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Original Article

Anaesthesia Section

Effect of Lignocaine Infiltration versus Intravenous Dexmedetomidine on Haemodynamic Response during Skull Pin Holder Application for Craniotomy: A Prospective Interventional Study

P SHAJLA[1], RASHMI RAVINDRAN[2], KT SHAFNA[3], S MINU[4]

ABSTRACT

Introduction: The use of a skull pin holder during craniotomy is a painful procedure that induces haemodynamic changes. This may lead to raised intracranial pressure, brain bulging, or intracranial haemorrhage. Effectively managing these stress responses is very important, as they may increase morbidity and mortality in neurosurgical patients.

Aim: To compare the effects of lignocaine infiltration and intravenous dexmedetomidine on the haemodynamic stress response to skull pin holder application during craniotomy.

Materials and Methods: This prospective interventional study was conducted on 122 patients over a period from June 2020 to June 2021 in the Neurosurgery operation theatre of Government Medical College, Kozhikode, Kerala, India, a tertiary care teaching centre. Patients were divided into two groups: Group-L received 3 mL of 2% lignocaine with 1 in 200,000 adrenaline at each premarked pin site, while Group-D received intravenous dexmedetomidine at a dose of 0.5 mcg/kg over 10 minutes before the induction of general anaesthesia. Heart Rate (HR), Systolic Blood Pressure (SBP), Diastolic Blood Pressure (DBP) and Mean Arterial Pressure (MAP) were recorded at baseline, after induction, post-intubation, pre-pin application and at intervals of 1, 2, 3, 4, 5, 10 and 15 minutes post pin application. Stata version 14.2 was used for data analysis. Statistical comparison between the groups was conducted using the independent t-test and Chi-square test.

Results: There was no statistically significant difference in HR and blood pressure between the groups immediately after skull pin application. However, Group-D exhibited a significant reduction in HR at 10 and 15 minutes ($p<0.001$) post pin application compared to Group-L. Additionally, a statistically significant reduction in SBP, DBP and MAP was observed among the groups at 5, 10 and 15 minutes post pin application, with a greater reduction in Group-D.

Conclusion: Intravenous dexmedetomidine at a dose of 0.5 mcg/kg was as effective as lignocaine infiltration at the premarked pin site in reducing the haemodynamic stress response to the application of the skull pin holder. Dexmedetomidine at this dosage also showed a greater reduction in HR and blood pressure after five minutes post pin application and did not require any additional pharmacological measures.

Keywords: Blood pressure, Intracranial pressure, Morbidity, Neurosurgery, Skull pin application

INTRODUCTION

Craniotomy is commonly indicated for conditions such as brain tumours, cerebral aneurysms, cerebral abscesses and cerebral arteriovenous malformations [1]. The application of the skull pin holder to secure the head for craniotomy is notably painful, often resulting in abrupt changes in HR and blood pressure. Such haemodynamic changes can precipitate perioperative complications, including increased intracranial tension, brain bulge, intracerebral haemorrhage and cerebral oedema. These changes pose a significant risk to patients with pre-existing cardiac conditions, intracranial aneurysms, or compromised intracranial compliance and autoregulation [2,3].

Given that this is an excruciating procedure, the haemodynamic response during pin application for craniotomy should be managed with appropriate measures. Intravenous fentanyl, alfentanil, sufentanil, ketamine at sub-anaesthetic doses, clonidine, pin site infiltration of local anaesthetic, scalp block with bupivacaine or ropivacaine and gabapentin premedication have all been tried previously to blunt this response, with variable success rates [4-14]. Dexmedetomidine, an alpha-2 agonist, provides stable haemodynamics along with sedative, analgesic and anaesthetic properties, all without causing respiratory depression. It can be administered intravenously in different doses (0.5 mcg/kg, 1 mcg/kg) to attenuate the haemodynamic responses during painful procedures [9,14-16].

As there is a chance of adverse effects with higher doses of dexmedetomidine, especially in neurosurgical patients, the present study aimed to compare the effect of intravenous dexmedetomidine at a lower dose of 0.5 mcg/kg with lignocaine 2% local infiltration at the pin insertion site in reducing the haemodynamic stress response due to pain during skull pin holder application [17,18].

Although there are studies that compare local anaesthetic infiltration and higher doses of intravenous dexmedetomidine (1 mcg/kg) for reducing stress response during craniotomy, there are no studies using a lower dose of dexmedetomidine, such as 0.5 mcg/kg intravenously. Hence, the present study is relevant [6,16,19]. The aim of the present study was to compare the effect of low-dose intravenous dexmedetomidine and lignocaine local infiltration on haemodynamic stress response during skull pin application. The objective of the study was to assess the changes in HR and blood pressure at various time points during skull pin application.

MATERIALS AND METHODS

The present prospective interventional study was conducted in the neurosurgery operating theatre of the Government Medical

College, Kozhikode, Kerala, India, a tertiary care teaching hospital, from June 2020 to June 2021. Patients were enrolled in the study after obtaining approval from the Institutional Research and Ethical Committee (Ref No: GMCKKD/RP2020/IEC/306).

Inclusion and Exclusion criteria: Patients scheduled for elective craniotomy under general anaesthesia, aged between 18 to 65 years, belonging to the American Society of Anaesthesiology Physical Status (ASA PS) 1 or 2, were included in the study. Patients with hypertension, ischaemic cardiac disease, pregnancy, features of raised intracranial pressure, those with traumatic brain injury and those allergic to dexmedetomidine or lignocaine were excluded from the study. A total of 122 patients were assessed for eligibility and after obtaining written informed consent, 61 patients were allocated to each group: Group-D (Dexmedetomidine group) and Group-L (Lignocaine group).

Sample size calculation: The sample size calculation was performed based on comparing MAP between two groups, as referenced in the study by Arshad A et al., [4]. With a 0.05 alpha error and 80% power, each group required 56 patients to achieve statistical significance ($p<0.05$).

$$n=\frac{(Z\alpha+Z\beta)^2 * SD^2 * 2}{d^2}$$

Here, SD=Standard Deviation, which is the mean of the SD of MAP between both groups post-drug administration using the given two types of anaesthetics: 86.13 (±9.73) mmHg and 104.03 (±12.95) mmHg. The variable d represents the effect size (d=6).

Considering potential dropouts and exclusions, a 10% inflation was applied to the sample size, resulting in 61 patients being included in each group.

Study Procedure

All patients were advised to fast for eight hours for solids and two hours for clear fluids. They were given tablet metoclopramide 10 mg and ranitidine 150 mg the night before and at 6 am on the morning of surgery. Electrocardiograms, pulse oximetry and non invasive blood pressure were monitored in the operating room. After premedicating the patients with an injection of midazolam 0.02 mg/kg, morphine 0.01 mg/kg and glycopyrrolate 0.2 mg intravenously, the radial artery was cannulated under local anaesthesia to facilitate invasive blood pressure monitoring.

Patients in Group-D received intravenous dexmedetomidine at a dose of 0.5 mcg/kg slowly over a 10-minute period in 100 mL of normal saline before the induction of anaesthesia [20]. Group-L received 2% lignocaine with 1 in 200,000 adrenaline, 3 mL on each pre-marked pin site as infiltration just before skull pin application.

After preoxygenation, general anaesthesia was induced with thiopentone sodium 5 mg/kg and muscle relaxation was achieved with 0.1 mg/kg of vecuronium intravenously. Oxygen, air and isoflurane were used to maintain anaesthesia following endotracheal intubation. Patients were positioned for craniotomy after securing the head with the skull pin holder. HR and blood pressure (SBP, DBP and MAP) were recorded at the following 11 time points: baseline, after induction, after intubation, pre-pin application and at 1, 2, 3, 4, 5, 10 and 15 minutes post-pin application.

Any adverse effects such as bradycardia and hypotension were monitored. A reduction of more than 20% in MAP from baseline was considered hypotension, while a HR of less than 50 beats per minute was considered bradycardia [21,22].

STATISTICAL ANALYSIS

Statistical analysis was performed using Stata version 14.2. Categorical parameters were presented as frequencies (percentages), while continuous variables were presented as means±SD. Statistical comparisons between groups were conducted using the Chi-square test for categorical variables and the independent t-test for continuous variables.

RESULTS

Demographic parameters such as age, sex and ASA PS were comparable in both groups. The majority of participants were between 50 to 60 years of age (n=41) and belonged to ASA PS 1 (n=88). Out of 122 patients, 60 were male and 62 were females [Table/Fig-1].

Parameters	Group-L (n=61)	Group-D (n=61)	p-value
Age (years)*			
<40	6 (26.1)	17 (73.9)	0.06
41-50	15 (46.8)	17 (53.1)	
51-60	25 (60.9)	16 (39.1)	
>60	15 (56.7)	11 (42.3)	
Sex*			
Male	33 (53.3)	29 (46.7)	0.36
Female	28 (46.8)	32 (53.2)	
ASA PS*†			
1	44 (50.0)	44 (50.0)	0.100
2	17 (50.0)	17 (50.0)	

[Table/Fig-1]: Comparison of demographic parameters between the groups.
*numbers and percentages; †American society of anaesthesiologists physical status

For outcome analysis, the evaluation of HR, SBP, DBP and MAP was conducted at 11 different time points among the groups. The baseline HR was comparable (p=0.51) across the groups. There was no statistically significant difference in HR and blood pressure among the groups immediately after skull pin application. However, a significant reduction in HR was observed after skull pin application at 10 and 15 minutes between the groups, with a greater reduction in Group-D [Table/Fig-2].

Variable Heart Rate (HR)*†	Group-L (mean±SD)	Group-D (mean±SD)	p-value
Baseline	75.1±13.4	76.7±14.2	0.51
After GA induction	78.5±11.9	78.2±13.9	0.90
After intubation	80.4±12.6	83.3±12.6	0.22
Pre-pin application	79.1±11.7	82.7±13.9	0.11
Post-pin application at 1 minute	78.3±10.7	80.7±11.3	0.23
2 minutes	77.1±12.0	78.2±11.2	0.59
3 minutes	75.6±12.1	75.0±11.1	0.78
4 minutes	73.4±10.9	72.1±10.6	0.51
5 minutes	72.5±10.8	69.1±11.1	0.08
10 minutes	71.2±10.7	65.8±10.6	<0.001
15 minutes	70.5±10.2	63.6±10.7	<0.001

[Table/Fig-2]: Comparison of Heart Rate (HR) at various time points between the groups.
*mean±SD; †HR: Heart rate

The SBP, DBP and MAP showed statistically significant reductions between the groups at 5, 10 and 15 minutes post pin application, with a greater reduction in Group-D. Both groups exhibited maximum MAP reduction from baseline at 15 minutes post pin application [Table/Fig-3-5]. In Group-D, four patients experienced bradycardia, while no patients in Group-L had bradycardia. The bradycardia in Group-D was not clinically relevant enough to require any rescue medication. Although hypotensive episodes among the groups (Group-L: n=50, Group-D: n=38) showed statistical significance (p=0.041), all these episodes responded to intravenous fluids and there was no requirement for vasopressors; hence, they were not clinically relevant.

Variable Systolic Blood Pressure (SBP)*†	Lignocaine Group-L (mean±SD)	Dexmedetomidine Group-D (mean±SD)	Unpaired t-test, p-value
Baseline	125.8±15.3	125.8±18.7	0.99
After GA induction	123.6±14.1	126.8±13.7	0.22
After intubation	120.4±16.9	129.2±14.6	0.001
Pre-pin application	117.4±17.7	124.7±14.9	0.01
Post-pin application at 1 minute	113.4±18.2	118.0±13.0	0.11
2 minutes	110.1±17.1	112.8±13.8	0.32
3 minutes	105.8±14.8	107.3±12.0	0.53
4 minutes	103.7±16.2	102.4±10.6	0.59
5 minutes	103.8±15.6	96.9±10.1	0.001
10 minutes	102.3±14.6	94.6±9.9	0.001
15 minutes	99.5±12.5	92.6±10.7	0.001

[Table/Fig-3]: Comparison of Systolic Blood Pressure (SBP) at various time points between the groups.
*mean±SD; †SBP: Systolic blood pressure

Variable DBP*†	Group-L (mean±SD)	Group-D (mean±SD)	p-value
Baseline	80.3±10.5	80.8±11.9	0.74
After GA induction	79.5±10.1	79.5±10.7	0.96
After intubation	77.7±9.4	80.1±11.7	0.22
Pre-pin application	75.5±9.5	76.4±12.1	0.64
Post-pin application at 1 minute	73.4±9.8	72.6±12.0	0.71
2 minutes	70.8±10.1	70.8±9.8	0.99
3 minutes	68.1±9.1	67.7±9.1	0.85
4 minutes	68.6±15.6	64.4±8.3	0.06
5 minutes	65.9±7.9	62.1±6.9	0.001
10 minutes	63.7±8.9	60.0±7.1	0.001
15 minutes	62.0±7.4	58.6±7.5	0.001

[Table/Fig-4]: Comparison of Diastolic Blood Pressure (DBP) at various time points between the groups.
*mean±SD; †DBP: Diastolic blood pressure

Variable MAP*†	Group-L (mean±SD)	Group-D (mean±SD)	p-value
Baseline	95.4±10.5	95.6±12.1	0.88
After GA induction	94.6±9.7	95.6±10.7	0.56
After intubation	92.1±10.5	96.9±10.8	0.01
Pre-pin application	89.6±10.9	93.4±11.5	0.08
Post-pin application at 1 minute	86.7±10.6	88.6±9.1	0.27
2 minutes	84.1±10.7	85.1±9.7	0.62
3 minutes	80.9±9.8	80.7±9.1	0.96
4 minutes	79.5±10.3	77.4±7.6	0.19
5 minutes	78.8±9.3	74.5±7.7	0.001
10 minutes	77.1±9.6	71.6±6.7	0.001
15 minutes	74.6±7.4	70.1±7.2	0.001

[Table/Fig-5]: Comparison of Mean Arterial Pressure (MAP) at various time points between the groups.
*mean±SD; †MAP: Mean arterial pressure

DISCUSSION

The haemodynamic stress response during skull pin application in neurosurgeries increases morbidity and mortality, particularly in patients with underlying medical conditions. Various methods have been tried worldwide to mitigate this. While lignocaine with adrenaline infiltration at the pre-marked pin site is commonly used, it often has drawbacks due to the need for readjustments in the pin application site, errors in infiltration techniques, improper head positioning during application and inadequate dosing. To overcome these drawbacks, other methods, such as scalp block, intravenous infusion of fentanyl, alfentanil, sufentanil, dexmedetomidine, oral clonidine, or gabapentin, have been compared with lignocaine infiltration, resulting in variable outcomes [4-14,23].

The present study compared a lower dose of dexmedetomidine (0.5 mcg/kg) versus 2% lignocaine local infiltration at the pin site to evaluate their effects on haemodynamic responses during skull pin application for craniotomy. Changes in HR and blood pressure were not statistically significant between the groups immediately after skull pin application. The dexmedetomidine group showed a greater reduction in HR, SBP, DBP and MAP at five, 10 and 15 minutes post-pin application compared to the lignocaine group ($p<0.01$). Hypotension in both groups was managed with intravenous fluids and did not require any vasopressors.

Dexmedetomidine, a selective alpha-2 receptor agonist, can be administered intravenously in varying doses (0.5 mcg/kg, 0.75 mcg/kg, 1 mcg/kg) to attenuate the haemodynamic stress response [9,12,14-16]. Higher doses of dexmedetomidine often result in bradycardia and hypotension [17,18].

Paul A and Krishna HM conducted a study comparing the use of intravenous dexmedetomidine, 1 mcg/kg diluted in 10 mL normal saline, given over 10 minutes, to lignocaine 2% infiltration at the skull pin site to reduce the haemodynamic stress response. The above study observed comparable changes in HR and MAP at all the study time points. However, the incidence of bradycardia and/or hypotension was significantly higher among patients receiving dexmedetomidine (n=19) compared to those receiving lignocaine (n=5) [6]. This may be due to the use of a higher dose of dexmedetomidine (1 mcg/kg). The current study used intravenous dexmedetomidine infusion at a lower dose of 0.5 mcg/kg over 10 minutes in 100 mL normal saline to reduce the incidence of bradycardia and hypotension. In the present study, four patients had bradycardia in the dexmedetomidine group, while the incidence was zero in the lignocaine group. Although there were statistically significant episodes of hypotension between the groups, this was improved with intravenous fluids without any medications; hence, it was not clinically relevant.

Kapoor C et al., observed that slow intravenous infusion of dexmedetomidine at 1 mcg/kg over 10 minutes is superior to lignocaine 2% local infiltration without causing bradycardia and hypotension to reduce the stress response. The study also noted that haemodynamics were better controlled with intravenous dexmedetomidine (1 mcg/kg) compared to the lignocaine group at 3, 5, 10 and 15 minutes post pin application [15]. Contrary to the above study, the current study showed that the dexmedetomidine (0.5 mcg/kg) group had a greater reduction in HR, SBP, DBP and MAP at 5, 10 and 15 minutes post-pin application compared to the lignocaine group ($p=0.001$), but the episodes of bradycardia and hypotension did not require any pharmacological measures.

The effectiveness of lignocaine with adrenaline infiltration at the skull pin insertion site was evaluated by Arshad A et al., in 30 craniotomy patients, who concluded that the stress response can be effectively prevented with pin site lignocaine infiltration. They also concluded that haemodynamic benefits were not obtained with lignocaine infiltration beyond 10 minutes post-pin application [4]. The current study observed that the haemodynamics with lignocaine infiltration up to 15 minutes post-pin application were within acceptable limits.

A comparison of intravenous dexmedetomidine bolus (1 mcg/kg) followed by infusion (1 mcg/kg/hour) versus scalp block using 0.5% ropivacaine was conducted by Singh G et al., to reduce haemodynamic effects during skull pin holder application. The present study concluded that the scalp block using 0.5% ropivacaine is not only effective but also superior to the dexmedetomidine infusion [24]. At the same time, observations from the present study indicate that both intravenous dexmedetomidine and lignocaine

local infiltration are equally efficacious in reducing the stress response during skull pin application.

Kondavagilu SR et al., evaluated the effectiveness of two doses of intravenous dexmedetomidine (0.5 mcg/kg versus 1 mcg/kg) along with bupivacaine 0.25% infiltration at the pin site to mitigate the haemodynamic response to head pin application. They observed that the low-dose dexmedetomidine (0.5 mcg/kg) was as effective as the 1 mcg/kg dose for controlling HR and MAP responses to pin application [14]. The present study also found that low-dose intravenous dexmedetomidine (0.5 mcg/kg) was effective in attenuating the haemodynamic response during skull pin insertion.

Bala R et al., compared intravenous dexmedetomidine at 0.75 mcg/kg given over 10 minutes after intubation versus pin site infiltration with ropivacaine 0.5% to reduce the stress response during pin application. The study concluded that both modalities are equally efficacious, but there was clinically and statistically significant hypotension requiring treatment in the dexmedetomidine group (31.8%) [19]. The present study showed that the stress response can be reduced without causing clinically relevant adverse effects with the use of a lower dexmedetomidine dose.

The present study included an adequate sample size and compared a lower dose of intravenous dexmedetomidine with lignocaine infiltration, which adds to the strength of the study. Since intravenous dexmedetomidine is efficacious in maintaining haemodynamics at 0.5 mcg/kg during skull pin holder application, further research on much lower doses can be conducted to achieve improved outcomes in the future. The combination of lower doses of dexmedetomidine with other modalities, such as scalp block or local anaesthetic infiltration, may also result in better control of haemodynamics without causing adverse effects.

Limitation(s)

The present study has selected patients for craniotomy for various indications; therefore, the differences in neurological pathology may have affected the haemodynamics. It would be better if patients with similar indications were selected for the study.

CONCLUSION(S)

Administering a low dose of dexmedetomidine (0.5 micrograms per kilogram) intravenously is as effective as local infiltration of lignocaine with adrenaline in minimising the haemodynamic response during the application of a skull pin holder for craniotomy. However, dexmedetomidine significantly reduced HR and blood pressure five minutes after pin application at this dosage, without causing clinically significant adverse effects. In comparison, lignocaine infiltration demonstrated more stable haemodynamics during this period.

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PARTICULARS OF CONTRIBUTORS:
1. Senior Resident, Department of Anaesthesia, Government Medical College, Kozhikode, Kerala, India.
2. Professor, Department of Anaesthesia, Government Medical College, Kozhikode, Kerala, India.
3. Assistant Professor, Department of Anaesthesia, Government Medical College, Kozhikode, Kerala, India.
4. Assistant Professor, Department of Anaesthesia, Government Medical College, Kozhikode, Kerala, India.

NAME, ADDRESS, E-MAIL ID OF THE CORRESPONDING AUTHOR:
Dr. KT Shafna,
Assistant Professor, Department of Anaesthesia, Government Medical College, Kozhikode-673008, Kerala, India.
E-mail: shafnakt87@gmail.com

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