Early Onset Seizures in Stroke

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Case Description
A 43-year-old woman with medical history of hypertension, myocardial infarction, ischemic cardiomyopathy, and systolic heart failure with an ejection fraction of 20% to 25% on a recent echocardiogram developed sudden onset of left hemiparesis, left hypoesthesia, left gaze deviation, and mutism. Two hours after symptom onset her initial National Institutes of Health Stroke Scale score was 25. A computed tomography of the head was unremarkable, but a computed tomography angiogram showed a distal right middle cerebral artery occlusion. She was given intravenous tissue-type plasminogen activator, but soon after tissue-type plasminogen activator administration, the patient had a generalized tonic clonic seizure. A repeat computed tomography of the head immediately after the seizure showed no hemorrhage, and the patient received mechanical thrombectomy for a her right middle cerebral artery occlusion. She was started on levetiracetam for secondary seizure prevention. MRI showed multiple areas of diffusion restriction involving all vascular territories consistent with a cardioembolic source and her history of dilated cardiomyopathy. She was started on anticoagulation. Importantly there were both cortical and subcortical areas with diffusion positive signal. Electroencephalography showed severe diffuse encephalopathy without epileptiform discharges. She made a good recovery and her discharge National Institutes of Health Stroke Scale score was only 4. She had no more seizures and was discharged home on warfarin, statin, and levetiracetam with seizure restrictions. Two months later she had a minimal hemiparesis and returned to work. She had no further seizures. Although it was explained that the plan had only been to continue anticonvulsants for 3 months, she wanted to drive as soon as possible and elected to continue anticonvulsants indefinitely.

Discussion
Stroke is the most common cause of seizures in the elderly, and seizures are the most common neurological sequelae of stroke. In the Seizure after Stroke Study,1 8.6% of ischemic stroke patients and 10.6% of hemorrhagic stroke patients had seizures. Seizures after stroke can be divided into 2 broad categories depending on their time of onset. Although definitions vary depending on the series, many groups define early onset seizures as occurring within 2 weeks of stroke onset and late onset seizures as occurring after 2 weeks. More than half of stroke-related seizures occur in the early period. Among the 8.6% of patients with ischemic stroke who had seizures in the Seizure after Stroke Study, 56% were early onset seizures and 44% were late onset seizures. Among the 10.6% of patients with hemorrhagic stroke who had seizures, 75% were early onset and 25% were late onset seizures. Most early onset seizures occur during the first 24 hours. The incidence of seizures after stroke is also increased with cortical location and larger volumes. The CA VE score has recently been developed to stratify patients more likely to develop late onset seizures after intracerebral hemorrhage. The score gives points for cortical location (1), age <65 years (1), volume >10 mL (1), and the presence of an early onset seizure (1). The risk of late onset seizures is ≈0.6%, 3.6%, 9.8%, 34.8%, and 46.2% for CA VE scores 0 to 4, respectively.2

The development of epilepsy, or recurrent seizures, after stroke occurs at varying rates, but it is higher after late onset seizures than early onset seizures. In patients with ischemic or hemorrhagic stroke, epilepsy developed in ≈30% of patients with early onset seizures and in 90% of patients with late onset seizures.3 The risk of epilepsy is higher in patients who experience an early onset seizure (30%) than in patients with stroke in general (5%–20%).2,3 Interestingly, patients with intracerebral hemorrhage tend to have a higher risk of developing epilepsy than those with ischemic stroke (10%–15% versus 6%–9%).1,3

The explanation for higher rates of late onset seizures progressing to epilepsy is likely because of the differing pathophysiology of early and late onset seizures. Early poststroke seizures are thought to be secondary to regional metabolic dysfunction and excitotoxic neurotransmitter (largely glutamate) production because of ischemia. This metabolic dysfunction is self-limited. However, late onset seizures are thought to be because of gliosis and meningocerebral scar, which is permanent.1

Management
There are no specific guidelines on optimal timing and type of antiepileptic drugs after poststroke seizure4 and little
Seizures are common after stroke. Early and late onset seizures differ in terms of the mechanism of seizure and the likelihood of developing epilepsy. Anticonvulsants can have serious side effects both during rehabilitation and long-term therapy. Withdrawal of anticonvulsants after recovery from stroke may be difficult because of restrictions placed on patients after withdrawal, especially driving restrictions.
Disclosures

None.

References


Key Words: antiepileptic agents ■ epilepsy ■ seizures ■ stroke
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Stroke. 2014;45:e249-e251; originally published online October 7, 2014;
doi: 10.1161/STROKEAHA.114.006974
Stroke is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0039-2499. Online ISSN: 1524-4628

The online version of this article, along with updated information and services, is located on the
World Wide Web at:
http://stroke.ahajournals.org/content/45/12/e249

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