artery, or the external carotid artery, or a cortical feeding artery, as well as cortical, temporal superficial, or parietal superficial nidus locations. Patients harboring such nidi likely warrant more rigorous neurological monitoring for signs of epilepsy [1, 4, 5].

AVM intervention (surgery, radiosurgery, and embolization) appears to afford favorable seizure outcomes in the majority of patients with AVM-associated epilepsy. Although in some studies, seizure remission appears to correlate with complete nidus obliteration [2, 3].

Cerebral Vein and Dural Sinus Thrombosis

Seizures are a frequent inaugural manifestation of cerebral vein and dural sinus thrombosis (10–61%). Seizures can be focal, generalised or both.

Some patients experience late-onset and, less often, recurrent seizures after the acute phase.

Remote seizures occurred only in patients with early seizures associated with haemorrhages diagnosed by CT/MRI on admission [1, 11].

Ischemic Strokes

6.5% to 8.5% of ischaemic strokes develop post-stroke seizures.

Stroke in Young Adults

Lupus, other vasculitis, the use of recreational drugs can cause both strokes and seizures. 2,8,10

Cerebral Amyloid Angiopathy

It remains unclear whether seizures reflect direct cortical ICH involvement or progressive small vessel damage related to cerebral amyloid angiopathy [9, 10].

Seizures after Carotid Endarterectomy and Angioplasty

Seizures are a rare complication of carotid endarterectomy (0.8%). They are a component of the so called 'hyperperfusion syndrome' that can develop hours or days after surgical relief of long-standing tight carotid stenosis. The vascular pathogenesis of such seizures is not always hyperperfusion; but also due to microembolism, hypoperfusion, or hypertensive encephalopathy [5, 11-12].

Moyamoya Disease

The 2 cardinal clinical signs of moyamoya disease are ischemic attacks or intracranial hemorrhage. Epilepsy is the third most common manifestation of moyamoya disease. About 20%-30% of cases present with seizures. Recurrent seizure attacks in moyamoya disease should be regarded as symptomatic localization-related epilepsy [5, 13].

Transient ischaemic attack

Is associated with the lowest incidence of seizures (3.7%16) [5, 6].

Clinic

Focal and Generalised Seizures

The clinical diagnosis of early seizures is sometimes easy, especially in cases of clonic manifestations on the side of hemiplegia. However, the symptomatology is more often non-specific, with fluctuating arousal, behavioural change or transient worsening on neurological examination. Authors described ictal and post-ic-

tal confusional states of up to seven to eight days. Thus, in these cases, diagnosis can be difficult and, in our experience, standard 20-min electroencephalography (EEG) is rarely contributory [1, 5, 11-12].

Gasparini et al. demonstrated that patients with leukoaraiosis frequently exhibit clinical and EEG signs suggestive of temporal lobe epilepsy, but patients with large vessel infarct had exhibit signs of frontal lobe epilepsy [2-3, 5].

Status Epilepticus (Se) After Stroke [Table 2]

Non convulsive status was the predominant SE type in the early onset post-stroke seizure patients (85%) and was as frequent as convulsive status (50%) in the late-onset group.

In both the acute and chronic situations, NCSE should always be considered in stroke patients in whom there is unexplained change in consciousness and behavior [1, 6, 13-14].

It is also unclear whether early post stroke status epilepticus is a risk factor for late-onset seizures. Several studies have confirmed that status epilepticus as a presentation does not predict subsequent development of epilepsy [2, 16].

Electroencephalogram (EEG)

Standard 20-min EEG recordings of the acute phase of stroke usually show non-specific abnormalities, such as slow waves in the side of the infarct (the most frequent finding) and periodic lateralized epileptiform discharges (PLEDs), but otherwise rarely contribute to a positive diagnosis of epileptic seizures.

EEG can be normal in about 5% of acute stroke and, therefore, normal EEG result does not exclude epileptogenicity. Nevertheless, EEG is necessary to establish the diagnosis of nonconvulsive status epilepticus, and detect persistent epileptiform activity after an apparently successful treatment of convulsive status epilepticus [1, 5, 7, 12-13].

Periodic lateralized epileptic discharges (PLEDs), often associated with metabolic abnormalities (hyperglycemia, fever...) and common abnormalities in the acute phase of severe cerebral ischaemia, are considered as an EEG signature of an unstable neurobiological condition that creates an ictal-interictal continuum in a sick brain, that can lead to seizures when any acute derangements coexist. However, PLEDs are considered as a true epileptic phenomenon (subtle status epilepticus) by some authors. Furthermore, PLEDs are more frequently observed in stroke patients with early-onset seizures and are rarely described in cases of late-onset seizures. Aggressive antiepileptic treatment is recommended by some to enhance recovery, but this attitude is contested by others [4, 13-14]. Focal slowing or diffuse slowing activities, rhythmic slow waves are associated with a low risk of early onset seizures whereas focal spikes, sharp waves, periodic lateralized, or periodic bilateral discharges are associated with a higher risk. Frontal intermittent rhythmic delta activities (FIRDAs) and diffuse slowing can be considered as possible indicators of late-onset seizures [1, 5, 7, 15-16].

Radiological Studies

When CT scan is used in conjunction with EEG and clinical findings, it is a useful diagnostic tool to establish the cause of the first seizure in adults [3, 4]. However, MRI brain is the imaging modality of choice, as it will show a number of abnormalities that may be missed on CT, for example, cortical malformations, hippocampal sclerosis, small mass lesions, and cavernoma, particularly in the temporal lobes [1, 5, 9-10]. Arterial spin-labeling (ASL), is very useful in early seizure and in the differentiation of late seizure due to simultaneous evaluation of changes to both cerebral perfusion and the primary stroke lesion [1, 5, 7, 13-14].

Late-onset epilepsy (LOE) patients have an increased volume of white matter lesions (WML) and lower cortical volumes, which is recognized to reflect cerebral small vessel disease in the absence of another disease process. Seizures may also prompt a vicious cycle, whereby areas of cerebrovascular damage continue to be disrupted by further seizure [1, 8, 11, 16].

Differential Diagnoses

In the context of recent stroke, metabolic and toxic disorders are purveyors of arousal fluctuations and movement disorders, which should not be confused with epileptic clonic seizures. In such cases, EEG recordings are essential for making the diagnosis and avoiding the unnecessary addition of an antiepileptic drug that can worsen encephalopathy [1, 7, 8, 14, 17].

Limb-shaking is clinically manifested by limb tremor mimicking myoclonia, but manifesting exclusively on standing, caused by transient hemodynamic ischemia observed in cases of tight stenosis of the internal carotid artery or, less frequently, the intracranial vessels

Midbrain strokes or acute insults of the subthalamic nuclei, whether ischemic or hemorrhagic, can provoke abnormal movements that mimic epileptic movements)

Treatment

Who should be treated?

Except of cerebral venous thrombosis, antiepileptic drugs (AEDs) are used after a second acute seizure, a prolonged first acute seizure or status epilepticus. Other neurologists, start AEDs after a first acute seizure, after excluding other causes for the seizures, based on the risk of status epilepticus and on the increased risk of remote seizures [1, 4, 11].

A first unprovoked late-onset seizure, is indicative of antiepileptic medication [2, 6, 17].

The choice of adequate treatment

Criteria for helping to select the most appropriate AED include: (1)

efficacy,; (2) tolerability in population who are often elderly and have comorbidities; (3) hepatic and renal function; (4) interactions between AEDs, and with other drugs (antithrombotic*); (5) cost and availability, and patient's preferences (6) [3, 8, 1, 4, 14].

Treatment options should be extrapolated from those of epilepsy in general. Classic AEDs (phenytoin 100-300 mg/day, carbamazepine 400-1600 mg/day or valproic acid 500-2500 mg/day) are the first treatment option. They have Poor safety profile; important drug interactions and adverse effects (negative cognitive effects, deleterious effects on neurological recovery). In cases of stroke associated with atheroma, it is necessary to be aware of classic antiepileptic drug-induced dyslipidemia

Carbamazepine is preferable in partial epilepsies.

The new AEDs (lamotrigine 100200 mg/day; gabapentine 900-3600 mg/day; levetiracetam 1000-3000 mg/day; topiramate 200-400 mg/day; zonisamide; oxcarbazepine and tiagabine) should be reserved for those patients who do not tolerate classic AEDs or in treatment failures with the classic AEDs [1, 5, 8, 11-12, 16-18]. They have good safety profile and less drug interactions than classic AEDs and may be preferable for patients taking several other medications, as is usually the case in stroke survivors. Can also be used in monotherapy or as add-on treatments. Evidence regarding their efficacy as an add-on treatment for drug-resistant partial epilepsy [5, 6].

New AEDs should be the first option in patients who need prolonged oral anticoagulation for secondary stroke prevention [5, 6]. [5,6]

Vascular epilepsy is usually easy to treat with monotherapy, as 80% of patients are seizure-free after 1 year [2, 14].

Treatment principles

An important principle of antiepileptic treatment in the elderly is to 'start low, go slow' [6, 13].

Individual evaluation of benefit-risk balance of antiepileptic medication: risk of seizures and the high probability of adverse effects due to high susceptibility of this population, some of which may be disabling; (e.g. somnolence, gait imbalance and ataxia, confusion, mental slowing, weight gain or loss of appetite and weight loss, and liver toxicity; cognition impairment)

If the residual deficit can be attributed to ischemia and not only postictal deficit, then seizures are no longer a contraindication for intravenous thrombolysis [4, 16, 17].

To be careful in the use of some drugs in stroke patients, this can decrease seizure threshold [1, 7, 12].

For how long we should maintain antiepileptic treatment?

It is recommended to stop medication once the acute stage is over or at the first post-stroke, 3-month visit after an early post-stroke seizure, because, early seizures are not predictive of later vascular epilepsy.1,6,13,17

For remote seizures, a seizure-free period of 2–3 years before a trial of AED withdrawal is recommended. Other neurologists start AED reduction after a seizure-free period of 6-12 months [1, 2, 11, 12].

Management of status epilepticus

Intravenous (IV) diazepam or IV lorazepam is used as a first option, followed by IV phenytoin or fosphenytoin if seizures recur and barbiturate if seizures are not controlled. IV valproic acid is an alternative in patients with contraindications to phenytoin and fosphenytoin [1, 3, 6, 14].

For 'subtle status epilepticus' (synonym: Myoclonic Status Epilepticus in Coma) some neurologist administer high dose intravenous AED therapy (barbiturate valproate, or levetiracetam), for 12 h only, to discontinue and if there is no improvement [1, 5, 11].

Guidelines of the French Neurovascular Society (SFNV)

To use only drugs with demonstrable efficacy in focal epilepsies, such as carbamazepine, oxcarbazepine, lamotrigine and levetiracetam

For the elderly patients, it is recommended to preferentially use lamotrigine, gabapentin, lacosamide and zonisamide [1, 6, 11].

European and North American Guidelines

"Standard IV and oral antiepileptic drugs are in general used (level of evidence: III) [6].

For ischaemic stroke, there is no evidence that prophylactic anticonvulsive treatment is beneficial (level of evidence: IV)" [2, 6].

For intracerebral haemorrhage, it is recommended that "seizure activity must be treated aggressively (level of evidence: V). Prophylactic antiepileptic therapy (preferably phenytoin) may be considered for a month and then tapered and discontinued if no seizure activity occurs during treatment" [8].

Prognosis

Mortality

Early onset of seizures after stroke is often associated with a higher mortality rate than late-onset seizures. The increased mortality may be due both to the severity of the underlying stroke and also the synergistic effect of combined injuries from seizures and cerebral vascular ischemia [8, 11].

-The immediate prognosis of status epilepticus is poor, in particular if of early-onset, with an increased mortality and higher morbidity [10, 33].

Morbidity

The occurrence of epilepsy in a patient with stroke significantly increases infarct size leading to an irreversible state of injury and will often further inhibit recovery and decrease quality of life, an effect that can be ameliorated with the administration of certain neuroprotective agents. Indeed, the seizures, even if infrequent, may result in dependency, increased social restriction, neurological deficit and psychological disability (depression and anxiety which can prove a hurdle in rehabilitation). These effects may be the direct biological consequence of the seizures on the ischaemic brain or secondary psychosocial effects [1, 14, 17-19].

Conclusion

Post-stroke seizures and epilepsy imposes a clinical dilemma in terms of diagnosis and its management is controversial. Randomised controlled trials of AED prophylaxis for acute and remote seizures are essential to improve the evidence level of current guidelines and recommendations for the management of post-stroke seizures

Competing Interests

The authors report no conflicts of interest.

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