ORIGINAL ARTICLE

Abdominal perfusion pressure is superior from intra-abdominal pressure to detect deterioration of renal perfusion in critically Ill patients

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ABSTRACT

BACKGROUND: Intra-abdominal hypertension (IAH) is a frequent cause of acute kidney injury (AKI) among critically ill patients who have risk factors. This study aimed to determine the relation between Abdominal Perfusion Pressure (APP) and AKI showed by the Doppler-based renal resistive index (RRI).

METHODS: In this study, 38 patients older than 18 years old who received mechanical ventilation and had risk factors for the development of IAH were prospectively studied. All measurements and parameters were divided into two groups according to renal dysfunction (Group I: RRI <0.72 vs Group II: RRI >0.72).

RESULTS: The mean IAPs were not significant between the groups, 11.5±6.9 mm Hg in Group I (n=35) and 13.5±5.8 in Group II (n=33), respectively. APPs were statistically higher in Group I (81.2±13.6) than Group II (66.4±9.5) (p<0.001). The AUC for the association between APP at RRI >0.72 was 0.802 (p<0.001), with the APP ≤72 mmHg having a sensitivity of the 76% (95% CI 58–89%) and a specificity of 71% (95% CI 54–85%).

CONCLUSION: Our findings suggest that an APP with a threshold of ≤72 mmHg is associated with a significant increase in renal RRI, which may be predictive of worsening of renal perfusion.

Keywords: Abdominal perfusion pressure; renal perfusion pressure; renal resistive index.

INTRODUCTION

Intra-abdominal hypertension (IAH) and abdominal compartment syndrome (ACS) have been shown to occur frequently and independently associated with morbidity and mortality among critically ill patients.[1] Given that both of them are associated with increased morbidity and mortality, identification, avoiding, or management of these conditions may improve patient outcomes.

The intra-abdominal pressure (IAP) measurement is a key point in diagnosing and managing critically ill ICU patients who have risk factors for the development of IAH and ACS. [2] IAP measurements are easy to perform and should be used routinely in daily follow ups of high-risk ICU patients. Normal IAP values are considered around 10 mm Hg in critically ill patients. IAH has accepted an increase in IAP ≥12 mmHg and ACS is defined elevation of IAP >20 mmHg with new onset of organ failure. Abdominal perfusion pressure (APP) is the difference between the MAP and IAP, a more accurate marker of resuscitation endpoint in patients with IAH. The APP correlates with visceral perfusion above the 60 mm Hg seems a good approach to maintain macro and microcirculation.[3]

An increase of the IAP can bring on several deleterious pathophysiologic consequences, which include vascular compression that reduces perfusion and venous drainage of intra-abdominal and other organs. Although mechanisms are not yet

completely understood, systemic effects of IAP may result in life-threatening for critically ill patients.[4] Increased IAP leads to many organ dysfunctions, mainly renal, cardiovascular, respiratory, gastrointestinal, and hepatic systems are affected.

The kidneys are more vulnerable to IAH than other organs due to the deleterious mechanical effects of the increased IAP in the blood supply. The etiology of renal dysfunction and probably one of the most important ones is diminished renal blood flow. Increment of renal vessel resistance occurs due to high IAP, which causes a decrease of microcirculatory flow in the renal cortex. This stimulates vasoconstriction of renal vessels with activation of the renin-angiotensin system that results in further diminished renal perfusion.[5]

Little evidence is known about renal monitoring during IAH. It is a frequent cause of acute kidney injury (AKI) in ICU and not commonly recognized by an intensivist. Hence, the identification and early recognition of patients in whom renal hemodynamics deteriorate are fundamental during critical care stay for achieving good clinical outcomes.[6] Renal resistive index (RRI), measured by renal interlobar artery Doppler ultrasonography, is a successful non-invasive bedside monitoring modality which directly reveals and quantifies modifications in renal vascular resistance. This method enables repeated assessments of the renal circulation at the bedside with high diagnostic accuracy following various therapeutic interventions in critically ill patients.[7] It has been widely performed monitoring parameter that is gaining more frequent use in daily ICU practice. RRI values are usually obtained by translumbar or transabdominal Doppler approach, but measurements are also accessible with the transesophageal echocardiography.[8] RRI >0.72 allows earlier detection of AKI before other biochemical parameters increase, such as creatinine. Thus, careful fluid management and hemodynamic adjustments with avoiding the use of nephrotoxic medication are possible.[9]

The primary purpose of this prospective single-center study was to investigate the relationship between APP and AKI showed by Doppler-based RRI. The secondary goal was to examine the correlation between the clinical and laboratory characteristics of the patients who had this pathologic condition.

MATERIALS AND METHODS

Data Collection

Age, gender, APACHE II (Acute Physiology and Chronic Health Evaluation) and SOFA (Sequential Organ Failure Assessment) scores were recorded. Simultaneous IAP, APP, Mean Arterial Pressure (MAP), cardiac output, cardiac index, heart rate, serum creatinine, lactate levels, mechanic ventilatory parameters; positive end-expiratory pressure (PEEP), Ppeak, FiO_2 and PaO_2 which were measured with arterial blood sampling (PaO_2/FiO_2 [PF] ratio) at baseline and at 24th hours were noted.

Patient Population

This prospective study was conducted between March 2018 to March 2019 in the Intensive Care Unit of the Marmara University Pendik Education and Research Hospital. The Clinical Research Ethics Committee of the Marmara University approved the study (registration number: 09.2018.105). All participants or legal representatives signed an Informed Consent Form.

In total, 38 mechanically ventilated with deep sedation (6 points on the Ramsay scale) ICU patients (>18 years old) who had risk factors for the development of IAH and ACS and expected to stay >24 h were included in this study. Exclusion criteria consisted of pregnancy, heart failure, non-sinus cardiac rhythm that affected renal resistive index, chronic renal failure or renal artery disease (unilateral kidney, renal stone disease, renal artery stenosis or having a contraindication for intravesical pressure measurement, eg. pelvic fracture, hematuria, or neurogenic bladder).

RRI ≥0.72 has been considered as a marker of renal dysfunction and acute kidney injury.[9] We divided all measurements into two groups, according to RRI. RRI <0.72 measurements were grouped as Group I (n=35). Group II (n=33) measurements included RRI above >0.72.

According to the Consensus definitions of the WSACS (www.wsacs.org), IAH is defined by the sustained or repeated elevation of IAP >12 mmHg. Abdominal perfusion pressure (APP) was calculated by subtracting the IAP from the mean arterial pressure (MAP): APP=MAP–IAP.[3]

IAP Measurement Techniques

The intra-vesicular pressure was measured through a closed-system Foley bladder catheter. It was measured at the end of the expiration with the patient in the supine position and the transducer placed at the mid-axillary line where it crosses the iliac crest. A transducer-based needle with an interposition T-piece was used directly to canulate the urinary catheter directly. The transducer (Pressure Set, Sasan Medical Disposable Products, Ankara, Turkey) was used to connect the system to bedside monitor (IntelliVue MX550, Philips Healthcare, Inc., Andover, MA, USA). The urinary drainage tube was clamped and a maximum of 25 mL of saline was instilled into the bladder through a foley catheter. Then, zero pressure on the monitor was achieved. IAP was measured on the monitor with stopcocks open to a pressure transducer, after a one-minute equilibration period. To the extent possible, the IAP was measured twice a day (basal and then at 24 h).

RRI and Cardiac Output (CO) Measurements

RRI measurements were performed using a Philips EPIQ 7 ultrasound system (Philips Healthcare, Inc., Andover, MA, USA) by a trained intensivist who is certified in ultrasonography.

Renal Doppler was performed on the interlobar arteries using a convex probe. Renal vasculature was identified using color Doppler, and then, the arterial waveforms were obtained by Doppler in the interlobar renal artery. RRI at the interlobular or arcuate artery near the border of the central echo complex was measured three times in the upper, middle, and lower portions of the kidney. In each patient, the following formula was used to calculate RI: (RRI = [peak systolic velocity – end-diastolic velocity]/peak systolic velocity). Three measurements were performed and the mean value of three measurements at each kidney calculated.[8]

Non-invasive Ultrasonic Cardiac Output Monitor (USCOM; USCOM Ltd., Sydney, Australia, 2005) was used to measure cardiac output transcutaneously via a probe applied to the suprasternal notch.

Statistical Analysis

Continuous parametric and nonparametric variables were presented as the mean (standard deviation) and median (25th; 75th percentiles) and were compared using the t-test and Mann–Whitney test, respectively. Categorical variables were expressed as absolute (n) and relative (%) frequency and were compared by the Chi-square test. The normality of variables was tested using the Shapiro-Wilk test for normality. Receiver operating characteristic curves were generated to test the predictive discrimination threshold of impaired renal perfusion (RRI ≥0.72) and normal renal perfusion (RRI <0.72) to APP. The area under the ROC curve (AUC) was calculated and compared using a Hanley- McNeil test. The optimal threshold value (the value that maximizes the sum of the sensitivity and specificity) was also defined for APP. The criterion associated with the Youden index is reported with its 95% CI based on bootstrapping using 1000 replications. The sensitivity and specificity values are also reported with the 95% CI. Significance was assumed for a two-sided p-value <0.05. All statistical analyses were performed using SPSS 21 (IBM SPSS Statistics, Chicago, IL, USA) and MedCalc 14 software (MedCalc, Mariakerke, Belgium). For all comparisons, a p-value of less than 0.05 was considered significant.

RESULTS

We enrolled 38 patients (17 female) fulfilling inclusion/exclusion criteria. In total, 68 IAP and RRI measurements were performed. Heart rate, cardiac output, cardiac index, P/F ratio, PEEP, Ppeak, lactate levels, pH and SpO$_2$ levels were comparable between the groups. The patients' clinical and laboratory parameters are reported in Table 1. In addition to this, the characteristics and diagnosis of the patients at inclusion who had risk factors for the development of IAH and ACS are shown in Table 2.

The mean of IAPs were not significant between the groups [11.5±6.9 mmHg in Group I (n=35) vs 13.5±5.8 in Group II

Table 1. Comparison of clinical and laboratory characteristics with and without acute kidney injury

	Group I (n=35)	Group II (n=33)	p
IAP (mmHg)	11.5±6.9	13.5 ±5.8	0.184
MAP (mmHg)	93±13	80±9	<0.001
APP (mmHg)	81.2±13.6	66.4±9.5	<0.001
Heart rate (bpm)	95±18	97±14	0.692
Cardiac output (L/min)	4.9 ±1.7	4.0±1.7	0.054
Cardiac index (L/min/m^2)	2.4±0.9	2.3±1.2	0.415
P/F ratio	302±99	340±189	0.309
PEEP (cmH$_2$O)	6.6±1.8	7.2±2.1	0.173
Ppeak (cmH$_2$O)	26.8±6.1	27.2±7.1	0.812
SpO$_2$ (%)	97.7±1.9	98.8±1.9	0.058
Serum creatinin (mg/dL)	1.12±0.9	1.78±1.23	0.029
Ph	7.44±0.07	7.41±0.09	0.111
Lactate (mmol/L)	1.78±1.39	2.47± 2.52	0.170
Renal resistive index	0.66±0.05	0.80±0.05	<0.001

IAP: Intrabdominal pressure; MAP: Mean arterial pressure; APP: Abdominal perfusion pressure; PEEP: Positive end-expiratory pressure.

Table 2. Characteristics and diagnosis at the inclusion of the 38 patients who had risk factors for the development of IAH and ACS

Parameter	Value
Age, (years)	55.3±17.7
Sex, n (%)	
Male	21 (55%)
Female	17 (45%)
APACHE II (inclusion)	20.3±8.4
SOFA (inclusion)	8 (5–10)
Diagnosis (n)	
Intrabdominal sepsis	10
Pneumonia sepsis	7
Intracerebral hemorrhage	7
Pancreatitis	2
Hepatic encephalopathy	2
Acute myocardial infarction	3
Intraabdominal tumor	3
Others	4

The data are presented as mean±standard deviation, median [interquartile range: P25 to P75] or count (percentage). APACHE II: Acute Physiology and Chronic Health Evaluation II; SOFA: Sequential Organ Failure Assessment.

(n=33)]. MAP measurements were higher in Group I (93±13 mmHg) compared to Group II (80±9 mmHg) (p<0.001). A significant difference in APP measurements was found between
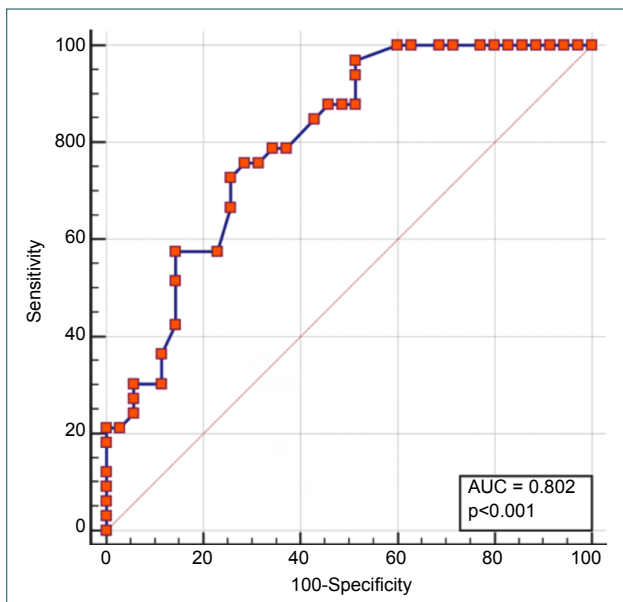
Figure 1. Receiver operating characteristic (ROC) curve for the association between the Abdominal Perfusion Pressure (APP) and an increased renal resistive index (>0.72). AUC: The area under of a ROC curve.

the groups. APPs were statistically higher in Group I than Group II (81.2±13.6 vs 66.4±9.5) (p<0.001). The AUC for the association between APP at RRI >0.72 was 0.802 (p<0.001), with the APP ≤72 mmHg having a sensitivity of 76% (95% CI 58–89%) and a specificity of 71% (95% CI 54–85%) (Fig. 1).

The serum creatinine levels were significantly higher in patients in Group II compared to Group I (1.12±0.9 vs 1.78±1.23) (p=0.029).

DISCUSSION

The principal finding of this study showed that an APP with a ≤72 mmHg threshold is associated with a significant increase in renal RRI. Thus Doppler ultrasonography with RRI calculation can be a useful method in managing these patients to determine the optimal APP level for renal perfusion. Our findings also suggest that the increase of this index represents renal impairment is possible although IAP is normal. An APP value ≤72 mmHg was the best cut-off for renal dysfunction prediction and a more reliable parameter than IAP. Thus, this threshold suggests that the kidney is a potential organ for adverse physiologic effects, so prompting intervention at an earlier stage should be started. In this study, we also reported significantly elevated creatinine levels concurrently together with the increasing levels of RRI. As such, early accurate assessment of renal dysfunction radiologically with RRI is possible and maybe essential in restoring kidney perfusion during the deterioration of APP.

IAH is an independent cause of mortality and very common in critically ill patients.[10] IAP exerts deleterious effects on various organs. Thus, accurate assessment and lowering of

IAP and establishing sufficient abdominal perfusion pressure appear essential strategies to maintain end-organ perfusion. IAH hypothetically directly or indirectly impairs nearly every organ system and is life-threatening for the critically ill patients, but the link between IAH and organ dysfunctions are not yet completely understood. Increased pressure may lead to compromised organ perfusion. For instance, cardiac contractility and ventricular function may decrease dramatically by reducing the preload of the heart. The presence of IAH is associated with an increase of intrathoracic pressures, and proportionally peak and plateau airway pressures. Transmission of abdominal pressures to the thoracic cavity may affect mainly respiratory mechanics.[11,12] Augmented airway pressures are necessary to overcome resistance in the airway due to increased rigidity of the thoracic wall. In addition to this, it was proposed that patients having a peak airway pressure >28 cm H_2O are more likely to have IAH.[13] The hypoxemia and hypercapnia may occur as a result of mechanic compression of the lungs. Impair perfusion of the liver may result in elevated serum lactate levels, and it may also give rise to the disturbance of cerebral perfusion.[14]

The APP is a new parameter that shows the circulatory compromise in the abdominal cavity. The APP appears as a better resuscitation endpoint compared to other macro and microcirculatory parameters and recommended by the current literature.[15] More importantly, it is associated with visceral perfusion instead of IAP alone if feasible and has sensitivity for decision making. Evidence suggested that it is superior to IAP or other hemodynamic parameters to predict mortality.[16] A study found that APP is better than MAP and lactate to discriminate survivors from nonsurvivors.[17] As APP <60 mmHg was considered as an indicator of abdominal hypoperfusion,[18] but at which APP early renal impairment occurs, radiologically remains unclear. In our study, the cut-off point APP ≤72 mmHg was found to be an indicator of early renal hypoperfusion because at this pressure, a significant increase in RRI was observed.

The adverse influence of IAH on renal function is often unappreciated by clinicians given that IAH is an independent cause of renal dysfunction.[19] Some new data claim that kidneys are particularly at high risk, and renal dysfunction occurs with much lower levels of IAP among critically ill patients.[20] IAH is still the cause of or contributing factor in AKI. Hence, in the early time, to find the etiology and management of critically ill patients with raised IAP should be concerned to prevent and avoid progression. IAH is associated with many negative effects on the kidneys that arise from multiple factors. It assumes that two main mechanisms during increased IAP, indirectly (systemic effects) or directly (renal effects), may affect kidney function.[21]

The kidneys are physiologically at risk of IAH mainly due to its susceptibility vasculature nature, but mechanisms underlying the vulnerability are not fully known.[22] The mechanisms

involved in the pathophysiology are complex, and one of the most consistently described effects is related to renal blood flow. It was suggested that renal vein compression with renal artery vasoconstriction seems to be the major cause of renal impairment.[5] In addition, direct compression of the renal cortex results in a decrease in blood flow. A large prospective observational study established IAH and low APP resulted as the best predictive factors for ARF.[23] Some studies concluded that an IAP 15–20 mmHg causes in oliguriaand anuria develops above 30 mmHg.[24] Besides, the rate of renal impairment can be raised according to the level of IAP; the incidence is doubling at 25 mmHg compared to 18 mmHg.[19] Moreover, Dalfino et al.[23] demonstrated that an association between IAH and acute kidney injury (AKI) in unselected ICU patients, IAH was a good predictor of AKI. It has also has been suggested that there is a clear link between IAH and the development of AKI in the kidney transplant patient. Early transplant dysfunction was treated using abdominal decompression in those patients.[25] However, in our study, we showed that elevated RRI and high levels of creatinine are possible with normal IAP.

Despite increasing interest in the use of ultrasound in critically ill patients, there has been a little report in the clinical examination of the patient with IAH. There are very limited data concerning aiming ideal APP to prevent acute kidney injury, and there is little evidence of radiologic measurement to show these subtle forms of organ dysfunction during IAH. RRI has been used for years in a variety of clinical settings, especially became apparent to differentiate acute and chronic obstructive renal disease. RRI provides useful information about changes in intrarenal perfusion and has been proposed to monitor renal perfusion in critically ill patients.[8,26,27] It was shown that this index might be affected by IAH and allowed earlier detection of renal impairment before the increase of other biochemical parameters.[9] In a porcine model, it was demonstrated that a linear relationship between increasing IAP and RRI and the author concluded that it might be a potential noninvasive, bedside screening tool to detect early deterioration of renal perfusion.[28] In 45 healthy volunteers, a mild increase in IAP compresses the major abdominal vessels was associated with a significant increase in RRI, suggesting that even mild IAH may affect intrarenal pressure.[5] However, in our study, the cut-off point of APP ≤72 mmHg is associated with a statistically significant increase of RRI suggesting that supporting renal function to maintain an APP >72 mmHg is to be advocated. Our findings suggest that an ideal APP determination is important and may be useful to prevent acute kidney injury.

Conclusion

Kidneys seem to be the most susceptible organ to the adverse consequences of increased IAP. Increased RRI may be a useful index for objective evidence of impending kidney injury that occurs before other end-organ dysfunctions. The results of our study show that an APP with a threshold of ≤72 mmHg is associated with a significant increase in renal RRI, which is predictive of worsening of renal perfusion. More importantly, our results suggest that kidneys may be particularly at risk with much higher levels of APP than believed previously. Measuring RRI using a noninvasive bedside renal Doppler ultrasound is a valuable tool to detect deterioration of renal perfusion, considering the importance of APP. In conclusion, it has an active role in helping to prevent potentially fatal complications of IAH/ACS and should be integrated while treating critically ill patients who have risk factors.

Conflict of interest: None declared.

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ORİJİNAL ÇALIŞMA - ÖZET

Kritik hastalarda abdominal perfüzyon basıncı renal perfüzyondaki bozulmanın saptanmasında karıniçi basınçtan daha üstündür

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AMAÇ: Karıniçi hipertansiyon (KH) risk faktörü olan kritik hastalarda akut böbrek hasarının (AKI) en sık nedenlerinden biridir. Bu çalışmada abdominal perfüzyon basıncı (APP) ile Doppler temelli renal rezistif indeks (RRI) ile gösterilen AKI arasındaki ilişkiyi saptamayı amaçladık.

GEREÇ VE YÖNTEM: Bu çalışmaya KH için risk faktörü taşıyan ve mekanik ventilasyon desteği altında 18 yaşından büyük 38 hasta alındı. Tüm ölçümler ve elde edilen parametreler renal disfonksiyona göre iki gruba ayrıldı (Grup I; RRI <0.72 ve Grup II; RRI >0.72).

BULGULAR: Grupların ortalama karıniçi basınçları (IAB) Grup I'de (11.5±6.9 mmHg, n=35) ve Grup II'de (13.5±5.8, n=33) benzerdi. APP; Grup I'de (81.2±13.6) Group II'ye göre (66.4±9.5) daha yüksekti (p<0.001). RRI'ya göre renal perfüzyondaki bozulma APP'nin (≤72 mmHg) belirlemdeki duyarlılığı %76 ve seçiciliği %71'dir. ROC eğrisi altında kalan alan 0.802'dir (p<0.001).

TARTIŞMA: Çalışmamızın sonuçları APP ≤72 mmHg eşik değerinde RRI'nın anlamlı şekilde arttığını ve bunun da renal perfüzyonun bozulmasını öngörebileceğini göstermiştir.

Anahtar sözcükler: Abdominal perfüzyon basıncı; renal perfüzyon basıncı; renal resistif indeks.