Stroke Mortality

- Is Mechanism a Biological Variable?: A Secondary Analysis of the PROPPR Trial
- Ezetimibe plus statin combination versus double-dose statin in patients with dyslipidemia and atherosclerotic cardiovascular disease risk: a comprehensive systematic review and meta-analysis of 47 randomized controlled trials
- Brain-Body Interactions in Ischemic Stroke: VNS Reprograms Microglia and FNS Enhances Cerebellar Neuroprotection
- Neutrophil Mobilization Triggers Microglial Functional Change to Exacerbate Cerebral Ischemia-Reperfusion Injury
- Analysis of outcomes in patients with HeartMate 3 with and without right ventricular assist device support
- Establishment of ABEI-based direct chemiluminescence immunoassays for PIC, TAT, tPAIC, and TM and their preliminary evaluation in thrombotic diseases
- Comparison of Mortality and Morbidity of Robotic Versus Laparoscopic Radical Nephrectomy for the Treatment of Renal Cell Carcinoma-An Analysis of the National Surgery Quality Improvement Program (NSQIP) Targeted Nephrectomy Database
- Predictors of Major Adverse Cardiovascular Events in Stable Patients After ST Elevation Myocardial Infarction

Stroke mortality refers to the incidence of death directly attributable to a stroke, including both ischemic stroke and hemorrhagic stroke subtypes.

It is typically measured as:

A proportion of deaths within a given population or cohort,

Or as a rate per 100,000 persons per year in epidemiological studies.

 In Clinical and Epidemiological Contexts

Stroke mortality reflects both stroke incidence and case fatality.

It is influenced by:

Age and comorbidities

Stroke type and severity

Access to acute care/intervention

Post-stroke complications

Preventive strategies (e.g., anticoagulation, antihypertensives)

 Measurement in Studies

May be reported as:

Crude mortality rate

Standardized mortality ratio (SMR)

Hazard ratio (HR) for death

In registry-based studies (e.g., SEER), stroke mortality is often derived from death certificate data, which can be imprecise.

⚠ Limitations

Stroke mortality data can be misclassified, especially in patients with multiple comorbidities (e.g., cancer), where the true cause of death is uncertain or multifactorial.

In a retrospective cohort study published in the Journal of Clinical Neuroscience, Ahmed et al. [1] analyzed data from over 5.9 million patients diagnosed with a first primary cancer, based on the SEER database (2000–2020). The study aimed to quantify the risk of stroke-related death (SD) in cancer patients and to identify temporal trends and associated clinical and demographic risk factors. Stroke-related mortality (SD) among cancer patients has significantly declined over the past two decades across all cancer types and both sexes. However, older age, non-white race, male sex, and specific cancer types—notably nervous system, respiratory, and head and neck cancers—are associated with a higher risk of stroke death. Conversely, patients receiving chemotherapy or radiotherapy had a lower risk of SD compared to those who received no treatment.

⚠ Fatal Methodological Flaws

No Clinical Stroke Classification

The authors report on “stroke mortality” without differentiating ischemic vs. hemorrhagic strokes, nor providing stroke etiology or timing relative to cancer diagnosis or cancer treatment—rendering any mechanistic or preventative inference purely speculative.

Reliance on Registry Data Without Validation

The study is built on SEER registry death certificates. These are known to be notoriously unreliable in classifying cause of death in complex patients, particularly in cancer, where the line between terminal illness and stroke is often blurred or misclassified.

Missing Core Clinical Variables

Absolutely no data on cardiovascular comorbidities (e.g., hypertension, atrial fibrillation), medications (e.g., anticoagulants, steroids), or functional status. Without these, attributing causality or understanding modifiable risks is scientifically irresponsible.

“No Treatment” Category is a Black Box

The study repeatedly highlights increased stroke mortality in patients receiving “no treatment”, but never interrogates why. Were they terminal? Frail? Untreated by choice? Refusing care? Without this, comparisons to treated groups are invalid.

Statistical Smoke Without Clinical Fire

The use of large numbers and Annual Percentage Change/SMR modeling creates an illusion of depth.

But in the absence of clinical granularity, the findings are epidemiologically flashy and clinically empty.

Cancer Type Associations Are Tautological

Finding increased SD risk in patients with nervous system tumors is hardly surprising, given the direct anatomical involvement. Presenting this as a novel association lacks critical insight and borders on disingenuous.

Temporal Trends Mask Structural Changes

Declines in stroke mortality are presented as a success story—but the study fails to account for changes in diagnostic criteria, coding practices, cancer treatments, and palliative care protocols over 20 years. These are not stable baselines.

 Conclusion: Big Data, Small Insight

This paper is a perfect example of data-driven illusion: a study that rides the wave of big epidemiological numbers without offering a single actionable or mechanistically sound conclusion. It identifies “associations” that are either already known, clinically irrelevant, or artefacts of poor data. The supposed protective effect of chemotherapy/radiotherapy is unadjusted for prognosis, functional status, or therapeutic intent—rendering the headline finding misleading at best.

⚠ If the purpose of research is to inform practice, this study falls spectacularly short. It tells us what we already suspect, adds confusion where clarity is needed, and fails to bridge data with clinical reality.

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Ahmed YB, Nan Feng AS, Alrawashdeh M, Ellaithy A, Khanduja S, AlBarakat MM, Alshwayyat S, Uchino K, Gusdon AM, Cho SM. Temporal trends and risk factors associated with stroke mortality among cancer patients. J Clin Neurosci. 2025 Jun;136:111249. doi: 10.1016/j.jocn.2025.111249. Epub 2025 Apr 18. Erratum in: J Clin Neurosci. 2025 Jun 17:111381. doi: 10.1016/j.jocn.2025.111381. PMID: 40252475.