Analysis of the influencing factors and nursing strategy for acute atelectasis after traumatic brain injury surgery

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Background: Acute atelectasis is common after traumatic brain injury (TBI), but the related factors and treatment are still unclear. This study is to analyze the independent risk factors for acute atelectasis after TBI and propose an interventional nursing strategy, in order to correct respiratory function and improve the prognosis of patients.

Methods: The clinical data of 93 patients with TBI admitted to our hospital from April 2015 to October 2019 were retrospectively analyzed. Clinical data were analyzed by single factor analysis, the cutoff value of influencing factors was obtained by receiver operating characteristic (ROC) curve analysis, and the influencing factors for acute atelectasis after TBI were examined by multi-factor logistic regression.

Results: Twenty-two patients (23.66%) were complicated with acute atelectasis during the observation period, while the remaining 71 patients (76.34%) did not have acute atelectasis. Univariate analysis showed that there were significant differences in the Glasgow Coma Scale (GCS), history of vomiting and aspiration, mannitol use, mechanical ventilation, hypoalbuminemia, and serum hypoxia-inducible factor-1α (HIF-1α) between the acute atelectasis group and the non-acute atelectasis group (P<0.05). The AUC area of HIF-1α level predicting acute atelectasis was 0.896 [95% confidence interval (CI): 0.814–0.978, P=0.042], and the cut-off value was 2.12 mmol/L, with a sensitivity of 76.9% and a specificity of 93.3%. Logistic regression analysis showed that a history of vomiting and aspiration [odds ratio (OR) 3.908, 95% confidence interval (CI): 1.201–12.712], mechanical ventilation (OR 3.250, 95% CI: 1.139–9.271), hypoalbuminemia (OR 5.741, 95% CI: 1.926–17.113), and HIF-1α ≥ 2.12 mmol/L (OR 6.623, 95% CI: 2.364–16.346) were independent risk factors for acute atelectasis after TBI.

Conclusions: A history of vomiting and aspiration, mechanical ventilation, hypoalbuminemia, and high expression of HIF-1α are all independent risk factors for postoperative acute atelectasis in patients with TBI. In clinical practice, patients should be guided to swallow properly, breathe smoothly, eat well, and regularly check the relevant indexes.

Keywords: Traumatic brain injury (TBI); acute atelectasis; influence factor; nursing

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Introduction

Traumatic brain injury (TBI) refers to cranial and brain injuries caused by external forces on the cranium, typically due to traffic accidents, falls from heights, slips, and falls (1). Upon suffering a TBI, patients usually exhibit symptoms such as a loss of consciousness, increased intracranial pressure, vomiting, and decreased sputum reflex. These symptoms can easily induce vomit and respiratory secretions to be inhaled into the lungs or block the airway, resulting in atelectasis (2,3). Clinical observations have found that acute atelectasis in patients with TBI is primarily manifested within 24–48 hours postoperatively. Atelectasis can cause cerebral ischemia and hypoxia, which will increase the risk of mortality in patients. Determining the influencing factors for acute atelectasis after TBI will help strengthen clinical monitoring, encourage the adoption of targeted intervention measures, reduce the incidence of acute atelectasis after TBI, and improve the prognosis of patients with TBI.

However, there are few reports regarding the factors affecting acute atelectasis after TBI. Therefore, this article will retrospectively analyze the clinical data of 93 patients with craniocerebral injury who underwent surgical treatment in our hospital, aiming to identify the influencing factors for acute atelectasis after craniocerebral injury. This study also aims to propose a corresponding nursing strategy to provide a reference for clinically reducing the incidence of acute atelectasis after TBI. The research results are reported as follows.

We present the following article in accordance with the STARD reporting checklist (available at http://dx.doi.org/10.21037/apm-21-790).

Methods

General information

A retrospective study design was employed to investigate patients treated for craniocerebral injury in Department of Neurosurgery, Shanghai Punan Hospital from April 2015 to October 2019. All procedures performed in this study involving human participants were in accordance with the Declaration of Helsinki (as revised in 2013). The study was approved by the Medical Ethics Expert Committee of Punan Hospital in Pudong New Area of Shanghai (No.: 2020004). Individual consent for this retrospective analysis was waived. The inclusion criteria were as follows: (I) patients diagnosed with TBI by imaging; (II) patients without lung injury, surgical contraindications, or severe underlying diseases prior to surgery; (III) a craniocerebral injury Glasgow Coma Scale (GCS) of 3–8 points; and (IV) an admission time <6 h after trauma. Patients were excluded based on the following criteria: (I) those with primary brain disease, pulmonary infection, bronchial injury, chronic obstructive pulmonary disease, and other related diseases that affect the study; (II) those who abandoned treatment or transferred in the middle; (III) those with poor compliance; (IV) dying patients; and (V) those with incomplete clinical data.

A total of 93 patients satisfied the above inclusion and exclusion criteria, and were grouped according to whether they had acute atelectasis during the postoperative hospital stay. Twenty-two patients with acute atelectasis were included in the acute atelectasis group, and 71 patients without acute atelectasis were included in the non-acute atelectasis group.

Study methods

Data collection

By consulting the electronic medical records, we obtained clinical data including the patient's age, gender, GCS score, admission time, injury site, surgical procedure, mannitol usage, operation time, history of vomiting and aspiration (including whether this was combined with underlying diseases such as myasthenia gravis, symmetry Polyneuritis, acute myelitis, etc.), mechanical ventilation, tracheotomy, hypoalbuminemia, use of analgesics, inflammatory factors [tumor necrosis factor-α (TNF-α) and interleukin-6 (IL-6)], serum hypoxia-inducible factor-1α (HIF-1α) level, and other information.

Diagnosis of acute atelectasis

The patients’ symptoms and lung radiographic imaging were collected during the postoperative hospitalization period. For symptoms, acute atelectasis patients can have various chest tightness, dyspnea, palpitation, dry cough, weakened lungs, breath sounds. Abnormal lungs in percussion examination (due to solid sound) have lung consolidation and decreased gas. For lung radiographic imaging, we may observe patients’ increased opacity of the affected lobe, bronchovascular crowding, narrowing of the ipsilateral intercostal spaces, compensatory hyperinflation, the compensatory shift of adjoining structures and/or diaphragm, and silhouette sign affecting contiguous mediastinal structures and so on (4).
Statistical analysis

SPSS 17.0 (SPSS Science Inc., Chicago, IL, USA) was used to analyze the data. Classification data was expressed by n (%), and the chi-square (χ²) test was used. Measurement data was expressed by the mean ± standard deviation, and the t-test was used. The clinical data of the patients were analyzed by univariate analysis, and the best cut-off value of related influencing factors was obtained by the area under the receiver operating characteristic curve (ROC) curve (AUC). Multivariate logistic regression analysis was used to test the influencing factors for acute atelectasis after TBI. P<0.05 was considered to be indicative of statistical significance.

Results

Univariate analysis of acute atelectasis after TBI

Of the 93 patients, 22 patients (23.66%) had acute atelectasis during the hospitalization observation period, while the remaining 71 patients (76.34%) did not develop acute atelectasis. In these two groups of patients, there were statistically significant differences in the GCS score, history of vomiting and aspiration, mannitol usage, mechanical ventilation, hypoalbuminemia, and serum HIF-1α levels (P<0.05) (Table 1).

ROC curve analysis of HIF-1α level predicting the risk of acute atelectasis

The AUC area of HIF-1α level predicting acute atelectasis was 0.896 [95% confidence interval (CI): 0.814–0.978, P=0.042], and the cut-off value was 2.12 mmol/L, with a sensitivity of 76.9% and a specificity of 93.3% (Figure 1).

Multivariate logistic regression analysis of acute atelectasis after TBI

Logistic multivariate analysis was performed with acute atelectasis as the dependent variable, and the GCS score, history of vomiting and aspiration, mannitol usage, mechanical ventilation, tracheotomy, hypoalbuminemia, and serum HIF-1α levels as the independent variables, combined with the cut-off value in Figure 1. Logistic multivariate regression analysis showed that a history of vomiting and aspiration [odds ratio (OR) 3.908, 95% CI: 1.201–12.712], mechanical ventilation (OR 3.250, 95% CI: 1.139–9.271), hypoalbuminemia (OR 5.741, 95% CI: 1.926–17.113), HIF-1α ≥2.12 mmol/L (OR 6.623, 95% CI: 2.364–16.346) were independent risk factors for acute atelectasis after TBI (Table 2).

Discussion

The injured brainstem in TBI may direct damage to respiratory function. Besides, TBI patients often have varying degrees of impaired consciousness and airway self-protection ability. Vomiting and aspiration events are frequent for TBI patients, which can easily lead to airway occlusion and lung infection, leading to atelectasis and severely reducing the patient’s oxygenation level (5). These reasons all increase the duration of mechanical ventilation in TBI patients, thereby increasing the risk of ventilator-induced lung injury.

Partial or complete reversible occlusion of airways can cause atelectasis. The degree of lung collapse and the prolonged time is related to the severity. Atelectasis can cause mechanical vascular occlusion and reactive vasoconstriction, leading to increased right ventricular afterload, making hemodynamic instability (6). Besides, atelectasis can cause the alveolar-blood exchange of CO₂ and O₂ to be hindered. Arterial blood gas abnormalities may affect brain metabolisms, such as hypercapnia and severe hypoxemia, which can reduce cerebrovascular resistance. Increased cerebral blood volume and intracranial pressure will result in unstable intracranial conditions and affect a lousy prognosis.

There is no consensus on the prevention and treatment of atelectasis (7). Atelectasis can be caused by various mechanisms. Airway obstruction can cause obstructive atelectasis. Large pleural effusion can cause passive atelectasis. Increased intra-abdominal pressure can cause compressive atelectasis. Increased alveolar surface tension can cause adhesive atelectasis. Therefore, atelectasis can be classified and treated according to the cause.

Reducing the secretory burden should focus on the recovery and maintenance of airway patency. Atomization and fiberoptic bronchoscope can restore obstructive atelectasis caused by mucus. Exercise, sighing, and deep breathing can help the sober patient. We can use a lung recruitment strategy to treat passive and adhesive atelectasis for patients with mechanical ventilation.

For TBI patients who need mechanical ventilation support, the lung-protective ventilation strategy may not protect the lungs while maintaining arterial partial pressure of oxygen (PaO₂) in the range needed by TBI patients, nor
can it prevent and treat atelectasis in TBI patients. The use of a lung-protective ventilation strategy in acute respiratory distress syndrome (ARDS) patients can significantly reduce ventilator-associated lung injury incidence (8). Lung-protective ventilation strategy advocates the appropriate reduction of ventilation (lower tidal volume and plateau pressure) in ARDS patients and acceptance of the resulting low partial pressure of blood oxygen (55–80 mmHg) (9). However, due to TBI characteristics, patients often need a higher level of PaO\textsubscript{2} to make oxygen enter the mitochondria of

Table 1 Univariate analysis of postoperative acute atelectasis in patients with TBI (\( \chi^2 \))

<table>
<thead>
<tr>
<th>Factor</th>
<th>Acute atelectasis (n=22), n (%)</th>
<th>Non-acute atelectasis (n=71), n (%)</th>
<th>( \chi^2/t )</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gender (cases)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>14 (20.90)</td>
<td>53 (79.10)</td>
<td>1.011</td>
<td>0.315</td>
</tr>
<tr>
<td>Female</td>
<td>8 (30.77)</td>
<td>18 (69.23)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age (years)</td>
<td>49.36±8.90</td>
<td>45.62±7.70</td>
<td>1.918</td>
<td>0.058</td>
</tr>
<tr>
<td>Admission time (h)</td>
<td>2.50±0.41</td>
<td>2.58±0.36</td>
<td>0.881</td>
<td>0.381</td>
</tr>
<tr>
<td>GCS score</td>
<td>4.21±0.85</td>
<td>5.52±1.05</td>
<td>5.329</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Injury site (cases)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Contusion of one frontotemporal lobe</td>
<td>6 (17.14)</td>
<td>29 (82.86)</td>
<td>2.907</td>
<td>0.420</td>
</tr>
<tr>
<td>Bilateral frontotemporal contusion</td>
<td>7 (22.58)</td>
<td>24 (77.42)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Diffuse brain swelling</td>
<td>6 (30.00)</td>
<td>14 (70.00)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Brainstem injury</td>
<td>3 (42.86)</td>
<td>4 (57.14)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Surgical procedure (cases)</td>
<td></td>
<td></td>
<td>0.012</td>
<td>0.911</td>
</tr>
<tr>
<td>Unilateral large craniectomy</td>
<td>13 (24.07)</td>
<td>41 (75.93)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bilateral large craniectomy</td>
<td>9 (23.08)</td>
<td>30 (76.92)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mannitol usage (cases)</td>
<td></td>
<td></td>
<td>5.835</td>
<td>0.016</td>
</tr>
<tr>
<td>&lt;500 mL</td>
<td>12 (38.71)</td>
<td>19 (61.29)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>≥500 mL</td>
<td>10 (16.13)</td>
<td>52 (83.87)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Operation time (h)</td>
<td>2.30±0.74</td>
<td>2.06±0.53</td>
<td>1.681</td>
<td>0.096</td>
</tr>
<tr>
<td>History of vomiting and aspiration (cases)</td>
<td>18 (32.14)</td>
<td>38 (67.86)</td>
<td>4.495</td>
<td>0.034</td>
</tr>
<tr>
<td>Combined with underlying diseases*</td>
<td>9 (31.03)</td>
<td>20 (68.97)</td>
<td>1.270</td>
<td>0.260</td>
</tr>
<tr>
<td>Tracheotomy (cases)</td>
<td>13 (35.14)</td>
<td>24 (64.86)</td>
<td>4.483</td>
<td>0.034</td>
</tr>
<tr>
<td>Mechanical ventilation (cases)</td>
<td>16 (33.33)</td>
<td>32 (66.67)</td>
<td>5.144</td>
<td>0.023</td>
</tr>
<tr>
<td>Indwelling gastric tube (cases)</td>
<td>16 (25.40)</td>
<td>47 (74.60)</td>
<td>0.328</td>
<td>0.567</td>
</tr>
<tr>
<td>Use of analgesics (cases)</td>
<td>11 (25.58)</td>
<td>32 (74.42)</td>
<td>0.164</td>
<td>0.685</td>
</tr>
<tr>
<td>Hypoalbuminemia (cases)</td>
<td>10 (52.63)</td>
<td>9 (47.37)</td>
<td>11.101</td>
<td>0.001</td>
</tr>
<tr>
<td>TNF-(\alpha) (ng/mL)</td>
<td>49.36±5.45</td>
<td>51.69±6.48</td>
<td>1.526</td>
<td>0.130</td>
</tr>
<tr>
<td>IL-6 (pg/mL)</td>
<td>37.66±4.13</td>
<td>36.57±3.80</td>
<td>1.520</td>
<td>0.252</td>
</tr>
<tr>
<td>HIF-1(\alpha) (mmol/L)</td>
<td>2.28±0.55</td>
<td>2.04±0.20</td>
<td>3.101</td>
<td>0.003</td>
</tr>
</tbody>
</table>

*, includes myasthenia gravis, symmetry polyneuritis, acute myelitis, etc. TBI, traumatic brain injury; HIF-1\(\alpha\), hypoxia-inducible factor-1\(\alpha\); TNF-\(\alpha\), tumor necrosis factor-\(\alpha\); IL-6, interleukin-6.
nerve cells across the concentration gradient to participate in aerobic metabolism. Dellazizzo et al. found that the minimum \(\text{PaO}_2\) required to maintain partial pressure of brain tissue oxygen above 20 mmHg in TBI patients is 94 mmHg (10), which was much higher than the \(\text{PaO}_2 > 60\) mmHg in patients with TBI proposed in the Guidelines for the Management of Severe Traumatic Brain Injury (11). \(\text{PaO}_2 = 94\) mmHg is only the lowest value that can maintain the essential oxygen supply of brain tissue. It may mean that TBI patients need a higher \(\text{PaO}_2\) than 94 mmHg to ensure that the brain gets enough oxygen for a good prognosis. Simultaneously, some researchers have reported that high blood oxygen concentration will also increase the mortality of patients with TBI (12). TBI patients’ respiratory support strategies need to prevent and treat atelectasis and maintain \(\text{PaO}_2\) within a reasonable range remain to be widely explored.

Active preventive measures for atelectasis can reduce the risk of atelectasis. It helps maintain the expected levels of arterial partial pressure of carbon dioxide and \(\text{PaO}_2\), maintain the expected level of aerobic metabolism of nerve cells and stabilize the intracranial pressure. Therefore, timely detection and effective treatment of atelectasis are significant for severe neurological patients. This article analyzed the epidemiology of whether acute atelectasis occurred in 93 cases of TBI, and found that 22 cases of acute atelectasis occurred [an incidence rate of 23.66% (22/99)].

At present, the mechanism of acute atelectasis after TBI has not been clarified and may be caused by the interaction of multiple factors. We conducted a univariate analysis of the clinical data of 93 patients with TBI, and found that there were significant differences in the GCS score, history of vomiting and aspiration, mannitol usage, mechanical ventilation, tracheotomy, hypoalbuminemia, and serum HIF-1\(\alpha\) level between patients with and without acute atelectasis. Multivariate logistic regression analysis found that a history of vomiting and aspiration, mechanical ventilation, hypoalbuminemia, and HIF-1\(\alpha\) \(\geq 2.12\) mmol/L were independent risk factors for acute atelectasis after TBI.

Pandian et al. reported that tracheotomy is a risk factor for lung infection in patients with TBI (13). The reasons for this are as follows. Firstly, atelectasis caused by vomiting is primarily a result of blockage of the bronchial ducts by food or respiratory secretions. Therefore, it is clinically recommended for patients with cranioencephalic injury and swallowing dysfunction to consume foods with uniform density, appropriate viscosity, and foods that are not easy to disperse (such as viscous semi-liquid, semi-solid, concentrated liquid food, etc.) as much as possible (14-16). At the same time, patients with TBI should be guided to avoid excessive mouth volume during feeding to prevent excessive food from leaking out of the mouth or causing aspiration. If the patient’s swallowing function is weak,

![Figure 1](image.png)

**Figure 1** ROC curve analysis of HIF-1\(\alpha\) level predicting the risk of acute atelectasis. ROC, receiver operating characteristic curve; HIF-1\(\alpha\), hypoxia-inducible factor-1\(\alpha\).

<table>
<thead>
<tr>
<th>Variable</th>
<th>(\beta)</th>
<th>Wald</th>
<th>(P)</th>
<th>OR</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>History of vomiting and aspiration</td>
<td>1.363</td>
<td>4.267</td>
<td>&lt;0.001</td>
<td>3.908</td>
<td>1.201–12.712</td>
</tr>
<tr>
<td>Mechanical ventilation</td>
<td>1.179</td>
<td>3.435</td>
<td>&lt;0.001</td>
<td>3.250</td>
<td>1.139–9.271</td>
</tr>
<tr>
<td>Hypoalbuminemia</td>
<td>1.748</td>
<td>1.480</td>
<td>&lt;0.001</td>
<td>5.741</td>
<td>1.926–17.113</td>
</tr>
<tr>
<td>HIF-1(\alpha) (\geq 2.12) mmol/L</td>
<td>1.891</td>
<td>1.346</td>
<td>&lt;0.001</td>
<td>6.623</td>
<td>2.364–16.346</td>
</tr>
</tbody>
</table>

TBI, traumatic brain injury; HIF-1\(\alpha\), hypoxia-inducible factor-1\(\alpha\).
nasal feeding care can be administered. Before nursing, the head of the bed should be raised appropriately, and nasal feeding should be given for 0.5 h after sputum suction. It is important to note that sucking sputum is strictly forbidden after nasal feeding. If the patient has a swallowing reflex or coughing reaction, nasal feeding should be stopped immediately, and appropriate sputum expectoration measures should be taken.

Secondly, during mechanical ventilation, if the intubation is too deep, it can quickly cause the entire lung to collapse. At the same time, it causes unilateral lung ventilation, leading to shrinkage of alveoli on the other side due to reduced or no ventilation, which will induce atelectasis (17). Therefore, it is recommended that before operating on patients who use mechanical ventilation, nursing staff should be fully trained in relevant knowledge to ensure that they master the relevant nursing methods and precautions of artificial airways for mechanically-ventilated patients. In order to prevent lung atrophy due to excessive suction of negative pressure, negative pressure should be controlled at 150–200 mmHg. If the suction time is too long, it is easy to interrupt positive pressure ventilation and cause iatrogenic atelectasis, and thus, the suction time should be less than 15 s. At the same time, nursing staff should not leave the ventilator during sputum suction to avoid airway pressure imbalance and interruption of ventilation.

Thirdly, the albumin (ALB) level can be used to assess the prognosis of cranial injuries (18). Under conditions of stress, such as head trauma or surgery, the body’s immune function declines. Also, especially after Enterobacteriaceae infection, the expression of ALB mRNA in patients is inhibited, and hypoproteinemia is prone to occur. Hypoproteinemia will weaken respiratory defenses and immune function, making patients with TBI more vulnerable to pathogenic microorganisms, viruses, and various inflammatory cells (19). The invasion of a large number of bacteria can aggravate lung infections, promote alveolar exudation, increase secretions, block alveoli, and induce atelectasis. Therefore, the patient’s recovery should be closely followed-up postoperatively and nutritional support should be strengthened according to individual circumstances. The intake of high-protein, high-calorie, and high-vitamin foods should also be increased. Based on the patient’s recovery, amino acid preparations, hydrolyzed protein, glucose, etc. should be administered to improve the immune function of the patient’s respiratory tract and prevent the occurrence of atelectasis.

Fourthly, HIF-1α plays an important regulatory role in erythropoiesis, glycolysis, and cell proliferation (20,21). Studies have confirmed that HIF-1α is closely related to the progression of lung disease under hypoxia (22-24). Intracranial hypoxia caused by TBI can enable HIF-1α to regulate the tolerance of epithelial cells by affecting the transcription and expression of nitric oxide synthase under hypoxic conditions (25,26). Decreased release of nitric oxide will cause continuous vasoconstriction and increase airway resistance. In this paper, it was found that the HIF-1α cut-off value of 2.12 mmol/L had a high sensitivity and specificity in predicting the occurrence of acute atelectasis after TBI. Therefore, for patients with abnormally elevated serum HIF-1α levels, we must closely monitor whether their airways are blocked. Diluting and draining respiratory secretions via airway humidification, ultrasonic atomization, vibration sputum, and other methods to keep the airway unobstructed. High-flow oxygen therapy is given to patients who are evacuated from the ventilator. Adequate oxygen therapy can effectively increase lung ventilation, lung volume, oxygen and ventilation, and prevent alveolar atrophy and atelectasis.

Even though our findings are interesting, there are two limitations. Firstly, this study is not a large sample size study. Although the current results have a particular significance, in the future, we plan to expand the sample size or join the multicenter study further to explore the study of TBI patients with atelectasis. Secondly, this study is a retrospective study. The data that could be collected was limited and prone to selection bias or recalls bias. However, this study’s result provides a basis for further design of prospective research to improve the reliability of the research results.

In summary, a history of vomiting and aspiration, mechanical ventilation, hypoalbuminemia, and high expression of HIF-1α are all independent risk factors for acute atelectasis after TBI. Medical staff should strengthen the monitoring of the postoperative condition of patients with TBI, instruct patients to swallow properly, keep the airway unobstructed, offer balanced nutrition, and regularly check related indicators.

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**Footnote**

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*Ethical Statement:* The authors are accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved. All procedures performed in this study involving human participants were in accordance with the Declaration of Helsinki (as revised in 2013). The study was approved by the Medical Ethics Expert Committee of Punan Hospital in Pudong New Area of Shanghai (No.: 2020004). Individual consent for this retrospective analysis was waived.

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