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A COMPARATIVE STUDY OF ADDITION OF CLONIDINE AND DEXMEDETOMIDINE TO EPIDURAL ROPIVACAINE IN LOWER ABDOMINAL AND LOWER LIMB SURGERY

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ABSTRACT The present study was conducted on 150 patients of ASA I and II physical status, of either sex and aged 20-60 years, scheduled for elective lower abdominal and lower limb surgery to compare effects of clonidine and dexmedetomidine as adjuvant to epidural 0.5%ropivacaine on the sensory and motor block characteristics, post-operative analgesia, sedation and adverse effects. The demographic profile of all patients viz. age, sex, body weight, height, ASA grade and duration of surgery did not show any statistical significance ($p>0.05$). The time of onset of sensory block at T10, time for complete motor block, time to maximum sensory block, time for regression to bromage 5, total duration of analgesia and sedation profile were superior in the dexmedetomidine group than in the clonidine group or ropivacaine alone group. To conclude, dexmedetomidine is superior to clonidine as an adjuvant to epidurally administered local anaesthetic.

KEYWORDS : Ropivacaine, Dexmedetomidine, Clonidine

Introduction:

Epidural administration of a local anaesthetic agent (LAA) is widely practiced technique for surgical anaesthesia in lower abdominal and lower limb surgeries.[1] In order to avoid the harmful effects of administering large volumes of LAA via epidural route to achieve optimal anaesthetic effect, use of adjuvant agents has been recommended. Several adjuvants have been used with epidurally administered LAA for the prolongation of intraoperative and postoperative analgesia and to mitigate the shortfall and adverse effects of LAA.[2] α-2 adrenergic receptor agonists have been used as adjuvants in regional anaesthesia due to their sedative, analgesic, sympatholytic, anaesthetic-sparing and haemodynamic-stabilizing properties.[3,4] Clonidine and dexmedetomidine both are α 2-adrenoceptor agonists, with dexmedetomidine having an affinity eight times greater than that of clonidine.[5] Ropivacaine is less lipophilic than bupivacaine and results in a relatively reduced degree and duration motor blockade. The present prospective, double-blind, randomized study was conducted to compare the haemodyanamic, analgesic and sedative effects of clonidine and dexmedetomidine when combined with epidurally administered Ropivacaine in lower abdominal and lower limb surgery.

Methods:

This prospective, double-blind, randomized controlled study was conducted from October 2010 to June 2012. Prior to commencing study, approval was obtained from hospital ethical research committee. After taking informed and written consent, 150 patients of ASA class I and II physical status between 20-60 years of age, of either sex, scheduled for elective lower abdominal and lower limb surgery were enrolled. Patients who had history of hypersensitivity to amide local anaesthetics, heart disease (heart block, bradyarrythmia, left ventricular failure, fixed cardiac output disease), local infection at injection site, coagulation or neuropsychiatric disorders, morbid obesity, pregnancy, spinal deformity and uncontrolled diabetes mellitus were excluded from the study. The patients were randomly divided by means of computer-generated table of random numbers into 3 groups, viz. RS, RC and RD, each group comprising 50 patients.

A day before surgery, a detailed pre anesthetic checkup was carried out. All participants were premedicated with oral Alprazolam 0.5 mg and oral ranitidine 150 mg on the evening before surgery and on the morning of surgery. All patients were kept fasting as per standard recommendations. On the day of surgery, intravenous (i.v.) access was established using a 18 gauge cannula and patients were preloaded with ringer lactate solution 10 ml/kg over 20 min. Standard monitoring devices such as non-invasive blood pressure, pulse oximetry (SpO2) and continuous ECG monitoring was instituted and the baseline parameters were noted. After positioning and skin preparation, epidural catheter was inserted at L3-4 intervertebral space using a 16 G Huber tipped Tuohy needle and was secured 3-4 mm into epidural space. After confirmation of correct placement of epidural catheter, the study drugs were administered. The study drugs were prepared by an anaesthesia technician who was not aware of the randomization of study groups.

Group RS (n=50): Received lumbar epidural block with 20 ml of Ropivacaine (0.5%) plus 1 ml of 0.9% saline.
Group RC (n=50): Received lumbar epidural block with 20ml of ropivacine (0.5%) and clonidine (1μg/kg) made upto 1 ml.
Group RD: Received lumbar epidural block with 20 ml of ropivacaine (0.5%) and dexmedetomidine(1μg/kg) made upto 1 ml.

All durations were calculated considering the time of epidural injection as zero. Cardiovascular parameters such as heart rate (HR) and systemic blood pressure (BP) were monitored continuously and recordings were made at 5 min,10 min. 15 min, 20 min and 30 min; then at 30 min-intervals upto 120 min and finally at 60 min-intervals till complete recovery from anesthesia. Intraoperative hypotension was taken as systolic blood pressure < 90 mm of Hg or 20% below baseline value and any such episode was treated with administration of oxygen, a bolus of 250 ml of Ringer's lactae solution over 10 min and intermittent doses of i.v. injection of mephenteramine (5 mg). Bradycardia or HR <50 beats per minue (bpm) was treated with incremental doses of i.v. injection of 0.3 mg atropine. Intraoperative nausea was treated with i.v. injection of 10 mg metoclopramide. Shivering was treated with injection tramadol 50 mg i.v. Patients were also monitored for any other side effects like rigor, itching, nausea, dry mouth, post dural puncture headache, dizziness, respiratory depression etc. during the perioperative period.

Level of sensory blockade was tested by loss of pain sensation to a

pinprick in the midline using a 22G blunt hypodermic needle at intervals of 2- min for the first 20 min, and thereafter at intervals of 5-min until no change in level was observed. Onset of sensory block to T10 dermatomal level, peak level of sensory block, and duration of sensory block (2-segment regression) were recorded. The degree of motor blockade was assessed by Bromage scale (0-3) as follows: 0-no block, 1-inability to raise the extended leg, 2-inability to flex knee, 3-inability to flex ankle and foot.6 Time taken for onset of complete motor blockade and time for regression to Bromage scale 1 was also noted. Likewise, sedation was assessed by by a five-point scale: 1-alert and wide awake, 2-arousable to verbal command, 3-arousable with gentle tactile stimulation, 4-arousable with vigourous shaking, 5-unarousable.[7] Sedation scores were recorded at every 5-min for first 30 min, and thereafter every 15 min till the completion of surgical procedure. Analgesia was monitored by using by a 10-point verbal rating scale (VRS), where 0 and 10 represented no pain and worst possible pain respectively. VRS was recorded by an anaesthesiologist unaware of the allocation groups 5 min before epidural, at the start of surgery and then every 15 min interval till the surgery was over. Postoperatively, VRS was recorded half hourly for first 1 h then one hourly for 12 h and then three hourly for next 12 h till 24 h.

After completion of surgery, patient was shifted to post anaesthesia care unit. All the vital and haemodynamic parameters were recorded in the recovery room at 1 min, 5 min, 10 min, 20 min, 30 min, 60 min and 120 min, and thereafter every 4-hourly till 24 hours. Postoperatively sensory and block characteristics were assessed at 30 min interval s till 6 h. Time to first dose of rescue analgesia, number of doses of rescue analgesia and the time at which it was repeated was recorded in both groups. The time at which patient demanded first dose of rescue analgesia was the primary end point of this study, because at this time the effect of epidural block had weaned off. The postoperative pain was managed by top-up doses of 8 ml of 0.2% ropivacaine. Top-up doses were administered epidurally whenever the VRS was 4. Rescue analgesia was provided by injection Diclofenac sodium, 1.5 mg/Kg body weight, deep intramuscular, when patients complained of inadequate analgesia even after 3 successive top-up doses given 30 minutes apart.

At the completion of study, all the observations were compared statistically using one-way analysis of variance (ANOVA TEST) and chi-square tests as appropriate, and analyzed by Statistical package for social science (SPSS) version 16 for windows (SPSS Inc, Chicgo, Illinois, USA). p-values< 0.05 and <0.001 were considered significant and highly significant repectively.

Results:

There was no significant difference among the groups with respect to age, gender, weight, height, ASA grade, type of surgery and duration of surgery. (p >0.05). (Table 1)

Table 1: Demographic parameters

Demographic parameters	Group RS	Group RC	Group RD	p-value
Age (years) MeanSD	41.82±6.838	40.76±5.452	42.04±7.322	0.58
Gender (Male/Female)	39/11	30/20	37/13	0.12
Weight (Kg) MeanSD	59.285.544	56.766.321	58.986.781	0.09
Height (cm) MeanSD	157.064.863	155.284.027	156.725.213	0.14
ASA grade (1/2)	23/27	19/31	22/28	0.71
Type of surgery i. Abdominal hysterectomy	25	30	28	0.78
ii. Inguinal herniorrhaphy	20	16	15	
iii. Femoral intramedullary nailing	05	04	07	
Duration of surgey (min) MeanSD	143.8415.513	147.7014.965	140.6816.021	0.08

Mean Heart rate at preoperative and at different time intervals intra-operative and postoperative between the three groups were comparable and statistically not significant (p>0.05) (Table 2). Similary, the was no significant difference among the three groups in terms of sysytolic BP (Table 3) and diastolic BP (Table 4)and SpO2 (Table 5) at different time intervals.

Table 2: Data showing heart rate (HR) (beats/ min) of patients in various group at different intervals (mean ±SD)

Time interval	Group RS Mean±SD	Group RC Mean±SD	Group RD Mean±SD	F-value	p-value
HR Baseline	75.549.683	75.329.430	75.109.195	.027	0.973
HR 5 min	72.589.344	72.188.928	72.329.106	.025	0.976
HR 10 min	72.227.181	71.966.815	71.986.882	.022	0.979
HR 15 min	71.846.976	71.586.590	71.646.706	.020	0.980
HR 20 min	72.187.170	71.866.716	71.986.912	.027	0.973
HR 30 min	68.423.887	67.243.695	67.163.749	1.744	0.178
HR 60 min	68.524.595	67.064.038	67.184.360	1.746	0.178
HR 90 min	68.423.913	66.863.709	67.103.615	2.512	0.085
HR 120 min	68.645.054	67.164.582	66.843.976	2.219	0.112
HR 180 min	67.264.557	66.764.128	67.004.267	.167	0.846
HR 240 min	67.805.253	66.324.753	66.625.054	1.212	0.300

Table 3: Data showing systolic blood pressure (SBP) (mm Hg) of patients in various group at different intervals (mean ±SD)

Time interval	Group RS Mean±SD	Group RC Mean±SD	Group RD Mean±SD	F-value	p- value
SBP Baseline	130.58±10.851	129.12±8.555	128.90±10.124	0.338	0.714
SBP 5 min	122.88±11.339	123.12±11.166	119.98±10.347	1.021	0.363
SBP 10 min	120.55±12.910	117.18±11.307	116.20±11.108	1.496	0.228
SBP 15 min	117.22±13.041	114.42±11.417	110.88±17.200	2.038	0.135
SBP 20 min	115.48±13.347	111.25±10.760	111.20±10.550	1.783	0.173
SBP 30 min	113.45±5.593	110.98±5.981	111.18±7.404	1.861	0.160
SBP 60 min	114.95±7.049	112.98±4.638	112.02±8.176	1.935	0.149
SBP 90 min	113.55±17.878	109.45±16.083	108.30±16.184	1.088	0.340
SBP 120 min	114.30±6.513	111.85±6.747	112.22±7.343	1.473	0.233
SBP 180 min	116.68±8.266	117.62±9.513	115.88±8.213	0.407	0.667
SBP 240 min	118.08±7.346	119.55±8.797	118.62±7.648	0.351	0.705

Table 4: Data showing diastolic blood pressure (DBP) (mm Hg) in various groups at various intervals (Mean ± SD)

Time interval	Group RS Mean±SD	Group RC Mean±SD	Group RD Mean±SD	F -value	P -value
DBP at Baseline	79.55±5.715	77.48±5.213	78.85±5.582	1.470	0.234
DBP 5 min	76.48±6.594	73.38±5.415	74.98±5.512	2.795	0.065
DBP 10 min	74.18±7.121	74.58±5.368	71.60±5.541	2.838	0.063
DBP 15 min	71.10±7.510	72.20±6.533	68.80±5.893	2.699	0.071
DBP 20 min	69.20±7.144	70.88±7.261	67.70±5.393	2.279	0.107
DBP 30 min	68.78±7.156	69.25±6.743	66.08±5.456	2.783	0.066
DBP 60 min	67.40±3.053	67.18±4.690	67.08±3.482	0.077	0.926
DBP 90 min	69.48±5.023	68.12±6.077	67.12±4.931	1.930	0.150
DBP 120 min	70.38±4.418	68.80±4.214	68.22±4.577	2.553	0.082
DBP 180 min	73.75±4.011	73.22±4.185	71.85±4.270	2.229	0.112
DBP 240 min	74.05±4.529	72.32±3.964	71.80±4.998	2.717	0.070

Table 5: Data showing pulse oximetry values (SpO2) in various groups at various intervals (Mean ± SD)

Time interval	Group RS Mean±SD	Group RC Mean±SD	Group RD Mean±SD	F -value	p - value
SpO2 Baseline	100.00.000	99.601.979	99.801.414	1.014	.365
SpO2 5 min	100.00.000	99.601.979	99.801.414	1.014	.365
SpO2 10 min	100.00.000	99.601.979	99.801.414	1.014	.365
SpO2 15 min	100.00.000	99.801.414	99.801.414	.500	.608
SpO2 20 min	100.00.000	99.801.414	99.601.979	1.014	.365
SpO2 30 min	100.00.000	99.801.414	99.601.979	1.014	.365

SpO2 60 min	100.00.000	99.601.979	99.601.979	1.021	.363
SpO2 90 min	100.00.000	99.601.979	99.601.979	1.021	.363
SpO2 120 min	100.00.000	99.402.399	99.402.399	1.564	.213
SpO2 180 min	100.00.000	99.402.399	99.601.979	1.447	.239
SpO2 240 min	100.00.000	99.601.979	99.601.979	1.021	.363

Time taken for complete motor blockade (i.e. time to achieve Bromage score 3) in group RS (28.92±4.593 min) was greater than those in group RC (16.86±1.938 min) and RD (13.28±1.604 min), and the difference among the groups was highly significant (p <0.001). In group RD, time taken maximum for regression to Bromage scale 1 was greater (191.62±9.608 min) than that in group RC (179.06±5.586 min) and group RS (163.54±9.370 min), and the same was statistically significant (p <0.001). (Table 6). Similarly, time of onset of sensory block and time to achieve highest level of sensory blockade was lowest in group RD, while the time for two segment regression and regression to S1 segment of the sensory blockade was maximum in group RD group among the three groups, and the difference in various groups was statistically significant (p <0.001). (Table 6). The maximum height of sensory block achieved was also higher in group RD (p=0.024). The duration of postoperative analgesia was maximum in group RD (337.7824.543 min) among the three groups (p=0.001). Total consumption of epidural Ropivacaine (mg) for postoperative analgesia in 24 h was lowest in the RD group, and when compared with the RS group, the difference was highly significant (p<0.001). (Table 6).

Table 6: Data showing characteristics of motor and sensory blockade in various groups.

	Group RS	Group RC	Group RD	p-value		
				Gp RS vs Gp RC	Gp RS vs Gp RD	Gp RC vs Gp RD
Time for complete motor blockade (in min) (Mean ± SD)	28.92±4.593	16.86±1.938	13.28±1.604	<0.001	<0.001	<0.001
Time for regression to Bromage scale 1 (in min) (Mean ± SD)	163.54±9.370	179.06±5.586	191.62±9.608	<0.001	<0.001	<0.001
Time of onset of Sensory blockade (upto T10) (in min) (Mean ± SD)	17.88±2.569	16.30±1.940	14.04±1.726	<0.001	<0.001	<0.001
Maximum height of sensory block achieved (Thoracic level) T6 T7 T8	12 18 20	22 15 13	28 12 10	p=0.024		
Time to achieve highest level of sensory blockade (in min) (Mean ± SD)	25.70±4.082	17.22±2.621	14.46±2.276	<0.001	<0.001	<0.001
Time for two segment regression of sensory blockade (in min) (Mean ± SD)	111.00±4.886	120.14±4.408	126.16±6.361	<0.001	<0.001	<0.001
Time to sensory regression to S1 segment (min) (Mean ± SD)	258.1215.231	281.9310.484	312.7613.985	<0.001	<0.001	<0.001
Time to first rescue epidural top-up (min) (Mean ± SD)	279.4422.117	316.2319.664	337.7824.543	<0.001	<0.001	<0.001
Total consumption of Ropivacaine (mg) for postoperative analgesia in 24 h (Mean ± SD)	90.7618.443	79.4817.851	68.1315.933	0.004	<0.001	0.004

In group RS, all of the 50 patients had sedation grade 1, while in group RC, 16 patients had sedation grade 1 and rest 34 patients had sedation grade 2 . But in group RD, out of 50 patients, 41 patients and 9 patients had sedation scale 2 and 3 respectively. Hence the level of sedation among the patients who received epidural dexmedetomidine was statistically highly significant (p<0.001).(Table 7)

Table 7 : Sedation scores

Sedation scale	Group RS		Group RC		Group RD	
	No.	%	No.	%	No.	%
1	50	100	16	32	0	0
2	0	0	34	68	41	82
3	0	0	0	0	9	18
Total	**50**	**100**	**50**	**100**	**50**	**100**

(p = <0.001)

Adverse effects such as hypotension, bradycardia, dizziness, dry mouth, nausea and shivering were comparable among all the three groups and no statiscal signicance was noted. (p= 0.917). (Table 8)

Table 8: Side effects

Side effects	Group RS		Group RC		Group RD	
	No.	%	No.	%	No.	%
Hypotension	2	4	4	8	3	6
Bradycardia	0	0	0	0	1	2
Dizziness	1	2	2	4	2	4
Dryness of mouth	0	0	8	16	6	12
Nausea	2	4	1	2	1	2
Shivering	2	4	1	2	1	2

(p= 0.917)

Discussion:

Regional anesthesia has many advantages over general anesthesia and is associated with lower incidence of pulmonary and cardiovascular complications, better post operative pain management, lower incidence of deep vein thrombosis and pulmonary embolism.[8] Studies have shown that α2 adrenergic agonists viz. clonidine and dexmedetomidine have both analgesic and sedative properties when used as adjuvants in regional anaesthesia.[9,10,11] Dexmedetomidine is a highly selective α2 adrenergic agonist with an affinity eight times greater than that of clonidine. Studies have demonstrated that the anaesthetic and analgesic requirements are reduced by use of these two adjuvants due to their analgesic properties and augmentation of local anaesthetic effects.[12,13,14] Analgesia produced by α2 agonists occurs as a result of decreased release of C-fibre transmitters and hyperpol arization of postsynaptic dorsal horn neurons. It has been postulated that binding of α2 agonist agents to the dorsal horn motor neurons results in the prolongation of motor blockade of local anaesthetics.[15,16] When administered as an adjuvant to epidurally administered LA, α2 agonists produce sedation, analgesia, anxiolysis, and a decrease in sympathetic activity.[4] They also exhibit anaesthetic-sparing and haemodynamic-stabilizing effects.[17,18]

The present study was conducted on 150 patients of ASA I & II physical status of either sex between 20-60 years of age scheduled for elective lower abdominal and lower limb surgery to compare the perioperative analgesia, sedation and haemodynamic stability produced by clonidine and dexmedetomidine when administered with ropivacaine. The demographic profile of the patients was comparable with respect to age, sex, height, weight, ASA grade and type and duration of surgery. The study evidently indicates that addition of

dexmedetomedine (1µg/kg) as an adjuvant to epidural ropivacaine resulted in a better perioperative analgesia and motor blockade as compared to clonidine(1µg/kg). Dexmedetomidine as an adjuvant leads to early onset of analgesia, rapid achievement of highest level of sensory and motor blockade and prolongation of duration of postoperative analgesia. Moreover it provides an acceptable level of sedation without producing any undue haemodynamic changes. In addition, use of α2 agonists as adjuvants in epidural anaesthesia causes faster onset of sensory and motor blockade, prolonged duration of analgesia and a higher level of dermatomal sensory spread compared to those with epidurally administered ropivacaine alone. Previous studies that have used clonidine and dexmedetomidine as an adjuvant to LA in neuraxial blockade too support the results of the present study.[19,20] An earlier study by Salgado et al demonstrated that the duration of motor blockade was significantly increased in the dexmedetomidine group, being approximately more than 30% greater than that observed in the control group.[21]

The primary outcome of the present study was rapid onset and prolonged duration of sensory and motor blockade and prolonged duration of post-operative analgesia with addition of 1 µg/kg of clonidine and dexmedetomidine to epidural ropivacaine. The secondary outcome was decreased need of rescue analgesia in the post-operative period, without any significant adverse effects associated with use of α2 agonists as adjuvant to ropivacaine.

Bajwa et al had studied a comparative evalution of addition of dexmedetomidine and clonidine) to epidural ropivacain (0.75%) in 50 adult female patients between age of 44 and 65 years underwent vaginal hysterectomy, found that time of onset of sensory and motor blockade was minimum in dexmedetomidine group. Unlike our study, Bajwa et al found that dexmedetomidine as an adjuvant had better perioperative analgesia and sedative and anxiolytic properties. The more rapid onset of analgesia and higher dermatomal level of sensory analgesia found by Bajwa et al in comparison to the present study could be due to higher amount of dexmedetomidine (1.5 µg/kg) and clonidine (2 µg/kg) used in their study.[16] Ogun CO et al also showed that addition of these two adjuvants promotes faster onset compared to established time of onset of sensory analgesia with ropivacain alone.[22] EL Saied AH et al also showed that addition of these two adjuvants promotes faster onset compared to established time of onset of sensory analgesia with ropivacain alone.[23] Bajwa et al in an another study have shown that caudal block with 0.25% of isobaric ropivacaine combined with 2µg/kg of clonidine provided an efficient analgesia intraoperatively and a prolonged duration of post-operative analgesia.[24] Van elstraete AC et al (2000), in a study with patients submitted to hemorrhoidectomy under caudal epidural anesthesia, observed that mean time for first analgesic request was longer in the group that received clonidine plus LA as compared to the group that received LA alone.[25] Similar results have been observed by other investigators as well.[26,27]

Dexmedetomidine produces hypnotic and supraspinal analgesic effects by hyperpolarization of noradrenergic neurons which, in turn, results in decreased neuronal firing in the locus coeuleus, inhibition of norepinephrine release in the descending medullospinal noradrenergic pathway. The results of the present study demonstrate the effectiveness of epidural dexmedetomidine as an adjuvant to ropivacaine in providing sedation. Sedation scores were significantly higher in the dexmedetomidine group in comparison to the other two groups. 82% of patients had a sedation score of 2 and 18% of patients had a sedation score of 3 who were arousable to a verbal command or by a gentle tactile stimulation respectively.

The group RD showed a significant advantage over the RC group in terms of several post-operative sensory and motor blockade characteristics, viz. increased time for two segment regression of sensory blockade, time to sensory regression to S1 segment, time for regression to Bromage scale 1 and time to first rescue epidural top-up. The total consumption of ropivacaine (mg) for postoperative analgesia during the first 24 h was also less in the group RD in comparision to RC group. The haemodynamic and oxygenation parameters remained stable in all the three groups that confirms the established effects of α2 agonists on perioperative oxygenation and haemodynamic state in humans.[28,29] There was a slight decrease in heart rate and mean arterial pressure in the RC and RD groups in comparison to RS group, but the decrease was not more than 15% of their baseline values. There was no respiratory depression noted in any of the three groups, which is also supported by the multiple earlier studies.[30]

The most common adverse effects associated with use of α2 agonists as adjuvant in neuraxial blockade are bradycardia and hypotension. 2 patients in group RS, 4 patients in group RC and 3 patients in group RD had developed hypotension which responded to administration of IV fluids alone. None of these patients required injection mephen teramine. Bradycardia occurred only in one patient in the RD group, which was treated with atropine (0.3 mg, i.v.). These side effects, however, were not significant probably due to the small dose of epidural clonidine and dexmedetomidine used in the present study. An increased incidence of dryness of mouth was noted in in groups RC and RD, although it was not statistically significant and produced only mild discomfort to the patients. Other adverse effects such as dizziness or nausea were comparable between the groups. A slightly decreased, although statistically insignificant, incidence of post-operative shivering was seen in the groups RC and RD. α2 agonists act on central thermoregulatory system, and hence decrease the vasoconstri ction threshold and prevent the post-operative shivering.31 We observed that a little higher incidence of drymouth , nausea
etc are more in group 3 than group 2and group 1 and found to be statistically insignificant.
($p>0.05$).

As the present study contributes to the existing knowledge on α2 agonists, certain limitations must be taken into consideration. All the patients included in the study were ASA physical status I and II, as such caution must be exerted while generalizing the results to ASA physical staus III and IV patients. It was conducted on patients scheduled for lower abdominal and lower limb surgeries, and it is possible that the level of surgery might alter the perception of post-operative pain. Therefore further clinical studies are needed to determine the equivalent doses of dexmedetomindine and clonidine for different types of neuraxial blockade.

Conclusion:

Dexmedetomidine is a better alternative to clonidine as an adjuvant in epidural anaesthesia as it produces rapid onset and prolonged duration of sensory analgesia and motor blockade, prolonged post-operative analgesia stable haemodynamics and acceptable sedation levels. It reduces the requirement of rescue analgesia needed in the post-operative without any serious adverse effects.

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