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Letter to the Editor

Use of Statins for Post-Stroke Epilepsy

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To the Editor,

We read with great interest an article by Tseng WJ et al.[1] "Post-Stroke Epilepsy: Current Understanding." Stroke is one of the leading causes of mortality and morbidity in adults in most countries.[2–4] Stroke is a common cause of acquired epilepsy, and nearly 50% of post-stroke epilepsy (PSE) patients are elderly. Hemorrhagic stroke has a higher incidence of post-stroke seizure than ischemic stroke, and reports were 7% overall.[1] Statins are inhibitors of 3-hydroxy-3-methylglutaryl coenzyme A reductase. In addition to lowering plasma levels of lipid, statins are also reported to have anti-thrombotic, anti-inflammatory and anti-oxidation effects, which have been suggested to relate to its anti-convulsant potential,[1] an interesting point. The cholesterol-lowering statins have garnered attention for possible neuroprotective effects, tied to their putative pleiotropic anti-excitotoxic, immunomodulatory, and antioxidant properties. These effects of statins have potential impacts on key processes in epileptogenesis, including reducing inflammatory mediators, protecting the blood-brain barrier, and preventing neuronal death. Several recent in vivo studies seem to support such potential, with findings associating statin use with decreased epilepsy incidence in some animal and geriatric human populations.[5] Pre-stroke statin use was not associated with PSE. However, post-stroke statin use was associated with lower risk of both early-onset seizures, and PSE. Review of 7 cohort studies suggested post-stroke, but not pre-stroke, statin use may be associated with reduced risk of early-onset seizures and PSE. Further research is warranted to validate these findings in broader populations and better parse the temporal components of the associations.[6] We believed that future investigations focus on: 1) Increasing understanding of the mechanisms underlying statins' apparent anticonvulsant and anti-epileptogenic effects following stroke, with important considerations being differential effects in ischemic versus hemorrhagic stroke, with lipophilic versus hydrophilic statins, and with high- versus low-intensity statin therapy; 2) Prospective, clinical observational studies to better assess early onset seizures, and important, potential confounders that were not readily available within claims data, including such key considerations as stroke severity, cortical involvement, and hemorrhage volume; and, 3) Further delineating the temporal component of prophylactic statin effects following stroke, particularly in terms of the optimal duration and timing of post-stroke statin use.[6]

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