Estimating the usefulness of inferior vena cava collapsibility index and caval aorta index to predict hypotension after spinal anaesthesia in adult patients undergoing elective surgery in a tertiary care hospital

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Abstract

Background and Aims: Post-spinal anaesthesia hypotension (PSAH) can occur in 25–75% of patients. The preload or volume status of a patient is an important contributor to PSAH, and coloading with fluids is advocated to prevent PSAH. Instead of blind volume loading, prediction of volume status using inferior vena cava (IVC) collapsibility index (IVCCI) and caval aorta index (IVC:Ao index) may be used to guide fluid administration.

Material and Methods: In our study, we used ultrasound in the immediate pre-operative period to calculate IVCCI and IVC: Ao index in patients scheduled for elective surgery in the supine position, under spinal anaesthesia. Spinal anaesthesia was given in the lateral position with 0.5% hyperbaric bupivacaine. Patients were placed supine thereafter, sensory blockade level was ascertained, and blood pressure (BP) was measured every 2 min for 30 min. Episodes of hypotension were treated with fluids or vasopressors as per the discretion of the treating anaesthesiologist. In the study, 73 patients were screened, out of which 69 were included.

Results: Totally, 23 participants out of 69 developed PSAH. The receiver operating characteristic (ROC) curve was made and the area under the curve analysis was done on our collected data. We found that IVC: Ao index has better sensitivity (0.696 for IVC: Ao index ≤ 0.810) and specificity (0.717 for IVC: Ao index ≤ 0.810) than IVCCI (sensitivity 0.522 and specificity 0.630 for IVCCI $\geq 33.32\%$) to predict PSAH.

Conclusion: IVC:Ao index is a better predictor of PSAH than IVCCI. Thus, it may be used to predict volume status and guide in coloading with fluids during spinal anaesthesia.

Keywords: Caval aortic index, IVC collapsibility index, post-spinal anaesthesia hypotension, spinal anaesthesia

Introduction

Spinal anaesthesia is routinely administered to patients all over the world. However, hypotension is a recognised adverse effect of it. Hypotension is said to occur in 25–75%

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of patients who receive spinal anaesthesia, with a higher incidence during caesarean section. Multiple risk factors have been identified for the occurrence of post-spinal anaesthesia hypotension (PSAH).^[1]

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Submitted: 02-Aug-2023 Accepted: 17-Dec-2023 Revised: 08-Dec-2023 Published: 23-May-2024 Reduction in cardiac output is implicated in the development of PSAH.^[2] As the fluid status of a person (i.e., preload) can determine cardiac output, it has been hypothesised that poor fluid status can contribute to PSAH. However, fluid status estimation is not carried out routinely in the pre-operative period.

Intensive care units and emergency rooms utilise fluid status estimation almost every day, to determine whether hypotension in a particular patient is due to decreased preload or not. Fluid status estimation is carried out using ultrasound guidance. With the increasing availability and ease of use of ultrasound, it may be feasible to perform such fluid status estimations in all patients posted for elective surgeries.

Under ultrasound-guided fluid status estimation, two parameters are of interest in this study: inferior vena cava (IVC) collapsibility index (IVCCI), and caval aorta index (IVC:Ao index). These parameters have been studied in various settings, including the emergency department, in paediatric populations, and before induction of general anaesthesia.

Our primary objective was to determine the usefulness of IVCCI and IVC:Ao index as predictors of PSAH, and our secondary objective was to determine which among the two indices is a better predictor for PSAH.

Material and Methods

The study was a prospective, single-centre, observational study conducted in the Department of Anaesthesiology of a tertiary care hospital from September 2020 to September 2021. The study began after obtaining approval from the departmental dissertation committee and the institutional ethics committee (IEC: 967/2019), and after registration at the Clinical Trials Registry, India (CTRI/2020/04/024489).

Our inclusion criteria were adult patients aged 18–65 years, American Society of Anaesthesiologists (ASA) physical status (PS) 1 or 2, and elective surgery scheduled under spinal anaesthesia in the supine position. Our exclusion criteria were body mass index (BMI) >30 kg/m², patients with baseline systolic blood pressure (SBP) <90 mmHg or mean arterial BP (MAP) <60 mmHg, pregnant women, patients taking angiotensin-converting enzyme (ACE) inhibitors/angiotensin receptor blockers (ARBs), patients with any contraindication to spinal anaesthesia, patients undergoing emergency surgery, and failure to perform spinal anaesthesia.

Participant identity was kept hidden by assigning random serial numbers, and hospital identification numbers were not written in data collection forms. In our pilot study, we found that the prevalence of PSAH was 45%, IVC: Ao had a sensitivity of 0.96 and specificity of 0.90 to predict PSAH. Substituting these values in the formula for calculation of sample size, keeping z = 1.96, we obtained two sample sizes: 41 and 62. The larger number of 62 was decided upon as an adequate sample size for our study. This ensured that the final sample size stayed above 41 even after dropouts and exclusions from the study at a later stage.

There were two observers involved in the study: Observer 1: The principal investigator who examined the patient on the day before surgery, ensured that inclusion criteria were met, and obtained written informed consent. Observer 1 also assessed the level of sensory blockade after spinal anaesthesia, monitored the patients during the study period, and collected data required for the study. Observer 2: The consultant anaesthesiologist who performed ultrasonography on the patient and obtained the values to evaluate the IVCCI and IVC:Ao index.

All patients were examined on the day before surgery by observer 1. The patients were kept NPO as per standard guidelines, and pre-medications were administered as per the discretion of the treating anaesthesiologist assigned to the operating room where the patients were scheduled for surgery.

In the pre-operative holding area, observer 2 performed ultrasonography on the subjects using a curvilinear probe (3–5 MHz frequency). B-mode was used. Inferior vena cava (IVC) and aorta were both scanned by longitudinal placement of the probe in the subxiphoid region. First, IVC was scanned. Using M-mode with the marker on the IVC, about 3–4 cm distal to the right atrium, the maximum internal diameter (IVCD_{max}) and the minimum internal diameter of the IVC (IVCD_{min}) were measured during the same respiratory cycle and noted down [Figure 1].

IVCCI was derived by the formula: $\left[\left(IVCD_{max} - IVCD_{min}\right) / IVCD_{max}\right] \times 100$

To the left of the IVC, the aorta was visualised. The maximum internal diameter of the aorta during systole (Ao_{max}) was measured and noted [Figure 2].

The IVC:Ao index was derived by the formula:

After ultrasonographic evaluation, the patients were shifted to the operating room and standard monitors were attached. Intravenous (IV) line was secured with an 18-G cannula and IV crystalloids were started as per institutional protocol. Spinal anaesthesia was performed as per the discretion of the treating anaesthesiologist assigned to the patient's operating room, using bupivacaine along with buprenorphine or fentanyl as the adjuvant. If dexmedetomidine or clonidine were used as additives, we excluded the patient from the study.

After performing spinal anaesthesia, patients were placed in the supine position, and they remained in the same position throughout the study period of 30 min and during the surgery. The level of sensory blockade was assessed by observer 1 with a pinprick test. If the level of sensory blockade was found to be T6 and above, we excluded the patient from the study.

Non-invasive BP was recorded every 2 min for the first 30 min and every 5 min thereafter. PSAH was defined as an absolute value of SBP <90 mmHg, or a decrease in SBP by more than 30% from the baseline value, or an absolute value of MAP <60 mmHg.

Episodes of hypotension, if they occurred, were treated as per the decision of the treating anaesthesiologist, using IV Mephentermine 3 mg, or IV Phenylephrine 100 mcg, or IV Ephedrine 6 mg. If bradycardia occurred, IV Atropine 0.6 mg was administered at the discretion of the treating anaesthesiologist.

The level of sensory block, volume of bupivacaine used, and number of episodes of PSAH were noted. In addition, we noted whether IV fluids alone were used to treat PSAH or whether IV drugs were needed. Any other complications were also noted.

Statistical analysis was done on the collected data. Mean IVCCI and IVC:Ao index were calculated and receiver operating characteristic (ROC) curves were plotted for the two indices. Area under the curve analysis was done for each index.

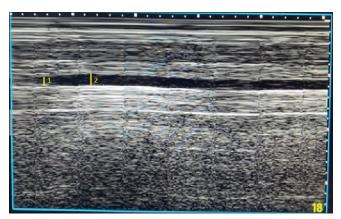


Figure 1: IVCCI measurement. Measurement 1: - IVC dDiameter at inspiration. Measurement 2: - IVC diameter at expiration

Results

A total of 73 patients were screened; out of which 69 (59 males, 10 females) were included as participants. Reasons for exclusion were surgery cancellation (1 patient), failed spinal anaesthesia (1 patient), and refusal for spinal anaesthesia on a table (2 patients). Of the 69 participants, 48 belonged to ASA PS 1, whereas the remaining 21 belonged to ASA PS 2.

Mean IVCCI in patients who developed PSAH was $35.63 \pm 17.01\%$ [Table 1]. Mean IVC:Ao index in patients who developed PSAH was 0.79 ± 0.25 [Table 2].

Area under the curve analysis was done for IVCCI and IVC:Ao and a logistic regression model was created. Figure 3 shows the ROC curve for IVCCI. IVCCI was found to be statistically insignificant in predicting PSAH [Table 3]. The sensitivity of IVCCI \geq 33.32% for predicting PSAH was found to be 0.522 and the specificity was 0.630. Figure 4 shows the ROC curve for IVC:Ao index. IVC:Ao index was found to be statistically significant in the prediction of PSAH.

Table 1: IVCCI in hypotensive v/s normotensive patients			
Parameter	BP	Mean±SD (%)	
IVCCI	Normotensive	31.66±12.80	
	Hypotensive	35.63±17.01	

Table 2: IVC:Ao in hypotensive v/s normotensive patients				
Parameter	BP	Mean±SD		
IVC: Ao Index	Normotensive	0.91±0.19		
	Hypotensive	0.79 ± 0.25		

Table 3: Area under the curve analysis for IVCCI			
Area	Standard error	Р	
0.562	0.082	0.401	

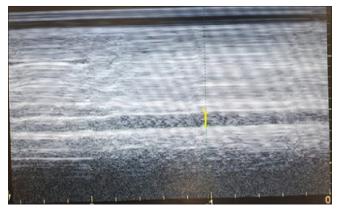


Figure 2: IVC: Ao measurement. Measurement 1: - Maximum diameter of aorta (during systole)

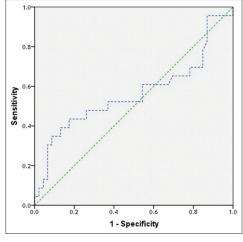


Figure 3: ROC curve for IVCCI

IVC:Ao index of ≤ 0.810 was found to have a sensitivity of 0.696 and a specificity of 0.717 to predict PSAH [Table 4].

Because our study had multiple other variables which could be confounding, the logistic regression model was created and the significance of each variable was calculated. We found that age and IVC:Ao index have a statistically significant contribution to PSAH, whereas sex, ASA PS, BMI, and IVCCI do not.

The findings of our logistic regression model were: (1) As age increases, the chances of PSAH increase 1.063 times. (2) As gender shifts from male to female, the chances of PSAH increase 1.934 times. (3) As ASAPS shifts from 1 to 2, the chances of PSAH increase 1.340 times. (4) As BMI decreases, the chances of PSAH increase 1.127 times. (5) As IVCCI increases, the chances of PSAH increase 1.031 times. (6) As the IVC:Ao index decreases, the chances of PSAH increase 37.037 times.

Discussion

The aim of our study was to estimate whether IVCCI and IVC:Ao are reliable predictors of PSAH, and to determine which of the two is superior. We found that IVCCI is not statistically significant and has a lower sensitivity and specificity compared to IVC:Ao in predicting PSAH.

In this study, we excluded patients in whom sensory blockade was at or above T6 level. This was done because it is known that sympathetic block occurs at higher levels than the sensory block level, as demonstrated by Zhang *et al.*^[3] With cardioacceleratory fibres being at T1-T4 level, at a sympathetic block of T4 or above one would expect sympathetic block and consequent hypotension regardless of fluid status of the patient.^[4] We excluded patients with BMI >30 kg/m² as

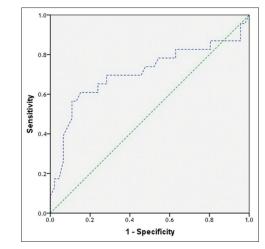


Figure 4: ROC curve for IVC:Ao index

Table 4: Area under the curve analysis for IVC:Ao index			
Area	Standard error	Р	
0.707	0.076	0.005	

well as patients taking ACE inhibitors and ARBs. This was done because previous studies have found these to be risk factors for patients to develop hypotension post-induction of anaesthesia.^[5,6]

Multiple studies have been conducted to compare the effects of various adjuvants used during spinal anaesthesia. Kaur *et al.*^[7] found that usage of dexmedetomidine could lead to hypotension, whereas no hypotension was noted with buprenorphine. Akanmu *et al.*^[8] found that there was no difference in the incidence of hypotension between fentanyl and saline used as adjuvants. Clonidine even at doses as low as 15 mcg was found to cause hypotension, and dexmedetomidine was also found to cause hypotension after spinal anaesthesia.^[9,10] Thus, buprenorphine and fentanyl were allowed, and clonidine and dexmedetomidine were not used in our study.^[7-10] As discussed previously by Singh *et al.*,^[11] advanced age was also found in this study to be a risk factor for PSAH. However, in our study, equidistribution was not present between the age groups.

In our study, we found that patients belonging to ASA PS 2 had more chances of developing PSAH than patients belonging to ASA PS 1. The same finding was also reflected in the study done by Singla *et al.*^[5] In our multiple logistic regression model, we saw that female sex was an independent risk factor for the development of hypotension after spinal anaesthesia. However, our study had a small sample size of female participants. We also found that patients with a BMI of <22.87 kg/m² had a higher chance of developing PSAH. Equidistribution between BMI categories and an overall

larger sample size may be needed to further comment on the significance of this result.

Many studies suggest that IVCCI and IVC: Ao can be used to predict fluid status and PSAH. However, Roy et al.^[12] found in their study that IVCCI is not a good predictor for PSAH. Gui et al.^[13] also concluded in their study that IVC: Ao is not superior to IVCCI in the estimation of fluid status of a patient. Thus, we wanted to find out through our study whether one or both of the parameters can be used in the future. In our study, we calculated the mean IVCCI in patients who developed PSAH. It was found to be $35.63 \pm 17.01\%$, which is lower than the optimal cutoff value of 43% calculated in the study done by Zhang and Critchley.^[14] It was also lower than the value of $49.9 \pm 6.1\%$ calculated by Salama and Elkashlan.^[15] The mean IVC: Ao index for patients who developed PSAH was 0.79 ± 0.25 , whereas it was 0.91 ± 0.19 in patients who did not develop PSAH. This correlates with the study done by Sridhar et al.^[16] The area under ROC curve analysis was done, and we found the area under the curve for IVCCI to be 0.562 (P value = 0.401) and that for IVC:Ao index to be 0.707 (P value = 0.005). From this, we can conclude that IVCCI is a poor predictor for PSAH, whereas IVC:Ao index may be considered a good predictor for PSAH.

We also calculated the sensitivity and specificity of each index to predict PSAH. IVCCI of \geq 33.32% had a sensitivity of 0.522, whereas IVC:Ao of \leq 0.81 was found to have a sensitivity of 0.696. Thus, IVC:Ao was found to be a more sensitive indicator for predicting PSAH. IVCCI \geq 33.32% was found to have a specificity of 0.630 in predicting PSAH, and the specificity of IVC:Ao \leq 0.81 for predicting PSAH was calculated to be 0.717. Thus, IVC:Ao also has more specificity than IVCCI for predicting PSAH.

In our study, we created a logistic regression model. The dependent variable was PSAH, and the independent variables were age, gender, ASA PS, BMI, IVCCI, and IVC:Ao index. From the model, we found that all the variables did contribute to PSAH. However, only age and IVC:Ao index had a statistically significant contribution to our logistic regression model.

We would like to add that in view of the COVID-19 pandemic, our sample size could not be sufficiently large to provide equidistribution among the different BMI groups, ages, as well as sexes. Further studies with larger sample sizes may be required to investigate all these variables separately.

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Conflicts of interest

There are no conflicts of interest.

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