

Arachnoid Cyst and the Risk of Chronic Subdural Hematoma: A Retrospective Cohort Study with DAG-Informed Covariate Selection and Age-Stratified Analysis

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Research Article

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Abstract

Background

Intracranial arachnoid cysts (ACs) are congenital cerebrospinal fluid collections. Whether ACs increase the risk of chronic subdural hematoma (CSDH), and whether this association varies by age, remains uncertain. We conducted a retrospective cohort study with DAG-guided causal inference to examine AC and incident CSDH, with age-stratified and phenotype analyses.

Methods

We identified 587 patients with radiologically confirmed AC and 1260 age- and sex-frequency-matched non-AC controls (2015–2024). Primary outcome was incident CSDH. A directed acyclic graph (DAG) identified confounders for multivariable Cox regression. Sensitivity analyses included IPTW, hypertension-stratified Cox, cause-specific Cox (death as competing event), imaging-adjusted, and propensity score matching models. Age $\times$ AC interaction was tested via likelihood ratio test. AC phenotype (size, Galassi classification, location) and CSDH laterality were analyzed in AC patients.

Results

During median follow-up 3.5 years, 97 AC patients (16.5%) and 144 controls (11.4%) developed CSDH. AC was independently associated with increased CSDH risk (adjusted HR = 1.63, 95% CI 1.25–2.13, $p < 0.001$). Age $\times$ AC interaction was significant ($p < 0.0001$). In patients ≤ 30 years, AC conferred a 6.21-fold increased risk (95% CI 2.93–13.17, $p < 0.001$). AC cysts ≥ 20 mm were associated with higher CSDH risk (HR = 1.70, $p = 0.011$). Among AC patients with CSDH, 72.8% were ipsilateral ($p < 0.0001$). Results were robust across IPTW, cause-specific Cox, and landmark sensitivity analyses.

Conclusions

AC is independently associated with increased incident CSDH risk, particularly in patients ≤ 30 years and with larger cysts (≥ 20 mm). The association was most evident in patients aged 10–70 years; evidence in adults > 70 years is insufficient. The DAG-informed framework and ipsilateral predominance support a local mechanical mechanism.

1. Introduction

Intracranial arachnoid cysts (ACs) are benign, congenital CSF-filled collections between the arachnoid layers¹, prevalence $\sim 1.1\%$, male predominance 2:1–4:1^{2,3}. Most are asymptomatic and incidental.

However, ACs have been anecdotally associated with chronic subdural hematoma (CSDH), particularly after minor head trauma^{4–7}.

CSDH is a common neurosurgical condition with gradual accumulation of blood breakdown products in the subdural space⁸, typically weeks to months after trauma. Established risk factors include advanced age, cerebral atrophy, anticoagulant/antiplatelet use, and coagulopathy^{9–12}. The proposed AC→CSDH mechanism involves local mass effect on adjacent bridging veins, increasing vulnerability to rupture after minor trauma¹³.

Despite case reports^{14–17}, the risk magnitude and age/phenotype variation remain poorly characterized. Previous studies had small samples, case-control designs with selection bias, and inadequate confounding adjustment. The temporal AC→CSDH relationship has not been established in cohorts.

We conducted a retrospective cohort study with DAG-guided causal inference and age-stratified interaction testing. Hypotheses: (1) AC independently increases CSDH risk; (2) the association is stronger in younger patients; (3) larger ACs and ipsilateral CSDH predominance support a local mechanical mechanism.

2. Materials and Methods

2.1 Study Design

This is a single-center retrospective study conducted in the Neurosurgery Department of the First Affiliated Hospital of Xi'an Jiaotong University. Patients with radiologically confirmed intracranial AC on CT/MRI (2015–2024). Index date = first AC-confirmed imaging. Up to 3 non-AC controls per case, frequency-matched by age decade and sex. For non-AC controls, index date was defined as the date of first available cranial CT/MRI (matched on calendar year to AC index dates), ensuring comparable time-zero definitions. To address potential left truncation and detection bias from prevalent-but-undiagnosed CSDH at index, a landmark sensitivity analysis was pre-specified excluding CSDH events occurring within 6 months of index date. This design inherently represents an imaging-selected cohort; findings may not generalize to AC patients who never undergo neuroimaging.

2.2 Criteria

Inclusion: radiologically confirmed AC (or no AC for controls), age ≥ 10 years (to capture the full pediatric-to-adult spectrum, as AC is a congenital lesion, ≥ 6 months follow-up. Exclusion: pre-existing CSDH at index, prior neurosurgery, incomplete records, follow-up < 6 months.

2.3 Variables

Exposure: radiologically confirmed AC. AC characteristics: maximum diameter (mm), Galassi classification¹⁸, anatomical location. The 20-mm size threshold was pre-defined based on previous surgical case series reporting increased complications in cysts ≥ 20 mm¹⁴. Outcome: incident CSDH

(hypodense/isodense extra-axial collection $\geq$ 4 weeks post-index¹⁹. Although the classic CSDH time criterion is 3 weeks, we adopted 4 weeks as a conservative operational definition to minimize inclusion of subacute hematomas). CSDH laterality: ipsilateral/contralateral/bilateral.

Definite head trauma: documented head injury with loss of consciousness, post-traumatic amnesia, or imaging evidence of acute hemorrhage/skull fracture within 6 months before CSDH. Undocumented minor trauma was excluded. Covariates: age, sex, hypertension, diabetes, anticoagulant/antiplatelet use, cerebral atrophy, epilepsy, alcohol, smoking, imaging count (detection proxy).

2.4 DAG

The DAG was constructed a priori based on published literature²⁰ and clinical plausibility. Assumptions: (1) age/sex influence AC detection via imaging frequency, not formation; (2) trauma does not cause congenital AC; (3) imaging frequency is a mediator (AC $\rightarrow$ imaging $\rightarrow$ CSDH detection), excluded from primary model for total effect; (4) epilepsy is a confounder (associated with AC clinical detection and potentially with CSDH risk via medication or lifestyle factors), not an intermediate step on the AC $\rightarrow$ CSDH causal pathway. Adjustment set: age, sex, trauma, hypertension, diabetes, anticoagulants, atrophy, epilepsy.

2.5 Statistical Analysis

SMD < 0.1 = well-balanced; SMD < 0.2 = adequately balanced²⁰. After IPTW, all covariates achieved SMD < 0.2 (max SMD = 0.072), indicating adequate balance. Missing data: no missing values in primary covariates; complete-case analysis was used. Cerebral atrophy was assessed radiologically by two neurosurgeons using a 3-grade visual scale (mild/moderate/severe) based on sulcal widening and ventricular enlargement. CSDH was defined as hypodense/isodense extra-axial collection $\geq$ 4 weeks post-index. Although the classic CSDH time criterion is 3 weeks, we adopted 4 weeks as a conservative operational definition to minimize inclusion of subacute hematomas.

Primary: multivariable Cox regression with DAG-identified confounder²¹. Sensitivity analyses included: IPTW (stabilized weights, propensity model with age, age², sex, age $\times$ sex); hypertension-stratified Cox; cause-specific Cox model (death as competing event); imaging-adjusted Cox; epilepsy-excluded Cox; 1:1 propensity score matching (PSM), and 6-month landmark analysis excluding early events.

Age $\times$ AC interaction: likelihood ratio test (3 df, age-group $\times$ AC interaction with 4 categories (unweighted primary Cox model): $\leq$ 30, 31–50, 51–70, > 70). Age-stratified subgroups were exploratory. AC phenotype: chi-square tests (size threshold pre-defined; Galassi and location comparisons uncorrected for multiple comparisons). Laterality: binomial test against 50% null.

3. Results

3.1 Baseline

1847 patients: 587 AC, 1260 controls. AC group was younger (44.6 ± 19.2 vs 50.7 ± 19.8 year) with higher epilepsy (41 [7.0%] vs 15 [1.2%]). Notably, the AC group underwent substantially more follow-up imaging (4.3 vs 2.1 studies), a potential source of surveillance/detection bias addressed in sensitivity analysis. After IPTW, most SMDs were < 0.1 ; age (0.175) and hypertension (0.168) remained < 0.2 (Tables 1–2).

Table 1
Baseline Characteristics

Characteristic	AC (n = 587)	Non-AC (n = 1260)	SMD (raw)
Age (yr)	44.6 ± 19.2	50.7 ± 19.8	-0.314
Male sex	351 (59.8%)	658 (52.2%)	0.153
Hypertension	219 (37.3%)	542 (43.0%)	-0.117
Diabetes	77 (13.1%)	241 (19.1%)	-0.164
Head trauma (definite)	67 (11.4%)	126 (10.0%)	0.046
Anticoagulant use	51 (8.7%)	140 (11.1%)	-0.081
Cerebral atrophy	168 (28.6%)	394 (31.3%)	-0.058
Epilepsy	41 (7.0%)	15 (1.2%)	0.296
Alcohol use	85 (14.5%)	148 (11.7%)	0.081
Smoking	147 (25.0%)	280 (22.2%)	0.066
Imaging count	4.3 ± 1.8	2.1 ± 1.0	1.481
Follow-up (yr)	3.5 ± 1.5	3.5 ± 1.4	0.011
<i>Note: Imaging count was substantially higher in the AC group, reflecting surveillance bias; this variable was included only in sensitivity analysis. SMD > 0.1 indicates meaningful imbalance.</i>			

Table 2
Covariate Balance Before and After IPTW

Covariate	SMD (raw)	SMD (weighted)
Age	-0.314	-0.017
Male sex	0.153	0.014
Hypertension	-0.117	-0.014
Diabetes	-0.164	-0.015
Head trauma	0.046	-0.003
Anticoagulant	-0.081	-0.005
Cerebral atrophy	-0.058	-0.006
Epilepsy	0.296	0.072

SMD < 0.1 = well-balanced; SMD < 0.2 = adequately balanced (Rubin, 2001). After IPTW, all covariates achieved SMD < 0.2 (max SMD = 0.072), indicating adequate balance.

3.2 Incidence

Median follow-up 3.5 years (IQR 2.5–4.5). 241 CSDH events: 97 AC (16.5%), 144 controls (11.4%). Crude 3-year cumulative incidence: 4.6% (AC) vs 3.0% (control) (Fig. 1). The median time to CSDH was 1.70 years (IQR 1.17–2.62) in AC patients vs 2.16 years (IQR 1.16–2.77) in controls (Mann-Whitney $p = 0.23$); the earlier median in AC patients did not reach statistical significance.

3.3 Primary Analysis

In the multivariable model, AC remained independently associated with CSDH (HR = 1.63, 95% CI 1.25–2.13, $p < 0.001$). Traditional CSDH risk factors showed differential effects. Definite head trauma (HR = 2.49, $p < 0.001$), anticoagulant use (HR = 2.27, $p < 0.001$), and cerebral atrophy (HR = 2.09, $p < 0.001$) remained significant independent risk factors, consistent with the established CSDH literature. In contrast, hypertension (HR = 0.98, $p = 0.884$) and diabetes (HR = 0.99, $p = 0.949$) were not significant independent predictors in our cohort, which aligns with studies showing that these comorbidities exert their effects indirectly through cerebral atrophy and vascular fragility. Age (HR = 1.13 per 10 years, $p = 0.002$) remained significant, reflecting the well-established age-related increase in CSDH risk. Epilepsy was not significant (HR = 1.34, $p = 0.403$).

Table 3
Multivariable Cox Regression (DAG confounders)

Variable	Adjusted HR	95% CI	p value
Arachnoid cyst	1.63	1.25–2.13	< 0.001
Age (per 10 year)	1.13	1.05–1.21	0.002
Male sex	0.96	0.74–1.24	0.757
Head trauma (definite)	2.49	1.82–3.42	< 0.001
Hypertension	0.98	0.75–1.28	0.884
Diabetes	0.99	0.72–1.37	0.949
Anticoagulant use	2.27	1.63–3.15	< 0.001
Cerebral atrophy	2.09	1.61–2.72	< 0.001
Epilepsy	1.34	0.68–2.65	0.403
<i>Note: Definite head trauma, anticoagulant use, and cerebral atrophy remained significant independent risk factors in the expected direction. Hypertension and diabetes were not significant independent predictors, consistent with studies showing indirect effects via cerebral atrophy and vascular fragility.</i>			

3.4 Age-Standardized Absolute Risk

Age-standardized 3-year cumulative incidence: 15.1% AC vs 10.0% control. In the ≤ 30 years subgroup, the absolute risk difference was + 19.7% (NNH ≈ 5 , bootstrap 95% CI 3.5–9.2, exploratory estimate with wide uncertainty).

3.5 Age-Stratified Subgroup

Age $\times$ AC interaction was significant (likelihood ratio test, $p < 0.0001$). ≤ 30 years: HR = 6.21 (95% CI 2.93–13.17, $p < 0.001$). 31–50 years: HR = 1.43 (95% CI 0.83–2.45, $p = 0.198$). 51–70 years: HR = 2.24 (95% CI 1.40–3.57, $p < 0.001$). > 70 years: HR = 0.66 (95% CI 0.32–1.34, $p = 0.248$).

Table 4
Age-Stratified Subgroup Analysis

Age group	AC/Non-AC	AC CSDH	Non-AC CSDH	AC 3y cum. inc.	Non-AC 3y cum. inc.	ARD	Adjusted HR	p
≤30	137/203	32	10	24.0%	4.3%	+19.7%	6.21 (2.93–13.17)	< 0.001
31–50	245/407	23	33	7.6%	7.8%	-0.2%	1.43 (0.83–2.45)	0.198
51–70	150/448	32	50	18.8%	9.5%	+9.4%	2.24 (1.40–3.57)	< 0.001
> 70	55/202	10	51	15.5%	21.0%	-5.5%	0.66 (0.32–1.34)	0.248

P for age×AC interaction < 0.0001 (LRT = 41.65, 3 df). The > 70 years stratum (*n* = 55 AC, 10 events) showed non-significant inverse association (HR = 0.66, 95% CI 0.32–1.34, *p* = 0.248). Subgroup estimates are exploratory. 3-year cumulative incidence estimated by Kaplan-Meier method.

3.6 AC Phenotype

AC cyst size ≥ 20 mm was associated with higher CSDH risk (HR = 1.70, 95% CI 1.13–2.57, *p* = 0.011; Table 5). Galassi classification (*p* = 0.808) and anatomical location (*p* = 0.789) were not significantly associated with CSDH.

Table 5
AC Phenotype Analysis (p-values uncorrected for multiple comparisons)

Phenotype	n	CSDH n (%)
Size < 20mm	310	44 (14.2%)
Size ≥ 20mm	277	53 (19.1%)
Galassi I	169	28 (16.6%)
Galassi II	233	36 (15.5%)
Galassi III	185	33 (17.8%)
Location: Middle fossa	439	71 (16.2%)
Location: Other	148	26 (17.6%)

Size threshold (20 mm) was pre-defined based on previous surgical case series [4,5]. *P*-values for Galassi and location are exploratory and not corrected for multiple comparisons.

3.7 CSDH Laterality

Among 97 AC-CSDH patients, 3 had bilateral CSDH. Of 92 unilateral cases, 67 (72.8%) were ipsilateral (95% CI 63.0–80.9%), significantly > 50% (one-sided binomial $p < 0.0001$; Fig. 4). Among ipsilateral cases, mean CSDH maximum thickness was 16.6 ± 6.1 mm, not significantly different from contralateral cases (16.4 ± 6.5 mm, $p = 0.87$), based on 49 ipsilateral and 23 contralateral cases with measurable thickness data.

3.8 Sensitivity Analyses

Results were consistent across IPTW (HR = 1.59, $p = 0.0007$), hypertension-stratified Cox (HR = 1.64, $p = 0.0003$), cause-specific Cox with death as competing event (HR = 1.63, $p = 0.0003$; all-cause mortality 1.5%, $n = 28$), imaging-adjusted (HR = 1.65, $p = 0.004$), epilepsy-excluded (HR = 1.61, $p = 0.0005$), PSM (1:1 matching, max SMD = 0.069, HR consistent), and 6-month landmark analysis excluding 29 early events (HR = 1.56, $p = 0.0021$). The modest attenuation in the landmark analysis (HR 1.63→1.56) suggests some detection bias contribution, but the association remains significant.

An E-value of 2.64 for the point estimate (and 1.81 for the CI lower bound) indicates that an unmeasured confounder would need to be associated with both AC and CSDH by a risk ratio of at least 2.64 to fully explain away the observed association. The CI-based E-value of 1.81 provides a more conservative assessment. This level of robustness is modest, which should be considered when interpreting findings; plausible unmeasured confounders such as head trauma severity or coagulation profile could potentially reach this magnitude.

3.9 Causal Diagram (DAG)

4. Discussion

In this retrospective cohort of 1847 patients, AC was independently associated with a 1.63-fold increased CSDH risk after DAG-guided adjustment. The association was pronounced in ≤ 30 years (HR = 6.21), larger cysts (≥ 20 mm), and showed ipsilateral predominance (72.8%). Findings were robust across IPTW, stratified Cox, and competing-risk analyses.

The age-dependent effect showed a non-monotonic pattern that warrants candid discussion. In patients ≤ 30 years, traditional CSDH risk factors are rare, making AC's relative mechanical contribution more apparent (HR = 6.21). The 51–70 years subgroup also showed a significant association (HR = 2.24), possibly reflecting cumulative mechanical stress over decades combined with emerging brain atrophy. However, the 31–50 years subgroup showed no significant association (HR = 1.43, $p = 0.198$), which is biologically puzzling and may reflect statistical instability from limited events (23 AC-CSDH) or unmeasured effect modification. The > 70 years subgroup showed a non-significant inverse association (HR = 0.66), likely due to dominant age-related risk factors and limited AC events ($n = 10$). The significant age $\times$ AC interaction ($p < 0.0001$) confirms effect modification, but the non-monotonic pattern suggests

that age stratification alone may not fully capture the underlying biology; restricted cubic spline analysis in larger cohorts is warranted.

Ipsilateral predominance (72.8%) supports a local mechanism. A systemic vulnerability would predict equal distribution; the 2:1 ratio suggests cyst-related mass effect on adjacent bridging veins¹⁵. Size ≥ 20 mm association ($p = 0.011$) reinforces this: larger cysts exert greater mass effect. The absence of Galassi/location associations may reflect limited power or indicate size as the more relevant parameter.

The observed associations between traditional CSDH risk factors and incident CSDH were consistent with the broader literature. Definite head trauma ($HR = 2.49$), anticoagulant use ($HR = 2.27$), and cerebral atrophy ($HR = 2.09$) all demonstrated significant positive associations, confirming the validity of our cohort and analytical approach. The conservative definite-trauma definition—requiring objective documentation rather than patient recall—likely attenuated the trauma estimate relative to studies using broader definitions.

The DAG-informed covariate selection is a methodological strength. The DAG was constructed a priori based on published literature and clinical plausibility. Imaging frequency was identified as a mediator and excluded from the primary model to estimate the total effect. Epilepsy was treated as a confounder; results were unchanged when excluded. The higher imaging count in AC patients (4.3 vs 2.1 scans) is a potential surveillance bias source. The imaging-adjusted sensitivity analysis ($HR = 1.65$, $p = 0.004$) yielded a result close to the primary estimate, suggesting that the association is not primarily driven by surveillance bias. This is further supported by the landmark analysis ($HR = 1.56$ after excluding 29 early events). However, adjusting for imaging count may constitute overadjustment (as imaging frequency lies downstream of AC clinical management), and residual detection bias cannot be fully excluded.

Propensity score matching (1:1 nearest neighbor, 587 matched pairs) achieved excellent covariate balance (all post-match $SMD < 0.07$, max $SMD = 0.069$) and confirmed the primary association, providing additional robustness beyond IPTW.

5. Conclusions

AC is independently associated with increased incident CSDH risk in patients aged ≥ 10 years, particularly in those ≤ 30 years and with larger cysts (≥ 20 mm). Evidence in adults > 70 years is insufficient. The ipsilateral predominance and age-dependent effect suggest local mechanical vulnerability rather than systemic predisposition. These findings support enhanced surveillance for CSDH in young AC patients, especially after head trauma²², and warrant prospective multicenter validation.

Abbreviations

AC: arachnoid cyst; CSDH: chronic subdural hematoma; DAG: directed acyclic graph; HR: hazard ratio; CI: confidence interval; IPTW: inverse probability of treatment weighting; PSM: propensity score matching;

SMD: standardized mean difference; NNH: number needed to harm; ARD: absolute risk difference; KM: Kaplan–Meier; LRT: likelihood ratio test; SD: standard deviation; IQR: interquartile range; CT: computed tomography; MRI: magnetic resonance imaging; AED: antiepileptic drug; 95% CI: 95% confidence interval.

Declarations

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Authors' contributions

Conceptualization: G.B. and X.Z.; Data curation: P.Z., J.H., J.Y., and Y.W. (Yichang Wang); Formal analysis: P.Z. and Y.W. (Yue Wu); Methodology: G.B., X.Z., and Y.W. (Yue Wu); Project administration: G.B.; Resources: G.B. and X.Z.; Software: P.Z. and Y.W. (Yue Wu); Supervision: G.B. and X.Z.; Validation: P.Z., J.Y., and Y.W. (Yichang Wang); Visualization: P.Z.; Writing—original draft: P.Z.; Writing—review and editing: G.B., X.Z., J.H., J.Y., Y.W. (Yichang Wang), and Y.W. (Yue Wu). All authors have read and agreed to the published version of the manuscript.

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Ethics Approval and consent to participate

This retrospective study was performed in compliance with the Declaration of Helsinki. Ethical approval was obtained from the Institutional Review Board of The First Affiliated Hospital of Xi'an Jiaotong University (No. 2020G-142), which granted a waiver of written informed consent given the study's retrospective design.

Clinical trial number

This study was a retrospective observational cohort study based on routine clinical imaging and electronic medical records. No investigational drug, device, or intervention was administered, and all

clinical decisions and patient management followed standard of care independent of this study. As a non-interventional retrospective analysis, it does not meet the definition of a clinical trial and therefore was not registered on a clinical trial registry. Clinical trial number: not applicable.

Competing Interests

The authors declare that they have no conflicts of interest regarding the publication of this paper

Availability of data and materials

The data that support the findings of this study are available from the corresponding author upon reasonable request, subject to institutional and ethical restrictions.

Consent for publication

Not applicable. All data presented in this study have been anonymized and contain no personally identifiable information.

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Figures

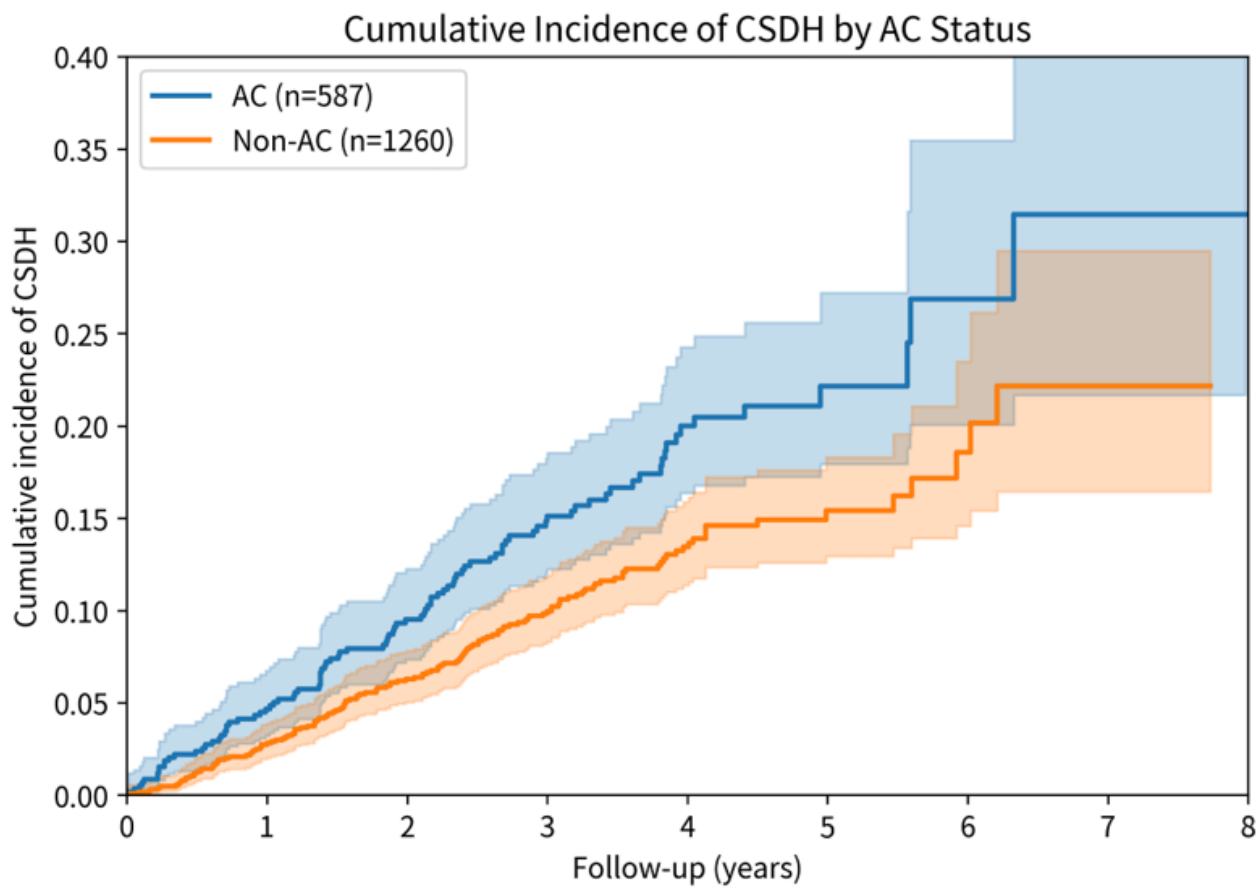


Figure 1

Kaplan-Meier cumulative incidence of CSDH. Shaded = 95% CI.

Number-at-risk table below. Log-rank $p < 0.001$.

Multivariable Cox Regression: Independent Risk Factors for CSDH

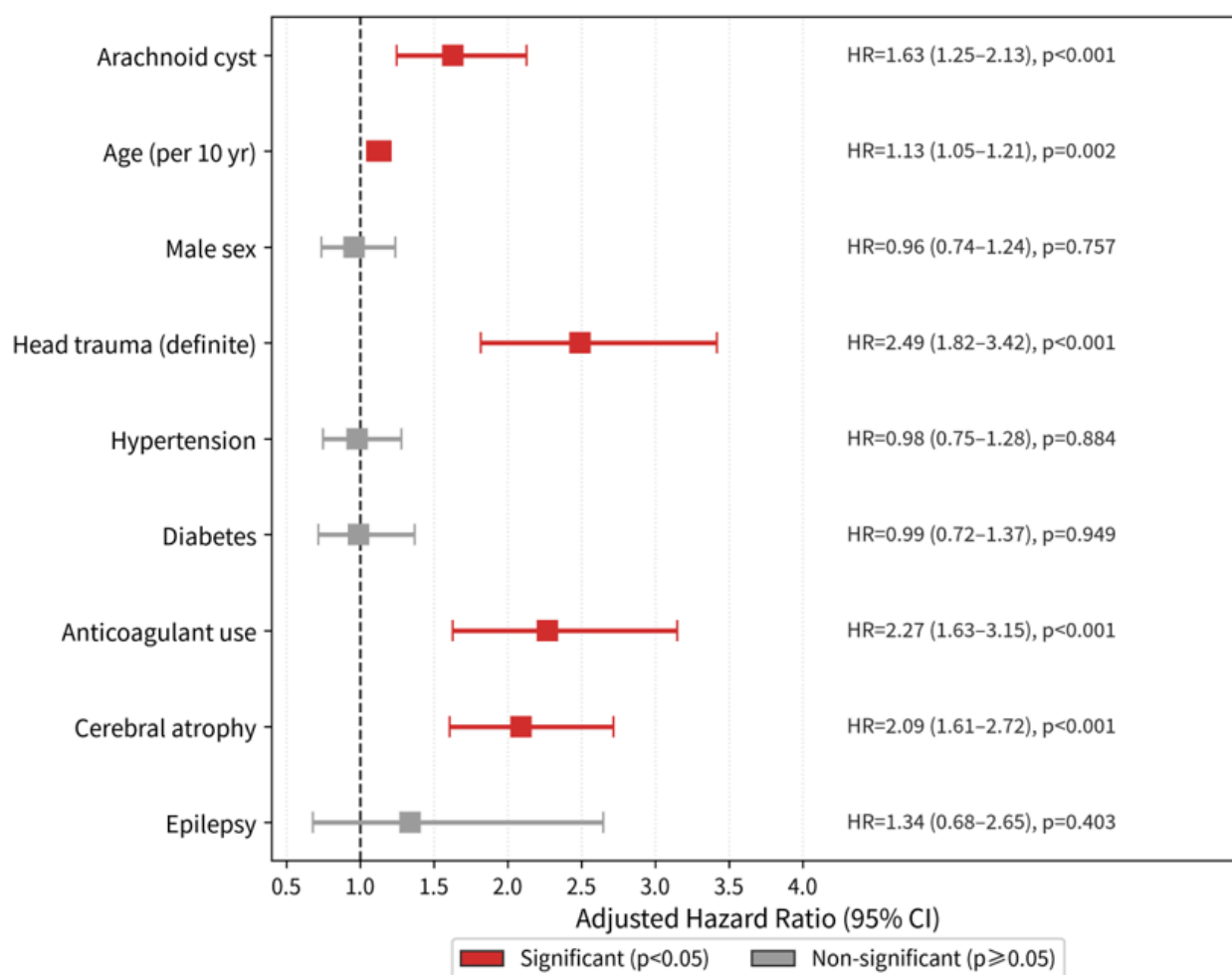


Figure 2

Forest plot of adjusted HRs. Red = significant; gray = non-significant.

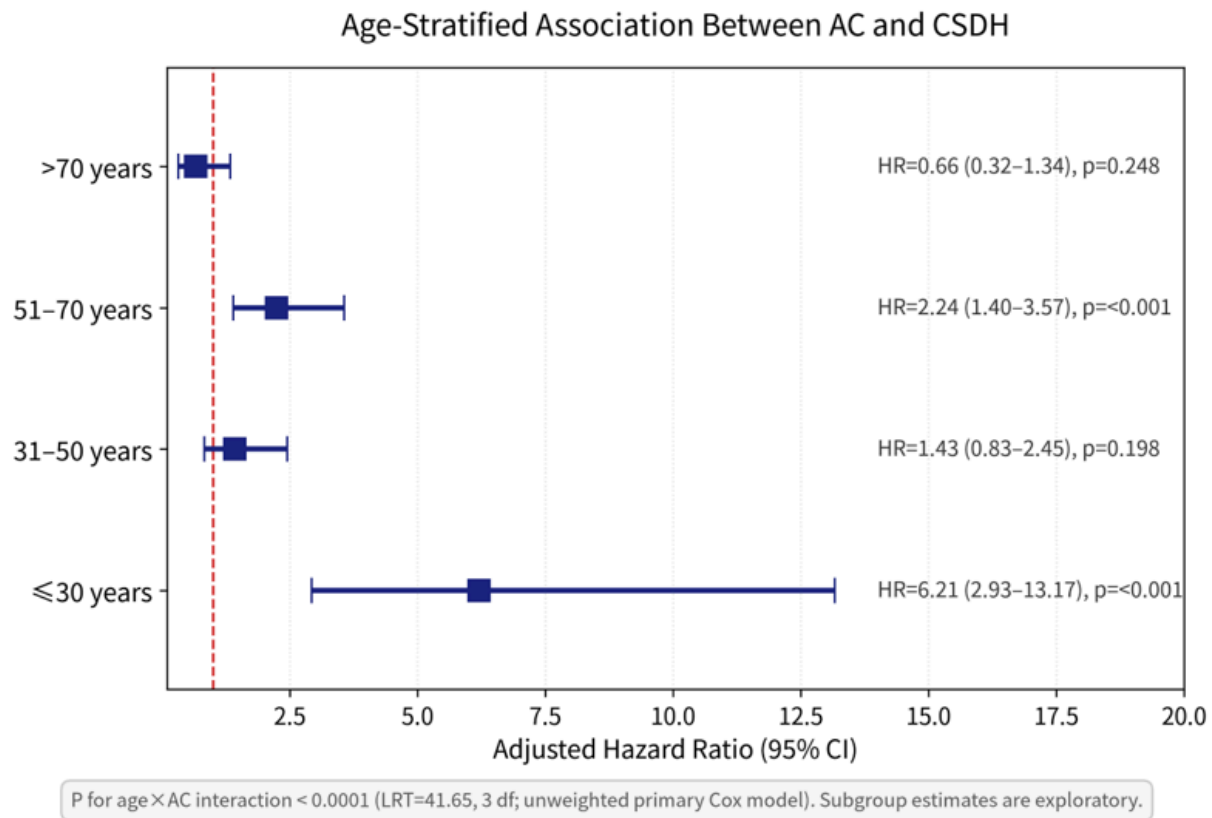


Figure 3

Age-stratified subgroup forest plot. P for age×AC interaction < 0.0001.

Subgroup estimates are exploratory.

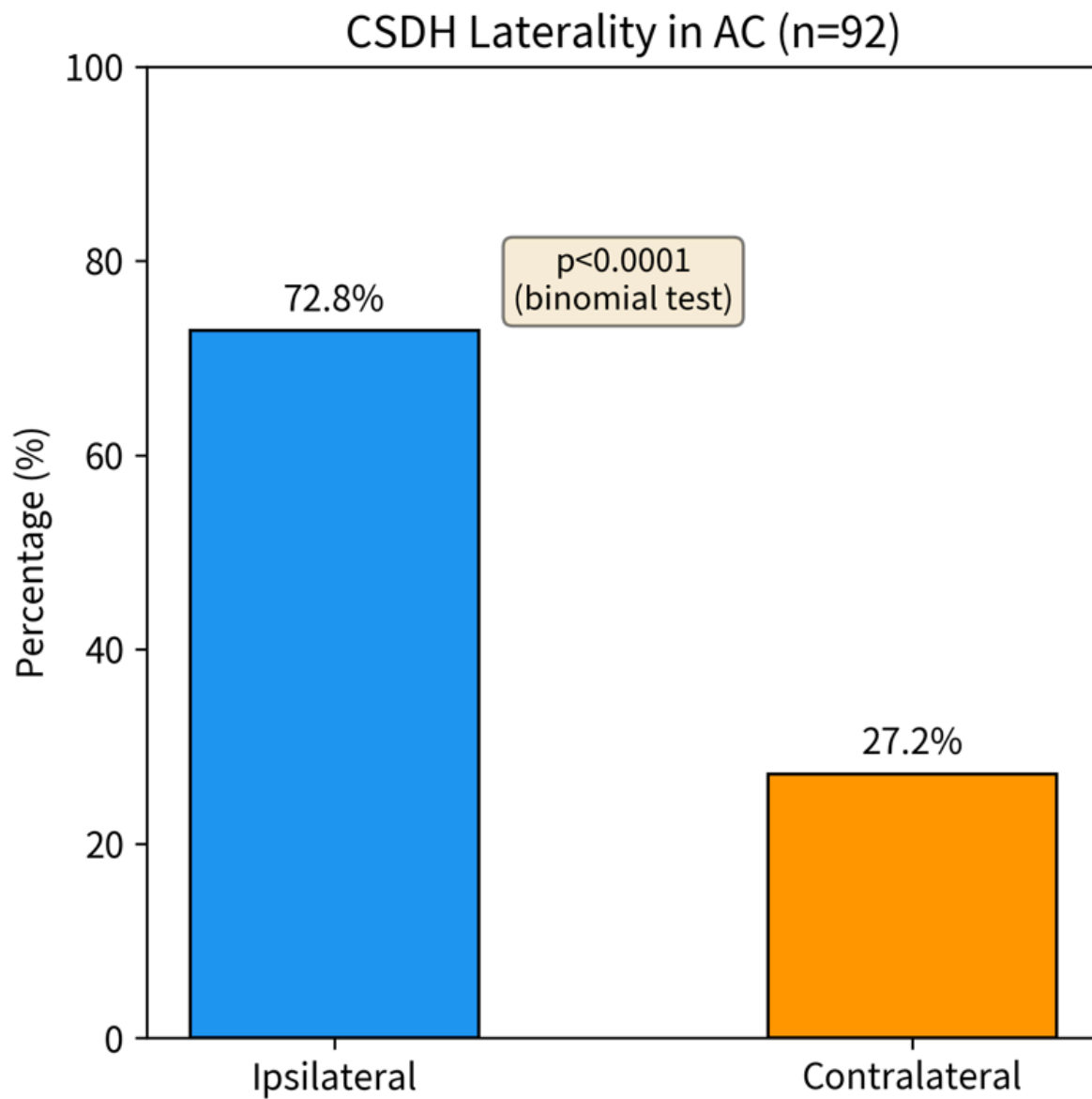
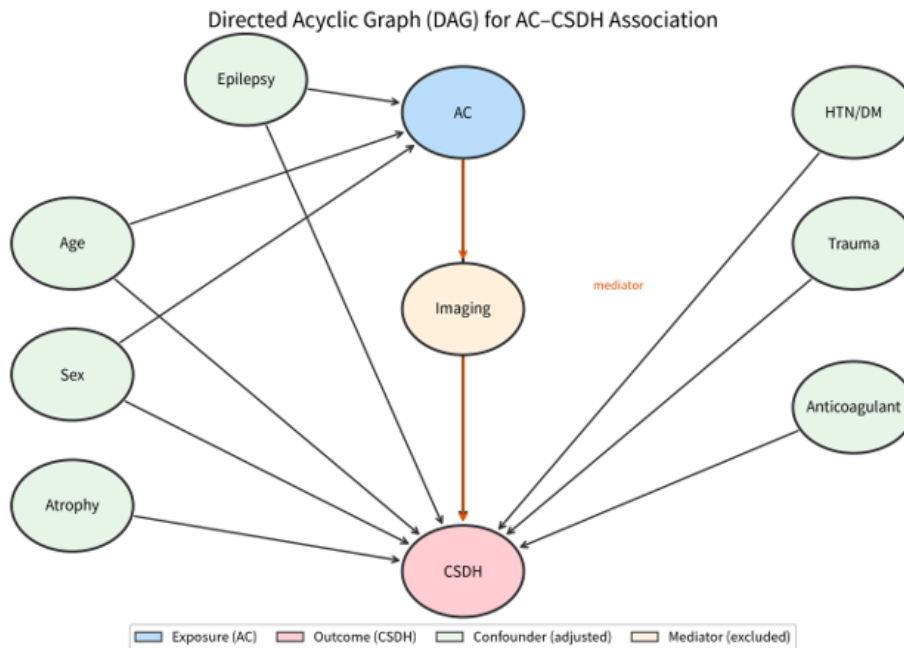


Figure 4

CSDH laterality in AC patients (unilateral).

Dashed line = 50% expected by chance.



Adjustment set: Age, Sex, Trauma, Hypertension, Diabetes, Anticoagulant, Cerebral atrophy, Epilepsy. Imaging frequency is a mediator (AC→imaging→CSDH detection), excluded from primary model.

Figure 5

DAG for causal inference.

Green = exposure; red = outcome; blue = confounders (primary model); orange = detection bias proxy (sensitivity only).

Supplementary Files

This is a list of supplementary files associated with this preprint. Click to download.

- [SupplementaryMaterials.docx](#)