Original Research Article

Observation of hemodynamic variability and level of blood cortisol while induction with propofol and etomidate: A prospective, randomized, double blind study

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A B S T R A C T

Background: To observe the effects of propofol and etomidate induction on patients undergoing laproscopic cholecystectomies regarding serum cortisol level, hemodynamic parameters, pain on injection, myoclonus, and apnea on induction.

Materials and Methods: It is a prospective, randomized, double blind study. After approval from Institute Ethics Committee, sixty patients of age between 18 and 60 years with ASA grade I and II scheduled for laparoscopic cholecystectomy under general anesthesia were randomly divided into two groups: Group A (n=30) would receive injection propofol 2 mg/kg i.v., Group B (n=30) would receive injection etomidate 0.3 mg/kg i.v. as induction agent. Vital parameters at induction, laryngoscopy and thereafter recorded. Pain on injection, myoclonus, apnea on induction were carefully watched. Serum Cortisol is measured at 1 hour before induction and at 2 hours and 24 hours after induction.

Results: Demographic variables and baseline parameters were comparable in both groups. Propofol group show significant decrease in heart rate and mean blood pressure after induction in comparison to etomidate group. Pain on injection was more in group A while myoclonus activity was higher in Group B. Serum cortisol level is significantly lower in Group B post induction compared to Group A which comes to above baseline after 24 hrs but in normal range.

Conclusion: Etomidate is a better induction agent than propofol in view of hemodynamic stability but also led to chemical evidence of adrenocortical insufficiency in patients with ASA grade I & II undergoing elective laparoscopic cholecystectomy under general anesthesia which returns to normal in 24 hrs.

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1. Introduction

In general anesthesia, airway management and patient safety is the most important aspect of patient management. Endotracheal intubation is the gold standard and safest method for protecting the airway, delivering anesthetic gases and ensuring protection against aspiration.1,2 Stress response during laryngoscopy and intubation leads to hemodynamic changes especially for patients who are under cardiac risk factors like hypertension and ischemic heart disease.3 The unavoidable effects of laryngoscopy and tracheal intubation includes dysrhythmia, hypertension, myocardial ischemia, infarction, hypoxia, hypercapnea, laryngospasm, and bronchospasm, and some rare side effects such as increased intracranial pressure and increased intraocular pressure.

Since the introduction of general anesthesia, no ideal induction agent has yet been discovered in term of providing a stable hemodynamics. In past many studies have been comparing different anesthetic induction agents, but studies
regarding comparison of propofol and etomidate are only few.

Propofol, 2,6-diisopropyl phenol is one of the commonly used drug for induction of general anesthesia. This is a short acting intravenous anesthetic agent. Recommended dose of propofol for induction is 1-2.5 mg/kg. Unwanted complication associated with this drug is hemodynamic instability and cardiovascular complications i.e. irregular heart rate, low blood pressure. Propofol can lead to bradycardia by increasing the production and release of nitrous oxide, also causes pain at injection site, as a respiratory depressant, it frequently produces apnoea on induction. As Propofol has been proposed to have several mechanisms of action both through potentiation of GABA receptor activity and also acting as a sodium channel blocker. Recent research has also suggested that endocannabinoid system may contribute significantly to propofol’s anaesthetic action and to its unique properties. Propofol did not reduce cortisol levels after surgery.

Etomidate, carboxylated imidazole, is a hypnotic agent which is cardio stable with no release of histamine, minimal respiratory depression and cerebral protective effects. It is short acting drug, used for induction and maintenance of anesthesia. Etomidate was first introduced in the early seventies, but was soon withdrawn, because of anaphylactic reactions to a stabilizing agent Cremophore EL. The most important side effects of Etomidate are nausea and vomiting that may lead to aspiration in patients. Intravenous injection of Etomidate would cause a burning sensation. One of the most important effect of this drug is the suppression of steroids production by reversible inhibition of 11-betahydroxylase enzyme. Its lack of effect on sympathetic nervous system, baroreceptor reflex regulatory system and its effect of increased coronary perfusion even on patients with moderate cardiac dysfunction makes it an induction agent of choice in cardiac disease patients. Induction of anaesthesia by Etomidate would lead to a stable hemodynamic condition for performing laryngoscopy and endotracheal intubation. Etomidate is known to cause a reduction in serum cortisol levels even after a single dose, leading to reduced cortisol levels for up to twenty-four hours.

The reduction in serum cortisol levels offered by etomidate may be beneficial provided it does not persist in the postoperative period, when the body’s circulatory reflexes need to be intact for the maintenance of hemodynamic parameters.

2. Materials and Methods

This study was a prospective randomized double blind clinical trial. After approval from institutional ethical committee, 60 patients aged between 18 to 60 years of either sex and ASA physical status I and II scheduled for elective laparoscopic cholecystectomy under general anesthesia were taken for study out of which 1 patient in group B was excluded from study on account of an episode of bronchospasm during extubation so given inj. hydrocortisone 100 mg iv. Written informed consent was taken from all patients. The patients were randomly divided into two groups of 30 patients each: Group A - Inj. Propofol (2 mg/kg) iv. And Group B - Inj. Etomidate (0.3 mg/kg) iv. Patients having following criteria were excluded from the study: Patient refusal to GA, ASA physical status III and IV, Emergency surgery, Patient with history of hypersensitivity to Propofol/ Etomidate, Bronchial asthma, Mallampati grade 3 and 4, Existence of considerable pathology in pharynx / larynx, Patient with GERD, Patient on steroid, Acute Pancreatitis due to gall stones, Severe acute Cholecystitis.

Pre anesthetic checkup of the patient was done a day before surgery and patients were counselled regarding adequate starvation, sedation, local anesthesia and operative procedure. Investigations were done according to the institutions protocol.

The patients were kept nil per orally for 8 hours prior to surgery. All patients were premedicated with tab. Alprazolam 0.25 mg, and tab. Ranitidine 150 mg at the night before surgery. All patients received inj. glycopyrrolate 0.2 mg IM 45 minutes before induction in the preoperative ward.

On arrival at operation theater, standard anesthesia monitors including electrocardiogram (ECG), non-invasive blood pressure (NIBP) and pulse oximetry were attached and hemodynamic parameters HR, Mean blood pressure, Oxygen saturation before induction, at induction and at 5 minutes, 10 minutes, 15 minutes, 30 minutes, 45 minutes and 60 minutes after laryngoscopy were recorded. An 18 G intravenous (IV) cannula in right hand secured and inj. ringer’s lactate infusion started. Inj. midazolam 0.025 mg/kg iv and Inj. fentanyl 2 μg/kg iv given 2 minutes before induction.

Anaesthesia was induced with either propofol 2 mg/Kg iv or etomidate 0.3 mg/Kg iv according to their group. After induction, adverse events like pain on injection, apnea on induction and myoclonus were recorded. Myoclonus movement on induction recorded as Grade 0 – No myoclonus movements, Grade 1 – Minor myoclonic movements, Grade 2 – Moderate myoclonic movements, Grade 3 – Major myoclonic movements. Pain on injection during induction measured as: Grade 0 – No pain, Grade 1 – Verbal complaint of pain, Grade 2 – Withdrawal of arm, Grade 3 – Both verbal complaints and withdrawal of arms

Later 60 seconds after loss of consciousness, which was confirmed by inability to respond to verbal commands and loss of eyelash reflex. Inj. vecuronium (0.1 mg/kg iv) was given, Laryngoscopy and endotracheal intubation was done after 3 min, confirmed with capnometry and bilateral auscultation of chest.
Anaesthesia was maintained by injection midazolam infusion 0.25-1μg/kg/minute iv and equal mixtures of oxygen-nitrous oxide along with intermittent bolus of vecuronium (maintenance dose 0.01-0.02 mg/kg iv) as required throughout the surgery.

At the end of the surgery residual neuromuscular block was antagonized with inj. neostigmine (0.05 mg/kg) iv and inj. glycopyrolate (0.01 mg/kg) iv and extubation was performed when respiration was adequate and patient was able to obey verbal commands.

Before 1 hr, after 2 hrs and 24 hrs of induction, 2ml blood sample is collected under aseptic precautions in serum separating tube (SST) vial and sent to laboratory where it was measured by chemiluminescent assay.

2.1. Statistical analysis

The sample size was calculated before beginning the study by calculating the power. With a power of 90% & an alpha error of 0.05% the sample size (n) came out to be 25 in each group but in our study, we selected estimated 30 patients in each group, considering if any patient left the study meanwhile or underwent any complications like bronchospasm etc.

All the data was presented as mean ± standard deviation (SD). Statistical Significant difference in the means between the groups was calculated using unpaired t test and ANOVA test. All difference were considered significant at p<0.05.

3. Result

The two groups were comparable in terms of demographic data as there were no statistically significant differences between the groups in terms of age, sex and weight. (Table 1)

Mean heart rate(HR), Mean Arterial Pressure(MAP) and mean percentage oxygen saturation is comparable in both the groups. Mean heart rate on induction in Group A is significantly decreased and in group B, the decrease is non significant and at 5 min after intubation, mean HR is more increased in group A but the difference in both groups is statistically non significant. (Table 2)

The mean MAP is significantly decreased in both the groups (p<0.001) but the fall is more in group A and at 5 min after intubation MAP is increased in both the groups which becomes comparable in both the groups at 10 min. Hypotension occurs with propofol is mainly due to reduction of sympathetic activity causing vasodilation or its direct effect on vascular smooth muscles. Sudden hypotension and bradycardia has deleterious effects on maintaining the circulation to vital organs in patients of coronary artery disease, valvular stenosis, uncontrolled hypertension and shock. On another side, hemodynamic stability observed with etomidate may be due to its unique effect of lack on the sympathetic nervous system and on baroreceptor functions.

Our findings were similar to Mehrdad et al used propofol and etomidate for anesthesia induction and found that propofol significantly reduced the MAP after induction. Schmidt et al found that hypotension caused by propofol is due to the reduction of heart’s preload and afterload, which are not synchronized with heart’s compensatory responses such as increased cardiac output and increased heart rate. This is in accordance with our finding of fall in blood pressure and slight decrease in heart rate.

On comparing the adverse effects, it was found that In Group A, 20 out of 30 patients had pain on receiving injection Propofol while 28 out of 29 patients had no pain on receiving injection Etomidate.

In group A, no myoclonic movement was seen at induction with inj propofol while 24% of patients i.e. 7 out of 29 was showing Grade I myoclonus movements and 2 patients (7%) was having Grade II myoclonic movements on induction with inj. Etomidate. In Group A, 22 out of 30 patients went into apnea on induction with inj propofol in comparison to Group B whereas 18 out of 29 patients went into apnea on induction with inj etomidate.

4. Discussion

In our study, the demographic variables and hemodynamic parameters in both the groups were comparable. In Group A patients, there was a significant (p value 1.9E-05) decrease in the heart rate after induction with a mean heart rate of 75.46±10.85 bpm whereas in Group B, it was non-significant (p value 0.0507) decrease in the heart rate with mean value 79.31±7.82 bpm. We found that after induction, there was a decrease in heart rate from the baseline in both the groups which on comparison was found to be non-significant (p value 0.0615). The findings of this study corroborates with the study report of Sarkar Molly et al. (2005), Shagun Bhatia Shah et al (2015), Kaushal Ram Prasad et al. (2015), Binod Pegu et al (2017) that decrease in heart rate is more on induction with injection propofol than with injection etomidate which on comparison found to be non-significant. The mean MAP is significantly decreased in both the groups (p<0.001) but the fall is more in group A and at 5 min after intubation MAP is increased in both the groups which becomes comparable in both the groups at 10 min. Hypotension occurs with propofol is mainly due to reduction of sympathetic activity causing vasodilation or its direct effect on vascular smooth muscles. Sudden hypotension and bradycardia has deleterious effects on maintaining the circulation to vital organs in patients of coronary artery disease, valvular stenosis, uncontrolled hypertension and shock. On another side, hemodynamic stability observed with etomidate may be due to its unique effect of lack on the sympathetic nervous system and on baroreceptor functions.

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Table 1: Demographic characteristics of patients

| S.No. | Variables       | Group A       | Group B       | P value |
|-------|-----------------|---------------|---------------|---------|
| 1.    | Sex(M:F)        | 7:23          | 9:20          |         |
| 2.    | Age             | 32.80 ± 6.15  | 34.31 ± 7.25  | 0.196   |
| 3.    | Weight          | 54.50 ± 10.47 | 55.66 ± 12.17 | 0.348   |
| 4.    | ASA Grade (I/II)| 27/3          | 25/4          |         |

Table 2: Comparison of heart rate between the groups A & B

| S.No. | Heart Rate | Group A (MEAN±SD) | Group B (MEAN±SD) | t       | P       |
|-------|------------|-------------------|-------------------|---------|---------|
| 1.    | Baseline   | 81.13±9.10        | 80.34±7.49        | 0.36    | >0.05   |
| 2.    | At induction| 75.46±10.85      | 79.31±7.82        | 1.56    | >0.05   |
| 3.    | 5 min      | 87.23±9.27        | 83.69±7.95        | 1.57    | >0.05   |
| 4.    | 10 min     | 81.66±9.09        | 81.72±7.27        | 0.028   | >0.05   |
| 5.    | 15 min     | 84.97±8.45        | 84.34±6.83        | 0.31    | >0.05   |
| 6.    | 30 min     | 80.96±7.33        | 83.1±9.50         | 0.98    | >0.05   |
| 7.    | 45 min     | 77.56±6.40        | 79.31±8.35        | 0.91    | >0.05   |
| 8.    | 60 min     | 75.36±6.34        | 77.82±7.31        | 1.39    | >0.05   |

Table 3: Comparison of mean arterial pressure between the groups A & B

| S.No. | MAP | Group A MEAN±SD | Group B MEAN±SD | t       | P       |
|-------|-----|-----------------|-----------------|---------|---------|
| 1.    | Baseline | 82.26±4.54     | 83.62±3.48      | 1.28    | >0.05   |
| 2.    | At induction | 72.63±4.19    | 81.86±3.35      | 9.34    | <0.001  |
| 3.    | 5 min | 80.56±3.83      | 82.45±3.21      | 2.04    | <0.05   |
| 4.    | 10 min | 81.23±4.44      | 81.58±2.51      | 0.38    | >0.05   |
| 5.    | 15 min | 84.06±3.87      | 84.55±2.83      | 0.55    | >0.05   |
| 6.    | 30 min | 81.33±4.13      | 82.31±3.15      | 1.02    | >0.05   |
| 7.    | 45 min | 78.96±4.02      | 79.72±3.09      | 0.81    | >0.05   |
| 8.    | 60 min | 79.06±4.10      | 78.86±2.85      | 0.22    | >0.05   |

*NC = Non-calculable

Table 4: Comparison of percentage oxygen saturation between groups A & B

| S. No | Oxygen saturation | Group A (MEAN±SD) | Group B (MEAN±SD) | t       | P       |
|-------|-------------------|-------------------|-------------------|---------|---------|
| 1.    | Baseline          | 98.33±0.75        | 98.17±0.75        | 0.82    | >0.05   |
| 2.    | At induction      | 99.7±0.46         | 99.86±0.35        | 1.5     | >0.05   |
| 3.    | 5 min             | 100               | 100               | NC      |         |
| 4.    | 10 min            | 100               | 100               | NC      |         |
| 5.    | 15 min            | 100               | 99.96±0.18        | 1.23    | >0.05   |
| 6.    | 30 min            | 100               | 100               | NC      |         |
| 7.    | 45 min            | 100               | 100               | NC      |         |
| 8.    | 60 min            | 100               | 100               | NC      |         |

Table 5: Comparison of effects on serum cortisol between groups A & B

| S. No | S. Cortisol | Group A (MEAN±SD) | Group B (MEAN±SD) | T       | P       |
|-------|-------------|-------------------|-------------------|---------|---------|
| 1.    | 1 hr before induction | 12.65±2.15        | 13.55±2.28        | 1.57    | >0.05   |
| 2.    | 2 hrs after induction | 16.42±2.40        | 7.46±1.75         | 16.43   | <0.001  |
| 3.    | 24 hrs after induction | 19.64±1.92        | 16.54±2.60        | 5.19    | <0.001  |
The difference in baseline serum cortisol in both the groups is statistically non-significant. In Group A, there is highly significant increase (p<0.001) in level of serum cortisol to 16.42±2.40 µg/dl from the baseline but within normal range, 2 hrs after induction. After 24 hrs, serum cortisol came out to be 19.64±1.92 µg/dl that also lie within normal range. In Group B, there is highly significant decrease (p<0.001) in level of serum cortisol to 7.46±1.75 µg/dl at 2 hrs after induction. After 24 hrs, serum cortisol came out to be 16.54±2.60 µg/dl that lie within normal range.

So, induction with propofol does not have any effect on serum cortisol but induction with etomidate decreases serum cortisol levels significantly at 2 hrs but returns back to normal within 24 hrs. This effect of etomidate is because of its ability to reversibly inhibit the enzyme 11β-hydroxylase in the pathway of steroidogenesis, which is a rate limiting enzyme. Our study is consistent with the findings of Pandey A. K. et al who found that serum cortisol level was significantly lower but still within normal level in the etomidate group (9.2 to 8.14 µg/ml) as compared to propofol group (11.4 to 28.8 µg/ml). The level of serum cortisol were returned to almost normal by 24 hrs.

On the other side, this suppression of the adrenal synthesis of cortisol though it is transient that lasts for at least 24 hrs after a single dose of etomidate administration, in septic patients has shown to be a risk factor (as cortisol play an important role in maintaining systemic vascular resistance) for increased mortality and can be detrimental in septic patients who may have a baseline adrenal insufficiency due to critical illness. This is favoured by Chan et al concluded that administration of etomidate for rapid sequence induction is associated with higher rates of adrenal insufficiency and mortality in patients with sepsis.

So, the use of etomidate should be done with caution in patients with critical illnesses where systemic vascular resistance is affected like septicemia.

Incidence of pain in Group A was 66% i.e. 20 out of 30 patients had pain on receiving injection Propofol while 1 out of 29 patients (3%) had pain on receiving injection Etomidate. Also, the severity of pain was more in Group A, 60% patients was showing grade I and 6% patients grade II of severity while in Group B, 3% patients showing only grade I of severity. This finding was very well-supported by Saricaoglu et al and Wu et al.

In Group A, no myoclonic movement was seen at induction with inj propofol while in Group B 24% of patients i.e. 7 out of 29 was showing Grade I myoclonus movements and 2 patients(7%) were having Grade II myoclonic movements on induction with inj. Etomidate. This finding was supported by Miner et al who concluded high incidence of myoclonus (20% vs. 1.8%) in etomidate in comparison to propofol group respectively. Similarly, Supriya Aggarwal et al also concluded that myoclonus activity was higher in etomidate group than propofol group.

Incidence of apnea in Group A was 74% i.e. 22 out of 30 patients went into apnea on induction with inj propofol while in Group B, it was 62% i.e. 18 out of 29 patients went into apnea on induction with inj etomidate. This finding is supported by Boysen et al who concluded that there was no significant difference between two groups (propofol and etomidate) as regard to apnea following induction.

5. Conclusion

Based on our findings, we can conclude that Etomidate is hemodynamically more stable than propofol as an induction agent associated with less incidence of pain on injection but have relatively higher incidence of myoclonic movements. It is also found that etomidate led to chemical evidence of adrenocortical insufficiency for shorter duration which become normal within 24 hours in patients with ASA grade I & II undergoing elective laparoscopic surgery under general anaesthesia.

On the basis of chemical evidence of adrenocortical insufficiency, we can also conclude that etomidate should be used with caution in specific group of patients where systemic vascular resistance is affected. E.g. Septicemia.

Further studies are needed to be done in such specific group of patients to get an evidence about etomidate relationship with increased risk of death, greater vasopressor use or duration of mechanical ventilation, or longer ICU or hospital stay.

6. Source of Funding

None.

7. Conflict of Interest

None.

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