
Stroke type (ischemic versus hemorrhagic) is often undetermined in resource-poor countries because of lack of access to CT scanners, yet low- and middle-income countries carry more than two thirds of the global burden of stroke. Berkowitz et al conducted a decision analysis using Markov models to evaluate the potential effect of aspirin therapy on long-term secondary prevention of stroke of undetermined type. Primary outcomes were mortality, incidence of ischemic stroke, incidence of intracerebral hemorrhage (ICH), and quality-adjusted life years. Sensitivity analyses were performed across the worldwide range of reported proportions of stroke caused by ICH (9% to 60%) and on all aspirin-associated relative risks (RRs).

Using the most stringent threshold of 34% of strokes caused by ICH (the highest proportion reported in a large epidemiological study), aspirin was predicted to prevent 11 ischemic strokes per 1000 patients per year at a cost of 4 more ICH per 1000 patients per year. Aspirin was also predicted to decrease yearly mortality from 103.32 to 102.97 per 1000 patients per year. Aspirin yielded 3.38 quality-adjusted life years, whereas no treatment resulted in 3.32 quality-adjusted life years. Based on the estimated 11 590 204 strokes in low- and middle-income countries in 2010, the model predicted that aspirin therapy for secondary stroke prevention in all patients with stroke in these countries could lead to an estimated yearly decrease of 84 492 recurrent strokes and 4 056 stroke-related mortalities. Sensitivity analysis of aspirin-associated RR of recurrent ICH after initial ICH would have to be 1.8-fold higher than the reported RR to favor not treating with aspirin, and the aspirin-associated RR of acute mortality after ICH would have to be 5.4-fold higher than the reported RR to favor no aspirin treatment. These findings are in favor of aspirin for secondary stroke prevention in patients with stroke of undetermined pathogenesis in resource-limited settings across a broad range of clinically plausible parameters.

This was a well-designed decision analysis to evaluate therapeutic effectiveness and safety of aspirin in resource-poor settings where neuroimaging is unavailable. The study has limitations related to the data imputed in the model. The figures imputed in the decision trees were collected predominantly in high-income countries and therefore may not necessarily reflect outcomes in low-income regions. However, given that more detailed epidemiological data on cerebrovascular disease in resource-poor countries are not currently available, authors used the best available data and accounted for inherent limitations in these data through rigorous sensitivity analyses.

As low- and middle-income countries now carry more than two thirds of stroke burden, there is increased urgency in conducting epidemiological studies and clinical trials in resource-poor nations to guide clinical practice in all aspects of stroke care.


Several studies have suggested a survival benefit among obese patients with stroke. This obesity paradox has led to uncertainty about the usefulness of weight loss in secondary stroke prevention. Dehendorff et al conducted a retrospective analysis of the Danish Stroke Registry from 2003 to 2012 (linked to Danish Registry of Causes of Death) to evaluate the relationship between body mass index (BMI) and death after stroke. To avoid selection bias, authors selected deaths within the first week or first month after stroke and adjusted for admission stroke severity using the Scandinavian Stroke Scale, in addition to other covariates.

Of 71 617 patients with stroke, 53 812 (75.1%) had information on BMI. The mean BMI was 25.7 kg/m²; 9.7% were underweight, 39.0% were normal weight, 34.5% were overweight, and 16.8% were obese. BMI was inversely related to mean age at stroke onset; compared with normal weight patients, stroke occurred 3 and 6 years earlier in overweight and obese patients. Stroke was the cause of death in 76% of the deaths within the first week and in 70% of the deaths within the first month post stroke. There was no difference in the risk for death in the first month among patients who were normal weight (reference), overweight (hazard ratio [HR], 0.96; 95% confidence interval [CI], 0.88–1.04), and obese (HR, 1.0; 95% CI, 0.88–1.13). Analysis of deaths within 1 week gave similar results (overweight HR, 0.97; 95% CI, 0.86–1.09 and obese HR, 1.05; 95% CI 0.88–1.24). Underweight status trended toward higher stroke mortality at 1 week (HR, 1.07; 95% CI, 0.93–1.24) and 1 month (HR, 1.16; 95% CI, 1.06–1.26).

These findings add to the growing body of literature showing no survival benefit associated with obesity. Numerous factors may explain the variability in obesity-related mortality estimates, including variable BMI definitions, differential effect of body fat distribution, obesity with and without metabolic syndrome, and inadequate adjustment for metabolic factors. This study’s strengths include large sample size, linkage with death records, adjustment of stroke severity and other confounders, and multiple imputations for cases with nonmissing and missing BMI. Despite best efforts, the study cannot exclude the possibility of bias caused by variables not recorded in the registry, such as the use of treatments (eg, tissue-type plasminogen activator, antithrombotic, statin) and interventions (eg, thrombectomy) that might have influenced survival.
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