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# Association between blood triglycerides and stroke-associated pneumonia: a prospective cohort study

Xiujuan Yuan[1†], Shicun Huang[2†], Jianqiang Ni[2*] and Wanli Dong[2*]

## Abstract

**Background** Cerebral infarction requires the reduction of blood lipids, but low triglycerides are associated with poor prognosis. stroke-associated pneumonia (SAP) can also lead to poor prognosis. Therefore, the purpose of this study is to investigate the correlation between triglycerides (TG) and SAP.

**Methods** This prospective cohort study was conducted at the First Affiliated Hospital of Soochow University and enrolled consecutive patients with acute ischemic stroke admitted between March 2019 and March 2021. Univariate analysis, Multivariable logistic regression analysis, subgroup analysis, curve fitting, inflection point analysis, stratified and interaction analyses was performed to examine the relationship between blood TG and SAP.

**Results** The study included 240 patients with acute ischemic stroke (92 females, mean age 68 years), of whom 94 developed SAP. Multivariate logistic regression analysis demonstrated that TG levels were independently associated with SAP. The fitting curve shows a linear relationship between TG level and SAP incidence, with a decrease in SAP incidence as TG increases. The inflection point value is TG = 2.6mmol/L.

**Conclusions** The findings suggest that TG levels may be inversely associated with the risk of SAP in elderly patients with acute ischemic stroke.

**Keywords** Stroke-associated pneumonia, Blood triglycerides, Acute ischemic stroke, Relative risk, Prospective cohort study

## Background

Acute ischemic stroke is characterized by high incidence, high disability, and high mortality [1]. Common complications associated with stroke include post-stroke deterioration, ischemic brain swelling, increased intracranial pressure, stroke-associated pneumonia (SAP), and cardiac complications [2, 3]. Each of these complications requires vigilant monitoring and appropriate management to optimize patient recovery and minimize further health risks.

Notably, SAP stands out as one of the primary causes of worsened outcomes and mortality following stroke, accounting for approximately one-third of early deaths

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and one-fifth of cases with unfavorable stroke outcomes [4, 5].The incidence of SAP among patients with acute stroke varies widely, ranging from 2 to 63%, with ICU studies reporting rates between 18% and 38% [5–7]. Currently, studies such as the Preventive Antibiotics in Stroke Study (PASS) and the Prophylactic Antibiotics after Acute Stroke for Reducing Pneumonia in Patients with Dysphagia (STROKE-INF) study, and even the Prevention of Infections and Fever to Improve Outcome in Older Patients with Acute Stroke (PRECIOUS) trial, have not provided evidence to support the use of prophylactic antibiotics to reduce the incidence of pneumonia [8–10]. This may be due to a failure to adequately identify patients at high risk of SAP. Consequently, early identification of patients at high risk of developing SAP after stroke is crucial for prompt and appropriate intervention. Although scores like the Ischemic Stroke-Associated Pneumonia Score (AIS-APS) [11], Oxford Acute Severity of Illness Score (OASIS) [12], and A2DS2 Score may predict SAP [13], no unified scoring standard exists. Early assessment is critical to determine whether SAP will occur, prompting extensive research into biomarkers associated with SAP in stroke patients. While hypertriglyceridemia is known to increase the risk of ischemic stroke by promoting atherosclerosis, thrombosis, and hyperviscosity [14], several studies have reported a paradoxical relationship between low blood triglyceride levels and poor stroke outcomes [15–18]. In addition, the pre-disease lipid profile has been found to influence the risk of SAP in clinical practice, and one study shows that higher TCBI (Triglycerides, Total Cholesterol, and Body Weight Index) triglyceride levels are associated with lower sepsis-related mortality, with higher triglyceride levels associated with lower sepsis-related mortality [19]. However, the relationship between blood triglycerides and SAP in elderly stroke patients remains

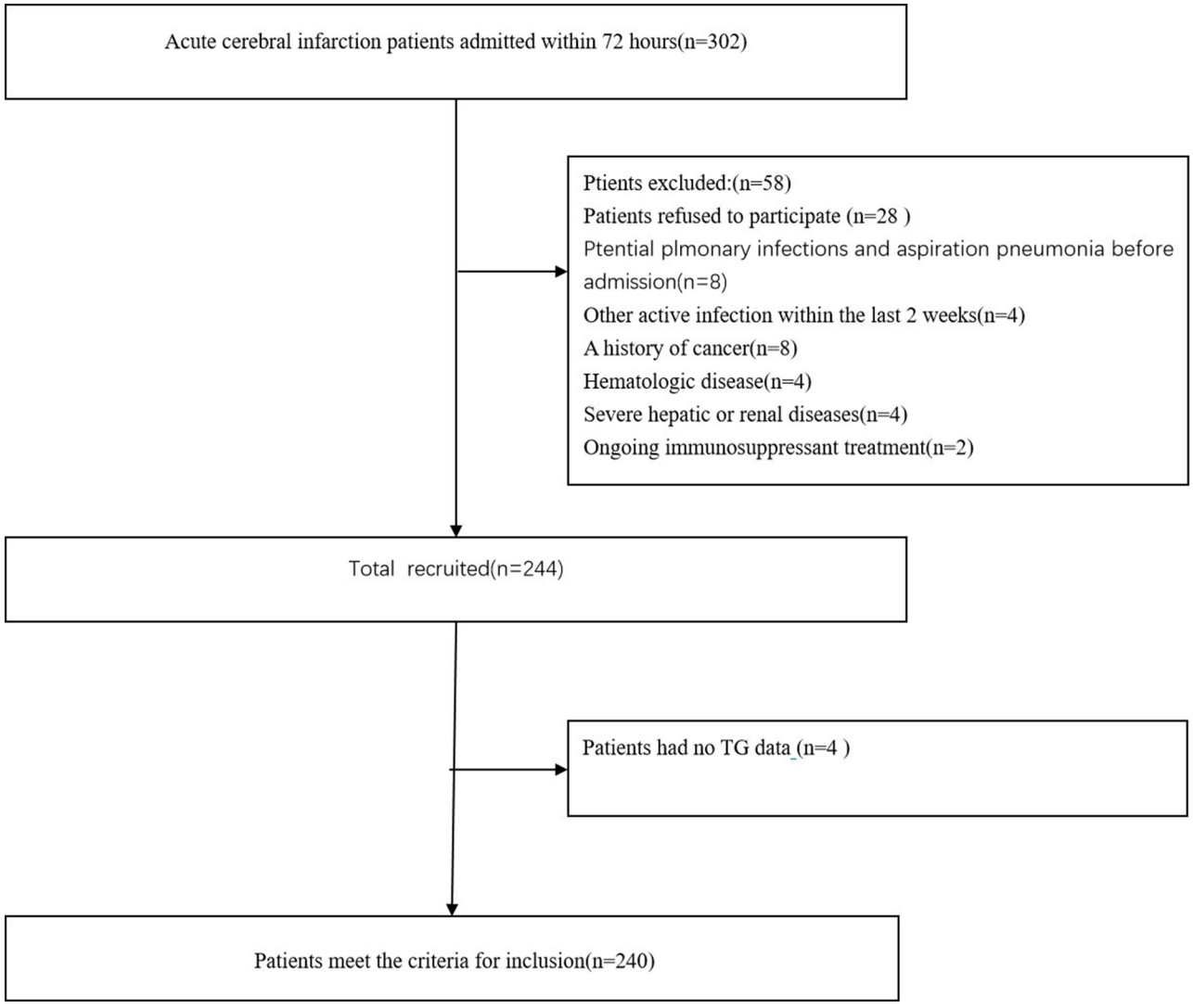


**Fig. 1** Study flowchart

**Table 1** Baseline characteristics of patients

| | TG (mmol/L) | | | | |
|---|---|---|---|---|---|
| **Characteristic** | **Total** | **T1 (≤ 1.7)** | **T2 (1.7–2.3)** | **T3 (≥ 2.3)** | ***p*-value** |
| NO. | 240 | 172 | 40 | 28 | |
| Age (year), Mean (SD) | 68.0 (56.0, 76.0) | 69.5 (58.8, 79.0) | 65.0 (52.0, 75.2) | 64.0 (52.5, 68.5) | 0.016 |
| Sex, n (%) | | | | | 0.019 |
| Male | 148 (61.7) | 111 (64.5) | 17 (42.5) | 20 (71.4) | |
| Female | 92 (38.3) | 61 (35.5) | 23 (57.5) | 8 (28.6) | |
| Hypertension, n (%) | 172 (72.3) | 120 (70.6) | 29 (72.5) | 23 (82.1) | 0.449 |
| Diabetes, n (%) | 70 (29.4) | 50 (29.4) | 10 (25) | 10 (35.7) | 0.634 |
| Smoke, n (%) | 80 (33.6) | 62 (36.5) | 6 (15) | 12 (42.9) | 0.019 |
| Alcohol drinking, n (%) | 35 (14.7) | 27 (15.9) | 4 (10) | 4 (14.3) | 0.701 |
| Infection, n (%) | 94 (39.2) | 79 (45.9) | 10 (25) | 5 (17.9) | 0.002 |
| NIHSS, Median (IQR) | 5.0 (2.0, 10.0) | 5.0 (2.0, 11.0) | 4.0 (2.0, 7.0) | 3.0 (2.0, 5.0) | 0.043 |
| Hs-CRP, Median (IQR) | 2.5 (0.9, 8.2) | 2.7 (0.9, 10.3) | 2.2 (1.3, 6.0) | 1.8 (1.0, 3.5) | 0.308 |
| NEU, Median (IQR) | 5.0 (3.9, 7.1) | 5.1 (3.9, 7.3) | 4.8 (3.9, 7.4) | 4.7 (3.5, 5.7) | 0.361 |
| LYC, modify, Median (IQR) | 1.5 (1.2, 2.0) | 1.5 (1.1, 2.0) | 1.5 (1.3, 2.1) | 1.7 (1.4, 2.5) | 0.034 |
| TG, Median (IQR) | 1.3 (1.0, 1.7) | 1.1 (0.9, 1.3) | 1.9 (1.8, 2.0) | 2.8 (2.5, 3.3) | < 0.001 |

SD: standard deviation; NIHSS: National Institutes of Health Stroke Scale; Hs-CRP: high-sensitivity C-reactive protein; IQR: interquartile range; TG: triglycerides; NEU: Neutrophil, LYC: Lymphocyte Count

poorly understood. Therefore, the aim of this study was to investigate the association between TG levels and SAP in elderly stroke patients.

## Methods

### Study design and population

This prospective cohort study was conducted at the First Affiliated Hospital of Soochow University, involving patients with acute ischemic stroke. Consecutive patients meeting the following inclusion criteria were admitted between March 2019 and March 2021: (1) diagnosed with acute ischemic stroke, (2) aged over 18 years, and (3) admitted within 72 h of symptom onset. Exclusion criteria encompassed: (1) missing data on blood triglyceride levels, (2) a history of cancer or hematologic disease, (3) ongoing immunosuppressant treatment, (4) severe hepatic or renal diseases, (5) pulmonary infections and aspiration pneumonia prior to admission, and (6) any other active infection within the previous two weeks. Diagnosis of acute ischemic stroke was established based on relevant ischemic lesions observed on diffusion-weighted imaging. Two independent and experienced neurologists made the diagnoses for each patient, with confirmation in accordance with the diagnostic criteria for acute ischemic stroke and pneumonia. Approval for the study was obtained from the ethics committee of the First Affiliated Hospital of Soochow University (approval #2021137), and written informed consent was obtained from the patients or their relatives. The study was conducted in adherence to the principles outlined in the Declaration of Helsinki and relevant regulatory guidelines.

### Procedures

This study included all patients admitted with acute ischemic stroke. Upon admission, demographic data such as age, sex, and history of cardiovascular risk factors (including hypertension, type 2 diabetes mellitus, smoking, and alcohol consumption) were recorded. And blood pressure was also measured. Stroke severity was assessed at admission using the National Institutes of Health Stroke Scale (NIHSS). The medical history was inquired about carefully, physical examinations were conducted, and if necessary, perform chest CT or X-ray to rule out potential pulmonary infections and aspiration pneumonia before admission, especially in older patients.

Routine laboratory investigations were conducted after overnight fasting on the first day after admission and included measurement of serum levels of glucose, total cholesterol (TC), high-density lipoprotein cholesterol (HDL-C), triglycerides, and TG levels were measured using a spectrophotometric assay (Olympus AU5400, Sekisui Diagnostic Ltd). Serum low-density lipoprotein cholesterol (LDL-C) levels were calculated using Friedewald's formula [20]. All patients underwent a computed tomography (CT) scan of the brain on admission to rule out ischaemic stroke mimics, such as brain hemorrhage. If a brain hemorrhage was found, the patient was referred to neurosurgery. Magnetic resonance imaging (MRI) on the second day was able to clearly diagnose a cerebral infarction and predict the prognosis of an ischaemic stroke. A second brain CT was performed if clinically indicated. Chest X-ray or CT and review of infection-related indicators were conducted when patients exhibited relevant lower respiratory clinical symptoms and signs. Data on stroke subtype, therapies during hospitalization, complications, blood chemistry examinations,

medical interventions, and imaging (CT and/or MRI) results were obtained upon discharge. Hypertriglyceridemia was defined as TG ≥ 1.7 mmol/L. According to the Chinese Guideline for Lipid Management, TG was divided into three groups: T1 (≤ 1.7 mmol/L) was normal, T2 (1.7–2.3 mmol/L) was the marginally elevated group, and T3 (≥ 2.3 mmol/L) was the heightening group [21].

### Outcomes

The primary outcome of this study was the TG levels and proportion of SAP. SAP was defined according to the recommended terminology for lower respiratory tract infections occurring within the first 7 days after the onset of stroke. The diagnosis of post-stroke pneumonia followed standardized criteria set by the US Centers for Disease Control and Prevention. In addition to pulmonary infiltrates observed on chest X-rays, the diagnosis required the presence of at least one criterion from the first category and at least two criteria from the second category: (1) fever (> 38.0 °C), leukocytosis (> 12 × 10^9/L) or leukopenia (< 4 × 10^9/L), and new-onset unexplained mental disorder in patients aged 70 years or older; (2) new or worsening cough, labored breathing, rapid breathing, abnormal respiratory tests, new or altered purulent sputum, and impaired gas exchange [6, 22]. The proportion of SAP cases was determined by dividing the number of SAP cases by the total number of participants in the study sample.

Data from the medical charts were collected, including information on age, sex, laboratory findings (complete blood count, high-sensitivity C-reactive protein [hs-CRP], blood triglyceride levels, glucose levels, liver function, and kidney function measured in the morning after admission), hypertension, diabetes mellitus, dyslipidemia, alcohol consumption, and smoking), stroke severity and subtypes, and imaging data (magnetic resonance imaging [MRI] cerebral infarction volume).

### Statistical analysis

All statistical analyses were conducted using R 4.0 (The R Project for Statistical Computing, www.r-project.org) and Free Statistics Software 1.9. Normally distributed continuous variables were presented as mean ± standard deviation and analyzed using Student's t-test. Skewed distributed continuous variables were reported as medians (interquartile range, IQR) and analyzed using the Mann-Whitney U-test. Categorical variables were presented as n (%) and analyzed using the chi-square test and Fisher's exact test. A linear regression analysis was used to analyze triglycerid level. We repeated the analyses for study-specific categorical exposures to analyze the TG. Univariable and multivariable logistic regression analyses were performed to assess the associations between TG levels and the risk of SAP. Fit curve analyses were utilized to explore the relationships between TG levels and SAP risk. Subgroup analysis to confirm

**Table 2** Association of covariates and infection risk

| Variable | OR (95% CI) | *p*-Value |
|---|---|---|
| Age (years) | 1.02 (1 ~ 1.04) | 0.029 |
| Sex, n (%) | 1 (reference) | |
| Male | | |
| Female | 0.86 (0.5 ~ 1.47) | 0.58 |
| Hypertension, n (%) | | |
| No | 1 (reference) | |
| Yes | 1.91 (1.04 ~ 3.53) | 0.038 |
| Diabetes, n (%) | | |
| No | 1 (reference) | |
| Yes | 1.22 (0.69 ~ 2.15) | 0.494 |
| Smoke, n (%) | | |
| No | 1 (reference) | |
| Yes | 1.12 (0.65 ~ 1.93) | 0.694 |
| Alcohol drinking, n (%) | | |
| No | 1 (reference) | |
| Yes | 1.03 (0.49 ~ 2.13) | 0.947 |
| Hs-CRP(mg/dl) | 1.04 (1.01 ~ 1.07) | 0.011 |
| NEU (%) | 1.24 (1.11 ~ 1.39) | < 0.001 |
| LYC.modify(%) | 0.77 (0.54 ~ 1.09) | 0.135 |
| NIHSS | 1.23 (1.15 ~ 1.3) | < 0.001 |
| TG (mmol/L) | 0.51 (0.33 ~ 0.8) | 0.003 |
| T1 (≤ 1.7) | 1 (reference) | |
| T2 (1.7–2.3) | 0.39 (0.18 ~ 0.85) | 0.018 |
| T3 (≥ 2.3) | 0.26 (0.09 ~ 0.7) | 0.008 |

NIHSS: National Institutes of Health Stroke Scale; HB: hemoglobin; Hs-CRP: high-sensitivity C-reactive protein; UA: uric acid; TG: triglycerides; NEU: Neutrophil, LYC: Lymphocyte Count

**Table 3** Association between TG and infection

| Tertiles | OR (95% CI) | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| | No. | Crude | *P*-Value | Model 1 | *P*-Value | Model 2 | *P*-Value | Model 3 | *P*-Value |
| TG (mmol/L) | 240 | 0.51 (0.33 ~ 0.80) | 0.003 | 0.54 (0.34 ~ 0.84) | 0.007 | 0.48 (0.30 ~ 0.77) | 0.002 | 0.56 (0.32 ~ 0.99) | 0.044 |
| T1(≤ 1.7) | 172 | 1(Ref) | | 1(Ref) | | 1(Ref) | | 1(Ref) | |
| T2(1.7–2.3) | 40 | 0.39 (0.18 ~ 0.85) | 0.018 | 0.43 (0.19 ~ 0.95) | 0.036 | 0.4 (0.18 ~ 0.89) | 0.025 | 0.47 (0.18 ~ 1.22) | 0.12 |
| T3(≥ 2.3) | 28 | 0.26 (0.09 ~ 0.70) | 0.008 | 0.28 (0.10 ~ 0.78) | 0.015 | 0.24 (0.09 ~ 0.68) | 0.007 | 0.44 (0.14 ~ 1.42) | 0.172 |
| Trend test | | | 0.001 | | 0.003 | | 0.001 | | 0.069 |

T, tertiles; OR, odds ratio; CI, confidence interval; Ref: reference. Model 1 was adjusted for age and sex. Model 2 was adjusted for age, sex, hypertension, diabetes and smoke. Model 3 was adjusted for age, sex, hypertension, diabetes, smoke, alcohol drinking, Hs-CRP, NEU., LYC.modify and NIHSS

the presence of interactions. Inflection point analysis was used. A two-sided P-value < 0.05 was considered statistically significant.

## Results

A total of 240 patients (148 males and 92 females) admitted for acute ischemic stroke were included in this study (Fig. 1). The patients were stratified into three groups according to their TG levels, T1(≤ 1.7mmol/L), T2(1.7–2.3 mmol/L), T3(≥ 2.3 mmol/L). The patients had an age of 68(56–76) years (Table 1). According to the diagnostic criteria for stroke-associated pneumonia, 94 people (39.2%) were diagnosed with SAP, with 79 people (45.9%) in the T1 group, 10 people (25%) in the T2 group, and 5 people (17.9%) in the T3 group (Table 3), indicating that as TG levels increase, the number of post-stroke pneumonia cases decreases. The median TG levels were 1.3 mmol/L (IQR 1.0-1.7). The average NIHSS score is 5.0 (IQR 2.0–10.0), T1 group is 5.0 (IQR 2.0–11), T2 group is 4.0 (IQR 2.0–7.0), and T3 group is 3.0 (IQR 2.0–5.0), indicating that the higher the TG level, the lower the NIHSS score [21].

Univariable logistic regression analysis showed that the factors might be associated with SAP included age (OR = 1.02; 95%CI: 1-1.04; $P = 0.029$), hypertension (OR = 1.91; 95%CI: 1.04–3.53; $P = 0.038$), NIHSS (OR = 1.23; 95%CI: 1.15–1.3; $P < 0.001$), hs-CRP (OR = 1.04; 95%CI: 1.01–1.07; $P = 0.011$), neutrophil (OR = 1.24; 95%CI: 1.11–1.39; $P < 0.001$), TG (OR = 0.51; 95%CI: 0.33–0.80; $P = 0.003$),T2(OR = 0.39; 95%CI: 0.18–0.85; $P = 0.018$),T3 (OR = 0.26; 95%CI: 0.09–0.7; $P = 0.008$) (Table 2).So, age, hypertension, NIHSS, hs-CRP, neutrophil and TG were associated with SAP.

When TG levels was analyzed as a continuous variable, a significant independent negative association was discovered between TG levels and the risk of SAP in the unadjusted model TG group[OR = 0.51; 95% confidence interval (CI): 0.33–0.80; $P = 0.003$], meanwhile, further adjustment did not significantly affect the results. With increased TG levels, the incidence of SAP decreased, and the OR of T3 was lower than that of T1 (OR: 0.26, 95% CI: 0.09–0.70). After adjusting for sociodemographic variables age, sex, hypertension, diabetes, smoke, alcohol drinking, hs-CRP, NEU, LYC.modify and NIHSS in model 3, the association between TG and SAP was marginally significant [OR = 0.56; 95% confidence interval (CI): 0.32–0.99; $P = 0.044$]. The impact of TG on SAP can be seen in unadjusted models, Model 1, and Model 2. In Model 3, due to adjusting for multiple confounding factors and a small sample size, no differences were observed between the T2 and T3 groups (Table 3).

In subgroup analysis, the relationship between TG and SAP remains stable. There was no interaction between age (P for interaction = 0.467), sex (P for interaction = 0.106), hypertension (P for interaction = 0.106), diabetes (P for interaction = 0.106), smoking (P for interaction = 0.106) and drinking (P for interaction = 0.106)(Table 4).

**Table 4** Subgroup analysis of the relationship between TG and infection

| Subgroup | OR (95% CI) | *p*-value | *P* for interaction |
|---|---|---|---|
| Age (years) | | | |
| ≤ 20 | 1 (reference)_ | | 0.467 |
| 20–50 | 0.12 (0.01 ~ 2.52) | 0.172 | |
| ≥ 50 | 0.59 (0.32 ~ 1.1) | 0.096 | |
| Sex | | | |
| Male | 0.7 (0.38 ~ 1.28) | 0.249 | 0.106 |
| Female | 0.27 (0.05 ~ 1.61) | 0.152 | |
| Hypertension | | | |
| No | 0.45 (0.04 ~ 5.46) | 0.534 | 0.676 |
| Yes | 0.6 (0.33 ~ 1.07) | 0.082 | |
| Diabetes | | | |
| No | 0.51 (0.24 ~ 1.07) | 0.076 | 0.636 |
| Yes | 0.59 (0.2 ~ 1.74) | 0.336 | |
| Smoke | | | |
| No | 0.4 (0.17 ~ 0.93) | 0.032 | 0.188 |
| Yes | 0.79 (0.36 ~ 1.72) | 0.547 | |
| Alcohol drinking | | | |
| No | 0.43 (0.22 ~ 0.84) | 0.014 | 0.092 |
| Yes | 1.39 (0.18 ~ 10.84) | 0.755 | |

Covariates were adjusted as in model III (Table 3)

There is a linear relationship between TG levels and the incidence of SAP. TG is approximately normally distributed, and as TG increases, the proportion of SAP decreases (Fig. 2). The inflection point value is TG = 2.6mmol/L. When TG > 2.6mmol/L, the risk of pneumonia significantly decreases with the increase of TG levels (Table 5).

Solid and dashed lines represent the predicted values and 95% confidence intervals, adjusted for Model 3 (age, sex, hypertension, diabetes, smoking, alcohol consumption, Hs-CRP, NEU, LYC.modify, and NIHSS). Only 99% of the data is shown.

## Discussion

This study demonstrates an inverse association between TG levels and the risk of SAP, indicating that lower TG levels are associated with a higher risk of SAP. This finding might provide cues for the prediction and prevention of SAP in elderly patients with acute ischemic stroke.

Previous studies have identified several factors linked to SAP, including stroke severity, comorbidities [23, 24]. Individuals with higher NIHSS scores at admission are at relatively higher risk of SAP [25]. Consistent with the literature, our study found that age, hypertension, NIHSS, hs-CRP, and neutrophil levels were independently associated with SAP, indicating alignment with existing research.

**Table 5** Threshold effect analysis of the relationship of TG with infection

| TG (mmol/L) | Adjusted model | |
|---|---|---|
| | OR (95% CI) | *p*-value |
| < 2.6 | 0.601 (0.288 ~ 1.254) | 0.1747 |
| ≥ 2.6 | 0.013 (0 ~ Inf) | < 0.001 |
| Likelihood Ratio test | - | 0.047 |

OR, odds ratio; CI, confidence interval. Adjusted for s Model 3 (age, sex, hypertension, diabetes, smoke, alcohol drinking, Hs-CRP, NEU, LYC.modify and NIHSS). Only 99% of the data is displayed

The benefits of lipid-lowering therapies in stroke prevention and management are well-established [26–29]. Elevated TG levels indicate the presence of triglyceride-rich lipoproteins with a higher atherogenic potential [30], and they are considered an independent risk factor for ischemic stroke [31, 32]. The 2013 meta-analysis showed that high-density lipoprotein cholesterol (HDL-C) and apolipoprotein A-I (apoA-I) levels were strongly associated with reduced cardiovascular risk, even in patients with very low-density lipoprotein cholesterol levels on statin therapy [33]. Furthermore, elevated TG levels have been linked to atherosclerosis, thrombosis, and increased blood hyperviscosity, all contributing to stroke development [14]. However, the association between TG levels and the prognosis of acute ischemic stroke remains controversial [34–36]. In hemorrhagic stroke, the recognized relationship between stroke severity and dyslipidemia includes the tendency of hypocholesterolemia to hemorrhagic stroke. For instance, a meta-analysis of twelve prospective studies(476,173 participants) identified a 3% decrease in hemorrhagic stroke risk for every 10 mg/dL increase in LDL-C levels [37]. In addition, in haemorrhage after intravenous thrombolysis, lower cholesterol levels were associated with a higher risk of such haemorrhage after thrombolysis [38], and in ischaemic stroke, recent studies of acute coronary syndrome have shown that patients with low blood lipid levels on admission have worse clinical outcomes and a significantly worse long-term prognosis [15–18, 39–41]. There is also evidence of a link between lipid metabolism and the host's

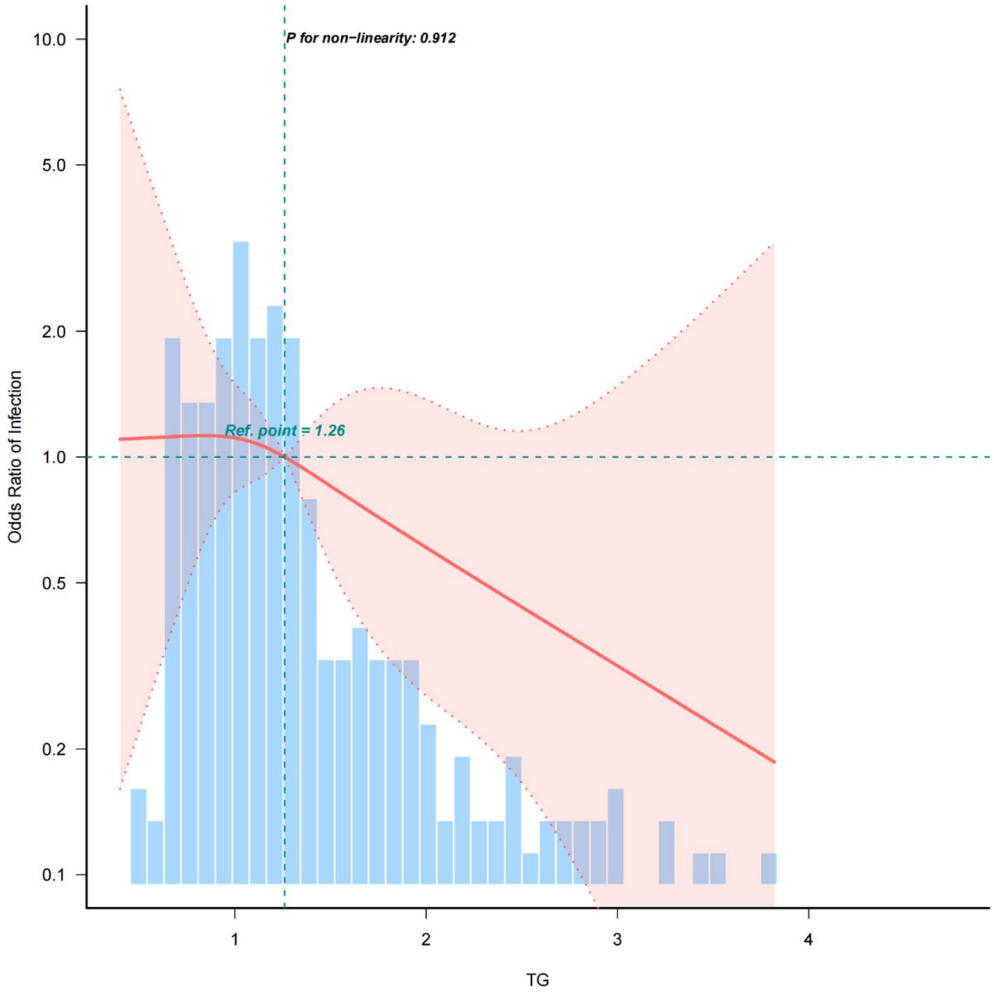


**Fig. 2** Linear relationship of TG and SAP

innate immune response to infection and acute injury [42, 43]. Low LDL-C levels have been associated with higher long-term rates of community-acquired SAP [44]. Decreased serum cholesterol is also considered a poor prognostic factor in patients with severe community-acquired SAP requiring intensive care unit admission [45]. However, the relationship between blood lipid levels and SAP is still not well understood.

In the present study, higher TG levels were found to be a protective factor associated with a reduced risk of SAP. This finding aligns with a previous retrospective cohort study ($n = 257$) that observed higher TG levels to be associated with a decreased incidence of stroke-associated infections (SAI) [46]. However, it is worth noting that the previous study was not a prospective study and the definition of SAI encompassed SAP, urinary tract infections (UTI), and other infections [46]. Additionally, Chan et al. [47] reported that hyperlipidemia was linked to a lower incidence of SAP in patients with chronic obstructive pulmonary disease (COPD). Low TG levels have also been associated with higher sepsis-related mortality [19]. In the context of the TG stroke paradox, low TG and low LDL levels during the acute phase have been shown to be significantly associated with higher 30-day and 1-year mortality in ischemic stroke patients [48]. Another study reported that elevated serum TG levels appear to be independently associated with less severe neurologic deficits in patients with acute ischemic stroke [34].

Several explanations for these differential effects are plausible. Firstly, lipid profile disorders may prime systemic innate immune responses by enhancing lipopolysaccharide induction of serum cytokines in vivo and macrophage cytokines ex vivo, thereby enhancing bacterial killing [42]. Secondly, it has been suggested that cellular accumulation of TG itself is not initially toxic; instead, the excess fatty acids accumulating in TG pools may divert these molecules from pathways that lead to cytotoxicity, thus serving as a buffer against lipotoxicity [49]. Thirdly, low lipid levels during the acute phase may be detrimental to ischemic cells experiencing acute injury, whereas low lipid levels in the stable phase could help prevent atherosclerosis progression and the occurrence of vascular events [48]. Lastly, low TG levels can reflect poor nutritional status, which may contribute to reduced resistance to injury.

This study has several limitations to consider. Firstly, it was conducted at a single study center, and the sample size was relatively small considering the high number of stroke patients. Secondly, there is a potential risk of bias due to patient selection and local policies and guidelines. Thirdly, we did not collect data on dysphagia after admission, which could potentially impact blood lipid levels. Fourthly, we did not collect data on the treatment of people with hypercholesterolemia and did not perform statistical analysis. Therefore, the timing between measurement and onset varied among patients, and changes in blood lipid levels were not assessed. Lastly, the subgroup analyses were primarily exploratory as some subgroups had small sample sizes and may lack sufficient statistical power.

## Conclusions

In conclusion, the findings of this study indicate an inverse association between blood TG levels and the risk of SAP in patients admitted for acute ischemic stroke. Specifically, lower TG levels were associated with a higher risk of SAP. However, given the small sample size, these findings could be due to chance. Further research is needed to better understand the implications of these results for implementing lipid-lowering strategies in stroke management.

### Abbreviations

| | |
|---|---|
| SAP | Stroke-associated pneumonia |
| TG | Triglycerides |
| WHO | World Health Organization |
| DALYs | Disability-adjusted life years |
| ICU | Intensive care unit |
| TC | Total cholesterol |
| HDL-C | High-density lipoprotein cholesterol |
| LDL-C | Low-density lipoprotein cholesterol |
| CT | Computed tomography |
| MRI | Magnetic resonance imaging |
| CI | Confidence interval |
| SAI | Stroke-associated infections |
| UTI | Urinary tract infections |
| COPD | Chronic obstructive pulmonary disease |

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Not applicable.

### Author contributions

Conceptualization: Wanli Dong, Jianqiang Ni; Data curation: Xiujuan Yuan; Formal analysis: Shicun Huang; Funding acquisition: Jianqiang Ni; Investigation: Xiujuan Yuan, Shicun Huang; Methodology: Xiujuan Yuan, Shicun Huang; Project administration: Wanli Dong, Jianqiang Ni; Resources: Xiujuan Yuan, Shicun Huang; Software: Shicun Huang; Supervision: Wanli Dong, Jianqiang Ni; Validation: Wanli Dong, Jianqiang Ni; Visualization: Wanli Dong, Jianqiang Ni; Writing-original draft: Xiujuan Yuan; Writing-review & editing: Wanli Dong, Jianqiang Ni.

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### Data availability

All data generated or analysed during this study are included in this published article.

## Declarations

### Ethics approval and consent to participate

The study was approved by the Ethics Committee of the First Affiliated Hospital of Soochow University (approval #2021137), and written informed consent was obtained from the patients or their relatives. I confirm that all methods were performed in accordance with the relevant guidelines. The study was carried out in accordance with the Declaration of Helsinki.

**Consent for publication**
Not applicable.

**Competing interests**
The authors declare no competing interests.

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