LIPIDS AND STROKE: THE LOWER THE BETTER (CON)

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In large epidemiological studies such as the Framingham study, elevated cholesterol is not a risk factor for stroke(1;2). The reason is that in contrast to myocardial infarction, there are many causes of stroke. Elevated LDL or a high ratio of LDL to HDL is clearly a risk factor for coronary artery disease, and for strokes due to carotid stenosis. However, lacunar infarctions (are due to hypertension and diabetes; intracranial haemorrhages are due to hypertension, amyloid angiopathy, vascular malformations or aneurysms; approximately 20% of strokes are cardioembolic (3). These patients, along with those whose strokes are due to vasculitis, venous infarction, paradoxical embolism, atrial myxoma, dissection and other causes of stroke are best treated specifically for the cause of stroke, and lipid-lowering therapy will not benefit them. Indeed, in the SPARCL trial atorvastatin was associated with an increased risk of hemorrhage: the risk was 1.68 (95 percent confidence interval, 1.09 to 2.59) (4;5).

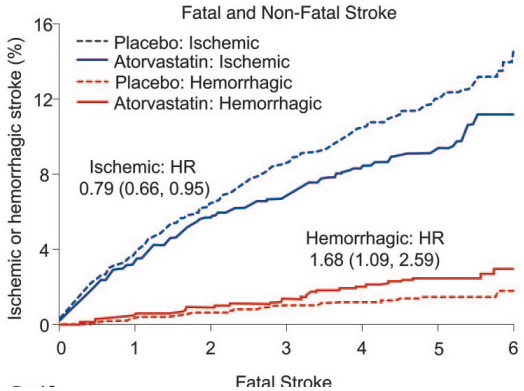

Figure 1. Risk of stroke with atorvastatin in the SPARCL trial: from Neurology® 2008;70:2364-2370

Furthermore, cholesterol is an integral part of myelin development, and lower cholesterol levels are associated with increased suicide, criminal behaviour, mortality and conduct disorders(6).

In our Urgent TIA clinic, among 1,500 new patients referred each year, only 10% are due to symptomatic severe carotid stenosis; those patients would clearly benefit from statin therapy, if only to reduce their significant risk of coronary events(7). However, the 5.5% that are from paradoxical embolism, and the additional 15-20% that are cardioembolic from other mechanisms require anticoagulation; those with giant cell arteritis require corticosteroids; etc, etc, etc.

The basic principle of stroke prevention is that in order to prevent the next stroke, it is necessary to define the cause of the incident stroke or TIA (8).

In secondary stroke prevention, intensive application of all the therapies available can achieve 80-90% reduction of stroke(9), but lipid lowering therapy represents only a small fraction of this benefit. To be properly applied the treatment must be specific to the cause of the TIA or stroke. In patients without coronary artery disease, statins only reduce ischemic stroke by 0.1-1.9%. Mindless treatment of all stroke patients with high-dose statins is not good Neurology, nor is it good Medicine.

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