European Journal of Molecular & Clinical Medicine

ISSN 2515-8260 Volume 10, Issue 1, Winter 2023

ORIGINAL RESEARCH

Severe Preeclampsia: Hemodynamic Effect Of Lumbar Epidural Anaesthesia

[1]Dr. Mohammad Ilyas, [2]Dr. Satyendra Uike, [3]Dr.Amit Jain, [4]Dr. Ajay Singh

[1,4]Assistant Professor, Department of Anaesthesia, Bundelkhand Medical College, Sagar, M.P., India
[2]Professor, Department of Emergency Medicine, Bundelkhand Medical College, Sagar, M.P., India
[3]Professor, Department of Anaesthesia, Netaji Subhash Chandra Bose Medical College, Jabalpur, M.P., India

Correspondence:
Dr Ajay Singh
Professor, Department of Anaesthesia, Netaji Subhash Chandra Bose Medical College, Jabalpur, M.P., India

Received: 09 November, 2022 Accepted: 13 December, 2022

ABSTRACT

Aim: To determine the hemodynamic effect of lumbar epidural anaesthesia in patients with Severe preeclampsia.

Material and methods: Following approval from the institutional ethics committee, 50 non-laboring ASA (physical status I or II, age 18-32 years, weight 44-68 kg) parturients carrying a singleton pregnancy and scheduled for elective caesarean section were included in our study, and written informed consent was obtained from each parturient in their own language. There were 25 normotensive women (Group I) and 25 severe pre-eclamptic women (Group II) with blood pressures of 160/110 who needed antihypertensive medication (either nifedipine, 10–20 mg BD or TDS, or labetalol, 800–1200 mg in 2 to 3 divided doses).

Results: Parturients in both groups were equivalent in terms of age, weight, height, and foetal gestational age. The pre-eclamptic group had greater baseline SBP, DBP, and MAP. The mean baseline HR was similar in both groups. Following SAB, SBP, DBP, and MAP all declined from baseline in both groups, but the lowest recorded SBP, DBP, and MAP in the normotensive group were lower than in the parturients with pre-eclampsia, which was statistically significant. The pre-eclamptic group consumed considerably less phenylephrine than the normotensive group. The percentage of decline in DBP and MAP estimated from the baseline was likewise lower in the pre-eclamptic group (34% and 32% in normotensive, respectively, against 30% and 32% in pre-eclamptics). Pre-eclamptics required considerably less phenylephrine to cure hypotension (150.99±61 g in normotensive patients vs 50.11±22.36 g in pre-eclamptics), which was statistically significant (P <0.0001).

Conclusion: In conclusion, hypotension after spinal anaesthesia for caesarean delivery was much lower in severe pre-eclamptics than in healthy pregnant women in the current research. Furthermore, pre-eclamptic parturients had lower phenylephrine needs, and newborn outcomes were equivalent across the two groups.

Keywords: hemodynamic, lumbar epidural anaesthesia, Severe preeclampsia.

INTRODUCTION

After 20 weeks of pregnancy, a previously normotensive woman may develop preeclampsia, a multisystem illness defined by new-onset hypertension (systolic blood pressure 140 mm Hg and/or diastolic blood pressure 90 mm Hg) and proteinuria >300 mg/24 h.[1] When proteinuria is more than 5 gm/24 hr and the systolic blood pressure is greater than 160 mm Hg or the diastolic blood pressure is greater than 110 mm Hg, then severe preeclampsia has developed.[2] Preeclampsia may occur in up to 7.6% of all pregnancies worldwide and in up to 21% of all cases involving twins.[3] About 7.6% of pregnancies are nulliparous, and preeclampsia is more prevalent in black women than in Caucasian women, in addition to being more common in twin pregnancies, chronic hypertension, multi-fetal pregnancies, advanced maternal age (>35 years), and obesity. Preeclampsia risk increases with maternal weight, with a 4.3% morbidity rate at a BMI 19.8 and a 13.3% morbidity rate with a BMI >35 kg/m2.[4,5]

The fetoplacental unit is implicated in the pathophysiology of preeclampsia, with aberrant placentation and placental function serving as primary risk factors. Preeclampsia's consequences include uteroplacental hypoXia, an imbalance of angiogenic and antiangiogenic proteins, oxidative stress, endothelial dysfunction in the mother, and increased systemic inflammation. There is an increase in thromboXane A2 and activation of the coagulation cascade after severe vasoconstriction.[6] Pre-eclampsia patients continue to provide a challenge in terms of anaesthetic administration. General anaesthesia (GA) and spinal anaesthesia are also viable choices (SA). General anaesthesia is an option for women with pre eclampsia, although it increases the risk of maternal complications. Airway complications from edoema exacerbated by tracheal intubation and the presser reaction to laryngoscopy and intubation are additional concerns related with GA.[7]

Five percent to eight percent of all pregnancies are affected by preeclampsia. It causes severe morbidity and death in both mothers and their newborns, affecting 5-7% of all pregnancies.[8] In 2006, it was linked to 54 of the 569 maternal fatalities that occurred in the United States.[9] About 1% of births and 5% of pregnancies are complicated in Ethiopia due to pre-eclampsia. Preeclampsia and eclampsia were responsible for 16% of maternal death that was directly attributable to them and 10% of maternal mortality overall. Pulmonary edoema, abrupt renal failure, convulsion, DIC, headache, postpartum haemorrhage, HELLP syndrome, visual disturbances, lower respiratory tract infection, and congestive heart failure are among complications associated with severe pre-eclampsia.[10]

Because of the dangers of airway edoema, trouble with the airway or failed intubation, hypertensive reaction to direct laryngoscopy, and aspiration pneumonitis, determining the optimum anaesthetic approach for caesarean delivery in cases of severe preeclampsia is a challenge for anesthesiologists. Magnesium sulphate may interact negatively with neuromuscular blocking medications, calcium channel blockers, and inhalational anaesthetics. Conversely, regional anaesthesia has been linked to severe hypotension, high motor neuronal inhibition, and the potential of convulsions.[11]

MATERIAL AND METHODS

Following approval from the institutional ethics committee, 50 non-laboring ASA (physical status I or II, age 18-32 years, weight 44-68 kg) parturients carrying a singleton pregnancy and scheduled for elective caesarean section were included in our study, and written informed consent was obtained from each parturient in their own language. There were 25 normotensive women (Group I) and 25 severe pre-eclamptic women (Group II) with blood pressures of 160/110 who needed antihypertensive medication (either nifedipine, 10–20 mg BD or TDS, or labetalol, 800–1200 mg in 2 to 3 divided doses). The research excluded parturients with heart illness, chronic hypertension, renal disease, diabetes mellitus,

coagulopathy, and those who rejected SAB. All parturients were given oral ranitidine (150 mg) and oral metoclopramide (10 mg) two hours before surgery. Furthermore, antihypertensive treatment was maintained in pre-eclamptic parturients. Prehydration was performed with 10 ml/kg body weight of lactated ringer (RL) solution after IV access was established with an 18 G cannula. Baseline hemodynamic data (HR, SBP, DBP, MAP) were obtained using a standard multichannel monitor. The mean of three measurements obtained 5 minutes after entering the operating room and before any invasive operations were used to calculate baseline blood pressure.

SAB was delivered using a 26G -spinal needle at the L3-4 interspace in sitting position with 12.5 mg hyperbaric 0.5% bupivacaine after adequate asepsis and draping. To avoid aortocaval compression, the patient was put supine with a 10-cm wedge under the right buttock. The RL infusion was maintained at a rate of 5 ml/kg/h. Surgery was permitted after the top level of sensory block reached T4. Oxytocin 3 IU was administered over 5 minutes after the baby's head was delivered, and oxytocin infusion was maintained at 0.16 IU/min for 12-18 hours unless there was postpartum haemorrhage.

Following SAB, SBP, DBP, MAP, and HR were measured every 2 minutes for 30 minutes, then every 5 minutes until the procedure was completed. Hypotension (defined as MAP less than 30% of baseline) was treated with a 50 g phenylephrine IV bolus, which was repeated at 5-min intervals if necessary to keep MAP within 30% of baseline. Bradycardia (HR 60 beats/min) was treated with 0.6 mg IV atropine if it was coupled with hypotension (maximum 1.8 mg). Each patient's lowest SBP, DBP, and MAP were recorded, as were the lowest and highest HR readings. It was also recorded how much phenylephrine was ingested in total. The baby's Apgar score at 1 and 5 minutes, birth weight, and gestational age were also compared.

All of the patients had enough caesarean section block, and none of them had to be omitted from the research owing to insufficient block. All of the pre-eclamptic mothers were stabilised with antihypertensive treatment and showed no clinical signs of pulmonary edoema.

STATISTICAL INVESTIGATION

Data was entered into a Microsoft Excel spreadsheet, and the student test was used to find significant differences in means and the Chi-square test was used to discover differences in proportions. P 0.05 was deemed significant.

RESULTS

Parturients in both groups were equivalent in terms of age, weight, height, and foetal gestational age. The pre-eclamptic group had greater baseline SBP, DBP, and MAP. The mean baseline HR was similar in both groups. Following SAB, SBP, DBP, and MAP all declined from baseline in both groups, but the lowest recorded SBP, DBP, and MAP in the normotensive group were lower than in the parturients with pre-eclampsia, which was statistically significant. The percentage drop in DBP and MAP from the baseline was lower in the pre-eclamptic group compared to the normotensive group, albeit this difference was not statistically significant. The pre-eclamptic group consumed considerably less phenylephrine than the normotensive group. The percentage of decline in DBP and MAP estimated from the baseline was likewise lower in the pre-eclamptic group (34% and 32% in normotensive, respectively, against 30% and 32% in pre-eclamptics). Pre-eclamptics required considerably less phenylephrine to cure hypotension (150.99±61 g in normotensive patients vs 50.11±22.36 g in pre-eclamptics), which was statistically significant (P <0.0001).

Table 1 Basic profile of the patients

	Group 1	Group 2	P value
Age in years	26.85±3.69	29.85±4.85	0.44
Weight in kg	64.52±4.85	69.85±4.96	0.21
Height in meters	1.65 ± 0.03	1.66± 0.05	0.74
Gestational age in weeks	37.4 ± 0.74	37.8 ± 1.74	0.63

Table 2 Trend of SBP in two groups

Time in min	Group 1	Group 2
2	118	157
4	115	153
6	110	150
8	110	147
10	107	140
12	105	135
14	104	130
16	103	125
18	105	123
20	115	120
22	110	122
24	108	125
26	106	126
28	105	126
30	100	128

Table 3 Trend of DBP in two groups

Time in min	Group 1	Group 2
2	79	169
4	78	158
6	75	155
8	73	149
10	70	150
12	72	154
14	74	150
16	72	148
18	72	146
20	70	148
22	69	145
24	67	145
26	65	143
28	63	141
30	60	140

Table 4 Trend of MAP in two groups

Time in min	Group 1	Group 2
2	80	183
4	78	175
6	70	170

8	68	166
10	63	160
12	64	162
14	65	164
16	66	160
18	63	158
20	61	155
22	62	154
24	62	153
26	63	158
28	64	154
30	62	155

DISCUSSION

In the past, EA was favoured over SAB for pre-eclamptics having caesarean section owing to the risk of rapid and widespread sympathetic block in them after SAB, which might lead to serious hypotension, endangering both mother and foetus. Large amounts of intravenous fluids used to treat this hypotension were also a source of worry due to the risk of developing iatrogenic pulmonary edoema. Several writers have since evaluated the effectiveness of SAB and EA in severe pre-eclamptics and found equivalent hemodynamic effects in both groups with comparable foetal outcomes. Vishalputra et al.,[12]investigated the hemodynamic effects of SAB and EA in severe pre-eclamptics and found that bouts of substantial hypotension (SBP 100 mmHg) were brief in both groups with similar newborn outcomes. As a result, they determined that using SAB in severe preeclampsia was safe. Chiu et al.,[13] discovered comparable safety of SAB in pre-eclamptics in a 2004 research. Hood et al.[14] discovered that parturients with SAB needed more fluid than the epidural group, with no deleterious consequences like as pulmonary edoema. As a result of its simplicity, speed, cost efficiency, and block intensity, SAB was selected as an appropriate therapy to execute in severe pre-eclampsia.

In this research, we examined the hemodynamic impact and vasopressor demand following SAB in normotensive and severe pre-eclamptic caesarean section patients. Blood pressure fell in both groups when SAB was established, however the lowest SBP, DBP, and MAP recorded throughout the observation period were always greater in the pre-eclamptic group than in the normotensive group, which was a statistically significant difference (P< 0.0001). The percentage of decline in DBP and MAP estimated from the baseline was likewise lower in the pre-eclamptic group (34% and 32% in normotensive, respectively, against 30% and 32% in pre-eclamptics). Pre-eclamptics required considerably less phenylephrine to cure hypotension (150.99± 61 g in normotensive patients vs 50.11± 22.36 g in pre-eclamptics), which was statistically significant (P 0.0001).

The findings are consistent with those of Aya et al2003.[15] research, which compared the incidence and severity of hypotension and ephedrine use in 25 pre-eclamptics and 25 healthy parturients. They discovered that pre-eclamptics had 6 times less SAB-induced hypotension and needed much less ephedrine to manage it. Other research have shown comparable findings.[16,17]

Dyer et al.[18] employed beat-to-beat monitoring of cardiac output (CO) to examine the hemodynamic reactions to SAB for caesarean delivery in 15 severe pre-eclamptic parturients. It was also discovered that phenylephrine had an influence on CO. They found that the changes in CO following SAB were minor in severe pre-eclamptics, and the fall in MAP was rapidly reversed by phenylephrine without any loss in maternal CO.

This decreased frequency of hypotension following SAB in severe pre-eclamptics was first ascribed to the parturients analysed having a lower gestational age, a smaller uterine mass,

and less aortocaval compression. However, after the study of Aya et al.[19] which compared the incidence and severity of hypotension between severe pre-eclamptics (n = 65) and parturients with preterm pregnancy (n = 71) undergoing SAB for caesarean delivery and concluded that small uterine mass was unrelated to the hypotension, and the probable reason behind the less fall in blood pressure was altered vascular response primarily due to humoral factors, this hypothesis was rejected.

In normal pregnancy, increased production of endogenous vasodilators such as prostaglandins (PGs) and nitric oxide (NO) results in a vasodilated state, with an increased reliance on sympathetic vasoconstriction for vascular tone regulation. This explains their immediate and severe hypotension after sympathetic blockage caused by SAB.[19]

In pre-eclampsia, vascular endothelial damage develops, resulting in an increase in endogenous vasopressors such as thromboxane and endothelin, which are important for maintaining vessel tone. Sympathetic blockade after SAB has no effect on this vascular response, reducing the excessive drop in BP in pre-eclamptics.[20-22]

Normal pregnancy has decreased sensitivity to exogenous vasoconstrictors, requiring more vasopressors to reverse the hypotensive impact of SAB. Preeclampsia patients are more sensitive to vasoconstrictor medications, requiring less vasopressor.[16] Despite variable incidences of hypotension and the use of IV phenylephrine to treat it, we observed similar and satisfactory neonatal outcomes in both groups in terms of Apgar score at 5 minutes after delivery and baby birth weight.

CONCLUSION

In conclusion, hypotension after spinal anaesthesia for caesarean delivery was much lower in severe pre-eclamptics than in healthy pregnant women in the current research. Furthermore, pre-eclamptic parturients had lower phenylephrine needs, and newborn outcomes were equivalent across the two groups.

REFERENCES

1. Lambert G. et al., Preeclampsia: an update, ActaAnaesth. Belg. 65 (2014) 137–149. Suman Chattopadhyay et al. 2014.
2. Bartsch E., Park A.L., Kingdom J.C., Ray J.G., Risk threshold for starting low dose aspirin in pregnancy to prevent preeclampsia: an opportunity at a low cost, PLoS One 10 (2015), e0116296.
3. Hauth J.C., Ewell M.G., Levine R.J., Esterlitz J.R., Sibai B., Curet L.B. et al., Pregnancy outcomes in healthy nulliparas who developed hypertension. Calcium for preeclampsia prevention study group, Obstet. Gynecol. 95 (2000) 24–28.
4. Brown M.A., Lindheimer M.D., de Swiet M., Van Assche A., Moutquin J.M., The classification and diagnosis of the hypertensive disorders of pregnancy: statement from the international society for the study of hypertension in pregnancy (ISSHP), Hypertens. Pregnancy 1 (2001) ix–xiv.
5. Terrone D.A., Isler C.M., May W.L., agann E.F., Norman P.F., Martin Jr. J.N., Cardiopulmonary morbidity as a complication of severe preeclampsia, HELLP syndrome, J. Perinatol. 20 (2) (2000) 78–81.
6. Sahin S., Hypertension in pregnancy and preeclampsia, in: Painless Labour and Anaesthesia in Cesarean Section, 1stedn, 2006, pp. 133–144. Bursa: Nobel&Gunes.
7. Gofton E., Capewell V., Natale R., Gratton R., Obstetric intervention rates and maternal and neonatal outcomes of women with gestational hypertension, Am. J.Obstet. Gynecol. 185 (2001) 798–803.
8. Chattopadhyay S., Das A., Pahari S., Fetomaternal outcome in severe preeclamptic women undergoing emergency cesarean section under either general or spinal anesthesia,

J pregnancy. 2014.
9. Hall G.H., Noble W.L., Lindow S.W., Masson E.A., Long-term sexual cohabitation offers no protection from hypertensive disease of pregnancy, Humanit. Rep. 16 (2001) 349–352.
10. Khan K.S., Wojdyla D., Say L., Gülmezoglu A.M., Van Look P.F., WHOanalysis of causes of maternal death: a systematic review, Lancet 367 (2006) 1066–1074.
11. Heron MP, Hoyert DL, Murphy SL, Xu J. Deaths: final data for 2006. Natl. Vital Stat. Rep.; vol 57, No 14.
12. Visalyaputra S, Rodanant O, Somboonviboon W, Tantivitayatan K, Thienthog S, Saengchote W. Spinal versus epidural anesthesia for cesarean delivery in severe preeclampsia: A prospective randomized, multicenter study. Anesth Analg 2005;101:862-8.
13. Chiu IU, Mansor M, Ng KP, Chan YK. Retrospective review of spinal versus epidural anaesthesia for caesarean section in preeclamptic patients. Int J Obstet Anesth 2003;12:17-23.
14. Hood DD, Curry R. Spinal versus epidural anaesthesia for Caesarean section in severely preeclampticpatients. A retrospective survey. Anesthesiology 1999;90:1276-82
15. Aya AG, Mangin R, Vialles N, Ferrer JM, Robert C, Ripart J, *et al*. Patients with severe preeclampsia experience less hypotension during spinal anesthesia for elective cesarean delivery than healthy parturients: A prospective cohort comparison. Anesth Analg 2003;97:867-72.
16. Clark VA, Smith SG, Stewart AV. Ephedrine requirements are reduced during spinal anaesthesia for caesarean section in pre-eclampsia. Int J Obstet Anaesth 2005;14:9-13.
17. Ishrat HM, Raja AT. Spinal anaesthesia in pre-eclamptic parturient- prospective cohort study. Internet J Anaesthesiol 2007;14:ISSN1092-406.
18. Dyer RA, Piercy JL, Reed AR, Lombard CJ, Schoeman LK, James MF. Hemodynamic changes associated with spinal anaesthesia for caesarean delivery in severe pre-eclampsia. Anesthesiology 2008;108:802-11.
19. Aya AG, Vialles N, Tanoubi I, Mangin R, Ferrer JM, Robert C, *et al*. Spinal anaesthesia-induced hypotension: A risk comparison between patients with severe pre-eclampsia and healthy women undergoing preterm caesarean delivery. Anesth Analg 2005;101:869-75.
20. Santos AC, Birnbach DJ. Spinal anesthesia in the parturient with severe preeclampsia: time for reconsideration. Anesth Analg 2003;97:621-2.
21. Khalil RA, Granger JP. Vascular mechanisms of increased arterial pressure in preeclampsia: Lessons from animal models. Am J Physiol-Reg I 2002;283:R29-45.
22. Redman CW, Sargent IL. Pre-eclampsia, the placenta and the maternal systemic inflammatory response: A review. Placenta 2003;24 (Suppl A):S21-7