Original Article

Role of IVC collapsibility index to predict post spinal hypotension in pregnant women undergoing caesarean section. An observational trial

ABSTRACT

Background: Postspinal anesthesia hypotension (PSH) in pregnant women is common and may lead to poor maternal and fetal outcome. Fluid loading in pregnant women before spinal anesthesia to prevent hypotension is of limited ability. We hypothesized that those women who are hypovolemic before spinal anesthesia may be at risk of PSH and inferior vena cava collapsibility index (IVCCI) will be able to identify hypovolemic parturients.

Methods: In this prospective observational study, $n = 45$ women undergoing elective lower segment cesarean section with singleton pregnancy were recruited and IVCCI in left lateral tilt (with wedge) and supine position (without wedge) were noted by M-mode ultrasound (USG) before spinal anesthesia. After spinal anesthesia, changes in blood pressure were noted till 15 min after spinal anesthesia.

Results: USG measurements were obtained in 40 patients and 23 of 40 patients (57.5%) had at least one episode of hypotension. Area under the ROC curve of IVCCI with wedge to predict PSH was 0.46 (95% CI 0.27, 0.64) and best cut-of value was 25.64 with a sensitivity and specificity of 60.9% and 35.5%, respectively. Area under the ROC curve of IVCCI without wedge to predict PSH was 0.38 (95% CI 0.19, 0.56) and best cut-of value was 20.4 with a sensitivity and specificity of 69.6% and 23.5%, respectively.

Conclusion: We conclude that IVCCI is not a predictor of PSH in pregnant women undergoing elective cesarean section.

Key words: C- section; hemodynamic monitoring; IVC collapsibility index; post spinal hypotension; volume responsiveness

Introduction

Fluid therapy in the operating theatre (OT), intensive care unit (ICU), and emergency department (ED) have been the most challenging tasks for the clinicians as both hypovolemia and volume overload increase the morbidity and mortality. A fluid challenge study suggests that only 50% of hemodynamically unstable patients (in the OT, ICU, or ED) are volume responders, i.e., they will increase their stroke volume (SV) by >10--15%.[1]

Postspinal hypotension (PSH) is common in obstetric anesthesia practice, with an incidence of up to 71%.[2] PSH can occur precipitously and, if severe, can result in both maternal

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and fetal/neonatal adverse events.[3] Pregnant women with predelivery hypovolemia are at risk of cardiovascular collapse and the sympathetic blockade may severely decrease venous return. Hence, prevention of PSH is an essential element in obstetric anesthesia and fasting for aspiration prophylaxis may further add up to the hypovolemia for the patients not on maintenance fluids.[4]

Several approaches have been investigated to prevent PSH, notably fluid loading, vasopressors, or both.[3] However, the efficacy of fluid loading for preventing spinal hypotension has been called into question. The influence of timing of administration of fluids showed that coloading is more effective than preloading to prevent PSH.[4] In contrast to fluids, the use of vasopressors has gained popularity as the primary technique for the prevention and treatment of maternal hypotension.[3] After 15 min of spinal anesthesia, it is demonstrated that maternal cardiac output, heart rate (HR), and SV increases.[2] In addition, a marked reduction in systemic vascular resistance is observed, which suggests that arteriolar dilatation is at least as important as aortocaval compression in contributing towards PSH. Therefore, maintaining systemic vascular resistance, venous capacitance, and splanchnic venous tone are likely to be key factors in preventing a decrease in maternal cardiac output, thus preventing PSH.[2,5]

Hemodynamic monitoring in obstetric patients has evolved during the last decade, with the development of minimally invasive and noninvasive continuous cardiac output (CO) monitors.[6] Ultrasound (USG) is a method for noninvasive hemodynamic optimization in the ICU and ED, and it may be more helpful than other noninvasive methods.[7,8] Transabdominal USG measurements of inferior vena cava (IVC) are noninvasive and thus are not associated with complications.[6] USG of the IVC diameter is a useful and easy method for assessing a patient's volume status by calculating the IVC collapsibility index (IVCCI).[9]

IVC ultrasound has been found to be feasible in pregnant women.[10] Volume responsive patients as suggested by IVCCI may be more susceptible to PSH. We hypothesized that IVCCI more than 36% may be associated with PSH. This study was planned to find a correlation between IVCCI and PSH in pregnant women.

Methods

After obtaining permission form the Institute Ethics Committee and written informed consent from the participants, n = 45 full term (37 week completed single ton pregnancy) pregnant women undergoing elective lower segment cesarean section (LSCS) under subarachnoid block were included in this single-center prospective observational study. Patients who refused, posted for emergency LSCS, had associated cardiovascular, respiratory, renal diseases or who received preloading of intravenous fluid were excluded from this study.

Study protocol

Pregnant women planned for elective surgery were advised for nil per oral after midnight and started on iv fluids in the ward in morning hours. After detailed preanesthetic checkup, on arrival in operating room, standard monitoring devices were attached (pulse oximeter, noninvasive blood pressure and 3-lead electrocardiography). A curvilinear USG probe (3.5--5 MHz, Sonosite Turbo) was used to measure IVC diameter in supine position without wedge during inspiration and expiration. M-mode USG was used to measure the end-expiratory ($IVCd_{min}$) and end-inspiratory ($IVCd_{min}$) diameter of IVC. A 4-chamber cardiac view was obtained in subcostal hepatic window in transverse plane, and then probe was rotated anticlockwise to visualize IVC. IVC measurements were taken in end-inspiration and end-expiration, just proximal to the IVC and hepatic vein junction approximately 3--5 cm distal to the right by putting M-mode coupled to 2D mode [Figure 1]. A custom-made wedge with an angle of 15° (as measured by goniometer) was placed thereafter, and readings were again taken after 3 minutes. The IVCCI was estimated as IVCCI = $(IVCd_{max}\text{-}IVCd_{min})/(IVCd_{max})$. IVC measurements were taken by either of the two authors (RKA or YS) and both had experience of at least 100 cases of such measurements.

The patients were positioned in sitting position to give the spinal anesthesia at L3-L4/L2-L3 intervertebral level in

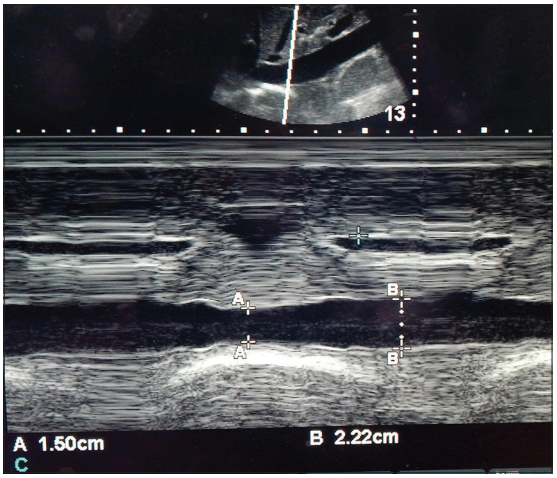

Figure 1: Ultrasound image (upper- 2D image, lower- M- mode) of IVC showing end-inspiratory and end-expiratory diameter

the midline approach. After local infiltration of skin and subcutaneous tissue with 2% lignocaine, 25 G Sprotte spinal needle was used to administer subarachnoid block (SAB) with 1.6 ml of hyperbaric bupivacaine (5%) and 20 μg of fentanyl after confirmation of free flow of cerebrospinal fluid (CSF) at the hub of the needle. Patients were coloaded with 10--12 ml/kg (over the period of 15 min) of Ringer's Lactate (RL) solution at the time of SAB. Thereafter, patients were placed in supine position with wedge under the right hip. HR, systolic blood pressure (SBP), diastolic blood pressure (DBP), mean arterial pressure (MAP), and SpO2 were recorded throughout the procedure every 3 min till 15 min of SAB. Level of sensory block was assessed by response to cold touch and surgery was allowed after sensory blocks reaches to T6 level. Hypotension (reduction in MAP more than 20% and/or MAP <65 mmHg) was treated with 6 mg of injection of ephedrine and a bolus of 250 ml of RL solution over 10 minutes. Number of boluses of ephedrine and fluids were recorded. Bradycardia (Heart Rate < 50 beats/min) was treated with 0.6 mg of injection atropine. Rest of the management was done as per standard protocol of this institute.

Data collection

IVC measurement data were collected by either of the two investigators (RKA or YS). Anesthesiologists who were not part of this study and blinded to the values of IVC diameter or IVCCI recorded all hemodynamic measurements.

Sample size estimation and plan for statistical analysis

No previous study has evaluated role of IVCCI in pregnant women to predict PSH; hence on pilot basis we have recruited $n = 45$ patients.

The normally distributed continuous variables (such as age, body weight, height, etc.) were expressed as mean and standard deviation (SD). Binary variables were expressed as absolute numbers and proportions. Independent sample *t*-test was used to compare normally distributed continuous variables in two groups and paired sample *t*-test was used to compare variables within the group. Receiver-operating characteristic (ROC) curves were constructed for IVCCI with wedge or without wedge as predictors of PSH and best cut-of values were obtained from Youden's index. All statistical analyses were conducted in STATA 12 software for Mac OS (*StataCorp. 2011. Stata Statistical Software: Release 12. College Station, TX: StataCorp LP*.).

Results

The study was conducted on $n = 45$ women and data of 40 pregnant women undergoing elective LSCS under SAB has been analyzed as it was not possible to visualize the IVC in rest of the women. Mean (SD) age of the women was 28.8 (4.5) years and mean (SD) period of gestation was 36.2 (1.9) weeks. Twenty-one out of forty-five were primiparous women and 24/45 were multiparous. Twenty-three out of forty patients (57.5%) had at least one episode of PSH (defined by reduction in MAP more than 20% and/or MAP <65 mmHg within 15 minutes of spinal anaesthesia). Comparison of baseline demographic and hemodynamic parameters in the patients who developed PSH ($n = 23$) and who did not have PSH ($n = 17$) has been depicted in Table 1. Baseline MAP was significantly higher in non-PSH pregnant women. HR was statistically similar in between the group ($P = 0.79$). IVC diameter at the end expiration ($IVCd_{max}$), end inspiration ($IVCd_{min}$) and IVCCI both with and without wedge in hypotensive and nonhypotensive pregnant women were statistically similar [Table 2]. Mean $IVCd_{max}$ was significantly higher with wedge than supine position [mean (SD) 1.36 (0.4) versus 1.26 (0.4); $P = 0.006$, paired sample *t*-test; Figure 2] but $IVCd_{min}$ was similar in those two positions [mean (SD) 0.95 (0.3) versus 0.90 (0.3); $P = 0.14$, paired sample *t*-test; Figure 2]. However, no difference was obtained in IVCCI in these two positions [mean (SD) 30.6 (12) versus 28.6 (13.3); $P = 0.35$, paired sample *t*-test]. $IVCd_{max}$ significantly lower without the placement of wedge (supine position) than with wedge in pregnant women who did not develop hypotension; but in women who had hypotension, it was statistically similar

Table 1: Comparison of baseline demographic and hemodynamic parameters in hypotensive (*n*=23) and non-hypotensive (*n*=17) patients

Parameters	All patients (*n*=40)	Hypotensive (*n*=23)	Non-hypotensive (*n*=17)	Significance
Age (years)	28.8 (4.5)	29 (4.1)	28.6 (5.2)	*P*=0.78
Body weight (kg)	64.9 (3.6)	64.5 (4.1)	65.6 (2.7)	*P*=0.36
Height (cm)	159.7 (3.6)	158.9 (4.0)	160.9 (2.5)	*P*=0.08
Period of gestation (weeks)	36.2 (1.9)	36.3 (1.6)	35.9 (2.3)	*P*=0.57
ASA PS (I/II)	27/13	16/7	11/6	*P*=0.75
Heart rate (beats per minute)	93.6 (15.6)	94.2 (14.1)	92.8 (17.8)	*P*=0.79
SBP	127.8 (18.1)	131.1 (20.4)	123.2 (13.6)	*P*=0.18
DBP	79.5 (11.7)	82.2 (10.5)	75.9 (12.6)	*P*=0.09
MAP	94.5 (13.8)	98.5 (14.1)	89.4 (11.8)	*P*=0.04

Table 2: Comparison of end-expiratory IVC diameter (IVCdmax), end- inspiratory IVC diameter (IVCdmin) and IVC collapsibility index (IVCCI) in hypotensive and non-hypotensive patients

Parameters	All patients ($n=40$)	Hypotensive ($n=23$)	Non-hypotensive ($n=17$)	Significance
With wedge				
$IVCd_{max}$	1.36 (0.4)	1.3 (0.4)	1.4 (0.3)	$P=0.31$
$IVCd_{min}$	0.95 (0.3)	0.92 (0.3)	0.99 (0.3)	$P=0.47$
IVCCI	30.6 (12)	29.6 (11.4)	31.9 (13)	$P=0.56$
Without wedge				
$IVCd_{max}$	1.26 (0.4)	1.24 (0.5)	1.29 (0.4)	$P=0.70$
$IVCd_{min}$	0.9 (0.3)	0.91 (0.4)	0.88 (0.3)	$P=0.81$
IVCCI	28.6 (13.3)	26.32 (13.3)	31.6 (12.3)	$P=0.22$

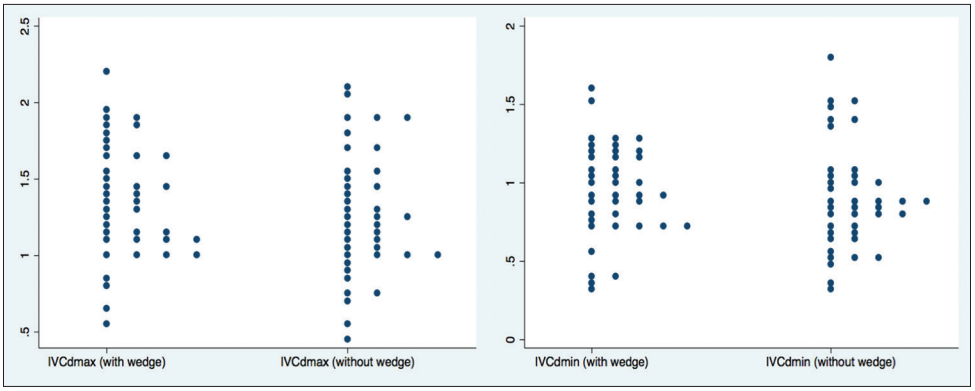

Figure 2: Dot plot showing distribution of $IVCd_{max}$ (left) and $IVCd_{min}$ (right) with wedge (left lateral tilt) and without wedge (supine) position

($P = 0.02$ and $P = 0.13$ respectively, paired sample *t*-test). $IVCd_{min}$ and IVCCI were similar with and without wedge both in hypotensive ($P = 0.85$ and $P = 0.20$ respectively, paired sample t- test) and nonhypotensive ($P = 0.07$ and $P = 0.93$ respectively, paired sample *t*-test) patients.

No correlation was found between percentage of change in mean arterial pressure at 3, 6, 9, 12, and 15 min and IVCCI with and without wedge.

Area under the ROC curve of IVCCI with wedge to predict PSH was 0.46 (95% CI 0.27, 0.64) and best cut-of value was 25.64 with a sensitivity and specificity of 60.9% and 35.5%, respectively. Area under the ROC curve of IVCCI without wedge to predict PSH was 0.38 (95% CI 0.19, 0.56) and best cut-of value was 20.4 with a sensitivity and specificity of 69.6% and 23.5%, respectively [Figure 3].

Areas under the ROC curve of $IVCd_{max}$ with wedge ≥1.4 to predict PSH was 0.4 (95% CI 0.22, 0.58) with a sensitivity and specificity of 43.5% and 47.1%, respectively. Area under the ROC curve of $IVCd_{max}$ without wedge to predict PSH was 0.45 (95% CI 0.27, 0.64) and best cut-of value was 1.53 with a sensitivity and specificity of 30.4% and 82.4%, respectively. Negative predictive value of this test was 73.3%. Areas under the ROC curve for IVCdmax in these two positions have been depicted in Figure 4.

Discussion

In this study, we found that IVCCI with and without wedge is not a predictor of PSH. Mean $IVCd_{max}$ and $IVCd_{min}$ were similar between pregnant women who had PSH and who didn't have. $IVCd_{max}$ was significantly higher with wedge than in supine position without wedge but $IVCd_{min}$ and IVCCI were similar in those two positions. IVCCI was not a reasonable predictor of PSH, but $IVCd_{max}$ had a negative predictive value of 73.3%.

PSH can result in both maternal and fetal/neonatal adverse events.[3] Several modalities have been used in obstetric anesthesia practice to prevent and treat PSH.[3] Recently, researchers have found that both reduction in preload from aortocaval compression and systemic vascular resistance from arteriolar dilation contributed to PSH.[5,11] Although its recommended to start prophylactic vasopressors, namely phenylephrine infusion to prevent PSH,[3] but we did not start it as we were interested in monitoring the episodes of hypotension, also we chose ephedrine as in our study as we were not anticipating major hemodynamic changes as ASA I/II women were enrolled and they were on maintenance iv fluid infusion. From physiological point, patients who are hypovolemic at the time of spinal anesthesia might have an exaggerated reduction in blood pressure due to reduction in preload. So, identification of the hypovolemic patients ("volume responders") and fluid optimization

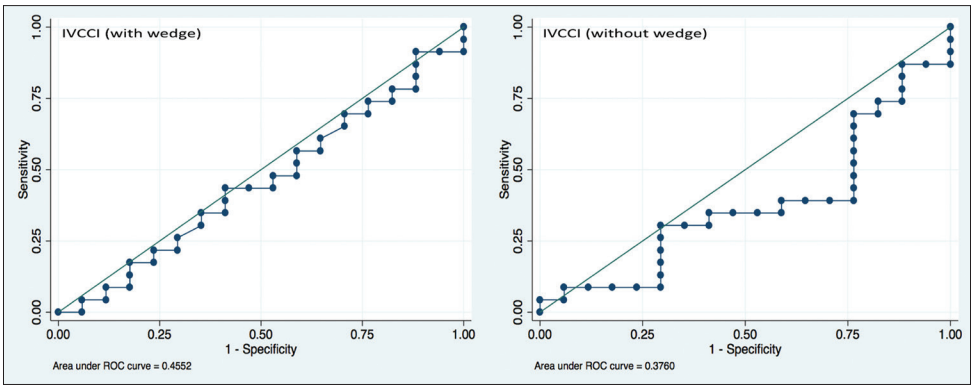

Figure 3: Receiver operating characteristics curve of IVCCI with wedge (left) and IVCCI without wedge (right) for predicting post- spinal hypotension

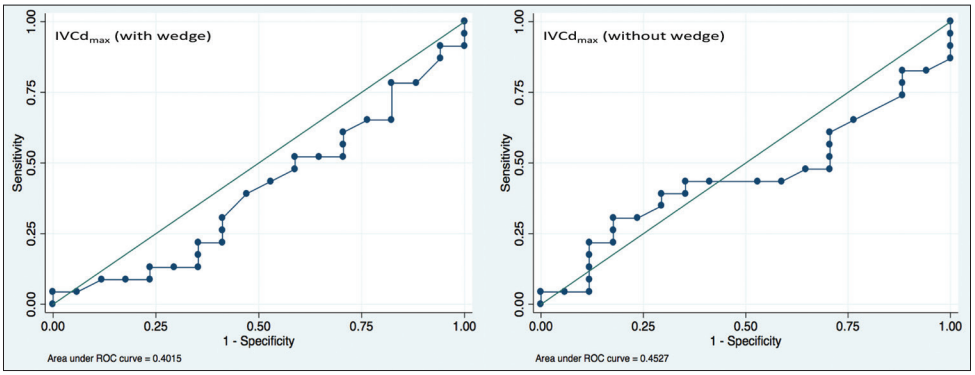

Figure 4: Receiver operating characteristics curve of IVCd$_{max}$ with wedge (left) and IVCd$_{max}$ without wedge (right) for predicting post- spinal hypotension

in this group of patients may reduce the incidence of hypotension.

IVCCI is a simple predictor of volume responsiveness both in mechanically ventilated and spontaneously breathing patients.[12-15] An observational study reported that IVCCI >42% has 97% specificity and 90% positive predictive value for fluid responsiveness spontaneously breathing adult patients.[12] Zhang *et al.* reported that preoperative IVCCI >43% is a reliable predictor of hypotension after induction of general anesthesia with a specificity of 91.7%.[16] A recently published randomized control trial reported that IVCCI guided fluid therapy is associated with a 35% reduced relative risk of PSH. In that study, an IVCCI >36% was considered as cut-off point of fluid responsiveness and fluid responsive patients received 500 ml of crystalloid bolus.[17] In this study, we have found that IVCCI whether in left lateral tilt position or supine position, is not a predictor of PSH. Reason for this difference may be multifactorial such as actual contribution of caval obstruction towards PSH may not be significant in all patients.[18] Second, normal pregnant women have an altered balance in vascular tone and increased synthesis of vasodilator prostaglandins and nitric oxide. So, sympathetic vacular tone may be more important for peripheral vascular resistance in these patients leading to a fall in blood pressure, following spinal anesthesia in pregnant women.

HR was not significantly associated with hypotension in our study. Preoperative baseline HR was found to be a predictor of PSH in several studies[17,19] but a few others have not supported this.[20,21]

The current literature suggests that proximal IVC measurements can be obtained in late pregnancy with failure rate of 3.9% due to nonvisualization,[10] although in our study failure rate was 11%, similar failure rate has been reported in general population also.[19] In our study, pregnant women were receiving maintenance fluids and they also received fluids during coloading because of which IVCCI was not significantly high. This suggests that the PSH in pregnant women is because of other reasons like more sensitivity to local anesthetics apart from hypovolemia.

We have found that IVCd$_{max}$ was statistically significantly higher with wedge (left lateral tilt position) as compared to without wedge (supine position), whereas there was no difference in IVCd$_{min}$ and IVCCI between these two positions. The decrease in IVCd$_{max}$ in supine position without wedge may be cause of decrease in venous return and central venous pressure.[22] IVCd$_{min}$ did not change as it is dependent both

upon amount of intravascular volume and the amount of negative intrathoracic pressure generated by the respiratory cycle.[22] Similar findings were reported in a cohort study in pregnant women with gestational age in between 30--42 weeks in labor room or delivery floor in left lateral lilt as compared with supine position.[10] However, they used a tilt of 30° as compared with 15° in our study. We used 15° tilt as high tilt leads to the difficulty in performing the surgery.

Limitations

There were several limitions in our study. First, our patients were on maintenance fluids of 50 ml/h and intravenous crystalloid boluses were administered at the time of spinal anesthesia which could have interfered with our results. Second, we have recruited only elective LSCS patients, so our results could not be extrapolated in patients undergoing emergency LSCS. Third, we have not assessed interobserver and intraobserver variability in IVC measurement in this study. Fourth, we could not assess the final level of block as surgery was started once it reached to T6 level.

Conclusion

IVCCI cannot be used to predict PSH in pregnant women undergoing elective cesarean section. Left lateral tilt with the help of wedge leads to increase in $IVCd_{max}$ without any change in $IVCd_{min}$ and IVCCI.

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Conflicts of interest
There are no conflicts of interest.

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