Section: Anesthesia

Original Research Article

DEXMEDETOMIDINE IN ATTENUATING ARTERIAL PRESSURE RISE DUE TO PNEUMOPERITONEUM IN ELECTIVE LAPAROSCOPIC CHOLECYSTECTOMY

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ABSTRACT

Background: Aims: To study the effect of dexmedetomidine in attenuating the arterial pressure increase due to CO2 pneumoperitoneum in patients posted for elective laparoscopic cholecystectomy undergoing general anaesthesia.
Materials and Methods: 62 Prospective observational study in 60 patients of age 18-60 years ASA 1 & 2 of both sex undergoing elective laparoscopic cholecystectomy under general anaesthesia are selected. Group A: 30 patients receiving normal saline IV infusion 10 minutes before pneumoperitoneum. Group B: 30 patients receiving IV dexmedetomidine (0.5µ/kg IV bolus followed by IV infusion of 0.5 µ /kg/hr) 10 minutes before pneumoperitoneum.
Results: We observed, that the systolic, diastolic and mean arterial pressure increased abruptly after induction of pneumoperitoneum and this response sustained during the entire pneumoperitoneum period in the control group (Group A). Dexmedetomidine group (group B) hemodynamic responses like heart rate and blood pressure levels to the induction of pneumoperitoneum were effectively blunted when compared to the control group (Group A). p Value <0.05 significant in HR, SBP, DBP, MAP during the entire duration of pneumoperitoneum and post extubation.
Conclusion: We conclude that intravenous administration of dexmedetomidine as an adjunct before induction of CO2 pneumoperitoneum in laparoscopic cholecyctectomy effectively attenuates the arterial pressure increase arising due to pneumoperitoneal response by suppressing central sympathetic outflow.
Keywords: End Tidal Carbondioxide(Pet CO2), Dexmedetomidine, Laparoscopic cholecyctectomy.

INTRODUCTION

Surgical procedures and anaesthetic techniques and gadgets have improved over decades with recent advances and there is drastic fall in the mortality and morbidity. As a result of that there is consequent reduction in health care cost. With the invent of better equipment and modern facilities, along with increased knowledge and better understanding of anatomy, physiology and pathophysiology, has lead to the development of laparoscopy for diagnostic and operative procedures. The pneumoperitoneum and the patient positions required for laparoscopy include a sequence of pathophysiologic changes in terms of increased intra abdominal pressure (IAP) and systemic CO2 absorption that can complicate anaesthesia. Hence better understanding of the CO2 pneumoperitoneum in laparoscopy is important for the anesthesiologist for better management of the patient. Moreover with the advancements in medical field there is increase in the life expectancy. So as anaesthesiologist, we are expected to anaesthetise elderly patients with associated co morbid conditions like diabetes, hypertension, Ischemic heart disease etc. So understanding the physiology of CO2 pneumoperitoneum becomes very much essential.[1,2] The multiple benefits like reduced hospital stay, post operative pain, respiratory complications and less cost reported after laparoscopy explains its increasing use and has now become the standard technique for

cholecystectomy. However, the CO2 pneumoperitoneum required for laparoscopy results in pathophysiologic changes particularly in cardiovascular system and respiratory system like 10-30 % decrease in cardiac output, significant increase in arterial pressure and systemic vascular resistances occurring soon after the beginning of intra abdominal insufflation, with no significant changes in heart rate (HR). Both mechanical and neurohumoral factors contribute to these hemodynamic changes. There is an increase in catecholamines, prostaglandins, renin and vasopressin levels.[3,4]

There are lots of anaesthetic methods and anaesthetic drugs have been used for attenuating the response associated with pneumoperitoneum. It has been already studied that Clonidine, alpha2-adrenergic agonist effectively attenuates the pneumoperitoneal response of the laparoscopy. Recently, dexmedetomidine is another drug of same family but more specific than Clonidine with better safety profile. We therefore tested the hypothesis, that dexmedetomidine might attenuate the hemodynamic changes induced by increased intra abdominal pressure due to CO2 pneumoperitoneum by reducing release of noradrenaline.

MATERIALS AND METHODS

62 Prospective observational study in 60 patients of age 18-60 years ASA 1 & 2 of both sex undergoing elective laparoscopic cholecystectomy under general anaesthesia are selected.

GROUP

Group A: 30 patients receiving normal saline IV infusion 10 minutes before pneumoperitoneum

Group B: 30 patients receiving IV dexmedetomidine (0.5µ/kg IV bolus followed by IV infusion of 0.5 µ /kg/hr) 10 minutes before pneumoperitoneum

Inclusion Criteria: cases 18 to 60 years in both sex of ASA 1 &2 posted Elective laparoscopic cholecystectomy without any co morbid condition Undergoing general anaesthesia

Exclusion Criteria: Patient on any drug treatment which may interfere with dexmedetomidine , Hypertension, Diabetes mellitus, Cardiovascular & kidney disease, Acute cholecystitis, Endocrine or metabolic diseases, Autonomic neuropathy and Patients on chronic β blocker therapy

Pulse oximetry NIBP, ECG, Et CO2, Airway pressure monitoring (PIP and mean pressure), Intra abdominal pressure (maintained around 12 mm of Hg). Urine output monitoring and Temperature monitoring are done. After ascertaining the inclusion criteria preoperative investigations were recorded which included complete hemogram, blood sugar, urea, creatinine, serum electrolytes, blood grouping, blood coagulation tests, urine routine, chest X-ray and ECG.

Preoperative instructions

All patients are explained about the study and written informed consent obtained.

Patients are advised a 6 hour period of absolute fasting. All patients receive an antacid prophylaxis of injection ranitidine 50 mg IV and injection ondansetron 4 mg IV on the morning of surgery. All the patients are premedicated with injection glycopyrrolate 0.2 mg IV one hour before surgery.

60 patients are randomized into two groups (group A and group B).

Conduct of anaesthesia

After shifting the patients to operation theatre patients are connected to ECG, pulse oximetry, and NIBP monitors. All patients are started with ringer lactate at 75 ml/hr. All patient given fentanyl 2µg/kg IV and pre oxygenated with 100% oxygen for 3 – 5 minutes. In all the patients, trachea was intubated after induction of anaesthesia with propofol 1.5-2 mg/kg and vecuronium 0.1 mg/kg. Anaesthesia maintained with 1.5-2% sevoflurane and 3:2 N2O/O2 at 5 litres/ minute.

After induction of general anaesthesia and 10 minutes before creation of CO2 pneumoperitoneum study group (group B) received IV dexmedetomidine 0.5µg/kg bolus dose over 10 minutes followed by 0.5µ/kg/min infusion and control group (group A) received normal saline at same infusion rate. In both the groups infusion was continued till dissection of gall bladder was complete.

Heart rate, SBP, DBP, MAP are measured before induction, pre pneumoperitoneum, at pneumoperitoneum(P0), at 2min, 5 min , 10 min, 15 min, 20 min, 30 min, 45 min after pneumoperitoneum and post surgery at 0,5,10 mins.

After completion of surgery, pneumoperitoneum deflated slowly and after the patient had adequate respiratory attempts patient reversed with glycopyrrolate and neostigmine IV. Adequate oral suctioning done and the patients are extubated. After adequate recovery from general anaesthesia, patients were shifted to recovery room where they remained and observed until there was complete recovery from general anaesthesia for 2 hours.

Statistical Analysis: Variables were analysed with Student ‘t’ test . Variables like age, sex, weight, height were compared using Levene‘s test for equality of variance. Sample size obtained according to previous background study. p‘ value less than 0.05 was taken as significant.

RESULTS

Table 1: Demographic and other parameters

Variable	Control (Group A)	Study (Group B)	P Value
Age	**42.9 ± 9.9**	**45.46 ± 9.26**	**0.3053**
Sex M/F	**16/14**	**15/15**	
Weight (Kgs)	**64.57 ± 9.30**	**68.33 ± 8.40**	**0.1057**
Height (Cms)	**164.06 ± 7.58**	**165.06 ± 7.63**	**0.6125**
Duration of surgery (minutes)	**70.33 ± 12.20**	**67.57 ± 12.38**	**0.3880**
Duration of anaesthesia (minutes)	**96.33 ± 13.95**	**92.60 ± 13.98**	**0.3052**
Pre OP HR (per minute)	**85.77 ± 10.2**	**85.47 ± 5.21**	**0.8864**
Pre OP SBP (mmhg)	**110.83 ± 11.12**	**111.80 ± 10.30**	**0.7272**
Pre OP DBP (mmhg)	**66.90 ± 9.17**	**69.93 ± 9.17**	**0.2057**
Pre OP MAP (mmhg)	**81.63 ± 9.54**	**83.80 ± 9.01**	**0.3688**

M - Male; F - Female; kgs - kilograms; cms - centimetres; mmhg - millimetre of mercury; SBP - Systolic Blood Pressure; DBP - Diastolic Blood Pressure;
MAP - Mean Arterial Pressure

Table 2: Heart Rate in present study

Variable	Control (Group A)	Study (Group B)	P Value
P0	88.40 ± 11.19	76.90 ± 4.99	<0.0001*
P2	88.46 ± 10.90	75.57 ± 4.87	<0.0001*
P5	84.87 ± 6.03	75.10 ± 4.36	<0.0001*
P 10	86.40 ± 11.03	75.80 ± 6.29	<0.0001*
P 15	85.13 ± 11.19	73.17 ± 4.76	<0.0001*
P 20	87.20 ± 10.30	75.53 ± 10.49	0.0001*
P 30	83.46 ± 11.35	76.03 ± 6.68	0.0031*
P 45	85.03 ± 10.98	78.86 ± 6.31	0.0099*
PE 0	92.60 ± 11.80	85.87 ± 7.17	0.0098*
PE 5	93.37 ± 11.41	74.20 ± 6.35	<0.0001*
PE 10	93.20 ± 11.33	72.43 ± 6.84	<0.0001*

P-value <0.05 - significant Test method - Student t test

The heart rate is significantly low in Study group (Group B) than the Control group (Group A) during the period of pneumoperitoneum at 0 min (77 vs 88), 2nd min (76 vs 88), 5th min (75 vs 85), 10th min (76 vs 86), 15th min (73 vs 85), 20th min (75 vs 87), 30th min (76 vs 83), 45th min (79 vs 85) and post extubation at 0 (86 vs 93), 5th min (74 vs 93), 10th min (72 vs 93).

Table 3: Systolic blood pressure in present study

Variable	Control (Group A)	Study (Group B)	P Value
P0	106.30 ± 8.43	99.83 ± 3.77	0.0003*
P2	105.93 ± 7.35	100.13 ± 3.81	0.0003*
P5	119.80 ± 10.13	108.10 ± 4.97	<0.0001*
P 10	128.67 ± 8.92	109.90 ± 6.80	<0.0001*
P 15	126.83 ± 8.79	107.90 ± 6.79	<0.0001*
P 20	124 ± 7.33	115.13 ± 4.91	<0.0001*
P 30	119.67 ± 8.35	111 ± 7.09	0.0001*
P 45	117.67 ± 8.35	109 ± 7.10	0.0001*
PE 0	120.20 ± 8.27	110.43 ± 7.03	0.0001*
PE 5	119.93 ± 8.67	108.93 ± 7.27	<0.0001*
PE 10	118.43 ± 8.98	106.43 ± 7.53	<0.0001*

p value <0.05 - significant Test method - Student t test

The systolic blood pressure is significantly low in Study group (Group B) than the Control group (Group A) during the period of pneumoperitoneum at 0 min (100 vs 106), 2nd min (100 vs 106), 5th min (108 vs 120), 10th min (110 vs 129), 15th min (108 vs 127), 20th min (115 vs 124), 30th min (111 vs 120), 45th min (109 vs 118) and post extubation at 0 (110 vs 120), 5th min (109 vs 120), 10th min (106 vs 118).

Table 4: Diastolic Blood Pressure in present study

Variable	Control (Group A)	Study (Group B)	P Value
P0	75.80 ± 3.24	71.86 ± 4.60	0.0003*
P2	75.07 ± 3.78	72.13 ± 4.72	0.0100*
P5	84.33 ± 5.48	76.27 ± 3.67	<0.0001*

P 10	91.43 ± 7.26	77.07 ± 4.86	<0.0001*
P 15	89.43 ± 7.26	75.07 ± 4.86	<0.0001*
P 20	89.53 ± 7.73	80.87 ± 4.67	<0.0001*
P 30	84.97 ± 9.47	76.47 ± 9.97	<0.0001*
P 45	82.96 ± 9.46	74.5 ± 9.99	0.0014*
PE 0	85.50 ± 9.44	75.93 ± 9.89	0.0003*
PE 5	85.50 ± 10.17	74.43 ± 9.79	0.0001*
PE 10	84.00 ± 10.56	71.93 ± 9.71	<0.0001*

p value <0.05 - significant Test method - Student t test

The diastolic blood pressure is significantly low in Study group (Group B) than the Control group (Group A) during the period of pneumoperitoneum at 0 min (72 vs 76), 2nd min (73 vs 75), 5th min (76 vs 84), 10th min (77 vs 91), 15th min (75 vs 89), 20th min (81 vs 90), 30th min (76 vs 85), 45th min (75 vs 83) and post extubation at 0 (76 vs 86), 5th min (74 vs 86), 10th min (72 vs 84).

Table 5: Mean Arterial Pressure in present study

Variable	Control (Group A)	Study (Group B)	P Value
P0	86.83 ± 4.95	81.23 ± 4.20	<0.0001*
P2	85.40 ± 3.62	81.53 ± 4.11	0.0003*
P5	96.13 ± 6.74	86.8 ± 3.13	<0.0001*
P 10	103.97 ± 7.17	87.97 ± 3.99	<0.0001*
P 15	102.00 ± 7.13	85.63 ± 4.26	<0.0001*
P 20	101.07 ± 7.00	92.33 ± 3.64	<0.0001*
P 30	96.63 ± 8.75	87.86 ± 7.18	0.0001*
P 45	94.64 ± 8.76	85.87 ± 7.17	0.0001*
PE 0	97.00 ± 8.70	87.33 ± 7.15	<0.0001*
PE 5	97.00 ± 9.43	85.83 ± 7.13	<0.0001*
PE 10	95.50 ± 9.79	83.33 ± 7.15	<0.0001*

p value <0.05 - significant Test method - Student t test

The mean arterial pressure is significantly low in Study group (Group B) than the Control group (Group A) during the period of pneumoperitoneum at 0 min (81 vs 87), 2nd min (82 vs 86), 5th min (87 vs 96), 10th min (88 vs 104), 15th min (86 vs 102), 20th min (92 vs 101), 30th min (88 vs 97), 45th min (86 vs 95) and post extubation at 0 (87 vs 97), 5th min (86 vs 97), 10th min (83 vs 96).

DISCUSSIONS

In this study, neurohormonal hemodynamic response of dexmedetomidine in attenuating the arterial pressure increase is studied. The results obtained showed an effective attenuation of blood pressure and heart ratei n patients who received dexmedetomidine as compared to the patients who received normal saline. Thus proving that dexmedetomidine acts by suppressing the central sympathetic outflow (sympatholytic) there by suppressing the hemodynamic changes induced by the CO2 pneumoperitoneum.

Many studies have been done on laparoscopic surgery and highlighted hemodynamic changes during pneumoperitoneum and also proved by endocrine co relates the reason for the pneumoperitoneal response. The landmark study which was conducted about the hemodynamic response of laparoscopy with CO2 pneumoperitoneum by Jean L. Loris et al,[5] showed that there is significant reduction of cardiac output and increase in mean arterial pressure and systemic vascular resistance. In the present study we also observed that systolic, diastolic and mean arterial pressure increased abruptly after induction of pneumoperitoneum and this response sustained during the entire pneumoperitoneum period in the control group (Group A) as observed by the previous studies.

Dexmedetomidine being a anxiolytic, sedative and sympatholytic effectively suppresses the stress response in various situations. In the study by Poonam S.Ghodki et al,[6] studied dexmedetomidine as an anaesthetic adjuvant in laparoscopic surgery and they concluded dexmedetomidine is an effective adjunct withoutthe fear of awareness under anaesthesia and resulted in 62.5% reduction in induction dose of propofol and 30% less end tidal isoflurane required. Similarly Clonidine which is congener of dexmedetomidine has been studied in attenuating the stress response in laparoscopic surgeries.

In the study by Jean L. Loris et al,[5] that found that Clonidine effectively attenuated the stress response due to pneumoperitoneum. Similarly in our study we found that in the dexmedetomidine group (group B) hemodynamic responses to the induction of pneumoperitoneum were effectively blunted and the heart rate and blood pressure levels when compared to the control group (group A). Even though study conducted by D.Jee et al,[7] studied the effect of magnesium sulphate on pneumoperitoneum response showed that there is no change in heartrate in the magnesium group when compare to control group. But Jean L,Loris showed that Clonidine significantly reduced both heart rate and blood pressure. Our study reports also show the same that dexmeditomidine

attenuates both heart rate and blood pressure significantly.

Loris et al,[5] in his study showed the endocrine correlation of pneumoperitoneal response. The study showed that the reason the increase in the peripheral vascular response is due to increase in vasopressin and catecholamines levels. More precisely the study showed that vasopressin levels correlated closely with changes in peripheral vascular response. Induction of pneumoperitoneum causes rapid and marked release of vasopressin and it well co related with changes in intra abdominal pressure, intrathoracic and right atrial pressure. The reason for release of vasopressin is not clearly known and it may be probably due to mechanical stimulation of peritoneal receptors. Catecholamines particularly noradrenaline which was released during pneumoperitoneum also contributes to increase in peripheral vascular resistance. The stimulus for the release of noradrenaline is not known. The reason may be due to surgical stress induced by the pneumooeritoneum in laparoscopy.

Prolonged intraoperative increase of 20 mm of Hg or more in mean arterial pressure can cause significant implications in cardiovascular system. It can cause increase incidence of myocardial ischemia, infarction and death. Soby attenuating these responses in laparoscopy surgeries, dexmedetomidine may be of immense use in decreasing the morbidity in high risk cardiac patients. Ours study concludes that dexmedetomidine can definitely be used for the attenuation of the hemodynamic responses arising due to CO2 pneumoperitoneum.

Intubation, pneumoperitoneum and extubation during general anesthesia are all harmful stimulus, which can cause a strong stress response. This can lead to increasing the concentration of catecholamines such as epinephrine and norepinephrine in the blood and make the HR and blood pressure elevate,[8] which causes a series of complications such as myocardial ischemia, arrhythmia and cerebrovascular accident in patients with cardiocerebrovascular diseases.[9] Intravenous application of dexmedetomidine in the perioperative period can inhibit the release of epinephrine and norepinephrine by activating the receptors in the medullary vasomotor center, thus reduce catecholamine level in the blood by more than 50%, which is beneficial to keep intraoperative hemodynamic stability.[10,11] Previous study found that continuous infusion of dexmedetomidine 0.2 μg/kg/h or 0.4 μg/ kg/h from 15 min before induction to the end of surgery could reduce the stress response during intubation, pneumoperitoneum and extubation, and the latter was better for maintaining hemodynamic stability with no significant changes in the incidence of bradycardia and hypotension.[8] A single dose of dexmedetomidine 0.5μg/kg or 0.75μg/kg administered before induction of anesthesia can also reduce the stress response during intubation, and there was no significant difference between group 0.5 and group 0.75. However, the incidence of bradycardia and hypotension was significantly higher in 0.75 μg/kg group than that in 0.5μg/kg group.[9,12] Before the end of the operation, intravenous infusion of dexmedetomidine can alleviate the fluctuation of HR and blood pressure during extubation, and the effect is the best at the dose of 0.5μg/kg with the lowest incidence of bradycardia.[13,14,15] The results of this study showed that intravenous infusion dexmedetomidine 0.4μg/kg before induction could not effectively inhibit the stress response, but dexmedetomidine 0.6μg/kg and 0.8μg/kg could effectively restrain the intubation reaction, attenuate the intraoperative stress response, and maintain the hemodynamic stability.

CONCLUSION

We conclude that intravenous administration of dexmedetomidine as an adjunct before induction of CO2 pneumoperitoneum in laparoscopic cholecyctectomy effectively attenuates the arterial pressure increase arising due to pneumoperitoneal response by suppressing central sympathetic outflow.

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