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Correlation Between Serum Procalcitonin Levels and the Severity of Pediatric Traumatic Brain Injury

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Abstract: *Objective:* To investigate the correlation between serum procalcitonin (PCT) levels and the severity of pediatric traumatic brain injury (TBI). *Methods:* Clinical data of 370 pediatric patients with TBI admitted to the Department of Neurosurgery, Jingzhou First People's Hospital between January 2018 and December 2025 were retrospectively analyzed. Based on the Glasgow Coma Scale (GCS) score, patients were divided into a mild TBI group (GCS 13–15, n = 250) and a moderate-to-severe TBI group (GCS 3–12, n = 120). Serum levels of PCT, C-reactive protein (CRP), white blood cell count (WBC), and neutrophil percentage (N%) within 24 hours of admission were compared between the two groups. The correlation between PCT and GCS score was analyzed. *Results:* PCT levels in the moderate-to-severe group were significantly higher than those in the mild group [1.82 (0.65, 3.46) vs. 0.06 (0.03, 0.12) μg/L, $p < 0.001$]. CRP, WBC, and N% were also significantly elevated in the moderate-to-severe group [18.6 (9.4, 34.2) vs. 3.2 (1.5, 6.8) mg/L; 14.7 ± 3.8 vs. 8.3 ± 2.1×10^9/L; 82.3 ± 6.7% vs. 64.2 ± 5.8%, all $p < 0.001$]. ROC analysis showed that the area under the curve (AUC) for PCT in discriminating moderate-to-severe TBI was 0.92 (95% CI: 0.88–0.96), significantly outperforming CRP (AUC = 0.85, 95% CI: 0.80–0.90; DeLong test, Z = 2.84, $p = 0.004$). PCT levels were negatively correlated with GCS scores (r = −0.542, $p < 0.001$). *Conclusions:* Serum PCT levels are negatively correlated with GCS scores in children with TBI, with higher PCT levels indicating greater injury severity. PCT demonstrates excellent diagnostic performance for discriminating moderate-to-severe TBI (AUC = 0.92), superior to CRP (AUC = 0.85), and may serve as a valuable adjunctive biomarker for assessing injury severity in this population.

Keywords: Procalcitonin; Children; Traumatic brain injury; Glasgow coma scale; Severity

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1. Introduction

Procalcitonin (PCT) is a precursor peptide of calcitonin. In healthy individuals, serum concentration is very low, usually below 0.1 μg/L [1,2]. It is worth noting that PCT is often used for early diagnosis and assessment of sepsis and severe infections, but it can also increase in non-infectious conditions such as major trauma and extensive surgery, and the degree of elevation is related to severity of tissue injury [3,4].

TBI is common neurosurgical emergency in children. It shows rapid clinical progression with high risk of secondary infection that we should pay attention to in clinical work because this can affect the treatment outcome and patient safety in a significant way. After TBI occurs in children, damaged brain tissue releases damage-associated molecular patterns

(DAMPs), and this triggers systemic inflammatory response that spreads through the body and leads to elevated levels of inflammatory biomarkers such as PCT and CRP in blood test [5,6] because the DAMPs act as signal for immune cells to start working. In theory, greater injury severity can result in more extensive DAMP release from the damaged cells in the brain tissue. Then the inflammatory response becomes stronger throughout the whole body, so finally the PCT levels can be higher than what we see in normal cases.

Previous studies examined PCT in the adult TBI and found that PCT can increase because of traumatic stress even when no infection exists, and this elevation cannot be seen as infection [7]. But children are different from adults. In fact, they have different PCT metabolism and baseline levels, and the inflammatory response pattern is also not the same. Studies about the correlation between PCT levels and injury severity in pediatric TBI are not many. Also, existing studies have small sample sizes.

This retrospective study analyzed clinical data from 370 pediatric patients with TBI to examine the correlation between PCT levels and Glasgow Coma Scale (GCS) scores, aiming to provide a reference for clinical assessment of TBI severity.

2. Materials and methods

2.1. Study population

A total of 370 pediatric patients with TBI admitted to the Department of Neurosurgery, Jingzhou First People's Hospital between January 2018 and December 2025 were enrolled. This study was approved by the Medical Ethics Committee of Jingzhou First People's Hospital (Approval No.: KY2026-019-01).

In this study, we include patients under 18 years old and complete clinical data is required. PCT should be measured within 24 hours after admission and we exclude those with concomitant dysfunction of vital organs including heart, lung, liver, or kidney, and autoimmune disease, malignancy, or hematological disorders. It is worth noting that antibiotic use prior to admission should be avoided. Also, confirmed infection (e.g., pneumonia, intracranial infection) should be excluded.

2.2. Grouping

Patients were stratified into two groups based on admission Pediatric Glasgow Coma Scale (GCS) scores [8]:

(1) Mild TBI group (n = 250)
GCS 13–15.

(2) Moderate-to-severe TBI group (n = 120)
GCS 3–12.

2.3. Laboratory measurements

Venous blood samples were collected from all patients within 24 hours of admission for measurement of PCT, CRP, WBC, and N%. PCT was determined by chemiluminescence immunoassay, CRP by rate nephelometry, and routine blood parameters by an automated hematology analyzer.

2.4. Statistical analysis

Statistical analyses were performed using SPSS 27.0. Normally distributed continuous variables are presented as mean ± standard deviation and compared using the independent-samples *t*-test; non-normally distributed variables are presented as median (25th percentile, 75th percentile) and compared using the Mann–Whitney U test. Categorical variables are expressed as counts (%) and compared using the chi-square test. The correlation between PCT and GCS scores was assessed using Spearman's rank correlation. A two-sided p value < 0.05 was considered statistically significant.

3. Results

3.1. Baseline characteristics

No significant differences in age or sex were observed between the mild and moderate-to-severe TBI groups ($p > 0.05$) **(Table 1)**.

Table 1. Comparison of baseline characteristics between the two groups

Variable	Mild group (n = 250)	Moderate-to-severe group (n = 120)	Statistic (Z/χ^2)	*p* value
Age (years), median (Q1, Q3)	5 (3, 8)	6 (3, 9)	Z = −1.608	0.108
Sex (male/female)	144/106	67/53	$\chi^2 = 0.109$	0.741

Notes: Data are presented as median (25th percentile, 75th percentile) or counts.

3.2. Comparison of PCT, CRP, WBC, and N% within 24 hours of admission

PCT, CRP, WBC, and N% levels were significantly higher in the moderate-to-severe group than in the mild group (all $p < 0.05$) **(Table 2)**.

ROC curve analysis demonstrated that the area under the curve (AUC) for PCT in discriminating moderate-to-severe TBI was 0.92 (95% CI: 0.88–0.96), with an optimal cutoff value of 0.52 μg/L, yielding a sensitivity of 87.5% and a specificity of 82.4%. The AUC for CRP was 0.85 (95% CI: 0.80–0.90), with an optimal cutoff of 12.3 mg/L, sensitivity of 72.3%, and specificity of 76.8% **(Table 3)**.

Table 2. Comparison of inflammatory biomarkers within 24 hours of admission between the two groups

Variable	Mild group (n = 250)	Moderate-to-severe group (n = 120)	*Z*/*t*	*p* value
PCT (μg/L)	0.06 (0.03, 0.12)	1.82 (0.65, 3.46)	$Z = -12.847$	< 0.001
CRP (mg/L)	3.2 (1.5, 6.8)	18.6 (9.4, 34.2)	$Z = -11.253$	< 0.001
WBC ($\times 10^9$/L)	8.3 ± 2.1	14.7 ± 3.8	$t = -7.381$	< 0.001
N% (%)	64.2 ± 5.8	82.3 ± 6.7	$t = -8.715$	< 0.001

Notes: PCT and CRP are presented as median (25th percentile, 75th percentile) and were compared using the Mann–Whitney U test (*Z* values); WBC and N% are presented as mean ± standard deviation and were compared using the independent-samples *t*-test (*t* values). $p < 0.05$ was considered statistically significant.

Table 3. ROC curve analysis of PCT and CRP for discriminating moderate-to-severe TBI

Biomarker	AUC (95% CI)	Optimal cutoff	Sensitivity	Specificity	Youden index
PCT	0.92 (0.88–0.96)	0.52 μg/L	87.5%	82.4%	0.699
CRP	0.85 (0.80–0.90)	12.3 mg/L	72.3%	76.8%	0.491

Notes: AUC, area under the curve; 95% CI, 95% confidence interval; Youden index = sensitivity + specificity − 1. Comparison of AUCs between PCT and CRP was performed using the DeLong test: $Z = 2.84$, $p = 0.004$.

3.3. Correlation between PCT and GCS score

Spearman correlation analysis revealed a significant negative correlation between PCT levels and GCS scores ($r = -0.542$, $p < 0.001$), indicating that lower GCS scores (greater injury severity) were associated with higher PCT levels **(Table 4)**.

Table 4. Distribution and correlation of GCS scores and PCT levels in all 370 patients

Variable	All patients (n = 370)
GCS score	13 (9, 15)
PCT (μg/L)	0.28 (0.05, 1.24)
Spearman's r	−0.542
p value	< 0.001

Notes: Data are presented as median (25th percentile, 75th percentile). Spearman's rank correlation was used to assess the association between GCS score and PCT level.

4. Discussion

We found serum PCT levels can be higher in moderate-to-severe TBI group than in mild group. The levels can have negative correlation with GCS scores. It is worth noting that PCT can reflect injury severity in pediatric TBI. These results match prior adult studies and PCT can be used as infection biomarker and it can also indicate post-traumatic systemic inflammatory response intensity [7,9].

4.1. Magnitude of PCT elevation far exceeds that of conventional inflammatory markers

From Table 2 we can see the median PCT level in moderate-to-severe group was 1.82 μg/L. This is about 30-fold higher than mild group (0.06 μg/L) ($Z = -12.847$, $p < 0.001$). Also, CRP increased by about 6-fold (18.6 vs. 3.2 mg/L, $Z = -11.253$, $p < 0.001$), while WBC increased by about 1.8-fold (14.7 vs. 8.3 × 10^9/L, $t = -7.381$, $p < 0.001$) and N% increased by about 1.3-fold (82.3% vs. 64.2%, $t = -8.715$, $p < 0.001$). It is worth noting that all four markers showed statistically significant differences, but the magnitude of PCT elevation can be regarded as most pronounced because the value changed from a near-undetectable level (0.06 μg/L) to stress-induced level (1.82 μg/L). This "from-absent-to-present" pattern can make PCT more sensitive than CRP, WBC, and also N% in reflecting trauma severity.

4.2. PCT outperforms CRP in discriminating moderate-to-severe TBI

We can see from Table 3 the ROC results. The AUC for PCT in discriminating moderate-to-severe TBI was 0.92 (95% CI: 0.88–0.96), and this value is higher than CRP which has 0.85 (95% CI: 0.80–0.90; DeLong test, $Z = 2.84$, $p = 0.004$). At optimal cutoff 0.52 μg/L, PCT has sensitivity 87.5% and specificity 82.4%. The Youden index is 0.699. By comparison, CRP at its optimal cutoff of 12.3 mg/L has sensitivity 72.3% and specificity 76.8%, with Youden index 0.491. It is worth noting that these findings indicate PCT can be more effective than CRP in distinguishing mild from moderate-to-severe injury in early assessment of pediatric TBI and we expect it has good clinical utility.

4.3. Negative correlation between PCT and GCS score confirms the "greater injury, higher PCT" relationship

Table 4 shows the results and we can see that the median GCS score for all 370 patients was 13 (9, 15) while the median PCT was 0.28 (0.05, 1.24) μg/L, and the Spearman correlation coefficient was r = −0.542 ($p < 0.001$). This moderate negative correlation (r = −0.542) is reasonable. In fact, PCT is influenced by GCS. It is also affected by factors such as age, surgery, or concomitant injuries, so the r value cannot approach −1.0. This result aligns with the previous adult studies. Also, it can support the feasibility of using PCT as biomarker for TBI severity assessment [7,10].

4.4. Clinical implications: Differentiating traumatic stress from infection

All patients in this study had no overt infection. In fact, PCT elevation can be attributed to traumatic stress, and infectious causes should not be the first reason. When elevated PCT is found in pediatric TBI patients, we should first consider that

this is caused by the trauma itself. We should not presume it is infection.

However, this does not mean PCT has no value for finding infection. If PCT stays high (e.g., > 2 μg/L) or keeps rising in serial monitoring, we should suspect secondary infection and a full evaluation is needed including clinical signs, imaging, blood tests, and microbiological investigations. Also, the dynamic change of PCT can provide more clinical guidance than single measurement. Future studies should examine value of serial PCT monitoring.

4.5. Limitations

This study has several limitations we should note. First, because of retrospective design, serial PCT data were not available for some patients, which means we cannot analyze the temporal trends of PCT in a full way and this can affect the understanding of how PCT changes over time in these patients. Second, the accuracy of GCS scores in infants and young children can be influenced by age. Future studies should use pediatric-specific trauma scoring systems (e.g., Pediatric GCS) for stratified analysis. Third, we only analyzed a single PCT measurement. It is worth noting that this limits the assessment of clinical value for dynamic monitoring. Fourth, factors that can influence PCT levels, such as surgery and blood transfusion, were not accounted for in the current analysis, and future multivariable analyses are needed to evaluate the independent predictive value of PCT in full way and can help remove the confounding effects from these factors.

5. Conclusion

Serum PCT levels are negatively correlated with GCS scores in children with TBI, with higher PCT levels indicating greater injury severity. PCT may serve as an adjunctive biomarker reflecting the intensity of the post-traumatic inflammatory response; however, its use for discriminating infection requires integration with comprehensive clinical assessment.

Disclosure statement

The authors declare no conflict of interest.

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