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ORIGINAL RESEARCH

Seasonal Variation in the Onset of Acute Cerebral Infarction and Its Association with Clinical Severity and Risk Factors: A Five-Year Single-Center Retrospective Study

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Objective: Winter low temperatures may affect the occurrence of acute cerebral infarction by affecting blood pressure, but little is known about the association between seasonal variation and the clinical severity of acute cerebral infarction. This study aims to explore the seasonal variation of acute cerebral infarction and its relationship with stroke severity and vascular risk factors.

Methods: A five-year retrospective study was conducted including 692 patients with acute cerebral infarction admitted to a second class A hospital. Patients were stratified by season of onset. Stroke severity was assessed using NIHSS, and multivariable logistic regression was applied to analyze associations and interactions.

Results: While incidence did not differ significantly by season, stroke severity was highest in winter. Spring onset might be independently associated with lower odds of severe stroke (OR = 0.38, 95% CI: 0.14–0.94, $p = 0.05$). Significant interactions were observed for alcohol use in summer (OR = 10.69, 95% CI: 1.42–222.84) and atrial fibrillation in spring (OR = 7.30, 95% CI: 1.13–66.12). Sex-stratified analysis revealed that spring onset was associated with lower incidence of moderate-to-severe stroke (NIHSS $\geq$ 8) among females, while atrial fibrillation and hypertension were risk factors for moderate-to-severe cerebral infarction in males.

Conclusion: A seasonal variation was found in the clinical severity of acute cerebral infarction and was highest in winter. Due to the lack of direct environmental exposure data collection (such as environmental temperature, humidity, air pollution), the specific mechanism still needs further exploration.

Keywords: acute cerebral infarction, stroke severity, seasonality, risk factors, retrospective cohort

Introduction

Cerebral infarction remains one of the foremost causes of mortality and long-term disability worldwide. According to the Global Burden of Disease Study 2019, the global age-standardized incidence of stroke reached 82.4 per 100,000 population, with a disproportionately high burden observed in low- and middle-income countries, especially in East Asia.[1] In China, recent national surveys have indicated a rising trend in stroke prevalence, with cerebral infarction accounting for over 75% of all stroke subtypes.[2] The increasing prevalence of aging, hypertension, diabetes mellitus, and atrial fibrillation has further amplified the disease burden, emphasizing the need for improved prevention, risk stratification, and timely intervention.

Growing evidence has suggested that cerebrovascular events, particularly cerebral infarction, may exhibit seasonal variation.[3] Ambient temperature is a widely discussed season-linked exposure that may plausibly influence both acute cerebral infarction occurrence and clinical severity. Previous studies have shown that extreme temperatures, both heat and cold, could pose a threat to people's health.[4,5] The frequency of such events tends to increase during colder months,

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which has been attributed to physiological responses such as elevated blood pressure, platelet activation, increased blood viscosity, vascular endothelial dysfunction, and heightened sympathetic nervous system activity.[6–8] In addition, behavioral and environmental changes, including reduced physical activity, increased indoor air pollution, and shifts in dietary habits during colder seasons, may also contribute to the observed seasonal disparity.[9] Cold spells were significantly correlated with increased hematocrit and fibrinogen levels, which may increase the tendency for thrombosis and lead to cerebral infarction in cold winter.[10,11] Nevertheless, most existing studies have focused solely on seasonal changes in incidence, often without exploring whether the severity of cerebral infarction or the distribution of key risk factors varies with seasonal patterns. Also, evidence from high latitudes where winter temperatures fluctuate widely may not be directly applicable to subtropical regions where winters are milder. Timely assessment of the severity of cerebral infarction is crucial for guiding clinical treatment and the better allocation of resources.[12,13]

Although seasonal fluctuations in stroke incidence have been documented, critical limitations remain in the current literature. Many studies lack sufficient sample sizes or long-term data to capture consistent trends over time. Furthermore, the relationship between seasonal variation and the clinical severity of cerebral infarction, assessed by standardized measures such as the National Institutes of Health Stroke Scale (NIHSS), has not been adequately investigated. The seasonal distribution of major clinical risk factors, and their potential interactions with seasonal effects, remains poorly understood. These limitations may hinder the development of season-specific risk assessment tools and evidence-based management strategies.

In response to these gaps, the present study investigates the seasonal variation of cerebral infarction based on a five-year retrospective cohort from a tertiary care hospital in China. The primary objectives are to describe the seasonal distribution of cerebral infarction onset, evaluate its association with clinical severity at admission, and examine whether seasonal variation interacts with established clinical risk factors such as drinking and atrial fibrillation. This study focuses on the interaction between clinical presentations and established risk factors under seasonal variation, rather than quantifying specific environmental exposures such as temperature, humidity, or air pollution. The findings are expected to enhance our understanding of the seasonal variation on stroke presentation and to provide a foundation for more precise and timely interventions in stroke care.

Materials and Methods

Study Design and Population

This was a single-center, retrospective observational study conducted at a second class A hospital in China. Consecutive patients admitted with a primary diagnosis of acute cerebral infarction between January 1, 2020 and December 31, 2024 were screened for eligibility. The diagnosis of cerebral infarction was confirmed by clinical symptoms in conjunction with neuroimaging findings, including brain computed tomography (CT) or magnetic resonance imaging (MRI).

Patients were included in the analysis if they met the following criteria: (1) age 18 years or older; (2) diagnosis of acute cerebral infarction confirmed by clinical and radiological evidence at the time of admission; (3) symptom onset occurred within seven days prior to admission; and (4) complete clinical data were available, including demographic information, vascular risk factors, NIH Stroke Scale (NIHSS) score at admission, and neuroimaging results.

Exclusion criteria were as follows: (1) diagnosis of hemorrhagic stroke or transient ischemic attack (TIA); (2) presence of severe systemic illnesses such as end-stage renal, hepatic, or cardiac failure; (3) incomplete clinical or radiological data; and (4) uncertain time of symptom onset.

All included patients were evaluated using a standardized data collection protocol and were followed through hospital discharge to record clinical outcomes. The study protocol was approved by the institutional ethics committee, and the need for informed consent was waived due to the retrospective design and anonymized data analysis.

Data Collection

Clinical data were retrospectively extracted from the hospital's electronic medical records using a standardized protocol. Variables collected included demographic characteristics (age, sex), lifestyle factors (current smoking, recent alcohol consumption), and vascular risk factors such as hypertension, diabetes mellitus, atrial fibrillation, and prior stroke. All diagnoses were based on standard clinical or laboratory criteria. Clinical severity was assessed using the NIHSS at admission, and moderate-to-severe stroke was defined as NIHSS ≥ 8, consistent with previous studies.[14,15] Neuroimaging was used to determine lesion laterality (left, right, bilateral, or corpus callosum involvement). Laboratory indicators included lipid profile (total cholesterol, triglycerides, HDL-C, LDL-C), metabolic markers (fasting glucose, homocysteine, uric acid, creatinine), and apolipoproteins (ApoA1, ApoB, Apo(a)). Derived indices included the atherogenic index of plasma (AIP = $\log_{10}$[triglyceride/HDL-C]), non-HDL-C (total cholesterol minus HDL-C), and the LDL/HDL ratio; these were used in supplementary analyses. Additional variables included admission blood pressure, hospital length of stay, swallowing function (Kubota test), and discharge outcomes (improved, not improved, death, transfer, or discharge against medical advice). The modified Rankin Scale (mRS) score was used to assess functional status at discharge. Due to the fact that this study mainly used routinely collected clinical data to explore seasonal variation in acute cerebral infarction and its relationship with stroke severity and vascular risk factors, we did not collect environmental exposure-related data (such as ambient temperature, humidity, or air pollution index).

Seasonal Definition and Classification

To investigate seasonal variation in the onset and severity of cerebral infarction, patients were categorized into four groups based on the date of symptom onset, following the conventional meteorological classification used in China. Spring was defined as March through May, summer as June through August, autumn as September through November, and winter as December through February. This classification reflects standard seasonal divisions based on regional climatic conditions and has been widely applied in previous epidemiological studies conducted in East Asian populations. All subsequent analyses, including comparisons of clinical severity, risk factor distribution, and outcomes, were performed according to these four seasonal categories.

Statistical Analysis

All statistical analyses were conducted using R (version 4.3.2) and IBM SPSS Statistics (version 26.0). Continuous variables were expressed as mean ± standard deviation (SD) or median with interquartile range (IQR), depending on distribution, and compared across seasons using one-way analysis of variance (ANOVA) or the Kruskal–Wallis test as appropriate. Categorical variables were presented as frequencies and percentages and compared using the chi-squared (χ^2) test. Multiple validation correction is performed using Bonferroni to adjust the test levels of pairwise comparisons.

To examine the association between season of onset and stroke severity, binary logistic regression analyses were performed with moderate-to-severe cerebral infarction (defined as NIHSS ≥ 8) as the dependent variable. Three models were constructed: an unadjusted model; a minimally adjusted model controlling for age and sex; and a fully adjusted model including lifestyle factors (smoking, alcohol use), vascular risk factors (hypertension, diabetes, atrial fibrillation, prior stroke), and laboratory indicators. Stepwise regression methods were employed to optimize variable selection and minimize collinearity.

Interaction and subgroup analyses were further conducted to explore effect modification by sex (male vs female), age (<65 vs ≥65 years), atrial fibrillation (present vs absent), and prior stroke history. Interaction terms (eg, season × atrial fibrillation) were included in logistic models to assess statistical significance. In subgroup analyses, separate logistic regression models were stratified by the above covariates to identify differential seasonal effects. A two-sided p-value <0.05 was considered statistically significant for all analyses.

Results

Patient Selection and Seasonal Grouping

A total of 692 patients diagnosed with acute cerebral infarction between January 2020 and December 2024 were included in the final analysis based on predefined eligibility criteria. Patients were subsequently stratified into four groups according to the season of symptom onset: spring (n = 139), summer (n = 189), autumn (n = 193), and winter (n = 171), using the standard meteorological classification applied in China (Figure 1).

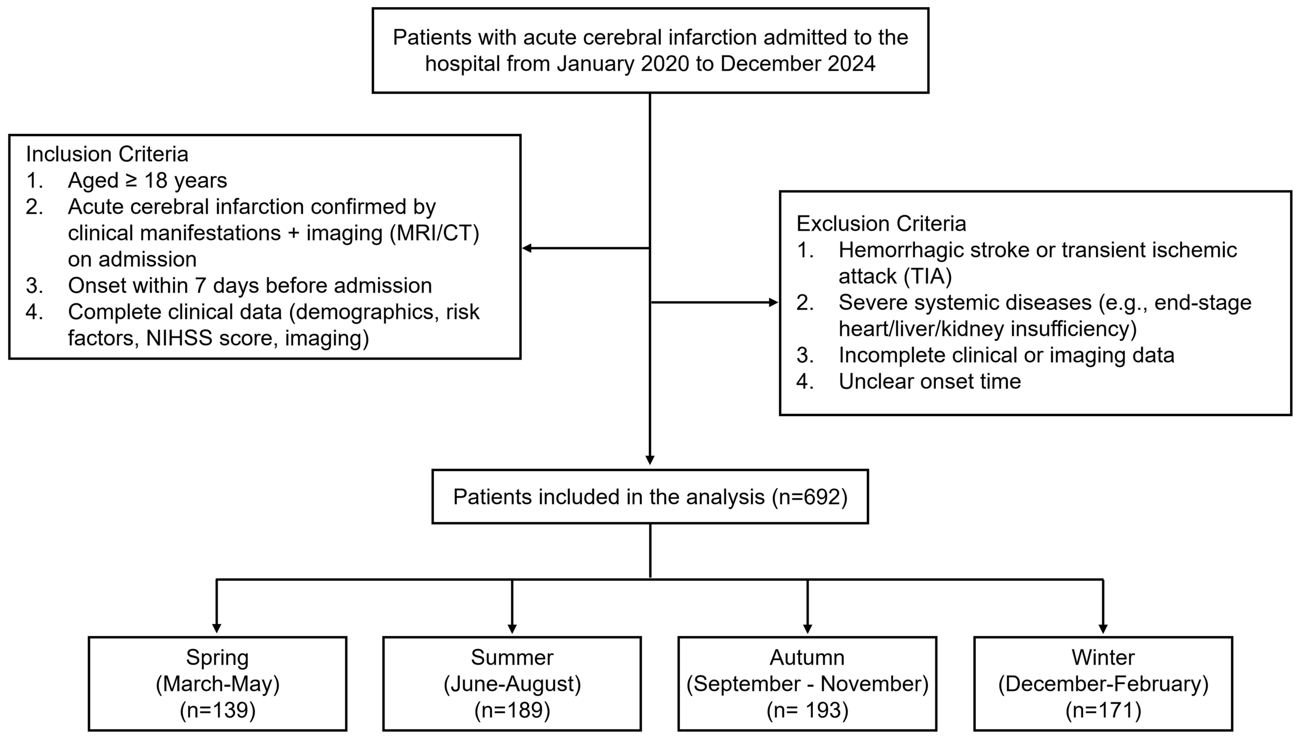

Figure 1 Flowchart of patient selection and seasonal grouping.

Baseline Clinical Characteristics of Patients Across Seasons

Baseline demographic, clinical, and laboratory characteristics by season are summarized in Table 1. Significant seasonal variation was observed in NIHSS scores at admission ($p = 0.018$), with higher values recorded in winter and spring compared to autumn, and higher in winter compared to summer. Length of hospital stay showed a non-significant trend toward longer

Table 1 Baseline Demographic, Clinical, and Laboratory Characteristics of Patients with Acute Cerebral Infarction According to Season of Onset

Parameters	Spring (N = 139)	Summer (N = 189)	Autumn (N = 193)	Winter (N = 171)	p-value
Age	66.18 ± 2.16	68.37 ± 1.77	65.18 ± 1.79	68.16 ± 1.95	0.06
Male	96(69%)	134(71%)	142(74%)	120(70%)	0.818
Length of hospital stay (days)	14.24 ± 1.52	11.92(±0.73)	13.16 ± 0.82	14.22 ± 1.67	0.059
NIHSS score	4.19 ± 1.04	3.22 ± 0.54†	2.99 ± 0.61‡†	4.3 ± 0.89	0.018*
mRS score	2.14 ± 0.22	1.97 ± 0.19	1.93 ± 0.17	2.26 ± 0.2	0.055
Water swallowing test (Good = 2)	9(6%)	20(11%)	16(8%)	20(12%)	0.158
Lesion location (Right = 2)	61(44%)	80(42%)	94(49%)	63(37%)	0.523
Discharge outcome (Not recovered = 2)	7(5%)	10(5%)	5(3%)	10(6%)	0.505
Blood pressure (high = 1)	65(47%)	65(34%)	82(42%)	79(46%)	0.074
Medical history					
Smoking (Yes = 1)	33(24%)	58(31%)	74(38%)	53(31%)	0.043*
Drinking (Yes = 1)	21(15%)	30(16%)	39(20%)	36(21%)	0.375
Stroke history (Yes = 1)	10(7%)	21(11%)	14(7%)	26(15%)	0.045*
Hypertension (Yes = 1)	116(83%)	151(80%)	157(81%)	146(85%)	0.549
Diabetes (Yes = 1)	55(40%)	72(38%)	67(35%)	67(39%)	0.774

(*Continued*)

Table 1 (Continued).

Parameters	Spring (N = 139)	Summer (N = 189)	Autumn (N = 193)	Winter (N = 171)	p-value
Atrial fibrillation (Yes = 1)	19(14%)	11(6%)	9(5%)	11(6%)	0.011*
Laboratory parameters					
Total cholesterol (mmol/L)	4.68 ± 0.22	4.47 ± 0.15	4.56 ± 0.16	4.49 ± 0.18	0.718
Triglyceride (mmol/L)	1.61 ± 0.19	1.64 ± 0.18	1.61 ± 0.14	1.55 ± 0.14	0.845
Blood glucose (mmol/L)	6.87 ± 0.56	6.64 ± 0.43	6.51 ± 0.34	6.84 ± 0.46	0.663
HDL–C (mmol/L)	1.12 ± 0.05	1.08 ± 0.04	1.11 ± 0.04	1.09 ± 0.04	0.805
LDL–C (mmol/L)	2.93 ± 0.19	2.89 ± 0.14	2.97 ± 0.14	2.87 ± 0.16	0.708
HCY (μmol/L)	16.33 ± 1.12	19.15 ± 2.13	20.12 ± 2.13	20.31 ± 2.94	0.142
Creatinine (μmol/L)	82.04 ± 7.25	76.58 ± 4.59	74.79 ± 4.2	82.05 ± 5.93	0.144
Uric acid (μmol/L)	330.62 ± 16.97	332.4 ± 15.29	342.87 ± 16.22	337.47 ± 17.13	0.863

Note: *p < 0.05. †Compared with spring; †Compared with winter.

duration in spring and winter (p = 0.059). Among categorical variables, smoking history (p = 0.043), alcohol consumption (p = 0.045), and the presence of atrial fibrillation (p = 0.011) differed significantly across seasons. However, after correction, the results showed that no pairwise differences remained statistically significant. No significant seasonal differences were noted in other demographic factors, comorbidities, or laboratory measures, including lipid profiles, glucose, homocysteine, creatinine, and uric acid. Therefore, these findings should be interpreted as exploratory.

Seasonal Distribution of Stroke Onset and Admission Severity

The seasonal distribution of acute cerebral infarction onset and initial stroke severity is shown in Figure 2. Panel A indicates that while autumn had the highest proportion of cases (27.89%) and spring the lowest (20.09%), no statistically significant difference was observed in the number of admissions across the four seasons (p = 0.2133). Panel B demonstrates significant seasonal variation in stroke severity, as measured by NIHSS scores at admission. The median NIHSS score was highest in winter, followed by spring, and lowest in autumn (p = 0.0178). In seasonal comparisons, the NIHSS scores of patients admitted in spring (p = 0.037) and winter (p = 0.0033) were significantly higher than those in autumn. The NIHSS score of winter patients was also significantly higher than that of summer patients (p = 0.046).

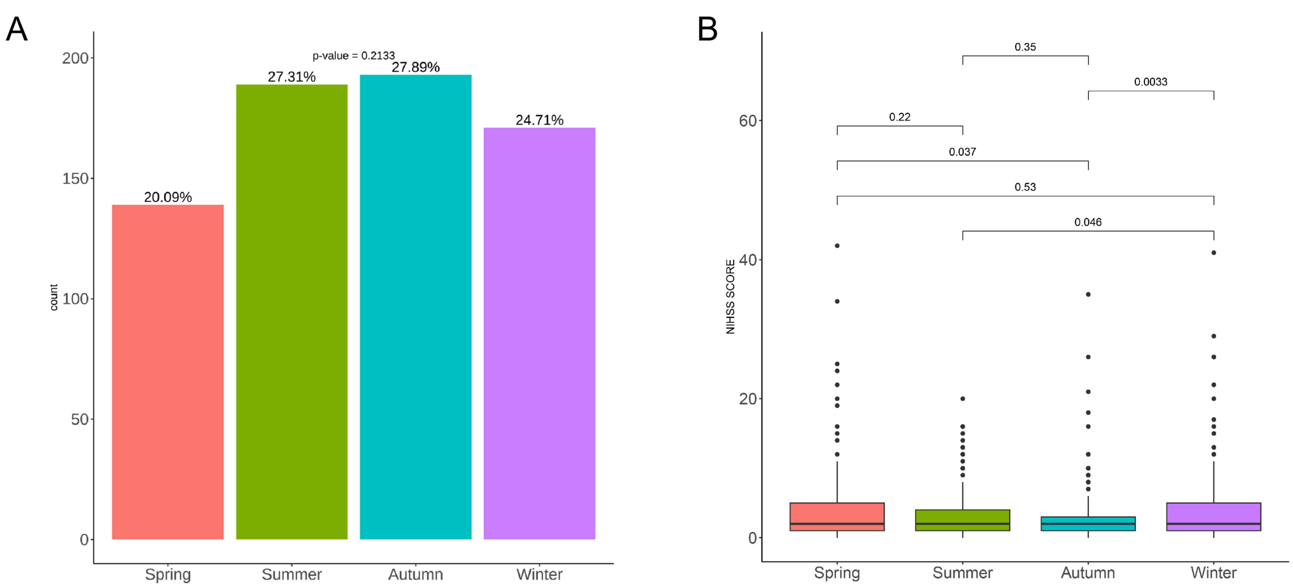

Figure 2 Seasonal distribution and admission severity of patients with acute cerebral infarction. (**A**) Bar plot showing the number of stroke cases by season. (**B**) Boxplot comparing NIHSS scores at admission across seasons.

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Table 2 Seasonal Comparison of Laboratory Parameters in Patients with Acute Cerebral Infarction

Variable	Spring	Summer	Autumn	Winter	p-value
Total cholesterol (mmol/L)	4.68 ± 0.22	4.47 ± 0.15	4.56 ± 0.16	4.49 ± 0.18	0.718
Triglycerides (mmol/L)	1.61 ± 0.19	1.64 ± 0.18	1.61 ± 0.14	1.55 ± 0.14	0.845
Blood glucose (mmol/L)	6.87 ± 0.56	6.64 ± 0.43	6.51 ± 0.34	6.84 ± 0.46	0.663
HDL-C (mmol/L)	1.12 ± 0.05	1.08 ± 0.04	1.11 ± 0.04	1.09 ± 0.04	0.805
LDL-C (mmol/L)	2.93 ± 0.19	2.89 ± 0.14	2.97 ± 0.14	2.87 ± 0.16	0.708
HCY (µmol/L)	16.33 ± 1.12	19.15 ± 2.13	20.12 ± 2.13	20.31 ± 2.94	0.142
Creatinine (µmol/L)	82.04 ± 7.25	76.58 ± 4.59	74.79 ± 4.2	82.05 ± 5.93	0.144
Uric acid (µmol/L)	330.62 ± 16.97	332.4 ± 15.29	342.87 ± 16.22	337.47 ± 17.13	0.863

Seasonal Variation in Laboratory Parameters Among Patients with Acute Cerebral Infarction

No statistically significant seasonal variation was observed in laboratory parameters among patients with acute cerebral infarction (Table 2). Levels of total cholesterol, triglycerides, blood glucose, HDL-C, LDL-C, HCY, creatinine, and uric acid were generally comparable across spring, summer, autumn, and winter groups (all $p > 0.05$). Although mean HCY values were slightly higher in winter (20.31 ± 2.94 μmol/L) and autumn (20.12 ± 2.13 μmol/L) compared to spring (16.33 ± 1.12 μmol/L), the differences did not reach statistical significance ($p = 0.142$). Similarly, there were no significant seasonal trends in lipid profiles or renal function markers.

Seasonal Variation in Vascular Risk Factors Among Patients with Acute Cerebral Infarction

The distribution of vascular risk factors across calendar months is illustrated in Figure 3. Overall, the prevalence of most risk factors remained relatively stable throughout the year, with modest fluctuations in specific subgroups. Notably, atrial fibrillation showed a seasonal pattern, with higher proportions observed from July to November, peaking in October.

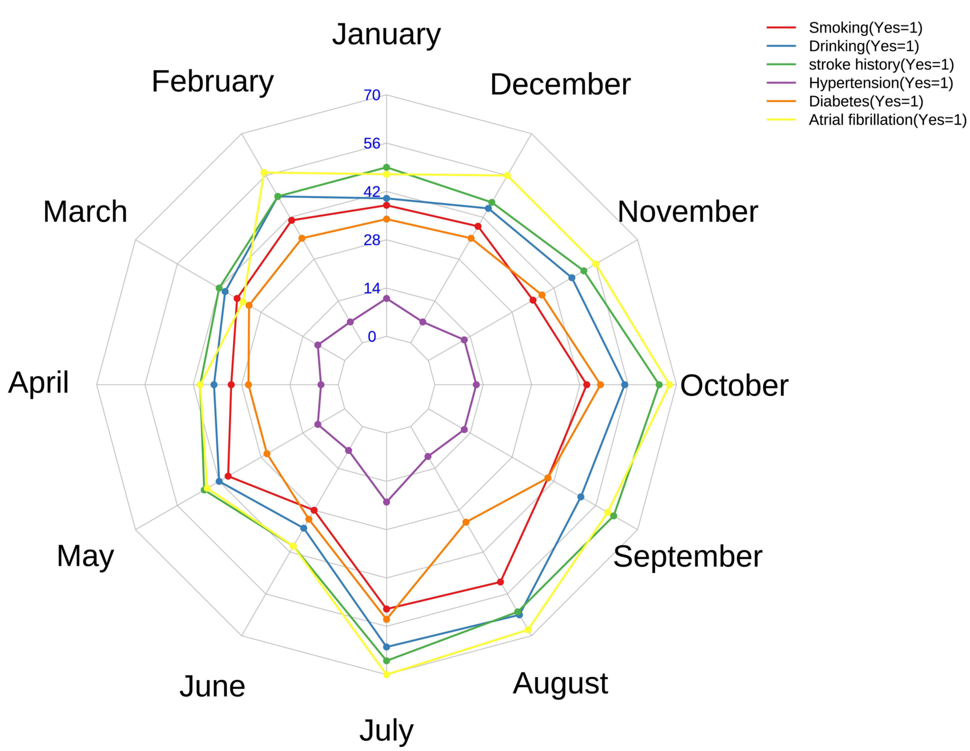

Figure 3 Monthly distribution of vascular risk factors in patients with acute cerebral infarction.

Smoking and alcohol consumption rates appeared slightly elevated during the summer and early autumn months (June to September), whereas the prevalence of hypertension and diabetes remained comparatively consistent across all months.

Association Between Season and Moderate-to-Severe Cerebral Infarction in Multivariable Logistic Regression Models

Logistic regression models were constructed to evaluate the independent association between season of onset and the likelihood of presenting with moderate-to-severe cerebral infarction (NIHSS ≥ 8). In the unadjusted model (Figure 4A), no statistically significant association was found between season and stroke severity. Compared with winter, the ORs for summer, spring, and autumn were 0.89, 0.80, and 0.67, respectively (all p > 0.05). In the age and sex adjusted model (Figure 4B), older age was significantly associated with higher odds of moderate-to-severe stroke (OR = 1.03, 95% CI: 1.01–1.05, p = 0.003), while male sex was inversely associated with severity (OR = 0.49, 95% CI: 0.30–0.80, p = 0.004). Seasonal effects remained non-significant in this model. In the fully adjusted model incorporating demographic, clinical, and laboratory variables (Figure 4C), spring onset was associated with significantly reduced odds of moderate-to-severe stroke compared with winter (OR = 0.38, 95% CI: 0.14–0.94, p = 0.05). Given the borderline p-value, this result should be interpreted with caution and considered as suggestive evidence, which is worth verifying using an independent cohort in the future. Age remained an independent risk factor (OR = 1.03, p = 0.02), and atrial fibrillation was significantly associated with increased severity (OR = 3.23, 95% CI: 1.29–7.73, p = 0.01). Other seasonal categories and total cholesterol were not significantly associated with severity.

Interaction Effects Between Season and Drinking History or Atrial Fibrillation on Stroke Severity

Interaction analysis was conducted to explore whether the association between season and stroke severity was modified by drinking status or atrial fibrillation (Table 3). The models revealed significant interactions in both domains. For drinking history, patients who consumed alcohol and experienced stroke in summer or autumn exhibited markedly increased odds of presenting with moderate-to-severe cerebral infarction. Specifically, the interaction terms summer × drinking (OR = 10.69, 95% CI: 1.42–222.84, p = 0.044) and autumn × drinking (OR = 10.67, 95% CI: 1.40–223.27, p =

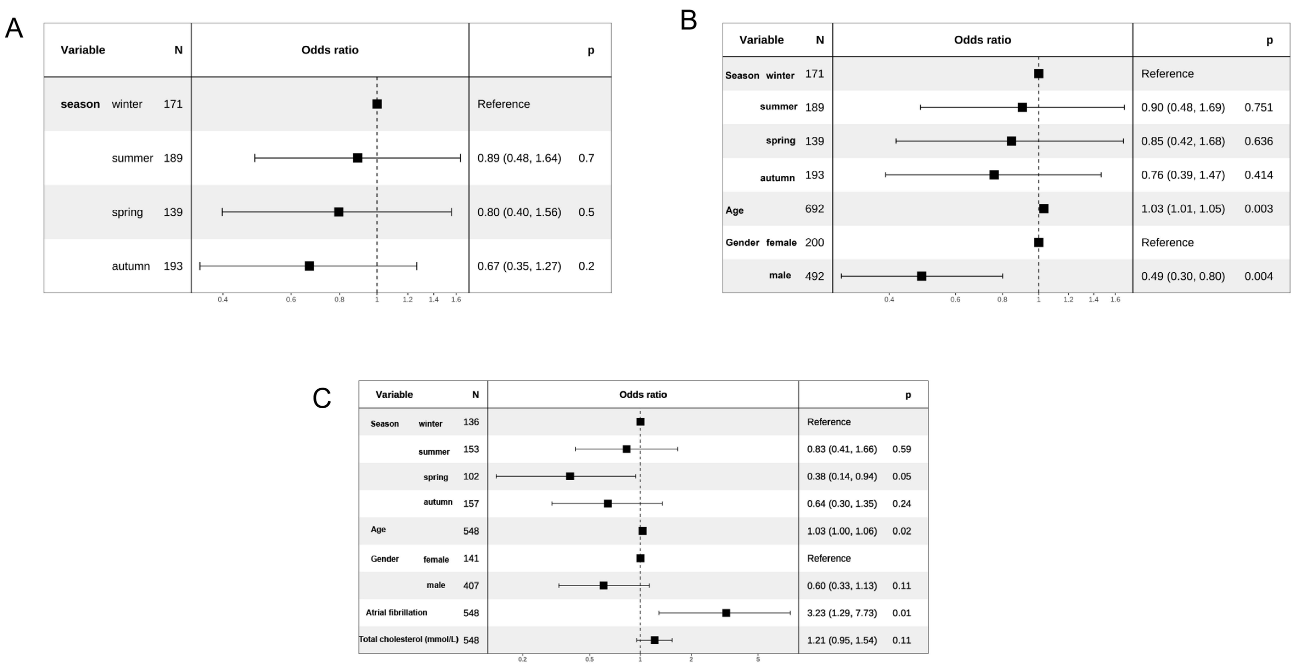

Figure 4 Multivariable logistic regression models evaluating the association between season and risk of moderate-to-severe cerebral infarction (NIHSS ≥ 8). (**A**) Unadjusted model (**B**) age- and sex-adjusted model (**C**) fully adjusted model including age, sex, atrial fibrillation, and laboratory markers. Odds ratios (squares) with 95% confidence intervals (horizontal lines) are shown.

Table 3 Interaction Analysis of Season with Drinking History and Atrial Fibrillation in Relation to Stroke Severity (NIHSS ≥ 8)

Variable	OR	Coef	SE (Coef)	p-value	95% CI
(Intercept)	0.207	–1.574	0.229	<0.001	[0.129, 0.318]
Summer	0.655	–0.423	0.335	0.207	[0.337, 1.261]
Spring	0.703	–0.353	0.359	0.326	[0.342, 1.409]
Autumn	0.483	–0.729	0.362	0.044	[0.232, 0.971]
Drinking	0.138	–1.981	1.04	0.057	[0.008, 0.692]
Summer × Drinking	10.688	2.369	1.175	0.044	[1.416, 222.844]
Spring × Drinking	2.49	0.912	1.486	0.539	[0.090, 69.272]
Autumn × Drinking	10.665	2.367	1.179	0.045	[1.403, 223.268]
(Intercept)	0.159	–1.836	0.23	<0.001	[0.099, 0.244]
Summer	0.794	–0.231	0.33	0.485	[0.413, 1.518]
Spring	0.448	–0.803	0.432	0.063	[0.181, 1.007]
Autumn	0.639	–0.449	0.343	0.191	[0.322, 1.246]
Atrial Fibrillation	1.394	0.332	0.815	0.684	[0.204, 5.869]
Summer × Atrial Fibrillation	3.239	1.175	1.055	0.265	[0.433, 31.137]
Spring × Atrial Fibrillation	7.304	1.988	1.007	0.048	[1.132, 66.12]
Autumn × Atrial Fibrillation	2.014	0.7	1.171	0.55	[0.183, 22.332]

0.045) were both statistically significant. This suggests that drinking alcohol may increase the stroke severity in summer and autumn. For atrial fibrillation, a significant interaction was observed only for the spring × atrial fibrillation term (OR = 7.30, 95% CI: 1.13–66.12, p = 0.048), indicating that atrial fibrillation was more strongly associated with moderate-to-severe stroke among patients whose onset occurred in spring. No significant interactions were found for other seasons in relation to atrial fibrillation (p > 0.05). However, the wide range of confidence intervals indicated limited accuracy of the results and should be treated with caution. A larger sample size is needed for validation in the future.

Subgroup Analysis by Sex on the Association Between Season and Stroke Severity

Subgroup analyses stratified by sex were performed to explore whether the association between seasonal onset and moderate-to-severe cerebral infarction (NIHSS ≥ 8) differed in male and female patients.

Among male patients (Figure 5A), atrial fibrillation (OR = 5.32, 95% CI: 1.52–19.12, p = 0.009) and hypertension (OR = 6.00, 95% CI: 1.29–53.60, p = 0.049) were significantly associated with higher odds of moderate-to-severe stroke. A protective

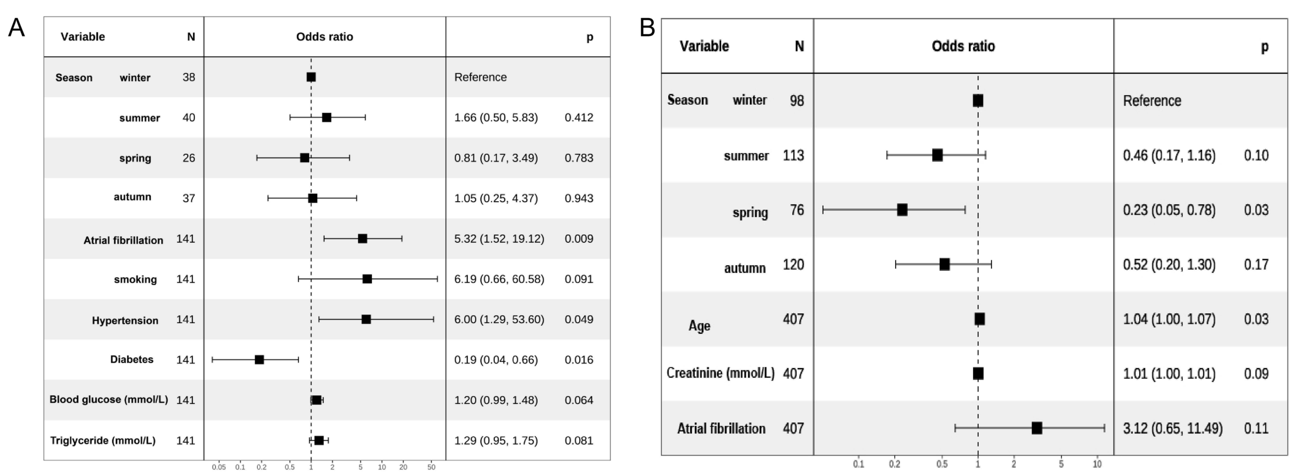

Figure 5 Forest plots of subgroup analyses stratified by sex for predictors of moderate-to-severe cerebral infarction (NIHSS ≥ 8). (**A**) Male subgroup: Atrial fibrillation, hypertension, and diabetes showed significant associations with stroke severity. (**B**) Female subgroup: Spring onset and age were significantly associated with stroke severity. Odds ratios are shown with 95% confidence intervals.

Table 4 Comparison of Length of Hospital Stay and Discharge Outcomes Among Patients with Acute Cerebral Infarction Across Four Seasons

Parameter	Spring (n = 139)	Summer (n = 189)	Autumn (n = 193)	Winter (n = 171)	p-value
Length of hospital stay (days)	14.24 ± 1.52	11.92 ± 0.73	13.16 ± 0.82	14.22 ± 1.67	0.059
Discharge outcome: Improved (1)	131 (94%)	175 (93%)	185 (96%)	157 (92%)	0.505
Discharge outcome: Not recovered (2)	7 (5%)	10 (5%)	5 (3%)	10 (6%)	0.505
Discharge outcome: Death (3)	0 (0%)	0 (0%)	0 (0%)	2 (1%)	0.505
Discharge outcome: Transferred (4)	1 (1%)	1 (1%)	1 (1%)	1 (1%)	0.505
Discharge outcome: Voluntary discharge (5)	0 (0%)	3 (2%)	2 (1%)	1 (1%)	0.505

effect was observed for diabetes mellitus (OR = 0.19, 95% CI: 0.04–0.66, p = 0.016). Seasonal variables did not reach statistical significance in this subgroup (all p > 0.05), indicating no strong seasonal influence on severity among males.

In contrast (Figure 5B), the spring season was significantly associated with a lower risk of moderate-to-severe stroke in female patients compared to winter (OR = 0.23, 95% CI: 0.05–0.78, p = 0.03). Age was also significantly associated with increased severity (OR = 1.04, 95% CI: 1.00–1.07, p = 0.03), while other seasonal categories and clinical variables, including atrial fibrillation and creatinine, did not reach statistical significance.

Seasonal Differences in Clinical Outcomes and Hospital Stay

Clinical outcomes and length of hospital stay across the four seasons are summarized in Table 4. Although not statistically significant (p = 0.059), the average length of hospitalization varied seasonally, with patients admitted in spring (14.24 ± 1.52 days) and winter (14.22 ± 1.67 days) experiencing longer stays compared to those admitted in summer (11.92 ± 0.73 days) and autumn (13.16 ± 0.82 days).

Regarding discharge outcomes, no significant differences were observed among the seasons (p = 0.505). The majority of patients in all groups were discharged with clinical improvement, ranging from 92% in winter to 96% in autumn. The proportion of patients not recovered at discharge ranged from 3% to 6%, while death was recorded only in the winter group (2 cases, 1%). Transfers and voluntary discharges were rare and evenly distributed.

Discussion

This five-year, single-center retrospective study aimed to explore whether the onset of acute cerebral infarction demonstrates seasonal variation and whether such variation is associated with differences in stroke severity and vascular risk profiles. Although the incidence of cerebral infarction remained relatively consistent throughout the year, patients admitted during winter presented with significantly higher NIHSS scores at admission, indicating more severe neurological impairment. In contrast, spring onset was independently associated with a lower likelihood of moderate-to-severe stroke, even after adjusting for age, sex, vascular comorbidities, and laboratory markers. This seasonal disparity in severity did not appear to be driven by differences in laboratory values or most conventional risk factors, which remained stable across seasons. However, interaction analysis revealed that certain risk factors had season-dependent effects. Specifically, the association between alcohol consumption and stroke severity was significantly amplified during summer and autumn, while the impact of atrial fibrillation on severity was more pronounced in spring. Subgroup analyses further indicated a sex-specific pattern: spring onset was associated with a decrease of stroke severity in female patients, whereas in males, atrial fibrillation and hypertension were the dominant predictors of moderate-to-severe presentation. These findings suggest that season may interact with both biological and behavioral factors to shape the clinical manifestation of cerebral infarction and that accounting for seasonal variation may enhance risk stratification in acute stroke care.

The seasonal variation of stroke onset and severity have been widely investigated in previous studies, particularly across different regions of Asia, Europe, and North America. Several large-scale studies from northern China and Korea have reported a marked increase in ischemic stroke incidence during winter months, often attributed to cold-induced vasoconstriction, elevated blood pressure, and increased thrombogenic potential.[8,16] Similar findings have been echoed in

Western populations. For instance, Lorking N et al[17] found a winter peak in stroke admissions across multiple European centers, while Chu S Y et al[18] observed higher stroke incidence and mortality during colder months in the United States. In contrast to these studies, our data showed no significant seasonal variation in the number of acute cerebral infarction cases, although patients presenting in winter exhibited significantly greater clinical severity.

This apparent discrepancy may be explained by several key differences in study design and population characteristics. First, many prior studies focused primarily on incidence trends without evaluating stroke severity or adjusting for comorbid conditions, whereas our analysis incorporated NIHSS scores and accounted for clinical and laboratory confounders. Second, geographic and climatic variations may play a role. Compared to northern regions with extreme temperature fluctuations, our study was conducted in a subtropical area with relatively milder winters, which may attenuate the physiological stress induced by cold exposure. Third, the use of a five-year longitudinal dataset in our study enhances stability in seasonal trend estimation, whereas some previous reports were based on shorter or interrupted time frames. In addition, earlier studies rarely investigated seasonal interactions with individual risk factors. By contrast, our findings suggest that season may modify the impact of behavioral and vascular risk factors such as alcohol consumption and atrial fibrillation on stroke severity, an aspect that has been largely underexplored in existing literature. Taken together, these differences underscore the importance of contextualizing seasonal stroke research within regional, clinical, and methodological frameworks.

The observed seasonal variation in stroke severity, particularly the greater neurological impairment seen during winter months, may be explained by several interrelated physiological and behavioral factors. Exposure to low temperatures has been shown to trigger vasoconstriction, elevate systemic blood pressure, enhance sympathetic nervous system activity, and increase blood viscosity, all of which can exacerbate cerebral ischemia.[19] Reduced sunlight radiation during the cold season leads to insufficient secretion of vitamin D, triggering parathyroid hormone secretion, intracellular calcium release, vasoconstriction, and sympathetic nervous system activity.[20] These mechanisms may contribute to larger infarct size and more severe deficits at the time of presentation. Conversely, the association between spring onset and lower stroke severity, particularly among female patients, may reflect a combination of moderate climate, restoration of circadian stability, and reduced inflammatory burden following winter.[21,22] Women have higher vulnerability of blood pressure variations compared to men. A study suggested that female was an independent predictor of a drop in blood pressure during the summer.[23] Therefore, the warm climate in spring leads to a decrease of blood pressure in women, which may have a protective effect against acute cerebral infarction in women. Spring may also coincide with improved lifestyle behaviors and hormonal balance, which could have protective effects on vascular function and neurological outcomes.[24] A study conducted in Shenzhen, a southern city in China, showed that diurnal temperature range (DTR) was significantly associated with first stroke, with elevated DTR in summer and winter increasing the risk of stroke.[10] One possible explanation was that large temperature differences can easily exceed the body's thermoregulatory ability, eventually causing stroke.

In addition to environmental influences, seasonal variation appears to interact with specific patient-level risk factors. The association between alcohol consumption and stroke severity was strongest during summer and autumn, possibly reflecting increased alcohol intake during social events or holidays, along with higher risks of dehydration under warmer conditions.[25,26] The enhanced impact of atrial fibrillation observed in spring may be linked to abrupt fluctuations in temperature, which could destabilize cardiac electrophysiology and increase the risk of embolic events.[27,28] These findings suggest that the effects of established vascular risk factors are not static but may be modified by seasonal context. From a public health perspective, these results highlight the need to incorporate seasonal variation into risk prediction and management strategies. Tailored preventive measures for patients with atrial fibrillation or frequent alcohol use, especially during vulnerable seasons, could help reduce the burden of severe stroke. At the healthcare system level, anticipatory resource planning during colder months may improve the readiness and efficiency of acute stroke services. Importantly, as this study was designed to investigate the relationship between seasonal variation and severity of acute cerebral infarction and vascular risk factors, we did not collect direct environmental exposure data (such as environmental temperature, humidity, air pollution). Therefore, the insights on seasonal specific risk assessment and management are provided in conjunction with the epidemiological findings of this study, however, the specific mechanisms still need further exploration.

This study has several limitations that should be acknowledged. It was conducted at a single center with a retrospective design, which may limit the generalizability of the findings. Some interaction effects have very wide confidence intervals, suggesting limited precision, likely due to sparse data and small numbers of moderate-to-severe events in certain season-by-risk-factor strata. The impact of environmental factors such as temperature, humidity, and air pollution could not be assessed because they were not directly measured, although they may influence seasonal variation in stroke severity. Key behavioral variables like smoking and alcohol consumption were collected categorically without accounting for intensity or frequency, potentially underestimating their true effects. In addition, factors such as physical activity, diet, and access to care, which may vary seasonally and affect clinical outcomes, were not included. Another point is that this study was conducted in the metropolitan Shanghai; therefore, the results of this study may not be generalizable to other regions such as non-urban and rural areas. Future research should adopt prospective, multicenter designs that incorporate real-time meteorological data, continuous assessments of behavioral risks, and biological markers such as inflammation and coagulation profiles. These enhancements could help clarify the mechanisms linking season to stroke severity and support the development of seasonally informed prevention and management strategies.

Conclusion

This study highlights the relevance of seasonal variation in shaping the clinical manifestation and risk profile of acute cerebral infarction. Recognizing season as a potential modifier of stroke severity could help to enhance individualized prevention strategies and guide more targeted allocation of clinical resources. These findings support the need for seasonally adaptive approaches in both research and stroke care delivery. However, due to the lack of direct direct environmental or biological measurements (such as temperature, humidity, and air pollution), this study lacks support for mechanistic inference and should be approached with caution.

Data Sharing Statement

The datasets generated during and/or analysed during the current study are available from the corresponding author on reasonable request.

Ethical Statement

The study protocol was approved by the institutional ethics committee of Anting Hospital (HXQK-2025-24), and the need for informed consent was waived due to the retrospective design and anonymized data analysis. The study was conducted in accordance with the Declaration of Helsinki.

Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

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The authors declare no competing interests in this work.

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