Prevalence and Correlation of Intra-abdominal Hypertension with Incidence of Acute Kidney Injury in ICU Patients: A Cohort Study from A Tertiary Care Centre

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ABSTRACT

Introduction: Intra-abdominal hypertension (IAH), a sustained and repeated steady state rise in intra-abdominal pressure (IAP) above 12 mmHg, was reported as a hidden cause contributing to morbidity and mortality in critically ill patients. This study was aimed to find out the possible relationship between IAH and acute kidney injury (AKI) in ICU patients.

Material and methods: Prospective cohort study was conducted among patients admitted in medical and surgical ICU. Consecutive patients coming under the inclusion criteria were selected and IAH was measured using pressure transducer technique. IAP was measured at the time of admission, at 24 hours, 48hours and also at the end of 72hours. Other parameters were collected based on a standard proforma prepared.

Results: Eighty patients were included in our study, of which 24 patients had IAH. Mean age in patients with IAH was obtained as 63.08 ± 12.37 years with a male predominance. Maximum patients were belonging to age group of 61-70 yrs (25%), 9 patients were below 40 yrs and 14 patients were above 81yrs. Mean IAP was calculated to be 11.65 ± 3.15. Only 9 patients were in grade IV (11.3%). Risk factors associated were found to be ascites, upper GI bleed and metabolic encephalopathy. IAH and AKI were found to be significantly correlated (odds ratio=2.666 with CI 0.98-7.25).

Conclusion: Rising intra-abdominal pressure is found to have higher incidence of renal failure than those with established IAH. IAH has been found to be a clinically significant risk factor for development of AKI in ICU patients.

Keywords: Abdominal compartment Syndrome, Acute Kidney Injury, Intra-Abdominal Hypertension, RIFLE Criteria, SOFA Score

INTRODUCTION

Intra-abdominal pressure (IAP) is defined as the steady-state pressure within the abdominal cavity, resulting from the interaction between the abdominal wall and viscera. IAP varies according to respiratory phase and abdominal wall resistance. Normal IAP ranges from 5 -10 mm Hg.[1] Intra-abdominal hypertension (IAH) was believed to seen in surgical and post trauma patients from time immemorial but its role in medical critical care was almost unrecognized until the last decade.[2] The World Society of Abdominal Compartment Syndrome (WSACS) has published a consensus statement including definitions and recommendations for the screening and management of IAH and abdominal compartment syndrome (ACS) in 2004 which proves its significance in today's management of critically ill patients.[3] IAH is responsible for both abdominal and extra abdominal effects. Vital organ systems become affected as the normal intra-abdominal tension becomes IAH and then to abdominal compartments syndrome.[4] Some of the risk factors proposed include ascites, pancreatitis, mechanical ventilation and on I.V. Fluids etc.

Acute kidney injury (AKI) refers to a syndrome comprising renal damage due to acute cause from mild injury to total loss of function that seriously disturbs the homeostasis of fluid and electrolyte balances and hence disturbing the homeostasis.[5] IAH occurs in 33%- 41% of patients after abdominal surgeries and is associated with AKI.[6] Kidneys are especially vulnerable for the deleterious effect of IAH because of its retroperitoneal position. The relationship between AKI and IAH has been a widely learned topic of today because of its role in worsening of critically ill patients.[6] So far no study has been conducted in this region to establish a correlation between the IAH and AKI. Hence, this study was aimed to find out the possible relationship between IAH and AKI in ICU patients

MATERIAL AND METHODS

A prospective cohort study was conducted among the patients who admitted in MICU and SICU during the period of one year from January, 2017 to December, 2017. All patients admitted in medical ICU and surgical ICU during the stipulated time period ,who satisfies the inclusion criteria such as Patients in the age group 18 and above admitted in MICU and SICU during time period Jan 2017 to Dec 2017 had been taken up for the study. Sample size was calculated to be 40. Patients above the age of 80, below the age of 18

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and those who were not willing to take part in the study were excluded. The study was approved by the Institutional Ethics Committee and written informed consent was obtained from the patients.

Study procedure: Intravesical pressure using pressure transducer method (Gold standard technique) was used for measuring the IAP.[7] Briefly, the patient was placed in the supine position for measurement. The height of the transducers was adjusted so that the top of the 3 way tap (atmospheric port) was leveled with the mid-axillary line at the iliac crest and zeroed the transducer. The drainage tube to the urine bag was clamped. After emptying urine from bladder, the bladder was filled with 1ml/kg (maximum 25ml) of 0.9% sodium chloride using the syringe. The volume of fluid in the bladder was assured to be constant for each measurement. The stopcocks of the syringe were closed and allow 30-60 seconds for equilibrium to occur. The mean pressure reading upon end expiration from the multipara-metre monitor was taken. The incidence of AKI was calculated and its association to IAH was analysed. IAP was measured for 80 consecutive patients at the time of admission, at 24 hours, 48 hour and at the end of 72 hour. Mean IAP was calculated for all time periods.

STATISTICAL ANALYSIS

Data were analyzed by chi square test using SPSS (v16, IBM, Illinois, US). P<0.05 was considered as significant.

RESULTS

In this study group of 80 patients (with mean age 62.21yrs) were included. Among the total, 20 patients were in the age group of 61-70 yrs (25%), 9 patients were below 40yrs and 14 patients above 81yrs (table 1). The co-morbidities were depicted in table 2. Among the 80 patients, 15 had chronic kidney disease (CKD) (18.4%), 45 had diabetes mellitus (56.3%), 50 had hypertension (62.5%), 17 had chronic liver disease (CLD) (21.3%), 28 patients had chronic obstructive pulmonary disease (COPD) (35%), 13 had Acute coronary syndrome (ACS) (16.3%) and 5 had acute respiratory distress syndrome (ARDS) (6.3). The risk factors assessed were sepsis (13/80); ascites (6/80); pancreatitis (4/80) ; UGI bleed (5/80) ; post-surgery (10/80) ;CCF (9/80) and metabolic encephalopathy (7/80) (Table 3). Mean ICU stay was for 4.26 ± 2.73 days and mean hospital stay was for 10.06 ± 5.67 days. Among total 80 patients 24 were expired (30%).

Mean IAP on day 0 was 10.63 ± 9.48, on day 1 was 11.51 ± 10.54, day 2 was 12.05 ± 12.011 and on day 3 was 12.45 ± 12.76 showing a steady state increase from time of admission to day 3. Mean IAP of 80 patients have come to 11.65 ±3.15. IAH was labeled even when at least one reading of IAP was above 12mm Hg. Of the 80 patients 24 patients had IAH and 56 patients had IAP below 12 mmHg. Among 24 patients who had IAH, 13 were males and 11 were females (P=0.434). All patients were graded based on the grading system as per WSACS 2012 Consensus. Grade 0 was allotted to 56 patients (70%) while, 9 patients were in Grade IV (11.3%).

Incidence of AKI was analysed based of different range of IAP. The grey zone between 8 -12 showed significant incidence of AKI accounting to about 15 patients out of total 80 and of which 10 had AKI. Less than 8 mmHg –there were 41 patients of which 14 had AKI and 27 didn't have AKI. IAH was found in 24 patients of which 16 had AKI and 8 did not have. This was analysed with chi-square test and p=0.015. The relationship of IAH and AKI was analysed statistically, it was found that out of 24 patients who had IAH, 16 had AKI and 8 did not have AKI (chi square test p = 0.051, Odds ratio 2.666 with confidence intervals of 0.98-7.25) (Table 4). Among 80 patients admitted 24 expired. Among 24 expired, 16 had IAH. Only 8 patients didn't have IAH and they were improved (p=0.0001).

Age	Frequency	Percent
≤20	3	3.8
21-30	3	3.8
31-40	3	3.8
41-50	9	11.3
51-60	15	18.8
61-70	20	25.0
71-80	13	16.3
≥81	14	17.5
Total	80	100.0

Table-1: Distribution of age

Total		IAH		P value (chi square test)
		no (n=56)	yes (n=24)	
CKD	15	12	3	.334#
DM	45	29	16	.219
HTN	50	39	11	.044
CLD	17	7	10	.003
COPD	28	14	14	.004
ACS	13	8	5	.475#
ARDS	5	4	1	.601#
# Fisher's exact test				

Table-2: Distribution of co-morbidities

Total		IAH		P value (Fisher's exact test)
		No (n=56)	Yes (n=24)	
Sepsis	13	9	4	.947
Ascitis	6	1	5	.005
Pncreatitis	4	4	0	.086
UGI bleed	5	1	4	.017
Post surgery	10	10	0	.005
CCF	9	6	3	.818
Metab encephalopathy	7	2	5	.017

Table-3: Distribution of risk factors

IAH	AKI		Total
	Present	Absent	
Present	16	8	24
Absent	24	32	56
Total	40	40	80
p=0.051 (Odd's ratio=2.666, Confidence interval 0.98 to 7.25)			

Table-4: Association of IAH and AKI

DISCUSSION

The result of the present study revealed that IAH was correlated with the incidence of AKI. Among 24 patients with IAH, 13 were males and 11 were females. But Deeren and De Potter(8(1)) along with Malbrain et al.[9] demonstrated that the prevalence is more common in females than males. In this study, among the total 17 CLD patients 10 had IAH and 7 did not have and was found to be statistically significant. Diebel et al.[10] has revealed liver pathology is an independent risk factor for IAH. Furthermore, study by Bioconfiore et al in liver transplant patients had shown to have an increased incidence of IAH in post-transplant patients.[11] Cullen et al.[12] conducted studies on the effect of IAH on pulmonary compliance and found that those lung conditions which affect the abdominal wall compliance is associated with an increased incidence of IAH. Various studies conducted by Malbrain et al.[13] in tertiary care centres identified UGI Bleed, presence of ascites and metabolic encephalopathy as the risk factor for IAH. We found UGI as the increased risk factor for the IAH. Studies conducted by Zhao and Liao[12] and Malbrain et al[14] demonstrated a positive correlation IAH with mortality. IAH adversely affects venous return and microcirculation and associated with significantly increased morbidity and mortality.[10] Our study also strongly points to fact that IAH is a strong association contributing to the mortality of the patients.

The ideal method of measuring IAP is still a controversy. Measurement of intra-vesical pressure as the indirect measurement of IAP is finally considered to be gold standard.[15] The abdominal compartment contains solid organs, hollow organs, fluid, gas, solids, and adipose tissue. A persistently increased IAP (more than 12 mmHg) can cause an impact on the intra-abdominal organs which resulted in multisystem failure.[3] Apart from etiologic condition causing AKI, a rise in IAH also contributes to the local inflammation leading to worsening of AKI.[15] Systemic inflammation as its well-studied earlier, can widely sustain AKI through circulating metabolic factors inducing apoptotic/necrotic damages to the renal parenchyma.[5] At IAP of 12–15 mmHg, oliguria develops and at 25–30 mmHg anuria appears in the presence of normovolemia and at lower levels of IAP in hypovolemic patients or sepsis or under mechanical ventilation with high levels of PEEP.[16] In critically ill group of patients, especially those with abdominal conditions such as pancreatitis, peritonitis, and abdominal trauma, and postoperative patients paralytic ileus a small increase in intra-abdominal volume has big impact on the IAP. [17] Main aim of surgical correction is maintenance of perfusion to organs is by invasive decompression laparotomy.[18]

Many studies have proved the efficacy in better outcome in patients undergoing surgery at the earliest rather than late intervention. Mid line laparotomy to relieve IAH is also being practised as from the xiphoid down to the pubis is the technique most commonly used, but other less invasive modalities have been developed, with subcutaneous linea-alba fasciotomy one of the most promising approaches.[19] Either ultrafiltration or diuretics can be used in those patients depending on the clinical scenario (87/20). Prokinetic agents such as metoclopramide or erythromycin are often used to overcome abdominal distention and ileus and thus are an indirect treatment option for IAH.[21] Studies conducted by Malbrain et al.[22] in tertiary care centres have shown a strong contribution of IAH with AKI. Though our study have also established a causal association, statistical significance would have been possible if a larger group has been included in the cohort.

CONCLUSION

Rising intra-abdominal pressure is found to have higher incidence of renal failure than those with established IAH. IAH has been found to be a clinically significant risk factor for development of AKI in ICU patients.

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