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Correlation of Peripheral And Central Venous Pressure Values In Intensive Care Patients With Acute Renal Failure

Akut Böbrek Yetmezlikli Yoğun Bakım Hastalarında Periferal ve Santral Venöz Basınç Değerlerinin Korelasyonu

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Öz

Amaç
Bu çalışmada yetişkin yoğun bakım ünitesindeki (YBÜ) hastalarda akut böbrek yetmezliği, santral ve periferik venöz basınç ölçümleri arasında herhangi bir ilişki olup olmadığını analiz etmeyi amaçladık.
Gereç ve Yöntem
Herhangi bir nedenle YBÜ'de en az 6 gün yatmış 39 hasta (16 K, 73.6 ± 13.9 yaş) dahil edildi. Bazal ve günlük kreatinin değerleri, günlük sistolik, diyastolik ve ortalama kan basınçları ile santral venöz basınç / periferik venöz basınç (CVP/ PVP) ölçümleri kaydedildi. İzlem sırasında kreatinin düzeylerinde %50'den fazla artış görülen hastalar, akut böbrek hasarı (AKI, n: 12) grubunda, stabil kreatinin değerleri olan hastalar ise no-AKI (n: 27) grup olarak kabul edildi.
Bulgular
Tüm hastalar göz önüne alındığında, PVP ve CVP ölçümlerinin pozitif yönde (r: .882, p: 0.0001) korele olduğunu tespit ettik. Ortalama PVP 10.6 ± 2.4, ortalama CVP 6.4 ± 2.4 ve ortalama PVP – CVP= 4.1 ± 1.1 mmHg idi. YBÜ'de yatış sürecinde ortalama CVP ve PVP ölçümleri arasındaki karşılaştırmada Bland-Altman diyagramı da mükemmel bir uyum (-4.3 farkı) olduğunu gösterdi. AKI ve no-AKI grupları demografik özellikler açısından benzerdi. AKI grubunda hem PVP (p: 0.009) hem de CVP (0.039) değerleri daha yüksekti. Ayrıca sistolik ve arteryal basınçları daha düşüktü (p <0.05). Böbrek yetmezliği olan hastalar no-AKI hastalarına kıyasla en yüksek CVP ve PVP değerlerine ve en düşük kan basıncına sahip olduğu görüldü (p: 0.01).
Sonuç
PVP ve CVP ölçümlerinin yüksek oranda korelasyona sahip olduğunu ve her ikisinin de venöz konjesyon, düşük arteriyel dolum ve ARF için erken belirteçler olarak kullanılabileceğini düşünüyoruz.

Anahtar Kelimeler: Akut böbrek yetmezliği, periferik venöz basınç, santral venöz basınç

Abstract

Objective
In this study, we aimed to analyze if there is any relationship between acute renal failure, central and peripheral venous pressure measurements in adult intensive care patients.
Materials and Methods
We included 39 patients (16 F, 73.6 ± 13.9 yrs old) who were hospitalized in intensive care unit (ICU) for at least 6 days for any reason. Basal and daily creatinine values, daily systolic, diastolic, and mean blood pressures, and central venous pressure / peripheral venous pressure (CVP/PVP) measurements were recorded. Patients who had more than 50% increase in creatinine levels during follow-up were accepted as acute kidney injury (AKI, n: 12) group while patients with stable creatinine values were accepted as no-AKI (n: 27) group.
Results
Considering all patients we found that PVP and CVP measurements were positively correlated (r:.882, p: 0.0001). Mean PVP was 10.6 ± 2.4, mean CVP was 6.4 ± 2.4 and mean PVP- CVP was 4.1 ± 1.1 mmHg. A Bland-Altman diagram for the comparison between mean CVP and PVP measurements during ICU hospitalization also indicated perfect agreement (difference of-4.3). AKI and no-AKI groups were similar in means of demographic characteristics. AKI group had both higher PVP (p: 0.009) and higher CVP (0.039) values. They also had lower systolic and mean arterial pressure (p < 0.05). Patients with renal failure has the highest CVP and PVP values and lowest blood pressures compared to noAKI patients (p: 0.01).
Conclusion
We think that PVP and CVP measurements are highly correlated and both could be used as early markers for venous congestion, lower arterial filling and ARF.

Keywords: Acute renal failure, peripheral venous pressure, central venous pressure

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Introduction

Central venous pressure (CVP) is a reflection of right atrial pressure, which is used as an estimate of left ventricular end-diastolic volume . CVP monitorization is a common practice in intensive care units (ICU) and is used to assess intravascular volume depletion of patients. An increased CVP value is accepted as a marker of hypervolemia while low CVP values are accepted as markers of decreased intravascular volume and possibly decreased tissue perfusion. There are two major clinical settings in which the CVP provides an unreliable estimate of the LVEDP. In patients with pure left-sided heart failure, the wedge pressure is increased, but the CVP may remain unchanged if right ventricular function is normal. On the other hand in setting of pure right-sided heart failure, CVP tends to exceed LVEDP. Central venous pressure is not a useful tool for assessing effective arterial volume (EAV) and could be both increased or decreased in patients with decreased EAV or decreased mean arterial pressure (MAP). A decrease in CVP associated with decreased EAV and/or MAP is usually accepted as true hypovolemia or dehydration while increased CVP associated with decreased EAV and/or MAP is a marker of hypervolemia associated with decreased arterial resistance (septic shock) or venous pooling (liver failure, heart failure). Central venous catheterization is required for CVP monitorization. This is an invasive procedure that could lead to some life threatening complications like hemothorax and pneumothorax. Long lasting catheters might also cause infections. Using peripheral venous pressure (PVP) measurement instead of CVP as a volume monitor could be an alternative that might decrease invasive procedure risks. In literature there are contradicting studies about the correlation between CVP and PVP measurements (1-3).

Acute renal failure (ARF) is a common problem in patients requiring ICU support. These patients are usually prone to hypotension due to true hypovolemia or septic complications. They also receive a group of drugs that have high nephrotoxic potentials like antibiotics and nephrotoxic opaque materials. Overall combining effect of these factors is a susceptibility to acute kidney injury (AKI) and ARF. There are some classifications and criterias that define AKI and ARF in these patients. Most common two classifications are RIFLE and AKIN that both accept a 50% increase in serum creatinine levels as the initial finding of AKI (4, 5). This definition puts patients who have very mild or moderate increases in serum creatinine levels into AKI / ARF group.

AKI / ARF is usually associated with venous congestion due to decreased urine output. However EAV / MAP could be increased or decreased depending on the underlying etiology. For example in a patient with septic shock and ARF, EAV / MAP would be decreased but in a patient who developed ARF due to nephrotoxic drug usage EAV / MAP might be increased, normal or decreased. So arterial blood pressure monitorization might not be an useful early predictor of kidney injury risk. So in this study we aimed to analyze if increased CVP values might be related development of AKI / ARF and if PVP could be considered as a suitable surrogate for CVP in ICU patients.

Material and Methods

Subjects

This study was designed as a prospective observational study. With the approval of the hospital research ethics committee, 39 patients (16 F, 73.6 ± 13.9 years old) were included who have been selected from a group of 108 patients who have been followed in ICU between October 2014 and February 2015 according to the following exclusion criterias; 1) pediatric patients (< 15 years age), 2) postoperative patients or requiring surgical intervention during study, 3) malignancy history, 4) severe malnutrition, 5) severe right or left sided heart failure, cor pulmonale, nephrotic syndrome or severe liver failure history, 6) myocardial infarction or acute coronary syndrome during study, 7) previous renal failure history or increased creatinine levels at ICU admission (> 1.5 mg/dL), 8) severe hypoalbuminemia at ICU admission (< 3 g/dL), 9) Inappropriate CVP and/or PVP measurements due to anatomical or technical reasons, 10) ICU follow-up duration is less than 6 days due to discharge or death.

Collected data

Demographic data (age, gender), reason for ICU hos-

pitalization, body weight were collected from patient charts. Initial albumin, and creatinine (sCr) levels were collected as initial biochemical characteristics. Daily sCR levels and urine output were collected for the following 5 days. Blood pressure measurements were done at hourly intervals and mean MAPs [(systolic blood pressure + 2x diastolic blood pressure) / 3] for 6 days including initial day were collected. Peripheral venous pressure and CVP measurements were done at 4 hour intervals and daily mean values for 6 days were collected. Method for CVP and PVP measurements were as follows;

Central and peripheral venous pressure monitorization: A central venous catheter appropriate for patients physical properties was inserted to either internal juguler vein or subclavian vein with standart methods. For measuring, catheter was attached to an intravenous fluid within a pressure bag that was inflated up to 300 mm Hg. Patient flat was placed in a supine position if possible or alternatively in a semi-recumbent position if a supine position was not possible. Transducer was taped to the phlebostatic axis or as near to the right atrium as possible. Then a Datex Ohmeda (Oregon, USA) monitorization system was used for automotized CVP measurement.

The PVP catheter site was maintained at midthorax height throughout each case. After flushing and room-air zero calibration, the transducer sets were mechanically flushed with saline and also maintained at midthorax level. Continuity of the PVP catheter with the downstream venous system was demonstrated at the beginning of each case by observing coincident pressure changes in the PVP waveform during circumferential, proximal arm occlusion.

Acute Kidney Injury definition

Patients with a 50% increase in any of their follow-up sCr values compared to initial values were accepted as AKI group. For classification of degree of renal failure we used both AKIN and RIFLE criterias which are summarized in Tablo-1. According to this classifications 12 patients were included into AKI and 27 patients were included into no-AKI groups. AKI group patients were included in this group if they developed renal failure in any day during ICU follow-up.

Tablo 1 RIFLE and AKIN criterias

RIFLE		AKIN		RIFLE and AKIN
Stage	sCr / GFR	Stage	sCr / GFR	Urine output
R	sCR increase > %50 or GFR decrease > %25	I	sCr increase > %50 or > 0.3 mg/dl	< 0.5 mg/kg/h for 6 hr
I	sCR increase > %100 or GFR decrease > %50	II	sCr increase > %100	< 0.5 mg/kg/h for 12 hr
F	sCR increase > %200 or GFR decrease > %75	III	sCr increase > %200 or > sCr > 4 mg/dL with an	< 0.3 mg/kg/h for 24 hr or anuria for 12 h
L	Need for RRT > 4 weeks		increase > 0.5 mg/dL or need for RRT	
E	Need for RRT > 3 months			

Satitistical methodology

Statistical Package for Social Sciences (SPSS for Windows, Chicago, IL, USA) version of 14.0 was used for data analysis. Data were submitted to a frequency distribution analysis by Kolmogorov-Smirnov's test. Values displaying normal distribution were expressed as the mean±SD and values with skew distribution were expressed as median (interquartile range). Differences between numeric variables were tested with independent samples Student's t-test or Mann Whitney U test where appropriate. Related data were compared with paired samples t or Wilcoxon tests where appropriate. Categorical data were compared by chi-square test. Correlation analyses were done by Pearson or Spearman correlation tests according to distribution status. Bland-Altman diagram was used to assess agreement between CVP and PVP values. A Kaplan-Meier survival analysis was used to compare AKI rates in high and low venous pressure groups. The value of statistical significance was accepted as: "$p<0.05$".

Tablo 2 Comparison of stduy groups in means of central and peripheral venous pressure

RIFLE	No-AKI group n:27	AKI group n:12	p value
Gender (fermale, n)	12/27	4/12	0.515
Body weight (kg)	76.1±8.6	76.3±10.1	0.662
Basal creatinine (mg/dL)	1.1±0.4	1.1±0.5	0.969
1st day creatinine (mg/dl)	0.97±0.21	1.14±0.2	0.022
2nd day creatinine (mg/dl)	1 (0.42)	1.67 (1)	0.0001
3rd day creatinine (mg/dl)	1(0.68)	1.71 (3.6)	0.0001
4th day creatinine (mg/dl)	0.9 (0.6)	1.95 (3.48)	0.0001
5th day creatinine (mg/dl)	0.8 (0.6)	2.2 (3)	0.0001
Basal albumin (mg/dl)	4.14	4.09 (0.31)	0.126
Basal MAP (mmHg)	97.9±12.8	97.1±13.1	0.871
1st day MAP (mmHg)	107.±14.1	92.3±12.9	0.002
2nd day MAP (mmHg)	104.7±13.7	87±11.7	0.001
3rd day MAP (mmHg)	94.9±12.5	85.5±11.4	0.032
4th day MAP (mmHg)	93.0±12.2	88.4±11.8	0.283
5th day MAP (mmHg)	91.1±11.9	80.4±11.4	0.013
Basal urine output (mL/kg/h)	0.95±0.46	1.09±0.53	0.406
1st day urine output (mL/kg/h)	1.14±0.62	0.77±0.45	0.406
2nd day urine output (mL/kg/h)	1.21±0.51	0.64±0.47	0.003
3rd day urine output (mL/kg/h)	1.21±0.64	0.66±0.56	0.014
4th day urine output (mL/kg/h)	1.13±0.43	0.66±0.52	0.005
5th day urine output (mL/kg/h)	1.1±0.41	0.68±0.56	0.013

Tablo 3 Comparison of study groups in means of central and peripheral venous pressure findings

RIFLE	No-AKI group n:27	AKI group n:12	P value
Basal CVP (mmH_g)	6.18±5.04	7.08±3.28	0.577
1st day CVP (mmH_g)	5.62±2.73	8.66±3.72	0.007
2nd day CVP (mmH_g)	5.77±2.02	7.33±2.49	0.002
3rd day CVP (mmH_g)	6.29±2.68	9.75±3.44	0.002
4th day CVP (mmH_g)	6.11±2.93	7.41±3.17	0.219
5th day CVP (mmH_g)	5.51±2.75	5.75±2.26	0.8
Basal PVP (mmH_g)	9.25±3.49	11.66±3.33	0.053
1st day PVP (mmH_g)	9.29±2.31	13.33±3.67	0.0001
2nd day PVP (mmH_g)	10±2.91	12.58±3.17	0.017
3rd day PVP (mmH_g)	10.96±2.95	13.5±2.74	0.016
4th day PVP (mmH_g)	10.59±3.76	10.75±3.13	0.9
5th day PVP (mmH_g)	9.7±2.58	10.83±2.03	0.189
Mean CVP (mmH_g)	5.91±2.29	7.66±2.5	0.039
Mean PVP (mmH_g)	9.96±2.29	12.11±2.04	0.009
Mean PVP - CVP (mmH_g)	4.04±1.22	4.44±1.08	0.343

Results

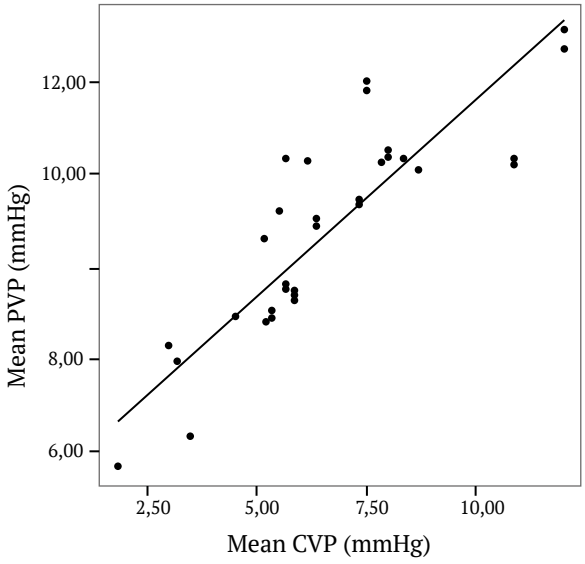
The reasons for ICU hospitalization of our patients were systemic infection (n:13), cerebrovasculer accident (n:5), hypotension caused by hypovolemia or hemorrhage (n:8), others (n:13; Chronic Obstuctive Lung Disease, pneumonia..).

AKI group had significantly higher creatinine values except basal creatinine value in follow-up period (Tablo-2, p: 0.022 – 0.0001). These patients also had lower urine outputs compared to patients without no-AKI in all 5 follow-up days (Tablo-2, p: 0.043 – 0.005). Mean arterial pressure measurements of patients with AKI were also lower compared to the-

ir counterparts without AKI except in days 0 and 4 (Tablo-2, p: 0.032 – 0.001).

Concerning CVP and PVP findings we observed that mean CVP was 6.45 ± 2.46 mmHg and mean PVP was 10.62 ± 2.41 mmHg. Difference between PVP and CVP was 4.17 ± 1.18 mmHg. All daily mean CVP measurements were significantly correlated with their daily mean PVP measurement equivalents (r: 0.919 – 0.681, p: 0.0001). Mean CVP and PVP values were also significantly correlated and the linear regression equation showed that CVP was equal to 0.91xPVP - 3.18 (Figure-1, r: 0.882, p: 0.0001). A Bland-Altman diagram for the comparison between mean CVP and PVP measurements during ICU hospitalization also indicated perfect agreement (difference of -4.3, Figure-2)

Figure 1 : Mean CVP and PVP measuremnts were significantly correlated (r:0.91, p:0.0001)



Patients with AKI had significantly higher CVP measurements between days 1-3 (p:0.047 – 0.002, Tablo-3). However days' 0, 4, 5 mean CVP measurements were indifferent. Similar to CVP findings, PVP measurements between days 1-3 were significantly higher in AKI group (p:0.017 – 0.0001). Mean CVP and PVP values were also significantly higher in patients with AKI (p: 0.039 and: 0.009 respectively). There was no significant difference between AKI and non-AKI groups in means of PVP-CVP difference. There was also no significant correlation between urine outputs and venous pressure findings.

Figure 2 : Bland-Altman diagram for the comparison between mean CVP and PVP measurements during ICU hospitalization. The horizontal line indicates perfect agreement (difference of -4.3), upper and lower lines indicate a clinically relevant difference of plus or minus 1.96. standard deviation (SD).

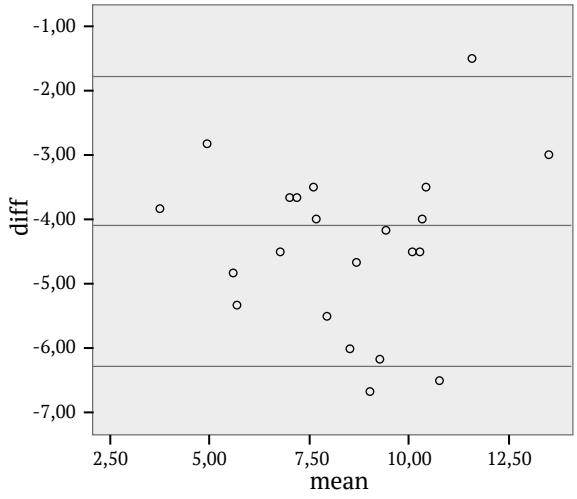
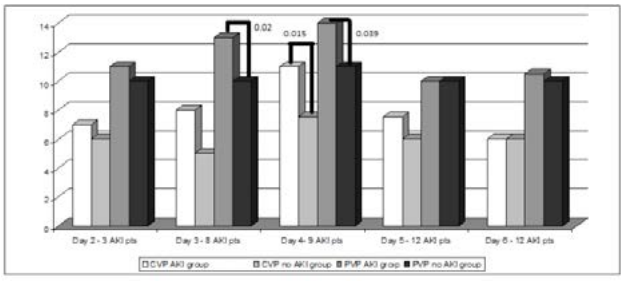


Figure 3: Comparison of median CVP and PVP values of patients with and without AKI in a daily basis. At day 3 we observed that patients with AKI had higher PVP (p: 0.02) and at day 4 higher CVP (p: 0.015) and PVP (p:0.039) values compared to non-AKI group



Comparison of median CVP and PVP values of pa tients with and without AKI in a daily basis is given in Figure-3. As there was not enough patients in AKI group at second day we did not perform any statistical analysis. Yet at third day we observed that patients with AKI had higher PVP (p: 0.02) and at day 4 higher CVP (p: 0.015) and PVP (p: 0.039) values compared to non-AKI group (Figure-3).

When subjects were grouped according to median values of CVP (6.5 mmHg) and PVP (11 mmHg) a Kaplan-Meier estimate for AKI rates revealed that AKI rates were significantly higher both in high CVP (p: 0.008) and high PVP (p: 0.05) groups (Figures 4, 5).

7 Patients were received invasive mechanical ventilation and AKI is found more frequent in the patients receiving invasive MV (p: 0.01).

Only 4 patients were received hemodialysis. Other patients with AKI treated hydration. There was no significant difference between AKI and non-AKI groups in means of albumin levels.

Figure 4: AKI rate was significantly higher in high CVP (p: 0.008) group.

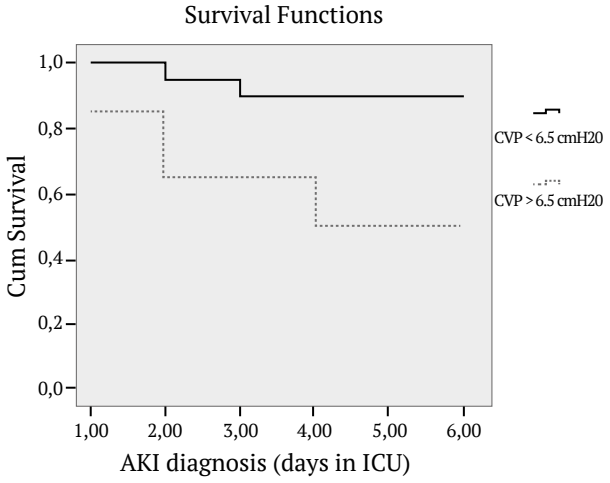
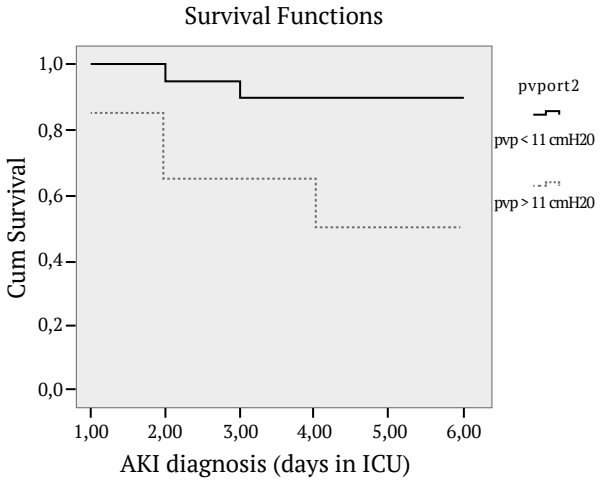


Figure 5: AKI rate was significantly higher in high PVC (p:0.05) group



Discussion

In this observational study including a group of ICU patients who were followed for at least 6 days and had no prior kidney failure history, we observed that PVP measurements were strongly correlated with CVP and both measurements were related with AKI.

In literature there are contradicting findings about the relationship between CVP and PVP. Yet there is a tendency in many authors to advice PVP as an alternative and accurate method that estimates CVP with less complication risk. Previously Kim et al analyzed data of 42 adult patients undergoing laparoscopic colorectal surgery and observed that CVP measurements were significantly lower than PVP measurements and there was a strong positive correlation between overall CVP and PVP (6). Similarly Memtsoudis et al, who analayzed data of 40 patients that underwent posterior spine surgery and reported that theses two measurements were strongly correlated (1). In a group of 50 pediatric surgery patients Anter and Bondok reported similar results and concluded that PVP showed a good agreement with CVP in the perioperative period and PVP monitoring may offer an alternative to direct CVP measurement for perioperative estimation of volume status and guiding fluid therapy in pediatric patients (2). Similarly Amoozgar et al reported that the aforementioned linear regression equation based on measurement of PVP gives a reliable estimate of CVP (7). Sahin et al reported that in patients under general anesthesia PVP measured from hand dorsum was still strongly correlated with CVP and concluded that catheter place is not an important factor that interferes with the relationship between these two parameters (8). As a contradictory finding Leipoldt studied in a group of 30 pediatric patients and reported, that despite of a strong correlation, PVP measured from an iv catheter in the hand predicts CVP poorly in pediatric patients (3).

In a recent ICU study Stoneking et al evaluate alternative methods to measure CVP to assess volume status, PVP and stroke volume variation (SVV) in a group of 20 patients that were been followed up for 1 hour in an ICU or emergency department (9). In this study authors concluded that PVP and SVV were mo-

derately good predictors of CVP, however as a weakness, study included a small group of patients with a short follow-up duration.

As can be seen above, most of the studies in literature seem to assess corelation between PVP and CVP in patients undergoing surgery with a follow-up duration usually less than 12 hours. In accordance to many of previous studies we also observed a strong correlation between CVP and PVP. As our knowledge our study has no match in literature as we followed a group of patients requiring ICU for 6 consecutive days and observed a relationship between increased venous pressure markers (CVP and PVP) and AKI. Depending on literature and our findings we recommend PVP as a reliable marker for CVP and venous congestion with less risk of complication and no need for central catheterization.

Central venous pressure is a good marker of venous congestion and is found to be increased in situations that are associated with low arterial blood pressure and venous hypervolemia. Chen et al (10) analyzed data of 86 septic shock patients and observed that in patients with high CVP levels (> 10 mmHg) AKI incidence was significantly higher comparing to patients with low CVP (75.6% vs 51.2%). Mortality rates were also significantly higher in patients with high CVP values (44.4% vs 22%). Depending on these findings authors concluded that a high CVP might increase the incidence and morbidity of AKI in septic shock and an excessively high CVP should be prevented. We also observed a similar finding for both increased CVP and PVP. According to our data when increased CVP value was defined as the median value (6 mmHg) we observed that in patients with increased CVP, AKI prevelance was significantly higher (52.6% vs 10%, p: 0.004). When a median value of 11 mmHg was accepted as a threshold for PVP we observed a similar result (47.6% vs 11.1%, p: 0.014).

We observed a tendency for decreased MAP in patients with AKI. Decreased arterial pressure results in decreased tissue oxygenation and kidney is the first and one of the most vulnerable organ to hypoperfusion. Acute tubular necrosis and ARF usually follows decreased arterial pressure of any cause. Yang et al reported increased incidence of AKI with different levels of MAP; 76.2% (90 - 100 mm Hg), 71.8% (80 - 90 mm Hg) and 94.4% (65 - 80 mm Hg) respectively (11). Despite of associations between increased venous congestion (CVP, PVP) and decreased arterial filling (MAP) markers and AKI seperately we did not observe any correlation between CVP / PVP and MAP. There was also no relationship between these three parameters and urine outputs.

As a contradictory finding Uthoff et al reported that both increased and decreased CVP values might be associated with AKI (12). However this study was conducted in acute heart failure patients that have different underlying pathophysiological mechanisms. In these patients both decreased MAP and venous pressure should clinically be interpreted as hypovolemia. However in our study none of the patients had previous or acute heart failure history and increased venous pressure was accompanied by decreased MAP and urine output. We think that these findings are secondary to fluid retention associated with cardiovascular collaps.

As a conclusion we think that PVP is a good and reliable marker of CVP in ICU patients. We recommend to use PVP as a volume status marker in these patients to decrease hemorrhagic or other complications related to central catheterization. We also observed that both increased PVP and CVP are important predictors of AKI and should be avoided if possible.

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