A prospective study of evaluation of pre and post operative intra-abdominal pressure monitoring and its association with post operative morbidity and mortality in emergency laparotomy

[1]Dr C Malarvizhi M.S, [2]Dr K Velmurugan M.S, [3]Dr L Lalith Kumar M.S, [4]Dr P Rajamoorthi M.S, [5]Dr D Aswin

[1]Associate Professor, [2,3]Assistant Professor
Department of General Surgery
Chengalpattu Medical College
Chengalpattu, India

I. INTRODUCTION

The incidence of complications arising from intra-abdominal pressure variation in critically ill patients suffering from acute abdomen conditions with unconfirmed diagnosis is high and justifies increasing intra-abdominal pressure (IAP) measurement requests.

Intra-abdominal pressure is defined as the steady state pressure concealed within the abdominal cavity and resulting from the interaction between the abdominal wall and viscera. IAP oscillates according to respiratory and abdominal wall resistance. Intra-abdominal pressure levels upto 5mmHg are considered physiological in adults. In obesity intra-abdominal pressure may range from 10 to15 mmHg without pathophysiological significance.

Intra-abdominal hypertension is defined by IAP elevation above 12 mmHg in three consecutive measurements taken at 4 to 6 hours interval. Intra-abdominal pressure may gradually progress to abdominal compartment syndrome with sustained IAP above 20 mmHg and associated with organ dysfunction or failure.

Abdominal compartment syndrome is a late manifestation of increased intra-abdominal pressure. The level of intra-abdominal pressure at which abdominal compartment syndrome occurs is unknown. The diagnosis is often made on a clinical basis. The finding of a tense abdomen with increased airway pressures and oliguria despite normovolemia and adequate cardiac output should lead to the diagnosis of abdominal compartment syndrome.

IAP measurements have been performed using a variety of techniques such as a Hamilton manometer in the stomach, trans rectally via a Millers-Abbott tube or nasogastric tube, direct intra peritoneal puncture, using a veress needle and using inferior vena cava pressure. In general terms there is high degree of correlation between intravesical, intraperitoneal, intragastric and IVC pressures as a measure of IAP in both animals and man. In clinical practice the most useful technique for measuring IAP involves the use of intravesical pressure.

Various condition such as accumulation of fluid, flatus, feces increase the intra-abdominal pressure and may lead to intra-abdominal hypertension first and later abdominal compartment syndrome. Most of the body systems affected by intra-abdominal hypertension and abdominal compartment syndrome, most markedly the renal, respiratory, cardiac and nervous systems. The prognosis of patient depends on the flow of blood to various organs which ultimately affected by ACS/IAH. Timely recognition of ACS/IAH, play a very important role in reducing morbidity or mortality of patients.

II. AIMS & OBJECTIVES

To assess the relationship between peri (pre and post) operative intra-abdominal pressure and post operative patient morbidity, (with special reference to kidney function and pulmonary atelectasis) and mortality.

III. MATERIAL AND METHODOLOGY

The study was be conducted on 50 cases of Emergency Laparotomy and 50 elective laparotomy patients admitted under Department of general surgery in CHENGALPATTU GOVERNMENT MEDICAL COLLEGE AND HOSPITAL. Ethical committee clearance was obtained from the institutional ethical committee of CHENGALPATTU GOVERNMENT MEDICAL COLLEGE AND HOSPITAL. This study was conducted between January 2020 and December 2020. Before the study conducted informed consent was obtained from all the patients.

METHOD OF COLLECTION:

Study design : A Prospective Comparative study.
Sample size : 100 admitted cases

Inclusion criteria:

1. Patients admitted to Chengalpattu medical college under General Surgery department and undergoing emergency and elective laparotomy .
2. Age group 18 yrs to 60 years.

Exclusion criteria:

1. Age group ≤ 18years and ≥ 60years.
2. Pregnancy.
3. Morbid obesity.
4. Spinal cord problems and fracture limbs who are unable to lie down supine.
5. Bladder complaints – neurogenic bladder, cystitis.
6. Patients with established causes of co-morbidity such as renal failure(oliguria), CAD, hypertension.
7. Patient not willing to give consent.

METHODOLOGY:

Two groups each with 50 surgical patients were selected. The experimental group had increased intra-abdominal pressure. The control Group had normal intra-abdominal pressure. The control group were selected from those undergoing elective surgery. The preliminary details collected from patients included, name, age, sex, diagnosis, operative procedure planned, BMI. Presence or absence of diabetes mellitus, hypertension. Examination findings of pulse, blood pressure, respiratory rate, temperature and specific systemic examination of respiratory system, cardiovascular system and abdomen were noted.

Investigation which included hemoglobin concentration, plasma urea, plasma creatinine and Chest X-Ray were done preoperatively and post operatively according to need. Intra-abdominal pressure and urine output were monitored pre-operatively and at every fourth hourly intervals in the post-operative period for 24 hours.

The intravesical route of measuring the IAP was done by catheterization of the urinary bladder using Foleys catheter. Equipments required to measure IAP were Intravenous infusion set,50ml syringe, suction catheter, measuring scale (length: 30cm), measuring tape and hemostat .Patient positioned in supine position And catheterized with Foley's catheter (size:16Fr /18Fr). The Infusion set was connected to a syringe filled with 50 ml of saline which was then connected with main drainage channel of Foley's catheter through the connector (suction catheter) and saline instilled into the empty bladder. Then the connector (suction catheter) clamped with hemostat; The empty syringe removed. The intravenous set tube and connector held vertically above the sympysis pubis. once the hemostat was released, the saline flows out of catheter drainage tube and reaches a height(in cm of saline)measured using a ruler scale marked in cm which corresponding to IAP. The value converted in mmHg by multiplication factor with 0.74.

Operative findings during laparotomy and the surgical procedure done were noted. Patient who underwent Post-operative mechanical ventilation and re-laparotomy were also noted. Also, cause of mortality was noted if the patient expired during the study period inspite of maximal supportive medical care. The results were documented and tabulated in MS excel.

IV. RESULTS AND DISCUSSION

Increased intra-abdominal pressure cause intra-abdominal hypertension and abdominal compartment syndrome. Radiological investigation such as ultrasound abdomen, chest x-ray, x-ray abdomen, computerized tomography, magnetic resonance imaging is also not used for quantify intra-abdominal pressure. Measurement of intra-abdominal pressure via the urinary bladder is considered to be the gold standard, because it is simple and cheap and easily reproducible.

An increased IAP can affect kidney negatively directly and indirectly by systemic effects (decreased cardiac output). Abdominal Perfusion Pressure (APP) is a clinical parameter to explain the circulatory compromise in abdominal cavity in the presence of intra-abdominal hypertension/ Abdominal compression syndrome. APP is the difference between the mean arterial pressure and intra-abdominal pressure. During Intra-abdominal hypertension, decrease of glomerular hydrostatic pressure (due to hypoperfusion) and increase of Bowman's space hydrostatic pressure (due to intra-abdominal hypertension) which might lead to acute reduction in glomerular infiltration. In this study, serum urea and creatinine were elevated pre-operatively in patients with increased IAP while patients and these values decreased post-operatively. Similarly, mean urine output increased post-operatively among the patients with increased IAP and these were found to be statistically significant.

Respiratory failure is among the most important consequences of IAH and it is originated by different mechanisms, such as chest wall elastance increase, functional residual capacity reduction, compression atelectasis and lung edema formation through reduction in lymphatic drainage. Pre-operatively in both the study group none of the patient had atelectasis. Post-operatively, out of 50 patients in normal IAP group, 2(4%) developed atelectasis and 1(2%) patient needed ventilatory support while among 50 patients in increased IAP group,3(6%) developed atelectasis and 2(4%) required ventilatory support. Post-operative ventilatory support among both groups had no statistical difference. This might be due the reversal of pulmonary detrimental effects by abdominal decompressive laparotomy by redistribution of aerated lung volumes. Surgical decompression was the standard treatment for the patients with Intra-abdominal Hypertension/ACS. Even after surgical decompression, IAP remains moderately to severely increased with improvement in physiological of organ, so all the patients requires careful observation. Re-laparotomy should be considered in a patient if IAP >20mmHg or the organ dysfunction persists. In this study, none of the patient had indication for re-laparotomy.

In this study, mortality rate was 0%, patients could be saved only when the organ dysfuntion in reversible phase. Delayed decompression was found to be the main cause for high mortality rate. Hence timely intervention and regular IAP monitoring post-operatively will reduce the mortality rate in patients with IAP/ACS.

Comparison of BMI with groups by using Pearson Chi square test which shows that there is no statistically significant difference in the BMI distribution between groups. This suggests overweight of the patient does not significantly increase the intra abdominal pressure of the patient.

Comparison of blood Urea between the groups by using Unpaired t-test which shows that there is highly statistically significant ($p=0.0005<0.01$) difference during all the time duration, blood Urea is significantly raised in all the cases of increased intra-

abdominal pressure as compared to the cases with normal intra-abdominal pressure in all time intervals (pre-operative, post – operative, 4th hourly, 12hourly, 24hourly.

Comparison of serum Creatinine between the groups by using Unpaired t-test which shows that there is highly statistically significant ($p=0.0005<0.01$) difference during (pre-operative, post – operative, 4th hourly) , whereas during (12hourly ($p=0.100>0.05$) , 24hourly ($p=0.151>0.05$)) no statistically significant) difference were found in serum Creatinine.

Comparison of Urine Output between the groups by using Unpaired t-test which shows that there is statistically significant ($p<0.05$) difference during (0 to 4 hours and 8 to 12 hours), whereas during other time period no statistically significant difference were found in Urine Output. This indicates that urine output might not be an ideal indicator.

Comparison of Atelectasis post-operatively with groups by using Pearson Chi square test which shows that there is no statistically significant difference in the Atelectasis post-operatively between groups.

V. CONCLUSION

ACS/ IAH is associated with profound physiological abnormalities both outside and within the abdomen. While treating these patients it is essential to identify the signs of increased abdominal pressure early and start the management accordingly. It is also important to monitor the intra-abdominal pressure of the affected patients and those with (more than two) risk factors either continuously or intermittently. Understanding the pathophysiology of ACS/IAH is of prime importance for applying patient tailored treatment. If needed, appropriate surgical intervention should be done at the stage of IAH itself and should not be postponed till the development of ACS.

Renal dysfunction is the most common complication of abdominal compartment syndrome. Pre-operative renal dysfunction was found to be high in all the case series ranging from around 40% to 80%. The fall in the post operative serum creatinine as compared to pre operative serum creatinine was also observed in all the case series. In many of the studies renal dysfunction became evident as oliguria and later progressed to anuria. Compression of the renal vein and parenchyma and reduced renal perfusion, lead to reduced microcirculation to the functioning glomeruli and cortex. This results in tubular and glomerular dysfunction and substantially reduced urine output since FG = MAP – 2 x IAP. Thus the IAH induced renal dysfunction and prerenal azotemia will neither be responsive to fluid resuscitation nor vasopressors. It improves dramatically by appropriately and promptly reducing the elevated IAP.

The awareness of the entity called Intra abdominal hypertension and abdominal compartment syndrome is spreading in recent times. Yet in many of the centers it is still under-diagnosed as strict protocols to monitor intra abdominal pressure in critical care patients both in the medical and surgical side have not been laid down. As recommended by the World Society of Abdominal Compartment Syndrome, all cases in the critical care wards should be assed for intra abdominal pressure immediately following admission and serially in cases of elevated initial pressure. Though the mortality rate is zero in this case series, it is probably due to the choosing patients with increased abdominal pressure selectively rather than monitoring all the patients in the critical care ward, some of whom may have died because of undiagnosed Intra abdominal hypertension.

Hence abdominal compartment syndrome is a treatable condition when it is timely diagnosed and appropriately managed. Both medical and surgical treatments play equally important role in their management. Awareness and recognition of this entity will go a long way in reducing the mortality of many critically ill patients and all it takes is a simple bedside test to make the difference between probable death and survival.

REFERENCES:

[1] Viana RA. Enfermagem em terapia intensiva: práticas baseadas em evidências. São Paulo: Atheneu; 2011. p.560.

[2] Viana RA, Whitaker IY. Enfermagem em terapia intensiva: práticas e vivências. Porto Alegre: Artmed; 2011. p.546.

[3] Knobel E. Terapia intensiva: enfermagem. São Paulo: Atheneu; 2006. p.656.

[4] Baird MS, Bethel S. Manual de enfermagem no cuidado crítico: intervenções deenfermageme condutas colaborativas. 6a ed. Rio de Janeiro: Elsevier; 2012. p. 1016.

[5] Bersani AL, Gomes JO, Braga IL, Guimarães HP, Lopes RD. Síndrome compartimental abdominal. Rev Bras Clin Med. 2009;7:313-21.

[6] Jeronimo RA, Cheregatti A. Técnicas de UTI. São Paulo: Rideel; 2010. p.344.

[7] Malbrain M. Abdominal compartment syndrome. F1000 Med Rep. 2009;1. pii: 86.

[8] Rafaela milanesi and Rita Catalina Aquino Caregnato .Intra abdominal pressure :an integrated review, https://doi.org/10.1590/S1679-45082016RW3088.

[9] Losada HM, Manterola CD, Vial MG, Pineda VN. Hipertensión abdominal y syndrome de compartimiento abdominal. Rev Chilena Cirugía. 2005;57(1):2-6.

[10] Bradley SE, Bradley GP. The effect of increased intraabdominal pressure on renal function in man. J Clin Invest 1947; 26: 1010-22. 12.

[11] Collee GG, Lomax DM, Ferguson C, Hanson GC. Bedside measurement of intra-abdominal pressure (IAP) via an indwelling naso-gastric tube: clinical validation of the technique. Intensive Care Med 1993; 19: 478-80.

[12] Fietsam R, Villalba M, Glover JL, Clark JL. Intra-abdominal compartment syndrome as a complication of ruptured abdominal aortic aneurysm repair. Am Surg 1989; 55: 396-402. 16.

[13] Wagoner RH. Studies on intra-abdominal pressure. Am J Med Sci 1926; 171: 697-705

[14] Iberti TJ, Lieber CE, Benjamin E. Determination of intraabdominal pressure using a transurethral bladder catheter: clinical validation of the technique. Anesthesiology 1989; 70: 47-50.

[15] Motew M, Ivankovich AD, Bieniarz J et al. Cardiovascular effects and acid base and blood gas changes during laparoscopy. Am J Obstet Gynecol 1972; 115: 1002-12.

[16] Mullane JF, Gliedman ML. Elevation of the pressure in the abdominal inferior vena cava as a cause of hepatorenal syndrome in cirrhosis. Surgery 1966; 59: 1135-46.
[17] Kron IL, Harman PK, Nolan SP. The measurement of intraabdominal pressure as a criterion for abdominal reexploration. Ann Surg 1984; 196: 28-30.
[18] Sugrue M, Buist MD, Lee A, Sanchez DJ, Hillman K. Intraabdominal pressure measurement using a modified nasogastric tube: description and validation of a new technique. Intensive Care Med 1994; 20: 588-90.
[19] Lacey JB, Brooks SP, Griswald J et al. The relative merits of various methods of indirect measurement of intraabdominal pressure as a guide to closure of abdominal wall defects. J Pediatr Surg 1987; 22: 1207-11.
[20] Kelly KM, Benjamin E, Hirsch S, Gentile DR, Premus G, Iberti TJ. A simple non invasive technique to measure intraabdominal pressure. Crit Care Med 1987; 366
[21] Volkmann R. On ischemic muscle paralysis and contraction. Centralblatt für Chirurgie. 1881;51:801–3
[22] Marey E-J. Paris: A Delahaye; 1863. Medical physiology on the blood circulation; pp. 284–93.
[23] Bert P. Paris: JP Baillière;; 1870. Lessons on the physiology of respiration.
[24] Emerson H. Intra-abdominal pressures. Arch Intern Med. 1911;7:754–84.
[25] Baggot MG. Abdominal blowout. Curr Res AnesthAnalg. 1951;30:295–9.
[26] Fietsam R, Jr, Villalba M, Glover JL, Clark K. Intra-abdominal compartment syndrome as a complication of ruptured abdominal aortic aneurysm repair. Am Surg. 1989;55:396–402.