Response to Letter by Sheikh

Response:

We appreciate Dr Sheikh’s thoughtful comments on our work. The key issue is whether or not total serum cholesterol (TSC) taken within 24 hours after a stroke reflects usual cholesterol levels. There are pros and cons. This is discussed in the article. It is important to note, however, that a correlation between stroke severity and an alteration in the lipid profile in the first few days after the stroke has not been demonstrated (Reference 30 in the article). Dr Sheikh claims that poststroke TSC levels are not representative of the levels before onset of the arterial disease, but he does not provide proof of his claim. It is premature, therefore, to deem our findings unreliable and misleading.

In line with others, we found that TSC measured within 24 hours after an ischemic stroke was inversely related to outcome (References 17 to 19 in the article). A neuroprotective role of cholesterol was suggested as an explanation of this finding. We observed an almost linear association between cholesterol and stroke severity—higher cholesterol levels were associated with smaller stroke. Hence, we instead propose the hypothesis that cholesterol primarily gives rise to smaller stroke (with a better prognosis).

When looking for the link between cholesterol and stroke, many large-scale studies have handled stroke as one entity (Reference 9 in the article). We now know that TSC in patients with hemorrhagic stroke is lower than in patients with ischemic stroke—this is widely accepted and also the finding in our study (unpublished). Therefore, hemorrhagic and ischemic strokes are now handled separately when studying the cholesterol/stroke association. We now propose that this should be done for ischemic strokes as well. It cannot just be assumed that cholesterol plays the same role in the development of embolic and thrombotic strokes and in large vessel and small vessel strokes. All ischemic stroke subtypes may be influenced by TSC, but we propose the hypothesis that TSC plays a more prominent role in some subtypes than in others.

Our hypothesis may help to explain paradoxes in the stroke-cholesterol relation. We may be wrong, we may be right, but still we find our hypothesis worth considering.

Disclosures

None.

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