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Management of stroke-related seizures

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Abstract

Although general guidelines exist for the diagnosis and the treatment of epilepsy, no specific recommendations are available concerning the management of seizures related to cerebrovascular disease.

The incidence of seizures after stroke was found to be 8.9% in the only published prospective study. This is an underestimation as seizures occurring at stroke onset were not taken in to account and non-convulsive spells are rarely recognized as such.

Risk factors, diagnosis, management and treatment will be different according to the time of onset of the seizures in relation to the stroke.

As, on one hand, repeated seizures and status epilepticus worsen the neurological and the mental condition of stroke patients and, on the other hand, antiepileptic drugs (AEDs) can also increase cognitive impairment, the main questions to be answered are for which patients AEDs should be prescribed and which drug should be preferred.

Key words: Hemorrhagic stroke; cortical infarction; lacunar infarct; partial anterior circulation syndrome; seizures; status epilepticus; non-convulsive spells; transient ischemic attack; antiepileptic drugs.

Introduction

Stroke-related seizures are a neglected topic and generally considered as a benign and a harmless complication occurring in the course of a longstanding and progressive cerebral and cardiovascular disease (Silverman *et al.*, 2002; Camillo and Goldstein. 2004). However, the occurrence of repeated seizures or status epilepticus worsen the neurological and the mental condition of stroke patients (De Reuck *et al.*, 2006a-b). On the other hand, most antiepileptic drugs also promote cognitive impairment in the elderly (Mula and Trimble, 2009).

So, it is important to balance the risk of occurrence of recurrent seizures, leading to epilepsy, against the risk of worsening the condition of the stroke patient by prescribing antiepileptic drugs.

Epidemiology

Stroke-related seizures are not only observed in patients with a cerebral infarct or a bleeding, but also those without visible brain lesions, but with significant vascular risk factors, have a higher incidence of epilepsy in population based surveys (Li *et al.*, 1997; Burn *et al.*, 1997).

In the only large prospective multi-center study an overall incidence of 8.9% of seizures after different types of stroke was observed. However, this study did not include patients who developed seizures at stroke onset (Bladin et al, 2000). In the Stroke Unit of the Department of Neurology of the Ghent University Hospital the overall incidence of stroke-related seizures during 2001 and 2007 was 9.8% (De Reuck 2007).

In the prospective study patients with an intracerebral hemorrhage were found to have a higher incidence of seizures (10.6%) than those with an ischemic stroke (8.6%), while in those with a subarachnoid bleeding the incidence was similar to that in patients with a brain infarction. A low incidence of 2.5% of seizures was observed in patients with lacunar strokes (Bladin *et al.*, 2000).

Due to the fact that ischemic strokes are far more frequent than hemorrhagic ones, the majority of stroke-related epileptic insults are due to cerebral infarction. In our series seizures caused by an intracerebral bleeding were only observed in 4.9% of our stroke patients (De Reuck, 2007).

Classification

Stroke-related seizures can be classified as those of early- and of late-onset. In a paradigm comparable to posttraumatic seizures an arbitrary cut point of

2 weeks after stroke onset will be considered to distinguish between early- and late-onset seizures (Jennett 1974). Most of the early-onset seizures occur within the first days (Arboix *et al.*, 1997). The first late-onset attack takes generally place between 3 months and 2 years after stroke but still 28% of the stroke patients develop their first late-onset seizure much later and even after several years. Also, twenty-three percent of late-onset seizures are the clinical expression of a recurrent infarction (De Reuck *et al.*, 2007a). So, stroke-related seizures are best classified as those of early-, late- and very late-onset and those due to recurrent stroke (De Reuck *et al.*, 2008a).

According to the clinical presentation one can subdivide the seizures in simple partial and complex partial ones, with or without secondary generalization and in those considered as primary clonic-tonic spells. As this classification will be mainly based on the description given by the family or caregivers, one has to be aware that the so-called primary clonic-tonic seizures are most probably of focal onset with rapid secondary generalization. Overall, simple partial seizures accounts for about 50% of the stroke patients, while complex partial spells and primary generalized clonic-tonic insults are observed in approximately 25% each (De Reuck *et al.*, 2006c). Early–onset seizures can remain restricted to the affected body side where as late-onset ones are more likely to generalize secondarily (Davalos *et al.*, 1992). However, status epilepticus occurs more frequently at onset of a severe stroke (Velioglu *et al.*, 2001; De Reuck and Van Maele, 2009b).

Risk factors

The vascular risk factors, such as arterial hypertension, coronary artery disease, isolated atrial fibrillation, cardiac valvular disorders, peripheral artery disease, diabetes, hypercholesterolemia and smoking have the same incidence in patients with stroke-related seizures as in those without seizures. Only chronic obstructive pulmonary disease (COPD) is an independent risk factor for seizures in stroke patients. The occurrence of seizures is not related to the severity of the COPD or to its type of treatment. Most probably it is associated to obstructive sleep apnea and to the accompanying nocturnal low oxygen saturation, which are frequently observed in COPD patients (De Reuck *et al.*, 2007b).

Early-onset seizures in ischemic stroke are mainly observed in patients with large cortical lesions and severe strokes (Arboix *et al.*, 2003).

Patients who present with a partial anterior circulation syndrome, according to the Oxfordshire classification of ischemic strokes (Bamford *et al.*, 1991), have the highest risk to develop late-onset seizures. Patients with a total anterior circulation syndrome have less chance of developing late-onset seizures not only due to their shorter life expectancy but also due to the fact that the large infarcts are more sharply demarcated (De Reuck *et al.*, 2005, 2008b).

Large cortical infarcts with irregular borders and located in the parietal and temporal regions represent an increased risk of developing late-onset seizures (De Reuck *et al.*, 2008c).

Patients who develop seizures at stroke onset have only a low risk to develop late-onset ones afterward, while the risk is high in those who develop their first epileptic spell between 3 months and 2 years after the cerebrovascular accident (Berges *et al.*, 2000; Lamy *et al.*, 2003). The recurrence rate is also very low when the seizure occurs many years after the stroke. A patient with a first primary generalized clonic-tonic spell after a subcortical infarct carries also a lower risk to develop epilepsy (De Reuck *et al.*, 2008c).

Status epilepticus is mainly observed in patients with a severe stroke. It occurs in 20.7% of patients at stroke onset, in 11.7% of those of late-onset and only in 2.1% of those of very late-onset (De Reuck *et al.*, 2008a). It occurs predominantly when the convex surface of the temporal lobe is involved in the infarct zone (De Reuck *et al.*, 2008c).

Seizures are also observed in patients with lacunar infarcts and white matter changes, demonstrated on computed tomography (CT) and magnetic resonance imaging (MRI) of the brain. However, no differences in number and location of the lacunes, and in the severity of the white matter changes on CT or MRI are found in the patients with and without seizures (De Reuck *et al.*, 2007c). Seizure occurrence correlates to the degree of cognitive impairment, suggesting that both are related to the same neurodegenerative changes in the cerebral cortex and not directly to the presence of the white matter changes and the lacunar infarcts (De Reuck and Van Maele 2009a).

The mechanism of seizure initiation by a hemorrhagic lesion is not very well established. Products of blood metabolism, such as hemosiderin, may cause a focal irritation, leading to seizures (Silverman *et al.*, 2002). Patients with a frontal lobar hematoma are more prone to develop early- (Vespa *et al.*, 2003) as well as late-onset seizures (De Reuck *et al.*, 2007d). Also hemorrhagic infarcts initiate more seizures than non-hemorrhagic ones (Lancman *et al.*, 1993).

There is no overall evidence from the literature that stroke-related seizures occur more frequently in

patients with cardiac- versus thrombi-embolic infarcts (Silverman *et al.*, 2002; Camilo and Goldstein 2004). A cardiac-embolic etiology of the stroke is only an increased risk factor in patients with seizures due to recurrent cerebral infarction (De Reuck *et al.*, 2007a).

A rare cause of transient focal seizures is the "reperfusion syndrome" that can occur after revascularization procedures, most commonly after carotid endarterectomy or stenting for chronic severe extracranial carotid stenosis (Ho *et al.*, 2000).

Finally, one has to take into account that a seizure episode is not always related to a previous stroke and that alcohol or drug abuse can also be a triggering factor (Loiseau *et al.*, 1990).

Etiology

The appearance of early-onset seizures can be explained by an unstable neurobiological condition during the instauration of the stroke, which creates an ictal-interictal continuum and leads to seizures when any acute derangements co-exist (Pohlmann-Eden *et al.*, 1996). Periodic lateralized epileptic discharges (PLEDs) on the electroencephalogram (EEG) are considered as the signature of this unstable condition. In a focal ischemic rat model PLEDs appeared over the penumbral regions, while intermittent rhythmic delta activities (IRDAs) recurred in the contralateral hemisphere with frontal and parietal dominance (Hartings *et al.*, 2003).

The pathogenesis of late-onset seizures is less clear. A common finding after an epileptic insult in a patient with sequels of a previous stroke is the temporary worsening of the neurological deficit, also called Todd's paresis. In some rare instances the increased deficit after the seizure can remain permanently, in particular after prolonged convulsions, without any new visible infarct on CT of the brain. It has been speculated that a prolonged convulsive attack can be responsible for additional ischemic damage (Bogousslavsky *et al.*, 1992).

Positron emission tomography (PET) studies have demonstrated that the severity of the ischemic changes within the borders of the infarct zone, seen on CT, is more pronounced in patients with seizures compared to a group of stroke patients without a history of seizures but with a similar degree of neurological deficit and a comparable infarct size. Patients with recurrent late-onset seizures have on ^{15}O PET scans a more severe decrease of regional blood flow and oxygen consumption, without increase in oxygen extraction fraction, than those with a single seizure (De Reuck *et al.*, 1995).

Delayed seizures after an ischemic stroke, are accompanied by an increase in lesion size on CT of the brain, showing increased cerebral damage due to the seizures themselves (De Reuck *et al.*, 2006a).

Diffusion-weighted and perfusion MRI have demonstrated parenchymal changes in partial status epilepticus (Szabo *et al.*, 2005). In a MRI study, performed within 8 days after a seizure following an ischemic stroke, 50% of the patients have a positive diffusion weighted imaging (DWI) with decreased apparent diffusion coefficient. A positive DWI rim is observed in 20.8% of the patients with late-onset seizures, to be considered as cytotoxic edema and leading to increase of the ischemic damage. In 22.6%, a large positive DWI zone is observed corresponding to a new recent infarct. All patients with early-onset seizures have a positive DWI corresponding to the establishing infarct (De Reuck *et al.*, 2007a).

Diagnostic procedures

In an elderly person who presents a first late-onset seizure a cerebrovascular cause has to be suspected. Every patient with a convulsive seizure following a previous stroke has also to be reinvestigated for the possibility of a new cerebrovascular accident. Admission to a stroke unit for 24 to 72 hours is advisable.

An EEG should be performed as soon as possible after the ictal event. PLEDs are only observed in a minority of cases of late-onset seizures. Focal slowing corresponding to the side of the infarct is the most frequent EEG finding (Ryglewicz *et al.* 1990; Niedzielska *et al.*, 2001). PLEDs are, however, frequently observed in patients with early-onset seizures, when the EEG is performed soon after the stroke onset. In our study PLEDs were observed in 25% of patients with early- and only in 1% of those with late-onset seizures. PLEDs, IRDAs and diffuse slowing together are seen in 26.5% of patients after a late-onset seizure compared to 6.2% in stroke patients without epileptic spells. A normal post-ictal EEG is only observed in 5.1% of the former compared to 53.8% of the latter group (De Reuck *et al.*, 2006c).

An urgent CT scan of the brain is necessary for different reasons. First of all, it can sometimes demonstrate a new infarct in patients who have already an old one. Secondly, a "silent" old infarct as cause of a late-onset seizure can be demonstrated. The third reason is that when a cortical infarct with a capricious distribution and with areas of apparently preserved cerebral tissue within the infarct region is demonstrated, there is an increased risk that the

patient can develop late-onset seizures than in those with a sharply demarcated infarct zone on CT of the brain.

MRI is more sensitive than CT of the brain and should be performed as early as possible. It is useful to exclude conditions other than a cerebrovascular accident as cause of the seizures. Also DWI can demonstrate additional secondary damage due to the seizure itself or an additional recent infarct as cause of the epileptic spell (De Reuck *et al.*, 2007a).

Patients who develop seizures after a stroke should also have an extensive blood examination in order to exclude or demonstrate possible metabolic or toxic provoking factors.

Patients with a post-stroke seizure should be reinvestigated in the same way as after their cerebrovascular accident. They should have an exhaustive cardiovascular examination, including a 24-hour electrocardiogram, cerebrovascular ultrasound of the carotid arteries and transthoracic echography or transoesophageal Doppler examination of the heart in order to investigate for possible sources of emboli. Conventional or MR angiography should be performed, if necessary.

The cognitive status of the patient should always be determined in all patients with a late-onset seizure at discharge from the hospital or after a reasonable time of recovery after the epileptic spell.

Non-convulsive or inhibitory seizures

A recent study has demonstrated that electric epileptic activity on EEG, in patients with a severe acute stroke, occurs more frequently than previously suspected even in the absence of focal convulsions (Carrera *et al.*, 2006). Also non-convulsive status epilepticus is found to be more frequent than the convulsive SE at stroke onset (Labovitz *et al.*, 2001).

Patients with delayed transient worsening of neurological deficits after an ischemic stroke have to be investigated for the possibility of inhibitory seizures. An inhibitory seizure episode is the cause of a transient worsening of the neurological signs in more than 22% of patients with a previous stroke. The subsequent occurrence of a similar attack, accompanied by focal convulsions, or the EEG recording elaborates the diagnosis (De Reuck *et al.*, 2006d).

Prognosis

The outcome of patients with early-onset seizures is poor with a high incidence of status epilepticus (De Reuck and Van Maele, 2009b) and with a high in-hospital mortality rate (Rumbach *et al.*, 2000).

Patients with late-onset seizures have a recurrence rate of more than 50% (Berges *et al.*, 2000; Lamy *et al.*, 2003). Recurrent seizures and status epilepticus increase the disability of stroke patients. Also vascular cognitive decline is promoted. A single seizure does not seem to affect the disability and the mental status to a significant degree (De Reuck *et al.*, 2006a-b).

Treatment

First of all, the best available vascular treatment should be established according to the etiology of the stroke. Also, the vascular risk factors should be drastically corrected.

In early-onset seizures and status epilepticus urgent treatment with intravenous administration of benzodiazepines should be started. As the occurrence of late-onset seizures is rather low in patients who had seizures at stroke onset, maintained antiepileptic drug treatment is not mandatory on discharge from the hospital after the stroke. Only when late-onset seizures eventually delay/appear, one has to start with sustained antiepileptic drug treatment.

The main question remains whether prevention with antiepileptic drugs should be started in some stroke patients who are particularly at risk of developing late-onset seizures (Labovitz and Hauser, 2003). As a first late-onset seizure does not seem to impair the cognitive status of the patient to a significant degree, it does not seem necessary to start with antiepileptic drugs as prevention for seizures after stroke, similar to the guidelines in traumatic brain disease (Temkin 2009).

Due to the high risk of recurrence after a first late-onset seizure (Berges *et al.*, 2000; Lamy *et al.*, 2003) treatment with antiepileptic drugs becomes mandatory after the first epileptic spell.

The choice of the anticonvulsive medication should be guided by the individual characteristics of each patient, including concurrent medications and medical co-morbidities.

The first-generation of antiepileptic drugs undergo significant hepatic metabolism. Phenytoin and valproate sodium are highly protein bounded. The well-recognized interaction of warfarin with phenytoin makes it difficult to maintain consistent therapeutic ranges of both agents in patients with atrial fibrillation. No controlled trials have been conducted to compare mutually the efficacy of these first-line agents in post-stroke epilepsy (Silverman *et al.*, 2002). Given the typical focal onset of late-onset stroke-related seizures and the moderate effects on cognition, until recent, our first-line option was to use carbamazepine as monotherapy (De Reuck, 2007).

In a trial of newly diagnosed epilepsy in the elderly, lamotrigine was demonstrated to be better tolerated and to maintain patients free from seizures for longer intervals than carbamazepine (Brodie *et al.*, 1999). In a small more restricted study on patients with post-stroke seizures, lamotrigine was also found to be better tolerated than carbamazepine (Gillad *et al.*, 2007).

In a small series Levetiracetam as monotherapy was also shown to be effective and well tolerated in elderly patients with post-stroke seizures (Kutlu *et al.*, 2008).

A recent overview proposes low-dose lamotrigine or gabapentine as the optimal first-line therapy for post-stroke seizure and epilepsy in elderly patients or in younger ones requiring anticoagulants. However, low-dose extended-release carbamazepine remains a reasonable and less expensive option (Ryvlin *et al.*, 2006).

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