Dynamic Changes and Clinical Significance of White Blood Cells and Platelets in Patients with Traumatic Brain Injury

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Traumatic brain injury (TBI) is becoming the main cause of death threatening human life because of its high disability rate, high mortality rate and great social harm. The purpose of this study was to investigate the dynamic changes and clinical significance of leukocytes (WBC) and platelets (PLT) in patients with traumatic brain injury. In the process of specimen collection, 4ml of peripheral venous blood was collected 24 hours, 4 days, 7 days, 14 days and 21 days after craniocerebral injury using sterile, non-heat source and non-endotoxin test tube. 2ml venous blood was added into EDTA anticoagulant blood collection vessel to detect the changes of peripheral blood leukocytes and platelets in patients with craniocerebral injury. In addition, 2 ml venous blood was added into the procoagulant blood collection vessel, and the serum was separated and frozen for the determination of CRP and TSP1. All patients were evaluated with Glasgow outcome score at discharge. When detecting PLT and WBC, Sysmex K4500 hematology analyzer was used to routinely measure the number of PLT and WBC in peripheral blood at different time points. In the process of CRP (C-reactive protein) measurement, adjust the pipette to 60 μl, and add 50 μl of developer a and developer B into the micropore respectively. Then it vibrates on a mini vibrator for 5 seconds to ensure mixing. Preheat the microplate reader for 30 minutes, place the microplate in the microplate reader, set the parameters and read the values. When measuring TSP1 (thrombin sensitive protein 1), adjust the micropipette to 50 μl, then add 50μl termination solution into the micropore respectively, and then vibrate on the micro vibrator for 30 seconds to ensure full mixing. Further analysis showed that there was no difference between the non-infection group and the normal control group on the 4th day (P > 0.05). This study is helpful to the timely treatment of patients with traumatic brain injury.

Key words: Traumatic Brain Injury, Patient White Blood Cells, Platelets, Dynamic Changes, C-reactive Protein
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With the development of medical level, the treatment success rate of patients with craniocerebral injury is increasing, and the incidence of traumatic hydrocephalus is gradually increasing. Traumatic hydrocephalus will seriously affect the prognosis of patients, and cause serious psychological and economic burden to patients' families and society. Therefore, neurosurgeons now pay more attention to traumatic hydrocephalus. The diagnosis and treatment of traumatic hydrocephalus is still difficult, and the prognosis of patients is highly uncertain. In the process of diagnosis and treatment of severe brain injury, there are many high-risk factors related to hydrocephalus. Further study of relevant high-risk factors is helpful to reduce the incidence of hydrocephalus and early diagnosis and intervention of hydrocephalus, so as to strive for a good prognosis for patients.

Modern medicine has made great progress in the treatment of severe craniocerebral injury, with remarkable curative effect, but there are still many

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problems that cannot be explained and treated, and many scholars expect to seek a breakthrough in traditional Chinese medicine. "Holistic concept, syndrome differentiation and treatment" is the core of traditional Chinese medicine. In this study, through the collection of TCM clinical symptoms of patients with severe craniocerebral injury, and the collection and analysis of PLT of patients, to explore the correlation between PLT and various syndromes, and to provide theoretical and experimental basis for TCM treatment of severe craniocerebral injury.

In patients with ventilatory head injury (TBI) in the intensive care unit (ICU), hyperoxaemia is not associated with a higher hospital mortality rate. Algarra N N determined that between 2003 and 2008, 61 hospitals in the United States suffered from arterial blood gas within 24 hours of admission to the ICU. He identified 1212 patients with ventilatory TBI, of which 403 (33%) were normoxemia, 553 (46%) were hypoxemia, and 256 (21%) were hyperxemia. The mortality rate was higher in the hypoxic group (224/553 [41%], followed by hyperoxemia (80/256 [32%]. Although his study was adjusted for other potential confounding factors in the multivariate analysis, possibility of exposure to hyperoxemia and the specific characteristics of the hospital, the possibility of exposure to hyperoxemia is independent, but his study is not prospective [1]. Wang W summarized the 2013 GBD injury estimates are being updated and provide detailed information on the incidence, mortality, DALYs, and rate of change from 1990 to 2013 for 26 causes of injury in the world, by region and country. The extensive GBD mortality database is used. Estimate, correct uncertain causes of death, and estimate using the overall cause of death modeling tool. Morbidity estimation is based on inpatient and outpatient data sets, 26 causes of injury and 47 categories of injury nature, and 7 long-term use of patient reports A follow-up study of outcome indicators. Although the global DALY rate of injury by age dropped by 31% between 1990 and 2013 in his study (UI dropped from 26% to 35%), his study time was too long [2]. Understanding the epidemiology of traumatic brain injury (TBI) is essential for formulating public health policies, implementing prevention strategies, and rationally allocating resources for TBI research, education, and rehabilitation. Nguyen R believes that there has not been a systematic review of all involved A population-based study of the epidemiology of TBI subtypes. He conducted a search at Medline and EMBASE on May 23, 2014. He used previously published tools to assess study quality and certainty bias in duplicate. Each item was recorded. The demographic data and incidence estimate of the study, as well as the stratification of age, gender, data collection year, and severity. Although his study believes that the incidence of TBI varies by age and country, the global epidemiological characteristics of TBI are still not very ideal [3]. Chao L believes that melatonin acts as an important mediator of sterile neuroinflammation. However, its basic mechanism is still poorly understood. Mitochondrial dysfunction is the main source of reactive oxygen species, which can affect inflammation. His research aimed to examine the effect of melatonin on inflammation by controlling cortical impact to eliminate damaged mitochondria (an in vivo model of traumatic brain injury (TBI)). He demonstrated that mitochondria, which inhibit autophagy, are damaged. The selective degradation of mitochondria significantly enhances TBI-induced inflammation. Melatonin treatment activates mitochondria through the mTOR pathway and then reduces TBI-induced inflammation. In addition, melatonin treatment can significantly improve neuronal death and behavior after TBI Defects, and 3-methyladenine reverses this effect by inhibiting mitochondrial phagocytosis. Although his results highlight the role of melatonin in protecting TBI-triggered immunopathology, the research cannot explain that it is autophagy of damaged mitochondria negatively regulates the mechanism of inflammation activation and IL-1β secretion [4].

This study mainly explores the dynamic changes and clinical significance of white blood cells (WBC) and platelets (PLT) in patients with traumatic brain injury. During the specimen collection process, a sterile, pyrogen-free, and endotoxin-free test tube was used to draw 4ml of peripheral venous blood 24 hours, 4 days, 7 days, 14 days, and 21 days after the patient's head injury. Among them, 2ml of venous blood is added to EDTA anticoagulated blood collection tube, which is used to detect the changes in the number of peripheral blood white blood cells and platelets in patients with craniocerebral injury. Another 2 ml

of venous blood was added to the procoagulant blood collection tube, the serum was separated, and the specimens were frozen for the determination of the patient's serum CRP and TSP1. Glasgow prognostic score was performed on all patients at discharge. In the detection of PLT and WBC, the Sysmex K4500 blood cell analyzer is used to routinely determine the number of PLT and WBC in the peripheral blood of patients at different time points. During the measurement of CRP (C reactive protein), adjust the pipette 60μl, and add 50μl of chromogenic reagent A and chromogenic reagent B to the microwells, the same. Then shake on a micro shaker for 5 seconds to ensure mixing. Warm up the microplate reader for 30 minutes, place the microplate in the microplate reader, set the parameters, and read the values. When measuring TSP1 (thrombin sensitive protein 1, thrombospondin l), adjust the micropipette to 50 ul, and then add 50 u1 of the stop solution to the micro wells, and then shake on the micro shaker for 30 seconds to ensure sufficient mixing.

DYNAMIC CHANGES OF WHITE BLOOD CELLS AND PLATELETS

Traumatic Head Injury

Traumatic craniocerebral injury has a rapid onset, rapid disease change, poor prognosis, and a high incidence. In China, about 600,000 people suffer from craniocerebral injury each year, ranking second in systemic injuries, and the fatality rate is the first. About 100,000 people die every year [5,6]. The age of the patients is 14-55 years old. It is an important cause of death and disability among young adults and brings serious harm and economic burden to the society [7]. Nerve damage after craniocerebral injury is divided into two forms: one is primary nerve injury. The direct "mechanical energy" of trauma can cause the destruction of neuron cell membranes and organelles and release inflammatory mediators, causing inflammatory cell infiltration and induction of cells Apoptosis; the second is secondary nerve damage, which is delayed nerve damage caused by multiple factors such as inflammation, immune response, cerebral vasospasm, and further ischemia and hypoxia. This process can last for several hours, days or more long time. If the motion characteristics of the controlled system can be described by a dynamic equation composed of discrete measurement variables, the expression of the discrete-time dynamic model of the unit z_{ij} output by $\sum_{ij}$ can be written as [8]:

$$z_{ij}(t+1) = F_{ij}\left(X_{ij}(t)\right) z_{ij-1}\left(t - d_{ij-1}^{ij}\right) \quad (1)$$

In the formula, $z_{ij} \subset Z$. Variable string [9,10]:

$$X_{ij}(t) = \left\{ z_{hm}\left(t - d_{hm}^{ij} - l_{hm}^{ij}\right) \middle| hm \in N_{ij} \right\} \quad (2)$$

Subscript set:

$$N_{ij} = \left\{ hm \middle| 0 \le d_{hm}^{ij} < \infty; m = 0,1,\dots,k_n, h = \{1,2,\dots,p_1\} \right\} \quad (3)$$

At present, clinical evaluation of the severity of the disease and the prognosis of neurological function in patients with brain injury mainly relies on neurological assessments such as the Glasgow Coma Scale (GCS), Glasgow Outcome Scale (GOS) and imaging examinations such as cranial. Brain CT, MRI scan, etc. [11]. However, in some cases, it is difficult to use neurological assessment and imaging examination to evaluate the condition of the patient early and predict changes in the patient's condition. First, down-sampling and feature extraction are required. Due to the large amount of redundant information in patients, down-sampling can effectively reduce the amount of data that needs to be processed [12,13].

$$S = \min_{i\epsilon[1,l]} \sum_{t=1,t\neq i}^{l} \left| |hi - ht| \right| \quad (4)$$

Another feature used in the algorithm is the feature of damaged image blocks [14].

$$M_r = \left| m_{ij} \right| \quad i,j = 1,2,\dots,\eta \quad (5)$$

$$m_{ij} = \frac{1}{l} \sum_{t=1}^{l} v_{i,jt} \quad (6)$$

Among them, $v_{i,jt}$ is the pixel value of the t position in the image block of the $\{i,j\}$damage site [15,16].

White Blood Cells and Platelets

Mean platelet volume (MPV) is an important indicator in blood cell analysis, and it is also a very important indicator of platelet activation. If the MPV increases, the platelet volume increases, and its activity is likely to increase. In most cases, PLT decreases, and MPV increases, mainly because the destruction of platelet count increases; PLT will be low, and colleagues' MPV will also decrease, which may be caused by bone marrow lesions. The indicators of platelet production, failure and death of patients can be detected by PLT, and the parameters of proliferation, metabolism and platelet production of macrophages in the patient's bone marrow can be detected by MPV, which can show the approximate age of platelets in the

circulation [17]. The related functions of the platelet body are related to the PLT count, and also closely related to its individual functional status, and the individual function of PLT is related to the morphology of platelets. When the overall function of the platelet is destroyed due to the decrease in the number of platelets, the PLT count is related to MPV. Relevant supplements will be made to work together to keep the body's bleeding function and coagulation function at a normal level [18,19]. Platelets are a nucleus formed part of blood. There are many glycoprotein molecules on the surface of resting platelet membrane to maintain the integrity of its structure and function. In addition, the various granular components contained in platelets are also essential elements for their normal functioning. When the vascular endothelium is damaged or under the action of certain pathophysiological stimulating factors, platelet adhesion, deformation, aggregation and release and other activation reactions occur. At this time, the expression of glycoprotein molecules on the platelet membrane surface increases [20]. At the same time, the cytokines IL-1 and IL-6 produced by inflammatory cells and other soluble cell adhesion factors such as ICAM-I, etc., can also indirectly stimulate the proliferation of smooth muscle cells; in addition, studies have shown that leukocytes after activation It can activate platelets and lung tissue cells, induce the synthesis and release of TxA2 and platelet activating growth factor (PAF) in the tissues of the blood caplet west I zone, especially the interaction between LTs, TxA2 and PAF, which promotes the increase of pulmonary artery pressure [21,22].

$$M_{t+1} = M_t + \mu(A_{t+1} - B_{t+1}) \quad (7)$$

Where I is the identity matrix and S is a soft threshold function [23]:

$$S_k(a) = a\,max\left\{\left(1 - \frac{k}{|a|}\right), 0\right\} \quad (8)$$

When updating patient data information, fix the reconstruction coefficient A and calculate it according to the following formula [24]:

$$\min_D F(X, A, D), s.t. \left\|d_j\right\|_2^2 < c, \forall j \in \{1,2,\dots,p\} \quad (9)$$

Among them, d_j is the jth column of patient data information.

$$p = \frac{2}{1+e^{(-2x)}} - 1 \quad (10)$$

p is the probability of recovery from surgery. The main role of interleukin 6 (IL-6) is to induce T lymphocyte differentiation and B lymphocyte growth in immune regulation. B lymphocytes must use IL-6 to secrete IgA, IgG and IgM, so IL-6 plays a vital role in humoral immunity. On the one hand, it induces the differentiation of T lymphocytes, enhances the release of cytokines, and promotes the occurrence of inflammation; on the other hand, it promotes the growth of B lymphocytes, induces their differentiation, and promotes the production of autoantibodies, leading to the occurrence of autoimmune diseases. Platelet activation and damage to endothelial cell function may be caused by the abnormal distribution of calcium ions inside and outside the membrane caused by shear stress and oxidative stress at the early stage of reperfusion. Factors, oxygen free radicals and adhesion molecules produce damage or activation effects, and there is also mutual activation between initially activated platelets and endothelial cells [25,26].

$$y = \frac{1}{1+e^{(-p)}} \quad (11)$$

The output value of the neural network model is denormalized as follows:

$$y_{ij} = y\frac{0.8 - y_{ij}}{0.6}(ymin_{max})_{max} \quad (12)$$

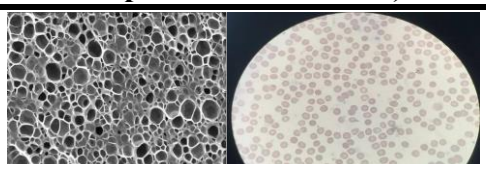
In the formula, y_{ij} represents the output value of the reaction temperature soft sensor model. White blood cells and platelets are shown in Figure 1.

WHITE BLOOD CELL AND PLATELET DETECTION EXPERIMENT IN PATIENTS WITH TRAUMATIC BRAIN INJURY

Experimental Materials

Experimental subjects

Figure 1.
White blood cells and platelets (http://alturl.com/zw6ns and http://alturl.com/2aact)



A total of 63 patients with craniocerebral injury who came to our hospital from May 2018 to October 2019 were selected, aged 19-72 years old, with an average age of 45 years old. There are 50 men and 13 women. Among the 63 patients, 45 cases of closed head injury (71.4%), 10 cases of internal open head injury (15.9%), and 8 cases of open head injury (12.7%). According to the GCS score after admission, 21 patients had mild head injury, 11 patients had medium head injury, and 32

patients had severe head injury. The diagnosis was confirmed by CT scan: Injuries included epidural hematoma, subdural hematoma, contusion and laceration of brain, traumatic subarachnoid hemorrhage). Among the 63 cases of craniocerebral injury, 28 cases were infected, including 25 cases of pulmonary infection, 2 cases of intracranial infection and 1 case of urinary tract infection. Patients were divided into infection group (28 cases) and non-infection group (35 cases). Another 10 healthy people were selected as controls, including 6 males and 4 females, aged 26-50 years old, with an average age of 38 years old. There was no significant difference in age and gender between the healthy group and the patient group. All patients and their families signed informed consent, and the experimental process was approved by the ethics committee.

Main experimental instruments:

1) Microplate reader BIO-RAD 680 American Bio-rad Company

2) 37℃ Constant temperature water baths Domestic

3)-80℃ ultra-low temperature refrigerator United States

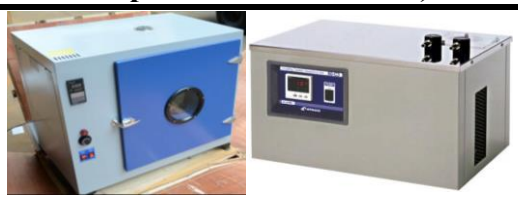
4) Electric heating drying oven

5) Hematology analyzer Sysmex K4500 Japan Sysmex Corporation

6) Low-temperature refrigerated high-speed centrifuge Hereaus, Germany

7) Beakers, measuring cylinders, etc. (BIO-RAD 680 microplate reader, shaker, multi-row pipette, 37℃ constant temperature water bath, electric drying oven, pipette). Electric heating drying box and 37℃ constant temperature water bath boxes are shown in Figure 2.

Figure 2.
Electric heating drying oven and 37℃ constant temperature water baths (http://alturl.com/ug9b9 and http://alturl.com/ahmak)



Main experimental reagents:

1) 96-well precision microplate, coated.

2) CRP standard product, divided into 5 bottles, each bottle is 1.0 ml, the concentration is 0 ng/ml, 10 ng/ml, 25 ng/ml, 50 ng/ml, 100 ng/ml.

3) Concentrated washing solution, 1 bottle of 50 ml, diluted 20 times in deionized water, used for repeated washing of microplates.

4) TSPI standard products, divided into 6 bottles, each bottle of 10 ml, the concentrations are 0 ng/ml, 50 ng/ml, 100 ng/m1, 250 ng/ml, 500 ng/m1, and 1000 ng/ml.

5) 0.01mol/L PBS buffer.

6) Hit 1000ml of ionized water.

Specimen Collection and Preservation

With the patient's informed consent, a sterile, pyrogen-free, and endotoxin-free test tube was used to draw 4 ml of peripheral venous blood 24 hours, 4 days, 7 days, 14 days, and 21 days after the patient's head injury. Among them, 2ml of venous blood is added to EDTA anticoagulated blood collection tube, which is used to detect the changes in the number of peripheral blood white blood cells and platelets in patients with craniocerebral injury. Another 2 ml of venous blood was added to the procoagulant blood collection tube, the serum was separated, and the specimens were frozen for the determination of the patient's serum CRP and TSP1.

Experimental Subjects

Among the 63 cases of craniocerebral injury, 28 cases were infected, including 25 cases of pulmonary infection, 2 cases of intracranial infection and 1 case of urinary system infection. The patients were grouped according to whether they were co-infected. There were 28 cases in the infection group and 35 cases in the non-infection group. All patients were discharged with Glasgow Outcome Score (GOS). GOS 1-3 is divided into poor prognosis group (including severe disability, plant survival and death), GOS 4-5 is divided into good prognosis group (including good recovery and Chinese disabled). According to GOS, 35 cases were in the good prognosis group and 28 cases were in the poor prognosis group. The patients were further divided into groups according to whether they were operated or not, including 34 cases in the operation group and 29 cases in the non-operation group. In addition, 10 healthy people were selected as controls, including 6 males and 4 females, aged 26-50 years old, with an average age of 38 years old. The age and gender

of the QC controls in the healthy group were not significantly different from those in the patient group.

PLT, WBC Detection

Patients with 2 ml of peripheral venous blood were drawn 24 hours, 4 days, 7 days, 14 days, and 21 days after the injury. Using the Sysmex K4500 hematology analyzer produced by Sysmex, Japan, the patient's peripheral blood PLT (platelets, platelets) was routinely measured at different time points.), the number of WBC (white blood cells).

Enzyme-linked Immunosorbent Assay

(1) CRP (C reactive protein) measurement process

1) Take out the various reagents in the kit, check the reagents, and place them at room temperature for 30 minutes. During the preparation of various experimental instruments such as pipettes, measuring cylinders, beakers, absorbent paper, timers, etc.

2) Specimen dissolution: Take out the frozen specimens from the -80℃ ultra-low temperature refrigerator, place them in a 36±2°C water bath and dissolve them quickly, and use them for testing.

3) Add standards and samples: adjust the pipette to 100μl, take out the microtiter plate, and add 100μl of samples and standards to the microwells in the order of numbering.

4) Add enzyme-labeled conjugate: adjust the pipette to 50 μl, and add 50 μl of enzyme-labeled conjugate to the microwells.

5) Incubation: In the ELISA, the antigen-antibody reaction, that is, after adding the sample and adding the enzyme-labeled conjugate, requires a certain temperature and time, that is, incubating. In this experiment, the ELISA plate was placed in a 36±2℃ water bath and incubated for 60 minutes.

6) Wash 5 times.

7) Color development: adjust the pipette to 60 μl, add 50 μl of color developer A and color reagent B into the microwells, the same. Then shake on a micro shaker for 5 seconds to ensure mixing.

8) Add stop solution: adjust the pipette to 50 μl, add 60 u1 of stop solution to the micro wells respectively, and then shake on the micro shaker for 30 seconds to ensure sufficient mixing and ensure that all the micro wells of the micro well plate are colored blue Turns to yellow.

9) Measuring and reading the absorbance value: warm up the microplate reader for 30 minutes, place the microplate in the microplate reader, set the parameters, and read the values.

TSP1 (thrombospondin l) measurement process

1) Take out the various reagents in the kit, check the reagents, and place them at room temperature for 30 minutes. During the preparation of various experimental instruments such as pipettes, measuring instruments, beakers, absorbent paper, timers, etc.

2) Specimen dissolution: Take out the frozen specimens from an 80'C ultra-low temperature refrigerator, place them in a 36±2°C water bath and dissolve them quickly, and use them for testing.

3) Add standards and samples: adjust the pipette to 50μl, take out the microtiter plate, and add 50μl samples and standards to the microwells in the order of numbering. When adding samples, add the added substance to the bottom of the well of the microplate, avoid adding it to the upper part of the hole wall, and be careful not to splash or generate bubbles. The suction nozzle should be replaced each time the specimen is added to avoid cross-contamination and affect the results.

4) Add biotin conjugate: adjust the pipette to 50 μl, add 50 μl of biotin conjugate to the micro wells, and then shake on the micro shaker for 15 seconds to ensure mixing.

5) Incubation: Place the ELISA plate in a 36±2℃ water bath and incubate for 60 minutes.

6) Add enzyme label conjugate: adjust the pipette to 50 μl, and add 50 μl of enzyme label conjugate to the microwells respectively. The adding rules are the same as above, and then shake on the micro shaker for 15 seconds to ensure mixing.

7) Washing: a. Thoroughly dry the reaction solution in the well; b. Wash it once with the washing solution (after filling the plate with the washing solution, shake it off); c. Soak, that is, to fill the plate with the washing solution and place 1-2 minutes, shaking intermittently, soaking time cannot be shortened arbitrarily; d. Shake off the liquid and pat dry on absorbent paper; e. Repeat operations c and d, and wash 5 times.

8) Color development: then shake on a micro

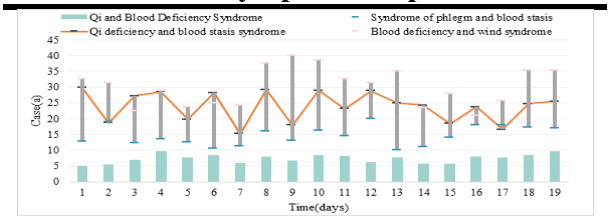
shaker for 5 seconds to ensure mixing.

9) Incubation: Color development is the last step of the ELISA incubation reaction, at which time the enzyme catalyzes the colorless substrate to produce a colored product. The temperature and time of the reaction are still important factors affecting the color development, so after adding the color reagent, the ELISA plate is placed in a 36±2°C water bath and incubated for 15 minutes.

10) Add stop solution: adjust the micropipette to 50 µl, and then add 50 u1 of stop solution to the microwells respectively, and then shake on the micro shaker for 30 seconds to ensure sufficient mixing and ensure that all the microwells of the microplate from blue to yellow.

11) Measure and read the absorbance value: warm up the microplate reader for 30 minutes, place the microplate in the microplate reader, set

Figure 3.
Different symptoms of patients



the parameters, and read the values. The absorbance value of thrombin-sensitive prostaglandin measured with a microplate reader is shown in Table 1.

Table 1.
The absorbance value of thrombin-sensitive prostaglandin measured with a microplate reader

Parameter	1	2	3	4
A	1.079	0.995	1.027	1.066
B	0.987	1.073	1.183	1.056
C	1.172	1.180	1.394	0.929
D	1.059	1.365	0.889	1.370
E	1.251	1.070	1.084	1.033
F	0.596	0.534	1.029	1.102
G	0.750	0.623	0.990	0.815

Statistical Processing

Image prooplus 6.0 was used for immunohistochemical image analysis, and spss25.0 was used for data analysis. The results were expressed as mean ± standard deviation (x ± s). One-way linear regression analysis was performed between groups. $P < 0.01$ means statistically significant intentional difference, $P < 0.05$ means statistically significant difference.

RESULTS AND DISCUSSION

Condition Analysis of Patients with Craniocerebral Injury

This study showed that on the 14th day of severe head injury, there were 35 cases of deficiency of both qi and blood in TCM syndrome, accounting for 50%; 17 cases of phlegm and blood stasis syndrome, accounting for about 24.3%; 13 cases of Qi deficiency and blood stasis syndrome, about accounted for 18.6%; 5 cases of blood deficiency and wind syndrome, accounting for about 7.1%. On the 21st day, there were 40 cases of Qi and Blood

Deficiency Syndrome, accounting for 57.1%; 13 cases of phlegm and blood stasis syndrome, accounting for 18.6%; 10 cases of Qi deficiency and blood stasis syndrome, accounting for 14.3%; Blood deficiency and wind syndrome 7 for example, it accounts for about 10%. On the 28th day, there were 41 cases of blood deficiency syndrome, accounting for 58.6%; 12 cases of phlegm and blood stasis syndrome, accounting for about 17.1%, 10 cases of qi deficiency and blood stasis syndrome, accounting for 14.3%, and blood deficiency syndrome was 7 cases. For example, it accounts for about 10%. The different symptoms of the patient are shown in Figure 3.

Table 2 shows the comparison of the HCT mean (%) among the various syndromes of severe head injury. As shown in Table 2, on the 14th, 21st, and 28th days of severe head injury, the three groups of data are in accordance with the normal distribution, and the variance is homogeneous. According to the q test, the different TCM syndrome HCT (hematocrit (hematocrit)) after a pairwise comparison between the means, it was shown that there was no significant difference between the mean of the TCM syndrome types of HCT ($P>0.05$).

Table 2.
Comparison of mean HCT among various syndrome types of severe head injury (%)

Syndromes	Day 14	Day 21	Day 28
Qi and Blood Deficiency Syndrome	45.1±7.85	42.3±8.64	45. 1±10. 01
Syndrome of phlegm and blood stasis	42.8±6.47	43. 7±6.54	46.2±6.47
Qi deficiency and blood stasis syndrome	44.3±5.76	44.2±9.82	43.3±8.59
Blood deficiency and wind syndrome	41.2±8.55	45.6±8.84	44.2±9.25

Table 3 shows the comparison of the mean PLT (1×10^9/L) between the various syndromes of severe head injury. As shown in Table 3, on the 14,

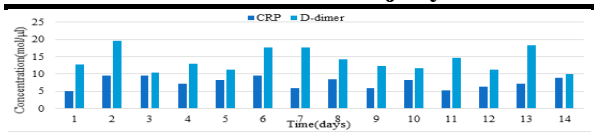
21, and 28 days of severe head injury, the data of each group conformed to the normal distribution, and the variance was homogeneous. After the q test, the mean PLT of each TCM syndrome type was compared in pairs. There was a difference in the mean PLT between the syndrome of stasis, qi deficiency and blood stasis, the syndrome of blood deficiency and wind, and the syndrome of qi and blood deficiency ($P<0.05$), while the mean PLT of phlegm and stasis syndrome and the syndrome of qi deficiency and blood stasis were different there was no significant difference between them ($P>0.05$), and there was no significant difference in the mean PLT of blood deficiency syndromes and Qi-blood deficiency syndromes ($P>0.05$).

Table 3.
Comparison of mean PLT among various syndrome types of severe head injury

Syndromes	Day 14	Day 21	Day 28
Qi and Blood Deficiency Syndrome	165.88±33.19	167.5±35.74	159.5 ± 30.23
Syndrome of phlegm and blood stasis	234.94±11.58	232.9±8.025	228.9±7.034
Qi deficiency and blood stasis syndrome	255.23±13.82	253.1±12.77	250.1±9.36
Blood deficiency and wind syndrome	171.40±15.45	169.7±5.648	168.7±5.54

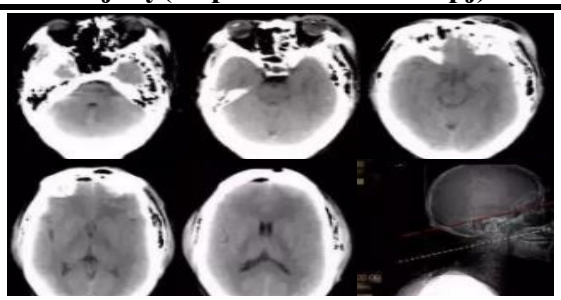
The results of the concentration of CRP and D-2 polymer after craniocerebral injury are shown in Figure 4. After craniocerebral injury, the detection results of CRP and D-2 polymer concentration were the highest on the first day, began to decline on the third day, and continued to decline on the seventh and 14th days. The detection values at each time point were higher than those of the control group. After one-way analysis of variance, $F=12.365$ and $F=17.689$, $P<0.01$, and further by least significant difference (LSD) t-test, the differences between the indicators at each time point and the control group were statistically significant $P<0.01$.

Figure 4.
The results of CRP and D-2 polymer concentration detection after craniocerebral injury



The CT examination of patients with traumatic head injury is shown in Figure 5. Neurological evaluation will be affected by factors such as the patient's age, own health, tolerance, and therapeutic drugs. In addition, in most hospitals, imaging examinations cannot be used for bedside examinations for critically ill or unsuitable patients. In recent years, many scholars at home and abroad have made great progress in basic and clinical research on the mechanism of traumatic brain injury, cerebral edema, secondary inflammation, and coagulation disorders.

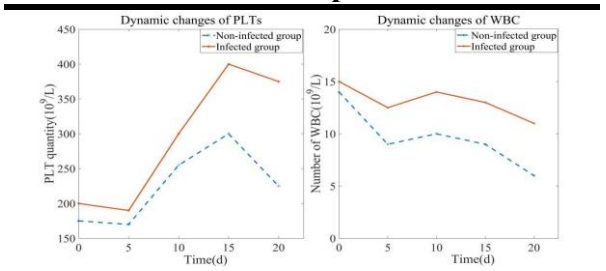
Figure 5.
CT examination of patients with traumatic head injury(http://alturl.com/ssapj)



Comparative Analysis of PLTs and WBCs

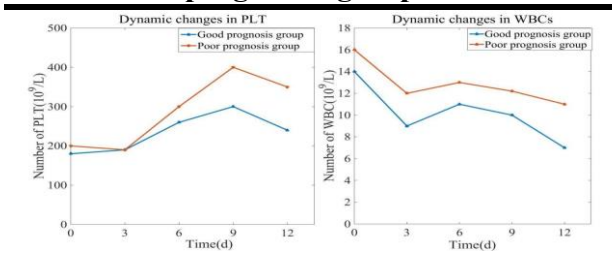
63 cases of craniocerebral injury were grouped and analyzed according to whether they were co-infected or not. It was found that PLTs in the infected group were significantly higher than those in the non-infected group and the normal control group 14-21 days after injury, and sustained high levels in the later stage of the injury ($P<0.01$). Regardless of whether the patient was co-infected or not, WBCs increased significantly within 24 hours ($P<0.01$), and then decreased rapidly in the following 2-4 days. According to the further analysis of co-infection grouping, it was found that the non-infected group had no difference compared with the normal control group at 4 days ($P>0.05$), and it fell below 10×10^9/L, but the infected group was still higher than normal at 4 days The control group ($P<0.05$), and greater than 10×10^9/L, showed a second increase in the following 7-14 days ($P<0.01$), and continued high levels. The comparison of PLTs and WBCs between infected and normal patients is shown in Figure 6.

Figure 6.
Comparison of PLTs and WBCs in infected and normal patients



Further analysis of 63 patients with craniocerebral injury grouped according to the GOS score found that the PLTs of the patients with poor prognosis in the later stage of injury (14-21 days) were significantly higher than those in the good prognosis group and normal control group ($P<0.01$). After the rapid decline of WBCs in 2-4 days, patients with poor prognosis showed a second increase in 7-14 days and continued to be high ($P<0.01$). The comparison of PLTs and WBCs in different prognosis groups is shown in Figure 7.

Figure 7.
Comparison of PLTs and WBCs in different prognostic groups



CONCLUSIONS

This study mainly explores the dynamic changes and clinical significance of white blood cells (WBC) and platelets (PLT) in patients with traumatic brain injury. During the specimen collection process, a sterile, pyrogen-free, and endotoxin-free test tube was used to draw 4ml of peripheral venous blood 24 hours, 4 days, 7 days, 14 days, and 21 days after the patient's head injury. Among them, 2ml of venous blood is added to EDTA anticoagulated blood collection tube, which is used to detect the changes in the number of peripheral blood white blood cells and platelets in patients with craniocerebral injury. Another 2 ml of venous blood was added to the procoagulant blood collection tube, the serum was separated, and the specimens were frozen for the determination of the patient's serum CRP and TSP1. Glasgow prognostic score was performed on all patients at discharge. In the detection of PLT and WBC, the Sysmex K4500 blood cell analyzer is used to routinely determine the number of PLT and WBC in the peripheral blood of patients at different time points. During the CRP measurement process, adjust the pipette to 60μl, and add 50μl of developer A and developer B into the microwells, the same. Then shake on a micro shaker for 5 seconds to ensure mixing. Warm up the microplate reader for 30 minutes, place the microplate in the microplate reader, set the parameters, and read the values. When measuring TSP1, adjust the micropipette to 50 μl, and then add 50 μl of stop solution to the micro wells respectively, and then shake on the micro shaker for 30 seconds to ensure sufficient mixing. This study is helpful for timely treatment of patients with traumatic brain injury.

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