Original Research Article

Assessment of Effect of Epidural Clonidine on Characteristics of Spinal Anaesthesia in Patients Undergoing Gynaecological Surgeries

Deepak S[1], Jitin George[2], Mohsin Mohammed Ali[3], Ashwin S John[4*], R. C Krishna Kumar[5]

[1]Assistant Professor, PK DAS Institute of Medical Sciences, Vaniamkulam, Ottapalam, Kerala, India
[2]Assistant Professor, PK DAS Institute of Medical Sciences, Vaniamkulam, Ottapalam, Kerala, India
[3]Assistant Professor, PK DAS Institute of Medical Sciences, Vaniamkulam, Ottapalam, Kerala, India
[4]Assistant Professor, PK DAS Institute of Medical Sciences, Vaniamkulam, Ottapalam, Kerala, India
[5]Medical Director, PK DAS Institute of Medical Sciences, Vaniamkulam, Ottapalam, Kerala, India

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Abstract

Background: To assess the effect of epidural clonidine on characteristics of spinal anaesthesia in patients undergoing gynaecological surgeries. **Materials & Methods:** One hundred twenty patients belonging to American Society of Anesthesiologists physical status I and II who underwent gynaecological surgeries were randomly divided into 2 groups. Group I received clonidine (C) and group II received saline (S). Hyperbaric bupivacaine (15 mg) was administered intrathecally for both groups after epidural injection. Sensory and motor block characteristics, analgesia, sedation and haemodynamics were recorded in both groups. **Results:** Group I and group II had 60 females. The mean height in group I was 158.4 cm and inn group II was 157.3 cm. The mean weight in group I was 65.2 Kgs and in group II was 66.2 Kgs. Duration of surgery was 78.4 minutes in group I and 77.2 minutes in group II. The difference was significant (P< 0.05). Onset of sensory block at L1 (s) was 38.2 and 50.4, time to bromage 3 (s) was 55.4 and 102.3, time to 2 segment regression was 192.8 minutes and 109.3 minutes, total duration of analgesia was 312.4 minutes and 150.6 minutes and duration of motor blockage was 345.2 minutes and 124.7 minutes in group I and group II. The difference was significant (P< 0.05). VAS at 30 minutes was 1.5 in group I and 2.3 in group II, at 60 minutes was 1.4 in group I and 1.8 in group II, at 120 minutes was 2.6 in group I and 2.9 in group II, at 180 minutes was 2.1 in group I and 2.0 in group II and at 240 minutes was 1.5 in group I and 1.9 in group II. The difference was significant (P< 0.05). **Conclusion:** Administration of clonidine epidurally, 10 min before SAB, caused early onset and prolonged duration of motor blockade and analgesia.

Key words: clonidine, spinal–epidural (CSE) anaesthesia, haemodynamics

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Introduction

Combined spinal–epidural (CSE) anaesthesia offers a safe and inexpensive technique with the advantage of both spinal and epidural anaesthesia. It provides faster onset of surgical anaesthesia and prolongs the duration of post-operative pain relief. Various adjuvants further increase its efficacy.[1]

Clonidine being a partial α2 adrenergic agonist has antihypertensive effects and can potentiate effects of local anesthetics.[2] It acts by opioids-independent mechanism, stimulates α2 adrenoreceptors reducing central neural transmission in spinal neurons, and inhibits the release of substance-P.[3] It acts pre-synaptically interfering with nitric oxide mechanisms and protein kinases as well as by stimulation of cholinergic interneuron. Anaesthesia was prolonged when clonidine was added to local anaesthetics for peripheral nerve blocks.[4]

The analgesic effect of clonidine is more potent after neuraxial administration which points to a spinal site of action, thus favouring neuraxial (intrathecal or epidural) administration.[5] Epidural or intrathecal administration of clonidine potentiates the anesthetic action and reduces the dose requirement of volatile or injectable general or regional anesthetic agents with correspondingly fewer side effects.[6] Studies comparing effects of the two α2 agonists – dexmedetomidine and clonidine on spinal and epidural anaesthesia – have found that both produce a similar prolongation in the duration of the motor and sensory block with preserved haemodynamic stability and sedation.[7] Considering this, we performed present study to assess the effect of epidural clonidine on characteristics of spinal anaesthesia in patients undergoing gynaecological surgeries.

Materials & Methods

We selected one hundred twenty patients belonging to American Society of Anesthesiologists physical status I and II who underwent gynaecological surgeries. Ethical approval from ethical review committee of the institute was obtained. All enrolled patients gave their written consent for the participation.

Data such as name, age, gender etc. was recorded. Patients were randomly divided into 2 groups. Group I received clonidine (C) and group II received saline (S). All patients received CSE anaesthesia. Ten minutes before subarachnoid block (SAB), group I received clonidine 150 µg diluted to 5 ml in normal saline (NS) and group II received NS epidurally. Hyperbaric bupivacaine (15 mg) was administered intrathecally for both groups after epidural injection. Sensory and motor block characteristics, analgesia, sedation and haemodynamics were recorded in both groups. The results were compiled and subjected for statistical analysis using Mann Whitney U test. P value less than 0.05 was set significant.

*Correspondence
Dr. Ashwin S John
Assistant Professor, PK DAS Institute of Medical Sciences,
Vaniamkulam, Ottapalam, Kerala, India
E-mail: happyash13@gmail.com

Result

Table I Patients distribution

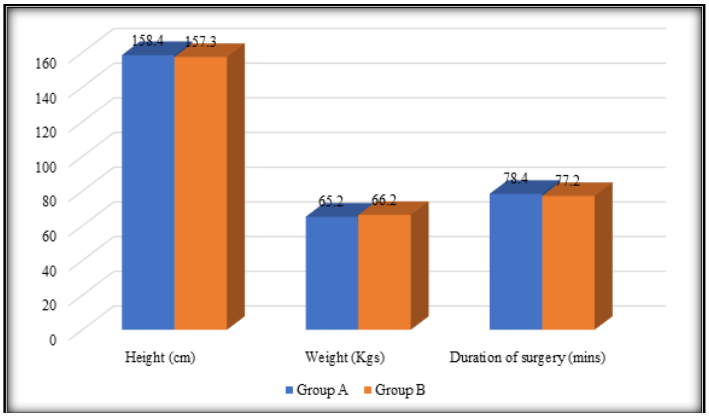
Parameters	Group A	Group B
Agent	Clonidine	Normal saline
Number	60	60

Group I comprised of 60 females and group II had 60 females (Table I).

Table II Demographic data

Parameters	Group A	Group B	P value
Height (cm)	158.4	157.3	0.82
Weight (Kgs)	65.2	66.2	0.94
Duration of surgery (mins)	78.4	77.2	0.95

The mean height in group I was 158.4 cm and inn group II was 157.3 cm. The mean weight in group I was 65.2 Kgs and in group II was 66.2 Kgs. Duration of surgery was 78.4 minutes in group I and 77.2 minutes in group II. The difference was significant (P< 0.05) (Table II, graph I).

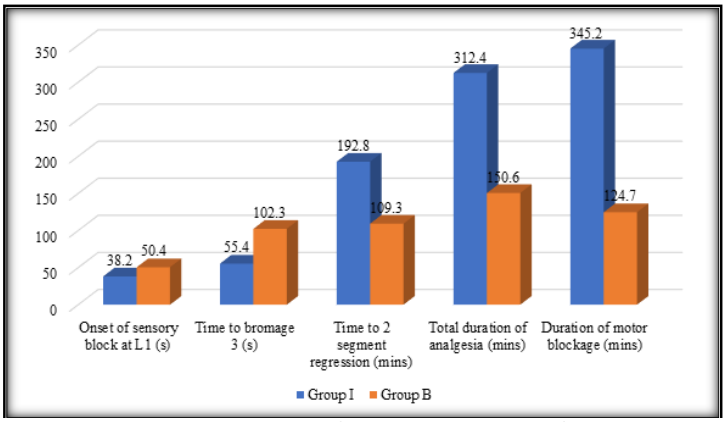


Graph I Demographic data

Table III Assessment of neuraxial blockade profile

Parameters	Group I	Group B	P value
Onset of sensory block at L1 (s)	38.2	50.4	0.04
Time to bromage 3 (s)	55.4	102.3	0.01
Time to 2 segment regression (mins)	192.8	109.3	0.05
Total duration of analgesia (mins)	312.4	150.6	0.02
Duration of motor blockage (mins)	345.2	124.7	0.01

Table III shows that onset of sensory block at L1 (s) was 38.2 and 50.4, time to bromage 3 (s) was 55.4 and 102.3, time to 2 segment regression was 192.8 minutes and 109.3 minutes, total duration of analgesia was 312.4 minutes and 150.6 minutes and duration of motor blockage was 345.2 minutes and 124.7 minutes in group I and group II. The difference was significant (P< 0.05) (Table III, Graph I).



Graph I Assessment of neuraxial blockade profile

Table IV Comparison of pain in both groups

VAS	Group I	Group B	P value
30 mins	1.5	2.3	0.04
60 mins	1.4	1.8	0.05
120 mins	2.6	2.9	0.05
180 mins	2.1	2.0	0.92
240 mins	1.5	1.9	0.03

Table IV shows that VAS at 30 minutes was 1.5 in group I and 2.3 in group II, at 60 minutes was 1.4 in group I and 1.8 in group II, at 120 minutes was 2.6 in group I and 2.9 in group II, at 180 minutes was 2.1 in group I and 2.0 in group II and at 240 minutes was 1.5 in group I and 1.9 in group II. The difference was significant (P< 0.05).

Discussion

inhibitory receptors) in the dorsal horn of the spinal cord.[8] This mimics the effects of noradrenaline which is released from the descending inhibitory pathways in the central nervous system.[9] Thus, decreased activity of the second-order neurons and wide dynamic range neurons in the dorsal horn occurs which in turn attenuates the input from peripheral nociceptive Aδ and C fibres. It does not affect proprioception or produce motor blockade.[10] Studies in rats show that clonidine partially inhibits voltage-gated sodium and potassium channels and suppresses generation of action potentials in tonic firing spinal dorsal horn neuron.[11] We assessed the effect of epidural clonidine on characteristics of spinal anaesthesia in patients undergoing gynaecological surgeries.

Our results showed that group I and group II had 60 females. The mean height in group I was 158.4 cm and inn group II was 157.3 cm. The mean weight in group I was 65.2 Kgs and in group II was 66.2 Kgs. Duration of surgery was 78.4 minutes in group I and 77.2 minutes in group II. Prasad et al[12] evaluated the effect of epidural clonidine on characteristics of spinal anaesthesia for gynaecological surgeries. Epidural clonidine produced faster onset (37.83 ± 8.58 s in Group C compared to 50.33 ± 8.80 s in Group S, P = 0.001) and prolonged duration of sensory block (241.17±18.65 minutes in group C compared to 150.33±19.16 minutes in group S, P = 0.001). Time for two segment regression of sensory block was193.67 ± 19.82 min in Group C and 109.33 ± 18.56 min Group S (P < 0.001). The duration of analgesia was 299.00 ± 43.38 min in Group C and 152.50±21.04 min in Group S (P< 0.001). Haemodynamics and sedation scores were comparable between two groups.

We observed that onset of sensory block at L1 (s) was 38.2 and 50.4, time to bromage 3 (s) was 55.4 and 102.3, time to 2 segment regression was 192.8 minutes and 109.3 minutes, total duration of analgesia was 312.4 minutes and 150.6 minutes and duration of motor blockage was 345.2 minutes and 124.7 minutes in group I and group II. Anandani et al[13] compared the onset, duration of sensory and motor block, hemodynamic effects, post-operative analgesia, and adverse effects of dexmedetomidine and clonidine with hyperbaric 0.5% bupivacaine for spinal anesthesia. 60 patients belonging to ASA Grade 1 and 2 undergoing elective gynecological surgery under spinal anesthesia. The patients were allocated in two groups (30 patients each). Group bupivacaine + clonidine (BC) received 17.5 mg of bupivacaine supplemented 45 mcg clonidine and Group bupivacaine+dexmedetomidine (BD) received 17.5 mg bupivacaine supplemented 5 mcg dexmedetomidine. The onset time of sensory and motor level, time to reach peak sensory and motor level, the regression time of sensory and motor level, hemodynamic changes, and side effects were recorded. Patients in Group BD had significantly longer sensory and motor block time than patients in Group BC. The onset time to reach dermatome T4 and modified Bromage3 motor block were not significantly different between two groups. Dexmedetomidine group showed significantly less and delayed requirement of rescue analgesic.

We found that VAS at 30 minutes was 1.5 in group I and 2.3 in group II, at 60 minutes was 1.4 in group I and 1.8 in group II, at 120 minutes was 2.6 in group I and 2.9 in group II, at 180 minutes was 2.1 in group I and 2.0 in group II and at 240 minutes was 1.5 in group I and 1.9 in group II. Eisenach et al[14] showed that 160 µg clonidine decreases arterial blood pressure by 18% and reduces HR by 5–20% and concluded that epidural clonidine does not induce haemodynamic instability.

Conclusion

Administration of clonidine epidurally, 10 min before SAB, caused early onset and prolonged duration of motor blockade and analgesia.

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