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"COMPARISON OF HEMODYNAMIC RESPONSE OF DEXMEDETOMIDINE VS PROPOFOL IN CASE OF AWAKE FIBREOPTIC INTUBATION : A RANDOMIZED INTERVENTIONAL STUDY"

Anaesthesiology

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ABSTRACT

Background: Providing adequate anxiolysis and sedation with a patent airway while performing fiberoptic bronchoscopic intubation is a challenging task to an anaesthetist. Ideal sedation would ensure calm and cooperative patient maintaining spontaneous ventilation. Dexmedetomidine is such a α2a adrenergic agonist with sympatholytic, analgesic, and sedative properties. Though its role is very well documented for sedation, proving it better than propofol for the procedure is being considered in this study. **Methods:** In total of 60 patients, after nebulizing with 5 ml of 4% lignocaine over 10 minutes, 30 patients were infused with dexmedetomidine @1µg/kg over 10 minutes followed by 0.3µg/kg and rest with propofol @1mg/kg. Fiber-optic bronchoscopy was done after 10 minutes of infusion. Monitoring was done considering heart rate, blood pressure, Ramsay sedation score and patient tolerance. **Results:** had shown successful intubation in both cases but dexmedetomidine had a better outcome with respect to sympathetic response and patient tolerance. P value was significant for sedation score, pre and post bronchoscopic intubation sympathetic response. No episodes of airway obstruction and hypoxia were noted with dexmedetomidine as compared with propofol. Mean Ramsay sedation score was 3.77 as compared to 2.33 with propofol. **Conclusion:** In our comparative study, Dexmedetomidine had offered better patient tolerance with adequate sedation and preservation of airway as compared to propofol and a reduced hemodynamic response to intubation.

KEYWORDS

Bronchoscopic Intubation, Dexmedetomidine, Difficult Airway, Propofol, Sedation Score.

INTRODUCTION

Fiberoptic bronchoscopic intubation is a gold standard for both anticipated and unanticipated difficult airway such as in unstable cervical spine injury, severe cervical stenosis, limited mouth opening, mandibular-maxillary fixation and severe facial burns. One of the challenges associated with this procedure is providing adequate anxiolytics and sedation while maintaining a patent airway, spontaneous ventilation and adequate oxygenation.

Propofol is widely used in anaesthetic practice to facilitate tracheal intubation. Dexmedetomidine is a selective alpha-2a adrenergic agonist with sympatholytic, analgesic, and sedative properties. By promoting natural sleep pathways, it creates a conscious, sedated patient who is arousable and has minimal respiratory depression.

MATERIALS AND METHODS

Study Location: The study will be conducted in the Department of Anaesthesiology (Plastic surgery O.T, TCOT, Oncosurgery OT), S.M.S. Medical College and Attached Group of Hospitals, Jaipur.

Study Design: Hospital based Randomized interventional study.

Study Period: After approval of research review board, from June 2019 till the sample size will be achieved.

Sample Size: The Sample size was calculated 71 subjects for each for the groups at 95% confidence interval and 80 % power of study to verify the expected significant fall in heart rate in Dexmedetomidine as compared to patients sedated with Propofol (19.369% versus 3.1728%), at the end of 10 minutes of infusion study groups (as per seed articl). [13] This sample size is adequate to cover all other study variables too.

Sampling Technique:

Randomization: It is a statistical procedure by which the participants will be allocated into 2 groups. In this study randomization will be done by computerized random number.

Study Universe: Cases undergoing surgery under general anaesthesia(Duration 1-2 hr).

Study Groups: The study will be conducted in following two groups of patients. Each group will consist of 71 patients.

Group A: Patients will receive I.V. dexmedetomidine (1 µg/kg) over 10 minutes.

Group B : Patients will receive I.V. propofol (1mg/kg) over 3 minutes followed by infusion @50 µg/kg/minute.

PROCEDURE

All patients will undergo preanaesthetic check up on the day before surgery. Informed written consent will be taken from each patient after providing complete explanation about the procedure and the study protocol.

After confirmation of identity, patient will be taken in operation theatre.PAC,fasting status, and informed written consentwill be checked. A peripheral I.V. line will be se-cured with 18 G/20 G cannula. Inj. Ringer Lactate 5 ml/kg will be started.Inj. ranitidine 50 mg I.V., inj. Metoclopramide 10 mg I.V.andInj. Glycopyrrolate (0.005 mg/kg I.V.) will be given as a premedication.

Nebulization will be done with 2% lidocaine 4 ml (80 mg) for topicalization. Xylometazoline nasal drops and lidocaine jelly will be applied to both the nostrils.

Baseline parameters such as Heart Rate (HR), Systolic blood pressure (SBP), Diastolic blood pressure (DBP), Mean Arterial Pressure (MAP), Rate pressure product and SPO2 will be recorded.

After that, by computerized random number table will be allocated to either of the 2 groups.

Group 1 patients will receive inj. Dexmedetomidine (1 µg/kg) I.V. over 10 minutes.

Group 2 patients will receive I.V. propofol(1mg/kg) over 3 minutes followed by infusion @50 µg/kg/minute.

Bronchoscope will be loaded with appropriate size cuffed endotracheal tube after proper lubrication.

Sedation will be assessed by Ramsay sedation score (RSS). When RSS will be achieved ≥2 the fibreoptic bronchoscopy will be performed. Oxygenation will be done via the nasal catheter with 100 % oxygen at the rate of 4 ltr/mnt. throughout the fibreoptic bronchoscopy.

Heart rate, Systolic blood pressure, Diastolic blood pressure, Mean B.P., Rate pressure product and SpO2 will be recorded at every 5 minutes interval during process of study drug infusion, pre and post intubation.

Assessment of cough score will be done to evaluate the intubation conditions.

Patient Tolerance will be assessed by a 5-point fibreoptic intubation comfort score during the placement of endotracheal tube in trachea.

General anaesthesia willbe induced with Inj. Thipentone sodium 5mg/kg I.V. slowly over 1 minute, followed by inj. Atracurium 0.5 mg/kg I.V. and surgery will be allowed to proceed.

Parameters (Heart rate, Systolic blood pressure, Diastolic blood pressure, Mean B.P., Rate pressure product and SpO2)will be recorded post intubation.

Anaesthesia will be maintained with 40 % O2+ 60 % N2O, Inj. Atracurium 0.1 mg/kg I.V., and Sevoflurane 1-2 MAC.

At the end of the surgery neuromuscular blockade reversal will be done with Inj. Neostigmine (0.05 mg/kg) and Inj. Glycopyrrolate (0.01 mg/kg). Patient will be extubated.

Any side effect will be noted.

RESULTS

A total of 142 patients were enrolled into the study. No statistically significant differences were found with respect to mean age, weight and gender between dexmedetomidine and propofol groups in our study (p>0.05) and thus the results were comparable.

Both the groups underwent successful fiberoptic intubation with good patient cooperation although; dexmedetomidine group had more favourable intubation scores as compared to propofol group. This was analyzed by scoring three parameters while performing bronchoscopy i.e. vocal cord movement (VCM), coughing and patient tolerance [Table 1]. 83.3% of patients in dex group had VCM score of one as opposed to propofol group where only 23.3% patients had a score of one. None of the patients in dex group showed closed vocal cords while performing bronchoscopy as compared to 23.3% patient in propofol group. This was highly significant result (p = 0.000) and has important implication for successful intubation. Similarly, coughing and patient tolerance score were more favourable for dex group. P value of 0.002 for coughing indicates absence of cough in 60% of dex group patients as opposed to 16.7% in propofol group. Patient tolerance score were also highly significant (p=0.000) as none of dex group patients showed any defensive movement of head or hands on contrary to propofol group where two of thirty patients have shown severe movement during the procedure.

MONITORING

GROUP A (DEXMEDETOMIDINE)

Parameters / Time	Heart rate	Systolic B.P.	Diastolic B.P.	Mean B.P.	SPO2
Base line	104	140	90	106.6	100
At 5 minutes	103	135	75	95	99
At 10 minutes	100	120	80	93.3	99
At Fiberoptic bronchoscope insertion	95	125	85	97	100
At Immediate post intubation	100	130	80	96	99

Raamsay Sedation Score (RSS)	5
Cough Score	1
Patient tolerance	2
Side effect (if any)	**Nil**

GROUP B (PROPOFOL)

Parameters / Time	Heart rate	Systolic B.P.	Diastolic B.P.	Mean B.P.	SPO2
Base line	112	140	90	106	100
At 5 minutes	110	130	80	96	99
At 10 minutes	106	120	90	93	99
At Fiberoptic bronchoscope insertion	100	110	76	87	100
At Immediate post intubation	115	140	90	106	99

Raamsay Sedation Score (RSS)	3
Cough Score	2
Patient tolerance	2
Side effect (if any)	Nil

Sedation scores as analysed by Man-Whitney test and Wilcoxon test for Ramsay Sedation Score showed p value of 0.0001 at pre and post intubation time. Heart rate and arterial pressure at three time points {i.e. baseline (0 minutes), pre (10 minutes) and post intubation} are shown in figures. Baseline heart rate 84 versus 83 beats/min, [Figure 1] did not differ significantly between the two groups, patients sedated with dexmedetomidine had significant fall in heart rate compared to patients sedated with propofol (19.369% versus 3.1728%), at the end of 10 minutes of infusion though there were no episodes of severe bradycardia (< 40 beats/min). Also, increase in mean heart rate of 5 and 18 beats/min was noted following bronchoscopy and intubation in dexmedetomidine and the propofol group respectively with a p value of 0.0001.

The variation in systolic, diastolic and mean arterial pressure did not differ significantly between the two groups [Figure 2&3] from baseline till preintubation (p=0.354, 0.899 and 0.524 for SBP, DBP and MAP respectively before intubation).But soon after the intubation, a significant increase in blood pressure was observed in propofol group (p=0.001 for SBP, DBP and MAP) as compared to dexmedetomidine infused patients.

DISCUSSION

The primary outcome of the study showed that dexmedetomidine provides a consistent and deeper level of sedation along with hemodynamic stability. One of the objectives of this study was to explore the possibility of a drug for better preservation of respiratory function. During management of difficult airway, it is safe to keep patient breathing spontaneously until an alternative airway is established. Dexmedetomidine activates the postsynaptic α2-adrenergic receptors in the locus cerulean, and induces sedation by activation of the endogenous sleep-promoting pathway. Moreover, it has sedative, analgesic, anxiolytic, and anti-sialagogue properties without predisposing to airway obstruction and respiratory depression. As it explains, dexmedetomidine has not been associated with any episode of airway obstruction, hypoxia or respiratory depression despite profound levels of sedation (sedation score of 4) in our study. Abdelmalak et al also reports a series of successful fiberoptic intubations using dexmedetomidine for sedation in patients with difficult airways caused by subglottic mass and a thyroid tumour without respiratory compromise. On the contrary, anecdotal reports describe incidents of respiratory depression (defined as a decrease in respiratory rate more than 25% or a decrease in oxygen saturation <90%) during infusions of propofol for sedation. Tsai et al has also reported episodes of airway obstruction and respiratory depression with propofol as compared to dexmedetomidine. In our study, two of thirty patients in the propofol group have shown transient hypoxia, with the lowest recorded oxygen saturation 88% (baseline 97%). Face-mask ventilation with 100% oxygen rapidly resolved the situation. Their data were analysed on an 'intention to treat' basis. There was no episodes of airway obstruction or hypoxia in the dexmedetomidine group.

Reduction in heart rate is expected with dexmedetomidine, a α2 adrenoceptor agonist. It may be due to two pathways: a vagomimetic effect and blockade of cardio-accelerator nerve. In a study by Tsai et al, significant decline in heart rate was observed with dexmedetomidine whereas same was not seen with the propofol. Other studies in healthy volunteers and in ICU patients, also showed a significant reduction in heart rate when compared to placebo. Our study has shown similar results.

Initial bolus dose of propofol has been associated with the occurrence of significant hypotension, bradycardia and respiratory depression. Previous work has demonstrated a powerful inhibitory effect of propofol on sympathetic outflow. Dexmedetomidine is also known to decrease sympathetic outflow and circulating catecholamine levels and would therefore be expected to cause decreases of blood pressure similar to those of propofol. However, larger doses of dexmedetomidine have a direct effect at the postsynaptic vascular smooth muscle to cause vasoconstriction, and it is possible that the sympatho-inhibitory effects of dexmedetomidine were slightly opposed by direct α_2-mediated vasoconstriction. Both the drugs

(dexmedetomidine and propofol) have shown consistent decrease in blood pressure (systolic, diastolic and mean arterial pressure) from baseline to intubation, but soon after the intubation, a significant increase in blood pressure was observed in propofol group whereas it was well controlled and stabilized in dexmedetomidine infused patients. This significant difference has emphasized the importance of dexmedetomidine in maintaining hemodynamic stability while performing fiberoptic intubation. Tsai et al also supports that dexmedetomidine has a favourable response than propofol with respect to hemodynamic stability.

R. M. Venn and R. M. Grounds have used RSS and BIS as a guidance of sedation for comparing dexmedetomidine and propofol for sedation in the ICU. They have found that RSS-guided sedation seems to be suitable and carries a good correlation with BIS. Their study shows dexmedetomidine as a safe and acceptable sedative agent for those requiring intensive care management. In our study, we have used Ramsay sedation score for assessing the level of sedation. Patients receiving dexmedetomidine were able to achieve a mean Ramsay sedation score (just before intubation) of 3.77 as compared to sedation score of 2.33 achieved in patients receiving propofol. This difference is highly significant ($p=0.001$) and thus implies the effectiveness of dexmedetomidine in providing adequate sedation for fiberoptic bronchoscopic intubation.

Dexmedetomidine infusion may also cause adverse effects such as atrial fibrillation, and hypoxia. Herr et al have reported ventricular tachycardia in 5% cases in propofol group but our study does not report any such observations. There were no incidents of ventricular tachycardia or atrial arrhythmia in both the groups in our study. None of the patients receiving dexmedetomidine required vasopressor or atropine. On the contrary, four patients in propofol group had bradycardia and hypotension requiring active management. Both symptoms were easily managed with atropine, ephedrine and intravenous fluid administration.

Both the groups underwent successful fiberoptic intubation with good patient cooperation although, dexmedetomidine group had more favourable intubation scores as compared to propofol group. Once tracheal intubation was complete and the naso-tracheal tube was secured, standard general anaesthesia was administrated. This result clearly shows smooth passing of bronchoscope into trachea with dexmedetomidine, as patient tolerance is better causing less sympathetic response and stable vitals. These findings clearly justify the use of dexmedetomidine over propofol for sedation in AFOI.

CONCLUSION

In our comparative study, sedation for fiberoptic bronchoscopic intubation was provided with dexmedetomidine and propofol. Dexmedetomidine offered better patient tolerance with adequate sedation, preservation of airway with spontaneous ventilation and a reduced hemodynamic response to intubation. These properties make it a drug of choice for providing sedation while performing fiberoptic bronchoscopic intubation.

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