

STATINS ARE MORE EFFECTIVE THAN ASPIRIN IN SECONDARY STROKE PREVENTION: YES

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It is a myth that cholesterol is not a risk factor for stroke; this is a relic from the days when hypertension dominated stroke risk to such an extent that the importance of cholesterol was masked. In a community where hypertensive strokes were reduced by improved blood pressure control¹, we showed² that cholesterol is clearly a risk factor for stroke.

The magnitude of risk reduction with cholesterol lowering can best be seen in patients with carotid stenosis. Although in the SPARCL trial³ the intention-to-treat analysis showed only a 16% reduction of stroke risk, many patients randomized to placebo received statins, so the intention-to-treat analysis underestimated the benefit of atorvastatin. The increase in intracerebral hemorrhage (ICH) in SPARCL among patients randomized to atorvastatin was not due to atorvastatin, since patients with ICH did not have lower levels of LDL; it was most likely due to patients stopping all their medications (including their antihypertensive drugs) when they stopped their study medication because of adverse effects. Statins probably do not cause either ICH⁴ or liver problems⁵.

Among patients in SPARCL with large artery disease⁶, atorvastatin reduced stroke by 33%, even in the intention-to-treat analysis. We found⁷ that in patients with asymptomatic carotid stenosis, intensive medical therapy based on maximizing the dose of statin, the two-year risk of stroke was reduced from 8.8% to 1%, and the 2-year risk of myocardial infarction was reduced from 7.6% to 1%. This is a much bigger effect than the 25% reduction of stroke with Aspirin. Other antiplatelet agents are only marginally better than aspirin, so instead of focusing on which antiplatelet agent to use, we should be focusing on reducing risk. Furthermore, the appropriate treatment for secondary stroke prevention depends on the underlying cause; just as not all patients with stroke require statins (for example, young people with normal arteries whose stroke is due to paradoxical embolism or dissection), not all patients should receive antiplatelet agents (for example, those with cardioembolic stroke, who should be anticoagulated). With the aging of the population and better blood pressure control, fewer strokes are lacunar strokes from small vessel disease, and a higher proportion are from large artery disease (which will benefit greatly from statins) or cardioembolic (which requires anticoagulation, not antiplatelet agents). Aspirin is therefore becoming less important in stroke prevention, and statins more important.

References

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