Stroke

ORIGINAL CONTRIBUTION

Incidence and Mortality of Chronic Subdural Hematomas: A Population-Based Study

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BACKGROUND: Despite a rising public health burden, there have been few population-based studies of chronic subdural hematomas (cSDH) in the United States. We provide the first estimates of cSDH incidence and mortality in a large, representative U.S. population.

METHODS: In a representative 5-county region of Southern Ohio and Northern Kentucky, all adults with cSDH in 2019 and 2020 were identified and adjudicated by study physicians. Incidence rates were estimated and standardized to the US population based on age, sex, and race; 30-day and 1-year mortality rates were also estimated. The cause of death was determined using the National Death Index.

RESULTS: A total of 353 patients with cSDH were identified. The median age was 76 (IQR, 65–85), 231 were men (65.4%), and 78 were Black (22.1%). Clinical frailty was prevalent among patients (the median retrospective score on the clinical frailty scale was 4), and only 128 (36.3%) were functionally unimpaired at baseline. The regional incidence rate was 16.3 cases/100 000 persons/y (95% CI, 13.9–19.0). Incidence was age- and sex-dependent, with men 85 and older having an incidence rate of 354.8 cases/100 000 persons/y (95% CI, 242.7–500.9). When adjusted to national demographics, the estimated overall US incidence rate was 17.3 cases/100 000 persons/y (95% CI, 14.7–19.9). The 30-day mortality rate after cSDH was 9.4% (95% CI, 6.5–12.9), and the 1-year mortality rate was 32.9% (28.0–38.0). Early mortality (≤30 days) was often partly or fully attributed to the cSDH (48.4% versus 16.1%; P=0.0004), whereas the most common causes of later mortality were neurodegenerative and cardiovascular diseases (27.2% and 28.4%, respectively).

CONCLUSIONS: Our contemporary population-level data show that cSDH is common in the US and primarily afflicts patients with a high degree of functional impairment and frailty. While short-term mortality is low, longer-term mortality is high and often related to comorbid illnesses.

GRAPHIC ABSTRACT: A graphic abstract is available for this article.

Key Words: cardiovascular diseases ■ cause of death ■ epilepsy ■ frailty ■ hemorrhagic stroke

In the setting of an aging population and widespread antithrombotic use, chronic subdural hematomas (cSDH) are increasingly common.[1,2] The public health effects of this rising incidence are considerable, as these patients are at a high risk of long-term cognitive impairment, epilepsy, and death.[3–5] Population-level study of cSDH incidence and outcome has been limited partly due to the challenges in studying the disease at a sufficient scale; administrative *International Classification of Diseases* codes do not reliably distinguish between acute SDHs (aSDH) and cSDHs, necessitating time-consuming manual review.[6] Distinguishing cSDHs is critical because they have a unique pathophysiology,[7] with different management paradigms[8] and outcomes.[9] Prior work has even suggested that many cSDHs develop without a preceding

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Nonstandard Abbreviations and Acronyms

aSDH	acute subdural hematoma
cSDH	chronic subdural hematoma
mRS	modified Rankin Scale
SDH	subdural hematoma

aSDH,[10] and relatively few patients with aSDH go on to develop symptomatic cSDHs.[11]

A better population-level study of cSDH would help clarify the risk factors and ideal medical management of the disease; it would also be helpful for policy planning as new treatments like middle meningeal artery embolization are implemented.[12,13] Unfortunately, most population-level studies of cSDH burden were conducted either in Europe[1,14] or in small, nonrepresentative populations in the United States.[2,15] As a result, the full burden of this disease and how it is distributed across the population is uncertain.

We sought to better characterize the burden of cSDH by determining its incidence, mortality, and the most common associated causes of death in the Greater Cincinnati Northern Kentucky Region. The population of the Greater Cincinnati Northern Kentucky region is representative of the United States and has been the source of national estimates of ischemic and hemorrhagic stroke burden for >30 years through an embedded epidemiological study.[16,17]

METHODS

This study was approved by institutional review boards at all participating hospitals with a waiver from informed consent, and follows the Strengthening the Reporting of Observational Studies in Epidemiology guidelines.[18] Because of the sensitive nature of the data collected for this study, requests to access the data set from qualified researchers trained in human subject confidentiality protocols may be sent to the corresponding author. Only anonymized data will be provided.

Population

The Greater Cincinnati Northern Kentucky region includes 5 counties that abut the Ohio river; the population is 1.4 million persons and is representative of the United States in terms of age, educational attainment, percentage of Black race, and income.[19] Our study region is essentially biracial, with White and Black individuals accounting for >98% of all stroke/SDH events, and thus only White and Black individuals (including those with and without Hispanic ethnicity) were considered for this analysis. cSDHs in Asians and other racial groups were not included for this analysis due to very low numbers in our population, making estimates unreliable.

Identification of cSDH Cases

This study methodology was adapted from and supported by the methodology of the Greater Cincinnati Northern Kentucky Stroke Study, a population-based study of stroke that has been ongoing for 30 years and has been validated extensively.[16] In the study region, there are 5 health systems with a total of 17 different hospitals/emergency departments that provide care to the entire population. Prior studies have shown that all patients in the region receive care at one of these hospitals.[16] There are 3 separate neurosurgery groups that cover these hospitals, with 1 being academic and the other 2 private practice. Discharge logs for all hospitals were screened for hospitalizations with SDH-related *International Classification of Diseases, Tenth Revision* codes (both traumatic and nontraumatic) in any diagnosis position in 2019 and 2020 (I62, S06). To ensure full ascertainment of cases, we used multiple overlapping methods of capture-recapture across institutions along with cold pursuit. All potential SDH-related hospitalizations were reviewed by a trained research nurse, and clinical data were extracted into a standardized case report form. We ensured cases were only counted once by following patients across interhospital transfers. For each patient, extensive data were abstracted. Retrospective estimates were made for the clinical frailty scale,[20] which is an ordinal scale ranging from 1 to 9, with higher scores suggesting a greater degree of frailty. Frailty is conceptualized as variability in vulnerability to adverse health outcomes, including after surgery.[21] We also assessed the premorbid modified Rankin Scale (mRS), a measure of functional status ranging from 0 (completely asymptomatic) to 5 (completely dependent). As a measure of the severity of symptoms related to the cSDH, we abstracted the Markwalder score, which ranges from 0 (no symptoms) to 4 (comatose with absent motor responses to painful stimuli).[22] Clinical data and imaging were then reviewed by a board-certified neurologist to confirm the presence of a cSDH. cSDHs were defined based on imaging characteristics, and included isodense, hypodense, and mixed density subtypes. This approach has good interrater reliability[23] and is consistent with the definition used in recent trials of MMA embolization.[12,13] Since acute subdural hygromas are common after high velocity trauma and can mimic the radiological characteristics of cSDH,[24] we excluded patients who presented acutely after severe high velocity trauma (eg, falls from >3 feet, motor vehicle collisions, assaults). Otherwise, all patients with a cSDH on imaging (including those with mild trauma, defined as a fall <3 feet or equivalent) were included. Patients with a hyperdense SDH were considered to have an aSDH and were thus generally excluded; however, those who were discharged and then returned to the hospital or emergency department with a symptomatic hypodense/isodense/mixed density SDH were also counted as cases. Patients with a remote history of high velocity trauma (>2 weeks in the past) who presented to the hospital with a new or evolving cSDH were also included as cases.

To determine whether our approach missed cSDH cases that were managed exclusively in the outpatient setting, we sampled 2 neurosurgery clinics in the region during 2020, 1 academic and 1 private practice. We reviewed every case with an SDH code presenting for any ambulatory visit to either clinic. This approach identified only 1 additional cSDH case, and this patient was entirely asymptomatic. Based on this analysis, pure outpatient cSDHs were excluded from this analysis.

Outcomes

Thirty-day and 1-year mortality were determined for all patients using the National Death Index. Since all causes of death were included in these estimates, they are best thought of as mortality

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rates rather than case-fatality rates. The National Death Index captures all deaths that occur in the territorial United States, including those that occur out of the hospital. All causes of death were included. For analysis of causes of death, we used death certificate data obtained from the National Death Index. The primary analysis used the Underlying Cause of Death, which is the primary cause extracted from the death certificate and is categorized with *International Classification of Diseases, Tenth Revision* codes. We grouped *International Classification of Diseases, Tenth Revision* codes similarly to prior work,[25] with the exception that SDH codes were considered their own category for this analysis (Table S1). As a second analysis, we specifically looked at how often an SDH-related code was present in any position on the death certificate, including as a contributory or related cause.

Statistical Analysis

Incidence rates were estimated for the overall population, and then by sex for different age subgroups. Only cases occurring within the calendar year 2020 were used to estimate the annual incidence rates. For each subgroup, an estimate of the incidence rate, estimates was calculated by using the raw frequency of cSDH cases as the numerator and the US census data for the 5-county area as the denominator. The exact 95% CIs for the incidence rates were calculated assuming a Poisson distribution. To generate estimates of the national incidence rates, we used direct standardization to match the demographic proportions of age, sex, and race in the overall population of the United States based on 2020 census data.[26] To standardize our rates to the United States, the age, race, and sex specific rates from our study are multiplied by the specific proportions in the reference adjusting population (in this case, the United States 2020 census population); the overall rate is then obtained by summing over all age, race, and sex groups. The SAS code used for this adjustment is available on the web (https://www.lexjansen.com/mwsug/2005/Pharmaceutical_Healthcare/PH600.pdf). This national incidence rate estimate was then multiplied by the total number of persons over the age of 20 (249 254 438) to estimate the total number of adults with cSDH-related hospitalizations across the United States in 2020.[27] For mortality rates, all cases in 2019 and 2020 were used for estimates. Estimates overall and by age group were calculated by using the number of patients who died (regardless of cause) as the numerator and the number of patients with cSDH as the denominator, along with exact 95% CI assuming a binomial distribution. To determine whether the causes of death were significantly different between those who died early (30 days or less) and late (31 days to 1 year), we initially used an Omnibus Fisher exact test. Pairwise comparisons were then conducted using either a χ^2 or Fisher exact test, depending on event numbers.

RESULTS

Demographics and Clinical Characteristics

Across 5 health systems and 19 hospitals, there were 3655 SDH-related hospital encounters in 2019 and 2020. 1982 of these visits were excluded because they represented duplicates (eg, between-hospital transfers, readmissions for the same SDH) or old/incorrect SDH-related codes, and 508 were excluded due to acute high velocity trauma or recent unrelated cranial neurosurgery. An additional 193 were excluded for other reasons (Figure 1). This left 972 patients with a suspected incident SDH during the study period who underwent full physician review. Among these patients, 312 patients initially presented with a cSDH as their index event, and 41 (11.5%) developed a symptomatic cSDH after initially being diagnosed with an aSDH, for a total of 353 patients with incident cSDHs.

Demographics and clinical characteristics of patients with cSDH are shown in Table 1. The median age was 76 (IQR, 65–85), and most patients were male (65.4%). Among patients with cSDH, there was a high prevalence of premorbid functional impairment and frailty. Only 128 patients (36.3%) were free from functional impairment at baseline (mRS scores of 0 or 1), and the median score on the clinical frailty scale was 4 (IQR, 3–5), which is considered vulnerable or very mildly frail. More than half of patients with cSDH took at least 1 antithrombotic before their incident cSDH (54.7%), most commonly an antiplatelet (43.9%). Most patients had a history of trauma (68%), typically a fall from <3 feet (64.3%). Eighty-seven patients (24.7%) were managed surgically during their hospitalization.

Incidence Rates

The incidence rate for cSDHs in 2020 was 16.3 cases/100 000 persons/y (95% CI, 13.9–19) in the Greater Cincinnati Northern Kentucky region. The incidence rate was higher in men when compared with women (22.7 cases/100 000 persons/y versus 10.4 cases/100 000 persons/y), and this was consistent across all age strata (Table 2). In both men and women, the incidence rates for cSDH were dramatically higher in older individuals. Among those 85 and older, the incidence rate was 354.8 cases/100 000 persons/y (242.7–500.9) in men and 115.3 cases/100 000 persons/y (70.4–178.1) in women.

Adjusted to the national population, the estimated incidence rate in the United States is 17.3 cases/100 000 persons/y (95% CI, 14.7–19.9), suggesting that ≈43 121 adults are hospitalized with cSDHs annually in the United States.

Mortality Rates and Causes of Death

The 30-day mortality rate after cSDH was 9.4% (95% CI, 6.5–12.9), and the 1-year mortality rate was 32.9% (28.0–38.0). The mortality rate was higher in older age groups, particularly at 1 year (Table 3).

We then looked at the primary causes of death among those who died early (0–30 days after diagnosis) versus

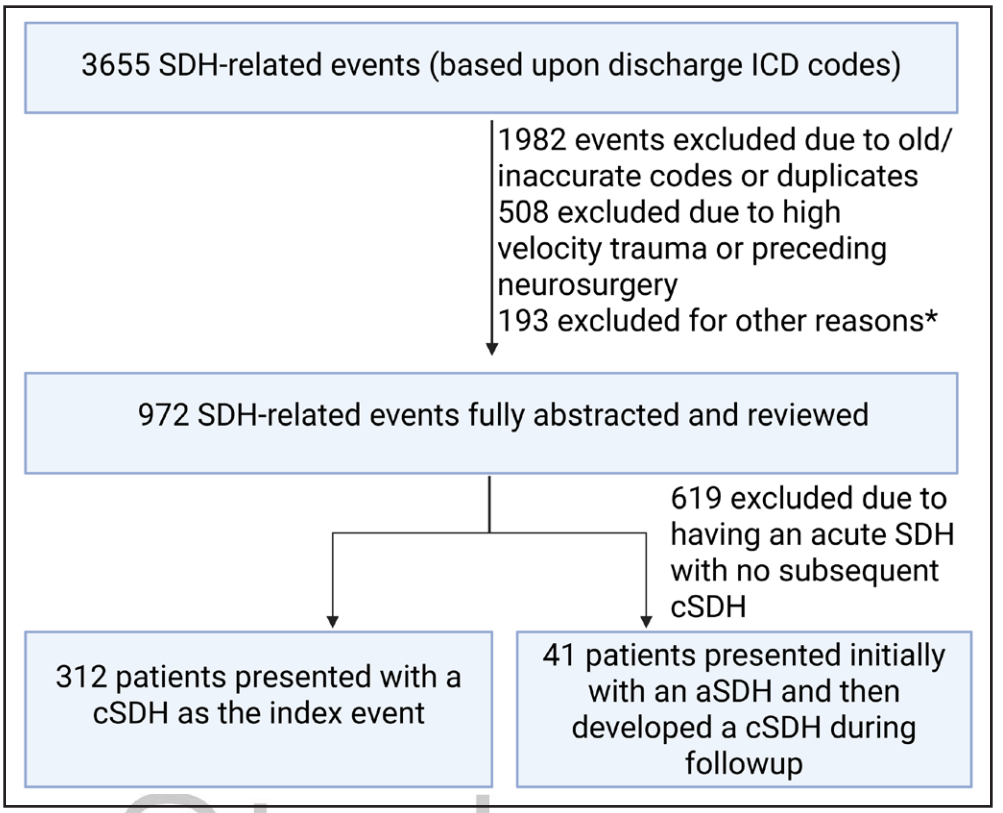

Figure 1. Flow diagram of case identification.
*Most common other reasons include that the patient did not actually reside in the study region on closer review, the event occurred outside of the study period, or that no subdural hematoma (SDH) was visualized on imaging. aSDH indicates acute subdural hematoma; and cSDH, chronic subdural hematoma.

those who died later (between 31 days and 1 year after diagnosis). Data on the cause of death were available in 112/116 deaths (96.6%). The causes of death were significantly different between the 2 time points (*P*=0.006; Figure 2), with cardiovascular disease, neurodegenerative disease, and respiratory illnesses more commonly causing late mortality, although only neurodegenerative disease reached statistical significance (pairwise, *P*=0.047). Early mortality was more commonly caused by an underlying malignancy, the SDH itself, or other causes, with other causes reaching statistical significance (*P*=0.002).

On review of the underlying diagnoses in the other causes category, almost all were accounted for by trauma (7/13, 54%) or cirrhosis (4/13, 31%) in the early period, whereas in the later period, these were most commonly kidney disease (4/12, 33.3%) or infections (3/12, 25%). Since a cSDH could be a secondary effect from cirrhosis and trauma and contribute towards mortality, we then compared the frequency of an SDH-related code anywhere on the death certificate between those who died early and those who died late. An SDH code was present somewhere on the death certificate in nearly half of all patients with early mortality (15/31 or 48.4%), and much less commonly present among those who died later (13/81, 16.1%; *P*=0.0004).

DISCUSSION

We present recent estimates of the incidence and mortality rates of cSDHs in a representative population of the United States. Our work builds on prior studies suggesting a rising incidence rate of cSDH in the United States over the past 3 decades, with our estimate of 17.3 cases/100 000 persons/y at the high end of historical projections for 2020.[2,28] Our data would suggest that in the current era, the burden of incident cSDH is in between the nontraumatic intracranial hemorrhages, more common than subarachnoid hemorrhage (≈9 cases/100 000 persons/y) but less common than intracerebral hemorrhage (≈29 cases/100 000 persons/y).[29,30] Given the profound age-dependence of this disease, its absolute burden will continue to rise as the United States population ages; our results thus highlight the importance of further research into cSDH prevention and treatment.

Our estimate of cSDH incidence rate is slightly lower than that reported in some other developed countries, particularly Japan (20.6/100 000).[14,31] Although methodological differences could explain some of this variation, the profound age-dependence of cSDH incidence likely also plays a role, with the median age of Japan being substantially older than the United States. Recent work in the US used several case definitions to estimate a

Table 1. Population Demographics and Clinical Characteristics of Patients With Chronic Subdural Hematoma (N=353)

Age, median (IQR)	76 (65–85)
Male sex, N (%)	231 (65.4)
Black race, N (%)	78 (22.1%)
Hispanic ethnicity, N (%)	4 (1.13%)
Hypertension, N (%)	279 (79.0%)
Diabetes, N (%)	114 (32.3%)
Hyperlipidemia, N (%)	234 (66.3%)
Dementia, N (%)	66 (19%)
Alcohol abuse, N (%)	34 (9.6%)
Chronic kidney disease, N (%)	69 (19.6%)
End-stage renal disease, N (%)	11 (3.1%)
Premorbid modified Rankin Scale of 0 or 1, N (%)	128 (36.3%)
Clinical frailty score, median (IQR)	4 (3–5)
Markwalder score on admission, median (IQR)	2 (0–2)
Antithrombotic use, N (%)	193 (54.7%)
Anticoagulant use	58 (16.4%)
Antiplatelet use	155 (43.9%)
Antiplatelet and anticoagulant use	20 (5.7%)
Surgically managed, N (%)	87 (24.7%)
Preceding acute subdural hematoma, N (%)	41 (11.6%)
Radiological characteristics on admission, N (%)	
Isodense	72 (20.4%)
Mixed density	139 (39.4%)
Hypodense	142 (40.2%)
SDH thickness ≥10 mm	231 (65.4%)
History of any trauma	240 (68.0%)
Minor trauma	227 (64.3%)
Remote major trauma*	31 (8.8%)

IQR indicates interquartile range; and SDH, subdural hematoma.

*Includes 13 patients with only a history of remote major trauma and 18 patients with both a recent minor trauma and remote major trauma.

Table 2. Incidence Rate of Chronic Subdural Hematomas in 2020, by Sex and Age

Age group	Males		Females	
	Number of cases	Incidence rate (cases/100 000 persons/y)	Number of cases	Incidence rate (cases/100 000 persons/y)
Overall (20 and above)	112	22.7 (18.1–26.6)	56	10.4 (7.8–13.5)
20–54	6	2.0 (0.7–4.3)	3	1.0 (0.2–2.8)
55–64	27	30.1 (19.8–43.8)	6	6.2 (2.3–13.4)
65–74	18	28.2 (16.7–44.5)	6	8.2 (3–17.8)
75–84	29	107.5 (72–154.3)	21	57.6 (35.6–88)
85+	32	354.8 (242.7–500.9)	20	115.3 (70.4–178.1)

range of potential incidence rates from 17 to 42 cSDHs cases/100 000 persons/y. Our estimate falls near the low end of this range, likely reflecting our strict case definition combined with differences in the respective study populations. Specifically, this prior work focused on a region of West Virginia, where the average age is higher than in the rest of the United States.[15] Meanwhile, our estimate is consistent with projections from work in the VA health care system.[2] Continued surveillance of this disease will be needed to fully understand its burden and how it is distributed.

Short-term case fatality rates in our population were higher than prior estimates, which were typically in the 3% to 4% range.[32,33] Prior estimates were not truly population-based and tended to favor surgically managed patients with cSDH, which may be biased towards younger and healthier patients. Our estimates of 1-year mortality, on the other hand, are largely concordant with historical estimates in the United States, which ranged from 30% to 32%.[3,34] Of note, these prior studies included patients with cSDH managed as far back as the 1990s. Although this could suggest that long-term survival after cSDH has not meaningfully changed over that span, caution is warranted when comparing rates since these prior studies were not population-based. Nevertheless, no recent cSDH trials have shown any interventions that improve long-term survival,[12,13,35] and our data emphasize that this is a vulnerable patient population in need of better evidence-based care.

When examining causes of death, we found significant differences between those who died early and late. Among those who died early, malignancy and other causes (typically a trauma or cirrhosis) were the most common, with the cSDH being the underlying cause in only 10%. Trauma is a known factor in the development of cSDHs,[7] while malignancy and cirrhosis are known to predispose to cSDHs through their effects on coagulopathy[36,37]; it is thus likely that these diseases caused death in part through a cSDH. Indeed, we found that almost half of all patients with early mortality had an SDH listed somewhere on their death certificate. Meanwhile, those who died later were much more likely to die from cardiovascular or neurodegenerative diseases/dementia, with the SDH rarely being contributory. Improving long-term outcomes in this population will thus require systems of care to adequately manage these comorbid chronic illnesses as patients recover from cSDHs. In particular, better guidance on managing antithrombotics is needed, with recent trials suggesting the safety of earlier aspirin resumption among surgically managed patients with cSDH[38]; future trials will need to examine when other antithrombotics (eg, anticoagulation) can be safely resumed and should include medically managed patients as well. More research is also needed to explore whether cSDHs can exacerbate an underlying neurodegenerative disease by causing progressive atrophy,[39] and whether this effect can be mitigated through existing or novel treatment paradigms.

Table 3. Thirty-Day and 1-Year Mortality Rate by Age, Including Both 2019 and 2020 Cases

Age group	30-Days		1-Year	
	Number deceased	Mortality rate, % (95% CI)	Number deceased	Mortality rate, % (95% CI)
Overall (N=353)	33	9.4 (6.5–12.9)	116	32.9 (28.0–38.0)
20–54 (N=23)	0	0	5	21.7 (7.5–43.7)
55–64 (N=63)	6	9.5 (3.6–19.6)	13	20.6 (11.5–32.7)
65–74 (N=74)	3	4.1 (0.8–11.4)	18	24.3 (15.1–35.7)
75–84 (N=97)	12	12.4 (6.6–20.6)	32	33.0 (23.8–43.3)
85+(N=96)	12	12.5 (6.6–20.8)	48	50.0 (39.6–60.4)

A key finding of this work is the high degree of functional impairment and clinical frailty among patients with cSDH. Nearly two-thirds of patients with cSDH in our study had some degree of premorbid functional impairment (defined as an mRS score >1), which is nearly double the rate seen in population-based estimates of individuals with ischemic stroke and intracerebral hemorrhage.[25] This needs to be considered in the context of some recent cSDH clinical trials that limited enrollment to those with a premorbid mRS of 0 or 1 only,[12] which would exclude the majority of patients with cSDH at the population level. Pragmatic cSDH trials will thus have to enroll patients with functional impairment at baseline[40] and also consider evaluating more patient-centered outcomes beyond mobility measures, such as the mRS.[41] Our data further suggests that most patients with cSDH are managed nonoperatively, highlighting the importance of further research into the optimal medical management of this disease.

There are significant strengths to our estimates of incidence and outcome, as they were developed using well-validated methodology with a standardized case definition. There are also important limitations to this work. First, we only captured patients with cSDH who had at least 1 interaction with an emergency department or hospital, potentially excluding cSDHs managed exclusively in the outpatient system and artificially lowering the incidence rate; however, we sampled two-thirds of

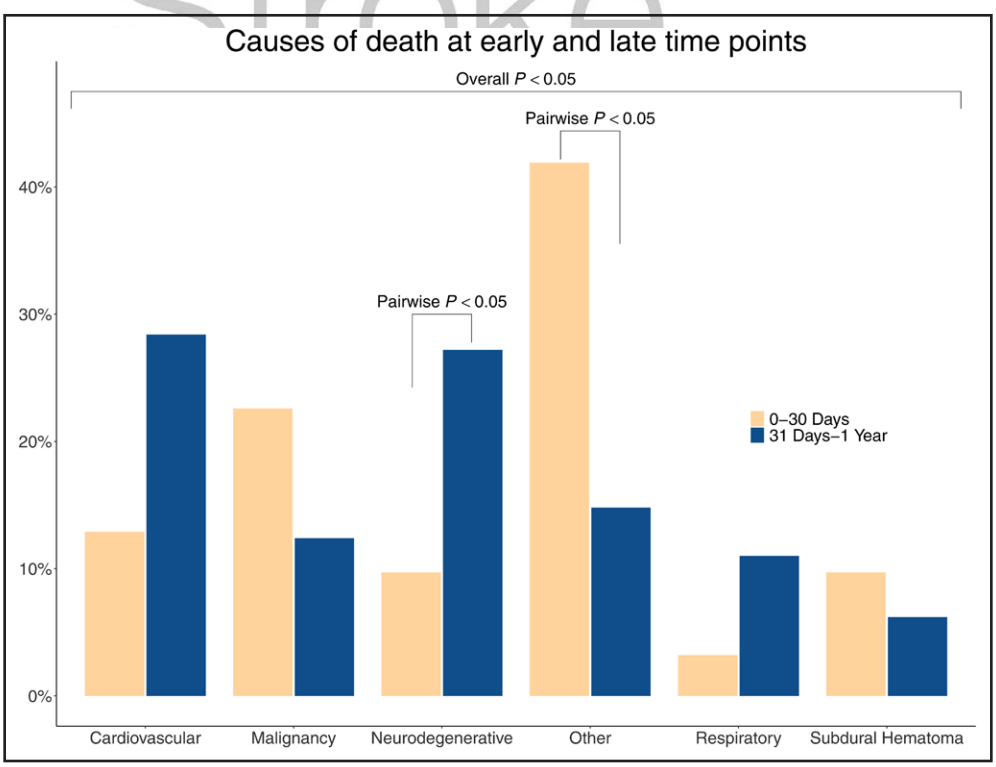

Figure 2. Causes of death at early (0–30 days, burlywood color) and late (31 days to 1 year, navy blue color) time points based on underlying diagnosis on the death certificate.

Cause of death was not available for 4 patients, and this chart reflects percentages of all deaths for N=31 in the early period and N=81 in the late period. Overall *P* value is an omnibus value comparing early vs late causes of death, and significant *P* values for subsequent pairwise comparisons are shown. Regarding other deaths, almost all were accounted for by trauma (7/13) and cirrhosis (4/13) at the early time point. At the late time point, the most common other causes were systemic infections (3/12) and kidney disease (4/12).

the neurosurgery clinics in the region and found only 1 additional potential case, suggesting that few patients with cSDH are managed exclusively outpatient in our region. Second, our study period overlapped with the 2020 COVID-19 pandemic; there were no dramatic differences in case numbers or mortality between 2019 and 2020, so it is unlikely that this meaningfully affected our findings. Third, our estimates may not be generalizable to every region of the United States, though our population is representative in terms of socioeconomic status, age, and percentage of Black race. Fourth, the cause of death was determined using death certificate data in our study, which may overestimate the burden of cardiovascular disease and underestimate the burden of dementia/neurodegenerative diseases.[42,43]

In conclusion, cSDH is common in the United States and frequently afflicts patients with premorbid functional impairment and frailty. Although short-term mortality is low, longer-term mortality is high and often related to comorbid illnesses.

ARTICLE INFORMATION

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Supplemental Material

Checklist
Table S1

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