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ORIGINAL RESEARCH

Association Between Serum Uric Acid to High-Density Lipoprotein Cholesterol Ratio and Pneumonia After Endovascular Treatment of Vertebrobasilar Artery Occlusion

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Background: The uric acid to high-density lipoprotein cholesterol ratio (UHR) is a novel marker of inflammation and metabolism. We aimed to explore the association of UHR with pneumonia after endovascular thrombectomy (EVT) in patients with vertebrobasilar artery occlusion (VBAO).

Methods: We retrospectively enrolled participants diagnosed with acute VBAO treated with EVT within 24 hours of estimated occlusion time from the multicenter PERSIST study. The primary outcome was pneumonia within 7 days after EVT according to the Pneumonia in Stroke Consensus Group recommendations. We utilized the restricted cubic spline curve to explore the dose–response relationship between UHR and pneumonia. We used multivariable logistic regression models to assess the association between UHR and the risk of pneumonia after EVT and verified the findings in subgroup analysis.

Results: Three hundred and seventy-eight patients were enrolled in this study, and 236 (62.4%) were diagnosed with pneumonia. In multivariable models, a higher UHR was associated with an increased risk of pneumonia after EVT (odds ratio [OR], 1.05; 95% confidence interval [CI], 1.01–1.10; $P = 0.020$; tertile 3 versus tertile 1: OR, 2.09; 95% CI, 1.15–3.82; $P = 0.016$). The dose–response relationship indicated that UHR was linearly associated with the risk of pneumonia ($P = 0.888$). The association between UHR and pneumonia remained significant in different subgroups.

Conclusion: This study demonstrated that a higher UHR was associated with an increased risk of pneumonia in VABO patients treated with EVT. Further studies were warranted to verify the prognostic values of UHR in pneumonia after EVT.

Keywords: pneumonia, vertebrobasilar artery occlusion, stroke, thrombectomy, uric acid to high-density lipoprotein cholesterol ratio

Introduction

Endovascular treatment (EVT) is considered an effective therapeutic approach for acute ischemic stroke due to vertebrobasilar artery occlusion (VBAO), which accounts for 10% to 20% of large artery occlusion strokes.[1–3] Although EVT has significantly improved clinical outcomes for patients with VBAO compared with optimal medical management, approximately 50% of these patients still do not achieve functional independence after 90 days, due to factors such as advanced age, a higher 24-h NIHSS and incomplete recanalization.[4–6] Pneumonia is a frequent complication in ischemic stroke, particularly

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in those with posterior circulation stroke and dysphagia, and it is a significant factor contributing to poor clinical outcomes.[7–10] Previous studies indicated that the incidence of pneumonia in patients with VBAO was approximately 70% and was significantly associated with early mortality.[11] Hence, it is important to identify biomarkers that could predict pneumonia in VBAO patients after EVT at an early stage, facilitating the recognition of high-risk patients and enabling timely intervention.

The uric acid (UA) to high-density lipoprotein cholesterol (HDL) ratio (UHR) is a novel marker of inflammation and metabolism recently proposed in studies.[12,13] UA can activate the natural immune response by acting as damage-associated molecular patterns and initiating innate immunity.[14–16] Conversely, HDL is an active lipid in the immune response and has a protective effect on infections.[17–19] As the ratio of UA to HDL, UHR may serve as a predictive marker for pneumonia risk in stroke patients. Previous studies reported that elevated UHR was associated with a reduced risk of osteoporosis.[20] Patients with atrial fibrillation and non-alcoholic fatty liver disease exhibited higher UHR levels than healthy populations.[21] UHR was associated with insulin resistance and metabolic disorders, particularly in patients with type 2 diabetes.[22] However, few studies had evaluated the prognostic value of UHR for pneumonia after EVT. Thus, we conducted a multicenter study to investigate the hypothesis that UHR was associated with pneumonia risk in VBAO patients treated with EVT.

Methods

Data that supported the findings of this study are available from the corresponding authors upon reasonable request.

Study Population

From December 2015 to December 2018, we enrolled participants diagnosed with acute VBAO from the PERSIST study, which was a retrospective, multicenter study conducted at 21 stroke centers in 13 provinces of China (ChiCTR2000033211). Details of the PERSIST study have been described in previous studies.[23–25] We included patients according to the following criteria: (1) ≥18 years; (2) diagnosed as acute symptomatic VBAO on computed tomographic angiography (CTA), magnetic resonance angiography (MRA), or digital subtracted angiography (DSA); and (3) treated with EVT within 24 hours of estimated occlusion time. The exclusion criteria were: (1) had pre-stroke mRS score >2; (2) had anterior circulation stroke; (3) had aneurysms or arteriovenous malformation; (4) had missing UA and HDL and imaging information; (5) had chronic inflammatory diseases or autoimmune diseases; (6) pregnancy or lactation. This study was approved by the Ethics Committee of The First Affiliated Hospital of USTC. Patient consent was waived due to the retrospective nature. This study was conducted in accordance with the ethical standards for patient confidentiality and the principles outlined in the 1964 Declaration of Helsinki.

Data Collection

Demographic data, medical history, laboratory results, and imaging information were retrospectively collected from the medical records. Stroke severity was assessed with the National Institute of Health Stroke Scale (NIHSS) score. Consciousness was assessed with the Glasgow Coma Scale (GCS). Stroke subtype was classified according to the Trial of Org 10172 in Acute Stroke Treatment (TOAST) criteria. All imaging files were independently reviewed by two neurologists who were blinded to this study. The occlusion site was classified as a basilar artery, vertebral artery, and vertebrobasilar artery occlusion. Cerebral ischemia was assessed with the posterior circulation-Alberta Stroke Program Early CT Score (PC-ASPECTS). Collateral status was assessed with the American Society of Interventional and Therapeutic Neuroradiology/Society of Interventional Radiology (ASITN/SIR) score. Successful reperfusion status was defined as the modified Thrombolysis in Cerebral Infarction (mTICI) scale 2b or 3. The blood samples were collected on admission including routine blood and blood chemistry. UHR was calculated with the following equations: serum UA (mg/dL)/HDL (mg/dL).[13]

EVT

EVT was carried out using mechanical thrombectomy with stent retrievers or thromboaspiration, balloon angioplasty, intra-arterial thrombolysis, stenting, or a combination of these techniques. The selection of specific EVT techniques was determined by the neurointerventionists, guided by local protocols and the preferences of the patients or their legal representatives at each institution.

Primary Outcome

Pneumonia within 7 days after EVT was defined according to the criteria published by the Pneumonia in Stroke Consensus Group:[26] (1) at least 1 of the following criteria: a. fever (>38°C) with no other recognized cause; b. leukopenia (<4000 white blood cell/mm^3) or leukocytosis (>12000 white blood cell/mm^3); c. for adults aged ≥70 years, altered mental status with no other recognized cause; (2) at least 2 of the following criteria: a. new onset of purulent sputum, or change in character of sputum over a 24 h period, or increased respiratory secretions, or increased suctioning requirements; b. new onset or worsening cough, or dyspnea, or tachypnea (respiratory rate > 25/min); c. rales, crackles, or bronchial breath sounds; d. worsening gas exchange. (3) and ≥2 serial chest radiographs with at least 1 of the following criteria: new or progressive and persistent infiltrate, or consolidation, or cavitation. Pneumonia was then classified as community-acquired pneumonia and hospital-acquired pneumonia.

Statistical Analysis

Categorical variables were presented as numbers (percentages), and continuous variables were presented as mean ± standard deviation or median (interquartile range, [IQR]) according to the normality in the Kolmogorov–Smirnov test. Categorical variables were compared with chi-square tests or Fisher's exact tests and continuous variables were compared with *t* test or Mann–Whitney *U*-test as appropriate. Multiple imputation with chain equations was used to deal with missing values.

We categorized UHR into low (<9.97%), medium (9.97%–14.61%), and high levels (≥14.61%) by tertiles, and presented the distribution of pneumonia types across different UHR levels. We utilized the multivariable logistic regression models to evaluate the association between UHR and pneumonia after EVT in VBAO with adjustment for potential confounders. Model 1 was adjusted for age and gender. Model 2 was adjusted for age, gender, systolic blood pressure, diastolic blood pressure, hypertension, diabetes mellitus, coronary heart disease, hyperlipidemia, atrial fibrillation, smoking, and stroke types. Model 3 was adjusted for variables with P <0.10 in the univariable analysis using the backward selection method, as well as for variables previously identified as predictors of pneumonia in prior research.

We used the receiver operating characteristic curve to evaluate the predictive ability of UHR for pneumonia after EVT of VBAO. We used the restricted cubic spline curve with 4 knots (5th, 35th, 65th, and 95th percentiles) to explore the dose–response relationship between UHR and pneumonia after EVT adjusted for variables included in model 3. In a sensitivity analysis, we validated the predictive value of UHR in hospital-acquired pneumonia after EVT. We performed subgroup analyses to verify the robustness of the association between UHR and pneumonia after EVT according to age, gender, hypertension, diabetes mellitus, and intravenous thrombolysis treatment subgroups.

All statistical analyses were performed using R software version 4.2.1. (R Foundation, Vienna, Austria). A two-sided P value <0.05 was considered to be statistically significant.

Results

In our study, a total of 378 patients (mean age, 63.3 ± 12.8 years; 73.3% male) were enrolled in the PERSIST study after excluding 182 patients with missing information and 17 with chronic inflammation diseases. The baseline characteristics of patients with or without pneumonia were shown in Table 1: 236 (62.4%) patients were diagnosed with pneumonia, 296 (78.3%) were diagnosed with basilar artery occlusion alone, 77 (20.4%) received intravenous thrombolysis treatment, and 333 (88.1%) had successful recanalization. The mean NIHSS score was 22.5 points, and the median UHR value was 11.8. Our results found that elevated inflammatory markers (white blood cell count, neutrophil-to-lymphocyte ratio, UHR, and reduced high-density lipoprotein cholesterol) were significantly associated with pneumonia. Additionally, factors reflecting stroke severity (NIHSS and GCS scores), larger infarcts (indicated by ASPECTS scores), intravenous thrombolysis, poor collateral circulation, and extended reperfusion times were also identified as significant predictors of pneumonia (all P < 0.05). Meanwhile, higher UHR values were significantly associated with increased inflammatory markers, larger infarcts, poorer consciousness, and a higher risk of pneumonia (all P < 0.05; Table S1), as well as hospital-acquired pneumonia (57.9% vs 44.4%; Figure 1).

Table 1 Baseline Characteristics of Participants with or without Pneumonia After EVT for VBAO

Characteristics	Total N = 378	With Pneumonia N = 236	Without Pneumonia N = 142	*P* value
Age, years; mean ± SD	63.3 (12.8)	64.1 (12.0)	62.0 (13.9)	0.125
Male, n (%)	277 (73.3)	175 (74.2)	102 (71.8)	0.708
SBP, mmHg; mean ± SD	150.9 (24.9)	150.5 (25.9)	151.5 (23.1)	0.710
DBP, mmHg; mean ± SD	85.6 (13.8)	85.8 (14.2)	85.3 (13.2)	0.757
Medical history, n (%)				
Hypertension	259 (68.5)	159 (67.4)	100 (70.4)	0.614
Diabetes mellitus	88 (23.3)	54 (22.9)	34 (23.9)	0.912
CHD	33 (8.7)	23 (9.7)	10 (7.0)	0.475
Hyperlipidemia	171 (45.2)	104 (44.1)	67 (47.2)	0.629
Atrial fibrillation	80 (21.2)	48 (20.3)	32 (22.5)	0.707
Smoking	124 (32.8)	74 (31.4)	50 (35.2)	0.509
Stroke	74 (19.6)	44 (18.6)	30 (21.1)	0.649
Stroke types, n (%)				0.155
LAA	248 (65.6)	161 (68.2)	87 (61.3)	
CES	74 (19.6)	39 (16.5)	35 (24.6)	
Others	56 (14.8)	36 (15.3)	20 (14.1)	
Scores, median (IQR)				
NIHSS	22.5 [14.0, 28.0]	23.0 [16.0, 28.0]	20.0 [10.0, 28.8]	0.013
ASPECTS	9.0 [8.0, 10.0]	9.0 [8.0, 10.0]	9.0 [8.0, 10.0]	0.015
GCS	8.0 [6.0, 12.0]	7.0 [6.0, 11.0]	10.0 [6.0, 12.8]	0.002
Treatment parameters				
IVT, n (%)	77 (20.4)	58 (24.6)	19 (13.4)	0.013
OTP, min; mean ± SD	421.1 (332.1)	428.9 (349.6)	408.2 (301.5)	0.544
PTR, min; mean ± SD	119.7 (67.5)	131.6 (69.2)	99.9 (59.7)	<0.001
mTICI 2b-3, n (%)	333 (88.1)	205 (86.9)	128 (90.1)	0.430
Occlusion site, n (%)				0.737
BA	296 (78.3)	183 (77.5)	113 (79.6)	
BA and VA	82 (21.7)	53 (22.5)	29 (20.4)	
Collateral status, n (%)				0.001
ASITN/SIR 0–1	281 (74.3)	190 (80.5)	91 (64.1)	
ASITN/SIR 2–4	97 (25.7)	46 (19.5)	51 (35.9)	
Laboratory test				
WBC, 10^9/L, median (IQR)	10.5 [8.1, 13.4]	10.9 [8.6, 13.8]	10.0 [7.5, 13.0]	0.008
NLR, %, median (IQR)	8.0 [4.8, 12.1]	8.4 [5.3, 12.3]	7.4 [3.7, 11.4]	0.026
CRP, mmol/L, median (IQR)	11.2 [4.2, 32.4]	11.2 [3.6, 38.4]	10.6 [5.2, 25.3]	0.614
UA, μmol/L, median (IQR)	315.5 [236.0, 385.8]	323.4 [242.5, 388.3]	298.4 [232.2, 369.2]	0.254
HDL, mmol/L, median (IQR)	43.2 [36.4, 52.9]	42.0 [34.3, 51.2]	45.7 [39.9, 53.4]	0.004
UHR, %	11.8 [8.9, 15.8]	12.7 [9.1, 16.8]	11.1 [8.3, 14.6]	0.005
SICH, n (%)	20 (5.3)	14 (5.9)	6 (4.2)	0.631

Abbreviations: ASPECTS, Alberta Stroke Program Early CT Score; BA, basilar artery; CES, cardioembolic stroke; CHD, coronary heart disease; CRP, C-reactive protein; DBP, diastolic blood pressure; EVT, endovascular treatment; GCS, Glasgow Coma Scale; HDL, high-density lipoprotein cholesterol; IVT, intravenous thrombolysis treatment; LAA, large artery atherosclerosis; mTICI, modified Thrombolysis in Cerebral Infarction; NIHSS, National Institute of Health stroke scale; NLR, neutrophil to lymphocyte ratio; OTP, estimated occlusion time to groin puncture; PTR, puncture to reperfusion; SBP, systolic blood pressure; SICH, symptomatic intracranial hemorrhage; UA, uric acid; UHR, uric acid to high-density lipoprotein cholesterol ratio; VBAO, vertebrobasilar artery occlusion; VA, vertebral artery; WBC, white blood cell.

The results of the univariable logistic regression models aligned closely with the findings from the baseline characteristics analysis (Tables 1 and S2). The area under the curve (AUC) for UHR demonstrated a modest predictive ability for pneumonia risk, with a value of 0.59 (95% confidence interval [CI], 0.53–0.64; Figure S1). Multivariable logistical regression analyses found that increased UHR was associated with a higher pneumonia risk in model 1 (odds

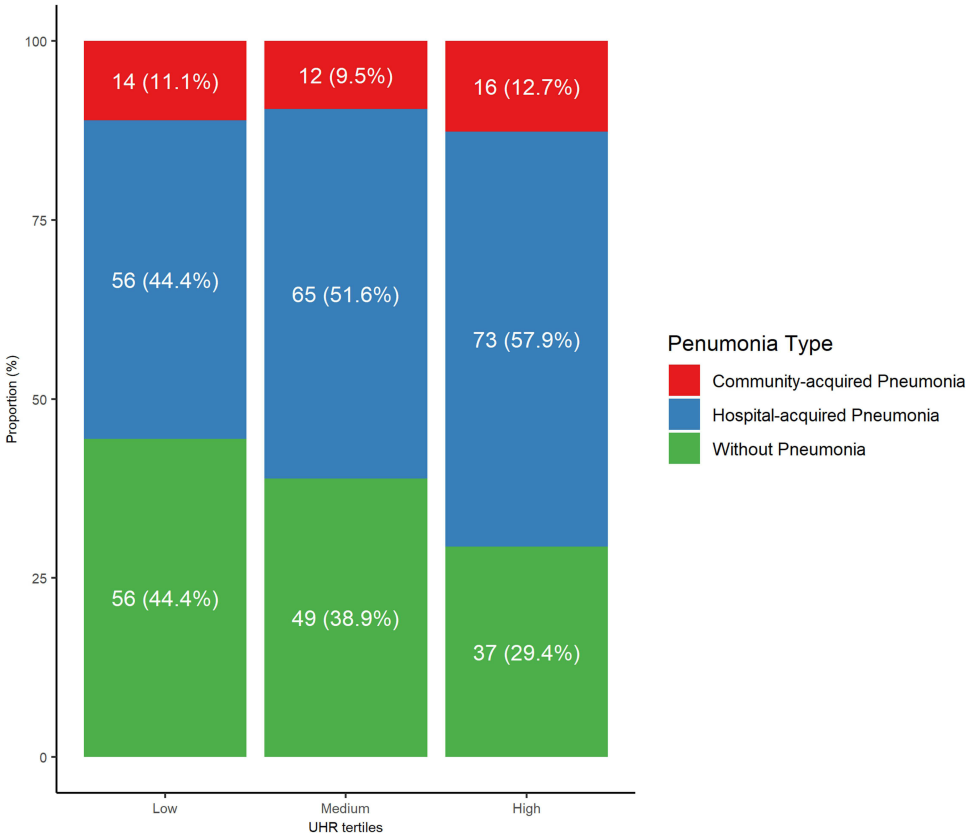

Figure 1 Distribution of pneumonia types across UHR tertiles. The distribution plot demonstrated that patients in higher UHR tertiles were more likely to suffer from pneumonia, including hospital-acquired pneumonia.
Abbreviations: UHR, uric acid to high-density lipoprotein cholesterol ratio.

ratio [OR], 1.05; 95% CI, 1.01–1.10; $P = 0.009$; tertile 3 versus tertile 1: OR, 2.04; 95% CI, 1.20–3.51; $P = 0.009$), model 2 (OR, 1.06; 95% CI, 1.02–1.11; $P = 0.004$; tertile 3 versus tertile 1: OR, 2.38; 95% CI, 1.37–4.20; $P = 0.002$), and model 3 (OR, 1.05; 95% CI, 1.01–1.10; $P = 0.020$; tertile 3 versus tertile 1: OR, 2.09; 95% CI, 1.15–3.82; $P = 0.016$; Table 2). The dose–response relationship indicated that UHR was linearly associated with the risk of pneumonia ($P = 0.888$; Figure 2).

Table 2 Association Between UHR and the Risk of Pneumonia After EVT for VBAO

UHR	Model 1		Model2		Model3	
	OR (95% CI)	***P* value**	**OR (95% CI)**	***P* value**	**OR (95% CI)**	***P* value**
Continuous	1.05 (1.01–1.10)	0.009	1.06 (1.02–1.11)	0.004	1.05 (1.01–1.10)	0.020
Tertile 1	Reference		Reference		References	
Tertile 2	1.28 (0.77–2.12)	0.347	1.37 (0.82–2.32)	0.232	1.46 (0.84–2.58)	0.185
Tertile 3	2.04 (1.20–3.51)	0.009	2.38 (1.37–4.20)	0.002	2.09 (1.15–3.82)	0.016
P for trend	0.009		0.003		0.016	

Notes: Model 1 was adjusted for age and gender; model 2 was adjusted for age, gender, SBP, DBP, hypertension, diabetes mellitus, CHD, hyperlipidemia, atrial fibrillation, smoking and stroke types; model 3 was adjusted for age, gender, ASPECTS, NIHSS, hypertension, diabetes mellitus, atrial fibrillation, OTP, PTR, IVT, collateral status, WBC, NLR, CRP, and smoking.
Abbreviations: ASPECTS; Alberta Stroke Program Early CT Score; CI, confidence interval; CHD, coronary heart disease; CRP, C-reactive protein; DBP, diastolic blood pressure; EVT, endovascular treatment; IVT, intravenous thrombolysis treatment; NIHSS, National Institute of Health stroke scale; NLR, neutrophil to lymphocyte ratio; OTP, estimated occlusion time to groin puncture; OR, odds ratio; PTR, puncture to reperfusion; SBP, systolic blood pressure; UHR, uric acid to high-density lipoprotein cholesterol ratio; VBAO, vertebrobasilar artery occlusion; WBC, white blood cell.

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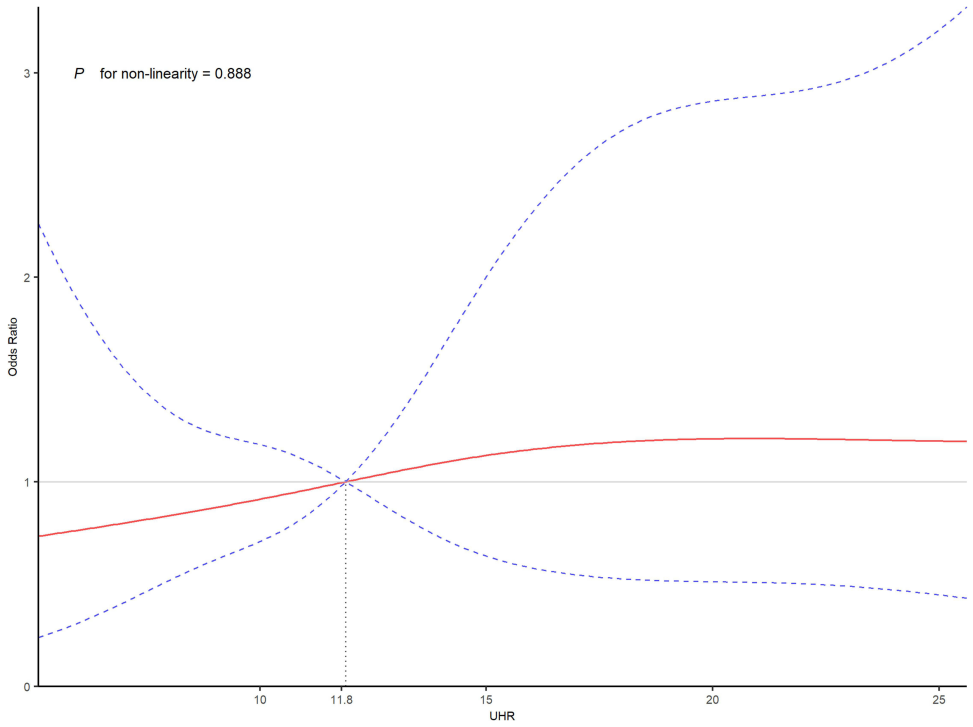

Figure 2 Dose–response relationship between UHR and the risk of pneumonia after EVT for VBAO. The dose–response relationship indicated that UHR was linearly associated with the risk of pneumonia (P = 0.888). OR was drawn in red solid lines, and the 95% CI was drawn in blue dashed lines.
Abbreviations: CI, confidence interval; EVT, endovascular treatment; OR, odds ratio; UHR, uric acid to high-density lipoprotein cholesterol ratio; VBAO, vertebrobasilar artery occlusion.

In sensitivity analyses, UHR was significantly associated with hospital-acquired pneumonia in model 1 (OR, 1.06; 95% CI, 1.02–1.10; P = 0.003; tertile 3 versus tertile 1: OR, 1.75; 95% CI, 1.05–2.93; P = 0.031), model 2 (OR, 1.07; 95% CI, 1.03–1.11; P = 0.001; tertile 3 versus tertile 1: OR, 1.99; 95% CI, 1.18–3.41; P = 0.011), and model 3 (OR, 1.06; 95% CI, 1.02–1.10; P = 0.008; tertile 3 versus tertile 1: OR, 1.74; 95% CI, 1.00–3.05; P = 0.051; Table S3). In addition, there was no significant interaction between UHR and pneumonia risk across different subgroups stratified by gender, hypertension, diabetes mellitus, and intravenous thrombolysis treatment (P > 0.05), except for the older age (P = 0.041; Figure 3).

Discussion

In this study, we found that about 62% of patients with VBAO developed pneumonia after EVT. The dose- response relationship curve suggested that a higher UHR was associated with an increased risk of pneumonia after EVT. The association of UHR and pneumonia risk remained significant in different subgroups and pneumonia types.

Pneumonia is a preventable complication associated with poor outcomes in patients with posterior circulation stroke. In our study, 62% of VBAO patients had pneumonia after EVT. In the Endovascular Treatment for Acute Basilar Artery Occlusion Study Registry, pneumonia occurred in approximately 70.7% of patients and was an independent predictor for early mortality.[11] The Berlin Stroke Register found that pneumonia was associated with mortality beyond 7 days and suggested that prophylactic antibiotics in high-risk patients might reduce early mortality.[9]

Early dysphagia screening can improve the detection rate of pneumonia.[27] VBAO patients were more likely to suffer from dysphagia compared to anterior circulation stroke.[28] Instrumental assessments, like fiberoptic endoscopic evaluation of swallowing and videofluoroscopy, were considered the gold standard for diagnosing dysphagia.[29] However, these tests required specialized staff and equipments. Previous studies suggested that the water swallow test may be a simpler method for identifying dysphagia in the hyperacute stroke.[30]

UHR is a novel biomarker for inflammation and metabolic diseases, easily calculated from routine clinical measures of UA and HDL. Previous studies suggested that UHR had been widely used in different diseases, such as poorly

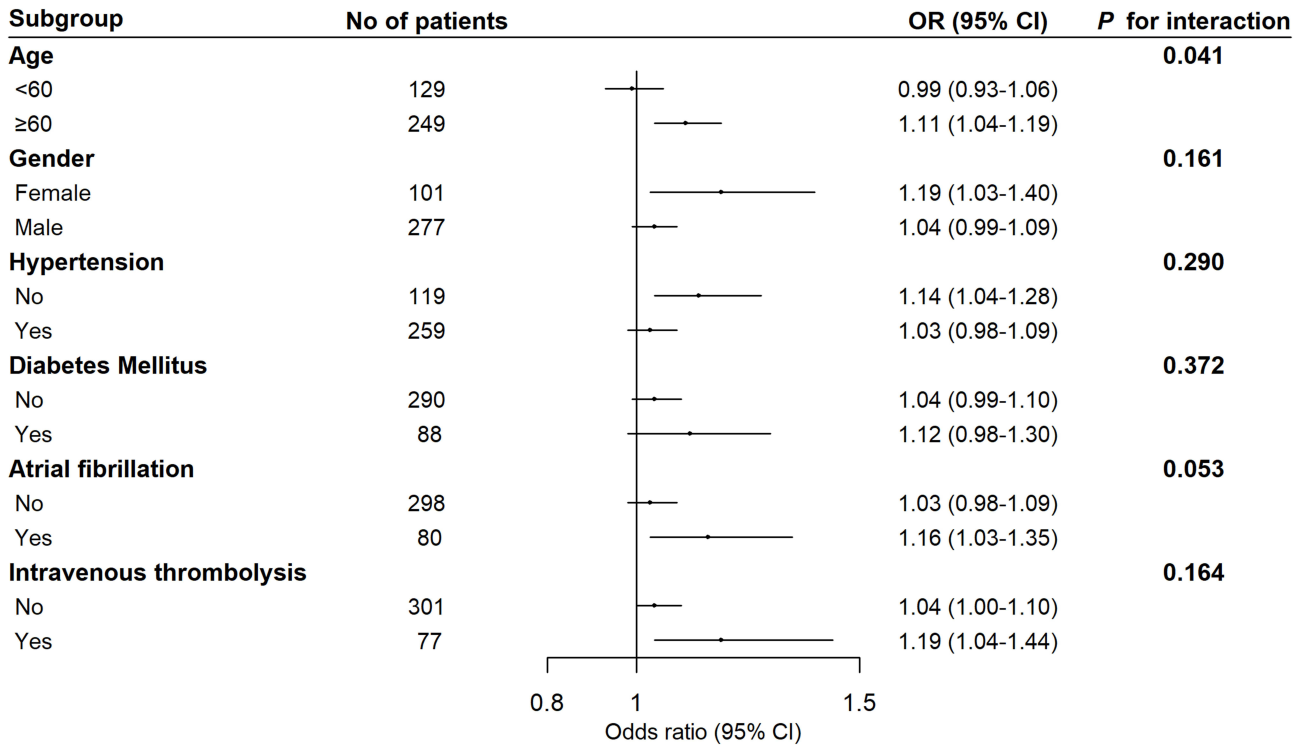

Figure 3 Subgroup analyses for UHR and of pneumonia after EVT for VBAO. There was no significant interaction between UHR and pneumonia risk across different subgroups stratified by gender, hypertension, diabetes mellitus, and intravenous thrombolysis treatment, except for the older age (P = 0.041).
Abbreviations: CI, confidence interval; EVT, endovascular treatment; OR, odds ratio; UHR, uric acid to high-density lipoprotein cholesterol ratio; VBAO, vertebrobasilar artery occlusion.

controlled hypertension, diabetes mellitus, and atrial fibrillation. In subgroup analyses, we found that UHR was significantly associated with pneumonia in patients with hypertension and atrial fibrillation. In a cross-sectional study, serum UHR was positively correlated with hypertension after adjusting for covariates, suggesting a link with hypertension through the pathophysiology of hypertension, including inflammation, oxidative damage, and endothelial dysfunction.[12,31] UHR levels were reported to be higher in patients with atrial fibrillation and non-alcoholic fatty liver disease compared to the control group, and when combined with age, it could improve the predictive ability for atrial fibrillation.[21] Another study found that UHR was positively associated with visceral fat in patients with type 2 diabetes and may reflect insulin resistance and metabolic disorders.[22]

Inflammatory markers have proven helpful in identifying high-risk patients, with previous studies confirming their predictive value for pneumonia after ischemic stroke. Hotter et al conducted a pooled analysis of 2 acute stroke studies and found that ultrasensitive copeptin and ultrasensitive procalcitonin were associated with functional outcomes at 3 months.[32] In a retrospective analysis, the neutrophil-to-lymphocyte ratio was found to be associated with an increased risk of severe pneumonia, as assessed by the Pneumonia Severity Index.[33] We found that a higher UHR was associated with an increased risk of pneumonia. Furthermore, when combined with recanalization status, neurological deterioration, and hemorrhagic transformation after EVT, UHR may provide a more robust prediction of outcomes and help identify VBAO patients who are more likely to benefit from more aggressive antibiotic therapy after EVT.[34]

The underlying mechanism regarding the association between UHR and pneumonia might be explained by the combined effects of elevated UA and decreased HDL levels. UA could decrease insulin-induced nitric oxide synthesis and activate the renin-angiotensin system of endothelial cells,[35] resulting in enhancing the proliferation of smooth muscle cells by mediating inflammation,[36] promoting the production of free radical species,[37] and suppressing oxidative stress reactions.[38] Meanwhile, UA at scale concentrations can help protect the endothelium against oxidative stress by acting as a selective free radical scavenger and functioning as an antioxidant.[39] HDL can reverse cholesterol transport, which exhibits anti-oxidation and anti-inflammation properties and protects endothelial cells from atherosclerotic burdens.[40,41]

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Hence, a high serum UA level and low HDL level may synergistically have detrimental effects on pneumonia after ischemic stroke.

To the best of our knowledge, this was the first study to investigate the association between UHR and pneumonia after EVT in VBAO patients. However, there were several limitations of this study. Firstly, this was a multi-center study conducted in China, introducing potential biases due to variations in the experiences and treatment strategies among the different centers. Secondly, due to the retrospective nature of this study, we were unable to collect preoperative information on systemic infections nor dynamically monitor changes in inflammatory indicators. This limitation may introduce confounding effects on pneumonia risk, as it hindered the ability to account for pre-existing infections as potential contributors and restricted the predictive ability of serial measurement of UHR. Third, we could not investigate other potentially confounding factors, such as calcium, and volume status, as they were not routinely assessed in patients treated with EVT. Fourth, although we used multiple imputation to handle missing data, the potential influence of missing values remained a significant source of bias. The sample size for subgroup analyses might have been insufficient to yield reliable results, and these findings should be interpreted with caution. Finally, the AUC for UHR in our study was relatively low, which might indicate a limited predictive capability.

Conclusion

In conclusion, our study revealed that approximately 62% of VBAO patients developed pneumonia following EVT. Elevated UHR levels were significantly associated with an increased risk of pneumonia after the procedure. Factors such as heightened inflammatory markers, larger infarcts, and reduced consciousness appeared to strongly correlate with UHR in influencing pneumonia risk. These findings highlight the potential utility of UHR as a biomarker for identifying high-risk VBAO patients, aiding in early screening and informing therapeutic interventions for pneumonia post-EVT. Further research is necessary to validate the prognostic role of UHR in outcomes of pneumonia after EVT in VBAO patients.

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Disclosure

The authors report no conflicts of interest in this work.

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