Platelet-to-Lymphocyte Ratio as a New Predictive Index of Neurological Outcomes in Patients with Acute Intracranial Hemorrhage: A Retrospective Study

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Background: Systemic inflammation plays a critical role in the pathophysiological process of intracranial hemorrhage (ICH). Recently, the platelet-to-lymphocyte ratio (PLR) has become a research focus that indicates inflammation in various diseases. Thus, this study aimed to investigate the predictive value of PLR in patients with acute ICH.

Material/Methods: This study was performed in a single teaching hospital. Glasgow coma scale at hospital discharge (GCS\text{dis}) and modified Rankin score (MRS) at 6 months were recorded as short-term and long-term neurological outcomes. Ordered and binary logistic regression methods were used to explore the associations.

Results: Finally, data on 183 ICH patients were included. A knot of PLR around 100 was detected and applied in the extended ordered logistic regression models. For PLR >100, PLR on ICU admission was significantly associated with worse GCS\text{dis} (from Model 1: OR: 1.004, 95% CI 1.001–1.007 to Model 4: OR: 1.006, 95% CI 1.002–1.009) while the PLR on Emergency Department (ED) admission was insignificant. For PLR \leq 100, neither the PLR on ICU or ED admission was associated with GCS\text{dis} level. In the quartile grouping analysis, PLR Q2 was used as a reference level. Both Q3 and Q4 on ICU admission were significantly associated with lower GCS\text{dis} level (OR, 3.30; 95%CI 1.38–7.88; and OR, 3.79; 95%CI 1.54–9.33, respectively), while Q1 was insignificant. All 4 quartiles of PLR on ED admission were not associated with GCS\text{dis}.

Conclusions: Only higher PLR value on ICU admission but not on ED admission was associated with worse GCS\text{dis}.

MeSH Keywords: Glasgow Coma Scale • Intracranial Hemorrhages • Lymphocyte Count • Platelet Count
Background

Intracranial hemorrhage (ICH) is a common subtype of stroke and is associated with extremely high morbidity and mortality. Efforts have been made to explore appropriate predictive factors for poor outcomes, including neurological outcomes and mortality, such as hyperglycemia [1], serum uric acid [2] and C-reactive protein level [3]. However, challenges remain. Studies have demonstrated that inflammatory system activation was one of the major pathological pathways contributing to ICH-induced secondary brain injury [4], such as brain edema formation [5] and hematoma enlargement [6]. Thus, identifying inflammatory indexes that can predict the prognosis of ICH patients has become a research focus.

Recently, the platelet-to-lymphocyte ratio (PLR) has emerged as a prognostic marker of inflammatory response in various conditions, such as acute pulmonary embolism [7], myocardial infarction [8] and various cancer [9]. However, the predictive value of PLR in patients with acute ICH has not been investigated. Thus, we performed this study to evaluate the prognostic value of PLR in predicting neurological outcomes in patients with acute ICH.

Material and Methods

Study population

This retrospective observational study was performed in a 20-bed Intensive Care Unit (ICU) of Dongyang People’s Hospital, a tertiary teaching hospital of Wenzhou Medical University. Patients diagnosed with acute ICH who were admitted to the ICU after intracranial surgery from January 2016 to June 2017 were initially screened. Inclusion criteria were: 1) older than 18 years; 2) Glasgow coma scale (GCS) ≥4 and ≤12 on admission; and 3), emergency intracranial surgery was performed within 24 h after onset of ICH. Patients who were pregnant, lacked sufficient information for PLR calculation, or were diagnosed with aneurysmal subarachnoid hemorrhage were also excluded from this study. All patients received standard treatment according to management guidelines [10,11] and were followed up through telephone interview. Modified Rankin score (MRS) at 6 months after hospital discharge was recorded. The study was approved by the Ethics Committee of Dongyang People’s Hospital and informed consent was waived due to the retrospective study design.

Data extraction

Demographic data of the included patients were extracted from the electronic medical records system. Clinical data were recorded within 24 h after ICU admission, including routine blood tests, serum biochemical indexes, comorbidities, bleeding sites, and Acute Physiology and Chronic Health Evaluation II (APACHE II) score, were recorded on ICU admission. Clinical outcomes, including GCS at hospital discharge, MRS at 6 months after hospital discharge, ICU length of stay, and ventilation duration, were also recorded.

Outcome definition

The primary endpoint was GCS at hospital discharge (GCS_{hospital}) and was divided into 3 levels: level 1: 3~8, level 2: 9~12, and level 3: 13~15. MRS at 6 months was also divided into 2 levels: favorable (0~2) and unfavorable (3~6). If a patient died within 6 months, the MRS at 6 months was recorded as 6.

Grouping methods for PLR in logistic models.

As a knot of PLR (around 100) was detected using Lowess smoother technique, linear spline function (PLR ≤100 and PLR >100) was initially applied in ordered logistic regression models. For better interpretation, quartile grouping method of PLR was also used in multivariate logistic regression models, using PLR quartile 2 as the reference level.

Missing data management

The percentages of most missing variables were less than 3% and were simply replaced by the mean value. More than 20% of C-reactive protein was missing and was not imputed.

Statistical analysis

Continuous data were expressed as mean ±SD or median (interquartile range). The variance analysis or Kruskal–Wallis test was used as appropriate. Categorical data were expressed as percent and compared using the chi² test. GCS_{hospital} was divided into 3 levels (level 1: 3~8, level 2: 9~12, and level 3: 13~15) and ordered logistic regression method was applied. MRS was divided into 2 levels (favorable (0~2) and unfavorable (3~6)), and ordinary logistic regression was applied. Grouping methods of PLR were described in the method section. As we aimed at adjusting for potential confounding factors, either clinically important or statistically significant variables were including in the logistic models. In order to achieve a robust conclusion, an extended model approach was used and is presented in Table 1: Model 2=Model 1+age, diabetes mellitus, hypertension. Model 3=Model 2+(bleeding sites). Model 4=Model 3+(serum sodium, hospital acquired pneumonia, APACHE II score on ICU admission). Multi-collinearity was tested using variance inflation factor (VIF) method, with VIF ≥5 indicating multi-collinearity existence. The two-tailed test was used, and p<0.05 was considered statistically significant. All statistical analyses were performed using STATA 11.2 software (College Station, TX, USA).
Table 1. Extended ordered logistic regressions of PLRs on ER/ICU admission using linear spline function.

| ER admission | PLR (≤100) | PLR (>100) | ICU admission | PLR (≤100) | PLR (>100) |
|--------------|------------|------------|---------------|------------|------------|
|              | OR (95% CI) |       p    | OR (95% CI)   |       p    | OR (95% CI) |       p    |
| Model 1      | 1.007      | 0.306     | 0.99          | 0.429      |           |           |
|              | (0.99–1.02)|           | (0.98–1.00)   |           |           |           |
| Model 2      | 1.005      | 0.448     | 0.99          | 0.490      |           |           |
|              | (0.99–1.02)|           | (0.99–1.00)   |           |           |           |
| Model 3      | 1.001      | 0.417     | 0.99          | 0.583      |           |           |
|              | (0.99–1.02)|           | (0.99–1.00)   |           |           |           |
| Model 4      | 1.005      | 0.450     | 0.99          | 0.879      |           |           |
|              | (0.99–1.02)|           | (0.99–1.00)   |           |           |           |

Ordered GCS scores (level one: 3–8, level two: 9–12, level three: 13–15) at hospital discharge was used as the dependent variable in all logistic models. Different associations between GCS score and PLR on ER/ICU admission were explored. Crude odds ratio was listed in model 1. Adjusted covariates: Model 2 = age, diabetes mellitus, hypertension. Model 3 = Model 2 + (bleeding sites). Model 4 = Model 3 + (serum sodium, hospital acquired pneumonia, APACHE II score on ICU admission). GCS – Glasgow Coma Scale; PLR – platelet to lymphocyte ratio; ER – Emergency Room; ICU – Intensive Care Unit; OR – odds ratio; CI – confidence interval.

Results

Baseline characteristics

Finally, data on 183 patients with acute ICH were included in this study after screening (Figure 1), and the demographic characteristics within 3 GCS levels are compared in Table 2. Patients with high GCS (59.9±15.1 vs. 51.0±14.8 vs. 48.5±13.7, p<0.001). Compared to patients with low GCS, the PLR on ICU admission (291.0±363.9 vs. 177.3±103.5 vs. 165.5±83.4, p=0.002) but not the PLR on ED admission (137.5±91.3 vs. 147.3±144.1, p=0.794) was significantly lower in patients with high GCS level. However, the crude comparisons of platelet count and lymphocyte count on ED or ICU admission were insignificant.

Crude comparisons within 4 PLR quartiles

Platelet count was significantly increased (from Q1 128.7±50.7 to Q4 190.0±51.7, p<0.001) while lymphocyte count was decreased stepwise (from Q1 1.97±0.07 to Q4 0.56±0.21, p<0.001) with increasing quartiles of PLR in Table 3. In this crude comparison, PLR Q2 was associated with highest GCS (13.10–15) and lowest percent (n=21 (45.6%)) of patients with MRS ≥3.

Association between PLR and short-term GCS

A knot around 100 of PLR on ICU admission was detected in the Lowess smoother curve between PLR and GCS in Figure 2. Thus, in the extended ordered logistic models (Table 1), linear spline function was applied using the cut-off value of 100. We found that for PLR >100, only PLR value on ICU admission was significantly associated with worse GCS level (from Model 1: OR: 1.004, 95% CI 1.001–1.007 to Model 4: OR: 1.006, 95% CI 1.002–1.009) while the PLR value on ED admission was insignificant in all 4 extended logistic models. For PLR ≤100, neither the PLR on ICU admission nor the PLR on ED admission were associated with GCS level. For better interpretation, quartile method was also applied for PLR and Q2 was used as the reference level (Table 4). The conclusion was similar. High PLR (Q3 and Q4) level on ICU admission was significantly associated with lower GCS level (OR: 3.30; 95%CI 1.38–7.88; and OR: 3.79; 95%CI 1.54–9.33, respectively) while low PLR (Q1) was insignificant. None of the 4 quartiles of PLR on ED admission were associated with GCS level.

Figure 1. Flow chart of patient selection.
## Table 2. Baseline characteristic comparisons within three GCS categories at hospital discharge.

| Variables                          | 3≤ GCS ≤8 (n=48) | 9≤ GCS ≤12 (n=69) | 13≤ GCS ≤15 (n=66) | P  |
|-----------------------------------|------------------|-------------------|-------------------|----|
| **Age (years)**                   | 59.9±15.1        | 51.0±14.8         | 48.5±13.7         | < 0.001 |
| **Gender (male) [n (%)]**         | 28 (58.3)        | 44 (63.7)         | 45 (68.1)         | 0.557  |
| **Alcohol drinking [n (%)]**      | 4 (8.3)          | 14 (29.1)         | 18 (26.1)         | 0.042  |
| **Smoking [n %]**                  | 12 (25.0)        | 18 (26.1)         | 19 (27.5)         | 0.891  |
| **Comorbidities**                 |                  |                   |                   |      |
| **Hypertension [n (%)]**          | 29 (50.0)        | 34 (49.2)         | 27 (39.1)         | 0.121  |
| **Diabetes mellitus [n (%)]**     | 2 (4.1)          | 3 (4.3)           | 3 (4.5)           | 0.995  |
| **Cardiac disease [n (%)]**       | 3 (6.2)          | 3 (4.3)           | 4 (6.1)           | 0.874  |
| **Kidney disease [n (%)]**        | 6 (12.5)         | 5 (7.2)           | 3 (4.5)           | 0.284  |
| **Liver disease [n %]**           | 1 (2.1)          | 11 (15.9)         | 8 (12.1)          | 0.057  |
| **Bleeding sites**                |                  |                   |                   |      |
| **Basalganglia [n (%)]**          | 11 (22.9)        | 21 (30.4)         | 16 (24.2)         | 0.595  |
| **Frontal lobe [n (%)]**          | 15 (31.2)        | 22 (31.8)         | 16 (24.2)         | 0.570  |
| **Parietal lobe [n (%)]**         | 21 (43.7)        | 9 (13.0)          | 9 (13.7)          | 0.070  |
| **Temporal lobe [n (%)]**         | 20 (41.6)        | 25 (36.2)         | 19 (28.7)         | 0.349  |
| **Epidural hemorrhage [n (%)]**   | 3 (6.2)          | 8 (11.6)          | 11 (16.7)         | 0.238  |
| **Subdural hemorrhage [n (%)]**   | 15 (31.2)        | 14 (20.2)         | 9 (13.6)          | 0.072  |
| **Trauma [n (%)]**                | 25 (52.1)        | 36 (52.1)         | 32 (48.5)         | 0.893  |
| **Blood loss during surgery (ml)**| 230.4±201.8       | 211.9±147.3       | 202.9±202.7       | 0.730  |
| **Fluid input/output**            |                  |                   |                   |      |
| **Fluid intake (ml/24 hr)**       | 4527±2750         | 3873±1783         | 3935±1712         | 0.195  |
| **Fluid balance (ml/24 hr)**      | 715±2298          | 518±1780          | 310±1736          | 0.533  |
| **Disease severity scores**       |                  |                   |                   |      |
| **APACHE II on ICU admission [median (IQR)]** | 23 (20–27)       | 18 (16–21)        | 16 (12–19)        | < 0.001 |
| **GCS on admission [median (IQR)]** | 5 (4–7)          | 7 (6–9)           | 10 (8–12)         | < 0.001 |
| **Outcomes on ER admission**      |                  |                   |                   |      |
| **Onset duration on ER admission (hour)** | 2.2±2.9          | 3.2±4.2           | 3.1±4.1           | 0.366  |
| **Platelet count (*10^9/L)**       | 208.7±48.4        | 207.2±72.9        | 240.0±21.4        | 0.334  |
| **Lymphocyte count (*10^9/L)**     | 3.36±2.05         | 2.15±1.60         | 2.46±1.69         | 0.529  |
| **PLR**                            | 137.5±91.3        | 154.2±140.3       | 147.3±144.1       | 0.794  |
| **C-reactive protein (mg/L)**      | 9.1±22.5 (n=27)   | 18.9±43.7 (n=48)  | 18.1±33.7 (n=36)  | 0.499  |
| **Outcomes on ICU admission**      |                  |                   |                   |      |
| **Onset duration on ER admission (hour)** | 2.2±2.9          | 3.2±4.2           | 3.1±4.1           | 0.366  |
| **Platelet count (*10^9/L)**       | 153.9±61.8        | 161.2±52.9        | 167.3±56.7        | 0.461  |
| **Lymphocyte count (*10^9/L)**     | 0.91±0.91         | 1.24±0.84         | 1.20±0.59         | 0.064  |
| **PLR**                            | 291.0±363.9       | 177.3±103.5       | 165.5±83.4        | 0.002  |
| **C-reactive protein (mg/L)**      | 84.9±50.9 (n=34)  | 105.0±53.1 (n=44) | 76.1±52.2 (n=47)  | 0.030  |

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We found a similar but insignificant pattern between PLR quartiles on ICU admission and MRS, shown in Supplementary Table 1.

Discussion

The major findings of our study are that in patients with ICH, only high PLR value (>100) on ICU admission was significantly associated with worse GCS, while low PLR (≤100) was insignificant. A similar but insignificant pattern between PLR on ICU admission and MRS at 6 months after discharge was also found. However, the high and low PLR values on ED admission were not associated with GCS or MRS. Further studies are needed to validate this relationship.

Table 3. Comparisons of clinical outcomes within four PLR quartiles on ICU admission.

| Variables                        | PLR Q1 (n=46) | PLR Q2 (n=46) | PLR Q3 (n=46) | PLR Q4 (n=45) | p     |
|----------------------------------|---------------|---------------|---------------|---------------|-------|
| Platelet count (*10^9/L)         | 128.7±50.7    | 154.5±50.8    | 173.5±55.7    | 190.0±51.7    | <0.001|
| Lymphocyte count (*10^9/L)       | 1.97±1.07     | 1.14±0.16     | 0.88±0.31     | 0.56±0.21     | <0.001|
| GCS at hospital discharge [median (IQR)] | 12 (9–13) | 13 (10–15) | 11 (8–13) | 10 (7–13) | 0.016 |
| MRS ≥3 [n (%)]                   | 25 (54.3)     | 21 (45.6)     | 25 (54.3)     | 26 (57.54)    | 0.688 |
| ICU length of stay (days)        | 10.1±9.4      | 9.7±8.6       | 9.5±8.5       | 11.0±10.5     | 0.860 |

PLR – platelet to lymphocyte ratio; ICU – Intensive Care Unit; GCS – Glasgow Coma Scale; MRS – Modified Rankin Scale; IQR – interquartile range.

Association between 4 PLR quartiles and long-term MRS

We found a similar but insignificant pattern between PLR quartiles on ICU admission and MRS, shown in Supplementary Table 1.

Figure 2. Crude relationship between PLRs on ED/ICU admission and GCS at hospital discharge. GCS was used as a dichotomous variable in Figure 1 (GCS ≤8 and GCS >8).
Table 4. Ordered logistic regressions of PLR on ER/ICU admission using quartile method.

| Variables          | ER admission | ICU admission |
|--------------------|--------------|---------------|
|                    | Adjusted odds ratio | 95% CI | p     | Adjusted odds ratio | 95% CI | p     |
| PLR Q1             | 0.72         | 0.31–1.67     | 0.459 | PLR Q1             | 1.21     | 0.51–2.28 | 0.657|
| PLR Q2             | Ref.         | –             | –     | PLR Q2             | Ref.     | –             | –     |
| PLR Q3             | 1.37         | 0.59–3.15     | 0.457 | PLR Q3             | 3.30     | 1.38–7.88 | 0.001|
| PLR Q4             | 0.77         | 0.33–1.77     | 0.541 | PLR Q4             | 3.79     | 1.54–9.33 | 0.004|
| Age (>65)          | 1.56         | 0.69–3.53     | 0.282 | Age (>65)          | 1.76     | 0.78–4.00 | 0.171|
| Diabetes mellitus  | 0.71         | 0.16–3.16     | 0.656 | Diabetes mellitus  | 0.65     | 0.14–2.93 | 0.583|
| Hypertension       | 0.99         | 0.52–1.88     | 0.977 | Hypertension       | 0.99     | 0.52–1.88 | 0.977|
| Extrudal hemorrhage| 0.47         | 0.17–1.27     | 0.141 | Extrudal hemorrhage| 0.42     | 0.15–1.16 | 0.097|
| Subdural hemorrhage| 2.31         | 1.05–5.12     | 0.037 | Subdural hemorrhage| 2.38     | 1.07–5.27 | 0.032|
| Serum sodium >140 mmol/L | 1.78     | 0.90–3.51     | 0.096 | Serum sodium >140 mmol/L | 2.02     | 1.00–4.05 | 0.047|
| Hospital acquired pneumonia | 1.73     | 0.96–3.14     | 0.067 | Hospital acquired pneumonia | 1.98     | 1.08–3.62 | 0.027|
| APACHE II score    | 1.19         | 1.11–1.27     | <0.001| APACHE II score    | 1.21     | 1.13–1.29 | <0.001|

Ordered GCS scores (level one: 3–8, level two: 9–12, level three: 13–15) at hospital discharge was used as the dependent variable in two logistic models. PLRs on ER/ICU admission were divided into four quartiles, and the second quartile was used as the reference level. PLR – platelet to lymphocyte ratio; ER – Emergency Room; ICU – Intensive Care Unit; CI – confidence interval; APACHE – acute physiology and chronic health evaluation.

Experimental researchers have indicated that inflammatory response plays a pivotal role in the pathophysiological process of ICH. A series of complex inflammatory responses are activated after the onset of hemorrhage, such as activation of microglia [12], increased secretion of cytokine, and infiltration of neutrophils and macrophages in the injury sites [13,14], which lead to edema progression, cell death, and permanent neurological damage. A clinical study also found that in ICH patients, inflammatory response was triggered and was associated with poor outcomes such as hematoma enlargement [6]. Thus, identifying new inflammatory indexes that predict the prognosis of ICH patients has become a research focus.

Increased evidence shows that PLR is a novel inflammatory indicator in many disorders, such as atherosclerosis [15], acute kidney injury [16], and cancers [17,18]. Yang et al. reported that high PLR level (>260 vs. <260) was an independent predictor of venous thromboembolism in patients with cancer, and Cetin et al. found that high PLR (>151 vs. <151) was associated with increased long-term major adverse cardiovascular events in patients with myocardial infarction. Another study [7] included 646 patients with acute pulmonary embolism, and reported that high PLR level (>149 vs. <149) was significantly associated with high simplified pulmonary embolism severity index score, which was directly related with high hospital mortality. However, due to the heterogeneity within different cohorts, the cut-off values were largely different in all these studies, which limit their application in other cohorts. Furthermore, in most studies the PLR was converted into a dichotomous variable using the respective cut-off value, which to a certain degree weakened the statistical efficiency. As different correlation trends were detected on the 2 sides of the cut-off value in the present study (Figure 1), simply using the low PLR as the reference level decreased the power to detect the true association between low PLR and clinical outcomes. To address this limitation, PLR value was used as a continuous variable by applying linear spline function in logistic models. A “converse-U”-shaped relationship was initially detected; however, only high PLR level was significantly associated with low GCS₉₀, while low PLR was insignificant after adjusting for confounding factors. Although the predictive value of PLR is widely reported in various diseases, the mechanism is largely unknown. We noticed that platelet counts increased stepwise and the lymphocyte counts decreased with increasing PLR quartiles. Studies have confirmed that platelets play a critical role in immunomodulatory and inflammatory processes [19,20] by inducing the release of inflammatory mediators.
cytokines [21] and interacting with various cells, including neutrophils, T lymphocytes, and macrophage, which contribute to the initiation or exacerbation of the inflammatory process [22]. Thus, high PLT may reflect the aggravated release of cytokines and increased thrombocyte activation, which lead to devastating inflammatory response. It was also reported that in patients with ICH, high PLT can predict elevated perihematomal edema and is associated with poor discharge outcome [23]. Furthermore, results published by Lattanzi et al. demonstrated that low lymphocyte count predicted worse 3-month outcome after ICH [24]. On the other hand, although PLT and lymphocyte were reported as predictive indexes in previous studies, we did not detect any statistical significance in the crude comparisons of platelet count and lymphocyte count within 3 GCSlevels. Thus, we speculated that PLR was superior to PLT or lymphocyte count alone in the prediction of neurological outcomes and may more accurately indicate a high level of inflammatory reaction [25] in ICH patients.

We also noticed that PLR on ED admission was not associated GCS or MRS at 6 months. Unlike other reported diseases such as hepatocellular carcinoma, acute kidney injury, and pneumonia [16,26,27], ICH is an acute severe disease and we noticed that the time interval between symptom onset and ED admission was quite short (2–3 h, Table 2). Thus, it was reasonable that both the PLR and the C-reactive protein (a common inflammation indicator) on ED admission were not significantly increased, as the inflammatory response may not be aggravated.

Based on these findings, PLR on ICU admission can be used as a prognostic indicator to identify patients who are at higher risk of poor neurological outcomes. In patients with high PLR, more intensive monitoring and rigorous prognosis evaluation may be arranged. On the other hand, as high PLR indicated strong inflammatory response, it is unclear if anti-inflammatory strategies would be beneficial or if this index could be used as an indicator of the efficacy of therapies.

Several limitations to our study should be noticed. First, this study was performed in a single teaching hospital, and the sample size was relatively small, which may limit the statistical power to detect a significant difference in long-term MRS. Second, as the PLR value was used as a continuous variable in the logistic regression model using linear spline function, the OR was small (1.004 to 1.006). However, this only represents the odds ratio based on 1 unit change of PLR (95% interval: 58–390). For better interpretation, quartile grouping method was also used and is shown in Table 4. However, as the prognosis of patients with ICH was affected by many confounding factors, it is difficult to predict clinical outcomes based on a single index. We hope that our findings will add to the prognostic tools for ICH in future studies.

**Conclusions**

In patients with ICH, high PLR value on ICU admission but not the value on ED admission was significantly associated with short-term neurological outcome. However, this prediction for the long-term outcome was insignificant. Prospective studies are needed to comprehensively explore the impact of inflammatory response on ICH.

**Conflict of interest**

None.

**Supplementary Table**

**Supplementary Table 1.** Associations between PLRs on ER/ICU admission and modified Rankin score using quartile method.

| Variables | ER admission | ICU admission |
|-----------|--------------|---------------|
|           | Adjusted odds ratio | 95% CI | p | Adjusted odds ratio | 95% CI | p |
| Q1        | 0.70 | 0.25–1.94 | 0.495 | Q1 | 1.40 | 0.51–3.85 | 0.508 |
| Q2        | Ref. | – | – | Q2 | Ref. | – | – |
| Q3        | 1.57 | 0.57–4.32 | 0.381 | Q3 | 2.16 | 0.79–5.89 | 0.129 |
| Q4        | 0.95 | 0.35–2.57 | 0.926 | Q4 | 2.50 | 0.88–7.09 | 0.085 |

Modified Rankin score (level one: 0–2, level two: 3–6) was used as a dichotomous variable. PLRs on ER/ICU admission were divided into four quartiles, and the second quartile was used as the reference level. Both the two models were adjusted for age, diabetes mellitus, hypertension, bleeding sites, serum sodium, hospital acquired pneumonia and APACHE II score on ICU admission. PLR = platelet to lymphocyte ratio; ER = Emergency Room; ICU = Intensive Care Unit; CI = confidence interval; APACHE = acute physiology and chronic health evaluation.

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