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Research Article

Correlation between Glasgow coma scale and Jugular venous oxygen saturation in severe traumatic brain injury

Mohammed Samy Sharf [a,*], **Mohammed A. El-Gebali** [b]

[a] *Anesthesia and Intensive Care Department, Al-Azhar Faculty of Medicine, Azhar University, Egypt*
[b] *Neurolosurgical Department, Al-Azhar Faculty of Medicine, Azhar University, Egypt*

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KEYWORDS

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Abstract *Background:* Glasgow Coma Scale (GCS) remains a key measure in neurological assessment after head injury and in most studies classification of the severity of the trauma is still based on the admission GCS.

The aim of the work: The aim of the work was to correlate between Jugular venous oxygen saturation (Sjvo2) with GCS in cases with severe traumatic brain injury.

Patients and methods: A 44 patients met the inclusion criteria, were included in the present study. They were selected from the neurosurgical and intensive care units at Al-Azhar University hospital during the period from June 2010 till June 2012. All therapeutic interventions were performed in accordance with Guidelines for the Management of Severe Traumatic Brain Injury. The following variables were collected: patients' demographics, Sjvo2, ICP, MAP, CPP and GCS. All pressures were monitored invasively and with identical transducers connected to monitors, and expressed numerically in mmHg. Measurements were always performed at 8.00 a.m. At the same time, patients were neurologically examined and these data were expressed as GCS score.

Results: There was statistically significant increase of GCS, MAP, CPP, Sjvo2 and Extended Glasgow Outcome Scale (GOSE) and decrease of ICP in survived in comparison to non-survived cases. In survived cases, there was positive significant correlation between Sjvo2 and GCS, MAP, CPP and GOSE, while there was significant negative correlation with ICT. On the other hand, in non-survived cases, there was only positive moderate, significant correlation between Sjvo2 and GCS. Running simple linear regression analysis, only GCS and Sjvo2 can predict mortality in studied cases.

* Corresponding author. Tel.: +20 101846798; fax: +20 572404035.
E-mail address: ms_sharf@hotmail.comm (M.S. Sharf).

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Conclusion: Results of the present study proved that, Sjvo2 is proportionally correlated with GCS and both can predict the prognosis of severe traumatic injury.

1. Introduction

Severe traumatic brain injury (TBI) is responsible for significant morbidity and mortality each year. TBI is the cause of about 40% of trauma-related deaths. The GCS remains a key measure in neurological assessment after head injury and in most studies classification of the severity of the trauma is still based on the admission GCS. A score less than or equal to 8 is used to differentiate between severe and moderate to mild head injury. In addition the patients' management is frequently dependent on this initial classification [1].

In addition to the efforts done to avoid brain injury, much of the management of TB has centered on the prevention of consequent insults related to edema, intra-cranial hypertension, cerebral hypoxia, and ischemia. The mainline of treatment is directed essentially to control the intra-cranial pressure (ICP) and cerebral perfusion pressure (CPP) through osmotic agents, vasopressors, ventilatory manipulation, drainage of cerebro-spinal fluid (CSF), craniectomy, and barbiturate-induced coma; as mortality and poor functional outcome are closely linked to high (ICP). However, poor outcomes have been documented even in the setting of normal ICP and CPP values [2]. Thus, it can be said that, the ICP and CPP are not responsible alone for brain tissue insults.

The main management strategy to limit secondary cerebral insults in TBI is cerebral oxygenation. Cerebral oxygenation can be evaluated through evaluation of the cerebral blood flow (CBF), arterial oxygen content and cerebral metabolic rate of oxygen consumption. The latter two are not practically in use now; so CPP is the mostly used as an alternative measurement for CBF measurements [3]. It is important to guide the therapy and evaluate the outcome according to the ICP and CPP values for the early management of TBI patients [4]. Preservation of autoregulation is essential to protect against secondary brain insults [5,6].

In an attempt to guide management of patients with TBI and prevent secondary brain insults, several monitoring strategies have been developed. One of these methods is brain tissue oxygen ($pBtO_2$) monitoring. A strong correlation between low $pBtO_2$ values and poor patient is demonstrated in number of observational studies [7,8].

The severity of the trauma is based mainly on the admission GCS, so the GCS remains a key measure in neurological assessment after head injury. Monitoring jugular venous oxygen saturation (Sjvo2) is a technique, which can be used to estimate the balance between global cerebral oxygen delivery and utilization. A catheter is inserted into the dominant internal jugular vein (IJV) and advanced to the jugular bulb. The global cerebral oxygenation is mostly monitored through Sjvo2 monitoring, when the dominant jugular bulb is cannulated and the right side is often chosen because it is usually dominant [9].

The catheter positioning is checked on a lateral cervical spine radiograph before measurement of Sjvo2 that can be made either continuously using a fibre-optic catheter or directly by aspirating blood samples and using a co-oximeter. Reduction in jugular Sjvo2 below physiological levels ($< 55\%$) indicates that cerebral oxygen delivery is inadequate to meet demand. In the context of TBI, this is most often related to the reduced CBF secondary to decreased CPP or hyperventilation-associated vasoconstriction. Conversely, raised jugular Sjvo2 indicates luxury perfusion caused by either raised CBF or reduced oxygen demand secondary to mitochondrial dysfunction or cell death [10]. Reduction in jugular Svo2 to $< 50\%$ after TBI is associated with poor outcome [11], and jugular Sjvo2 is responsive to changes in CPP [12]. There is some evidence to suggest that the use of jugular Sjvo2 monitoring may improve outcome after TBI [10], however jugular venous oxygen saturation is limited by its lack of sensitivity to regional changes [6].

1.1. Aim of the work

The aim of this study was correlating between Sjvo2 with GCS in cases with severe traumatic brain injury and outcome as regard morbidity and mortality.

2. Materials and methods

The present study was done as – an observational study – in the neurosurgical and intensive care units at Al-Azhar University hospital during the period from June 2010 till June 2012. Fifty-four patients met the inclusion criteria and thus, were included in the present study.

2.1. Inclusion criteria

- Patients presented with closed traumatic brain injury with GCS $\leqslant 8$.
- Patients required a neurocritical care with continuous monitoring of ICP and invasive arterial blood pressure (ABP), lasting usually longer than 24 h.

2.2. Exclusion criteria

- Patients presented with Glasgow coma scale 3 after resuscitation.
- Dilated, reactive pupils.
- Patients who were recognized as dead in the field.
- When data for more than 24 h were missing.

Standard interventions were performed, e.g., radial artery cannulation, central venous access, and internal jugular vein bulb cannulation. All therapeutic interventions were performed in accordance with Guidelines for the Management of Severe Traumatic Brain Injury [13]. ICP monitoring was

performed via ventriculostomy. The procedure was carried out at bedside or in the operating theater as part of extensive neurosurgical procedures (evacuation of subdural or epidural hematomas, decompressive craniotomy).

Sjvo2 determination: A catheter is inserted into the dominant internal jugular vein (IJV) and advanced to the jugular bulb on the right side as it is the dominant jugular bulb. Once catheter positioning has been checked on a lateral cervical spine radiograph, measurement of jugular Svo2 was made by aspirating blood samples and using a co-oximeter [6].

Arterial hypotension was promptly treated with fluids and continuous infusion of inotropes. Episodes of increased ICP controlled with medical treatment whenever possible, including sedation, mild hyperventilation, hypothermia, and thiopentone infusion. Surgical treatment added if persistent or uncontrolled intracranial hypertension developed. It should mention that the assessment of the patient's conscious level by GCS was done before administration of sedating agents.

The following variables collected: patients' demographics, Sjvo2, ICP, MAP, CPP and GCS. All pressures were monitored invasively and with identical transducers connected to monitors, and expressed numerically in mmHg. Measurements were always performed at 8.00 a.m. At the same time, patients were neurologically examined and these data were expressed as GCS score. All data were recorded in the patient's sheet and entered in database.

Extended Glasgow Outcome Scale (GOSE), which is a global assessment of independent living and social reintegration that is widely used as an outcome measure in brain injury research, to analyze long-term functional outcome (3 months postoperatively). The assessment was carried out using the structured interview for the GOSE, with questions covering the following areas: (1) consciousness; (2) independence inside and outside the home; (3) resumption of normal social roles (work, social and leisure activities, personal relationships); and (4) residual symptoms interfering with daily life. The GOSE does not require a detailed psychological or neurologic examination and can be administered by professionals from different backgrounds. The GOS consists of five categories: dead (score 1), vegetative state (score 2), severe disability (conscious, but disabled) (scores 3, 4), moderate disability (disabled, but independent) (scores 5, 6), and good recovery (scores 7, 8). For the GOSE, the latter three categories are divided into upper and lower bands (GOSE was applied 3 months postoperatively).

Data was analyzed using SPSS (Statistical Package for Social Sciences), version 10. Qualitative data presented as number and percent. Comparison between groups was done by Chi-Square test. Student *t*-test was used to compare between two variables. *F*-test (One-Way ANOVA) was used to compare between more than two variables. Spearman's correlation coefficient (*r*) was used to analyze the association between the different variables. Values of $p < 0.05$ were considered to be statistically significant.

3. Results

The present study included 54 cases, 39 (72.5%) were males and 15 (27.8%) were females; age ranged from 21 to 61 years with a mean of 48.59 ± 8.79; mortality rate was 31.5% (Table 1). The most common cause of reported injuries was motor car accident (represented in 63.0% of case), then fall from height in 33.3% and gunshot injury was only reported in 3.7% of cases; the most common associated injury was subarachinoid hemorrhage (presented in 48.1%), then skull fracture in 22.2%, cerebral edema in 14.8%, intracerebral hemorrhage in 13.0%) and brain contusion in 1.9%; 45 cases (83.3% needed surgical interference and epileptic fits developed in 9.3% of cases during their hospital stay (Table 2). The results of the present work showed statistically significant increase of GCS, MAP, CPP, Sjvo2 and GOSE in survived in comparison to nonsurvived cases. On the other hand, there was statistically significant decrease of ICP in survived when compared to non-survived cases (Table 3). In survived cases, there was positive significant correlation between Sjvo2 and GCS, MAP, CPP and GOSE, while there was significant negative correlation with ICT. On the other hand, in non-survived cases, there was only positive moderate, significant correlation between Sjvo2 and GCS (Table 4). Running simple linear regression analysis, only GCS and Sjvo2 can predict mortality in studied cases (Table 5).

Table 1 Demographic characteristics of studied cases.

Variable	Statistics
Gender (*m*/*f*; *n*, %)	39/15; 72.2%/27.8%
Age (mean ± SD; minimum–maximum) per year	48.59 ± 8.79; 21–61
Mortality rate (*n*, %)	17 (31.5%)

There were 54 cases, 39 (72.5%) were males and 15 (27.8%) were females; age ranged from 21 to 61 years with a mean of 48.59 ± 8.79; mortality rate was 31.5%.

Table 2 Descriptive statistics of TBI.

Variable	Statistics
Cause of TBI (n, %)	
Fall from height	18 (33.3%)
Motor car accident	34 (63.0%)
Gunshot injury	2 (3.7%)
Associated injury (n, %)	
Cerebral edema	8 (14.8%)
Subarachinoid hemorrhage	26 (48.1%)
Skull fracture	12 (22.2%)
Intracerebral hemorrhage	7 (13.0%)
Brain contusion	1 (1.9%)
Cases need surgical interference (*n*, %)	45 (83.3%)
Epileptic fits at hospital (*n*, %)	5 (9.3%)

The most common cause of reported injuries Motor car accident was the most common cause of reported injuries (represented in 63.0% of case), then fall from height in 33.3% and gunshot injury was only reported in 3.7% of cases; the most common associated injury was subarachinoid hemorrhage (presented in 48.1%), then skull fracture in 22.2%, cerebral edema in 14.8%, intracerebral hemorrhage in 13.0% and brain contusion in 1.9%; 45 cases 83.3% needed surgical interference and epileptic fits developed in 9.3% of cases during their hospital stay.

4. Discussion

Traumatic brain injury (TBI) and stroke are the main pathological scenarios that result in neuronal loss with subsequent

Table 3 Comparison between survived and non-survived cases regarding GCS, MAP, ICP, CPP and GOSE.

Variable	Survived	Non-survived	(*t*) Test	*P* value
GCS	6.0 ± 1.11	3.94 ± 0.89	6.71	<0.001*
MAP (mmHg)	92.16 ± 14.97	59.12 ± 3.17	8.95	<0.001*
ICP (mmHg)	18.32 ± 7.51	31.23 ± 6.26	6.15	<0.001*
CPP (mmHg)	73.83 ± 21.08	27.88 ± 7.63	8.69	<0.001*
Sjvo2 (%)	65.10 ± 4.90	50.52 ± 1.84	11.83	<0.001*
GOSE	5.41 ± 1.53	1.00 ± 0.00	10.41	<0.001*

There was a statistically significant increase of GCS, MAP, CPP, Sjvo2 and GOSE in survived in comparison to non-survived cases. On the other hand, there was statistically significant decrease of ICP in survived when compared to non-survived cases.
** Highly significant $P < 0.0$.
* Significant $P < 0.05$.

Table 4 Correlation between Sjvo2 and different parameters.

		Age	GCS	MAP	ICP	CPP	GOSE
Sjvo2 in whole cases	*r*	0.08	0.85	0.91	−0.79	0.90	0.86
	p	0.52(NS)	<0.001*	<0.001*	<0.001*	<0.001*	<0.001*
Sjvo2 in survived cases	*r*	−0.15	0.74	0.78	−0.69	0.82	0.52
	p	0.36(NS)	<0.001*	<0.001*	<0.001*	<0.001*	0.001*
Sjvo2 in non-survived cases	*r*	−0.34	0.66	−0.12	0.35	−0.34	
	p	0.17(NS)	0.004*	0.62(NS)	0.15(NS)	0.17(NS)	–

In survived cases, there was positive significant correlation between Sjvo2 and GCS, MAP, CPP and GOSE, while there was significant negative correlation with ICT. On the other hand, in non-survived cases, there was only positive moderate, significant correlation between Sjvo2 and GCS.
** Highly significant $P < 0.0$.
* Significant $P < 0.05$.

Table 5 Regression analysis of studied variable to detect predictors of mortality.

	Beta	*P* value	95% Confidence interval for B	
			Lower bound	Upper bound
Age per year	0.003	0.964	−0.007	0.008
GCS	−0.396	0.008	−0.224	−0.035
ICP (mmHg)	0.120	0.269	−0.005	0.017
MAP (mmHg)	−0.241	0.207	−0.015	0.003
Sjvo2 (%)	0.800	<0.0001*	0.028	0.064

Running simple linear regression analysis, only GCS and Sjvo2 can predict mortality in studied cases.
** Highly significant $P < 0.0$.
* Significant $P < 0.05$.

disability, and the common mechanism is inadequate cerebral blood flow (CBF) for the neurons [14].

The ideal CBF monitoring device should have the following characteristics: a steep learning curve with minimal training, ability to provide point of care measurement, allow for continuous assessment with minimum interference to patient care, noninvasive or minimally invasive with no adverse effects, a high temporal and spatial resolution, consistent and reproducible, and cost-effective. Currently, no CBF monitor fulfills these criteria [15].

Cannulation of the jugular bulb allows assessment of the global oxygenation status of the brain and adequacy of CBF [16]. Normal jugular venous oxygen saturation (Sjvo2) range is between 55% and 75%. The ischemic threshold has been reported to be a Sjvo2 < 50% for at least 10 min [11]. Low Sjvo2 indicates either an increase in oxygen demand as in fever and seizures or a reduction in oxygen delivery due to vasospasm or hypotension, or inadequate CPP. A high Sjvo2 indicates the opposite and may be consistent with benign hyperemia [15].

The present study was designed to correlate between Sjvo2 with GCS in cases with severe traumatic brain injury, and, in the majority of the cases, the injury was caused by traffic accidents and these results are in agreement with that reported by [17]. Post-traumatic epilepsy is one of the most important complications after TBI [18], and our finding that 16.7% of patients suffered from posttraumatic epilepsy is consistent with the findings of [19].

The mortality rate in the present study was 31.5% and this is in agreement with previous report in the literature [14] where they reported that, TBI affects 2 million individuals per year in the USA alone, and in the first 72 h the mortality rate exceeds

30%. In addition, Martini et al. [20] reported that, the cumulative hospital mortality rate was 29% and it is slightly lower than that reported in the present work. On the other hand [17], reported that, the overall in-hospital mortality was 58%, and [21] reported 52% mortality rate. These results are much higher than that of the present study and this can be attributed to the more severe TBI in their study.

In the present study, GCS showed a good predictive value of mortality in studied populations. In contradiction to these results, Balestreri et al. [1], in a report of 10 years' data on head injured patients shows a loss in correlation between admission GCS and EGOS from 1997 onwards, suggesting a reduction in power of the GCS score as predictor of outcome after brain trauma. In the same group of patients, age remains an important factor in prognostic modeling after head injury. The possible explanation for converse with results of present work may be attributed to the facts that they included data of all head injuries regardless its severity.

In the present work, there was positive correlation between Sjvo2 and GCS especially in non-survived cases with a good predictive power. These results are in agreement with those reported that, a significant association exists between jugular venous desaturation and poor neurological outcome. However, it has the disadvantage that a relatively large volume of tissue must be affected, approximately 13%, before Sjvo2 levels decrease below 50% [22,23]**.** Furthermore, Tisdall and Smith [6] reported that, reduction in jugular Svo2 below physiological levels ($<55\%$) indicates that cerebral oxygen delivery is inadequate to meet demand. In the context of TBI, this is most often related to reduce CBF secondary to decreased CPP or hyperventilation-associated vasoconstriction. Confirming these results, Robertson et al. [11] reported that, reduction in jugular Svo2 to $<50\%$ after TBI is associated with poor outcome, and jugular Svo2 is responsive to changes in CPP.

Finally, there is some evidence to suggest that the use of jugular Svo2 monitoring may improve outcome after TBI [10]**.** Conversely, McLeod et al. [24] reported that, the baseline value for Sjvo2 was high, and this may have been related to hyperemia. Increased Sjvo2 values are usually considered to represent excess (''luxury'') perfusion or decreased cerebral oxygen consumption [25]**.** However, Cormio et al. [26] reported that, increases of Sjvo2 occur in almost 20% of patients after head injury, and it has been suggested that this is a heterogeneous condition that cannot automatically be associated with hyperemia.

Also, results of non-survived cases showed no significant correlation between GCS, Sjvo2 and intracranial tension, and this supports results of Bulger et al. [27] who reported that, the use of ICP monitoring is not universal and there exists no Class I evidence supporting its efficacy.

Furthermore, results of the present study showed no association between age and mortality outcome in studied populations. In addition, age had no predictive power. These results are in contradiction to those reported by Livingston et al. [28] who reported that, age is an exceedingly important parameter affecting recovery from TBI. The mean age of TBI victims varies from 32 to 49 years. Older patients, after isolated TBI, have poorer functional status at discharge and make less improvement at 1 year compared to all other patients. These worse outcomes occur despite less severe TBI in elderly patients as measured by a higher Glasgow Coma Scale (GCS) score upon admission.

In short, results of the present study proved that, Sjvo2 is proportionally correlated with GCS and both can predict the prognosis of severe traumatic injury.

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