Low Levels of Low-Density Lipoprotein Cholesterol Increase Hemorrhagic Transformation but Not Parenchimal Hematoma in Large Artery Atherothrombosis

To the Editor:

We read with interest the article by Kim and colleagues reporting on the effect of low level of total cholesterol and low low-density lipoprotein cholesterol (LDLC) on hemorrhagic transformation (HT) after acute ischemic stroke. The authors found that low levels of LDLC are independently associated with the risk of HT after acute ischemic stroke attributable to large artery atherothrombosis (LAA) but not to cardioembolism (CE). The authors commented on the results saying that they should be interpreted with caution in consideration of several points: the study was conducted in a retrospective manner, some of the patients had total cholesterol levels without LDLC levels, they did not distinguish between symptomatic and asymptomatic HT, the cholesterol level may change in the acute stages of stroke, and finally, the long-term outcome of HT was not evaluated in the study. We showed that only parenchimal hematoma (PH) but not hemorrhagic infarction is associated with an adverse outcome at 3 months. In our study, of 1125 stroke patients included, 191 had stroke attributable to LAA and 300 to CE. HT was more prevalent in CE subgroup (51/300, 17%; 30 hemorrhagic infarction and 21 PH) than in LAA subgroup (21/191, 11%; 15 hemorrhagic infarction and 6 PH). In those patients with CE the mean value of LDLC was 111.2±34.0 mg/dL; 108.0±28.3 mg/dL in patients with HT (P=n.s.) and 107.1±26.9 mg/dL in patients with PH (P=n.s.). In patients with LAA, the mean value of LDLC was 114.1±33.9 mg/dL; 100.5±29.5 mg/dL in patients with HT (P=0.05) and 97.1±16.1 mg/dL in patients with PH (P=n.s.). Lower LDLC levels were related to an increased risk of HT after ischemic stroke due to LAA, with the risk of HT increasing by 2% for each 1 mg/dL decrease in the LDLC level (treating LDLC as a continuous variable), after adjusting for other risk factors (odds ratio 0.98, 95% CI 0.96 to 1.00, P=0.05). This was not the case for the association between PH and the LDLC level after adjusting for other risk factors (odds ratio 0.98, 95% CI 0.95 to 1.02, P=n.s.). A low level of LDLC was not associated with HT or PH in patients with stroke due to CE (odds ratio 0.99, 95% CI 0.98 to 1.00, P=n.s.; odds ratio 0.99, 95% CI 0.98 to 1.01, P=n.s. respectively). Also our data should be interpreted cautiously because they ultimately rest on just 6 PH occurrences in LAA cases.

In conclusion, the association among HT, PH, low levels of LDLC and outcome in stroke attributable to LAA should be considered in clinical stroke research in the future.

Disclosures

None.

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Stroke. 2009;40:e544; originally published online July 23, 2009;
doi: 10.1161/STROKEAHA.109.556399

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

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World Wide Web at:
http://stroke.ahajournals.org/content/40/9/e544

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