Original Article

Impact of anesthetic induction with etomidate, thiopentone, and propofol on regional cerebral oxygenation: An observational study in patients with traumatic brain injury

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Abstract

Background and Aims: Anesthetic induction plays a pivotal role in determining the operative course and the outcome in patients with acute traumatic brain injury (TBI). The present study compared the effect of anesthetic induction with etomidate, thiopentone, and propofol primarily on systemic hemodynamics and regional cerebral oxygenation ($rScO_2$) and secondarily on the serum cortisol levels in TBI patients.

Material and Methods: In this prospective observational study, eligible patients were recruited and divided into three groups as per the induction agent received. Data collected were hemodynamic parameters and $rScO_2$ levels at baseline, following 3 min of preoxygenation, and over 10 min of induction. Serum cortisol levels were measured before and after 24 h of induction. The statistical analysis was done using R software.

Results: A total of 115 patients were included: 32, 33, and 50 in thiopentone, propofol, and etomidate groups, respectively. A significant increase ($P < 0.001$) in bilateral $rScO_2$ was noted in all three groups following anesthetic induction. Intergroup comparison of the propofol and the etomidate groups revealed significantly lesser increase in contralateral $rScO_2$ ($P = 0.019$) and a greater fall in mean arterial pressure ($P = 0.003$) on using propofol as an induction agent. Trend changes in bilateral $rScO_2$ and hemodynamic parameters were comparable between thiopentone and etomidate groups. An insignificant fall in serum cortisol was observed in etomidate ($P = 0.332$) and thiopentone ($P = 0.364$) groups, but a significant increase was observed in the propofol group ($P = 0.004$). The Glasgow coma scale (GCS) score at discharge improved significantly in all the groups ($P < 0.001$).

Conclusions: In TBI patients, anesthetic induction with etomidate resulted in least hemodynamic changes compared to induction with thiopentone and propofol. The $rScO_2$ increased in all three groups after induction, with the maximal increase observed with etomidate compared to propofol and thiopentone. Insignificant fall in serum cortisol was observed with etomidate and thiopentone, but not with propofol. Outcome at discharge, assessed with GCS, was comparable in all the groups.

Keywords: Blood pressure, cerebral oxygen saturation, cortisol, etomidate, propofol, thiopentone, traumatic brain injury

Key Messages: Anesthetic induction with etomidate produced least hemodynamic changes compared to induction with thiopentone and propofol in patients with traumatic brain injury. Consequently, maximal increase in cerebral oxygen saturation occurred with etomidate among the three induction agents. An insignificant decrease in serum cortisol was observed with etomidate and thiopentone, but not with propofol. Neurological outcome at discharge was similar with all drugs.

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Introduction

The compromise of cerebral perfusion and oxygenation in vulnerable acute brain injury patients consequent to postanesthetic induction hypotension is a serious concern in neurosurgery. Prevention of secondary brain insults like cerebral hypoperfusion and hypoxemia to have an optimal patient outcome is one of the prime anesthetic goal in the management of traumatic brain injury (TBI).[1]

The existing literature suggests the benefits of stable hemodynamics with the use of etomidate for anesthetic induction.[2,3] This advantage of etomidate over conventional induction agents is yet to be fully elucidated in patients with TBI, probably due to the concern over postoperative adrenal insufficiency[4] in this population. However, there is evidence suggesting that a single dose of etomidate does not increase adrenal insufficiency in patients with TBI.[5]

One of the methods of assessing cerebral oxygenation in patients with brain trauma is by monitoring regional cerebral oxygen saturation ($rScO_2$) using near-infrared spectroscopy (NIRS) technique.[6] As the physiological changes in the hemodynamic[7] and respiratory parameters[8] can alter cerebral perfusion and oxygenation in patients with acute brain injury, monitoring of $rScO_2$ may help in detecting the cerebral effects occurring as a consequence of anesthetic induction with different anesthetic induction agents.

We hypothesized that etomidate induction, compared to thiopentone and propofol induction, in TBI patients will lead to stable hemodynamics, and thus better cerebral oxygen saturation. The primary objective of our study was to compare the effects of three different intravenous (IV) anesthetic induction agents, that is, thiopentone, propofol, and etomidate, on the systemic hemodynamic parameters and $rScO_2$ in patients with acute brain injury undergoing emergency surgery. The secondary objectives were to evaluate the effect of anesthetic induction agents on serum cortisol levels and outcome at discharge.

Material and Methods

All the adult patients (>18 years) with acute TBI requiring emergency cranial surgery were clinically evaluated for the eligibility and were included after obtaining informed written consent from the next of their kin. Patients who had contraindications to the anesthetic drugs used in the study, those who were intubated outside the operation theater (OT), and those having pulmonary aspiration before anesthetic induction or having extracranial injuries at the site of placement of NIRS sensor, thereby preventing the procurement of baseline $rScO_2$ value, were excluded from the study cohort. This study was carried out in the emergency OT of a tertiary care neurosciences center in South India from December 2020 to December 2021.

The baseline demographic data and data on hematological and biochemical investigations of each patient were collected. Standard anesthesia monitoring as per the American Society of Anesthesiologists recommendations was applied to the patients. In addition, two NIRS sensors (Nonin Equanox Medical, Inc. Plymouth, MN, USA) were placed on the forehead. All patients were preoxygenated for 3 min with 100% oxygen.

Anesthesia was induced with IV fentanyl citrate 1–2 μg/kg followed by preservative-free lignocaine 1 mg/kg. Thereafter, an IV anesthetic induction agent, as per the discretion of the attending anesthesiologist, was administered in the following dose range: etomidate 0.2–0.3 mg/kg, thiopentone 3–5 mg/kg, and propofol 1–2 mg/kg. To facilitate tracheal intubation, rocuronium 1–1.2 mg/kg was administered. Anesthesia was maintained with oxygen: air (50:50) mixture, with the fraction of inspired oxygen (Fi_{O2}) of 50%, and sevoflurane at a minimum alveolar concentration (MAC) between 0.5 and 1.

The patients were divided into three groups depending on the induction agent they received: Group 1- thiopentone, Group 2- propofol, and Group 3- etomidate. In all the patients, hemodynamic parameters of heart rate (HR), mean arterial blood pressure (MAP), end-tidal carbon dioxide ($EtCO_2$), peripheral oxygen saturation (SpO_2), ipsilateral $rScO_2$ on the side of injury ($IrScO_2$), and contralateral $rScO_2$ on the relatively less-injured/uninjured side ($CrScO_2$) were recorded at the following time intervals: (a) baseline, at room air; (b) after 3 min of preoxygenation; (c) after anesthetic induction (at 1, 2, and 3 min); (d) after intubation (at 0, 2, 4, 6, 8, and 10 min); (e) at the end of the surgery, and (f) after extubation or on-tube room air. A baseline serum cortisol sample was taken before the anesthetic induction and subsequently 24 h later. Serum cortisol levels were tested with a SPARK microplate reader (Tecan Group Ltd., Männedorf, Switzerland) using the Calbiotech ELISA kits, which were subsequently analyzed by Spark Control Software. In the event of postinduction hypotension (>20% fall in blood pressure [BP] from baseline), the patient was first resuscitated with normal saline and in case of inadequate response, a bolus dose of 3 mg IV mephentermine was administered. It was repeated if required to a maximum dose of 12 mg. In the event of persistent hypotension, an infusion of noradrenaline (0.05–0.15 μg/kg/min) was started and titrated to maintain a MAP of around 70 mmHg. For refractory hypotension, a

bolus of 100 mg IV hydrocortisone was considered as a last resort. Glasgow coma scale (GCS) scores of all the patients were noted at baseline, immediately after surgery, and at discharge. Patients being extubated immediately after the surgery in each group were also noted.

This study was approved by the Institute Ethics Committee (NIM/DO/IEC (BS and NS DIV)/2019-20 dated 01.27.2020) and it also received a research grant from the Indian Council of Medical Research. This study was registered with the Clinical Trial Registry of India (CTRI/2020/12/029813).

Statistical analysis was performed using the R software version 4.1.2. Since the study was planned as a prospective observational study to maintain pragmatism over induction agent allocation, formal sample size calculation was not conducted. We included all the patients meeting the eligible criteria over a period of 1 year and receiving any of the three anesthetic induction agents. Interval scale and ordinal scale variables are represented by median and interquartile ranges, while nominal variables are represented by frequencies and percentages. Baseline and demographic variable differences were analyzed using the Kruskal–Wallis test or Chi-square test as appropriate for the variable type. Between-group analysis for longitudinal data was conducted using linear mixed effect models (package "lmerTest" of R). Longitudinal time effects were analyzed to observe separate slopes for each time point relative to the baseline. Random effects were specified as random intercepts by the subject. An unstructured covariance matrix was assumed. Results are presented as estimates with 95% confidence levels. Estimates for main effects represent the mean difference relative to the reference for that effect, while the estimate for interaction effect represents a difference of slopes of effect relative to the reference for that effect. The correlation between hemodynamic variables and $rScO_2$ was determined using repeated measures correlation based on generalized linear modeling (package "rmcorr" of R). P value < 0.05 was considered statistically significant.

Results

The total number of patients recruited was 116, of which 115 patients were included in the final analysis. Over the studied period, 32, 33, and 50 patients received thiopentone (Group 1), propofol (Group 2), and etomidate (Group 3), respectively [Figure 1]. The baseline demographics and the variables affecting $rScO_2$, such as region of the injury, hemoglobin levels, SpO_2, and HR, across the three groups were comparable, except for MAP. Also, between the groups, a significant difference was noted in the bilateral baseline $rScO_2$ [Table 1].

Compared to etomidate, changes in MAP and HR were significant with thiopentone (MAP, $P < 0.001$; HR, $P < 0.001$), but not with propofol (MAP, $P = 0.313$; HR, $P = 0.854$).

Change across time average across all groups (main effect of time) was statistically significant for MAP ($P < 0.008$), but not for HR ($P < 0.067$). Compared to etomidate, in the propofol group, the trend changes of MAP across the study time points were statistically significant with an observed decrease in MAP (estimate = –0.44 (–0.72 to –0.15); $P = 0.003$), though the trend changes in HR were insignificant ($P = 0.412$). In the thiopentone group, compared to the etomidate group, trend changes in MAP and HR across the study time points were insignificant (MAP, $P = 0.770$; HR, $P = 0.081$) [Table 2, Figure 2].

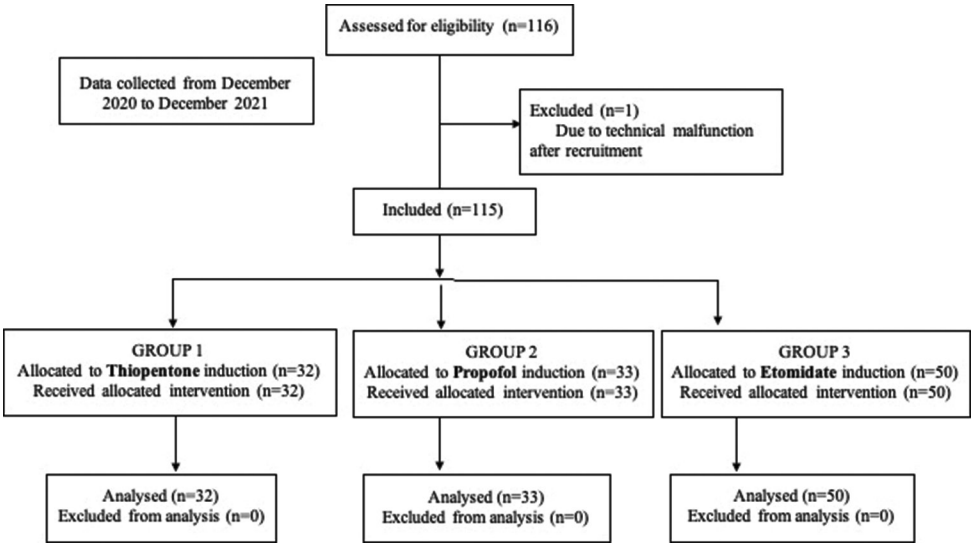

Figure 1: The study flow diagram in line with the Strengthening the Reporting of Observational Studies in Epidemiology(STROBE) guidelines

Table 1: Demographics and baseline variables

Variable	Etomidate *n*=50	Propofol *n*=33	Thiopentone *n*=32	*P*
Age (years)	41.4±13.6	37.4±14.2	39.6±13	0.438
Gender (F:M), *n* (%)	12 (24%):38 (76%)	3 (9.1%):30 (90.9%)	8 (25%):24 (75%)	0.178
Preoperative GCS	13 (11–15)	13 (11–15)	13 (11–14)	0.446
Predominant area of injury				
Frontal	14 (28%)	9 (27.3%)	10 (31.3%)	0.933
Temporal	30 (60%)	18 (54.5%)	17 (53.1%)	
Other	6 (12%)	6 (18.2%)	5 (15.6%)	
Midline shift (cm)	0.6±0.3	0.5±0.3	0.5±0.4	0.272
Hb (g/dl)	13.4±2.3	14.1±1.7	13.9±2.4	0.363
International Normalized Ratio (INR)	1.2±0.2	1.2±0.2	1.2±0.3	0.798
Serum creatinine (mg/dl)	0.9±0.3	0.9±0.2	0.9±0.3	0.575
GRBS (mg/dl)	144.6±41.8	135.3±39.4	151.4±42.8	0.182
Ipsilateral baseline $rScO_2$ (%)	63.5±7.7	67.3±6.1	63.6±7	0.014
Contralateral baseline $rScO_2$ (%)	68.3±5.4	70.7±5	69.1±4.3	0.032
Baseline SpO_2 (%)	96.4±0.9	96.7±0.9	96.3±0.8	0.137
Baseline HR (bpm)	91±16.9	89.9±15.7	83.5±15.3	0.087
Baseline MAP (mmHg)	94±9.5	96.5±9.3	88±8	<0.001
Baseline $EtCO_2$ (mmHg)	35.36±1.64	35.91±1.33	35.28±1.91	0.093

bpm=beats per minute, $EtCO_2$=end-tidal carbon dioxide, F=female, GCS=Glasgow Coma Scale, GRBS=general random blood sugar, Hb=hemoglobin, HR=heart rate, M=male, MAP=mean arterial pressure, $rScO_2$=regional cerebral oxygen saturation, SpO_2=peripheral oxygen saturation. Values are expressed as mean±standard deviation, median (interquartile range), or as number (percentage)

Table 2: Comparison of study variables between groups and across time

	Ipsilateral $rScO_2$ (%)		Contralateral $rScO_2$ (%)		MAP (mmHg)		HR (bpm)	
	Estimate	*P*	Estimate	*P*	Estimate	*P*	Estimate	*P*
(Intercept)	62.15 (61.12–63.18)	<0.001	66.45 (65.68–67.23)	<0.001	93.13 (91.91–94.35)	<0.001	90.57 (88.52–92.62)	<0.001
Propofol (Ref=etomidate)	3.16 (1.49–4.84)	<0.001	2.18 (0.94–3.41)	0.001	1 (-0.94–2.94)	0.313	-0.31 (-3.56–2.95)	0.854
Thiopentone (Ref=etomidate)	0.58 (-1.09–2.26)	0.493	1.11 (-0.13–2.36)	0.080	−5.77 (−7.73 to -3.81)	<0.001	−7.25 (-10.53 to -3.97)	<0.001
Time (Ref=baseline)	1.61 (1.46–1.76)	<0.001	1.65 (1.54–1.77)	<0.001	-0.24 (-0.42 to -0.06)	0.008	-0.28 (-0.59–0.02)	0.067
Propofol*Time (Ref=Etomidate*Time)	-0.18 (-0.42–0.07)	0.162	-0.22 (-0.4 to -0.04)	0.019	-0.44 (-0.72 to -0.15)	0.003	-0.2 (-0.68–0.28)	0.412
Thiopentone*Time (Ref=Etomidate*Time)	0.03 (-0.22–0.27)	0.838	-0.06 (-0.24 to 0.12)	0.529	-0.04 (-0.33–0.25)	0.770	0.43 (-0.05–0.92)	0.081

bpm=beats per minute, HR=heart rate, MAP=mean arterial blood pressure, rScO2=regional cerebral oxygen saturation. Values are expressed as mean±standard deviation

The change in bilateral $rScO_2$ was significantly higher in the propofol group compared to etomidate group ($IrScO_2$: estimate 3.16, 95% confidence interval [CI] 1.49–4.84, $P < 0.001$ and $CrScO_2$: estimate 2.18, 95% CI 0.94–3.41, $P < 0.001$). The thiopentone group did not show any difference in the bilateral $rScO_2$ relative to etomidate ($IrScO_2$: $P = 0.493$ and $CrScO_2$: $P = 0.080$, respectively). Changes in $IrScO_2$ and $CrScO_2$ across time average across all groups (main effect of time) were statistically significant ($P < 0.001$). Compared to etomidate, in the propofol group, trend changes in $IrScO_2$ across the study time points were not statistically different ($P = 0.162$), though the trend changes in $CrScO_2$ were significant (estimate = –0.22 (–0.4 to –0.04), $P = 0.019$) with a lesser increase in $CrScO_2$ with propofol compared to etomidate.

In the thiopentone group compared to etomidate, trend changes in bilateral $rScO_2$ across the study time points were insignificant ($IrScO_2$: $P = 0.838$, $CrScO_2$: $P = 0.529$) [Table 2, Figure 2].

At 24 h after anesthetic induction, an insignificant decrease in median serum cortisol levels was observed in the thiopentone ($P = 0.364$) and etomidate ($P = 0.332$) groups, whereas the propofol group revealed a statistically significant increase in serum cortisol levels after surgery ($P = 0.004$) [Table 3]. On pairwise intergroup comparison, a significant difference was observed in the changes in serum cortisol levels between the etomidate and propofol groups ($P = 0.006$), but it was comparable between thiopentone and etomidate ($P = 0.184$) [Table 3].

Table 3: Comparison of baseline and 24-h postinduction serum cortisol levels between groups and within groups

	Group	Preinduction serum cortisol (ng/ml)	24-h postinduction serum cortisol (ng/ml)
Descriptive	Etomidate	223.55 (157.7–353.075)	198.996 (152.522–298.8)
	Propofol	172.918 (119.236–288.625)	223.872 (165.948–321.84)
	Thiopentone	245.846 (166.016–334.653)	228.914 (179.275–353.475)
Type of test	**Pairs**	**Effects**	***P***
Omnibus test	Overall	Group	0.519
		Time points	0.099
		Group*time points	0.021
Pairwise tests	Etomidate: propofol	Group	0.554
	Etomidate: propofol	Group*time points	0.006
	Etomidate: thiopentone	Group	0.544
	Etomidate: thiopentone	Group*time points	0.184
	Propofol: thiopentone	Group	0.302
	Propofol: thiopentone	Group*time points	0.140
Within group effects	Etomidate	Time points	0.332
	Propofol	Time points	0.004
	Thiopentone	Time points	0.364

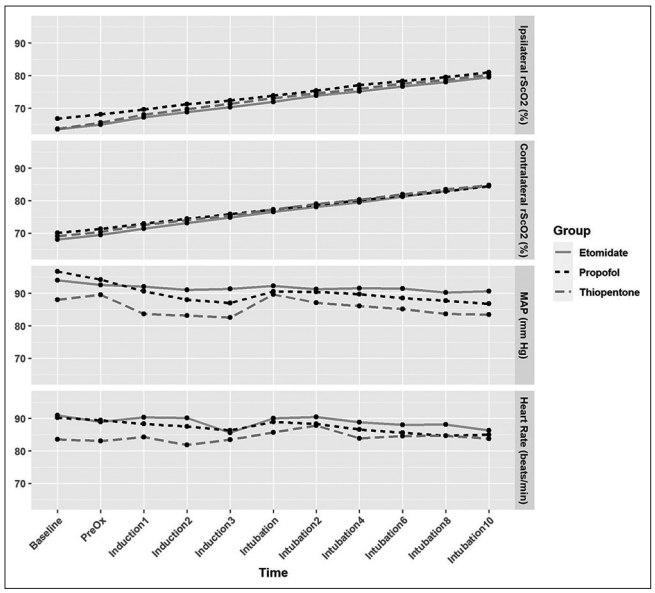

Figure 2: Line diagram showing trend changes in bilateral $rScO_2$ and hemodynamic parameters among the three groups over the induction period, that is, at baseline, 3 min of preoxygenation, postinduction at 1, 2, and 3 min, and postintubation immediately after and then at 2-min intervals till 10 min. $rScO_2$ = regional cerebral oxygen saturation

In all three groups, the GCS score showed a significant improvement immediately after the surgery and at discharge compared to the preinduction state ($P < 0.001$). Between-group comparison did not reveal any significant difference in the GCS change [Table 4]. On observing the number of patients who got extubated on table immediately after the surgery, a total of 57 patients were extubated, with the observed rate of extubation being 54% (27/50), 48% (16/33), and 43% (14/32) in etomidate, propofol, and thiopentone groups, respectively. Although the percentage of extubation in etomidate group was highest, the intergroup difference was insignificant ($P = 0.656$).

Discussion

The ideal anesthetic induction agent in patients with TBI maintains stable systemic hemodynamics and intracranial physiology with regards to cerebral perfusion and intracranial pressure (ICP). Thiopentone and propofol are commonly used anesthetic induction agents in patients undergoing surgery for TBI. However, despite etomidate being a cardiostable drug, its use in TBI remains uncommon due to concerns regarding involuntary muscle movements and adrenal suppression associated with cortisol deficiency.[9,10]

In our study in patients with TBI, we observed stable hemodynamics (MAP and HR) in the etomidate group and the changes were comparable with thiopentone induction. However, there was a statistically significant decrease in MAP, but not HR across the studied time points, with the use of propofol compared to etomidate, though it was not clinically significant. Similar observations were noted by Uygur *et al.*[11] who studied the effects of these three induction agents in 45 adult patients (15 patients in each group) undergoing elective surgery. They observed a significant decrease in BP and cardiac output in the propofol group compared to the thiopentone and etomidate groups, with no difference in HR changes, suggesting better hemodynamic stability with etomidate.

In patients with a unilateral intracranial space-occupying lesion (ICSOL), a significant decrease in the systemic and cerebral hemodynamic parameters was noted following induction with propofol, but not etomidate.[12] In another study, in eight adult patients undergoing surgery for ICSOL, the authors observed a significant reduction in ICP

Table 4: Comparison of baseline, immediate postoperative, and at discharge GCS scores between the groups and within the groups

	Group	Immediate preoperative GCS	Immediate postoperative GCS	Discharge GCS
Descriptive	Etomidate	13 (11–15)	12 (9–14)	15 (13–15)
	Thiopentone	13 (9.5–13.5)	11.5 (7.25–13)	14.5 (11–15)
	Propofol	13 (11–15)	11 (9–13)	14 (13–15)
Type of test		**Pairs**	**Effects**	***P***
Omnibus test		Overall	Group	0.659
			Time points	<0.001
			Group*time points	0.520
Pairwise tests		Etomidate: propofol	Group	0.863
		Etomidate: propofol	Group*time points	0.697
		Etomidate: thiopentone	Group	0.388
		Etomidate: thiopentone	Group*time points	0.495
		Propofol: thiopentone	Group	0.509
		Propofol: thiopentone	Group*time points	0.307
Within group effects		Etomidate	Time points	<0.001
		Propofol	Time points	<0.001
		Thiopentone	Time points	<0.001

GCS=Glasgow Coma Scale

without any significant changes in MAP, cerebral perfusion pressure, and HR following induction with etomidate to electroencephalogram burst suppression.[13] Thus, the findings of these studies including our study establish the safe hemodynamic profile of etomidate compared to other induction agents, even in the neurosurgical population.

Further, we observed a significant increase in bilateral $rScO_2$ after anesthetic induction with all three drugs. On comparing the trend changes between the groups, changes in $CrScO_2$ were significantly more with etomidate compared to propofol, though they were not clinically significant, whereas changes in bilateral $rScO_2$ were comparable between thiopentone and etomidate. In 36 patients undergoing elective day care surgeries, authors observed the impact of anesthetic induction agents, that is, etomidate, propofol, and thiopentone, on cerebral oxygenation using a NIRO 500 spectrophotometer.[14] Cerebral oxygenation was observed to increase following induction with thiopentone and propofol, whereas it decreased with etomidate in their study. In contrast, we observed an increase in $rScO_2$ in all the patients, including those receiving etomidate. This increase in $rScO_2$ in the relatively normal side of the brain was more in the etomidate group. The contrasting findings could be due to the difference in the doses of anesthetic agents used, the study population, and the monitoring device used for assessing $rScO_2$.

Changes in $rScO_2$ with anesthetic induction are a net result of multiple factors including the FiO_2 used, the degree of metabolic suppression achieved, the status of cerebral autoregulation, and hemodynamic changes that accompany anesthetic induction and tracheal intubation. The decrease in $rScO_2$ may be from a disproportionate decrease in cerebral blood flow (CBF) compared to the decrease in metabolism ($CMRO_2$). Reduction in ICP, CBF, and $CMRO_2$ has been observed with the use of etomidate.[15] Decrease in CBF with etomidate has been attributed to the reduction in $CMRO_2$[16] and/or its direct vasoconstrictive action mediated by nitric oxide synthase (NOS) inhibition.[17] The latter observation was made in the setting of focal cerebral ischemia induced by mechanical obstruction of the cerebral vessel in rats, where etomidate was administered as a continuous infusion over 6 h. Acute brain injury presents with a myriad of altered physiological changes consequent of the site of hematoma.[18] Though NOS inhibition has been described in the pathophysiology of acute TBI impeding CBF in the acute stage of injury,[19] the role of single-dose anesthetic induction with etomidate in further inhibiting NOS and reducing CBF is not currently clear.

A single dose of etomidate at anesthetic induction has not led to adrenal suppression in patients with sepsis.[20] Currently, etomidate is used for rapid sequence induction and intubation in the emergency department as well as in the intensive care units.[21,22] TBI *per se* can lead to adrenal suppression. In patients with polytrauma ($n = 49$), on studying the impact of TBI ($n = 36$) versus non-TBI ($n = 13$) on the total cortisol, corticosteroid-binding globulin (CBG), and free cortisol levels at baseline and 12 and 24 h of trauma, significantly lower concentrations of total and free cortisol were observed in TBI patients.[4] This difference was attributed to the different concentration dynamics in TBI patients compared to non-TBI patients. Further, a decreasing trend in serum cortisol levels correlated with the severity of GCS and cortisol levels strongly

correlated with age, where inadequate post-TBI cortisol levels were observed in older patients.

On studying the effect of a single dose of etomidate ($n = 15$) used for intubation in adults patients of moderate to severe TBI, compared to other anesthetic drugs ($n = 25$), etomidate did not increase the cumulative incidence of adrenal insufficiency when tested by adrenocorticotropic hormone (ACTH) stimulation test done at 24, 48, and 168 h following the intubation.[5] Though a significant difference in delta cortisol was observed only at 24 h in the etomidate group, its clinical impact was not studied. In our study, we observed an insignificant decrease in postoperative serum cortisol levels with the use of etomidate and thiopentone and, contrarily, an increase in the propofol group at 24 h postinduction.

The increase in the propofol group could be due to the relatively younger age of the study cohort in the propofol group (37.4 ± 14.2 years) in comparison to the thiopentone group (39.6 ± 13 years) and the etomidate group (41.4 ± 13.6 years), although the age difference was insignificant ($P = 0.438$). On studying the trend of GCS till discharge, we observed an improvement in the median GCS in all three studied groups, with the highest median GCS of 15 (11–15) at discharge observed in the etomidate group.

Serial serum cortisol and blood glucose levels in TBI patients were found to be elevated in the comatose patients who passed away within 48 h of brain trauma (serum cortisol = 1600 nmol/l, blood glucose = 11 mmol/l).[23] The majority of the patients in our study had favorable GCS scores at discharge. Out of 115 patients, only four died during the hospital stay, of which two patients died within 24 h of surgery before their serum cortisol samples could be collected, whereas the other two deceased patients had 24-h postoperative serum cortisol values of 211.3 and 401.32 ng/ml, respectively.

Strengths and limitations: To the best of our knowledge, this is the first study evaluating the effects of anesthetic induction agents on $rScO_2$ in patients with TBI. This study provided insight into the interplay between drugs used for anesthetic induction, systemic hemodynamics, and $rScO_2$. The study limitations include a single-center (generalizability), observational design (bias) and a small sample of the study population (power).

Conclusions

In TBI patients, on comparing the three induction agents, the hemodynamic fluctuations were least with the use of etomidate. All three anesthetic agents led to an increase in bilateral $rScO_2$ after induction and intubation, with a significant increase being observed in the etomidate group compared to propofol and thiopentone. Insignificant fall in serum cortisol was observed with etomidate and thiopentone, but not with propofol. Outcome at discharge, assessed with GCS, was comparable in all the groups. Thus, all the three anesthetic induction agents maintain cerebral oxygenation in TBI patients undergoing emergency surgery.

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Data availability

The data that support the findings of this study are available from the corresponding author, RMS, upon reasonable request.

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Conflicts of interest

There are no conflicts of interest.

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