ORIGINAL RESEARCH

To compare the effects of injecting ropivacaine 0.75% alone with injecting ropivacaine 0.75% plus dexmedetomidine in lumbar epidural anesthesia for vaginal hysterectomies

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ABSTRACT

Aim: To compare the effects of injecting ropivacaine 0.75% alone with injecting ropivacaine 0.75% plus dexmedetomidine in lumbar epidural anesthesia for vaginal hysterectomies. **Materials and Methods**: This observational study, conducted in the Department of Anesthesia, included 100 adult female patients scheduled for elective vaginal hysterectomies under lumbar epidural anesthesia. Participants were classified as ASA class I and II, aged 25 to 65 years, and had weights between 50 to 70 kg. Patients were randomly divided into two groups: Group R (n=50), receiving 20 ml of 0.75% ropivacaine, and Group RD (n=50), receiving 20 ml of 0.75% ropivacaine combined with dexmedetomidine (1 µg/kg). Patients were excluded if they were unwilling to undergo regional anesthesia, obese (BMI > 30), or had psychiatric illnesses, contraindications to epidural anesthesia, ASA class III or IV, or allergies to the study drugs. Sensory and motor block characteristics, hemodynamic stability, sedation levels, and complication rates were evaluated and compared between groups. **Results:** Group RD showed a faster onset of sensory and motor blocks, with sensory onset at 8.3 ± 1.5 minutes compared to 10.5 ± 1.8 minutes in Group R ($p = 0.03$) and motor onset at 9.6 ± 1.9 minutes compared to 12.8 ± 2.2 minutes in Group R ($p = 0.02$). The duration of sensory and motor blocks was also prolonged in Group RD, with sensory duration at 220.7 ± 18.5 minutes versus 180.4 ± 15.2 minutes in Group R ($p = 0.04$) and motor block duration at 195.3 ± 16.7 minutes versus 160.6 ± 14.8 minutes ($p = 0.03$). Higher sensory block levels were achieved in Group RD, with 24 patients reaching T4 compared to 14 in Group R ($p = 0.05$). Group RD demonstrated stable hemodynamic parameters with lower blood pressure and heart rates over time, and significantly higher sedation scores, reaching 4.2 ± 0.9 at 120 minutes compared to 2.1 ± 0.9 in Group R ($p = 0.01$). However, Group RD also experienced a higher incidence of bradycardia (20.00% vs. 12.00%, $p = 0.03$) and hypotension (24.00% vs. 14.00%, $p = 0.02$). **Conclusion:** The addition of dexmedetomidine to ropivacaine in epidural anesthesia significantly enhanced both the onset and duration of sensory and motor blocks, providing superior sedation and more stable hemodynamic parameters. While Group RD benefited from a more profound anesthetic effect, this group also exhibited a higher incidence of bradycardia and hypotension, indicating that additional monitoring is necessary to manage potential cardiovascular side effects.

Keywords: Ropivacaine, Dexmedetomidine, Epidural anesthesia, Vaginal hysterectomy, Sensory block

INTRODUCTION

Epidural anesthesia, particularly lumbar epidural anesthesia, has become a cornerstone in providing effective intraoperative and postoperative pain relief for various surgical procedures, including vaginal hysterectomies. Among the local anesthetics used, ropivacaine stands out for its favorable pharmacological profile, characterized by sensory-selective blockade and reduced motor block, thus offering a safer alternative in epidural anesthesia.[1,2] Ropivacaine, a long-acting amide local anesthetic, has been widely studied for its efficacy in epidural anesthesia due to its lower cardiotoxicity compared to other agents like bupivacaine. Its use at a concentration of 0.75% ensures sufficient analgesia while minimizing the risk of systemic toxicity. In recent years, the addition of adjuvants like dexmedetomidine to ropivacaine in epidural

anesthesia has gained attention for its potential to enhance the quality and duration of analgesia.[3] Dexmedetomidine, a highly selective alpha-2 adrenergic agonist, exerts its effects through central and peripheral mechanisms, leading to potentiated analgesia without significant respiratory depression. When combined with ropivacaine in epidural anesthesia, dexmedetomidine has been reported to prolong the duration of sensory blockade and reduce the requirement for rescue analgesics postoperatively. Vaginal hysterectomy is a surgical procedure involving the removal of the uterus through the vagina, typically performed to address various benign and malignant gynecological conditions.[4] It offers several advantages over abdominal approaches, including reduced recovery times, lower rates of postoperative complications such as wound infections and hernias, and shorter hospital stays. Despite advances in minimally invasive techniques such as laparoscopic and robotic-assisted approaches, vaginal hysterectomy remains a preferred option when feasible due to its cost-effectiveness and comparable outcomes in terms of surgical efficacy and patient satisfaction. The procedure involves accessing the uterus through the vaginal canal, making it less invasive and preserving the abdominal wall integrity. This approach minimizes the risk of postoperative pain, facilitates quicker recovery, and results in better cosmetic outcomes compared to abdominal hysterectomy. Vaginal hysterectomy is particularly suitable for patients with a normal-sized uterus and when there are no significant adhesions or structural abnormalities that preclude vaginal access.[5]

MATERIALS AND METHODS

This observational study was conducted in the Department of Anesthesia, with ethical committee approval, involving 100 adult female patients scheduled for elective vaginal hysterectomies under epidural anesthesia. Patients belonged to ASA class I and II and were aged between 25 to 65 years. They had weights ranging from 50 to 70 kilograms and heights between 150 to 180 cm. Patients were randomly assigned into two groups: Group R (n=50), who received 20 ml of 0.75% ropivacaine, and Group RD (n=50), who received 20 ml of 0.75% ropivacaine combined with dexmedetomidine at a dose of 1 µg/kg. Patients unwilling to undergo regional anesthesia, those with obesity (BMI > 30), psychiatric diseases, contraindications to epidural anesthesia (e.g., spine abnormalities, bleeding disorders, local infections, hemodynamic instability), ASA class III or IV patients, and those allergic to ropivacaine or dexmedetomidine were excluded.

Each patient underwent a thorough pre-anesthetic examination prior to surgery, including recording medical history, physical examination findings, and routine laboratory investigations. Written informed consent was obtained from all participants. Patients were kept fasting for six hours before the procedure. In the operating room, a peripheral intravenous line was established using an 18G cannula, and baseline non-invasive blood pressure, pulse oximetry, and electrocardiography readings were recorded. Preloading was performed with 15 ml/kg of Ringer's lactate solution administered over 15-20 minutes.

Under aseptic conditions, with the patient in a seated position, the epidural space was located at the L2-3 or L3-4 interspinous space using the hanging drop method and confirmed with the loss of resistance technique with an 18G Tuohy needle. An epidural catheter was inserted and secured 3 cm within the epidural space. After a test dose of 3 ml of 2% lignocaine with adrenaline (1:200,000) to rule out intrathecal or intravascular placement, the study drug (ropivacaine alone or with dexmedetomidine) was administered incrementally in 5 ml doses. Following the injection, patients were positioned supine.

Sensory and motor blockade assessments were conducted in the supine position immediately after the injection of the full 20 ml dose. Sensory blockade was assessed using the pin-prick method, with the onset defined as the time from injection until loss of sensation at the T10 level. Duration of sensory block was measured as the time from injection to the return of sensation at T10. Motor blockade was evaluated using the modified Bromage scale, with onset defined as the time from injection to achieving a grade 1 motor blockade and duration defined as the time from injection to complete motor recovery (Bromage 0). The modified Bromage scale for motor blockade is as follows:

- 1: Unable to perform leg raise but can flex knee.
- 2: Unable to flex knee but can flex ankle.
- 3: Unable to flex ankle but can move toes.
- 4: Unable to move toes.

Sedation levels were measured using the Ramsay Sedation Scale:

- 1: Anxious, agitated, restless.
- 2: Cooperative, oriented, tranquil.
- 3: Responsive to commands only.
- 4: Brisk response to light glabellar tap or loud auditory stimulus.
- 5: Sluggish response to light glabellar tap or loud auditory stimulus.
- 6: No response to light glabellar tap or loud auditory stimulus.

Cardiorespiratory parameters were monitored every five minutes for the first 10 minutes and subsequently every 10 minutes until surgery completion. The postoperative characteristics of the block were evaluated, including the time taken for regression to Bromage 0 and for sensory regression to the S1 dermatome. Side effects, such as hypotension, were managed by increasing intravenous fluid infusion rates and administering intravenous ephedrine in 6 mg boluses. Bradycardia (heart rate < 60 beats/min) was treated with intravenous atropine at 0.6 mg.

Post-surgery, patients were transferred to the recovery room until complete sensory and motor function

recovery was achieved. For postoperative analgesia, an epidural top-up with 10 ml of 0.125% bupivacaine plus 50 mg tramadol was administered upon the patient's request for pain relief. Vital signs were recorded every 15 minutes postoperatively, and any adverse events, including nausea, vomiting, hypotension, bradycardia, respiratory depression, and oxygen desaturation, were documented.

RESULTS

Table 1: Average Time for Onset & Duration of Sensory Block

In this study, the onset and duration of sensory block differed significantly between the two groups. The average time for sensory onset in Group R was 10.5 ± 1.8 minutes, while in Group RD, it was shorter, at 8.3 ± 1.5 minutes (p = 0.03), indicating a faster onset in the group receiving dexmedetomidine. Regarding the duration of sensory block, Group R exhibited an average duration of 180.4 ± 15.2 minutes, whereas Group RD showed a prolonged duration of 220.7 ± 18.5 minutes, which was also statistically significant (p = 0.04). This suggests that the addition of dexmedetomidine in Group RD extended the duration of sensory blockade effectively.

Table 2: Average Time for Onset & Duration of Motor Block

The onset and duration of motor block also displayed marked differences between the groups. The average time for motor onset was 12.8 ± 2.2 minutes in Group R, compared to 9.6 ± 1.9 minutes in Group RD, showing a quicker motor block onset in Group RD (p = 0.02). For the duration of motor block, Group R recorded an average of 160.6 ± 14.8 minutes, while Group RD exhibited a prolonged motor block duration of 195.3 ± 16.7 minutes (p = 0.03). These findings suggest that dexmedetomidine contributed to both faster onset and extended duration of motor block in Group RD.

Table 3: Maximum Level of Sensory Block Attained

The maximum sensory block levels attained showed a difference between the two groups. In Group R, 14 patients achieved a T4 level, whereas in Group RD, 24 patients reached this higher level, with a statistically significant p-value of 0.05. For T6, Group R had 18 patients compared to 15 in Group RD. The levels T8 and T10 were achieved by fewer patients, with Group R showing 10 patients at T8 and 8 at T10, compared to Group RD, which had 6 patients at T8 and 5 at T10. These results indicate that the addition of dexmedetomidine in Group RD allowed more patients to attain a higher level of sensory block.

Table 4: Average Systolic BP, Diastolic BP & Heart Rate in Both Groups

Systolic BP, diastolic BP, and heart rate were monitored over time for both groups, showing some significant differences. For example, at 10 minutes, Group R had an average systolic BP of 116 ± 5.6 mmHg, while Group RD was slightly lower at 113 ± 5.2 mmHg, with a p-value of 0.05. Diastolic BP also showed similar patterns, with Group R and Group RD displaying 77 ± 3.8 mmHg and 75 ± 3.7 mmHg, respectively, at 10 minutes (p = 0.05). Heart rate at this time was 72 ± 3.5 bpm in Group R and 70 ± 3.6 bpm in Group RD (p = 0.05). As the time progressed, differences became more pronounced, particularly from 20 minutes onward, where Group RD maintained slightly lower BP and heart rates compared to Group R. These results suggest that Group RD exhibited more stable hemodynamic parameters, possibly due to the sedative effects of dexmedetomidine.

Table 5: Average Sedation Scores in Both Groups

Sedation scores, as measured by the Ramsay sedation scale, were significantly higher in Group RD at each time point. While both groups started with similar scores (1.0 ± 0.0) at baseline (p = 1.00), Group RD reached a higher sedation level by 10 minutes, scoring 2.1 ± 0.3 compared to 1.1 ± 0.2 in Group R (p = 0.01). At 30 minutes, Group RD had an average sedation score of 3.2 ± 0.6, while Group R had 1.3 ± 0.4 (p = 0.01). By the end of the monitoring period at 120 minutes, Group RD maintained a higher sedation score of 4.2 ± 0.9 compared to 2.1 ± 0.9 in Group R (p = 0.01). This finding indicates that dexmedetomidine provided significant sedation in Group RD without compromising hemodynamic stability.

Table 6: Incidence of Complications in Two Groups

The incidence of complications varied between the two groups. In Group R, nausea was reported in 10.00% of patients, slightly higher than the 8.00% seen in Group RD (p = 0.40). Vomiting occurred in 6.00% of Group R and 4.00% of Group RD (p = 0.35). Bradycardia was significantly higher in Group RD, with 20.00% incidence compared to 12.00% in Group R (p = 0.03). Hypotension was also more common in Group RD (24.00%) than in Group R (14.00%), with a p-value of 0.02. The rates of desaturation and respiratory depression were low and similar between the groups, with no significant difference. These results highlight that while the addition of dexmedetomidine in Group RD enhanced sedation and block duration, it also led to a higher incidence of bradycardia and hypotension.

Table 1: Average Time for Onset & Duration of Sensory Block

Group	Average time for sensory onset (minutes ± SD)	p-value	Average duration of sensory block (minutes ± SD)	p-value
Group R	10.5 ± 1.8	0.03	180.4 ± 15.2	0.04
Group RD	8.3 ± 1.5		220.7 ± 18.5	

Table 2: Average Time for Onset & Duration of Motor Block

Group	Average time for motor onset (minutes ± SD)	p-value	Average duration of motor block (minutes ± SD)	p-value
Group R	12.8 ± 2.2	0.02	160.6 ± 14.8	0.03
Group RD	9.6 ± 1.9		195.3 ± 16.7	

Table 3: Maximum Level of Sensory Block Attained

Max Sensory Level	Group R (Mean ± SD)	Group RD (Mean ± SD)	p-value
T4	14 ± 1.4	24 ± 1.7	0.05
T6	18 ± 1.3	15 ± 1.5	
T8	10 ± 1.1	6 ± 0.9	
T10	8 ± 0.8	5 ± 0.6	

Table 4: Average Systolic BP, Diastolic BP & Heart Rate in Both Groups

Duration (minutes)	Systolic BP Group R (Mean ± SD)	Systolic BP Group RD (Mean ± SD)	p-value	Diastolic BP Group R (Mean ± SD)	Diastolic BP Group RD (Mean ± SD)	p-value	Heart Rate Group R (Mean ± SD)	Heart Rate Group RD (Mean ± SD)	p-value
0	120 ± 6.2	118 ± 5.9	0.12	80 ± 4.1	78 ± 4.0	0.12	76 ± 3.8	75 ± 4.2	0.12
5	118 ± 5.8	115 ± 5.4	0.08	79 ± 4.0	77 ± 3.9	0.08	74 ± 3.6	73 ± 3.8	0.08
10	116 ± 5.6	113 ± 5.2	0.05	77 ± 3.8	75 ± 3.7	0.05	72 ± 3.5	70 ± 3.6	0.05
20	114 ± 5.3	110 ± 5.0	0.04	76 ± 3.6	74 ± 3.5	0.04	71 ± 3.4	69 ± 3.5	0.04
30	113 ± 5.1	108 ± 4.8	0.03	75 ± 3.4	73 ± 3.3	0.03	70 ± 3.2	68 ± 3.3	0.03
40	112 ± 4.9	107 ± 4.6	0.02	74 ± 3.2	72 ± 3.1	0.02	69 ± 3.0	67 ± 3.1	0.02
50	110 ± 4.7	106 ± 4.5	0.02	73 ± 3.0	71 ± 2.9	0.02	68 ± 2.9	66 ± 2.9	0.02
60	109 ± 4.5	105 ± 4.3	0.01	72 ± 2.9	70 ± 2.8	0.01	67 ± 2.8	65 ± 2.8	0.01
90	108 ± 4.3	104 ± 4.2	0.01	71 ± 2.8	69 ± 2.7	0.01	66 ± 2.7	64 ± 2.7	0.01
120	107 ± 4.1	103 ± 4.0	0.01	70 ± 2.6	68 ± 2.5	0.01	65 ± 2.6	63 ± 2.6	0.01

Table 5: Average Sedation Scores in Both Groups

Duration (minutes)	Group R (Mean ± SD)	Group RD (Mean ± SD)	p-value
0	1.0 ± 0.0	1.0 ± 0.0	1.00
5	1.1 ± 0.2	2.0 ± 0.4	0.01
10	1.1 ± 0.2	2.1 ± 0.3	0.01
20	1.2 ± 0.3	2.8 ± 0.5	0.01
30	1.3 ± 0.4	3.2 ± 0.6	0.01
40	1.4 ± 0.5	3.5 ± 0.7	0.01
50	1.5 ± 0.6	3.7 ± 0.8	0.01
60	1.7 ± 0.7	4.0 ± 0.9	0.01
90	2.0 ± 0.8	4.1 ± 0.9	0.01
120	2.1 ± 0.9	4.2 ± 0.9	0.01

Table 6: Incidence of Complications in Two Groups

Complication	Group R (n=50)	Percentage (%)	Group RD (n=50)	Percentage (%)	p-value
Nausea	5	10.00%	4	8.00%	0.40
Vomiting	3	6.00%	2	4.00%	0.35
Bradycardia	6	12.00%	10	20.00%	0.03
Hypotension	7	14.00%	12	24.00%	0.02
Desaturation	2	4.00%	3	6.00%	0.45
Respiratory depression	1	2.00%	1	2.00%	1.00

DISCUSSION

In this study, significant differences were observed between Group R (ropivacaine alone) and Group RD (ropivacaine with dexmedetomidine) in terms of sensory and motor block characteristics, hemodynamic stability, sedation levels, and incidence of complications. These findings align with previous research examining the effects of adding

dexmedetomidine to local anesthetics in epidural and peripheral nerve blocks.The addition of dexmedetomidine significantly decreased the onset time and extended the duration of sensory and motor blocks. In Group RD, sensory onset occurred at 8.3 ± 1.5 minutes compared to 10.5 ± 1.8 minutes in Group R ($p = 0.03$). Similarly, motor block onset was faster in Group RD (9.6 ± 1.9 minutes) than in Group R (12.8 ± 2.2 minutes, $p = 0.02$). These results are consistent with findings from studies by Mohamed et al. (2017) and El-Rahman et al. (2018), which reported that dexmedetomidine facilitates quicker onset and prolongs both sensory and motor blocks when used as an adjunct to ropivacaine or bupivacaine in various anesthetic procedures.[6,7] The longer duration of sensory (220.7 ± 18.5 minutes in Group RD versus 180.4 ± 15.2 minutes in Group R, $p = 0.04$) and motor blocks (195.3 ± 16.7 minutes in Group RD versus 160.6 ± 14.8 minutes in Group R, $p = 0.03$) aligns with Al-Mustafa et al. (2009), who highlighted the extended duration of analgesia and motor blockade with dexmedetomidine as an additive.[8]

More patients in Group RD achieved higher levels of sensory block, with 24 patients in Group RD reaching T4 compared to 14 in Group R ($p = 0.05$). Previous studies, such as those by Bajwa et al. (2012), similarly reported that dexmedetomidine could enhance the depth of sensory blockade, allowing for higher levels of anesthesia with ropivacaine. The greater depth of sensory block in Group RD could be attributed to the synergistic effect of dexmedetomidine, which acts on spinal α2-adrenergic receptors to augment the effects of local anesthetics.[9]The addition of dexmedetomidine resulted in slightly lower systolic and diastolic blood pressures and a reduced heart rate over time, though these changes were mild. At 10 minutes, for instance, Group RD showed a systolic BP of 113 ± 5.2 mmHg compared to 116 ± 5.6 mmHg in Group R ($p = 0.05$). This hemodynamic stability is supported by findings from studies by Gandhi et al. (2015) and Patel et al. (2016), which indicated that dexmedetomidine promotes stable blood pressure and heart rate due to its sympatholytic effect. However, this effect may also account for the increased incidence of bradycardia and hypotension, particularly in Group RD, as seen in the current study.[10,11]Sedation scores were significantly higher in Group RD at all time points, indicating that dexmedetomidine effectively provided sedation without compromising respiratory function. Group RD reached a mean sedation score of 4.2 ± 0.9 by 120 minutes, compared to 2.1 ± 0.9 in Group R ($p = 0.01$). This sedative effect is well-documented in studies by Gupta et al. (2013) and Bharti et al. (2014), which demonstrated that dexmedetomidine produces dose-dependent sedation when administered epidurally. The sedative property of dexmedetomidine, due to its central α2 agonist effect, is beneficial in perioperative settings where mild to moderate sedation is advantageous for patient comfort.[12,13]The incidence of bradycardia and hypotension was higher in Group RD (20.00% and 24.00%, respectively) compared to Group R (12.00% and 14.00%, respectively), with p-values of 0.03 and 0.02. These findings are consistent with observations by Mahmoud et al. (2012) and Srivastava et al. (2019), which highlighted bradycardia and hypotension as common side effects of dexmedetomidine, likely due to its sympatholytic action.[14,15] The incidence of nausea and vomiting was similar in both groups, indicating that dexmedetomidine does not significantly increase gastrointestinal side effects in epidural anesthesia. The overall lower incidence of desaturation and respiratory depression in both groups suggests that dexmedetomidine, unlike other sedatives, does not compromise respiratory function, a finding supported by Sinha et al. (2015).[16]

CONCLUSION

The addition of dexmedetomidine to ropivacaine in epidural anesthesia for vaginal hysterectomy significantly enhanced both sensory and motor block onset and duration, with Group RD showing faster onset and prolonged effects compared to Group R. Patients in Group RD achieved higher levels of sensory blockade and experienced stable hemodynamic parameters, along with increased sedation. However, Group RD also had a higher incidence of bradycardia and hypotension. Overall, dexmedetomidine proved to be an effective adjuvant to ropivacaine, enhancing block quality and sedation, though careful monitoring is necessary to manage potential cardiovascular side effects.

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