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"EVALUATION OF INTRAVENOUS LIGNOCAINE AS A COMPONENT OF BALANCED ANAESTHESIA FOR SPINE SURGERY."

Anaesthesiology

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ABSTRACT

Background: A major spine surgery represents a major trauma. Inflammatory mediators released from damaged tissues initiate cascade reactions leading to a systemic inflammatory response syndrome, organ dysfunction and pain.[2] In our institute we routinely use systemic lignocaine in major surgery during perioperative period, as it has analgesic, anti -inflammatory, antinociceptive and immunomodulating properties.[1] So, we planned to study the effects of systemic lignocaine as a component of balanced anaesthesia in major spine surgeries. **Method:** All patients were divided in 2 groups: **Group L:** Received Inj. Lignocaine as a bolus of 1.5mg/kg just before induction followed by infusion at 2 mg/kg /hr up to last skin suture. **Group C:** Includes patients who did not receive intravenous lignocaine infusion. All patients were induced with Inj. fentanyl 2mcg/kg, Inj. Propofol 1.5 - 2 mg/kg and Inj. Vecuronium 0.12 mg/kg and maintained with O2, N2O and inhalational sevoflurane to keep BIS between 40-60. Hypertension and tachycardia were treated with intermittent bolus of Inj. fentanyl 1 µg/kg IV. **Results:** we observed that sevoflurane requirement in group L was statistically significantly lower as compared to group C ($p < 0.05$). In group L, mean duration of post operative analgesia was 202.4±150.48 minutes and 90.68±47.52 minutes in group C. ($P < 0.05$). The mean time to achieve Aldrete score ≥9 after extubation in group L was 1.92±0.90 minutes as compared to 2.24±1.47 minutes in group C. Intraoperative requirement of fentanyl bolus dose was same in both groups. **Conclusion:** Perioperative systemic lignocaine significantly reduces sevoflurane requirement and increases duration of post operative analgesia with no side effects during major spine surgeries.

KEYWORDS

Lignocaine, Sevoflurane, BIS (Bi -Spectral Index), Major spine surgery.

INTRODUCTION:

Lignocaine is an amide type local anaesthetic agent, widely used as a local anaesthetic agent and for the treatment of life threatening ventricular arrythmias.

During perioperative period IV lignocaine in a bolus dose is used to reduce sympathetic response to laryngoscopy, alleviate pain of propofol injection and to prevent postoperative airway complications.[1]

Intravenous lignocaine infusion causes a significant reduction in release of inflammatory mediators which has implications in not only the return of bowel function but also thrombosis, post-operative myocardial infarction and sepsis.[1]

Systemic administration of lidocaine tends to decrease interleukin-1 (IL-1), tumor necrosis factor-alpha (TNF-α), intercellular adhesion molecule-1 (ICAM-1), mucosal COX-2 (cyclooxygenase-2), and plasma prostaglandin E2. In vitro studies have demonstrated the modulatory effect of lignocaine on potassium channels, calcium channels, G-coupled protein receptors, N-methyl-D-aspartate (NMDA) receptors and the glycinergic system. Such mechanisms are thought to contribute to the anti-neuroinflammatory effects of lignocaine and may explain its clinical benefits in the management of acute and chronic pain.[1]

In our institute, no study has been done to evaluate the effect of IV lignocaine on requirement of inhalational agent. So, we planned this study to assess the effect of intravenous administration of lignocaine with primary objectives of its effect on sevoflurane requirement and on hemodynamic parameters. Secondary objectives of our study were to assess effects of intravenous lignocaine on recovery profile and post-operative analgesia in patients undergoing spine surgery.[4][5]

MATERIAL AND METHOD: -

- After obtaining institutional ethical committee approval, this prospective observational study was conducted on 50 adult patients with 25 in each group, undergoing major spine surgeries.
- We used convenient purposive sampling technique to calculate our sample size.
- Patients scheduled for Spine Surgery under ASA class I, II & III belonging to 18-65 years of age were included. We excluded Patients who refused to participate in study, Allergy to lignocaine, Patient having Liver disease or renal impairment, Epilepsy, Arrhythmia, long QT syndrome, BMI (body mass index) > 30.
- All the adult patients scheduled for spine surgery during study period were screened for eligibility during PAC.
- All patients received premedication half an hour before surgery with Inj. Glycopyrrolate 0.004 mg/kg IV, Inj. Tramadol 1.5 mg/kg IV and Inj. ondansetron 4 mg IV.
- Patients were preoxygenated with 100% oxygen for 3 minutes. Induction was done with Inj. fentanyl 2mcg/kg IV, Inj. Propofol 1.5-2 mg/kg IV, Inj. Vecuronium 0.12 mg/kg IV. Patients who received Inj. lignocaine 1.5mg/kg/hr IV followed by 2 mg/kg/hr up to completion of surgery as per departmental protocol formed group L while patients who did not receive lignocaine formed group C. Intermittent bolus dose of fentanyl 1 µg/kg was used to maintain hemodynamic parameters within 20 % of baseline.
- Anaesthesia was maintained with intermittent Vecuronium, Sevoflurane with an aim to keep BIS between 40 to 60. During intraoperative period pulse rate, systolic blood pressure, diastolic blood pressure and sevoflurane dial concentration were recorded. BIS value was kept between 40-60 to prevent intraoperative awareness.
- At the end of the surgery, Sevoflurane & Lignocaine were stopped at the last stich & patients were reversed with injection Neostigmine & Glycopyrrolate.
- To compare recovery profile, the time to achieve Alderate score of 9 after extubation was noted.
- In postoperative period when patient complained of pain (VAS>3), Inj. Tramodal 2 mg/kg/hr was given as a rescue analgesia. Time to first rescue analgesic requirement from stopping IV lignocaine infusion was noted.

OBSERVATION AND RESULTS:

To assess the effect of lignocaine on sevoflurane requirement, the dial setting of sevoflurane vaporizer was noted at the time of induction, during intubation, at skin incision, 5 minutes after skin incision and at every 10 minutes up to 30 minutes and subsequently at every 15 minutes during intraoperative period. Pulse rate, SBP and DBP were also recorded at above time points.

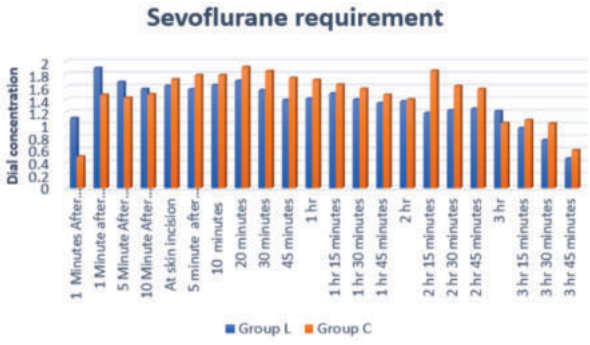
Sevoflurane dial concentration at different time interval:

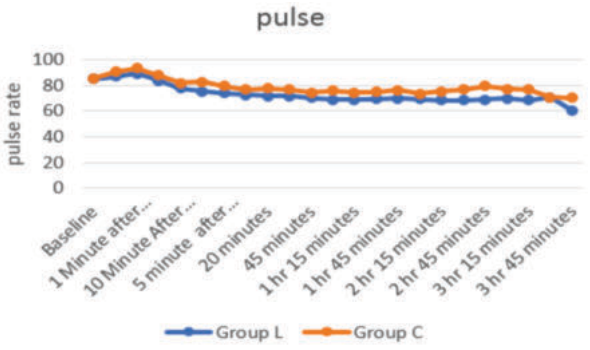
In group L, sevoflurane requirement was significantly lower at 30 minutes, 45 minutes, 60 minutes,135 minutes ,150 minutes, 165 minutes, and at 210 minutes as compared to group C. ($p < 0.05$)

Pulse rate at different time interval:

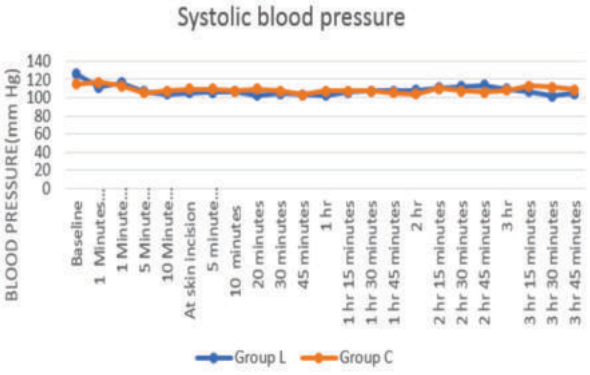
The mean heart rate remained comparable in both groups throughout study period except at 60 minute ,105 minute,135 minute ,150-minute, 165 minute ,180 minute, 195 minutes ,225 minutes. ($P < 0.005$). At

above time points, mean heart rate was statistically significantly higher in group C as compared to group L. However, difference was clinically not significant.

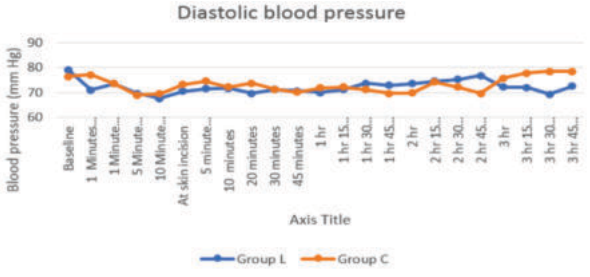




Systolic blood pressure (mm Hg) at different time interval:
When all the readings of mean systolic blood pressure of both groups were compared, we observed no statistically significant difference between the groups except at 20 minutes, 165 minutes, 210 minutes (p>0.05). it was higher in control group; however, the difference was clinically not significant.



Diastolic blood pressure (mm Hg) at different time interval:
Mean diastolic blood pressure was comparable throughout time period except at 165 minute and 210 minutes. However, difference between the DBP in both the group was clinically not significant throughout the study period.



Mean duration of postoperative analgesia after surgery:

	Group L (minutes)	Group C (minutes)	P VALUE
Duration	202.4±150.48	90.68 ±47.52	<0.05

In group L, duration of post operative analgesia was statistically significantly longer as compared to group C(p<0.05).

In each group, 8 patients required supplemental dose of fentanyl to maintain SBP within range..

Bradycardia (HR<20% of baseline or HR<50/minute) for more than 2 minutes not responding to decrease in sevoflurane concentration was treated with Inj. Glycopyrrolate 0.2mg IV. None of the patients in group L required glycopyrrolate while 1 patient in group C required glycopyrrolate. The difference between the two groups was statistically insignificant.

None of the patients in either group required ephedrine.

Time to achieve Aldrete score ≥9:
The mean time to achieve Aldrete score ≥9 after extubation is shorter in group L as compared to group C. (P>0.05) (1.92±0.90 in group L VS 2.24±1.47 in group C)

	GROUP L	GROUP C	P VALUE
Duration (minutes)	1.92±0.90	2.24±1.47	> 0.05

DISCUSSION:
Lignocaine is perhaps the most frequently used drug by an anaesthetist. It is not only used for local anaesthesia but also for reduction of sympathetic response to laryngoscopy, reduction of pain associated with propofol injection, prevention of postoperative airway complications and to treat perioperative arrythmia.[1] On reviewing literature, we found that it also reduces intraoperative sevoflurane requirement, helps in postoperative pain control and Enhanced recovery after surgery (ERAS). It is a part of ERAS protocol.

During spine surgery, challenges for anaesthesiologist are to maintain hemodynamic stability, to minimize blood loss, prevent neuronal ischemia and rapid recovery to allow neurological assessment in immediate post operative period.

The mechanism through which lignocaine reduces requirement of inhalational agents is not well understood but different theories are proposed by different authors for its anaesthetic-sparing effects like facilitation of hypnotic agents via GABA-receptor effects in the central nervous system (CNS) and a direct sedative and hypnotic action. It also blocks sensory transmission or exhibits anti-nociception.[4] Brain activity was reduced by systemic lignocaine administration and it has inhibitory effect at the central nervous system.

All mechanisms are supported by a study done in 2006 by Gaughen CM et al in which they studied the effect of high dose of intravenous Lidocaine on bi-spectral index. In this case report, lidocaine was administered at a dose of 100 mg per min for 7–8 min and BIS reached to zero.[5] The effect of IV lidocaine on MAC of inhalational agent may be due to its motor response blocking effect at spinal level,[5] which is enhanced by its central and peripheral analgesic effect.

In 2022, Ilona Batko et al used lidocaine as a bolus of 1.5 mg/kg over 30 minutes before incision followed by infusion at 1mg/kg/hour up to 6 hours after surgery and observed that End-tidal sevoflurane requirement to maintain intraoperative hemodynamic stability and proper depth of anaesthesia was reduced by 15% in lidocaine group as compared to control group.

In 2017 Laurence Weinberg and Jae Jang used IV lignocaine 1.5 mg/kg loading dose followed by infusion at 1.5 mg/kg/hr till duration of surgery observed that the average end tidal sevoflurane concentration was lower in lignocaine group (1.49%) as compared to saline group (1.89%) (p<0.001). In2013, Ahmed m. Omar and Osama h. aboushanab used IV lignocaine bolus 1.5 mg/kg over a period of 1 minute during induction followed by infusion at 2mg/kg/hour started

after 5 minutes of induction observed that End-tidal sevoflurane concentration were significantly higher in control group as compared to lignocaine group.

Intravenous lignocaine suppresses neuronal excitability in dorsal horn neurons, depresses spike activity, amplitude, and conduction time in both myelinated A and unmyelinated C fibres. It causes blockade or inhibition of nerve conduction which in turn decreases the neural response to post-operative pain.[11]

Another theory for analgesic property says that after damage to peripheral nerves, there is a high expression of sodium channels on their cell membranes which causes persistent spontaneous discharges that maintain the central nervous system in a state of hyperactive. Lidocaine probably by the inhibition of Na channels (all isoforms) as well as NMDA and G-protein-coupled receptors suppresses spontaneous impulses generated from injured nerve fibres and the proximal dorsal root ganglion.[2] Lidocaine also reduces neurogenic inflammation at the site of tissue injury, by inhibiting granulocyte migration, and reducing the release of lysosomal enzymes and thus pro-inflammatory and anti-inflammatory cytokines.This effect probably is achieved by inhibition of VGSC, G-protein coupled receptors and ATP-sensitive potassium channels. This systemically administered local anaesthetic also has a desensitizing effect on TRP (transient receptor potential) channels, which are key components in nociception and neurogenic inflammation, what explains its long-lasting analgesic effect.[2] Lidocaine limits peripheral and central sensitization. This anti-hyperalgesic effect is mainly the result of suppression of the neuro-inflammatory response to pain, blockade of neural transmission, inhibition of NMDA receptor and modulation of the glycinergic system.[2]

In 2020 Ilona Batko et al lidocaine as a bolus of 1.5 mg/kg over 30 minutes before incision followed by infusion at 1mg/kg/hour up to 6 hours after surgery and observed that lidocaine treatment reduced the demand for morphine and there was no difference in fentanyl requirement during intraoperative period. In 2017 Krishna Murthy et al used lignocaine 1.5 mg/kg as a bolus over 10 minutes before induction followed by 1.5 mg/kg/h continuous infusion till 1 hour post operatively and observed that there was significant increase in duration of postoperative analgesia in patients who received IV lignocaine. In 2016 S. Weibel et al observed that lidocaine reduces post operative pain (VAS 0-10) at 1- 4 hours and at 24 hours compared to control group after surgery.

The attenuating effects on haemodynamic has been attributed to lignocaine induced arteriolar vasodilatation, mitigation of autonomic reaction, increased depth of general anaesthesia and it also might be due to its cough suppressant activity, which could increase BP and PR at the time of extubation due to tracheal irritation.

In 2015 Shruti Jain and Rashid M khan used IV lignocaine 1.5 mg/kg IV bolus (diluted up to 6 ml with normal saline) over 10 minutes before induction followed by 1.5 mg/kg/h continuous infusion (diluted with normal saline at rate of 6 ml/hr.) till 1 hr postoperatively and observed that during laryngoscopy and intubation and extubation, the haemodynamic parameters (PR and MAP) increased significantly in both the groups. Though the rise of both in lignocaine group was significantly less than control Group.

CONCLUSION:

Thus, from this study we conclude that; Intra operative administration of systemic lignocaine reduces sevoflurane requirement to maintain anaesthesia in major spine surgery. It also increases the duration of postoperative analgesia with no significant effect on intraoperative opioid requirement when used as component of balanced anaesthesia during major spine surgery.

Despite this, our study has some limitations like vaporizer dial setting was used to calculate sevoflurane requirement. Gas monitoring can give more precise information than dial setting. Inability to measure plasma lignocaine levels prevented us from knowing that during our study, plasma level of lignocaine reached a toxic level or not. Our study did not include parameters such as total hospital stay, early ambulation, benefits in early postoperative cognitive functions and total cost effectiveness for the patient.

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