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Glasgow Coma Scale Improvement after Lidocaine Infusion in Moderate Traumatic Brain Injury

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Abstract

Since the Glasgow Coma Scale was developed 40 years ago it has been accepted throughout the world as a method for assessing impaired consciousness.

Fourty moderate TBI patients were simple randomly divided into 2 groups: group A take normal saline infusion without lidocaine as control group (N=20), and group B take normal saline with lidocaine at rate 1 mg/kg/BW infusion as treated groups (N=20). Serum samples were collected at before induction and 2 hours after giving infusion treatment to measure serum IL-6 and PLA2 level, and lidocaine serum concentration. GCS were measured before induction and after 24 hours surgery. Data were analyzed using analytic statistic with paired t test to measure serum IL-6, PLA2, & lidocaine serum level before induction and after 2 hours. The correlation of serum IL-6, PLA2 and GCS was measure with Separman correlation. A P value less than 0.05 were considered significant.

The result are both serum IL-6 and PLA2 levels were significantly lower in saline with lidocain infusion group ($p<0.005$) with Mann Whitney and Dependent t test statistically analyses. The lower serum IL-6 and PLA2 levels, the higher GCS value. GCS measurement was significantly higher in lidocaine infusion group ($p<0.05$) with dependent t test. The decrese in serum IL-6 and PLA2 was correlated with the dosage of lidocaine. Medium doses of lidocaine achieved the optimum decreases in the serum IL 6 and PLA2 level. Therefore, inhibition of the composition and secretion of IL-6 and PLA2 levels by Lidocaine might be one of the mechanisms involved in decreasing inflammatory reaction and cellular damage in brain cells, and may be responsible for the neuroprotective effect after TBI. Glasgow Coma Scale was improving after infusion with lidocaine 1 mg/ kg/ hr during traumatic brain injury surgery by decreasing IL-6 and PLA2 plasm.

Keywords: Glasgow coma scale; Traumatic brain injury; Lidocaine

Introduction

The Glasgow Coma Scale is an integral part of assessing levels of consciousness. It uses a simple standardized approach. The scale has been revised to make sure it remains an accurate tool. The overall coma score should not be used to convey clinical findings. The scale can be used with children who are over 5 years old part of the care of patients with acute brain injury from head trauma, intracranial haemorrhage and many other causes. The GCS reflects the initial severity of brain dysfunction, while serial assessments demonstrate the evolution of the injury. Each is crucial for decision making. The GCS is also a guide to prognosis and an essential tool for research studies. Four decades after its introduction, the GCS has gained worldwide acceptance. It is now employed in more than 80 countries, has been translated into more than 60 languages and there are more than 18,000 references to its use [1-4].

Head injury or Traumatic Brain Injury (TBI) is defined as a disorder non degenerative (not aging) and non-congenital (not inherited) that occurs in the brain due to the mechanical strength of the outside/trauma, risk of causing disruption temporarily or permanently in the case of noble function (cognitive), physical, or psychosocial functioning, with accompanying reduction in or loss of consciousness [5]. So far have not found a standard pharmacologic therapy for a primary brain injury. Treatments currently available only to limit or reduce the occurrence of secondary brain injury that would enable the various series of the ischemic cascade and release of proinflammatory cytokines and free radicals product that determine the effects of a brain injury later. This is done with the approach of a series of procedures and stages of brain protection [6].

Local anesthetic lidocaine is one of the drugs that are used as adjuvants in neuroanestesia, which also affects brain protection. Clinical effect is determined by the dose of lidocaine. At the recommended clinical dose (1.5 mg/kg), lidocaine can reduce the damage of ischemic [7]. Intravenous lidocaine has been known to have a protective effect because the brain works by inhibiting sodium channels, improve recovery through deceleration and a reduction in anoxic depolarization/ischemic and reduce the influx of sodium [8-10].

Several studies conducted to evaluate the outcome of TBI choices of the level of consciousness by using a scoring system on the Glasgow Coma Scale (GCS). Rate GCS can describe the effect of the power of brain injury in the form of compression power, cut outs, or secondary injuries including a series of inflammation that occurs in a brain injury [11-13]. Glasgow Coma Scale (GCS) score is measured on three parameters that is the eye, verbal and motor. Where the highest score was 15 and the lowest value is 3. Determination of brain injury in this study was based on the GCS score 9-12 (moderate TBI).

In addition to GCS, monitoring ICP can also be used for prognostic outcome of a TBI [14]. In one study it was found that GCS determine

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the prognostic value by measuring the Odds Ratio (OR), where the value of GCS more than 8 when the first patient received indicating outcomes 6.58 times better than the value the GCS 8 downward (OR 6.58; 95% CI 1.87 to 23.14), as observed in a guideline for TBI [15].

In the TBI can be increased proinflammatory cytokines IL-6 and PLA2 [16-20]. Increased IL-6 and PLA2 is signified their cerebral ischemia after TBI [21-30]. Cerebral ischemia caused by TBI can cause cognitive impairment [31-35]. Energy deficit due to supply O_2 lower than the need would cause cognitive impairment [36-43]. The more severe the TBI, the lower the score of GCS. TBI more light, the higher the score GCS [44].

The GCS carries valuable information about the neurological status of patients and constitutes an element of surveillance of their evolution. Yet, by no means should it replace a through neurological examination. The same is true for a number of other tools to assess level of consciousness that have been brought forward the previous years. Nonetheless, none of these seems able to replace the GCS. Moreover, even though it was designed for the evaluation of severe TBI, the GCS is currently used in assessment of coma due to any etiology. However, full knowledge of this scale's strengths and limitations is essential in order to assure its proper use [1-4].

Subject and Methodology

Subjects are the patients with moderate TBI (GCS 9-12) with diagnoses epidural hematoma (EDH) undergoing craniotomy surgery with general anesthesia in the operating room emergency room RSU. Prof. R.D. Kandou Manado. Criteria for inclusion are age 17-55 years, ASA physical status I and II, new TBI traumatic experience within a maximum of 48 hours first, no pro-coagulant therapy. The exclusion criteria are patients who experience worsening of traumatic TBI (loss of consciousness or signs of cerebral herniation) during observations, during the observation period decreased general condition that requires intervention by certain drugs that can interact with the type of treatment used and the family refused to participate. Criteria Drop Out are patients TBI traumatic experience complications during administration of intervention in the form of hemodynamic instability during the observation and intervention, surgery craniotomy with operating time of more than 3 hours.

Using simple random method, samples divided into 2 groups. First group takes sodium chloride infusion after intubation, and second group takes sodium chloride with lidocaine infusion 1 mg/kg/hour with syringe pump. All samples take 2 hours of infusion during surgery. Anesthesia maintenance with isoflurane (no more than 1.5 MAC or 2 volume %) and propofol titration by syringe pump 1-2 mg/kg. We give muscle relaxant every 20 minutes with rocuronium 10 mg IV boluses.

Procedures for work

Before being given the treatment, all patients were given nasal cannula oxygenation with 3 liters/min, 1 hour before the start of induction. The subjects were divided into two study groups. The first group (control group) received infusion of Normal Saline 0.9% without lidocaine and the second group (treatment group) received intravenous saline with lidocaine. Each-each sample was induced with fentanyl 2 mg/kg and propofol 2 mg/kg body weight. Facilities rocuronium intubation with 0.9 mg/kg body weight. Thereafter, the patients were divided two groups, the first group was given a bolus of lidocaine 1 mg/kg after intubated followed by infusion of lidocaine 1 mg/kg/h (using a syringe pump), the second group was given a bolus of lidocaine 1 mg/kg after intubated followed by infusion of Normal saline 0.9% (using a syringe pump) at the same speed.

Infusion of lidocaine or Normal Saline was continued for 2 hours while the surgical procedure is still running. Maintenance of anesthesia with isoflurane and propofol titration (syringe pump) 1-2 mg/kg body weight/hour. The type of surgery to be included in the study is limited to craniotomy operation with the operating time of no more than 3 hours, which is calculated from the start until the skin incision to start sewing leather. Inhalation anesthetics (isoflurane) is given no more than 1.5 MAC (2 volume %).

Muscle relaxants are given every 20 minutes with rocuronium 10 mg intravenous bolus. GCS score measure in pre induction period and 24 hour postoperative.

Data processing and analysis

Analisys processing and distribution of data when normal population using statistical tests dependent-test for independent variables numerical scale that lidocaine levels in plasma, but to the data with the population distribution is not normally distributed, used Mann Whitney test. For categorical variables such as the assessment scale used GCS chi-squared test.

Ethical issues

This research is performed without violating ethics, because:

The samples were given lidocaine does not endanger patients because the drug is often used both in the field of neurosurgery as well as in neuroanesthesia in general as a protective agent penumpul brain and sympathetic response.

Patients are given anesthesia and analgesia drugs are also routinely used in anesthesia action.

The study was conducted after obtaining approval from the ethics committee.

Result

Characters of demographic and clinical factors

Of the 40 samples that meet the criteria, divided into treatment and control groups at random; each 20 people per group. With the division of random groups expected demographic and clinical characteristics of research subjects divided equally in both groups or in other words occur homogenization demographic and clinical characteristics between the two groups before treatment, so that the demographic characteristics did not affect differences in the expected effect on both group (Table 1).

Table 1 shows that there are strong negative linier significance ($p<0.05$) correlation between lidocaine with heart rate, systolic blood pressure, diastolic blood pressure, IL-6, and PLA2. The higher lidocaine concentration, the lower heart rate, systolic blood pressure, diastolic blood pressure, IL-6 and PLA2. There is positive linier correlation between lidocaine concentration with GCS and MMSE.

Table 2 shows correlation analysis between IL-6, PLA2, and GCS. Also between IL-6 with PLA2. There are strong linier significance correlation ($r=0.763$, $p<0.05$) between IL-6 and PLA2. The higher IL-6, the higher PLA2. There are moderate negative linier correlation ($r=-0.508$) between PLA2 with GCS. The higher PLA2, the lower GCS; the higher IL-6, the lower GCS.

Figure 1 shows that there is negative correlation between lidocaine concentration with IL-6; the higher lidocaine concentration, the lower IL-6.

Variables	Lidokain concentration	
	Correlation Coeficient (r)	Significance (p)
HR	r=-0.701	p=0.000
SBP	r=-0.823	p=0.000
DBP	r=-0.788	p=0.000
IL-6	r=-0.677	p=0.000
PLA2	r=-0.734	p=0.000
GCS	r=0.356	p=0.012

Table 1: Correlation between lidocaine with IL-6, PLA2, HR, SBP, and DBP 60 minutes after extubation.

Correlation	Spearman Correlation	
	Correlation Coeficient (r)	Significance (p)
IL-6-CS	r=-0.298	p=0.031
PLA2-CS	r=-0.508	p=0.000
IL-6-PLA2	r=0.763	p=0.000

Table 2: The correlation of IL-6, PLA2 with GCS 60 minutes after extubation.

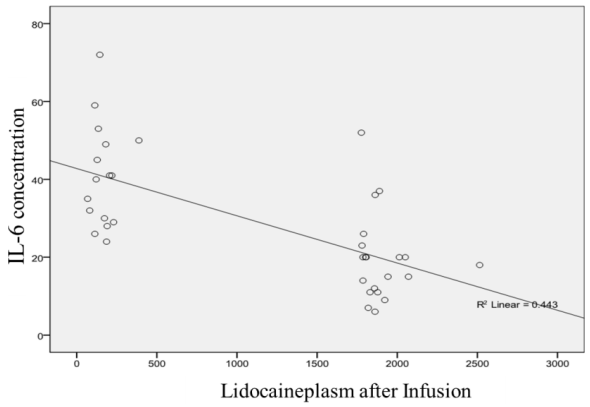

Figure 1: Correlation between lidocaine with IL-6.

Figure 2 shows there is negative correlation between lidocaineplasmconcentration with PLA2 plasm; the higher lidocaineplasm concentration, the lower PLA2 plasm.

Figure 3 shows there is positive correlation between IL-6 plasm with PLA2 plasm concentration; the higher IL-6 plasm concentration, the higher PLA2 plasm concentration.

Table 3 shows that there is no side effect which are bradycardia and hypotension between control and lidocaine group.

Table 3 shows that heart rate in normal limit during surgery until 60 minute after extubation. Therefore, as dyastolic blood pressure. The systolic blood pressure approximately in normal limit, just once less than 90 mmHg but the decrease is no more than 20% from baseline. So there is relative no hypotension effect in this research.

Levels of serum lidocaine

The treatment group was the group that received lidocaine infusion in a liquid, NaCl 0.9% and the control group only received 0.9% NaCl infusion without lidocaine. Two hours post-infusion, examined serum lidocaine levels in both groups. Comparison of serum lidocaine levels between the two groups were tested with independent sample test. The results can be seen in Table 4.

Table 4 shows that serum lidocaine levels 2 hours post-infusion was higher in the treatment group (1901.7 ± 169.6 ng/ml) than the control group (171.9 ± 75.8 ng/ml). Results of independent t test showed very significant differences (p=0.000).

Changes GCS

GCS changes in the control and treatment groups were tested by paired t test. GCS comparison between the two groups before surgery, as well as GCS after extubation and the difference of change in GCS before surgery until after extubation between the two groups were tested by the Mann Whitney test. The results can be seen in Table 5.

Table 5 shows that in the control group there was an increase of 0.1 of GCS (10.1 ± 0.8) to (10.2 ± 0.8) but paired t test results showed no significant changes (p>0.05), while the treatment group increased by 1.3 of (10.1 ± 0.6) to (11.4 ± 1.3) and significant (p<0.05). Mann Whitney test results showed that the GCS between the two groups prior to surgery showed no significant difference (p=0.083), but after two hours of infusion was significantly different (p<0.001), as well as differences in changes in GCS, significant (p<0.05). Thus, changes in GCS were significantly different between the two groups. GCS increased in the treatment group, whereas the control group did not change.

Figure 4 shows that the same GCS (coincident) before induction of anesthesia, and after a 2 hour infusion of GCS levels increased in the treatment group, while the control group remained or increased slightly.

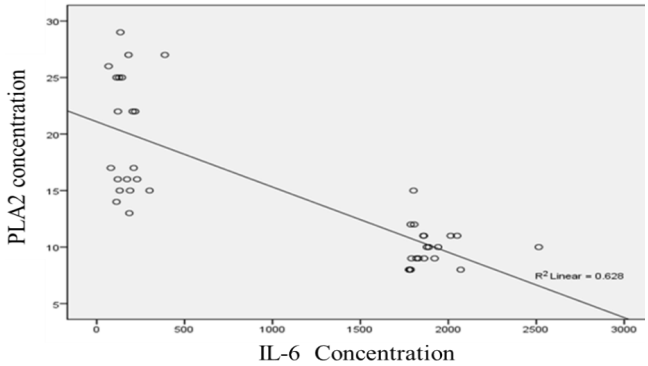

Figure 2: Correlation between lidokain with PLA2.

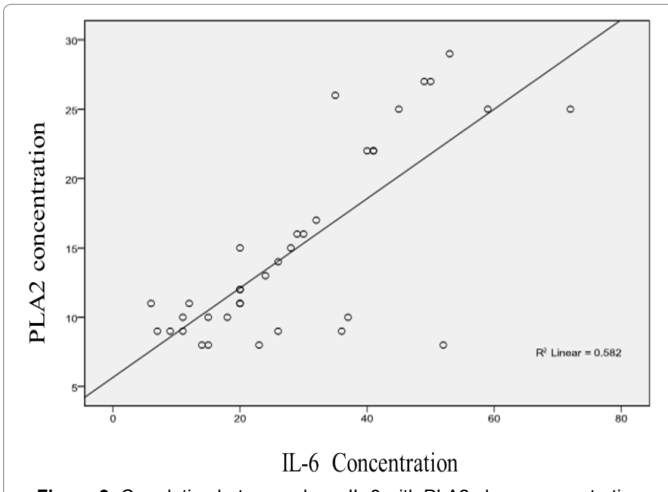

Figure 3: Correlation between plasm IL-6 with PLA2 plasm concentration.

Time Observation	Minimal-maximal Heart Rate (x/minute)		Minimal-Maxinal SBP (mmHg)		Minimal-Maximal DBP (mmHg)	
	Control	Lidocaine	Control	Lidocaine	Control	Lidocaine
Before surgery	78-105	78-102	115-144	112-147	56-100	59-104
Awal infus	78-100	79-102	112-140	115-145	54-98	61-100
30'infus	78-103	71-100	131-154	92-136	67-100	48-78
60' infus	78-102	70-92	128-152	92-136	78-102	50-72
90' infus	78-101	70-98	128-158	85-126	78-102	52-79
120' infus	78-100	68-90	135-150	92-136	44-103	48-78
Saat ekst	78-100	66-93	132-149	92-136	73-100	48-78
30' post ekst	78-102	65-90	132-150	92-136	72-100	48-78
60' post ekst	78-100	63-91	132-150	92-136	80-100	48-78

Table 3: Distribution of rangeheart rate, SBP, DBP between control group and lidocaine group.

Level Lidocaine Serum (ng/ml)	Group		Independent Sample t test
	Treatment (n=20)	Kontrol (n=20)	
Mean ± SD	1901.7 ± 169.6	171.9 ± 75.8	p=0.000
Minimal/Maximal	1776-2514	68-388	

Table 4: Comparison of serum lidocaine levels between 0.9% NaCl infusion group (control) and Group NaCl+0.9% Lidocaine (treatment).

Group	GCS			Paired t- test
	Preinduction	2 hours post-operative	Perubahan	
Control (n=20)	(10.1 ± 0.8)^{p}	(10.2 ± 0.8)^{p}	Increase (0.1)^{a}	p=0.083
Treatment (n=20)	(10.1 ± 0.6)^{p}	(11.4 ± 1.3)^{q}	Increase (1.3)^{b}	p=0.001
Superscript are not different in the same column shows the results of Mann Whitney test was not significant (p> 0.05), and if different, Mann Whitney test results showed significant (p<0.05).				

Table 5: Comparison of GCS changes between the two groups.

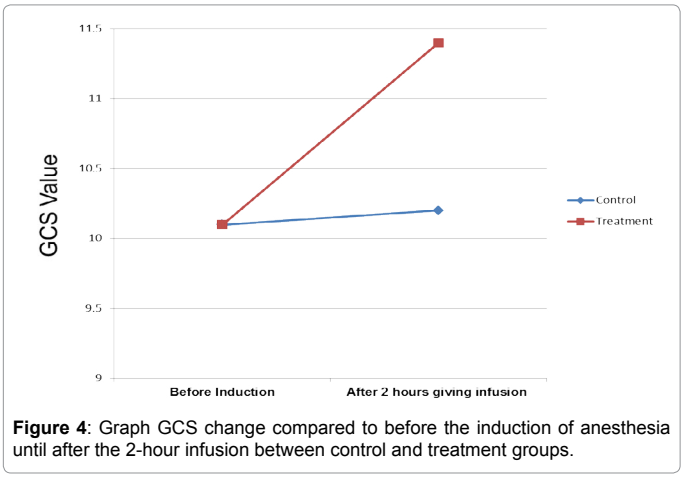

Figure 4: Graph GCS change compared to before the induction of anesthesia until after the 2-hour infusion between control and treatment groups.

Pulse rate changes and average artery pressure

To assess the side effects that may occur, observed on cardiovascular responses (heart rate and mean arterial pressure). Analysis results can be seen in Table 6.

Correlation between GCS improvement with IL-6 and PLA2 decreasing

Table 7 show there are strong correlation (p=0,000) between IL-6 plasm decreasing with GCS increasing with coefficient correlation r=-0.530. The lower IL-6 plasm concentration, the higher GCS. The lower PLA2 plasm concentration, the higher GCS (r=-0.351 and p=0.031) (p<0.05).

Discussion

General characteristics

General data of patients including age, height, weight, BMI, initial pressure systolic, pressure early diastolic blood pressure on average, respiratory rate, heart rate, SpO_2, core temperature, blood sugar levels, and GCS showed no difference significant between the two groups, which means both relatively homogenous groups.

The average patient age was 30 years; mean traumatic effect will be the same as its value is the same GCS. In contrast to when one group over the age of 60 years. Although it weighs the same primary injury but because of different brain tissues, the therapeutic effect will be different.

Lidocaine effect on intracranial component

Perhaps the most widely used of this intravenous lidocaine, administered before rapid sequence intubation. Although the exact mechanism by which lidocaine works is unclear, it is thought to be related to its ability to blunt the increase in pulse rate and blood pressure associated with laryngoscopy and tracheal intubation, which subsequently cause an increase in intracranial pressure. Lidocaine has also been shown to reduce cerebral blood flow and cerebral vascular resistance. Finally, lidocaine as a sodium channel blocker, may decrease cerebral metabolism and stabilize neuronal membranes, which may lead to a decrease in secondary brain injury in traumatic brain injury. Evidence suggests that using intravenous lidocaine 2 to 3 minutes before rapid sequence intubation is safe and may help attenuate the increase in intracranial pressure associated with intubation in the traumatically brain-injured patient, potentially improving long term outcome. An intracranial pressure greater than 20 mmHg and occuring within the first 72 hours of patient care is associated with an increased morbidity and mortality [45-47].

Each channel for ion transport pathways in the cell membrane of neurons can only be traversed by a certain ion. In the resting, state of cells no stimulation to the cell membrane potential in a state of rest. In the circumstances that led to the opening of voltage-gated channels (usually very fast) changes in the membrane potential and this is what

causes the onset of electrical signals called action membrane potential. There are two types of channels in addition to the canals mentioned above, namely that can open and close. Channels that provide an answer to their chemical signals responsible for the formation of an electrical signal, called a ligand-gated channels. This channel is opened when there is stimulation of extracellular molecules. Some are some channels that respond to molecules in the cell, for example of a molecule "second messengers" such as Ca^{2+} or other molecules such as cyclic GMP, and G protein subunits that are activated by cell surface receptors. The action potential is a cycle of depolarization, hyperpolarization and return to a resting state. Specifically, the electrical changes caused by the Na+ channels and voltage-gated K+ that open and close in response to changes in membrane potential [45-47].

In the process, the Krebs cycle takes a number of enzymes found in the mitochondrial matrix that will give the final result of CO_2. During this process the ion H will be released that will be captured by coenzyme nicotinamide adenine dinucleotide (NAD). Electrons are derived from the hydrogen moving through some respiratory enzymes (flavoproteins and cytochromes) that eventually combine with oxygen to form protons from water molecules (H_2O). Of the latter is to be obtained by the energy stored in the bonds of ATP are derived from ADP. Being necessary enzymes bound to the inner mitochondrial membrane wall composed of sections called inner membrane sub-units (unit globular). Each subunit consists of a round building with a diameter of 9 nm which is connected with the handle along the 3.5 to 4.5 nm [48,49].

A chemical signal is delivered from outside the cell through a receptor on the cell surface, and then sent to the cell nucleus. This process is very complex because it involves a variety of chemical and physical properties of a signal protein, which is not another because of changes in the electrical charge of protein molecules [46]. In a tissue, damage will occur inflammatory process characterized by increased proinflammatory cytokines IL-1B, IL-6, TNFα. Lidocaine effects that reduce cell death as a result of barriers to the Na+ channels will cause reduced the inflammation process.

Magnesium-lidocaine combination administered according to the above regimen is safe and well tolerated after severe head injury. Patient in the magnesium-lidocaine group showed a more favorable outcome and lower mortality rate when compared to the control group. Lidocaine infusion (IV, 3 mg/kg) triggers a 5-minute 10% reduction of cerebral O_2 consumption within 2 to 3 minutes [49]. Lidocaine reduces in a dose dependent manner brain metabolism, suppresses synaptic transmission and stabilizes membranes by blocking pathologic K+ efflux, Na+ channels, and demand for Na+-K+ transport [50].

Classification of traumatic brain injury

Traumatic brain injury division based on a few things in between based on the mechanism of occurrence, based on the severity (severity), and based on its morphology. Based on the mechanism of injury, the TBI is divided into: blunt injury and injury penetrans. Based on the severity (severity) TBI divided based on the criteria of the Glasgow Coma Scale (GCS), namely: mild TBI (GCS 14-15); moderate TBI (GCS 9-13); and severe TBI (GCS 3-8) in patients examined [49].

Based on the morphology TBI is divided into: 1. Focal injuries, ie injuries identified suitable areas affected; divided into: Cerebral contusion, extradural hemorrhage, scalp lacerations, fractures calvaria, subarachnoid hemorrhage, subdural Hemorrhage; 2. Diffuse injuries, ie injuries affecting the whole brain tissue, consisting of: Concussion and Brain Injury Diffuse axonal (Table 8) [47,50].

Glasgow coma scale

Glasgow Coma Scale was first introduced by Teasdal and Jennett as the standard method for evaluating health care givers and describe the degree of alteration of consciousness or coma in patients who suffered a head injury. Glasgow Coma Scale to test three different components, namely: open your eyes, verbal response, and motor response. In each category there is a range of ratings to determine the specific components. Each observation is connected with the scores recorded by health care providers as a standard classification of the patient's

Time of Observation	Minimal-Maximal Heart Rate (x/minutes)		Minimal-Maximal MAP (mmHg)	
	Control	Treatment	Control	Treatment
Before surgery	78-105	78-102	96-127	97-130
60' infusion	78-102	70-92	114-130	78-113
120' infusion	78-100	68-90	109-132	77-114
MAP: Mean Arterial/Blood Pressure				

Table 6: The range of distribution of heart rate and mean arterial pressure in the control and treatment.

The correlation between variables	Spearman Correlation	
	Coefficient correlation (r)	Significance (p)
IL-6 plasm decreasing with GCS increasing	r=-0.530	p=0.000
PLA2 plasm decreasing with GCS increasing	r=-0.351	p=0.013
IL-6 plasm decreasing with PLA2 plasm decreasing	r=0.453	p=0.002

Table 7: The correlation between IL-6 and PLA2 plasm decreasing with GCS increasing.

	Mild TBI	Moderate TBI	Severe TBI
Beginning GCS value	14-15	9-13	3-8
Presentation	80	10	10
Abnormality of CT Scan (%)	5-15	30-50	60-90
Demand of Neurologic intervention (out of ICP Monitoring)	1-3	5-30	30-50
Mortality (%)	<1	10-15	30-50
Good Functional Outcome/GEO (%)	>90	20-90	<20

Table 8: There is also a division of TBI based on the outcome produced.

level of consciousness. Components eyes open there are 4 scale ranging from no response (score 1) through the eyes open spontaneously (score 4). Motor component No 6 scale, ranging from no response to the movement (score 1) to comply with orders observer movement (score 6). No verbal components 5 components, ranging from no verbal response at all (score 1) to be able to communicate the appropriate / right orientation (score 5). From each category are then summed to be rated as GCS [49].

Traumatic brain injury is a heterogeneous condition in terms of aetiology, severity, and outcome. The most useful classification of severity is based on the level of consciousness as assessed by the Glasgow Coma Scale after resuscitation. The GCS comprises the sum score of the values from three components: eye, motor, and verbal scales. However, factors such as hypoxia, hypotension, and alcohol intoxication can affect GCS, leading to diagnostic confusion. Therefore, the patient should be resuscitated and reversible causes corrected before GCS assessment. The ability to assess eye responses is influenced by sedative agents or tracheal intubation, leading some to suggest the use of the motor score alone.

Score GCS can describe the effect of the power of brain injury in the form of compression power, cutouts, or secondary injuries including a series of inflammation that occurs in a brain injury [11-13]. In addition to GCS, monitoring ICP can also be used for prognostic outcome of TBI [10]. In one study it was found that GCS determine the prognostic value by measuring the Odds Ratio (OR), where the value of GCS more than 8 when the first patient received indicating outcomes 6.58 times better than the value of GCS 8 downward (OR 6.58; 95% CI 1.87 to 23.14), as observed in a guideline for TBI [15].

Neurological injury progresses over hours and days, resulting in a secondary injury. Inflammatory and neurotoxic processes result in vasogenic fluid accumulation within the brain, contributing to raise intracranial pressure, hypoperfusion, and cerebral ischaemia. Much of this secondary injury may be amenable to intervention, as almost one-third of patients who die after a TBI will talk of obey commands before their death. Secondary injury also occurs as a result of further physiological insults. Hypoxia, hypotension, hypercapnia or hypocapnia, hyperglycemia or hypoglycaemia has all been shown to increase the risk of secondary brain injury. Overall those processes involve and influence the value of GCS [45-49].

Conclusion

Glasgow Coma Scale was improving after infusion with lidocaine 1 mg/kg/hr during traumatic brain injury surgery by decreasing IL-6 and PLA2 plasm.

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