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# EFFECT OF EPIDURAL CLONIDINE ON SPINAL ANAESTHESIA CHARACTERISTICS IN PATIENTS UNDERGOING GYNAECOLOGICAL SURGERIES: A RANDOMIZED CONTROLLED TRIAL

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**ABSTRACT** **Background:** Combined spinal–epidural (CSE) anaesthesia is widely employed in gynaecological surgeries for its rapid onset and extended postoperative analgesia. Clonidine, an α2-adrenergic agonist, has shown potential as an epidural adjuvant to enhance spinal block characteristics. **Materials and Method:** This randomized, double-blind controlled study evaluated the impact of epidural clonidine on sensory and motor block parameters in 60 ASA I–II female patients aged 25–60 years undergoing gynaecological procedures. Group C received 150 µg clonidine epidurally; Group S received normal saline. Both groups subsequently received 15 mg hyperbaric bupivacaine intrathecally. **Results:** Group C showed significantly prolonged analgesia (287.33 ± 20.79 min vs. 180.50 ± 17.83 min), faster onset of sensory (43.57 ± 5.56 sec) and motor block (62.20 ± 7.51 sec), and extended motor block duration (301.83 ± 22.91 min). Two-segment regression was delayed in Group C (183.00 ± 13.87 min vs. 102.50 ± 13.69 min). Sedation scores were higher in the clonidine group without evidence of over-sedation. Hemodynamic parameters remained stable across both groups, with minimal incidence of hypotension and bradycardia. **Conclusion:** Administration of epidural Clonidine provides longer duration of post-operative analgesia and has faster onset of sensory and motor blockade of spinal anaesthesia, longer duration for sensory regression of dermatomal levels, prolonged duration of motor block when compared to epidural saline without significant hemodynamic adverse effects in combined spinal epidural technique in patients undergoing gynaecological surgeries.

**KEYWORDS :** Epidural Clonidine, Combined Spinal–Epidural (CSE) Anaesthesia, Postoperative Analgesia

## INTRODUCTION

Regional anaesthesia offers a safe and economical method of achieving prolonged postoperative analgesia. Adequate pain control after surgery helps suppress autonomic, somatic, and endocrine stress responses, thereby enhancing recovery. Since no single agent has been found to selectively block nociception without side effects, multimodal pharmacological strategies are now routinely employed for postoperative pain management. [1]

The combined spinal–epidural (CSE) technique has seen a rise in clinical use due to its dual-action approach: a rapid onset of anaesthesia via intrathecal injection, coupled with the ability to extend or adjust the block through an epidural catheter. [2]

Clonidine is an α2 agonist that mediates its analgesic effect via the α2 receptor located post-synaptically on the dorsal horn of the spinal cord. Stimulation of the α2 receptor reduces afferent transmission of pain producing analgesia.[3]

This study aims to evaluate the role of epidural clonidine as an adjuvant in CSE anaesthesia for patients undergoing gynecological surgery, focusing on its effectiveness in enhancing block characteristics and postoperative pain relief.

## MATERIALS AND METHODS

This single-centre, prospective randomized controlled study conducted after approval from Institutional Review Board and following prospective trail registration. Written informed consent obtained from all patients. Female patients belonging to ASA grade I & II undergoing elective gynaecological surgery under general anaesthesia were considered. P atient were excuded if they met any of the following crieteria: refusal by patients for the procedure, patients for whom central neuraxial block is contraindicated, ASA III & IV patients, patients with known allergy to study drugs, Pregnant and breast-feeding women.

After obtaining personal informed consent from these patients, general demographic data regarding name, age, sex, height, weight and contact information were noted. All patients in the study underwent a detailed pre-anaesthethic evaluation, history regarding duration of disease and previous treatment modalities, general physical and systemic examination. Patients were randomly allocated to 2 groups Group C (Clonidine group) and Group S (Saline group) as per computer generated randomization table. The randomization scheme was generated by using https://www.sealedenvelope.com. Allocation concealment was done using sequentially numbered, opaque sealed (SNOSE) technique. After confirmation of adequate fasting status patients were shifted to Operation Theatre, intravenous access was placed, American society of anesthesiologists (ASA) monitors were connected and baseline vitals noted. Patients were preloaded with 500ml of Hartmann's solution. All patients received CSE anesthesia. GROUP C received injection clonidine 150 mcg, diluted to 5ml in Normal saline (NS) via epidural catheter 10 minutes before subarachnoid block (SAB). Group S: received 5ml NS 10 minutes before SAB. Lumbar puncture was performed and hyperbaric bupivacaine (15mg) was administered intrathecally 10 minutes after epidural injection.

### Parameters Measured

1. Onset of sensory blockade
2. Onset of motor blockade
3. Time taken for 2 segment regression
4. Duration of motor blockade
5. Duration of analgesia is defined as the duration of time (in minutes) starting from administering the block to the need for first rescue analgesia when VAS score is >4
6. Intraoperative and postoperative sedation score
7. Intraoperative and postoperative pain score
8. Intra operative hemodynamics
9. Adverse effects if any noted

Sensory block was assessed by loss to sensation of pin-prick using a sterile hypodermic needle. Motor block assessed using Modified Bromage scale[5]. Grade 0- full flexion of knees and feet, Grade 1- Just able to flex knees, full flexion of feet. Grade 2- Unable to flex knees, but some flexion of feet possible. Grade 3- Unable to move legs or feet. Pain was assessed using Visual Analogue scale (VAS)[6] (0: No pain, 2–4: Mild pain, 5–7: Moderate pain, 8–10: Worst pain). Sedation was assessed using Ramsay Sedation scale (RSS)[7] (1: Anxious or restless or both, 2: Co-operative, oriented and tranquil, 3: Responding to commands, 4: Asleep, brisk response to light, glabellar tap or auditory stimuli, 5: Asleep, sluggish response, 6: Asleep, unarousable).

Data was analyzed with SPSS® Version 28.0 (IBM SPSS statistics for Windows) software. The Qualitative data represented in the form of frequency and percentage. Associations between variables was

analyzed using Chi square test. Quantitative data represented using mean± SD. Unpaired t test was used to compare the mean difference between groups. P value <0.05 was considered statistically significant. Tables and graphs were created with the help of MS Word and MS Excel 2010.

The sample size was calculated to be 60 using www.openepi.com software. The sample size was calculated using duration of analgesia as primary outcome. Keeping power of study 80% and confidence interval 95%, sample size required was 28 in each group. We enrolled total 60 patients considering losses and dropout rates for better validation of results.

## RESULTS

All 60 patients enrolled completed the study. The demographic data were comparable.

**Table – 1 Demographics Data**

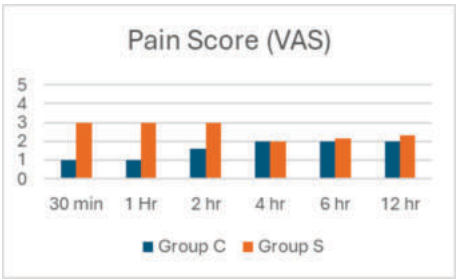
| Demographics | Group C | Group S | P |
|---|---|---|---|
| Age (years) | 47.37±2.76 | 47.93±2.35 | 0.39 |
| Height (cm) | 153.9±3.57 | 154.7±3.45 | 0.97 |
| Weight (Kg) | 60.17±7.19 | 57.17±6.32 | 0.09 |
| BMI (Kg/m2) | 25.42±3.06 | 23.99±1.89 | 0.07 |
| Duration of surgery (min) | 105±20.47 | 111±17.88 | 0.23 |

Onset of sensory block at L1 was much faster in Group C (43.57±5.56s) compared to Group S (57.67±3.14s) and was statistically significant. Onset of motor block was faster Group C (62.20±7.51 s) compared to Group S (95.20±12.38s) and was statistically significant. The time taken for 2 segment regression of sensory block was slower in Group C (183±13.87 min) compared to Group S (102.50±13.69 min) and was statistically significant. The highest sensory level achieved in Group C was T4 and in Group S was T6. Duration of motor blockade was more in Group C (301.83±22.91 min) compared to Group S (211.50±16.41 min). Duration of analgesia which was main criteria was more in Group C (287.33±20.79 min) compared to Group S (180.50±17.83 min) was statistically significant with p value of 0.001.

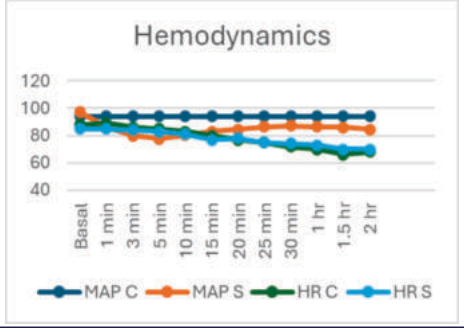
**Table – 2 Neuraxial Blockade Profile**

| Parameters | Group C | Group S | P |
|---|---|---|---|
| Onset of sensory block at L1 (s) | 43.57±5.56 | 57.67±3.14 | 0.001 |
| Time to Bromage 3 (s) | 62.20±7.51 | 95.2±12.38 | 0.001 |
| 2 segment regression time (min) | 183±13.87 | 102.5±13.69 | 0.001 |
| Duration of motor block (min) | 301.83±22.91 | 211.5±16.41 | 0.001 |
| Duration of analgesia (min) | 287.33±20.79 | 180.5±17.83 | 0.001 |

VAS scores were significantly lower after 1,2,4,6 hours in Group C compared to Group S (figure 1). Patients in Group C were well sedated and comfortable than in Group S (Graph 1).




Intraoperative hemodynamics such as heart rate, SBP, DBP, MAP and SpO2 were monitored in all patients. It was found that there were no significant changes between the two groups (Graph 2).




## DISCUSSION

This randomized controlled trial showed that administering epidural clonidine (150 µg) prior to the spinal anaesthesia in combined spinal–epidural anaesthesia in patients undergoing gynecological surgery significantly prolonged the duration of analgesia, facilitated a faster onset of both sensory and motor blockade, and enhanced postoperative comfort, without producing clinically significant hemodynamic instability.

Clonidine is an $\alpha_2$ agonist that mediates its analgesic effect via the $\alpha_2$ receptor located post-synaptically on the dorsal horn of the spinal cord. Stimulation of the $\alpha_2$ receptor reduces afferent transmission of pain producing analgesia[3,4,8]

The mean duration of analgesia in the clonidine group (287.33 ± 20.79 min) was markedly longer than in the saline group (180.50 ± 17.83 min; p = 0.001). This finding corroborates earlier work in of the study[9], who also reported a significant prolongation of analgesia in the clonidine group (299 ± 43.38 min vs. 152.50 ± 21.04 min; p < 0.001). Such consistency across trials reinforces the role of clonidine as a valuable adjuvant in regional anaesthesia. The observed prolongation of analgesia and block duration can be attributed to the synergistic interaction between clonidine and local anesthetics at both spinal and supraspinal levels. Clonidine, an α2-adrenergic agonist, reduces sympathetic outflow, enhances inhibitory interneuronal activity, and modulates nociceptive transmission, thereby intensifying and prolonging neuraxial blockade.

We observed a significantly faster onset of sensory block in the clonidine group (43.56 ± 5.56 s vs. 57.66 ± 3.14 s; p = 0.001). Similar findings were reported other studies[9,10], suggesting that α2-adrenergic agonists enhance the action of local anaesthetics by facilitating Aδ and C-fiber blockade[11]. The onset of motor block was also faster in our study with clonidine (62.2 ± 7.50 s vs. 95.2 ± 12.3 s) similar to one of the study.[10]

Clonidine significantly delayed two-segment sensory regression (183 ± 13.87 min vs. 102.50 ± 13.69 min), similar to findings in a previous study.[12] Furthermore, we observed prolonged motor block duration (301.83 ± 22.91 min vs. 211.5 ± 16.40 min), consistent with findings in study done by Gupta et al .[13] Taken together, these results strengthen the evidence that clonidine not only accelerates the onset but also sustains both sensory and motor blockade.

In terms of postoperative analgesia, patients receiving clonidine reported significantly lower VAS scores up to 6 hours postoperatively. This observation is in line with one study[9], indicating that clonidine provides superior early postoperative pain relief. Enhanced sedation, as reflected by higher Ramsay Sedation Scale scores, was also observed in our clonidine group, with patients remaining more comfortable compared to controls. The improved sedation scores may be explained by central α2 receptor-mediated effects in the locus coeruleus, promoting anxiolysis and comfort without excessive respiratory depression.

Hemodynamic stability was maintained in both groups. The incidence of hypotension (10% vs. 13%) and bradycardia (10% vs. 3%) was comparable and statistically insignificant. These findings align with study[14], who demonstrated that small doses of clonidine, even in combination with other agents, do not jeopardize cardiovascular stability. Clonidine stimulates alpha2 adrenoreceptors in the brain and spinal cord, resulting in reduction of sympathetic outflow from the central nervous system and in decreased in peripheral resistance, renal vascular resistance, plasma renin activity, heart rate, cardiac output, and blood pressure. Normal postural reflexes are intact; therefore, orthostatic symptoms are mild and infrequent.[15,16]

Our findings highlight the potential utility of clonidine as an adjuvant in gynaecological surgeries requiring regional anaesthesia. The prolonged analgesic duration and enhanced patient comfort may reduce the requirement for systemic opioids in the immediate postoperative period, thus minimizing opioid-related adverse effects. The maintenance of stable hemodynamics suggests that clonidine can be safely used in ASA I–II patients, though vigilance is warranted in higher-risk populations

### Limitations of Study

There are only a few literatures and studies that precisely show the mechanism of action of clonidine in epidural route and its action on

SAB by hyperbaric bupivacaine. Thus, more studies are needed to better understand the mechanism of action. Further studies with larger sample sizes, varied dosing regimens, and inclusion of higher ASA status patients are needed to validate the safety profile of clonidine.

## CONCLUSIONS

Epidural clonidine administered prior to spinal anaesthesia significantly enhances the quality of block by shortening onset times, prolonging sensory and motor blockade, and providing superior postoperative analgesia, while maintaining hemodynamic stability. Our results, in concordance with existing literature, support the incorporation of clonidine as a useful adjuvant in regional anaesthesia for gynaecological surgeries.

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**Conflicts of Interest**: Nil

## REFERENCES

[1] Sirvinskas E, Laurinaitis R. Use of magnesium sulfate in anesthesiology. Medicine 2002; 38: 147-150.
[2] Continuing Education in Anaesthesia Critical Care and Pain, Volume 7, Issue 2, April 2007, Pages 38-41, https://doi.org/10.1093/bjaceaccp/mkm003.
[3] Filos K.S., Goudas L.C., Patroni O., Polyzou V. Intrathecal clonidine as a sole analgesic for pain relief after cesarean section. Anesthesiology. 1992; 77:267–274.
[4] Parker RK, Connelly NR, Lucas T, Serban S, Pristas R, Berman E et al. Epidural clonidine added to a bupivacaine infusion increases analgesic duration in labor without adverse maternal fetal effects. J Anaesth. 2007; 21:142-7.
[5] Bromage PR. A comparison of the hydrochloride and carbon dioxide salts of lidocaine and prilocaine in epidural analgesia. Acta Anaesthesiol Scand Suppl. 1965;16: 55-69. doi:10.1111/j.1399-6576.1965.tb00523.x. PMID: 5322004
[6] Huskisson EC. Measurement of pain. Lancet. 1974 Nov 9;2(7889):1127-31. doi:10.1016/s0140-6736(74)90884-8.PMID: 4139420.
[7] Ramsay MA, Savege TM, Simpson BR, Goodwin R. Controlled sedation with alphaxalone-alphadolone. Br Med J. 1974 Jun22;2(5920):656-9.doi: 10.1136/bmj.2.5920.656. PMID: 4835444; PMCID: PMC1613102.
[8] Eisenach JC, De Kock M, Klimscha W. alpha(2)-adrenergic agonists for regional anesthesia. A clinical review of clonidine (1984-1995). Anesthesiology. 1996 Sep;85(3):655-74. doi: 10.1097/00000542-199609000-00026. PMID: 8853097.
[9] Prasad R, Rao RR, Turai A, Prabha P, Shreyavathi R, Harsoor K. Effect of epidural clonidine on characteristics of spinal anaesthesia in patients undergoing gynaecological surgeries: A clinical study. Indian J Anaesth. 2016 Jun;60(6):398-402.doi: 10.4103/0019-5049.183395. PMID: 27330201; PMCID: PMC4910479.
[10] Kanazi GE, Aouad MT, Jabbour-Khoury SI, Al Jazzar MD, Alameddine MM, Al-Yaman R, Bulbul M, Baraka AS. Effect of low-dose dexmedetomidine or clonidine on the characteristics of bupivacaine spinal block. Acta Anaesthesiol Scand. 2006 Feb;50(2):222-7. doi: 10.1111/j.1399-6576.2006.00919.x. PMID: 16430546.
[11] Guan M, Fanelli D, Verbeek T, Warfield DJ, Liu H. Alpha-2 Adrenergic Agonists. In First Aid Perioperative Ultrasound: Acute Pain Manual for Surgical Procedures. Springer International Publishing. 2023. p. 169-194 doi: 10.1007/978-3-031-21291-8_12.
[12] Sathesha M, Raghavendra Rao RS, Hassan SJ, Sudheesh K. Clonidine as a Sole Epidural Adjuvant in Combined Spinal-epidural: Clinical Study. Anesth Essays Res.2018 Apr-Jun;12(2):309-312. doi: 10.4103/aer.AER_18_17. PMID: 29962588; PMCID: PMC6020595.
[13] Gupta S, Raval D, Patel M, Patel N, Shah N. Addition of epidural Clonidine enhances postoperative analgesia: A double-blind study in total knee- replacement surgeries. Anesth Essays Res. 2010 Jul-Dec;4(2):70-4. doi: 10.4103/0259-1162.73510. PMID: 25885233; PMCID: PMC4173343.
[14] Förster JG, Rosenberg PH. Small dose of clonidine mixed with low-dose ropivacaine and fentanyl for epidural analgesia after total knee arthroplasty. Br J Anaesth. 2004 Nov;93(5):670-7. doi: 10.1093/bja/aeh259. Epub 2004 Sep 17. PMID: 15377579.
[15] Houston MC. Clonidine hydrochloride: review of pharmacologic and clinical aspects. Prog Cardiovasc Dis. 1981 Mar-Apr;23(5):337-50. doi: 10.1016/0033-0620(81)90020-7. PMID: 7012927.
[16] Robinson ES, Nutt DJ, Hall L, Jackson HC, Hudson AL. Autoradiographical and behavioural effects of a chronic infusion of antisense to the alpha2D-adrenoceptor in the rat. Br J Pharmacol. 1999 Oct;128(3):515-22. doi: 10.1038/sj.bjp.0702823. PMID: 10516627; PMCID: PMC1571666.