INTRA-ABDOMINAL HYPERTENSION AS A RISK FACTOR FOR ACUTE KIDNEY INJURY IN CRITICALLY ILL PATIENTS

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ABSTRACT

BACKGROUND AND AIMS

Increased intra-abdominal pressure (IAP), also referred to as intra-abdominal hypertension (IAH), affects organ function in critically ill patients. The prevalence of IAH is between 32% - 65% in intensive care units. Normal IAP is ≈ 5–7 mmHg. According to WSACS definition, IAH = IAP ≥12 mmHg and is divided into 4 grades. They are Grade I (12-15 mmHg), Grade II (16-20 mmHg), Grade III (21-25 mmHg), Grade IV (>25 mmHg). Transvesical measurement of IAP currently is the most popular technique. Several systems with or without the need for electronic equipment are available that allow IAP measurement. The aim is to study the incidence of IAH in critically ill patients, to assess the risk factors for development of IAH, to study the role of IAH as a risk factor for Acute Kidney Injury (AKI), to assess the role of IAH as a risk factor for increased (Intensive Care Unit) ICU mortality.

SUBJECTS AND METHODS

This is a prospective observational study. Study period was six months. The study included 52 patients admitted to Medical ICU in Government Medical College, Kozhikode, Kerala.

RESULTS AND CONCLUSION

There was a very high incidence of intra-abdominal hypertension in critically ill patients. IAH was significantly associated with risk factors like sepsis, mechanical ventilation, pancreatitis, capillary leak, ascites, cumulative fluid balance and cirrhosis. IAH is an independent risk factor for development of acute kidney injury. IAH is an independent predictor of mortality in critically ill patients.

KEYWORDS

Abdominal Compartment Syndrome, Acute Kidney Injury, Critically Ill Patient, Intra-abdominal Hypertension, Intravesical Pressure Monitoring.

HOW TO CITE THIS ARTICLE: Melemadathil S, Puthiyottil NT, Krishnan JE. Intra-abdominal hypertension as a risk factor for acute kidney injury in critically ill patients. J. Evid. Based Med. Healthc. 2016; 3(38), 1899-1901.
DOI: 10.18410/jebmh/2016/422

INTRODUCTION: Increased intra-abdominal pressure (IAP), also referred to as intra-abdominal hypertension (IAH), affects organ function in critically ill patients and may lead to abdominal compartment syndrome (ACS). Although initially described in surgical patients, IAH and ACS also occur in medical patients without abdominal conditions. IAP can be measured easily and reliably in patients through the bladder using simple tools.

The effects of increased IAP are multiple, but the kidney is especially vulnerable to increased IAP because of its anatomic position. Although the means by which kidney function is impaired in patients with ACS is incompletely elucidated, available evidence suggests that the most important factor involves alterations in renal blood flow. IAH should be considered as a potential cause of acute kidney injury in critically ill patients.

Its role in other conditions, such as hepatorenal syndrome, remains to be elucidated. Because several treatment options (both medical and surgical) are available, IAH and ACS should no longer be considered irrelevant epiphenomena of severe illness.

The harmful consequences of increased IAP initially were reported more than 100 years ago, and effects on the kidney were among the first described. In 1876, Wendt reported that an increase in IAP was associated with a decrease in urine output, and in 1947, Bradley and Bradley published a comprehensive experimental article describing the effect of IAP on kidney perfusion and function. Several investigators have since noted similar effects in animal models and clinical studies in the critically ill. Presumably because measurement of IAP was cumbersome and clinicians were unaware of the dangers, clinical effects of IAP were not reported again until the early 1980s. It was not until the landmark report by Kron et al, which reported that IAP could be monitored objectively and relatively easily through an indwelling intravesical catheter, that more

Financial or Other, Competing Interest: None.
Submission 31-03-2016, Peer Review 13-04-2016,
Acceptance 23-04-2016, Published 12-05-2016.
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DOI: 10.18410/jebmh/2016/422

clinical evidence was rapidly forthcoming concerning the deleterious effects of increased IAP on different organ systems.

IAH is defined as sustained or repeated IAP ≥12 mmHg and is divided into 4 grades. The clinical picture involving sustained IAP ≥20 mmHg with the development of new organ dysfunction or failure constitutes ACS. ACS can be categorised as primary ACS (referring to an intra-abdominal cause), secondary ACS (extra-abdominal cause), and recurrent ACS (recurrence despite previous treatment). The incidence of IAH was 64% and that of ACS was 12%. Maximal IAP was identified as an independent predictor of mortality. Overall mortality was 43%.[1] Development of IAH was identified as an independent risk factor for death. Patients with IAH demonstrated significantly higher ICU and 90-day mortality. Mortality among patients with primary IAH was significantly higher than for those with secondary IAH.[2] A prospective observational comparison of intermittent and continuous IAP measurements that confirms previous work demonstrating the validity of continuous IAP monitoring.[3]

Normal IAP is ≈5–7 mmHg, with baseline levels in morbidly obese individuals often ranging from 9–14 mmHg. Although this degree of IAH may affect organ function in other patients, it often appears to be tolerated in obese individuals. Normal IAP usually is lower in children. In general, an individual patient's physiologic state must be taken into account when interpreting IAP measurements.

IAP typically is expressed in millimetres of mercury and conversion from centimetres of water may be necessary. Transvesicular measurement of IAP currently is the most popular technique, and several systems with or without the need for electronic equipment are available that allow IAP measurement in a non-ICU environment. All transvesicular IAP measurement techniques are based on the same principle; namely, that a fluid column in the bladder catheter and tubing to the collector serve as a pressure transducing medium. The pressure in this closed system can be measured either by means of an electronic system using a pressure transducer or by measuring the height of the fluid column in the tubing.[4]

The presence of IAH as a risk factor for acute kidney injury (AKI) has been shown in many clinical settings. Blood flow both to and from the kidney is impaired in patients with IAH because IAH decreases cardiac output (to a variable extent) leading to decreased arterial blood flow to the kidney. In addition to decreased cardiac output and systemic perfusion, local arterial inflow to the kidney may be impaired by IAH. Highlighting the importance of IAP to kidney perfusion, kidney perfusion pressure can be defined as MAP minus IAP. Therefore, in theory, decreased kidney function can be prevented by either decreasing IAP or increasing MAP, either of which will increase kidney perfusion pressure. Given the frequency of IAH in critically ill patients, the dose-dependent effect of IAH on kidney function, and the identification of IAH as an independent risk factor for AKI, IAH should be considered in every patient with AKI in the ICU. When IAH is present in an oliguric patient, fluid resuscitation can be continued, but IAP should be monitored carefully and crystalloid use should be avoided or limited. Specific medical treatment options to decrease IAP should be considered.

AIMS AND OBJECTIVES:

- To study the incidence of IAH in critically ill patients and to investigate the role of IAH as a cause of AKI in such patients.
- To assess the risk factors for development of IAH/ACS in such patients.
- To study the value of measures to decrease IAH/ACS in the development of ACS.

MATERIALS AND METHODS: A prospective, observational cum interventional study performed over six-month period in a tertiary care medical ICU.

Inclusion Criteria:

- Age>18 yrs.
- Stay in ICU>24 hour.
- Those requiring an indwelling bladder catheter.

Exclusion Criteria:

- Bladder Injuries.
- Neurogenic Bladder.
- Chronic Kidney Disease.
- Pregnancy.
- Surgical causes of Intra-Abdominal Hypertension.

METHODS: Demographic data, pre-existing comorbidities, primary diagnosis and APACHE II score are collected during admission. Clinical aetiological factors and predisposing conditions for increased IAP as well as associated conditions are also recorded for each patient. IAP measured via a Foley's bladder catheter according to a bedside standardised technique; IAP to be always measured at end expiration in complete supine position and in stable conditions with the base of the tubing positioned at the level of iliac crest in midaxillary line. Measurements are made at admission and at least every 24 hours. IAH is defined as pathological elevation of IAP at or above 12 mmHg on at least two consecutive measurements performed at 24-hour intervals. Abdominal perfusion pressure [APP] measured as MAP-IAH is computed daily. AKI defined and staged according to KDIGO guidelines. IAH and AKI considered potentially related when the time interval between their onsets is less than 48 hrs. Relevant interventions wherever appropriate are done and their effects analysed. Patients are followed up until death or hospital discharge whichever is earliest.

Categorical data are expressed as number and percentage and compared using the chi-square test. Logistic regression analysis was performed with AKI as the dependent outcome variable to assess independent risk factors for the same. Logistic regression analysis was performed with mortality as the dependent outcome variable. Receiver Operator Characteristic (ROC) curves were plotted to identify the threshold values of IAP and optimised the sensitivity and specificity for predicting AKI.[5]

RESULTS: The study included 52 patients. Mean age of the study population was 54.8 yrs. There were 29 males (55.7%) and 23 females (44.23%). Mean APACHE II score at ICU admission was 21.2. Out of all admissions, 33 patients (63.46%) fulfilled criteria for sepsis.

Out of 52 patients, 25 (48.1%) developed IAH during ICU stay. Prevalence of IAH on admission was 21.2%. Mean time of onset for development of IAH was 2.1 days.

Out of 52 patients, 16 patients (30.8%) had AKI. Among 25 patients with IAH, 14 of them (56%) had AKI. Mean time of development of AKI was 2.7 days after onset of IAH.

Patients in the IAH group had more severe renal failure. In the IAH group, 13 patients had AKI of grade 2 or 3 while only one patient had grade 1 AKI. In the non-IAH group, only two patients had AKI, both had grade 1 AKI. About six patients needed RRT support (37.5%), all in IAH group.

The ROC curve analysis obtained by plotting worst IAH values versus the presence or absence of AKI for each patient showed a significant Area under the Curve (AUC) of 90.5% (SE 0.046; CI 0.805-0.984; p value of 0.000). The ROC analysis implies that a random patient with AKI has a higher value of IAP than a random patient without AKI in 90.5% of the cases.

CONCLUSION: There was a very high incidence of intra-abdominal hypertension in critically ill patients. IAH was significantly associated with risk factors like sepsis, mechanical ventilation, pancreatitis, capillary leak, ascites, cumulative fluid balance and cirrhosis. IAH is an independent risk factor for development of acute kidney injury. IAH is an independent predictor of mortality in critically ill patients. Some of the limitations of the study was the sample size was not large enough to compare the predictive value of all the known potential promoting factors of AKI. A more standardised approach to IAP measurement might have improved the IAH definition and its relationship with AKI.

ABBREVIATIONS:

ACS- Abdominal compartment syndrome.
AKI- Acute Kidney Injury.
APP- Abdominal perfusion pressure.
AUC- Area under the curve.
IAH- Intra-abdominal hypertension.
IAP- Increased intra-abdominal pressure.
ICU- Intensive Care Unit.
RAC- Receiver Operator Characteristic.

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