Review Article

Haemodynamic predisposition to acute kidney injury: Shadow and light!

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Abstract

Acute kidney injury (AKI) could well be regarded as a sentinel complication given it is relatively common and associated with a substantial risk of subsequent morbidity and mortality. On the aegis of 'prevention is better than cure', there has been a wide interest in evaluating haemodynamic predisposition to AKI so as to provide a favourable renoprotective haemodynamic milieu to the subset of patients presenting a significant risk of developing AKI. In this context, the last decade has witnessed a series of evaluation of the hypotension value and duration cut-offs associated with risk of AKI across diverse non-operative and operative settings. Nevertheless, a holistic comprehension of the haemodynamic predisposition to AKI has been a laggard with only few reports highlighting the potential of elevated central venous pressure, intra-abdominal hypertension and high mean airway pressures in considerably attenuating the effective renal perfusion, particularly in scenarios where kidneys are highly sensitive to any untoward elevation in the afterload. Despite the inherent autoregulatory mechanisms, the effective renal perfusion pressure (RPP) can be modulated by a number of haemodynamic factors in addition to mean arterial pressure (MAP) as the escalation of renal interstitial pressure, in particular hampers kidney perfusion which in itself is a dynamic interplay of a number of innate pressures. The present article aims to review the subject of haemodynamic predisposition to AKI centralising the focus on effective RPP (over and above the conventional 'tunnel-vision' for MAP) and discuss the relevant literature accumulating in this area of ever-growing clinical interest

Keywords: Acute kidney injury, central venous pressure, congestive renal failure, effective renal perfusion pressure, haemodynamics, mean arterial pressure

Introduction

"The eye is always caught by light, but shadows have more to say." We commence our review on this clinically pertinent topic with a succinct quote of renowned American novelist, Gregory Maguire, in our endeavour to unveil a holistic haemodynamic predisposition to acute kidney injury (AKI) in the times witnessing a paradigm shift from a pressure-centric to a perfusion-centric management approach.

AKI occurs in 5-7.5% of the hospitalized patients with the incidence reaching up to 12% following major operative procedures, and 20% in the critically ill patients in the intensive care unit (ICU) setting.[1-5] Although the incidence of postoperative AKI is greatly influenced by the underlying operative procedure and the classification system employed (RIFLE criterion and the staging established by

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the Acute Dialysis Quality Initiative (ADQI) and Acute Kidney Injury Network (AKIN)),[6-8] certain predisposed settings such as cardiac surgery associated-AKI (CSA-AKI) are associated with 5-42% AKI incidence, representing a significant cause of AKI in the ICU, second only to sepsis.[9-11]

The Table 1 summarizes the predispositions to a heightened risk of AKI. The development of AKI is independently associated with adverse clinical outcomes, including an elevated mortality rate (escalates ICU-mortality by 50-60%) and contributes to an enhanced progression to chronic kidney disease, duration of ICU stays and thereby, imposes a huge burden and unrealistic challenge on the health care infrastructure.[1,12,13]

Search Strategies

The non-systematic review was planned after comprehensive analysis of the literature from textbooks, journals, internet resources using keywords "Acute kidney injury", "haemodynamic", "congestive renal failure", "central venous pressure", "effective renal perfusion pressure", "mean perfusion pressure", "diastolic perfusion pressure", and "mean arterial pressure". The filters were case reports, clinical trials, controlled trials, randomised control trials, observational study and text articles using search engines such as PubMed, EMBASE, Medscape, Google Scholar Medline Scopus, Science Direct and many others.

Risk modulation: *The Pivotal Role of Haemodynamics....*

Despite ongoing improvements in perioperative care, AKI continues to present a steady management concern to the physicians leading to the requirement of renal replacement therapy (RRT) in severe AKI and an attributable eight-fold escalation in the odds of mortality.[14] Considering a wide gamut of potential perioperative ischaemic renal insults, there is an ever-increasing focus on the risk-factor identification and favourable management modulation as the cornerstone of AKI management.[15]

In this context, a range of research reports on the association of intraoperative hypotensive episodes with postoperative renal insult following cardiac and non-cardiac surgical, and other critically ill cohort have promoted the interest of the fraternity in evaluating haemodynamic optimization as a potentially modifiable AKI risk factor.[5,16-21] The specific associations of the hypotensive epochs with Kidney Disease Improving Global Outcomes (KDIGO) stage I injury are presented in Table 2.[5,16-21]A very recent description of post-cardiopulmonary bypass mean arterial pressure (MAP) <65 mmHg for ≥ 10 minutes entailing a heightened risk of de novo RRT in the postoperative period in a retrospective single-centre cohort study involving 6,523 patients by Ngu *et al.*,[22] adds to the aforementioned literature on the pivotal role of haemodynamics in modulating the involved AKI risk.

Renal Perfusion Pressure (RPP): *The Concept*

Akin to the cerebral perfusion and myocardial perfusion dynamics,[23]wherein there is a pressure head promoting the downstream perfusion (MAP in the case of cerebral perfusion and diastolic arterial pressure (DAP), in the case of myocardium) and a pressure head impeding the net perfusion (intracranial pressure in the case of cerebral perfusion and left-ventricular end-diastolic pressure, in the case of myocardium), the mere assurance of a sufficient MAP cannot ensure an adequate renal perfusion. Lack of acknowledgement to the pressure forces which impede renal perfusion has led to the subsequent underemphasized

Table 1: The Subset of Patients Predisposed to Acute Kidney Injury

Pre-existing co-morbidities (advanced age, DM, HT, CKD, anaemia, PVD, major atherosclerotic disease) or contrast-enhanced preoperative investigations
Post-major non-cardiac surgery (particularly, major vascular surgeries or transplantation procedure with major intraoperative hypotension, significant fluid shifts, massive blood and blood product transfusion, intravascular haemolysis due to mismatched blood transfusion, etc.)
Critically ill hypotensive patients on vasopressor support
Septic shock
Post-cardiac surgery (extracorporeal circulation, embolic phenomenon, haemodilution, etc.)
Congestive cardiac failure (Left, right or biventricular heart failure)
Cardio-renal syndrome
Acute respiratory distress syndrome
Hypovolemic shock
Cardiogenic shock
Perioperative myocardial infarction
Abdominal compartment syndrome
Mechanical circulatory assistance (ECMO, IABP, VAD)
Nephrotoxic drugs

CKD: chronic kidney disease; DM: diabetes mellitus; HT: hypertension; PVD: peripheral vascular disease; ECMO: extracorporeal membrane oxygenation; IABP: intra-aortic balloon pump; VAD: ventricular assist device

concept of the renal perfusion pressure (RPP). Moreover, kidney being an intra-capsular organ, the combination of the underlying intra-capsular and extra-capsular forces present a potential threat to renoprotection by compromising RPP, and simultaneously mitigation of these also provide an opportunity to improve renal outcomes in addition to the conventional notion of optimizing volume status in order to augment cardiac output to ensure the adequacy of renal perfusion.[24]

Factors Affecting an Effective RPP: *As 'Effective' is 'Important'!*

From a conceptual point of view, effective RPP is intricately determined by the net resultant of the forces between the MAP or renal arterial pressure (particularly in situation of impaired renal autoregulation) and, transmitted renal venous pressure (RVP) (estimated by central venous pressure (CVP)), intra-abdominal pressure (IAP) as well as mean airway pressures generated owing to mechanical ventilation [Figure 1]. The importance of the later variables in collectively contributing to an elevated renal interstitial pressure becomes all the more important in the critically ill subset of patients. Nevertheless, the interdependency between the variables compounds the situation in most practical scenarios.

The first experimental human study aimed at deciphering the role of these forces impeding renal perfusion, was contemplated in 1947 by Bradley and Bradley, wherein the creation of a 70-80mmHg pressure inside a balloon surrounding the healthy volunteer's abdomen, resulted in an IAP of around 20 mmHg.[25] The setting escalated the RVP to 18-20 mmHg from the initial normal value of 5-6 mmHg without affecting the arterial blood pressure. Thereby, the renal plasma flow (RPF) and glomerular filtration rate (GFR) declined by 24.4% and 27.5%, respectively, subsequent to the venous stasis.[25]A few years later, Maxwell and colleagues outlined a significant rise in RVP to almost 20 mmHg in patients of congestive cardiac failure (CCF). They revealed that these patients were characterized by a 2/3rd diminution of RPF and a 1/3rd attenuation of the filtration rate compared to their healthy counterparts.[26] Patients with pulmonary hypertension with high baseline CVP values, awaiting lung transplantation and a large cohort of cardiac disease subjects also depicted a declined GFR in respective independent studies.[27,28]

Adding over and above the intra-capsular forces, IAP constitutes an extra-capsular pressure from the renal standpoint, which potentially influences the renal interstitial pressure. The renowned World Society of the Abdominal Compartment Syndrome (WSACS) proposes the subtraction

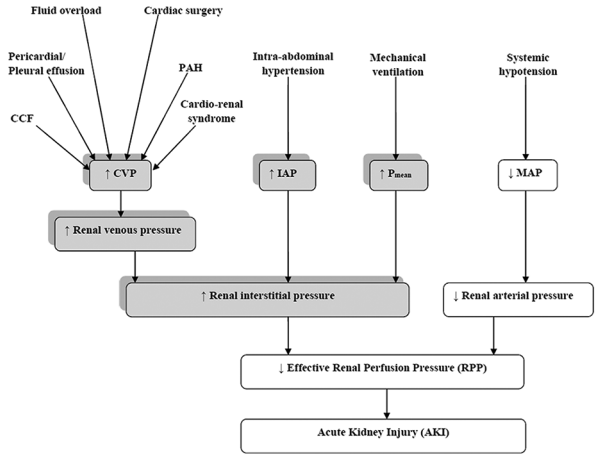

Figure 1: The illustration depicting the dynamic interplay of innate pressures in determining the effective renal perfusion pressure (RPP) wherein, the shaded parts of the figure represent the relatively under-emphasized areas in the context of haemodynamic predisposition to AKI. (CCF: congestive cardiac failure; CVP: central venous pressure; IAP: intra-abdominal pressure; P_{mean}: mean airway pressures; MAP: mean arterial pressure; PAH: pulmonary artery hypertension)

of IAP from MAP to estimate the abdominal perfusion pressure (APP) which is often employed as the surrogate for adequacy of the renal perfusion.[24] The grading of intra-abdominal hypertension (IAH) is outlined in Table 3. In circumstances wherein IAP exceeds 20 mmHg with APP declining below 60 mmHg, abdominal compartment syndrome is diagnosed with the imminent risk of organ dysfunction, particularly the kidneys.[24,29] Similarly, Moore *et al.*,[30] demonstrated the sensitivity of the renal blood flow to positive-pressure ventilation in infant primates whose renal dynamics were strikingly similar to those of the humans.

While the importance of renal haemodynamicsin the AKI-pathophysiology cannot be undermined yet on an optimistic note, most of the basic sciences explorations and the clinical investigations reflect an elevated oxygen-extraction and consumption in settings of AKI despite diminished GFR and tubular reabsorptive load.[31]However, relying on the former can be perilous in certain peculiarly predisposed subset like the diabetic counterparts where renal hypoxia is seen to exist.[32]

Relevant Literature: *Beyond a 'Tunnel-vision' for MAP*

(i) Role of CVP in predicting AKI in the critically ill
A recent 2020 meta-analysis by Chen *et al.*,[33] including 15 cohort studies with a characteristically broad spectrum inclusion of critically ill (predominantly septic and certain post-cardiac surgical subset) revealed that an elevated CVP is associated with a heightened risk of '*congestive renal failure*' and mortality in ICU patients.

Table 2: The associations of the hypotensive epochs with Kidney Disease Improving Global Outcomes (KDIGO) stage I injury.[5,16-21]

MAP <55 mmHg for 1-5 minutes
MAP <60 for >10 min
MAP <65 for 10-20 min, or
Relative MAP decrease >20% from baseline value for ≥90 min

Table 3: Grading of Intra-Abdominal Hypertension (IAH).[24,29]

Severity	Intra-Abdominal Pressure
Grade I	12-15 mmHg
Grade II	16-20 mmHg
Grade III	21-25 mmHg
Grade IV	>25 mmHg
Grade I	12-15 mmHg

Any evidence of ongoing IAH with end-organ failure is defined as abdominal compartment syndrome

On a dichotomous scale, elevation in CVP entailed an augmented mortality risk (969 participants from 3 studies; odds ratio of 1.65) and AKI (689 participants from 2 studies; odds ratio of 2.09). Whereas, on a continuous scale, escalated CVP accounted for a greater mortality risk (7837 participants from 5 studies; odds ratio of 1.10) and AKI (5446 participants from 6 studies; odds ratio of 1.14). In addition, for every 1 mmHg increase in CVP, the odds of AKI increased by 6% (5150 participants from 4 studies; odds ratio of 1.06).[33]Interestingly, a popular study by Palomba and colleagues (included in the aforementioned meta-analysis) aimed at developing the 'Acute Kidney Injury in Cardiac Surgery' (AKICS) score adequately outlined that the risk of AKI increases to 2-fold once the postoperative CVP reaches a threshold value of 14 mmHg across the cardiac surgical population.[34,35]

Moreover, independent researchers have also revealed a significant association of the estimated CVP at 6 h postoperatively to CSA-AKI even in the non-chronically congested cardiac surgical subset.[35-37]A retrospective evaluation of a considerably large database (n = 9,090 following exclusions) coined as Multi-parameter Intelligent Monitoring in Intensive Care (MIMIC-III) delineated an elevated 28-day mortality in participants with CVP > 10 mmHg in the first 72 ICU hours.[38] Despite the elucidation of the importance of the role of CVP in determining renal outcomes, a number of recent studies evaluating hypotensive predisposition to AKI connote a definite '*tunnel-vision*' by not accounting for the perioperative CVP values. A representative example of the abovementioned is the previously described study by Ngu *et al.*,[22] wherein as high as 245 out of 336 patients (72.9%) requiring RRT in their study underwent combined valvular procedures, where systemic venous congestion is a peculiar postoperative feature particularly in scenarios compounded by right heart dysfunction.

(ii) Role of novel perfusion pressure parameters as compared to MAP

A remarkably interesting set of studies have endeavoured to evaluate the deficits in the novel perfusion parameters such as mean perfusion pressure (MPP = MAP-CVP) and diastolic perfusion pressure (DPP = DAP-CVP), in comparison to the MAP deficits in predicting the progression to AKI.[39-41]An observational study by Legrand *et al.*,[42] depicted that a lower DAP and a higher CVP are more closely associated to septic AKI in comparison to the MAP.Another observational study outlined the association of a decreased MPP with AKI while MAP failed to demonstrate such an association, strengthening the role of CVP in predicting septic AKI furthermore.[39]In addition, a recent post-cardiovascular surgical evaluation by Saito *et al.*,[40] revealed a significant association between decreased MPP, DAP and DPP (and not MAP) with CSA-AKI in vasopressor dependent patients. The decline in DPP resulted owing to the elevation of CVP in 23.8% of the study subjects and was contributed by a diminished DAP in the rest 76.2%.Jin and colleagues also highlighted the independent association of the postoperative nadir DPP with CSA-AKI ahead of MAP in their retrospective analysis of 300 surgical patients.[41]

(iii) Role of IAP and mechanical ventilation in predicting AKI

A range of perioperative studies have employed APP as a surrogate for RPP.[29,43]Demarchi*et al.*,[44] outlined the importance of IAP in predicting postoperative AKI following abdominal surgeries.The incidence of IAH in the first 72 ICU hours in setting of septic shock has also been demonstrated to be associated with an escalated risk of AKI.[45]With regards to the effect of the positive pressure ventilation on AKI, a retrospective cohort study by Tojo and colleagues outlined that an intraoperative low tidal volume ventilation is associated with a reduced incidence of AKI following cardiovascular surgery.[46]Out of the 338 included participants, a total of 105 study participants developed postoperative AKI. In the patients stratified to receive <7, 7-8, 8-9, and >9 mL/kg predicted body weight (PBW) mean tidal volume, the cumulative AKI incidence was discovered to be 12.8%, 29.9%, 38.7%, and 34.5%, respectively.[46]Argalious *et al.*,[47] also revealed an estimated AKI odds ratio of 1.05 (confidence interval: 1.02-1.08, $P = 0.001$) for every mL increase in the tidal volume per kilogram PBW

in their very recent evaluation of the association between the intraoperative tidal volumes and AKI.Moreover, a recent evaluation of a large cohort of ICU patients from theMIMIC-III database outlined a strong relationship between mechanical ventilatory settings and worsening of the renal function. Interestingly, this relationship was intricately linked to the renal venous congestion emanating as a result of the underlying cardio-pulmonary-renal interaction.[48]

Practical considerations and the future directions

Despite the elaboration upon the concept, importance and the factors regulating RPP, there is a dearth of literature on the practical application of the effective RPP in the context of AKI.[49,50]Kopitkó and colleagues are credited for their evaluation of the collaborative value of haemodynamics, IAP and mean airway pressure (P_{mean}) monitoring in predicting AKI in the first 12 hours following major abdominal surgery wherein a neoteric formula for the effective RPP being equivalent to MAP- (CVP + IAP + P_{mean}) was employed by the research group.[49]In a cohort of 84 patients, the group discovered significant RPP differences between the AKI and the non-AKI groups at 12 h post-ICU admission (median and interquartile range: 40 (36-52) v/s 57 (42-64); $P < 0.05$). Below the median RP*P* value of 40.7 mmHg, postoperative AKI developed in all the study patients.[49]The same group of researchers compared the AKI predictive value of a range of permutation for the RPP formulas wherein, the aforementioned formula [MAP- (CVP + IAP + P_{mean})] revealed the highest sensitivity and specificity.[50]

Although the effective RPP computed using this formula can be monitored continuously, enabling timely optimization of the haemodynamic therapies and mechanical ventilation, the predictive value requires additional validation in more diverse and larger subset of the patient populations.[50]In addition, the suitability of the formula in predicting the renal recovery following the initiation of RRT, (either in the setting of conventional haemodialysis or continuous RRT) and the value of the computed effective RPP in the de-resuscitative phase of septic shock remain to be established.

While the relevance of the venous circulation to the overall renal perfusion dynamics continues to be increasingly recognised, novel imaging parameters such as a renal ultrasound based evaluation of the venous impedance index (VVI = maximum velocity − minimum velocity/maximum velocity, described initially in CCF) can provide exciting future avenues given the renal veins portray a high capacitance with pulsations synchronous with the phases of respiration and the right atrial contraction.[24,51]

Conclusion

To conclude, the renal haemodynamics and optimum oxygenation play an important role in the genesis of AKI, albeit the concept of effective RPP is underemphasized in the existing literature which continues to simplistically provide magical cut-offs of MA*P* values and duration in predicting AKI. Not only the heterogeneous settings confound the extrapolation of these MAP cut-offs [52], the interplay of other innate pressures such as CVP, IAP and P_{mean} can also significantly alter renal perfusion in the critically ill. Therefore, an individualized haemodynamic optimization aimed at favourably modulating the effective RPP,[53-55] in order to minimize the overall AKI incidence, is certainly the need of the hour, particularly pertinent in this era of precision medicine.

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Conflicts of interest
There are no conflicts of interest.

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