Research Article

A comparative study of nebulized versus intravenous lignocaine to suppress the haemodynamic response to endotracheal suction in patients on mechanical ventilation

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ABSTRACT

Background: Cardiovascular response to tracheal suction is decreased by intravenous lignocaine. Tracheal suction is a powerful stimulus that causes intense haemodynamic changes in patients on mechanical ventilation. In the present study, we compared the effect of nebulized and intravenous lignocaine on haemodynamic response to tracheal suctioning in patients on mechanical ventilation through an endotracheal tube.

Methods: A prospective randomized cross over study was conducted in Lokmanya Tilak Municipal Medical College and Government Hospital, Sion, Mumbai, India during the period of January 2012 to September 2013. Sixty patients requiring tracheal suction through an endotracheal tube received 1.5 mg/kg of lignocaine in the nebulized form or as an intravenous injection on two different occasions. Heart rate (HR), mean arterial pressure (MAP), systolic and diastolic blood pressure (SBP and DBP) and peripheral capillary oxygen saturation (SPO2) were recorded at baseline, after the administration of lignocaine, after two successive suctions and once in two minutes for the next 16 minutes.

Results: In the present study, SPO2 decreased in response to ETT suctioning in both the study groups as compared to the pre-suctioning value. However the changes in the SPO2 were not significant when compared between the groups. Changes in HR, SBP, DBP and MAP were not significantly different between the two routes of lignocaine administration.

Conclusions: From the present study, we can conclude that the abolition of haemodynamic response to tracheal suction is similar with both intravenous and nebulized lignocaine. But the return of MAP towards baseline value was observed to be earlier with nebulized lignocaine than with intravenous lignocaine which favours use of nebulized lignocaine over intravenous lignocaine. With built-in nebulizer facility in the current intensive care ventilators, this technique should be easy, more effective and assure better haemodynamic stability than intravenous lignocaine during tracheal suction.

Keywords: Tracheal suction, Lignocaine, Endotracheal tube, HR, MAP, SPO2, Nebulizer

INTRODUCTION

Tracheal suction is a powerful stimulus that causes intense haemodynamic changes in patients on mechanical ventilation. Traditionally, intravenous lignocaine has been used to control the haemodynamic response to tracheobronchial stimulation. While this technique is generally considered safe, in critically ill patients on mechanical ventilation requiring tracheal suctioning, there are potential risks with intravenous lignocaine. It may cause hypotension. In patients with low cardiac output, transient high plasma concentration of lignocaine with associated systemic toxicity may occur. At the same time, there is evidence to show that intravenous
Nebulized lignocaine has been used in clinical practice for a variety of indications. It has been tried in patients with bronchial asthma to decrease the airway reactivity. Awake fiberoptic intubation has been achieved by combining nebulized lignocaine with other lignocaine supplements to suppress the airway reflexes. The efficacy of nebulized lignocaine on cardiovascular response to tracheal suction has not been investigated.

In the present study, we compared the effect of nebulized or intravenous lignocaine on haemodynamic response to tracheal suctioning in patients on mechanical ventilation through an endotracheal tube.

**METHODS**

A prospective randomized cross over study, from January 2012 to September 2013 was conducted, sixty haemodynamically stable patients who were on mechanical ventilation in the Trauma Critical Care (Eward) of Lokmanya Tilak Municipal Medical College and General Hospital, Sion, Mumbai, India were enrolled in the study. Approval by the Institutional Medical Ethics Committee and written informed consent was obtained from the every patients family included in the study.

**Inclusion criteria**

- Age between 18 to 70 years.
- Either sex.
- Haemodynamically stable patients.
- Patient not receiving any vasoactive drugs.
- Patient on mechanical ventilation with endotracheal intubation.

**Exclusion criteria**

- Unwilling for consent
- Age <18years, and >70years.
- Patient who have already received lignocaine by any other route.
- Hypersensitivity to lignocaine.
- History of convulsions.
- Patient receiving vasoactive drug support.
- Patient on mechanical ventilation with tracheostomy tube.

**Study protocol**

**Nebulized lignocaine**

Baseline heart rate (HR), mean arterial pressure (MAP), peripheral capillary oxygen saturation (SPO2>90%) were recorded initially. Nebulized lignocaine (4% solution) 1.5 mg/kg body weight diluted to 4 mL was administered by using a jet nebulizer connected to the ventilator, which had a facility for nebulization. The nebulizer was connected to the inspiratory limb of ventilator circuit close to the Y-piece and the drug was delivered only during the inspiratory phase of respiration. HR, SPO2, SBP, DBP and MAP were recorded at the end of nebulization following which two tracheal suctions were carried out at an interval of two minute by introducing a 16 gauze polyvinyl chloride (PVC) catheter up to the carina.

Each suction was carried out for 15 sec. Following the first suction, the patients were connected to the ventilator until 2 minute time has elapsed and then a second suction was done. HR, SPO2, SBP, DBP and MAP were recorded at one minute and after each suction and at 2,4,6,8,10,12,14 and 16 minute intervals afterwards.

**Intravenous lignocaine**

The protocol was similar to the nebulized lignocaine protocol but for the study intervention, after recording the baseline HR, SPO2 >90%, and MAP the patients were administered 1.5 mg/kg of 2% intravenous lignocaine over a period of 60 seconds. HR, SPO2, SBP, DBP and MAP were noted at 2 minute after lignocaine injection. The rest of the suction procedures and data collection were similar between the two protocols.

**Side effects**

Complications if any with calculated dosages of Intravenous administration of lignocaine are mild and well known, which include symptoms such as numbness of the tongue and mouth, light-headedness, tinnitus. The patient was observed during the study and managed accordingly.

**Statistical analysis**

Data entry was done in Excel. Data analysis was done with the help of SPSS Software version 15, Statistical version and Sigma plot version 11. Quantitative data was presented with the help of mean, standard deviation, comparison between study groups was done with the help of Unpaired T test and Intra group comparison was done with the help of paired T test. Qualitative data was presented with the help of Frequency and Percentage table, association among study group was assessed with the help of Chi-Square test. P value less than 0.05 was taken as significant level.

**RESULTS**

For this study, 60 patients were selected and each patient was a part of both the study groups.

**Group “N”** - Nebulized lignocaine (4% solution) 1.5mg/kg diluted to 4ml will be administered using a nebulizer connected to the ventilator.
Group “I” - Intravenous lignocaine (2% solution) 1.5mg/kg will be administered over a period of 60 seconds.

Figure 1 shows comparison of mean heart rate among study groups from baseline up to 16 minutes of observation. There is no statistically significant difference found in the baseline heart rate between two groups. The Intergroup P value is found to be statistically not significant (P >0.05) at any level.

Figure 3 shows comparison of mean of DBP (mmHg) among study groups from baseline up to 16 minutes of observation. There is no statistically significant difference found in the baseline DBP between two groups. The Intergroup P value is found to be statistically not significant (P>0.05) at any level.

DISCUSSION

Tracheal suction is a potent stimulus that causes cough and haemodynamic response in intubated intensive care patients. In addition, it may also cause bronchoconstriction in any patient with increased airway reactivity. Intravenous lignocaine has been used to
suppress cough during tracheal intubation, laryngospasm and cough during extubation and airway reflexes elicited by the irritation of tracheal mucosa. It has also been used to suppress airway hyper reactivity and mitigate bronchoconstriction after tracheal intubation.

Increase in the heart rate is a part of hemodynamic response to tracheal suctioning. In the present study Heart rate increased in response to ETT suctioning in both the study groups as compared to the pre-suctioning value, the p values were not significant (p>0.05). There were however significant differences within the group. Prasad JR et al who compared nebulized versus intravenous lignocaine to suppress the haemodynamic response to endotracheal suction in patients on mechanical ventilation had similar findings. In their study the HR changes were not significantly different between the nebulized lignocaine and intravenous lignocaine group but there were significant within group differences.

In the present study, both systolic and diastolic blood pressure increased in response to ETT suctioning in both the study groups as compared to the pre-suctioning value which signifies the intensity of the stimulus. The difference was not significant in between the groups. However the intra group changes were significant in both the study groups.

In the present study mean arterial pressure (MAP) increased in response to ETT suctioning in both the study groups as compared to the pre-suctioning value which signifies the intensity of the stimulus. The difference was not significant in between the groups. However the intra group changes were significant in both the study groups. Prasad JR et al who compared nebulized versus intravenous lignocaine to suppress the haemodynamic response to endotracheal suction in patients on mechanical ventilation. In their study the Significant within group differences of MAP were found. But the changes were not significantly different between the groups.

In the present study, SPO2 decreased in response to ETT suctioning in both the study groups as compared to the pre-suctioning value. However the changes in the SPO2 were not significant when compared between the groups.

Though use of intravenous lignocaine to suppress the airway reflexes caused by tracheal irritation has been an accepted procedure, an effective suppression may actually require a very high plasma lignocaine concentrations bordering on to toxic levels. In humans anaesthetized with enflurane, airway irritation elicited cough, and other respiratory reflexes such as expiration, apnoea and spasmodic panting. After administration of intravenous lignocaine, plasma concentrations of lignocaine exceeded 4.7ug/ml, for abolition of all the responses except brief apnoea. The apnoeic reflex was not eliminated even at plasma lignocaine concentrations greater than 7.0ug/ml. In a volunteer study of abolition of histamine-induced bronchospasm also, the effective lignocaine plasma concentration required to decrease bronchoconstriction, ranged at low antiarrhythmic concentrations, but caused mild central nervous system side effects in about a third of the volunteer’s tested. In another study comparing intravenous with inhaled lidocaine, both the techniques attenuated reflex bronchoconstriction significantly. But lignocaine plasma concentrations were significantly lower after inhalation.

High plasma concentrations of lignocaine are fraught with certain potential complications, which include central nervous system symptoms such as numbness of the tongue and mouth, light headedness, tinnitus, visual disturbances, slurring of speech, muscular twitching, irrational conversation, unconsciousness, grand mal convulsion, coma and apnoea, cardiovascular symptoms such as hypotension and myocardial depression. The incidence of such toxicity is low in normal individuals. Critically ill patients however, have certain risk factors such as hypovolemia and acidosis that may enhance the likelihood of increased plasma lignocaine concentration. In addition, rapid injection or inadvertent arterial injection also may be associated with systemic toxicity. In contrast, nebulized lignocaine used to provide surface anaesthesia might produce the required suppression of the response to the tracheobronchial stimulation at lower plasma concentration. This has been observed in many studies.

Most of the studies found no significant difference between the efficacy of Nebulized Lignocaine in suppressing the haemodynamic responses to that of the other regional techniques used for suppression of airway reflexes during airway interventions. Thus, Nebulized lignocaine seems to be clinically effective at plasma concentrations that are below toxic threshold and it can be a safer alternative to Intravenous Lignocaine.

The results of the present study indicate that the haemodynamic stimulation caused by tracheal suction can be effectively suppressed by both nebulized and intravenous lignocaine. While this seems to suggest that both these interventions may be used with equal efficacy, we find at least two reasons to prefer nebulization to intravenous administration. For a given dose of lignocaine, the plasma concentration will be lower with nebulization as inferred from the previous studies. Secondly, the return of MAP towards baseline value was observed to be earlier with Nebulized Lignocaine than with Intravenous Lignocaine which favors use of Nebulized Lignocaine over Intravenous Lignocaine.

We used a cross-over design in this study with each patient acting as his own control. This model decreases the influence of other confounding factors that might have affected the results if the study was carried out in two different groups of patients. The two interventions were carried out within less than 24 hours to avoid any
gross changes in the clinical condition of the patients between the two studies. Also, we ensured that patients were haemodynamically stable before the study. Considering the short duration of action of lignocaine, there is little chance for the carry-over effect.

The effect of the two study interventions is similar in the present investigation. Given the earlier evidence supporting suppression of haemodynamic response to airway stimulation by lignocaine, we may infer that the response would have been more intense without these interventions. Lack of difference between the two modes of administration of lignocaine suggests that nebulization may conveniently replace the intravenous route.

CONCLUSION

The present study shows that the abolition of haemodynamic response to tracheal suction is similar with both intravenous and nebulized lignocaine. But the return of MAP towards baseline value was observed to be earlier with Nebulized Lignocaine than with Intravenous Lignocaine which favors use of Nebulized Lignocaine over Intravenous Lignocaine. With built-in nebulizer facility in the current intensive care ventilators, this technique should be easy, more effective and assure better haemodynamic stability than intravenous lignocaine during tracheal suction. Nebulization may also help to loosen the secretions and facilitate better clearance of secretions.

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REFERENCES
