ABSTRACT

Introduction: Central Venous Pressure (CVP) measurement is the recommended method for assessment of intravascular status in paediatric shock. The role of Ultrasonography guided measurement of respiratory collapsibility in inferior vena cava diameter (IVC-CI) as a newer, non-invasive adjunct to CVP measurement has been evaluated. This study was done to determine the effectiveness of CVP and IVC-CI in predicting fluid responsiveness in cases of paediatric shock.

Material and Methods: This prospective observational study was done in 107 fluid refractory shock patients aged 1-14 years. An informed consent was obtained. Baseline vitals, CVP and IVC-CI were measured before and after a crystalloid Fluid Bolus of 20ml per Kg BW. The changes in CVP and IVC-CI were noted and were correlated to the clinical response. A rise of ≥15% in Cardiac Output was taken as positive fluid response.

Results: Mean age of the patients was 7.6 years (±4.153). The mean CVP in fluid responders and non-responders was 6.58 (±2.64) and 11.22 (±6.12), while the mean IVC-CI was 46.57% (±23.34) and 25.62% (±23.28) respectively. There was significant inverse correlation between CVP and IVC-CI (P<0.01) in both fluid responders and non-responders. At CVP ≥8.25, sensitivity was 80% and specificity was 99% for predicting fluid responsiveness. When IVC-CI was ≥33.5%, sensitivity was 87% and specificity was 86%. Thus IVC-CI has better sensitivity and poor specificity to predict fluid responsiveness than CVP.

Conclusion: Both CVP and IVC-CI are good predictors of volume responsiveness. A shift from hypovolemic to euvoletic status was associated with gradual fall in mean IVC-CI values with progressive rise of CVP values. IVC-CI can provide a useful guide for non-invasive intravascular volume status assessment in critically ill patients.

Keywords: Shock, Fluid Responsive, Inferior Vena Cava, Central Venous Pressure, Paediatrics.

INTRODUCTION

Shock in paediatric population is a major problem associated with high mortality and organ failure.1,2,3 Aggressive and early fluid boluses is the initial treatment of choice.4,5 Determining intravascular volume status and fluid responsiveness based on clinical examination is challenging. Clinicians often use invasive hemodynamic monitoring as an adjunct to the physical examination to arrive at a fluid management strategy. Central Venous Pressure is an extensively used hemodynamic parameter. It gives an approximation of right atrial pressure which in turn correlates with right ventricular filling. Therefore, CVP indicates right ventricular preload. However, there are complications associated with invasive nature of CVP insertion like failure of catheter insertion, pneumothorax, arterial puncture. Bedside ultrasound is a non-invasive technique to estimate the intravascular status by measuring inferior vena cava diameter. Ultrasonography evaluation of Inferior vena cava dimensions during inspiration and expiration (Inferior vena cava collapsibility index)6 provides data to guide the clinician on the bedside to decide whether or not more fluid boluses can be given.7,8,9,10 This study was done to determine the effectiveness of CVP and IVC-CI in predicting fluid responsiveness in cases of paediatric shock.

MATERIAL AND METHODS

This prospective observational study was done in Paediatric intensive care unit (PICU) of Jawaharlal Nehru Medical College and Hospital (JNMCH), Aligarh Muslim University (AMU), India from December 2015 to July 2017. Ethical clearance was obtained from Institutional Ethics Committee, JNMCH, AMU.

Informed consent was taken for PICU admission and placement of central venous catheter. During the study period, patients who were in shock despite fluid bolus of 60 ml per kg of normal saline were shifted to PICU and included in the study. Patients with clinical signs of elevated abdominal pressure, moderate to severe tricuspid regurgitation, CVP inserted for more than 24 hours, and patients in whom the supine position was contraindicated were not included in the study. Patients were intubated and ventilated with 6 ml/Kg tidal volume and were sedated and paralysed with appropriate drugs. Central venous access was obtained in either internal jugular vein or subclavian vein. Bedside x-ray was done to ensure the tip of the catheter is at the superior vena cava – right atrium (SVC-RA) junction. CVP was transduced and measured using 7 Para monitor.
(Nihon Kohden) at the level 4th intercostal space in mid clavicle line.

Bedside echocardiography was done using GE vivid model.

The ultrasound examination of the inferior vena cava was done by a paediatric critical care specialist in all the cases, who was blinded to CVP monitoring during the collection of ultrasound data.

The ultrasound images were obtained with patient in supine position to determine the dimensions and collapsibility of IVC. The ultrasound gel was applied to the sub-xiphoid region. Then, the transducer in the sub-xiphoid position and IVC was imaged in a longitudinal plane. The intrahepatic segment of the IVC entering the right atrium was visualized. The IVC diameter was measured 2 cm caudal to the hepatic vein-IVC junction, or approximately 3 cm from the junction of the IVC and right atrium. This measurement location was preferred as IVC collapsibility in the intrahepatic segment was not influenced by the activity of the muscular diaphragm compared to that at the IVC-right atrial junction. M-mode was used to capture the IVC over two or three respiratory cycles. The maximum IVC diameter (IVC Dmax) was measured as the maximum anterior-posterior dimension at end-expiration using the leading edge technique (inner edge to inner edge of the vessel wall). In addition, the minimum IVC diameter was measured at end-inspiration (IVC Dmin).

The IVC collapsibility index was the difference between the maximum and minimum IVC diameters divided by the maximum IVC diameter, expressed as a percentage ((IVC Dmax – IVC Dmin) / IVCDmax × 100)%

Cardiac output was measured using echocardiography by measuring the aortic orifice diameter and aortic blood flow velocity.

Baseline clinical variables with CVP and IVC-CI were recorded in all patients. Fluid bolus of 20ml/kg of Normal Saline was started in all patients. Subject was considered to be Fluid Responder when increase in Cardiac Output ≥ 15% from the baseline without evidence of fluid overload on completion of bolus. Fluid Non-Responder was when hemodynamic parameter either worsened or patient demonstrated features of fluid overload. Bolus was stopped immediately in patients who demonstrated features of fluid overload. CVP and IVC-CI was compared in fluid responders and non -responders.

**STATISTICAL ANALYSIS**

The data was entered and analysed on SPSS version 21. Descriptive statistics were calculated for both qualitative variables. Pearson correlation coefficient was used to assess the significance between CVP and IVC-CI (%). A p-value less than 0.05 was considered to be significant.

**RESULTS**

The mean age of the patients was 7.6 yrs (±4.153). Majority of the patients were in female group (54%), (n=58). Septic Shock (93%) was the most common cause of shock.

Most of the enrolled patients were fluid responders (66%) . Fluid responder group had lower mean CVP value and higher

<table>
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<th>Table-1: Overview of responders and non-responders</th>
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<tr>
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mean IVC-CI value than those of in fluid non-responder group. There was significant inverse correlation between CVP and IVC-CI in both responders and non-responders. From the ROC curves in figure 1and 2 it was observed that when CVP was ≤ 8.25 sensitivity was 80% and specificity was 99%, When IVC-CI was ≥ 33.5% sensitivity was 87% and specificity was 86%. Thus IVC-CI has better sensitivity put poor specificity to predict fluid responsiveness.

Area under curve: For IVC-CI ROC curve 0.908
For CVP ROC curve 0.963

Figure 3 shows the mean CVP value in fluid responder group is much lower than that of the fluid non-responder group, and this relation is statistically significant (P<0.01)

Figure 4 shows the mean IVC-CI value in fluid responder group is higher than that of the fluid non-responder group, and this relation is statistically significant (P<0.01)

Figure 5 shows that in fluid responder group the mean IVC-CI value is around 60% when CVP value is below 5. As there is shifting from hypovolemic to euvolemic range after fluid boluses there is gradual fall in mean IVC-CI values with progressive rise of CVP values. (P<0.01)

Most of the enrolled patients were fluid responders. Fluid responder group had lower mean CVP value and higher mean IVC-CI value than those of in fluid non-responder group. The significant inverse correlation between CVP and IVC-CI was there. As there is shifting from hypovolemic to euvolemic range after fluid boluses in fluid responders there is gradual fall in mean IVC-CI values with progressive rise of CVP values. (P<0.01) it was observed that when CVP specificity was 80% and IVC-CI sensitivity was 99%, When IVC-CI was ≥ 33.5% sensitivity was 87% and specificity was 86%. Thus IVC-CI has better sensitivity put poor specificity to predict fluid responsiveness.

DISCUSSION

CVP is the most commonly used variable for volume status. More than 90% of intensivist and anaesthesiologist use CVP to guide fluid management. In a meta-analysis of 5 studies, pooled correlation between mean CVP and measured blood volume was 0.16 and pooled area under the curve was only 0.56. CVP is dependent upon venous return to heart, RV compliance, peripheral venous tone, posture, pulmonary vascular disease, RV disease, isolated LV failure and valvular heart disease. Further CVP may actually fall with fluid bolus and sympathetic vascular constriction is relieved. Furthermore CVP catheter insertion is time consuming requires expertise and may involve complications.

Respiratory variation in Inferior Vena cava is easy, reliable and non-invasive method to evaluate the intravascular volume status and predicting fluid responsiveness in patients with shock. Central Venous Pressure is a time honoured static variable to assess fluid status. Both CVP and IVC-CI were statistically different in responders and non-responders. There was a decreasing trend of IVC-CI in both groups after the fluid bolus. Various studies in adults have shown that IVC-CI has reasonable sensitivity and specificity for detecting fluid responsiveness. Various thresholds were 24.6% 48%, 12% 25%, 36.5%. Very few paediatric studies have come up in this regard. One such study had IVC-CI of 27%. Of late CVP is considered to be a poor marker for assessing fluid responsiveness. However in this study we found that it has reasonable sensitivity and specificity at a cut-off of less than 8.25. Both CVP and IVC-CI are good predictors of volume responsiveness. IVC-CI at >33.5% had more sensitivity but lesser specificity than CVP of <8.25 mm Hg as predictor for volume responsiveness.

CONCLUSION

IVC-CI is a valid and good variable for assessing fluid responsiveness in cases of paediatric shock. It is simple bedside tool and requires minimal training and correlates well with the time honoured static variable i.e CVP. The non-invasive nature of IVC-CI compared to CVP makes it even more lucrative in patients in whom central venous access could not be taken.

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Figure-5: CVP VS IVC-CI in Fluid Responder Group