
Little is known about long-term survival after stroke in individuals with atrial fibrillation (AF). Fang et al conducted a retrospective study involving 13,559 individuals with AF, aged ≥18 years, in 1996 to 1997 in the Kaiser Permanente Northern California database, followed for a median of 6 years. Subjects with and without a history of ischemic stroke (n=1025 in each group) were matched for demographics (age, sex, race), congestive heart failure, hypertension, age >75 years, diabetes mellitus, stroke (CHADS$_2$) score, congestive heart failure, hypertension, age, diabetes mellitus, stroke, vascular disease, and sex (CHA2DS$_2$-VASc) score, and time of entry into the cohort. Primary outcome was all-cause mortality. Long-term mortality was compared between stroke and nonstroke patients and by severity of stroke using modified Rankin Scale at discharge.

Among stroke patients with AF, 30-day mortality was 24.7%, 1-year mortality was 40.3% (95% confidence interval [CI], 37.3%–43.5%), 2-year mortality was 51.9% (48.7%–55.1%), and 5-year mortality was 72.8% (69.4%–76.1%). Median survival in those with and without stroke was 1.8 versus 5.7 years (hazard ratio, 2.5; 95% CI, 2.5–3.2). The survival difference persisted among those who survived beyond 6 months after index stroke (hazard ratio, 2.0; 95% CI, 1.7–2.5). The severity of stroke was strongly associated with subsequent survival. Subjects with minor or no deficit after stroke were 1.7-fold more likely to die than those without stroke. In contrast, survival was dramatically worse with more severe strokes: hazard ratio of 2.9 (95% CI, 2.3–3.5) for strokes resulting in major deficits and hazard ratio of 8.3 (95% CI, 5.2–13.2) for strokes resulting in severe deficits compared with matched comparators without stroke.

The high 30-day mortality of strokes because of AF is well known; however, this study is one of the first to show that increased mortality persists long after 30 days. This information is helpful for decision making regarding the risks and benefits of anticoagulant therapy for stroke prevention among individuals with AF. Strengths of the study include the large sample size, extended follow-up, and robust study design with >98% of cases matched with nonstroke controls on a multitude of factors. Limitations include lack of clinical details on severity of comorbid conditions, proportion of time adequately anticoagulated, and type of anticoagulants used because these might affect mortality. Furthermore, the majority of subjects in the study were white and limited to northern California. Additional studies are needed to investigate long-term survival after stroke among individuals with AF in more diverse populations, in broader geographic regions.


Numerous epidemiological studies have demonstrated racial disparities in stroke mortality; however, little is known about the factors that contribute to racial disparities. Using a nationally representative sample from the Health and Retirement Study with linked Medicare data from 1991 to 2007, Levine et al conducted a case-crossover study to evaluate the risk of ischemic stroke death and hospitalization after acute infection. In their crossover design, the frequency of infection during the at-risk period (14 days and 30 days) before stroke hospitalization was compared with the frequency of infection during 4 corresponding comparison periods within the same person, each period being 14 days with a 30-day washout.

Nine hundred sixty-four adults with ischemic stroke were included. Acute infection increased the 30-day risk of ischemic stroke death by 5.8-fold and ischemic stroke hospitalization by 1.9-fold. The most frequent sites of infection were the genitourinary tract (34.6%), respiratory tract (31.6%), and skin (15.8%). Acute infection increased the short-term risk of stroke similarly across racial groups. However, acute infection disproportionately increased stroke death in blacks (odds ratio, 39.2; 95% CI, 9.3–166.0) than in whites (odds ratio, 4.5; 95% CI, 3.1–6.4) or Hispanics (odds ratio, 5.2; 95% CI, 1.3–20.0), even after adjusting for atrial fibrillation.

The finding of a link between infection and subsequent stroke is consistent with previous studies. This study, however, was the first to report a differential effect of acute infection
on stroke death by race. Furthermore, this was the first large cohort involving ≈1000 subjects to explore precipitants that might explain racial disparity in stroke death. The crossover cohort design minimized unmeasured confounding because each patient served as his or her own control. The study has the inherent limitations of administrative data sets, such as lack of detailed clinical information. *International Classification of Diseases, Ninth Revision* coding for infection, stroke, and death could also introduce ascertainment biases. Future studies may explore why acute infection is a more potent trigger of stroke death in blacks. Potential areas of exploration include genetic susceptibility, environmental factors, and evaluation of immunization as a potential strategy to reduce racial disparities in stroke death.
The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://stroke.ahajournals.org/content/45/7/e123