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Original Article

Effect of Dexmedetomidine Versus Midazolam on Intracranial Pressure in Traumatic Brain Injury Through Ultrasonographic Measurement of Optic Nerve Sheath Diameter

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ABSTRACT

Background: Traumatic brain injury (TBI) is brain injury triggered by extrinsic mechanical stress leading to behavioral, cognitive, physical, and emotional symptoms. Outcome can vary from recovery to disability or mortality. The current study aim was evaluating the impact of dexmedetomidine versus midazolam on intra cranial pressure through ultrasonographic assessment of optic nerve sheath diameter (ONSD) in cases of TBI. **Methods**: This randomized clinical trial study was performed on one hundred thirty-six TBI cases who were divided equally into; Group dexmedetomidine (Group DEX): 68 patients received a loading dose of DEX (1 µg/kg over 10 minutes) followed by a maintenance infusion dose (0.2-0.7 µg/kg/h) for 24 hours as a sedative agent. Group Midazolam (Group MDZ): 68 patients received a loading dose of Midazolam (0.05-0.1 mg/kg) followed by a maintenance infusion dose (30-120 mcg/kg/hr) for 24 hours as a sedative agent. All cases were subjected to clinical assessment and laboratory, and ONSD examination. **Results**: Right and left ONSD post sedation at (at 6, 12, 18 and 24h) were significantly lower in DEX group compared to MDZ group ($P<0.05$). HR at 6 h, 12 and 18 h post sedation was notably reduced in DEX group compared to MDZ group ($P=0.004$, <0.001, <0.001 respectively). Concerning the adverse events, vomiting occurred in 3 (4.41%) cases in group D and 5 (7.35%) cases in group M, hypotension occurred in only 7 (10.29%) cases in group D and not reported in group M. **Conclusion**: dexmedetomidine may be a more effective sedative option for reducing ICP in TBI patients compared to midazolam. However, the administration of dexmedetomidine was correlated with a higher risk of bradycardia, which may require closer monitoring and potential intervention. Both medications were found to cause a decrease in blood pressure, although the magnitude of the effect was not substantially varied between the groups. **Keywords**: Dexmedetomidine, Midazolam, Intracranial Pressure, Ultrasonographic, Optic Nerve

INTRODUCTION

Traumatic brain injury (TBI) is described as brain injury caused by a mechanical impact that produces physical, cognitive, and behavioral symptoms. The outcome may vary from full recovery to severe impairment or death. TBI is a notable cause of illness and mortality globally, primarily among adolescent and children [1]. Detection of post-traumatic increased intracranial pressure (ICP) may enable in early momentary operations and final therapy in the neurologically impaired cases and is connected with better outcomes [2]. Cerebral edema and elevated ICP are the most serious immediate outcomes of TBI, accounting for the majority of early deaths. A rise in ICP may block cerebrospinal fluid (CSF) and result in ischemic injury, and its severity

and persistence are correlated with the outcome following TBI [3]. Cases with TBI require sedation to produce anxiolysis, reduce agitation, and enable mechanical respiration [4].

Dexmedetomidine (DEX) has sedative, analgesic, and sympatholytic impacts. It lowers neuronal mortality by defending against neuro-inflammation, and autophagy and improving motor and cognitive function after TBI, and alleviating cerebral ischemic reperfusion injury [5]. It lowers oxygen consumption, brain metabolic rate, and could decrease ICP by reducing CSF pressure while posing no danger of cerebral ischemia. In traumatic cases, α2-agonist action may cause arterial vasoconstriction, resulting in lowering ICP, in addition to decreased volume of cerebral blood [6].

Midazolam (MDZ) is commonly used in clinical practice, as it lowers the ICP by lowering the cerebral metabolism [5]. Midazolam, an affordable benzodiazepine, is best suited for sedation in TBI cases. It has a short half-life that is sensitive to context and has a rapid onset and offset of action [7].

The optic nerve is covered by a sheath that extends directly into the subarachnoid space, therefore any increase in ICP causes dilatation of the optic nerve sheath, resulting in a rise in sheath diameter due to higher CSF pressure. Ultrasonography (US), a non-invasive and quick approach for identifying increased ICP, can be utilized to quantify the optic nerve sheath diameter (ONSD) [8].

The present work aim was evaluation the effect of dexmedetomidine versus midazolam on intra cranial pressure through US measurement of ONSD in cases of TBI.

METHODS

This randomized clinical trial study was conducted for six months from October 2023 to April 2024 at the emergency and surgical intensive care units, Department of Anesthesia, intensive care and pain management, Zagazig university hospital.Verbal and written informed consent was obtained from 1st degree relatives of participants after an explanation of the procedure and medical research. The research was conducted under the World Medical Association's Code of Ethics (Helsinki Declaration) for human research. This study was carried out after the approval of the Institutional Review Board (IRB#101037).

Cases with the following criteria were included; Acceptance of patient relative first degree. Patients classified as ASA (American Society of Anesthesiologists) physical status I to III, including those with trauma. Cases between 18 and 64years. Restless and agitated cases who were in need for close follow-up and sedation. TBI patient have CT brain with elevated ICP abnormalities, TBI patients with agitated and restless patients who were in need for sedation and close follow up with Glasgow Coma Scale (GCS) from 6 to 10. Cases with the following characteristics were excluded; History of allergy to studying drugs. Patients with history of ocular pathology, ocular trauma, and/or history of a previous eye surgery. Patients with advanced liver diseases. AV block with HR< 45 beat/min Patients with severe preadmission hemodynamic instability, and patients with known elevated ICP before the trauma. All patients of the studied groups were subjected to Initial assessment: The initial resuscitation happened simultaneously with the primary evaluation. When a life-threatening problem was discovered, prompt corrective measures were implemented before proceeding to the next phase. All polytrauma cases in the resuscitation room were subjected to the following: FAST (Focused Assessment Sonography for Trauma cases): for potential internal hemorrhage. Chest and pelvic X-ray: Anteroposterior supine view for unstable cases with a portable X-ray machine. After resuscitation and stabilization of patient, CT brain was done for looking for signs of increased ICP according to Rotterdam classification [9]. ***Secondary survey:*** Following the initial resuscitation attempt, all individuals were subjected to full history within the first hour after arrival.

Clinical Evaluation within the first hour after arrival: Vital signs (pulse, mean arterial blood pressure, respiratory rate), neurological examination (sensory and motor), and complete general assessment. These evaluations were measured as standard for the patients on admission and repeated every 6 hours for 24 hours. After initial assessment and neurological examination and pre sedation measurement of optic nerve sheath diameter. One hundred thirty-six traumatic brain injury patients were allocated randomly by using computer randomization program (Random-allocation software) into two equal groups. Group dexmedetomidine (Group DEX): 68 patients with TBI received a loading dose of DEX followed by a maintenance infusion dose. The loading dose was 1 µg/kg over 10 minutes, and the maintenance infusion dose was 0.2-0.7 µg/kg/h for 24 hours as a sedative agent. Group Midazolam (Group MDZ): 68 patients with TBI received a loading dose of Midazolam followed by a maintenance infusion dose. The loading dose was 0.05-0.1 mg/kg, and the

maintenance infusion dose was 30-120 mcg/kg/hr for 24 hours as a sedative agent.

Laboratory tests: Blood grouping and complete blood count. Liver functions test (Bilirubin, ALT, Albumin, and AST). Kidney functions test (serum creatinine, BUN). Coagulation profile (INR, PT, and PTT). ONSD measurement performed by U/S for all patients for patients who had increased intracranial pressure confirmed by CT brain. Eye scans were performed for patients using portable color ultrasounds (Mindray Z 60) within 15 minutes after brain CT scans. The ONSD was measured bilaterally at 3 mm before entrance of the optic nerve to the globe. The patient's closed eyelids were covered with a sterile transparent dressing. The high-frequency linear array probe was evenly applied with a conductive gel to scan the cross-sectional and sagittal surfaces of one side of the eyeball. The ultrasound probe was placed horizontally or vertically on the patient's eyeball. The probe angle and depth during operation was slightly adjusted to obtain a clear optic nerve sheath (ONS) image without pressing the eyeball. The optic nerve sheath diameter was measured in both eye pre sedation and 6 hours, 12 hours, 18 hours,24 hours post sedation. The final ONSD value was the average of the four measurements.

Statistical Analysis: The statistical analysis was performed using SPSS v28 (IBM Inc., Armonk, NY, USA). Quantitative variables were expressed as mean and SD and compared between the two groups using the unpaired Student's t-test. Qualitative variables were provided as frequency and percentage (%) and examined using the Fisher's exact test or Chi-square test, as appropriate. A two-tailed P value of <0.05 was deemed significant. Repeated measures ANOVA is used to examine groups of related dependent variables that represent various measurements of the same property.

RESULTS

In this study, 179 cases were examined for eligibility; 24 did not match the requirements, and 19 declined to participate. The rest of the 136 cases were split randomly into 2 groups of 68 patients each. All cases were monitored and statistically assessed. (Fig. 1)

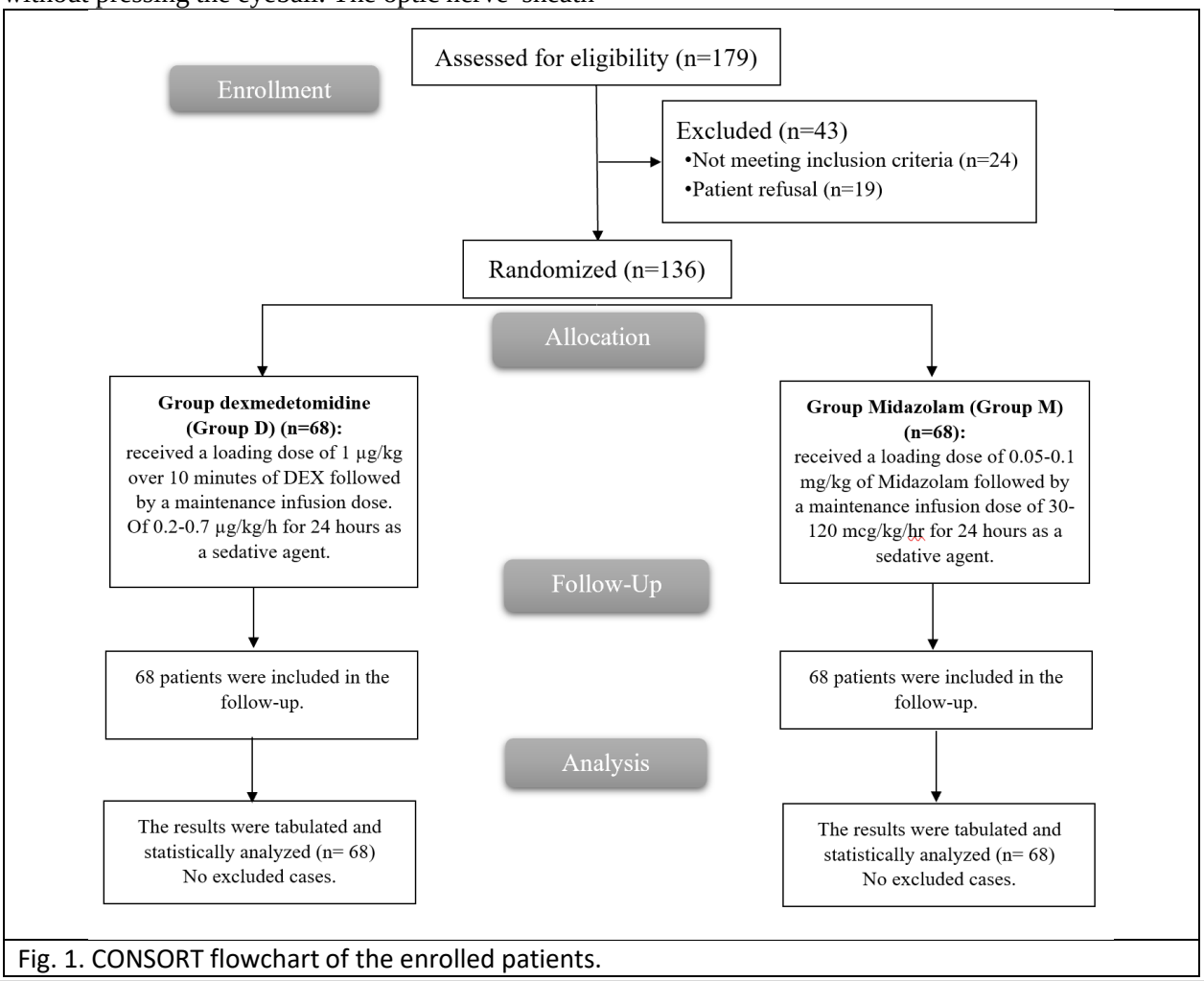

Fig. 1. CONSORT flowchart of the enrolled patients.

There was non-substantial variation between the groups concerning demographic, anthropologic, and laboratory data (P>0.05). (Table 1,2).

Table 1: Demographic and anthropologic data of the studied groups

		Group D (n=68)	Group M (n=68)	P value
Age (years)		40.06 ± 11.02 (21 – 60)	39.66 ± 11.19 (21 – 60)	0.835
Sex	Male	52 (76.5%)	51 (75%)	0.841
	Female	16 (23.5%)	17 (25%)	
Weight (Kg)		75.29 ± 10.23 (55 – 93)	76.35 ± 9.42 (55 – 93)	0.531
Height (m)		1.71 ± 0.06 (1.55 - 1.82)	1.7 ± 0.06 (1.55 - 1.82)	0.576
BMI (Kg/m2)		25.7 ± 2.77 (22.34 - 32.21)	25.9 ± 2.5 (22.34 - 32.21)	0.660
BMI: Body Mass Index				

Table 2: Laboratory investigations pre sedation of the studied groups

	Group D (n=68)	Group M (n=68)	P value
Hb (g/dL)	13.01 ± 0.84 (11.2 - 14.6)	12.79 ± 0.74 (10.9 – 14)	0.103
PLT (*109/L)	280.16 ± 60.78 (156 – 380)	288.4 ± 69.32 (169 – 399)	0.463
Hct (%)	45.49 ± 4.78 (38 – 53)	44.69 ± 4.95 (36 – 52)	0.343
RBCs (*1012/L)	4.95 ± 0.58 (3.9 - 5.8)	4.91 ± 0.57 (3.8 - 5.9)	0.744
WBCs (*109/L)	7.48 ± 1.9 (4.5 - 10.9)	7.91 ± 1.68 (4.7 - 10.8)	0.163
Neutrophil (*109/L)	4.93 ± 1.31 (2.66 – 7)	4.6 ± 1.24 (2.52 - 6.84)	0.140
Lymphocyte (*109/L)	2.56 ± 0.86 (1.04 - 3.93)	2.55 ± 0.82 (1.01 - 3.85)	0.920
Eosinophils (*109/L)	0.25 ± 0.14 (0.02 - 0.5)	0.23 ± 0.16 (0 - 0.49)	0.454
INR	0.9 ± 0.06 (0.8 – 1)	0.91 ± 0.06 (0.81 – 1)	0.675
ALT (U/L)	34.49 ± 8.43 (20 – 47)	33.38 ± 7.17 (22 – 45)	0.412
AST (U/L)	32.88 ± 5.52 (23 – 43)	34.34 ± 6.33 (24 – 45)	0.155
Hb: hemoglobin, PLT: platelets, Hct: hematocrit, RBCs: red blood cells, WBCs: white blood cells, INR: intranational normalized ratio, ALT: alanine aminotransferase, AST: aspartate aminotransferase.			

Twenty-four hours post sedation CT brain findings substantially varied between both groups (P=0.027), they were substantially better in group D compared to group M. In the present study, 31.6% of patients have had a GCS of 10/15, 50% of patients presented with a GCS of 9/15, while 18.4% of patients were presented with a GCS of 8/15. There was non-significant variance between groups concerning GCS, pupils, lateralization and reflexes. (Table 3).

Heart Rate (HR) at 6 h, 12 and 18 h post sedation was remarkably reduced in group D compared to group M (P=0.004, <0.001, <0.001 respectively), with non-substantial variation between both group regrading HR pre sedation and 24 h post sedation. (Table 4)

Table 3: CT brain findings based on Rotterdam score and neurological examination pre sedation

		Group D (n=68)	Group M (n=68)	P value
Pre sedation	Score 2	4 (5.88%)	0 (0%)	0.163
	Score 3	36 (52.94%)	32 (47.06%)	
	Score 4	25 (36.76%)	29 (42.65%)	
	Score 5	3 (4.41%)	6 (8.82%)	
	Score 6	0 (0%)	1 (1.47%)	
24 h post sedation	Score 2	36 (52.94%)	20 (29.41%)	0.027*
	Score 3	25 (36.76%)	37 (54.41%)	
	Score 4	7 (10.29%)	9 (13.24%)	
	Score 5	0 (0%)	2 (2.94%)	
	Score 6	0 (0%)	0 (0%)	
GCS	Mean ± SD	7.87 ± 0.79	8.03 ± 0.83	0.246
	Range	7 - 9	7 - 9	
	8/15	12 (17.65 %)	13 (19.12 %)	0.940
	9/15	35 (51.47 %)	33 (48.53 %)	
	10/15	21 (30.88 %)	22 (32.35 %)	
Pupils	Not reactive	64 (94.12 %)	65 (95.59 %)	1.00
	NAD	4 (5.88 %)	3 (4.41 %)	
Lateralization	NAD	64 (94.12 %)	66 (97.06 %)	0.680
	Yes	4 (5.88 %)	2 (2.94 %)	
Reflexes	Hyporeflexia	6 (8.82 %)	3 (4.41 %)	0.493
	NAD	62 (91.18 %)	65 (95.59 %)	

CT: computed tomography, GCS: Glascow coma scale, NAD: no abnormality detected *: statistically significant as p value <0.05.

Table 4: Heart rate (bpm) of the studied groups

	Group D (n=68)	Group M (n=68)	P value
Pre sedation	80.6 ± 11.52 (60 – 100)	80.31 ± 11.94 (61 – 100)	0.884
6 h post sedation	75.87 ± 9.07 (60 – 90)	80.66 ± 9.9 (63 – 96)	0.004*
12 h post sedation	71.21 ± 8.73 (58 – 87)	77.63 ± 7.28 (66 – 89)	<0.001*
18 h post sedation	68.19 ± 8.05 (55 – 80)	77.07 ± 7.51 (65 – 90)	<0.001*
24 h post sedation	86.62 ± 8.77 (72 – 100)	88.65 ± 7.02 (77 – 100)	0.138
*: statistically significant as p value <0.05.			

There was non- substantial variation between groups concerning the systolic blood pressure (SPB) and Diastolic blood pressure (DPB), pre- and post-sedation (at 6, 12, 18 and 24h). (Table 5). Right and left ONSD post sedation

at (at 6, 12, 18 and 24h) were significantly lower in DEX group compared to MDZ group (P<0.05). (Table 6). Concerning the adverse events, vomiting occurred in 3 (4.41%) cases in group D and 5 (7.35%) cases in group M, hypotension occurred in only 7 (10.29%) cases in group D and not reported in group M. (Table 6)

Table 5: Systolic and diastolic blood pressure (mmHg) of the studied groups

	Group D (n=68)	Group M (n=68)	P value
SBP (mmHg)			
Pre sedation	119.01 ± 6.26 (110 – 130)	119.81 ± 6.7 (110 – 130)	0.476
6 h post sedation	124.53 ± 3.14 (120 – 130)	125.43 ± 3.09 (120 – 130)	0.095
12 h post sedation	120.04 ± 5.69 (110 – 130)	121.24 ± 6.13 (111 – 134)	0.242
18 h post sedation	124.07 ± 4.16 (110 – 130)	125.12 ± 3.82 (120 – 135)	0.130
24 h post sedation	120.1 ± 6.69 (110 – 130)	121.78 ± 5.72 (110 – 130)	0.119
DBP (mmHg)			
Pre sedation	77.66 ± 1.88 (75 – 80)	77.18 ± 2.15 (75 – 85)	0.164
6 h post sedation	74.68 ± 2.83 (70 – 80)	75.22 ± 3.19 (70 – 80)	0.295
12 h post sedation	77.51 ± 4.5 (70 – 85)	76.65 ± 4.67 (70 – 85)	0.272
18 h post sedation	77.53 ± 1.91 (75 – 80)	77.54 ± 1.62 (75 – 80)	0.962
24 h post sedation	77.19 ± 1.55 (75 – 80)	77.51 ± 1.72 (75 – 80)	0.251
SBP: systolic blood pressure, DBP: diastolic blood pressure.			

Table 6: ONSD (mm) assessment and adverse events of the studied groups

		Group D (n=68)	Group M (n=68)	P value
ONSD				
Right ONSD (mm)	Pre sedation	8.53 ± 0.63	8.73 ± 0.82	0.113
	6 h post sedation	8.31 ± 0.41	8.54 ± 0.71	0.022*
	12 h post sedation	8.13 ± 0.37	8.39 ± 0.61	0.003*
	18 h post sedation	7.56 ± 0.23	8.17 ± 0.32	<0.001*
	24 h post sedation	7.15 ± 0.43	7.97 ± 0.62	<0.001*
Left ONSD (mm)	Pre sedation	8.48 ± 0.46	8.61 ± 0.73	0.216
	6 h post sedation	8.23 ± 0.22	8.42 ± 0.17	<0.001*
	12 h post sedation	7.89 ± 0.76	8.29 ± 0.75	0.002*
	18 h post sedation	7.45 ± 0.39	8.09 ± 0.95	<0.001*
	24 h post sedation	7.12 ± 0.64	7.8 ± 0.66	<0.001*
Adverse events				
Vomiting		3 (4.41%)	5 (7.35%)	0.718
Hypotension		7 (10.29%)	0 (0%)	0.115
Local anesthetic toxicity		0 (0%)	0 (0%)	---
ONSD: optic nerve sheath diameter, *: statistically significant as p value <0.05.				

DISCUSSION

TBIs are a serious public health concern. In accordance with the World Health Organization, TBIs from road collisions was found to be the 3rd leading cause of illness and disability globally [10]. Elevated ICP and cerebral edema are the most serious immediate outcomes of TBI, accounting for the majority of early mortality. A rise in ICP may hinder cerebral blood flow (CBF) and contribute to ischemia, and its severity and persistence are correlated with outcomes following TBI [11].

The aim of the study was to assess the impact of DEX versus MDZ on ICP through US measurement of ONSD in cases of TBI. In the current study, the demographic features of the study individuals, including age, gender, and anthropometric measures, were comparable between the groups, with no statistically substantial variations. This closeness assures that any observed variations in outcomes are credited to the study interventions rather than confounding factors.

These findings agreed with Huang et al. [5] who performed a single-center randomized investigation to compare the impact of DEX and MDZ in 115 cases with TBI. They reported no notable variation between both groups concerning demographic data, weight, height, and BMI. In same line with our findings, Peng et al. [12] performed a study to evaluate DEX against MDZ in 456 cases subjected to peripheral operations with mild TBIs. They found that there was no significant difference in BMI, weight, and height and demographical measurements except age between both cohorts.

Also, Gong et al. [13] investigated the impacts of DEX on recurrence and sedation following hematoma removal in 161 cases with postoperative hypertensive intracerebral hemorrhage (HICH). The cases were separated into two groups: DEX (86 cases) and MDZ (78 cases). No major variations in monitoring indices, including age, sex, and weight were found between both groups. In this study, there was no substantial variation between groups concerning SBP and DBP pre and post sedation. However, HR mean values were remarkably reduced in DEX group compared to group M at 6, 12, 18 hr post-sedation ($p= 0.004$, 0.001, and 0.001 respectively). The observed bradycardia in the dexmedetomidine group is a well-known side effect of this medication [14].

In agreement with our findings, Khalili et al. [15] performed a study including 138 cases with moderate and severe TBI to assess the impacts of DEX on functional outcome of cases. Cases were allocated equally into 2 groups (DEX and control groups). No significant variations in SBP and DBP were detected between DEX and control groups. Similarly, Zhu et al. [16] demonstrated that the variance in HR between the DEX and control groups was not remarkable ($P>0.05$) at 5 minutes pre-anesthesia in supine position. After pneumoperitoneum in Trendelenburg position, the DEX group had decreased HR than the control group ($P < 0.05$). MAP showed no remarkable variance between the groups at any time interval ($P>0.05$). In same line with the present study, Peng et al. [12] found that there was no remarkable variance regarding HR and MAP between dexmedetomidine and midazolam groups before the begin of anesthesia.

Gong et al. [13] reported that no significant variances of monitoring indices, including HR and MAP were found between dexmedetomidine and midazolam groups before induction of anesthesia. However, Schomer et al. [17] reported that no significant hemodynamic alterations were detected in heart rate, blood pressure, or central perfusion pressure when pre- DEX data were compared to DEX data.

A large retrospective case series examined the sedative and analgesic needs, neurological condition, and physiological alterations in 85 cases with severe TBI who received DEX. Despite the median levels of SBP, DBP, MAP, and HR reduced following DEX infusion, the difference was not substantial. Hypotensive episodes occurred in 35% of TBI cases throughout DEX; nevertheless, the number of cases suffering hypotension was not substantially different before, during, and after infusions [18].

In contrast with our findings, Devabhakthuni et al. [19] study evaluated the clinical results of extended infusions with standard- or high-dose DEX to propofol in emergent traumatic cases. They determined that greater doses of DEX could outcome in an elevated risk of hypotension and higher associated analgesic and sedative, which requires further investigation in trauma cases. In the present study, 31.6% of patients have had a GCS of 10/15, 50% of patients presented with a GCS of 9/15, while 18.4% of patients were presented with a GCS of 8/15. There was non-significant variance between groups concerning GCS, pupils, lateralization and reflexes. These findings came in line with Huang et al. [5] who included 115 cases with brain injury and reported no significant variance between MDZ and DEX groups respecting GCS.

In parallel with our results, Peng et al. [12] studied 456 cases with TBI and found no significant variance between MDZ and DEX groups concerning GCS. In the current study, there was non- substantial variation between both groups concerning the Pre sedation CT brain findings based on Rotterdam score. 24 h post sedation was CT brain findings remarkably varied between both groups (P=0.027), was notably better in DEX group compared to MDZ group. In agreement with our findings, Khalili et al. [15] determined the impacts of DEX on functional outcome of cases with severe and moderate TBI. They reported that majority of cases, 42.1% and 31.2% had Rotterdam score of 2 and 3 respectively.

In the current study, right and left ONSD post sedation at (at 6, 12, 18 and 24h) were notably lower in DEX than MDZ group (P<0.05). Zhu et al. [16] showed that there was no substantial variation in ONSD at 5 min before anesthesia in supine position between the dexmedetomidine and control groups, while the ONSD after pneumoperitoneum in trendelenburg position in both groups were higher than before anesthesia induction. The ONSD after pneumoperitoneum in Trendelenburg position in DEX group was obviously less than that in control group (P<0.05). Moreover, Xia et al. [20] demonstrated a study to assess the impact of DEX on ICP in 93 pediatric cases receiving laparoscopic operation by ONSD assessment by ultrasound. Cases were allocated into 2 groups (control group, n=45) and (DEX group, n=48). Patients in the DEX group had increased decline in ONSD than the control group (p<0.01). They concluded that DEX can decrease the progression of elevated ICP and had no impact on the postoperative recovery.

However, Sahay et al. [21] assessed the impact of DEX on ICPs during laparoscopic operations and revealed that cases received DEX revealed a lower elevation in the mean ONSD than normal saline group; however, this was not remarkable (P = 0.075).On the other hand, Khallaf et al [3] evaluated some hemodynamic and ICP alterations in TBI and showed no substantial variations between the groups (propofol, DEX, and mixed groups) concerning the ICP values and central perfusion pressure. In disagreement with our findings, Pajoumand et al. [22] assessed DEX as an adjunct cases with TBI and showed that DEX did not lower the highest ICP in the 1st and 2nd day of the trauma compared to propofol or a DEX and propofol . DEX had its optimum impact on lowering ICP on day 4 post-trauma. In our study, regarding the adverse events, vomiting occurred in 3 (4.41%) cases in group D and 5 (7.35%) cases in group M, hypotension occurred in only 7 (10.29%) cases in group D and not reported in group M. Local anesthetic toxicity was no reported in any of the studied groups, with non-significant variance between groups concerning the adverse events..

Peng et al. [12] found no significant difference regarding vomiting, shivering, hypotension, bradycardia, and headache between dexmedetomidine and midazolam groups. DEX lead to bradycardia (P = .019) and hypotension (P = .033), while, MDZ caused respiratory events (P = .003) as negative outcomes during hospitalization.

CONCLUSION

The present study suggests that dexmedetomidine may be a more effective sedative option for reducing Intracranial pressure in traumatic brain injury patients compared to midazolam. However, the use of dexmedetomidine is correlated with a higher risk of bradycardia, which may require closer monitoring and potential intervention. Both medications were found to cause a decrease in blood pressure, although the magnitude of the effect was not significantly varied between the two groups.

Conflict of Interest or financial disclosure: No potential conflict of interest to be reported by the authors.

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