Changes of Blood Leukocyte and Blood Glucose in Patients with Acute Craniocerebral Trauma and Nursing Effect

Junyi Yang

*Junyi Yang, Department of Nursing, The Second Affiliated Hospital of Shenyang Medical College, Shenyang 110035, Liaoning Province, China, *Corresponding author: Junyi Yang, No. 64 Qishan West Road, Huanggu District, Shenyang City, Liaoning Province, China, Email: (hlpx2015@126.com)*

Acute craniocerebral injury is one of the most common injury types, and its mortality rate ranks first among all kinds of injuries. The changes of white blood cells and blood glucose play an important role in the treatment and nursing of patients. The purpose of this study is to analyze the changes of white blood cells and blood glucose in patients with acute craniocerebral trauma and nursing effect. In this study, 65 patients with craniocerebral injury admitted to the Department of Neurosurgery of our hospital from January 2017 to September 2019 were selected as the research objects. After admission, appropriate nutritional treatment was given according to the condition of patients. Blood samples were collected on fasting in the morning of 2, 8 and 15 days after admission, while those in the control group were collected by fasting vein for detection of white blood cells, blood glucose, blood lipid, blood uric acid and other indicators. There was no significant difference in the number of neutrophils in each group ($P < 0.05$, $P < 0.05$, $P < 0.05$, $P < 0.05$, $P < 0.05$). After acute traumatic brain injury, the white blood cells and blood glucose of patients increased, and the more severe the degree of brain injury, the higher the white blood cell count and blood glucose. This study will contribute to the analysis of white blood cell and blood glucose changes and nursing care of patients with craniocerebral injury.

Keywords: Craniocerebral Injury, White Blood Cell Count, Blood Glucose Change, Medical Care
Tob Regul Sci.™ 2021;7(5): 1670-1680
DOI: doi.org/10.18001/TRS.7.5.90

Craniocerebral trauma is a common and serious disease in emergency department. The disability rate and mortality rate are very high. Trauma not only directly causes damage to the body, but also has an important impact on the survival and prognosis of patients. Acute traumatic coagulation disorder (ATC) is known as acquired coagulation disorder, early traumatic coagulation dysfunction and acute traumatic coagulation dysfunction. It has been described with simple head injury (ITBI) for a long time. In the initial studies, ATC was thought to be caused by the consumption of coagulation factors in chronic intravascular coagulation (DIC), but the pathophysiology of ATC is still under debate.

Acute craniocerebral injury will increase the morbidity and mortality in peacetime and war. Generally speaking, the incidence rate is second only to limb fracture, and the mortality rate is the first. Previous studies have confirmed that the function of the anterior pituitary will change greatly after traumatic brain injury.

In the study of the changes of blood leukocyte blood glucose in patients with acute craniocerebral trauma, the study of Du X was to explore the dynamic changes of serum α - melanocyte stimulating hormone (α - MSH) in patients with traumatic brain injury.He used enzyme-linked immunosorbent assay (ELISA) to measure serum α - MSH and tumor necrosis factor (TNF) - α levels, and Pearson correlation analysis was used to analyze the correlation between α - MSH and TNF - α. His method is not stable [1]. Objective to investigate the protective effect of

dexmedetomidine (DEX) on rats with severe brain injury. He divided 120 male Sprague Dawley rats into dexamethasone group, sham operation group and control group randomly. The acute severe brain injury model was established by hydraulic percussion method, and the rats were treated 2 hours after injury. The behavioral changes were observed before and after modeling. The accuracy of his method is low [2]. Objective to investigate the protective effect of Jiang Won patients with severe craniocerebral injury during perioperative period. Methods 80 adult male SD rats were divided into dexmedetomidine injury group (experimental group) and sodium chloride injury group (control group), with 40 rats in each group. The modified Feeney free fall method was used to establish the model of severe craniocerebral injury in the two groups, his method is not practical [3].

This study first introduced the basic situation and mechanism of craniocerebral injury, and elaborated the surgical and non-surgical treatment methods of craniocerebral injury. The study continues to explain the risk factors for white blood cells and brain damage, as well as the role of automatic blood analyzers. This article also introduces the coagulation mechanism, treatment and nursing care after craniocerebral injury. In this study, 65 patients with traumatic brain injury in our hospital from 2017 to 2019 were selected as the research objects. The blood samples of patients were detected, and the changes of white blood cells and blood glucose were observed and analyzed by leukocyte genomic DNA extraction and leukocyte telomere calculation. Through the analysis of the results, the results of serum total protein in different periods were analyzed, blood glucose and white blood cell count and their changes were analyzed, and the changes of white blood cells and blood glucose between different hematoma volumes were analyzed. The conclusion is that brain injury can lead to increased white blood cells and blood glucose levels.

CRANIOCEREBRAL INJURY AND NURSING

Brain Injury

Brain injury (TBI) is a general trauma, which may occur alone or other injuries at the same time. Now, for various reasons, the incidence of TBI is increasing, which is a serious problem for doctors all over the world. The mortality of TBI is very high, and the prognosis of the most severe patients is poor. In China, more than 60000 people suffer from TBI every year, and the death toll may reach 100000, bringing millions of economic losses to society and families. Large scale retrospective analysis showed that TBI patients had the first mortality and disability rates. Brain injury includes one-time and two-time. The former occurs at the time of injury and cannot be avoided. Methods to reduce secondary damage are now the focus of discussion. The intracranial causes of secondary injury include brain edema, high intracranial pressure, vasospasm, etc. Systemic causes include hypoperfusion, hypoxemia, fever, increased blood glucose, abnormal blood coagulation, etc. Among them, coagulation dysfunction as an independent risk factor of death in trauma patients has caused a lot of attention [4, 5].

Common Mechanisms of Craniocerebral Injury

The injury caused by external forces directly acting on the skull and brain tissue is called primary cranial brain injury. The external force mechanism determines the degree and type of head injury, and is affected by the time and effect of the action. According to their different effects, craniocerebral injury can be divided into three categories: indirect violence injury, direct violence injury and head contusion.

The severity of secondary head injury mainly depends on the amount of intracranial hematoma and the degree of traumatic edema. Traumatic brain edema has a very complex formation mechanism, the specific mechanism of stroke is still unclear, need further investigation [6, 7].

Treatment of Craniocerebral Injury

Surgical treatment

At present, the most commonly used surgical treatment for patients with intracranial injury is craniotomy and craniotomy decompression. These two methods can effectively reduce intracranial pressure in most cases. In order to save the lives of

patients, patients with obvious brain swelling and malignant brain tissue swelling can choose bilateral craniotomy decompression. Some scholars pointed out that for patients with severe craniocerebral injury or severe craniocerebral injury, craniotomy is suitable for standard large bone flap craniotomy. Intraoperative intracranial pressure decreased rapidly, but it may continue to decline. The effective tissue perfusion increased, the blood supply of brain tissue and the degree of cerebral blood oxidation were improved, and the prognosis of patients was improved. Of course, some scholars believe that bilateral forebrain craniotomy is more suitable for the treatment of adult patients with severe brain injury, which greatly improves the survival rate of patients and improves the prognosis of patients. The project shortened the length of stay in ICU and reduced the economic burden of patients' families, but the proportion of patients with poor prognosis increased. Standard decompression and decompression have been proved in many clinical treatments in patients with craniocerebral injury. This surgical method can improve the prognosis of patients and improve the success rate of rescue. This is the main method of neurosurgery. For patients with craniocerebral injury, surgical treatment is the most important link in the whole treatment process, and other links are also very important, which also affects the prognosis and nursing of patients with non-surgical treatment[8].

(2) Non operative treatment

The purpose of non-surgical treatment of patients with craniocerebral injury is to reduce the degree of brain edema, reduce the water content in brain tissue, and finally achieve the goal of eliminating edema. In order to reduce the intracranial pressure of patients, lumbar puncture or ventricular puncture puncture can also achieve this goal. Dehydration is the most common treatment for patients with craniocerebral injury. Glycerin fructose alcohol and mannitol solution are widely used in clinical application. For patients with craniocerebral injury, neurotrophic drugs play an important role. The main mechanism of neurotrophic drugs is to protect the nerve cells of patients and promote the functional recovery of damaged cells. From the perspective of drug selection, licorice can be determined according to the state of patients or the combined use of multiple drugs [9, 10].

Relationship between Leukocytes and Risk Factors of Craniocerebral Injury

The risk factors of white blood cells and brain injury are high blood pressure, diabetes, obesity and hyperlipidemia. Hypertension, diabetes, obesity and hyperlipidemia are important risk factors for brain injury or poor prognosis. Among them, hypertension is the main risk factor of stroke. Recently, the systematic analysis of the worldwide burden of disease in foreign countries indicates that 73% of the disease burden of head injury may be caused by hypertension. But the cause of hypertension is not clear. Previous studies have shown that inflammation can cause endothelial dysfunction and blood pressure may rise further. Epidemiological studies have also studied the relationship between white blood cells and hypertension. Hypertension is associated with an increase in the number of white blood cells. If the number of white blood cells increases, the risk of hypertension will increase. Similarly, the society investigated the relationship between white blood cell count and the risk of hypertension and the cumulative risk of hypertension in 15 years. After adjusting for other risk factors, the increase of white blood cell count was also related to the risk of hypertension and disease [11, 12].

Accuracy of WBC Count by Automatic Blood Analyzer

By analyzing the test results of XE-5000 automatic blood analysis device, compared with manual detection method of white blood cell count in joint fluid, it can be seen from table 1 that XE-5000 automatic blood analysis device is compared with manual method. The difference between the two results was $p > 0.05$, and the difference was not statistically significant. According to the linear regression analysis of automatic blood analysis device and manual counting method, the R value between the two was 0.821, showing a good correlation. However, 80%

of the patients, 50% and 66.6% of the patients with femoral neck fracture, femoral head necrosis and correction surgery had significant difference (50% difference) between the two counting results. In this category of patients, automatic blood analysis device was used for detection, and the accuracy of clinical diagnosis was improved. In order to prevent missed diagnosis and misdiagnosis, manual calculation was needed. XE-5000 automatic blood analysis device includes a variety of detection modes and a new generation of blood cell detection and analysis device with detection channels. In this mode, only 200 μ l of joint fluid is needed. The samples can be tested.

Coagulation Mechanism after Craniocerebral Injury

With the increase of intracranial hemorrhage after TBI, in many cases, the rapid development of blood from hypercoagulability state to low coagulation state will cause hemorrhage. Brain injury can lead to the release and consumption of coagulation factors and the increase of intracranial hemorrhage. The pathophysiological mechanisms of coagulation disorder after traumatic brain injury include platelet dysfunction, endothelial cell activation, fibrinogen changes, inflammation and hyperfibrinolysis, which may lead to the development of intracranial hemorrhage. These institutional hypotheses are mainly based on the relevant research of various variables, and causality has not been established.

Treatment and Nursing of Craniocerebral Injury

For patients with acute craniocerebral trauma, we should carefully detect pulse, respiration, blood pressure, consciousness, pupil, accurately implement GCS score, and strengthen the basic nursing of respiratory tract, digestive tract, various drainage pipes and various complications. In particular, it is necessary to monitor blood glucose and blood circulation according to the state, report abnormalities to doctors, and formulate corresponding nursing countermeasures. According to the doctor's instructions, antibiotics should be used timely and accurately. According to the changes of blood glucose, doctors can adjust the dosage of insulin and the concentration and dosage of sugar solution. Control the use and use of sugar water. Observe the drug reaction carefully. For patients with nasal nutrition and diet, the ratio of total calories to protein, fat and carbohydrate in a day must be calculated according to the patient's standard weight and physiological status. In addition, patients should be instructed not to drink sugary drinks and food, and the intake and output of water in 24 hours should be recorded. To control hyperglycemia, observe the balance of water and electrolyte. In the case of hyperosmolar coma, 150-200 ml of cold water will be provided from the nasogastric tube every 1-2 hours.

Helping patients to build self-confidence is the key to psychological nursing. Medical staff must make patients understand that, through continuous improvement of people's lives and continuous improvement of the disease, reliable guarantee of effective TB countermeasures is provided. "Preventive measures" of the disease, if early detection, regular medication, regular reassessment, can be cured. Use words of encouragement to fully identify what the patient has done and improve the patient's trust in overcoming the disease.

Due to the warm reception of patients in hospital, the patients feel warm and at the same time, the relationship with the nurses and patients of the patients is also gradually deepened, which deepens the understanding of medical treatment and the psychological state is also well understood. Please talk with them more and more, care and care in life, comfort them in various ways, compare their inner feelings, experience difficulties, convey their understanding of the disease, and talk about their attitude towards life. Deal with psychological entanglement, stimulate the resonance of patients, eventually lose the burden, rejuvenate themselves, and actively assist in treatment.

Medical Model Scoring Method

Simple moving average method:

$$F_{t+l} = \frac{\sum_{i=t-n+1}^{r} A_i}{n} (1)$$

F_{t+1} is the demand forecast of period t + 1; n is the number of time periods for calculating moving

average (here n = 4); A_i is the actual demand in period I; w_i is the weight of period I ($\sum \omega_i = 1$).

Weighted moving average method:

$$F_{t+1} = \sum_{i=t-n+1}^{t} \omega_i Ai (2)$$

The Experiment of Detecting Blood Glucose and Leukemia after Craniocerebral Injury

Subjects

65 patients with craniocerebral injury admitted from January 2017 to September 2019 were selected. Selection criteria: hospitalization within 3 days after injury, massive hemorrhage, no shock; no severe combined injury; cerebrovascular disease, epilepsy, no history of diabetes, no severe heart, liver, no history of kidney disease; no history of intracranial disease, other organ infections, 18 to 65 years old, except for those with malnutrition and without immunosuppressants. All patients did not appear persistent high fever and multiple organ dysfunction syndrome after injury, and survived for more than 15 days. At the time of hospitalization, the patients were divided into groups according to Glasgow's criteria. Patients with scores of 3-8 were considered as severe head injuries, patients with scores of 12 were moderate head injuries, and patients with scores above 13 were considered as mild head injuries. At the same time, 20 healthy people with the same age as the physical examination center of our hospital were randomly selected as the control group.

Experimental Design

After hospitalization, the patients received appropriate nutritional therapy according to their status. Blood was drawn from the vein on the second, eighth and fifteenth days after hospitalization. Blood was drawn from the vein on an empty stomach in the control group. Serum total protein, albumin and prealbumin were determined. The plasma levels of IL. 6, TNF. A and benzoic acid were measured by ELISA. The collected data were processed by SPSS16.0 software. The statistical explanation was expressed by the mean standard deviation. $P < 0.05$ was considered to be statistically significant.

At the time of hospitalization, the score will be given according to Glasgow grade standard. The score of 3-8 points was severe head injury, 9-12 points was moderate head injury, and more than 13 points was mild head injury. According to the score, the patients were divided into three groups: mild, moderate and severe. At the same time, 20 healthy people aged 18-65 were randomly selected as control group. As shown in Table 1, Glasgow Coma Score.

Table 1.
Glasgow Coma Score

Eyes open	Score	Language response	Score	Motor response	Score
Automatic eye opening	4	Correct answer	5	Correct execution	6
Call to open your eyes	3	Wrong answer	4	Location of tingling pain	5
Tingling open eyes	2	incoherent speech	3	Tingling retraction	4
No response	1	Only pronunciation	2	Limb reaction	3
		No response	1	Limb hyperextension	2
				No response	1

Sample Preparation

The standard serum was used to make tips alone. When modeling each model, all samples must be mixed (active control and control). All samples were randomly arranged using the rand function in Excel. Each chip contained seven samples and one standard serum (the standard serum was also randomly placed on the chip), and each model was added with empty control (using deionized water instead of sample).

The samples were melted on ice (30 minutes to 1 hour), centrifuged at 10000 rpm for 2 minutes, and the samples were arranged in the order of the experiment plan (on ice). Take 96 wells of cell culture plate, put it into the refrigerator, add 10 UL of processed serum to each well (directly Pierce, stick on the wall, do not touch the bottom), and vibrate with 10ml 600rpm for 5 minutes.

Experimental Methods

Extraction of genomic DNA from leukocytes

Collect the basic data of the research object. In the morning of the next day, the blood pressure of the subjects was tested. 4ml venous blood was taken from fasting to detect blood glucose, blood lipid, uric acid and other indicators. At the same time, blood samples were collected with edta-k3 anticoagulant tube, centrifuged at 3000 rpm for 5 minutes. The upper serum, the lower layer of blood cells and the upper layer of serum were put into EP tube, and the blood cells were frozen in - 80 ° C cryopreservation box. Please extract the genomic DNA of venous leukocytes and follow the instructions.

Calculation of relative telomere length of leukocytes

The relative telomere length (T / s) = 2 - [CT (telomere) - CT (β - globin) sample] / 2 - [CT (telomere) - CT (β - globin) standard].

Determination of albumin

Albumin was quantified by brominated sol green method. Plasma albumin was positively charged with buffer memory at pH 4.2. In the presence of nonionic surfactants, a green complex with 630 rim absorptions can be formed by combining it with a negatively charged pigment, namely, cresol bromide green. Compared with the albumin standard of the same treatment, the albumin content in plasma can be obtained.

Determination of IL-6

Enzyme linked immunosorbent assay (ELISA) was used. Human leukocyte-6 (IL-6) samples, standards, hi labeled detection antibodies were added to the microl pre coated with capture antibody in turn, and then were completely cleaned by incubate. The color of TMB is the color of TMB, which turns blue and finally yellow according to the catalysis of acetamide. There was a positive correlation between the intensity of color and human leukocyte 6 (IL-6) in the samples. The absorbance (OD value) was measured at 450 removable wavelengths using a microplate reader. The concentration of human IL-6 in the sample can be obtained by drawing a standard curve.

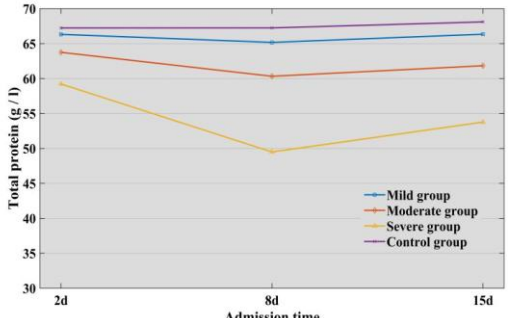
Detection Steps

Remove the necessary padding from the aluminum foil bag that has been balanced at room temperature for 20 minutes and put the remaining pad in the zipper bag to recover to 4 ℃. The standard well and sample well were set as the standard with different concentrations of 50ml. After adding 10ml of test sample, the sample was added to the sample hole of 40ml sample diluent. The reaction hole was sealed with sealing film, and it was carried out in a 37 ℃ water bath or indoor baking machine for 60 minutes. Throw away the liquid, gently tap with absorbent paper, fill each hole with cleaning solution, let stand for 1 minute, throw off the cleaning fluid, gently tap with absorption paper, and clean repeatedly for 5 times. 50 ml of matrix A and B were added to each well and incubated in the dark at 37 ℃ for 15 minutes. Add 50ml of stop liquid in each well within 15 minutes, and measure the OD value of each well with the wavelength of 450rim.

ANALYSIS OF THE CHANGES OF LEUKOCYTE AND BLOOD GLUCOSE IN PATIENTS WITH CRANIOCEREBRAL INJURY

Analysis of Serum Total Protein Results at Different Time Points

Figure 1.
Dynamic changes of serum total protein levels in patients with traumatic brain injury in each group



As shown in Table 2, serum total protein levels were compared at different time points after admission.

From Table 2 compared with the normal

control group, the total serum protein level of patients with craniocerebral trauma in each group decreased on the 2nd, 8th and 15th day after hospitalization, and the difference between groups was significant (F = 5.545, 33.522, 18.292, P < 0.01). All groups had the lowest decrease on the 8th day after hospitalization and increased on the 15th day, but still lower than usual. The overall level of serum total protein in patients with severe traumatic brain injury was lower than that in mild and moderate groups (P < 0.05), and that in moderate group was lower than that in mild group (P < 0.05). As shown in Figure 1, the dynamic changes of serum total protein levels in patients with traumatic brain injury in each group.

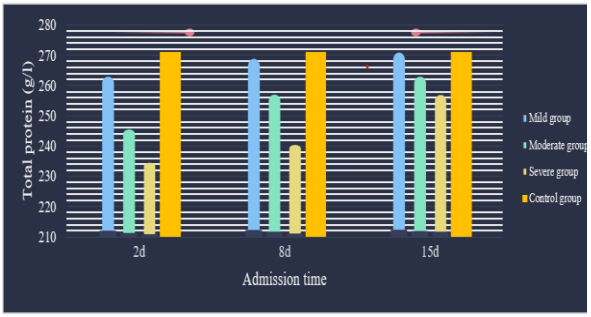
Table 2.
Comparison of serum total protein levels at different time points after admission (g / L)

	2d	8d	16d
Mild group	66.33±7.66	65.18±7.84	66.35±7.42
Moderate group	63.76±6.21	60.33±5.79	61.84±5.46
Severe group	59.23±9.44	49.50±7.42	53.77±8.24
Control group	67.24±3.99	67.25±4.01	68.11±3.89

As can be seen from Figure 1, the serum total protein level of the severe group on the second day of hospitalization was significantly lower than that of the normal control group and the mild group (P < 0.05), and the lowest on the eighth day: compared with the normal control group, the serum total protein level of the moderate severe group and the severe group was significantly decreased (P < 0.01). The serum total protein level of patients with moderate and transferred traumatic brain injury was also significantly lower than that of mild group (P < 0.05). The serum total protein level of severe group on the 15th day was higher than that on the 8th day. However, the serum total protein level of normal control group, mild group and moderate group (P <0.05) was significantly lower than that of mild group. Other groups (P > 0.05).

As shown in Figure 2, the serum prealbumin levels of each group were compared at different time points after admission.

Figure 2.
Comparison of serum prealbumin levels in different groups at different time points after admission

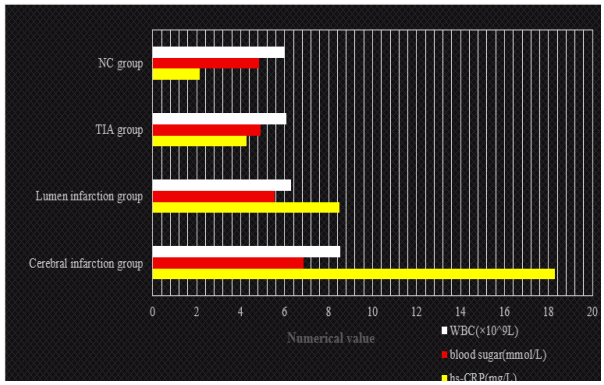


According to Figure 2, compared with the normal control group, the serum prealbumin level of patients with traumatic brain injury in each group decreased on the second day after admission, increased on the eighth day, and returned to the normal level on the 15th day. The results of inter group comparison showed that there was significant difference between the groups on the 2nd and 8th day after admission (F = 4.088, 3.069; P = 0.009, 0.032 < 0.05), but there was no significant difference between the groups on the 15th day (F = 0.646, P = 0.588 > 0.05).

On the second day after hospitalization, the concentration of prealbumin in the severe group was significantly lower than that in the control group and mild group (P < 0.05), but there was no significant difference between the two groups (P > 0.05). The difference between the moderate group and the control group was obvious. Even on the 8th day, the serum albumin level of the severe group was significantly lower than that of the moderate disease group and the mild disease group, which may be the cause of the patient's severe state. Prealbumin levels did not return to normal levels. On the 15th day, the level of prealbumin in the severe group was lower than that in the other groups, but there was no statistical difference (P > 0.05), indicating that the recovery of prealbumin level can be achieved through moderate nutritional treatment.

Analysis of Blood Glucose and White Blood Cell Number in Serum of Craniocerebral Injury

Figure 3.
Comparison of serum Hs-CRP, blood glucose and white blood cell count in each group



As shown in Figure 3, the comparison of serum hs CRP, blood glucose and white blood cell count in each group.

It can be seen from Figure 3 that the comparison of serum hs CRP, blood glucose and white blood cell count in each group is shown in Figure 4. The serum hs CRP, blood glucose and white blood cell count in cerebral infarction group were significantly higher than those in the other three groups (all $P < 0.05$). The serum hs CRP in the cavity infarction group was significantly higher than that in the TIA group and NC group, and that in the TIA group was significantly higher than that in the NC group (all $P < 0.05$).

CRP is a kind of acute phase reaction protein, when tissue injury, infection, inflammation, serum concentration increases sharply, can be used as a sensitive indicator of inflammatory response. In this study, the levels of serum hs CRP in patients with ICVD from high to low were cerebral infarction group, lumen infarction group, TIA group, and were significantly higher than NC group (all $P < 0.05$). This suggests that acute ICVD is an inflammatory process in vivo, and the serum HS CLP level reflects this state to some extent. In addition, the blood glucose and white blood cell count in the cerebral infarction group were significantly higher than those in the other three groups, which may be related to the extensive field of cerebral infarction and severe brain tissue damage. In conclusion, the changes of serum hsCRP, blood glucose and white blood cell count in patients with acute ICVD reflect the state of patients to a certain extent. The use of target therapy can reduce the brain injury and improve the prognosis of patients with ICVD.

Analysis of Changes of White Blood Cells and Blood Glucose in Different Therapeutic Groups of Craniocerebral Injury

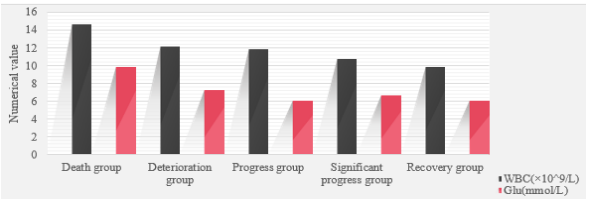
The changes of leukocyte count, neutrophil and blood glucose in different therapeutic groups are shown in Table 3. (WBC refers to white blood cells, n refers to neutrophils, and Glu refers to blood glucose level)

Table 3.
Changes of WBC, N and Glu in different therapeutic groups

Group	Total	WBC(×10⁹/L)	N(%)	Glu(mmol/L)
Death group	17	14.67±4.21	83.07±8.66	9.86±5.37
Deterioration group	14	12.13±5.25	82.19±6.15	7.28±3.64
Progress group	18	11.84±4.09	77.55±6.79	6.02±1.37
Significant progress group	27	10.77±4.53	71.03±12.94	6.63±1.33
Recovery group	33	9.84±2.43	70.66±11.02	6.05±1.22
Normal value		4~10	50~75	3.92~6.16

It can be seen from Table 3 that the F values of leukocyte count, neutrophil count and blood glucose were 4.06, 5.66 and 6.87 respectively, $P < 0.01$. It can be considered that there are significant differences in the number of leukocytes, neutrophils and blood glucose among different prognosis groups. With the increase of the degree of deterioration, the values of the three indicators also increase. As shown in Figure 4, white blood cells and blood glucose changes.

Figure 4.
Changes in leukocytes and blood glucose values



The results of Figure 4 show that acute traumatic brain injury is often accompanied by the increase of white blood cell, mesoglobulin and blood glucose, with the increase rates of 59.6%, 60.6% and 48.6%, respectively. The higher the value of the three indicators, the more blood loss, the greater the damage to the brain, on the contrary, the state will gradually return to normal. This mechanism is not only related to the injury of the inferior colliculus, but also to the pressure response. Under pressure, the granulospheres in the bone marrow storage pool are mobilized and released, and the granular balls in the marginal groove enter the human blood circulation. As a result, the increased granulocytes almost turn into mature leaves. In this granulocyte or more mature rod granulocyte group, the number of neutrophils increased in most patients, and the number of good and medium cells also increased. Of course, this may also be related to the direct or indirect release of specific substances after acute traumatic brain injury.

In this study, the initial blood glucose rise rate of acute traumatic brain injury reached 48.6%. In addition to the death group, the fasting blood glucose level of survivors returned to normal after repeated examination before discharge. It is speculated that the cause of the increase of blood glucose is caused by stress reaction.

The mortality and deterioration rate increased significantly with the increase of WBC, neutrophils and blood glucose at the early stage of the disease. When WBC > 12×10^9 / L, n > 0.85, Glu > 10 mmol / L, the prognosis was poor. Therefore, paying attention to the changes of white blood cells and blood glucose is helpful to observe the state and judge the results.

Analysis of Leukocyte and Blood Glucose Changes among Different Hematoma Volumes

As shown in Table 4, the changes of WBC and blood glucose in different hematoma volume groups were compared.

Table 4.
Comparison of WBC and blood glucose in different hematoma volume groups

	Number of cases	WBC($\times10^9$/L)	Glu(mmol/L)
0~30ml group	28	20.64±8.02	8.90±2.98
31~60ml group	11	16.81±4.12	5.97±0.94
≥60ml group	11	13.09±5.74	5.98±1.11

It can be seen from Table 4 that the number of blood cells and blood glucose in patients with acute brain injury increased significantly. In general, catecholamine and cortisol in the blood increase during acute craniocerebral injury due to changes in the body's stress state and central nervous system function. The rise of paediatric olamine may cause a large number of white blood cells in the marginal cistern to be discharged into the blood flow, while the rise of cortisol will cause a large number of good mesosphere preserved in the bone marrow to flow out into the blood. Brain contusion or brain laceration may cause cerebral microcirculation disorder, which will gather in the damaged part of blood cells, further worsen the local cerebral ischemia, and may cause brain edema. The more severe the brain damage, the more serious the reaction. As a result of overreaction, the pellet will release inflammatory medical institutions, which will worsen brain tissue damage[13].

This study shows that in addition to the increase in the number of white blood cells, there is also a significant increase in blood glucose after traumatic brain injury. This may be the cause of severe damage to brain tissue, body pressure response to impaired glucose metabolism, or damage to the hypothalamus and pituitary system. Blood glucose increased. Its occurrence is related to the injury of pituitary stem, the injury of hypothalamus and hypophysis. During stress response, in order to ensure the function and metabolism of important tissues such as the heart and brain, the body will limit the glucose metabolism of other tissues. Increase blood sugar level. The increase of paediatric amine will lead to the increase of glucose secretion and decrease the secretion and function of insulin. Cortisol will reduce the utilization of glucose in tissues. These factors promote the rise of blood glucose.

CONCLUSION

This study shows that the more severe the brain injury, the higher the pituitary hormone level after injury, and the dynamic observation will also drop sharply, almost all below the normal value. The death group, severe and moderate group replacement group, and brain contusion group were the same. The increase of pituitary hormones is small in the patients with low severity of the brain, and the dynamic changes gradually decrease. Most of them are in the normal range. The dynamic changes of pituitary hormones in patients with craniocerebral injury can be used as an important prognostic indicator. Pituitary hormone increased significantly after injury and decreased significantly after 48 hours, which can be considered as an indicator of severe injury and poor prognosis.

The study showed that the more severe the brain injury, the higher the white blood cell count and blood glucose level. The average WBC of patients with severe and severe brain injury in gcs3-5 and 6-8 groups was 20.00×10^9 / L (the normal value of WBC was $4.00\text{-}10.00\times10^9$ / L). The average blood glucose level was above 8 mmol / L (usually 3.9-6.3 mmol / L). There was significant difference between gcs9-12 and 13-15 subgroups ($P < 0.05$). The increase of white blood cell count and blood glucose value is related to the prognosis of patients. This indicated that WBC (22.28×10^9 / L) and blood glucose (10.24 mmol / L) in death group were significantly higher than those in ineffective group (13.07×10^9 / L and 5.98 mmol / L) and good group (18.05×10^9 and 7.12 mmol / L) ($P < 0.05$). These results suggest that the increase of white blood cell count and blood glucose in patients with acute head injury can be used as an important indicator to judge the prognosis of patients.

The sum of the pituitary hormone values detected shortly after hospitalization and on the third day after hospitalization is an important index to evaluate injury and prognosis. When the ratio is greater than 3 or the total is greater than 10, it is an important indicator of severe injury and poor prognosis. On the other hand, when the ratio is less than 1.5 or the total is less than 8, the injury is mild and the prognosis is good. The increase of white blood cell number and blood glucose value caused by acute brain injury is related to many clinical factors: the increase of white blood cell number and blood glucose value is related to the degree of brain injury.

REFERENCES

1. Du X, Dong B, Li C, et al. Dynamic changes of α-melanocyte-stimulating hormone levels in the serum of patients with craniocerebral trauma. Exp Ther Med. 2017/09/01 2017;14(3):2511-2516. doi:https://doi.org/10.3892/etm.2017.4793
2. Liao X, Liu Y, Yang L, Wu W, Zhou M. Protective effect and mechanism of hydrochloride dexmedetomidine on the severe craniocerebral trauma in rats. International Journal of Clinical and Experimental Medicine. 2017;10(5):8522-8528.
3. Jiang W-W, Wang Q-H, Liao Y-J, Peng P, Xu M, Yin L-X. Effects of dexmedetomidine on TNF-α and interleukin-2 in serum of rats with severe craniocerebral injury. BMC Anesthesiology. 2017/09/20 2017;17(1):130. doi:https://doi.org/10.1186/s12871-017-0410-7
4. Grelat M, Madkouri R, Bousquet O. Acute isodense subdural hematoma on computed tomography scan – diagnostic and therapeutic trap: a case report. Journal of Medical Case Reports. 2016/02/24 2016;10(1):43. doi:https://doi.org/10.1186/s13256-016-0822-x
5. Zemek R, Barrowman N, Freedman SB, et al. Clinical Risk Score for Persistent Postconcussion Symptoms Among Children With Acute Concussion in the ED. JAMA. 2016;315(10):1014-1025. doi:https://doi.org/10.1001/jama.2016.1203
6. Mesa Galan LA, Egea-Guerrero JJ, Quintana Diaz M, Vilches-Arenas A. The effectiveness and safety of pharmacological prophylaxis against venous thromboembolism in patients with moderate to severe traumatic brain injury: A systematic review and meta-analysis. Journal of Trauma and Acute Care Surgery. 2016;81(3):567-574. doi:https://doi.org/10.1097/TA.0000000000001134
7. Clauberg R, Hattingen E. Schädel-Hirn-Trauma. Craniocerebral Injuries. Radiologie up2date. // 24.11.2017 2017;17(03):215-226. doi:https://doi.org/10.1055/s-0043-117790
8. Brawanski N, Baumgarten P, Konczalla J, Seifert V, Senft C. Cerebral foreign body granuloma in brain triggering generalized seizures without obvious craniocerebral injury: A case report and review of the literature. Surgical neurology international. 2016;7(Suppl 29):S775-S778. doi:https://dx.doi.org/10.4103%2F2152-7806.193924
9. Silverberg ND, Panenka W, Iverson GL, et al. Alcohol Consumption Does not Impede Recovery from Mild to Moderate Traumatic Brain Injury. Journal of the International Neuropsychological Society. 2016;22(8):816-827. doi:https://doi.org/10.1017/S1355617716000692
10. Van Deynse H, Van Belleghem G, Lauwaert D, et al. The incremental cost of traumatic brain injury during the first year after a road traffic accident. Brain Injury. 2019/07/29 2019;33(9):1234-1244. doi:https://doi.org/10.1080/02699052.2019.1641224
11. Komol'tsev IG, Levshina IP, Novikova MR, Stepanichev MY, Tishkina AO, Gulyaeva NV. Changes in the

Cerebral Cortex after Dosed Craniocerebral Trauma in Rats of Different Ages. Neuroscience and Behavioral Physiology. 2016/09/01 2016;46(7):826-832. doi:https://doi.org/10.1007/s11055-016-0317-7

12. Lang JM, Krauss JK. Denied Suicide Attempt: Penetrating Cranial Cerebral Injury with a Nail Gun. Indian Journal of Neurotrauma. // 07.09.2017 2017;14(01):035-038. doi:https://doi.org/10.1055/s-0037-1602721

13. S. Arul Pragassame, V. K. Mohandas Kurup JK. Efficacy of Sustained Natural Apophyseal Glides Mulligan Technique on Mobility and Function in Patients with Cervical Spondylosis: An Experimental Study. J Nat Sci Biol Med. 2020;11(2):128-134. doi:10.4103/jnsbm.JNSBM