

**PREVALENCE AND PREDICTORS OF INTRA-ABDOMINAL
HYPERTENSION AND COMPARTMENT SYNDROME IN SURGICAL
PATIENTS IN CRITICAL CARE UNITS AT KENYATTA NATIONAL
HOSPITAL**

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**DISSERTATION SUBMITTED IN PART FULFILMENT OF THE
REQUIREMENT FOR THE AWARD OF MASTER OF MEDICINE IN
GENERAL SURGERY DEGREE AT THE UNIVERSITY OF NAIROBI**

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DECLARATION

I hereby certify that this is my Original work and has not been presented for a Degree in any other University.

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ACKNOWLEDGEMENT

I would like to sincerely thank Professor Ndaguatha and Dr Daniel Ojuka for their invaluable input and advice when developing this dissertation.

I'm sincerely grateful to KNH Research and Programs department for funding this work.

Special thanks to my colleagues for their input in proposal development and carrying out of the study.

Finally to the relatives of the patients involved in this study for voluntarily agreeing for their kin to participate in this study.

DEDICATION

To my wife Celia and my daughter Celleste for their patience,love and support during this process.

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ABBREVIATIONS

ACS-	Abdominal compartment syndrome
APP-	Abdominal perfusion pressure
BIPAP-	Biphasic positive airway pressure
BMI-	Body Mass Index
BSA-	Burnt Surface Area
BP-	Blood Pressure
CCU-	Critical care unit
CPAP-	Continuous Positive Airway Pressure
CT-	Computed Tomography
HB-	Hemoglobin
IAH-	Intra-abdominal hypertension
KNH-	Kenyatta National Hospital
IAP-	Intra-abdominal pressure
IP-	Intra-peritoneal
IV-	Intravenous
MAP-	Mean arterial pressure
MmHg-	Millimeters of mercury
NG-	Nasogastric
PSV-	Positive pressure ventilation
RAAS-	Renin angiotensin aldosterone system
SD-	Standard Deviation

SIMV-	Synchronized Intermittent Mandatory Ventilation
SIRS-	Systemic Inflammatory Response Syndrome
SPSS-	Statistical Package for Social Sciences
UON-	University Of Nairobi
WSACS-	World society of abdominal compartment syndrome
TAC-	Temporary Abdominal Closure

ABSTRACT

Background

The harmful effects of intra-abdominal hypertension (IAH) have been documented on almost every organ system. It may be under-diagnosed since it mainly affects patients who are critically ill and whose organ dysfunction may be incorrectly attributed to progression of the primary disease. The prevalence of IAH and abdominal compartment syndrome (ACS) at Kenyatta National Hospital (KNH) is not known. In addition, no much attention is paid to the problem and routine monitoring is not practiced in our critical care units (CCU). The purpose of this study was to determine the prevalence and factors associated with development of IAH/ACS among critically ill surgical patients.

Objective

The aim of this study was to determine the prevalence and possible predictors of IAH and ACS among surgical patients in critical care units at Kenyatta National Hospital.

Materials and Methods

Design and setting

This was a cross sectional descriptive study involving surgical patients in critical care units at Kenyatta National Hospital carried out over a duration of seven months from March 2015 to October 2015.

Patients

One hundred and thirteen critically ill and ventilated patients aged 13 years and above admitted to various critical care units and with no contraindication to transvesical intra-abdominal pressure measurements were recruited into the study.

Measurements

World society of abdominal compartment syndrome (WSACS) consensus guidelines 2013 criteria and Krohn's intravesical method were used to measure and describe prevalence of IAH and ACS. IAP was measured at first contact, then at 12 and at 24 hours. Additional parameters recorded included: Base excess, serum bilirubin, total blood count, serum urea

and creatinine, urine output, vital signs, peak airway pressure and amount of resuscitation fluid administered and fluid balance in 24 hr. as recorded in the patient's input-output chart.

Results

A total of 113 consecutive surgical patients admitted to the critical care units were studied. Demographic, clinical data, laboratory data and maximal (max) and mean intra-abdominal pressure (IAP) were recorded. Frequency, means and standard deviation were used to describe the data. With level of significance set at <0.005 , categorical variables i.e. age, gender and diagnosis categories were analyzed using Chi square test. Continuous variables were analyzed using student 't' test and Mann Whitney test as appropriate. Of our study population 71.7% (by IAP max) and 67.3 % (by IAP mean) had IAH. Abdominal compartment syndrome (ACS) developed in 4.4% of the group. The following factors were significant determinants of risk of IAH : amount of IV fluids over 24 hours (3949.6 vs. 2931.1, $p=0.003$,adjusted OR 1.0[1.0-1.002]), hemoglobin values at admission (9.9 vs. 12.0 , $p=<0.012$,adjusted OR 0.6[0.4-0.9]) peak airway pressure (28.4 vs. 17.3 ; $p=0.018$,adjusted OR 1.6[1.1-2.4]) and synchronized intermittent mandatory ventilation (SIMV) (60 vs. 32; $p=0.041$,adjusted OR 1.4[0.78-2.04]).

Of those who had IAH, age, amount of IV fluids in 24 hours, fluid balance and ventilator mode were significant determinants of risk of progression to ACS .

Conclusion

In Kenyatta National Hospital among critically ill surgical patients, the prevalence of intraabdominal hypertension and abdominal compartment syndrome is high. Clinical parameters pertaining to fluids administration and ventilator mode are significant determinants.

1.0 CHAPTER ONE: INTRODUCTION

Intra-abdominal pressure (IAP) is the steady-state pressure concealed within the abdominal cavity. The normal pressure range is 5-7mmHg¹ High grade IAH and ACS are independent predictors of multiple organ failure and without prompt and adequate treatment carries a mortality rate of 70-80%. Early detection is the cornerstone of management and regular measurement of IAP is key^{2,3}.

IAH and both primary and secondary ACS can be predicted early by measurement of IAH and physiological parameters such as fluid balance, acid-base status, haemodynamic status and respiratory parameters^{4,5}.

IAH still remains largely under diagnosed in spite of widespread awareness about the problem in many critical care units.⁶

The aim of this study was to establish the prevalence of IAH and ACS and factors associated with high risk of developing the same among critically ill surgical patients at KNH.

1.1 LITERATURE REVIEW

IAH is defined by a sustained or repeated pathologic increase in IAP ≥ 12 mm Hg while ACS is defined as a sustained IAP >20 mm Hg (with or without APP < 60 mmHg) that is associated with new organ dysfunction/failure. ACS resulting from injury or disease in the abdominal-pelvic region is called primary ACS and frequently requires early surgical or interventional radiologic intervention. Secondary ACS refers to ACS resulting from conditions that do not originate from the abdominal-pelvic region. A condition, in which ACS redevelops after previous surgical or medical treatment of primary or secondary ACS, is referred to as recurrent ACS^{1,7}.

The harmful effects of IAP in various organ systems have been studied over the past century. It has been shown to have deleterious effects in virtually all critical organs. The harmful effects of IAH occur long before the manifestation of compartment syndrome.⁸ The abdominal cavity can be considered as a closed space and the pressure within this cavity influenced by its compliance. It is partially rigid (pelvis, spine and costal margin) and partially flexible (abdominal wall, viscera and diaphragm).⁹ Intra-abdominal hypertension occurs when tissue fluid within the peritoneal and retroperitoneal space (edema,

retroperitoneal blood, free fluid in the abdomen and excessive gas within the intestines) builds up to such an extent that the abdominal wall compliance threshold is crossed and the abdomen can no longer stretch, at which point continued accumulation results in very high pressure within this closed space⁹.

Initially this increase in pressure does not cause organ failure because there is initial attempt at compensation. If the pressure build up goes over 20 mm Hg and organs begin to fail, the problem has now progressed to the end stage i.e. Abdominal compartment syndrome. In a significant proportion of cases, the underlying pathological process is capillary permeability caused by the systemic inflammatory response syndrome (SIRS) that is the common denominator in critically ill patient. Fluid leaks into the gut wall, mesentery and retroperitoneal tissue⁹.

At the cellular level, reduced perfusion and impaired oxygen delivery develops leading to ischemia and anaerobic metabolism. This leads to acidosis which triggers release of vasoactive substances such as histamine and serotonin which increases endothelial permeability causing further capillary leakage, a vicious circle sets in⁹.

As pressure rises, abdominal compartment syndrome impairs not only visceral organs but the other systems as well. In the cardiovascular system IAH causes reduced cardiac output¹⁰. It is associated with high airway pressures and decreased chest wall compliance leading to respiratory failure¹¹. It causes renal vein compression and renal artery vasoconstriction resulting in renal failure¹². The liver's ability to remove lactic acid is impaired by increased intraabdominal pressure as small as 10 mmHg¹³. Intracranial pressure (ICP) is elevated in the presence of persistent IAH causing critical decrease in cerebral perfusion and progressive cerebral ischemia¹⁴. Elevated IAP also interferes with GIT functions as has been demonstrated among critical care patients on enteral feeding. It has been associated with enteral feed intolerance¹⁵

Although the detrimental effects of elevated IAP are numerous, it is not without some benefits. Intraperitoneal (IP) chemotherapy with increased IAP has been associated with improved antitumor effect of Cisplatin when compared with conventional IP or Intravenous (IV) chemotherapy¹⁶.

Originally thought to be a disease solely of the traumatically injured, IAH and ACS have now been recognized to occur in a wide variety of disease entities ¹⁷. Fiesta et al coined the term abdominal compartment syndrome after they observed the condition in four patients operated on for abdominal aortic aneurysm ¹⁸.

Injured and post-surgical patients requiring aggressive infusion of crystalloid intravenous fluids in a bid to correct hypovolemic shock are at increased risk for ACS ¹⁹. An unusual presentation of slow leaking ectopic pregnancy leading to elevated IAP and leading to ACS at a referral hospital in Nigeria has been reported ²⁰. Several other risk factors for the development of pathological IAH and ACS have been identified among critically ill patients ¹⁷. They can be categorized as follows:

- i. Those related to diminished abdominal wall compliance
- ii. Those related to increased intra-abdominal contents
- iii. Those related to capillary leak and fluid resuscitation

1.1.1 Factors related to diminished abdominal wall compliance

- High BMI

- Pregnancy

- Mechanical ventilation

- Basal pneumonia

- Pneumoperitoneum

- Abdominal surgery particularly with tight abdominal closures

- Pneumatic anti shock garments

- Prone positioning

- Abdominal wall bleeding or abdominal hematoma

- Burns with abdominal eschars

1.1.2 Factors related to increased intra-abdominal contents

- Gastro paresis
- Gastric distension
- Ileus
- Volvulus
- Bowel pseudo obstruction
- Abdominal hematoma
- Intra-abdominal or retroperitoneal hematoma
- Damage control laparotomy
- Liver dysfunction with ascites
- Abdominal infection (peritonitis, pancreatitis)
- Hemoperitoneum
- Pneumoperitoneum
- Major trauma
- Peritoneal dialysis
- Laparoscopy

1.1.3 Factors related to capillary leakage and fluid resuscitation

Acidosis (pH below 7.2)

- Hypothermia (core temp below 33°)
- Coagulopathy
- Multiple transfusions/trauma (>10 units in 24 hours)
- Sepsis, severe sepsis or bacteremia
- Septic shock

- Massive fluid resuscitation (>5 L colloid or > L crystalloid in 24 hours in the presence of capillary leak and a positive fluid balance)
- Major Burns
- Head injury
- Spinal cord injury

Given the wide range of potential risk factors and the significant attendant morbidity and mortality of IAH/ACS, a high index of suspicion and low threshold for IAP measurement is vital in the patient possessing any of these risk factors. The world society for abdominal compartment syndrome (WSACS) strongly recommends that patients should be screened for IAH/ACS risk factors upon ICU admission and in the presence of new or progressive organ failure²¹.

Early predictors of both primary and secondary ACS have been identified in a bid to improve early detection of the problem. These include: hemoglobin concentration, base excess, volume of resuscitation fluid used, fluid balance, urine output, temperature and peak airway pressure^{4,5,22}.

Majority of the patients with ACS have a tense distended abdomen but, physical examination of the abdomen is a poor predictor of ACS. In one series of 42 adult blunt trauma victims, physical examination of the abdomen identified a significantly elevated intraabdominal pressure (defined as >15 mmHg) with a sensitivity of 56 percent, specificity of 87 percent, positive predictive value of 35 percent, negative predictive value of 94 percent, and accuracy of 84 percent²².

Other clinical manifestations of IAH and ACS include: Progressive oliguria, increased ventilator requirements, hypotension, tachycardia, an elevated jugular venous pressure, jugular venous distension, peripheral edema, abdominal tenderness, and acute pulmonary decomposition. There may also be evidence of hypo perfusion, including cool skin, obtundation, restlessness, or lactic acidosis^{9,22}.

Recognizing that persistent IAH leads to ACS, early corrective measures once IAH diagnosis is established can halt progression of IAH to ACS. These includes; reduction of intraabdominal volume through evacuation of intraluminal contents(e.g. nasogastric and

rectal drainage), evacuation of intraabdominal space-occupying lesions (e.g., ascites, hematoma) when possible, and measures to improve abdominal wall compliance²³ .

Khron et al introduced abdominal decompression as a management strategy in patients presenting with a tense abdomen and noted dramatic improvement in outcomes²⁴ .

In established ACS, surgical decompression by performing a laparotomy is considered definitive management ²⁵. Temporary abdominal closure (TAC) is required to protect the abdominal contents after surgical decompression. This is done using e.g. ‘vacuum pack closure’, ‘Bogota bag’, Wittmann pouch and vacuum-assisted closure. All of these techniques involve encasing exposed bowel to create an air-tight seal but allowing for intra-abdominal volume to increase. It is important to have a system that allows for drainage of peritoneal fluid, to avoid build-up of pressure ²⁶ .

Not all cases of suspected ACS need operative intervention. Some of the exceptions include, escharotomy to relieve mechanical limitations due to burn scars and percutaneous catheter decompression to relieve tense ascites ²⁷ .Increased recognition of its incidence among the critically ill combined with advances in both the diagnosis and management of intra-abdominal hypertension (IAH) and abdominal compartment syndrome (ACS), have resulted in significant improvements in patient survival²⁸ .

Among post laparotomy patients in critical care units in Harare Zimbabwe, incidence of ACS has been reported to be eight(8).A sample size of thirty eight(38) was used ²⁹.The table below summarises the incidence of Intra-abdominal Hypertension (IAH) and Abdominal Compartment Syndrome (ACS) Among ICU Patients ¹⁷ .

Table 1: IAH and ACS among ICU patients

Population	IAH	ACS
medical	18-78%	4-36%
surgical	32-43%	4-8%
trauma	2-50%	0.5-36%
burn	37-70%	1-20%
paediatric	***	0.6-19%

*** - no data available

The studies done so far to document the incidence of IAH and ACS among surgical patients in critical care units have mainly focussed on general surgical patients with abdominal pelvic conditions requiring laparotomy with IAP being measured after laparotomy. The studies on secondary IAH and ACS have focussed mainly on post traumatic haemorrhagic shock. This study will involve a heterogeneous population of patients in critical care units with various surgical diagnoses (abdominopelvic and others) to determine incidence and early predictors of intra-abdominal hypertension and both primary and secondary abdominal compartment syndrome.

1.1.4 Intra-abdominal pressure

If abdominal cavity is treated as a fluid compartment, the pressure within it obeys Pascal's hydrostatic law: when pressure is applied to a contained fluid, the force is transmitted equally in all directions. In this regard, pressure measured at any point within the cavity at any given time can be taken to represent IAP in the entire abdomen.³⁰ Abdominal perfusion pressure-analogous to cerebral perfusion pressure-has emerged as a more reliable indicator of visceral perfusion and a good resuscitation end point³¹. $APP = MAP - IAP$ Where MAP is mean arterial pressure, APP is abdominal perfusion pressure.

1.1.5 Intra-abdominal pressure measurement techniques

There are many techniques of measuring IAP - intermittent and continuous measurements. So far, no continuous IAP measuring technique has been validated for routine clinical use³². The gold standard is direct needle puncture with a transducer connected to intraperitoneal catheter (e.g. during peritoneal dialysis and laparoscopy). It is invasive and not always practical. Intraabdominal pressure can be measured indirectly using intragastric, intracolonic, intra-uterine, intravesical (First described by Khron), or inferior vena cava catheters. The wall of the hollow viscus or vascular structure acts as a membrane to transduce pressure. These methods are relatively non-invasive and the IAP thus obtained correlates well with directly measured IAP^{30,32}.

Khron's method (Intravesical method) has become the most widely used because it is reliable, easy to use, reproducible, has minimal cost and there is strong correlation between the bladder pressure and directly measured intraabdominal pressure in animals and humans^{30,33}.

1.1.6 Intravesical technique

A continuous fluid column is used with a small volume of transducing medium (25ml). Higher volume, distends the bladder leading to detrusor contraction which can give spuriously elevated IAP^{30,32,33}. The pressure measured is expressed in mmHg and is measured in supine position with transducer zeroed at the mid axillary line. Previous studies had investigators using various reference points such as pubic symphysis and phlebostatic axis and mid axillary line as zero point each with a different IAP reading³⁴

Some physiological conditions such as obesity and pregnancy are associated with chronically elevated IAP of 10-15mmhg with no pathological consequence. Hence, the need to establish a base line IAP for individual patient where applicable^{30,34}.

1.1.7 Grading of IAH

Pathological IAH will range from mild elevations with no clinical sequelae to markedly elevated values attended to by organs dysfunction. Pathological IAH is defined as >12mmhg. One Classification system is based on duration of symptom and has four categories i.e. hyperacute, acute, subacute, and chronic. Hyperacute occurs within seconds to minutes as in sneezing or physical activity, while acute takes hours and is the form seen among surgical patients. Subacute occurs over days in medical patients e.g. with ascites. The chronic form is seen in pregnancy and abdominal tumours³⁵.

Another classification stratifies patients based on the IAP values³⁶ i.e.:

Grade 0 <12mmhg

Grade 1 12-15mmhg

Grade 2 16-20mmhg

Grade 3 21-25mmhg

Grade 4 >25mmhg

2.0 CHAPTER TWO :STUDY JUSTIFICATION

Intraabdominal hypertension and compartment syndrome carries significant morbidity and mortality in critically ill patients¹². ACS is an independent predictor of multiple organ failure and without prompt and adequate treatment carries a mortality rate of 70-80%^{2,3}.

The frequency of IAH and ACS among surgical patients at risk in our critical care units is not known. Early recognition of significant IAH(grades 2,3 and 4) through IAP measurement ,and timely intervention prevents ACS and overall is associated with good clinical outcomes

3.0 CHAPTER THREE: METHODOLOGY

3.1 Study Design

This was a prospective cross sectional study that was conducted in seven months from march 2015 to October 2015

3.2 Study Site

The study site was KNH, intensive care units

(ICUs):MainICU,cardiacICU,NeurosurgeryICU,Burns unit and Accident&Emergency department ICU.

3.3 Study Population

Patients being cared for by the surgical team admitted in the various Intensive critical care units

3.4 Inclusion Criteria

The following cases were considered eligible for inclusion in the study:

1. ≥ 13 years and older
2. Surgical patients admitted in the critical care units,intubated and on mechanical ventilation.
3. Patients whose kin consented for them to participate in the study

For the purpose of this study, a surgical patient was defined as one, who based on the diagnosis, would have been admitted to the general surgical, orthopaedic or any of the specialty surgical units i.e.neurosurgery, cardiothoracic and plastic surgery units, were it not for the critical nature of their illness. This excluded gynecological and obstetric patients

3.5 Exclusion Criteria

The following cases were excluded from the study:

1. pregnant patients
2. Patients with suprapubic catheter.
3. Patients already known to have bladder outlet obstruction e.g. from benign prostatic enlargement.

Patients with burst abdomen or those who have already undergone damage control laparotomy and temporary abdominal closure(TAC) already done before admission to CCU

3.6 Sampling Method

Consecutive patients who met the inclusion criteria and their kin consented for them to take part in the study were recruited.

For a descriptive study.

Assumptions made include:

Estimated incidence of ACS is 8%¹⁷.

Confidence level set at 95%

Using the formula :

$$n = \frac{Z^2 p(1-p)}{e^2}$$

Where n = sample size,

Z = Z statistic for a level of confidence,

P = expected prevalence or proportion

and

e = precision

(in proportion of one; if 5%, $e = 0.05$).

For the level of confidence of 95%, which is conventional, Z value is 1.96

$$n = (1.96^2 * 0.08 * 0.92) / 0.0025 = 113$$

3.7 Data Collection

The study was based at KNH in the Intensive care units unit and commenced once approved by the department of surgery and Ethical Research Committee (ERC) - KNH/UON. I, the principal investigator was assisted by two research assistants who were at the level of general surgical residency in clinical rotations. Recruitment of participants who met the inclusion criteria was done where next of kin of the study participants were informed of the nature, purpose, potential benefits and harmful effects of the study. Those who agreed for their kin to

participate in the study, informed written consent was obtained and subsequently enrolled in the study. Information obtained included bio data, diagnosis, clinical parameters such amount of fluids administered, pints of blood given, fluid balance and vital signs(blood pressure,temperature,pulse and respiratory rate)laboratory tests(white cell count,haemoglobin,bilirubin ,urea and creatinine)IAP at first contact,IAP at 12 hours and IAP at 24 hours. Only data from patients who had all the clinical, laboratory parameters and all three IAP measurements completed was included in the final analysis.

3.8 Measurement of Intra-Abdominal Pressure

The abdominal pressure was determined using indirect method whereby urinary bladder pressure is measured with a Foley's catheter. Patients were catheterized with a 16-gauge two- way Foley's catheter, bladder drained and then filled with 25cc of sterile saline through the Foley's catheter. The tubing of the collecting bag were clamped and catheter connected to a saline manometer using two three-way B-BRAUNTM stopcocks connected in series .With the patient in a supine position with abdominal muscles relaxed ,the point along the midaxillary line at the level of anterior superior iliac spine was used as the zero reference point. IAP was then measured in centimeters of water at end-expiration 30-60 seconds after instillation of the priming 25cc of saline into the bladder. A conversion factor of 1.36 was used to convert the pressure into millimeter of Hg.

Based on IAP,intraabdominal hypertension was graded as follows:

Grade 0 <12mmhg

Grade 1 12-15mmhg

Grade 2 16-20mmhg

Grade 3 21-25mmhg

Grade 4 >25mmhg

Abdominal compartment syndrome was defined as a sustained IAP >20 mm Hg (with or without APP < 60mmHg) that was associated with new organ dysfunction/failure.

3.9 Patient Care

Those with grade 2-4 IAH were recommended for non-surgical interventions to reduce IAP and those with ACS decompressive laparotomy.

3.10 Data Analysis

Intra-abdominal pressure, number of patients with IAH and number of patients with ACS were taken as the independent variables while the clinical and laboratory parameters listed above were the dependent variables.

The data was analyzed using Statistical Package for Social Sciences (SPSS) for Windows Version 21(Chicago, IL, 3)

Measures such as frequency, mean and standard deviation were used to describe the data . Correlates of elevated IAP were determined using Chi square test for categorical variables and Student 't' test and Mann Whitney for continuous variables as appropriate.Univariate and multivariate analysis and logistical regression were used to correlate IAP to the statistically significant factors with p value set at <0.05.

3.11 Study Limitation

1. I used saline manometer because of lack of transducers. Though this could affect accuracy of measurements, every attempt was made to zero the manometer properly before each IAP measurement.
2. The study required multiple calibrations and measurements of the IAP.This was mitigated by having the research assistants applying the same technique of zeroing the manometer and measurement for each of the three readings.
3. Diagnosis of ACS required presence of IAH with at least one organ dysfunction/failure. It was not possible to attribute the organs dysfunction/failure to development of ACS.
4. We did not perform the full coagulation assessment, only relied on platelet count which is not a full representation of the coagulation status.
5. We didn't relate the presence of IAH/ACS to patient outcomes,something that can help in making a case for routine IAP measurement for high risk groups.

3.12 Ethical Considerations

The study commenced upon KNH/Upon ERC approval. All data in soft copy was in a password protected database whereas hard copy was kept in a locker and secured. Access was controlled by the principle researcher and limited to the two research assistant on authorization by principle researcher. At completion of study, raw data on hard copy was destroyed.

Feedback of information; all participants next of kin were informed of the IAP measurements and further care needed depending on the severity.

4.0 CHAPTER FOUR: RESULTS

4.1 Characteristics of the study population:

Out of the one hundred and forty seven patients recruited, 34 patients died before all the three IAP measurements in 24 hours could be done. Of the 113 analyzed, 70.8% were male. They ranged in age from 15-90 years with a mean age of 37.2 years (Table 1)

Clinical parameters pertaining to fluid homeostasis, vital signs and ventilator mode and settings were recorded. The laboratory measurements included white cell count, platelet count, and haemoglobin count and blood biochemistry (Table 1)

To be able to estimate the magnitude of IAH and ACS, three IAP measurements were done that is, at admission (baseline), at 12 hours and at 24 hours. From these measurements, maximal (highest in 24hrs) and mean IAP were recorded. Using mean IAP, the number of patients considered to have IAH were 76(67.3 %). The prevalence rose to 81(71.7%), when maximal IAP is considered. Of the 113, five were deemed to have ACS-based on presence of severe IAH and documented organ failure. This gives ACS prevalence of 4.4%. Of those who had IAH, 39.5% (using IAP mean) and 40.7 % (using IAP max) had primary IAH. Considering IAP mean, 60.5% had secondary IAH and the figure is similar at 59.3% when IAP max is considered (Table 2)

Table 2: Sociodemographic details and clinical Parameters of the study population

Variable	Frequency (%)	
Gender		
*Male	80 (70.8)	
Female	33 (29.2)	
Age in years		
#Mean (SD)	37.2 (12.8)	
Min-Max	15-90	
Clinical and laboratory parameters		
Variable	Mean (SD)	Min-Max
^u Amount of IV fluids over 24 hours in ML	3616.1 (1416.8)	1800-6900
Urine output in 24 hours	1949.9 (598.3)	800-3800
^y Fluid balance over 24 hours	1698.8 (1368.1)	100-8500
Number of pints of blood transfused over 24 hours	1.4 (1.6)	0-6
Pulse rate	101.9 (31.4)	55.3-147.3
Systolic Blood Pressure	105.7 (20.4)	56.0-164.0
Respiratory rate	22.8 (10.3)	11.0-38.0
[€] PAP	18.4 (4.1)	6.0-38.5
Temperature	36.1 (5.3)	3.0-40.2
^Ω WBC	11.8 (9.9)	3.4-30.0
[≠] Ventilation mode, n (%)		
biped	5 (4.4)	
cap	16 (14.2)	
skim	92 (81.4)	
Amount of positive end expiration pressure (PEEP) administered	4.5 (0.7)	4-8
Haemoglobin	10.6 (3.0)	4.1-21.9
Platelet count	321.7 (117.1)	4.1-791.0
Serum keratinise	118.6 (70.8)	4.0-723.0
Serum urea	10.6 (11.0)	2.3-87.0
Serum bilirubin	18.0 (12.8)	5.2-36.0
Base excess	-2.5 (6.2)	-26.5-9.4
[∞] Prevalence and grade of IAH and ACS		
Variable	Mean IAP in 24 hours Frequency (%)	Maximum IAP in 24 hours Frequency (%)
Grade 0	37 (32.7)	32 (28.3)
Grade 1	28 (24.8)	22 (19.5)
Grade 2	29 (25.7)	32 (28.3)
Grade 3	13 (11.5)	19 (16.8)
Grade 4	6 (5.3)	8 (7.1)
IAH	76 (67.3)	81 (71.7)
ACS	5 (4.4)	
Primary and secondary IAH based on diagnosis at admission		
^v pathology	N=76 using IAP mean(%) Frequency (%)	N=81using IAP max(%)
Abdominal pelvic(primary)	30 (39.5%)	33(40.7%)
Non abdominal pelvic(secondary)	46 (60.5%)	48(59.3%)

*Majority of the patients were male 70.8%

#the mean of the study population was 37.2 years

μ the amount of intravenous fluids administered over 24 hours as recorded in input-output chart.

¥ The fluid balance was derived from the difference of the total amount of fluids administered (sum of IV fluids and enteral and parenteral feeds) and the output (urine output plus 700ml of estimated insensible fluid losses)

€ Peak airway pressure (PAP) in cm of H₂O as displayed on the ventilator.

Ω White cell count (WBC) one of the parameters from total blood count profile others considered being hemoglobin and platelets count.

≠ Ventilation mode as set by the intensive care team. Biphasic positive airway pressure (Bipap), Continuous airway pressure (CPAP), Synchronized intermittent mandatory ventilation (SIMV)

∞ Of the 113 patients analyzed, 76(67.3%) had intraabdominal pressure (IAH) when the mean intraabdominal pressure in 24 hours was considered. This number rose to 81(71.7%) when the maximal (highest reading in 24 hours) is considered. The IAH was categorized in severity from mildest (grade 0, no IAH) to most severe level (grade 4) based on the intraabdominal pressure readings in mm Hg after conversion from cm of H₂O.

∇ The patients were categorized based on diagnosis at admission into those whose primary pathology was in the abdominal pelvic region and the others to be able to generate data on primary (of those with abdominopelvic conditions) and secondary (those with other [non abdominopelvic] conditions) IAH and ACS. When mean IAP is considered, of those who developed IAH, 60.5% had non abdominopelvic conditions therefore secondary IAH. This number is similar when maximal IAP is considered.

Of the 5 patients who met the criteria for ACS, 4(80%) had primary ACS and 1(20%) had secondary ACS. The patients were broadly categorized into two: those with abdominal pelvic diagnosis and those whose pathology or disease entity affected other areas of the body, that is, on-abdominal pelvic diagnosis. Those with abdominopelvic conditions would generate data on primary IAH and consequently primary ACS while those with non-abdominopelvic diagnosis expected to have secondary IAH and ACS. of the 113 patients, 33 had abdominopelvic diagnosis representing 29.2% of the total. Those with non-abdominopelvic conditions were 80, accounting for 70.8% of the group sampled. Figures 1 and 2

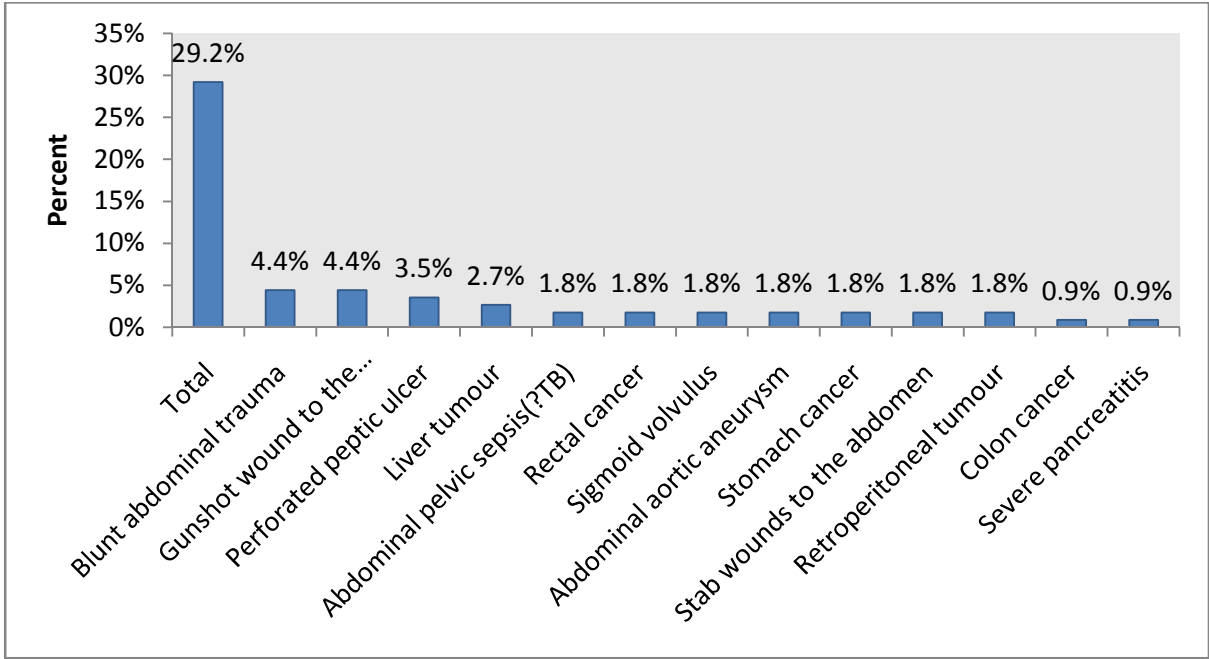
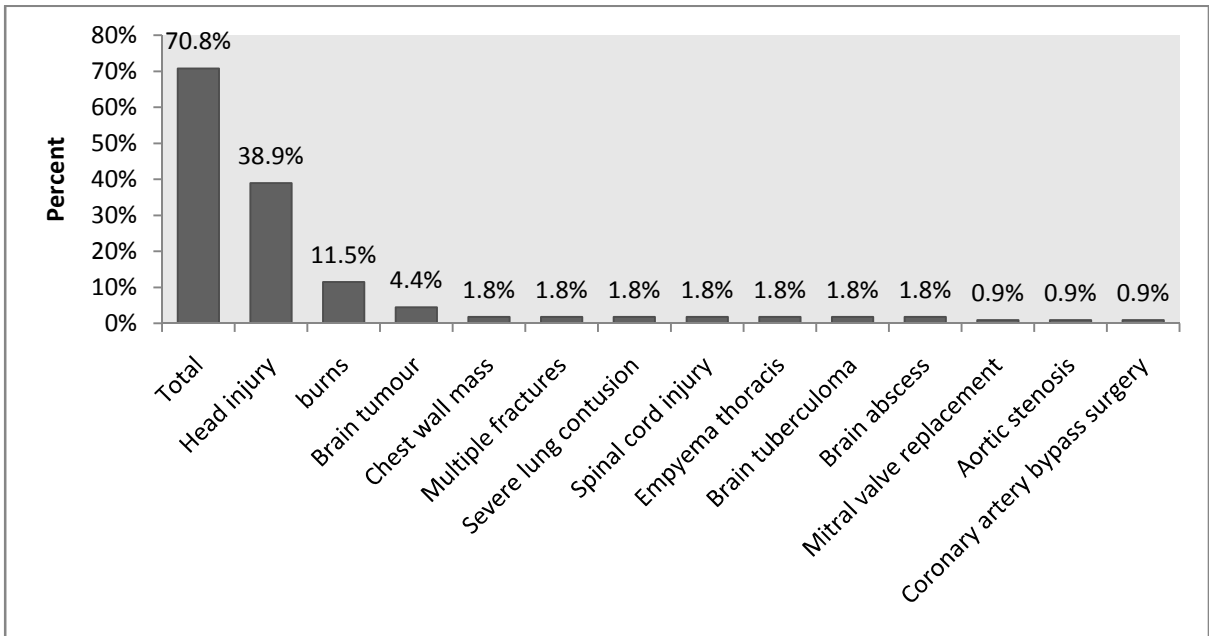


Figure 1: Patients with abdominopelvic diagnosis

On the x axis the bars represents the diagnosis categories as recorded at admission to critical care unit. Of the 113 patients analyzed 29.2% had abdominopelvic conditions and the specific disease entities are enumerated.

The Y axis has the proportion of those with abdominopelvic conditions in % out of the total of 113.

Figure 2: Patients with other (non-abdominopelvic) diagnosis



On the x axis the bars represents the diagnosis categories as recorded at admission to critical care unit. Of the 113 patients analyzed 70.8% had non abdominopelvic conditions and the specific disease entities are enumerated. The Y axis has the proportion of those with non - abdominopelvic conditions in % out of the total of 113.

IAH can be graded from the mildest to most severe form based on IAP readings. Grade 0 meaning no IAH through to grade 4, which is the most severe. Using the mean IAP, 37 (32.7%) had grade 0 and 6 (5.3%). The picture was similar when considering maximal IAP with 32 having no IAH and 8 having grade 4, representing 28.3% and 7.1% of the sample respectively. Figure 3 and 4

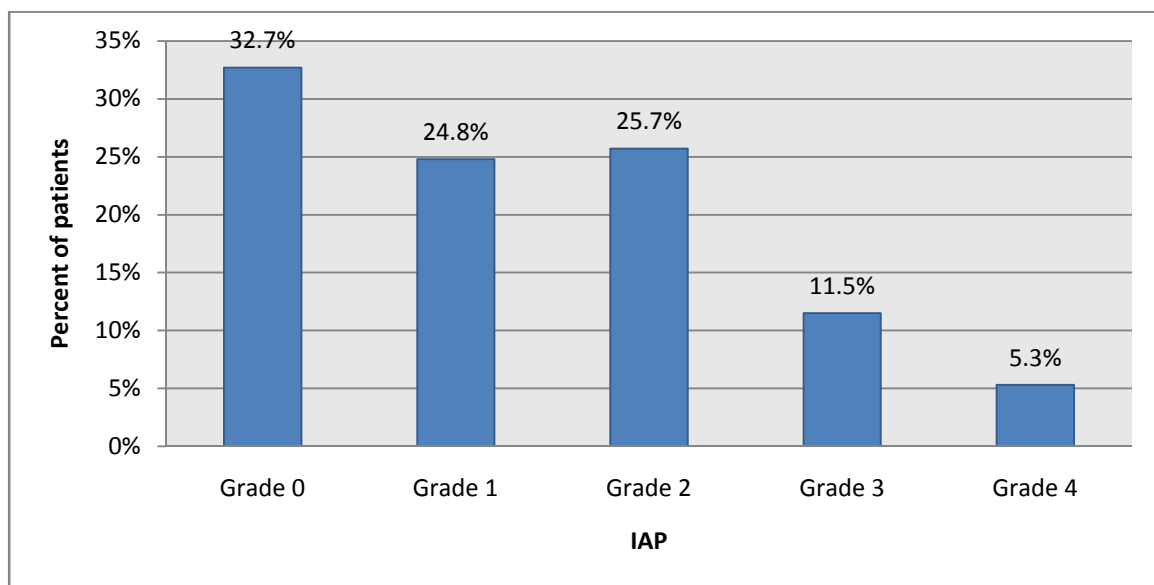


Figure 3: Patients with IAH when mean IAP is considered

The x axis represent the severity of IAH as categorized in four grades ranging from no IAH (grade 0) to most severe IAH (grade 4) based on mean of the three IAP readings in the 24 hour period. The Y axis is the proportion of patients with IAH in % when mean IAP is considered. This number is out of the total of 76 who had IAH when mean IAP is considered.

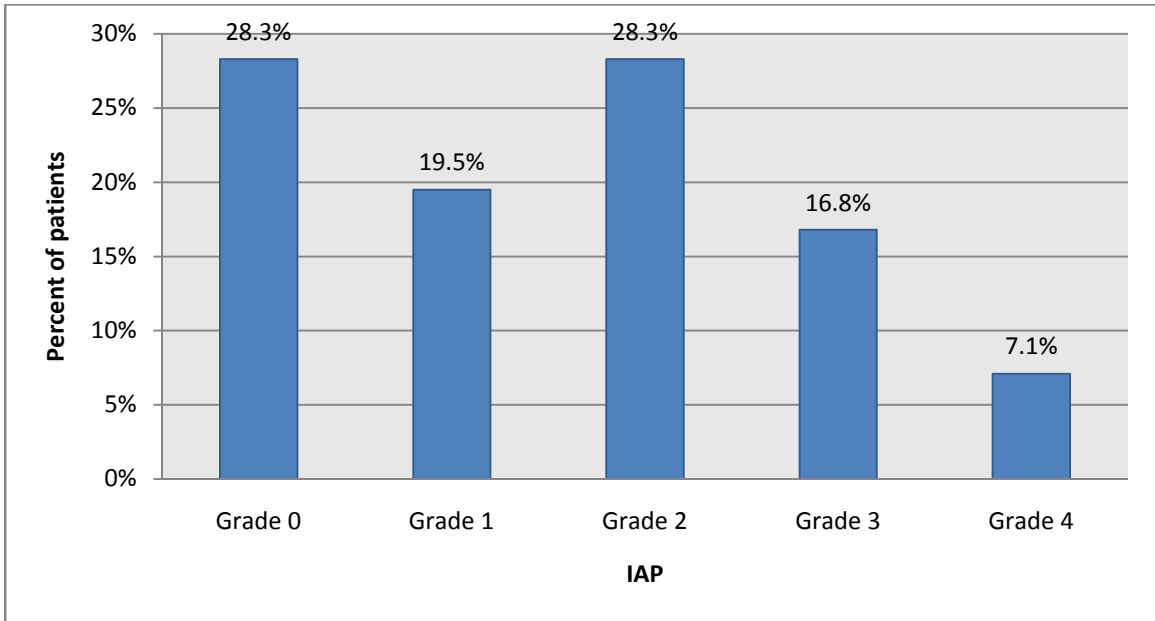


Figure 4: Patients with IAH when maximal IAP is considered

The x axis represent the severity of IAH as categorized in four grades from no IAH(grade 0) to most severe IAH (grade 4) based on the highest reading(maximal IAP) of the three IAP readings in the 24 hour period.The Y axis is the proportion of patients with IAH in % when maximal IAP is considered. This number is out of the total of 81 who had IAH when maximal IAP is considered

There were patient characteristics, clinical parameters and laboratory parameters that were found to be significantly associated with presence of IAH using chi square test for categorical variables(Gender) and Student t test for continuous variables with a significance level set at <0.005.When means IAP is considered; amount of IV fluids given in 24 hours, Hb, peak airway pressure (PAP) and ventilator mode were significant determinants of IAH development(Table 2)When considering maximal IAP; amount of IV fluids given in 24 hours, and hemoglobin levels were significant determinants of IAH(Table 3). In contrast; gender, age, maximal peak airway pressure, baseexcess, white cell count and platelets were not significant determinants of IAH

Table 3: Factors associated with IAH when considering mean IAP in 24 hours

	IAH		P value	Adjusted OR (95% CI)	P value
	Yes	No			
Gender					

Male	51 (64.6)	28 (35.4)	0.351	2.2 (0.5-10.3)	0.334
Female	25 (73.5)	9 (26.5)			
Age in years	38.0 (13.3)	35.4 (11.6)	0.316	1.04 (0.98-1.10)	0.249
^a Amount of IV fluids over 24 hours in ML	3949.6 (1431.5)	2931.1 (1121.8)	0.003	1.0 (1.0-1.002)	0.030
Number of pints of blood transfused over 24 hours	1.5 (1.7)	0.5 (1.2)	0.003	1.04 (0.59-1.80)	0.904
Fluid balance over 24 hours	1992.9 (1454.0)	1094.6 (927.6)	0.001	1.0 (1.0-1.002)	0.907
^b Peak airway pressure 24 hours	28.4 (1.7)	17.3 (1.9)	0.008	1.6 (1.1-2.4)	0.018
Ventilation mode(%)					
Bipap	1(20.0)	4(80.0)	0.039	1.23(0.8-1.69)	0.218
Capp	15(93.8)	1(6.3)	0.015	1.1(1.0-1.22)	0.328
ΣSIMV	60(65.2)	32(34.8)	0.034	1.4(0.78-2.04)	0.041
Base excess	-3.2 (7.2)	-1.0 (3.1)	0.085	1.10 (0.94-1.28)	0.230
WBC	12.2 (7.8)	11.1 (13.2)	0.606	1.01 (0.97-1.07)	0.505
[±] Hb	9.9 (3.2)	12.0 (1.9)	<0.001	0.60 (0.40-0.90)	0.012
Platelets	332.8 (128.0)	294.5 (90.3)	0.109	1.01 (1.00-1.02)	0.661

When mean IAP is considered with p value set at <0.05 at 95 % confidence interval, the following parameters were found to be significant determinants of risk of IAH: amount of iv fluids in 24 hours, number of pints of blood transfused in 24 hours, ventilation mode , peak airway pressure and hemoglobin level. Upon multivariate logistic regression, the following parameters remained significant:

A amount of IV fluids administered over 24 hour period.

B the peak airway pressure inch H₂O as displayed on the ventilator.

Σ The SIMV ventilation mode

± the hemoglobin levels in g/dl

Table 4: Factors associated with IAH when considering maximal IAP

Variable	IAH		P value	Adjusted OR (95% CI)	P value
	Yes	No			
Gender					
Male	54 (68.4)	25 (31.6)	0.231	1.0 (0.2-4.5)	0.954
Female	27 (79.4)	7 (20.6)			
Age in years	38.3 (13.3)	34.4 (11.2)	0.150	1.05 (1.00-1.12)	0.076
^π Amount of IV fluids over 24 hours in ML	3861.4 (1435.0)	2995.3 (1176.1)	0.003	1.00 (1.00-1.001)	0.025
Number of pints of blood transfused over 24 hours	1.4 (1.7)	0.5 (1.2)	0.10	0.96 (0.57-1.62)	0.881
Fluid balance over 24 hours	1914.3 (1443.0)	1153.1 (979.5)	0.007	1.00 (1.00-1.001)	0.797
Peak airway pressure 24 hours	27.2 (1.9)	17.5 (1.7)	0.135	1.2 (0.9-1.7)	0.253
Ventilation mode(%)					
Bipap	4(26.0)	1(33.0)	0.139	1.03(0.6-1.59)	0.308
Capp	22(73.8)	2(6.0)	0.241	1.1(0.97-1.20)	0.151
SIMV	44(55.2)	27(30.7)	0.444	1.0(0.78-2.24)	0.607
Base excess	-2.9 (7.1)	-1.3 (3.1)	0.226	1.1 (1.0-1.3)	0.183
WBC	11.9 (7.6)	11.5 (14.2)	0.843	1.01 (0.95-1.06)	0.786
^λ Hb	10.0 (3.1)	12.1 (2.0)	0.001	0.60 (0.41-0.87)	0.007
Platelets	331.6 (125.1)	291.4 (93.3)	0.108	1.01 (1.00-1.02)	0.119

When the highest IAP reading in 24 hours(maximal IAP) is considered with p value set at <0.05 at 95 % confidence interval, the following parameters were found to be significant determinants of risk of IAH:amount of iv fluids in 24 hours,haemoglobin level and fluid balance. Upon multivariate logistic regression, the following parameters remained significant:

Π The amount of IV fluids given over 24 hours

λ Hemoglobin level in gram per deciliter

4.2 Abdominal compartment syndrome(ACS)

Of the 113 patients sampled, five met the criteria for ACS in that they had severe IAH and at least one organ dysfunction/ failure. This represents a prevalence of 4.4 %.This was a small but heterogeneous group of patients with the following diagnosis/clinical impression: There was a middle aged man who was admitted following repair of ruptured slow leaking abdominal aortic aneurysm, a patient with acute pancreatitis with severe sepsis, one with extensive third degree burns, a polytrauma patient with missed blunt abdominal trauma and an elderly lady admitted after colectomy for gangrenous sigmoid volvulus.

All five patients had variable degree of multiple organ dysfunction/failure as evidenced by hematological profile, blood biochemistry and ventilator requirements and the team in critical care was notified of the high IAP readings and suspicion for ACS.Non-surgical interventions including insertion or repositioning of nasogastric tube(NG), insertion of flatus tube, careful titration of IV fluid requirements and appropriate adjustments of ventilator setting were instituted.

The patient with severe pancreatitis had been transferred to the critical care unit when he developed respiratory failure suspected to be due to ARDS.While in CCU he had progressive abdominal distention but bowel sounds were present and he was passing stool. He had oliguria, elevated urea and creatinine and serum bilirubin. The mean IAP over 24 hour period was 25mmhg with maximal IAP of 27mmhg.Non-surgical interventions i.e. NG tube, flatustube,optimizing analgesia and titrating IV fluids to needs and response did not work. He underwentdecompressivelaparotomy, debridement of necrotic pancreatic tissue and temporally closure of the abdomen with Bogota bag. Following the intervention there was dramatic improvement in urine output, reduction in serum bilirubin and urea and creatinine.

The burns patients had sustained open flame mixed second and third degree burns to the trunk, upper limbs (estimated burnt surface area (BSA) 40%) and had signs of inhalational burns as result of a kerosene stove explosion. Before admission to our burns critical care unit, he had received 14l of normal saline and had been catheterized and central venous line placed at the referring facility where he had been admitted for 48 hours. On examination he was found to have extensive firmeschars on the anterior chest and abdomen. He was sedated,

intubated and mechanically ventilated and continued to have IV fluids resuscitation. In spite of aggressive fluid replacement with ringer's lactate, urine output remained at <30ml per hour, mean systolic BP at 84mmhg, creatinine and urea were markedly elevated at 256 micromole/l and 23mmol/l. He had a peak airway pressure of 47cmH₂O. During the 24 hours following admission, the mean IAP was 23mmhg and maximal IAP 28mmhg. Diagnosis of ACS was suspected and measures to reduce the IAP instituted i.e. nasogastric decompression, flatus tube, crystalloids reduced and colloids introduced, diuretics administered and escharotomy done on the chest and abdomen. There was only modest reduction in mean and maximal IAP and modest improvement on renal status, BP and peak airway pressure and a decision for decompressive laparotomy was made but he succumbed before this could be done.

The polytrauma patient was a 43 year old motorcyclist who collided head on with a speeding car and was thrown off the motor cycle, sustaining severe head injury (suspected diffuse axonal injury on brain CT scan), multiple rib fractures with haemopneumothorax and lung contusions, lower limb fractures and abdominal trauma. He was seen at a county hospital and admitted to the intensive care unit, he was intubated and started on mechanical ventilation, received IV fluids, had chest drain inserted to drain pneumohaemothorax, the fractures were splinted and received five pints of packed red blood cells to correct hemorrhagic shock. He stayed at the county facility for 72 hours before being referred to the main referral hospital when he continued to deteriorate. At admission to our CCU, he had systolic BP of 68mmhg, no urine output, massively distended abdomen and initial IAP reading was 33hg with a mean IAP of 30mmhg and a maximal IAP of 41mmhg. A diagnosis of ACS was entertained and immediate decompressive laparotomy performed where the findings were: massive bowel distension, edema and 300ml of blood in the pouch of Douglas with the solid organs intact, temporary closure of the abdomen was done using Bogota bag. There was gradual increase of blood pressure, reduction in IAP and he started diuresis though he remained intubated and ventilated for the severe head injury. External fixators were used for temporary fixation of the lower limb fractures.

There was a 52 year old man who developed ACS following colectomy for gangrenous sigmoid colon. He presented in shock, metabolic acidosis and oliguria. He had fluid resuscitation using crystalloids and haemacel before undergoing the operation. On the second postoperative day, he was noted to be restless, tachypnoeic, SBP 88mmhg, oliguric in spite of adequate IV fluids and had moderate abdominal distension. He had urea of 29mmol/l, creatinine 230micromol/l, PH 7.32, pO₂ 50mmhg, pCO₂ at 60mmhg, hco₃ 21mmol/l. He was

admitted to CCU, where his intubated and ventilated. His mean IAP was 24mmhg with maximal IAP at 29mmhg. Noninvasive means to reduce IAP did not succeed and he had abdominal exploration where massively distended edematous intestines were the only positive finding. The abdomen was closed temporarily with Bogota bag. He responded well with IAP readings dramatically falling, and renal and respiratory parameters also improving. He was intubated 3 days later and definitive abdominal closure done after 12 days.

The fifth ACS patient was readmitted to CCU three days following repair of slow leaking ruptured abdominal aortic aneurysm. The patient had been referred to the cardiothoracic unit from a county hospital with a diagnosis of infrarenal abdominal aortic aneurysm measuring 5.9cm in greatest diameter. He was 56 years old, overweight (BMI 31), a heavy smoker with poorly controlled diabetes and hypertension, who had presented at the accident and emergency department in shock. Preoperatively, he had received 8l of crystalloids, 2.5 l of haemacel and 6 pints of packed red cells. He underwent emergency open repair of the aneurysm, with aortic cross clamp time reported as 1 hour and 40 minutes. He was taken to critical care unit where he stayed for 3 days then discharged to the general ward. While in the ward he was noted to have oliguria, tachypnoea, tachycardia, oxygen saturation at 77% and had a systolic BP of 80mmhg, with abdominal distension and reduced bowel sounds. At this point the serum urea was 44mmol/l, creatinine at 660micromol/l, haemoglobin at 7.8g/dl, PH 7.28, PO2 at 53mmhg and PCO2 at 32mmhg with a HCO3 at 19mmol/l. He was readmitted to CCU, where he was sedated, intubated and mechanically ventilated, had NG tube placed, diuretics administered. The mean IAP was 22mmhg with a maximal reading of 31mmhg. A diagnosis of ACS was made, he had re-exploration of the abdomen. Intraoperatively, he had massively distended small bowel, distended colon with multiple ischemic patches on splenic flexure, descending and sigmoid colon. The vascular graft was in place and the repair was intact. There was a small amount of serosanguinous fluid in the pouch of Douglas. The abdomen was temporarily closed with a Bogota bag. Post operatively he did well, with the IAP falling dramatically, the urine output increased and his respiratory parameters improved and was off the ventilator after four days. The definitive closure of the abdomen was done 14 days later. Table 5 summarizes the patient characteristics for the five patients deemed to have ACS.

Table 5: Clinical, laboratory and ventilator parameters for patients with ACS

Variable	Mean (SD)	Min-Max
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Mean IAP in 24 hours	26.1 (3.0)	22.3-30.0
Maximum IAP in 24 hours	30.0 (2.4)	27.0-31.0
^σ BUN	36.0 (13.7)	20.0-56.0
^ρ Serum creatinine	194.4 (13.0)	179.0-213.0
^ϕ Platelet	128.2 (26.8)	97.0-150.0
^Φ Total Bilirubin	39.8 (17.4)	21.0-68.0
Peak airway pressure	49.2 (7.2)	57.0-23.0

The laboratory tests were performed once, at the time of admission. The other parameters were recorded with each of the three intraabdominal pressure (IAP) measurements. Blood urea nitrogen and creatinine were used to assess renal function, platelet count as a marker of coagulation status and total bilirubin as a test of liver function.

^σ Blood urea Nitrogen (BUN) the normal range for our lab 3.1-8.3mmol/l

^ρ serum creatinine, the normal range for our lab is 40-80 μ mol/l

^ϕ normal platelet count is 150-450 $\times 10^9/l$

^Φ Total bilirubin reference range is 2-22mmol/l

When means IAP is considered, of those with IAH, age, ventilator mode, amount of IV fluids in 24 hours and fluid balance determined risk of progression to ACS. Table 5 below summarizes the factors considered.

Table 6: Factors associated with increased risk of progressing IAH to ACS

Variable	ACS	IAH	P value
Gender			
Male	4 (80.0%)	51 (66.2%)	1.000
Female	1 (20.0%)	26 (33.8%)	
Ventilation mode			
bipap	2 (40.0%)	1 (1.3%)	0.149
cpap	0 (0.0%)	63 (83.1%)	0.441
[‡] simv	3 (60.0%)	12 (15.6%)	0.041
[Ⓜ] Age in years	53.2 (7.6)	38.5 (13.4)	0.018
[Ⓝ] Amount of IV fluids over 24 hours in ML	5800 (5700-6200)	3500 (2700-4900)	0.005
Fluid balance over 24 hours	2100 (1900-3800)	1300 (900-2700)	0.051
Number of pints of blood transfused over 24 hours	2 (2-2)	0 (0-2)	0.324
Peak airway pressure 24 hours	18 (16.5-20.5)	19 (17-19)	0.942
White blood cell count	12.1 (10.7-13.4)	10.4 (8.0-13.8)	0.783
Haemoglobin	9.3 (8.4-9.7)	10.4 (7.4-11.9)	0.651
Platelet count	112 (94-163)	313 (287-401)	0.191
Base excess	2.0 (-8.6-2.1)	-2.4 (-7.4-2.3)	0.807

[‡]Of the ventilation modes, synchronised intermittent mandatory ventilation (SIMV) was significantly associated with progression of IAH to ACS.

[Ⓜ]of those with IAH,those who progressed to ACS were significantly older.

[Ⓝ] similar to IAH,amount of IV fluids in 24 hours was a significant determinant IAH progressing to ACS

5.0 CHAPTER FIVE: DISCUSSION

The main aims of this study were to document the prevalence of intraabdominal hypertension and abdominal compartment syndrome and factors significantly associated with development of the same. The prevalence of IAH and ACS differed depending on whether mean or maximal IAP was used. Mean IAP showed an IAH prevalence of 67.3% and when maximal IAP was considered 71.7%. Malbrain et al, noted that mean IAP tend to down grade intra-abdominal pressure values and may lead in some cases of IAH and ACS being missed¹⁷. To improve on accuracy and reliability of mean IAP would require frequent IAP measurements (at least every four hours and more frequent if IAP >12mmHg) or continuous measurement^{22,32}. In the absence of automated IAP measurement devices and in a resource constrained set up, like KNH, that would strain the critical care unit workforce. While maximal IAP may be seen as over-diagnosing IAH and ACS, the overall result is positive in terms of diagnosing and prognosticating these patients^{29,37}.

This study revealed an IAH prevalence that is remarkably higher than that quoted in other studies^{17,29,37}. The prevalence of ACS is however comparable with what is published literature. Taurai studied a small population of post laparotomy surgical patients in critical care, where among the 38 patients studied, the prevalence of IAH was 57% when considering mean IAP and 60% when maximal IAP was considered with ACS prevalence stated as 8%²⁹. Malbrain et al carried out the largest multicenter prevalence prospective study of IAH in 13 intensive care units (ICUs) using maximal IAP and found the prevalence of IAH to be 65% and the prevalence of ACS to be 5% among surgical^{36,37}. A possible explanation of such a high occurrence of IAH in our study is the fact that due to pressure for bed space in our critical care units, at any given time, the patients in these units are more sick and therefore at a higher risk for IAH than centers with more and bigger CCUs. The prevalence of ACS in our study population may have been higher given that the length of follow up of patients with IAH but not deemed to have ACS was restricted to the 24 hour period of monitoring IAP.

Large amount of IV fluids administered over 24 hours and the attendant positive fluid balance were significantly associated with development of IAH and ACS. This is in keeping with findings by other investigators^{38,39}. This results from excessive extracellular fluid accumulation within the intestine and the contents there in⁴⁰. This is best avoided by calculating and adhering to individual patient fluid needs and response.

Low haemoglobin and the number of pints of blood transfused to correct the same were positively associated with risk of development of IAH and ACS. A preresuscitation Hb value that is 8g/dl or less has been reported to be associated with high risk of developing IAH and ACS in acutely ill patients both in the emergency department and in the first 24 hours of their care in critical care units^{4,41}. In addition, severely anaemic patients requiring transfusion of at least three pints of packed red cells have high risk of developing IAH and ACS⁴¹. Although aggressive use of blood and blood products can contribute to fluid overload and cause metabolic acidosis hence worsening the capillary leakage, low crystalloids to blood products ratio help to minimise the total volume of fluid required to restore effective circulating volume⁴².

When mean IAP is considered, the subset of patients who had IAH had significantly higher peak airway pressure (PAP) readings compared to those without. This is in keeping with other published work that showed that when considering mean IAP both peak inspiratory and mean airway pressures are significantly increased in patients with IAH and ACS⁴³. Though a positive finding in IAH and ACS, airway pressures do not accurately reflect IAP and cannot be substituted for IAP measurements in patients at risk for IAH/ACS because there are lung and airway diseases that affect peak inspiratory and mean airway pressure¹¹.

Ventilatory mode had an influence on risk of developing IAH and ACS. SIMV mode was associated with higher odds of developing IAH when mean IAP was considered. Mehrdad et al reported a significant relationship between ventilation mode and IAP, demonstrating that IAP is mostly affected by SIMV, followed by BIPAP and CPAP in that order⁴⁴. This is explained partly by the finding that pressure support ventilation (PSV) is associated with less IAP elevation and CPAP, BIPAP, SIMV have highest PSV in that decreasing order^{45,46}.

In this group, there was no statistically significant difference in base excess between patients with IAH and those without. Base excess and lactate are useful markers for assessing resuscitation adequacy and response among critically ill patients. G. Arabadzhiev et al evaluated a cohort of 43 surgical patients at risk of IAH and ACS and demonstrated that patients with grade two and grade three IAH had high base excess⁴⁷. Base excess and lactate, as resuscitation end points and biochemical markers of cellular metabolic derangements, have been shown to be useful prognostic indicators in critically ill patients⁴⁸.

The incidence of IAH in patients with severe acute pancreatitis (SAP) is high (60-80% depending on the population considered), with 1 in 3 of those with IAH developing full blown

ACS, with mortality rate nearly 70%⁴⁹. Factors responsible include: pancreatic and peripancreatic edema (aggravated by excessive IV fluids), ascites, ileus, abdominal wall edema and abdominal pain⁵⁰.

Although surgery is very effective in managing ACS, it is reserved for those who fail to respond to non-surgical therapeutic interventions such as gut decompression, minimising IV fluids given and using more of hypertonic solutions and colloids, use of muscle relaxants, percutaneous drainage and reducing enteral feeds. All that considered, prompt recognition of failed medical management should lead to timely surgical decompression to ensure favourable outcome^{21,51}.

Though ACS is characterized by a tensely distended abdomen, this may not be seen in patients with major torso burns with eschar formation. The risk factors for IAH and ACS in major burns patients are: Inhalational burns, burns surface 70% or greater, massive fluid resuscitation and deep circumferential torso burns⁵². In these situations, a combination of SIRS, capillary leak and third spacing and extrinsic compression of chest and abdomen by the eschars contribute to development of IAH and ACS⁵³.

While diuresis, sedation, adequate analgesia, escharotomy and use of colloids and hypertonic saline-dextran may help to reduce IAP in mild and moderate cases of IAH, in established ACS decompressive laparotomy is the only treatment option that help relieve the pressure and improve organ function^{52,53}. Even with such drastic and invasive intervention, in severe burns complicated by ACS, mortality remains very high⁵³. Since major burns patients are prone to IAH and ACS, routine IAP monitoring is key in preventing this fatal complication^{53,54}.

Polytrauma patients are at risk of ACS from SIRS causing massive capillary leak and third spacing. Another contributing factor is massive blood loss necessitating aggressive resuscitation with IV fluids and blood transfusion, and intraperitoneal and retroperitoneal bleeding⁵⁵. Even in these critically ill patients, decompressive laparotomy reduces IAP and may also discover major bleeding which can be treated surgically⁵⁶.

ACS incidence following open repair of a ruptured AAA is reported to be as high as 30% with a mortality approaching 70%⁵⁷. Massive fluid transfusion, shock at admission and prolonged cross clamp time are recognised risk factors for ACS development. Abdominal decompression is vital to achieve favourable outcome when managing these patients⁵⁸. Prevention of the ACS, with routine measurement and early recognition of rising IAP and

expedited decompression of the tense abdomen can lead to mortality reduction after aneurysm repair ⁵⁹.

We had a case of an elderly lady who developed ACS following sigmoid colectomy and Hartmann's colostomy for gangrenous sigmoid volvulus. This is a very rare occurrence; Mehmet et al reported on a case of ACS due to a distended rectal stump following colectomy for gangrenous sigmoid volvulus ⁶⁰.

Though the number of patients who had ACS is small(five),on subgroup analysis, there were significant differences in age, ventilatorymode, amount of iv fluids in 24 hours and fluid balance between IAH and ACS groups. Those with IAH who went on to develop ACS,wereolder,had higher fluid balance ,received more iv fluids in 24 hours and more were on SIMV ventilatorymode.Advanced age is associated with higher risk of IAH and progression to ACS and poor outcomes ⁶¹.

The ventilation mode had an effect on risk of developing both IAH and ACS, with SIMV showing positive correlation. It has been shown that IAP is mostly affected by SIMV, followed by BIPAP and CPAP ⁴⁴.Pressure support ventilation(PSV) is associated with less IAP elevation and CPAP,BIPAP,SIMV have highest PSV in that decreasing order ^{45,46}.

5.1 CONCLUSION

In this mixed population of surgical patients, the prevalence of intraabdominal hypertension and abdominal compartment syndrome is high and could be a significant cause of morbidity and mortality. This is due to the deleterious effects of IAH and ACS in virtually all organ systems causing altered organ perfusion and end organ function.

Amount of IV fluids administered over 24 hours,fluidbalance,haemoglobinlevels,high transfusion requirements and SIMV ventilation mode are determinants of IAH.Of those with IAH,age,amount of IV fluids, fluid balance and ventilation mode are important determinants of risk of progression to ACS.

5.2 RECOMMENDATIONS

1. There is need for further research with larger sample size, more frequent IAP measurements and a follow up period to determine if there are more risk factors, document response to various interventions and to establish contribution of IAH/ACS to clinical outcomes i.e. morbidity and mortality of surgical patients in KNH ICUs.
2. In a bid to ease measurement of IAP, it would be important to have transducers instead of using the more cumbersome and possibly less accurate saline manometer.
3. This study has revealed a high prevalence of IAH/ACS and justifies teaching CCU teams on how to measure IAH, risk factor reduction and to start routine IAP measurement for at risk patients.
4. We could not assess the possible contribution of BMI to risk of IAH/ACS due to lack of ICU beds that are fitted with weighing device. It would be important to acquire these special beds because weight measurement is important to guide fluid administration and other therapies.
5. It would be important to explore possibility of developing and testing a risk prediction model for at risk patients using patient characteristics, clinical and laboratory data.

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APPENDICES

Appendix I :Consent

INTRA-ABDOMINAL HYPERTENSION AND COMPARTMENT SYNDROME IN SURGICAL PATIENTS ADMITTED IN CRITICAL CARE UNITS AT KENYATTA NATIONAL HOSPITAL.

English version

This Informed Consent form is for surgical patients aged thirteen years and older admitted at the Kenyatta National Hospital critical care units. This consent will be administered to the patient's next of kin. We are requesting these patients to participate in this research project whose title is "Intra-abdominal hypertension and compartment syndrome in surgical patients admitted in critical care units at Kenyatta National hospital".

Principal investigator: Dr. Alex MuturiMwihaki.

Institution: School of Medicine, Department of surgery- University of Nairobi

Supervisors: Professor Peter L.W Ndaguatha and Dr Daniel Ojuka.

This informed consent has three parts:

1. Information sheet (to share information about the research with you)
2. Certificate of Consent (for signatures if you agree to take part)
3. Statement by the researcher

You will be given a copy of the full Informed Consent Form.

Part I: Information sheet

My name is Dr Alex MuturiMwihaki, a post graduate student at the University of Nairobi's School of Medicine,department of surgery. I am carrying out a study to determine incidence and early predictors of intra-abdominal hypertension and abdominal compartment syndrome in surgical patients admitted to critical care units at Kenyatta National Hospital. These are conditions that result from buildup of pressure inside the abdomen from accumulation of air,blood,pus or due to swelling of tissues within the abdomen. This will be determined by data collection through filling of questionnaires and measurement of intra-abdominal pressure using intravesical catheter method-urinary catheter will be connected to a tube attached to manometry ruler with pressure readings. The measurements will be taken thrice: when we

first see the patient, twelve hours later and at twenty-four hours. Blood specimen will also be collected for Total blood count, serum bilirubin, serum urea, serum creatinine, and base excess-the tests assesses the functioning of the kidneys, liver and blood circulation. The blood tests will also be performed thrice. The information obtained will help doctors know the magnitude of the problem in our critical care units and determine the surgical diagnoses and patient characteristics associated with intra-abdominal hypertension and abdominal compartment syndrome, in a bid to improve on early recognition and timely intervention crucial to successful treatment and improvement of outcome. Any patient confirmed to have ACS will be recommended to have decompressive laparotomy and patient with grade 2 and 3 IAH in whom organ dysfunction hasn't set in, will be recommended to undergo aggressive non-operative treatment measures to reduce IAP and prevent ACS

I am inviting you to participate in my study and you are free to either agree immediately after receiving this information or later after thinking about it. You will be given the opportunity to ask questions before you decide on behalf of your kin and you may talk to anyone you are comfortable with about the research before making a decision. After receiving this information concerning the study, please seek for clarification from either myself or my assistant if there are words or details which you do not understand.

All the information which you provide regarding your kin will be kept confidential and no one but the researchers will see it, their name will not appear in any document or any specimen container. The information about them will be identified by a number and only the researchers can relate the number to patient. The information will not be shared with anyone else unless authorized by the Kenyatta National Hospital/University of Nairobi – Ethics and Research Committee (KNH/UoN-ERC).

Your kin's involvement in this research will be through an interview and clinical evaluation and they will not expose themselves to any risks if you consent on their behalf, to participate. There will be no extra cost incurred for participating in the study. Participation in this study is out of your own free will, your kin will not be denied medical care in case you refuse to participate in the study. You may stop the participation at any time with no consequences whatsoever. All the information that you give us will be used for this research only.

This proposal has been reviewed and approved by the KNH/UoN-ERC which is a committee whose work is to make sure research participants are protected from harm. The contact information is given below if you wish to contact any of them for whatever reason;

- **Secretary, KNH/UoN-ERC**

P.O. Box 20723 KNH, Nairobi 00202

Tel 726300-9

Email: uonknh_erc@uonbi.ac.ke

- University of Nairobi research supervisors

- 1. Professor Peter L.W Ndaguatha**

MBChB, M.Med F.C.S (ECSA) Fellow of Urology (U.K)

Thematic Head of Urology

Department of Surgery, School of Medicine, University of Nairobi

P.O. Box 19676 KNH, Nairobi 00202

Tel # 0202726300

- 2. Dr Daniel Ojuka**

MBChB (U.O.N), M.Med Surgery (U.O.N), F.C.S (ECSA)

Department of Surgery, School of Medicine, University of Nairobi

P.O. Box 19676 KNH, Nairobi 00202

Tel # 0202726300

- **Principle researcher:**

Dr. Alex MuturiMwihaki

Department of Surgery, School of Medicine, University of Nairobi

P.O. Box 19676 KNH, Nairobi 00202

Mobile phone 0721811483

Part ii: Consent certificate by patient

I.....freely give consent of my kin, named.....to take part in the study conducted by Dr. Alex MuturiMwihaki, the nature of which has been explained to me by him/his research assistant. I have been informed and have understood that my participation is entirely voluntary and I understand that I am free to withdraw my consent at any time if I so wish and this will not in any way alter the care being given to my child kin. The results of the study may directly be of benefit to my kin and other patients and more significantly to the Medical professionals to better understand the disease namely Intra-abdominal Hypertension and Abdominal Compartment Syndrome that finally translate to early diagnosis and better management of patients who will in future present with this disease .

.....

Signature/left thumb print (Next of kin)

Date.....

<p>Thumb print of next of kin if Unable to sign due to illiteracy</p>

Statement by the witness if next of kin is illiterate

I have witnessed the accurate reading of the consent form to the participant, and the individual has had the opportunity to ask questions. I confirm that the individual has given consent freely.

Name of witness.....

Signature of witness.....

Date.....

Part iii: Statement by the researcher

I have accurately read out the information sheet to the participant, and to the best of my ability made sure that the participant understands the following:

- Refusal to participate or withdrawal from the study will not in any way compromise the quality of care and treatment given to the patient.
- All information given will be treated with confidentiality.
- The results of this study might be published to enhance knowledge and to help improve early recognition of pathological intra-abdominal hypertension and compartment syndrome and timely corrective intervention in order to improve outcome.

I confirm that the participant was given an opportunity to ask questions about the study, and all the questions asked by the participant have been answered correctly and to the best of my ability. I confirm that the individual has not been coerced into giving consent, and the consent has been given freely and voluntarily.

A copy of this Informed Consent Form has been provided to the participant.

Name of researcher taking consent.....

Signature of researcher taking the consent.....

Date.....

2 (b) Kiswahili version'

Fomu ya idhini

(i) Sehemu ya kwanza – Maelezo ya Doktorimtafiti.

Mimi ni Dkt. Alex Muturi Mwiwaki, kutoka shule ya Elimu ya Afya idara ya upasuaji Chuo Kikuu cha Nairobi (University of Nairobi). Ninafanya utafiti wa kuangalia kiwango cha presa ndani ya tumbo na hali inayotokana na presha hiyo kupanda kupita kiasi, katika wagonjwa wanao hudumiwa katika idara ya upasuaji haswa wale waliolazwa chumba cha wagonjwa mahututi.

Ningependa kumjumuisha mwanao / jamaa wako katika utafiti huu wangu. Ninafanya hivyo kwa kuuliza maswali fulani ya afya, kupima viwango vya presha ndani ya tumbo kwa kutumia mpira maluum uliotiwa ndani ya kibofu. Katika utafiti huu utatakiwa kutoa tariifa inayomhusu mwanao / jamaa wako. Habari zote zitakazo kusanywa zitashughulikiwa kwa siri na hazita sambazwa ila tu kwa ruhusa kutoka kwa mkurugenzi mkuu wa utafiti wa chuo kikuu cha Nairobi na hospital kuu ya Kenyatta.

Utafiti huu utawasaidia madaktari kuelewa tatizo hili kwa kina, hivyo basi kuweza kuwahudumia wagonjwa wao kwa ubora zaidi na kuwapa mbinu ya kutambua tatizo hili kwa haraka iwezekanavyo, kupatiana tiba ya dharura, katika juhudi za kupunguza maafa yanayo tokana na shidahii. Kuhusika kwa mwanao / jamaa wako kwenye utafiti huu hakuna malipo yoyote ila ni kwa hiari yako mwenyewe na pia unaweza kujiondoa kushiriki katika utafiti wakati wowote bila kuhatarisha matibabu ya mwanao / jamaa wako katika Hospitali Kuu ya Kenyatta.

Naomba mimi ama wasaidizi wangu katika utafiti wa kuulize maswali ambayo yatajibiwa kwa fomu maalum. Habari yote ambayo utatuarifu ni ya siri kati yako nasisi watafiti na haitaenezwa kwa watu wengine. Jina la mwanao / jamaa wako halitaandikwa kwenye fomu yoyote wala kwenye vipimo vyovyote. Unaweza kuuliza maswali yeyote kuhusu utafiti huu na ukiridhika tafadhali ijaze fomu ya idhini iliyopo hapa chini. Unaweza pia kuuliza swali lolote baadaye kwa kupiga simu kwa mtafiti mkuu ama mkuu wa idara ya upasuaji katika chuo kikuu cha Nairobi ama walimu wasimamizi wa utafiti ukitumia nambari za simu zifuatazo;

Katibu wa utafiti,

Hospitali kuu ya Kenyatta na Chuo kikuu cha Nairobi.

Sanduku la Posta 20723 KNH, Nairobi 00202.

Nambari ya simu 726300-9.

Walimu wa kuu wa Chuo kikuu cha Nairobi:

1. Professor Peter L.W Ndaguatha,

MBChB, M.Med, F.C.S (ECSA) Fellow of Urology (U.K)

Sanduku la Posta 19676 KNH, Nairobi 00202. Nambari ya simu: 0202726300

2. Dkt Daniel Ojuka,

MBChB (U.O.N), M.Med Surgery (U.O.N), F.C.S (ECSA)

Sanduku la Posta 19676 KNH, Nairobi 00202. Nambari ya simu: 0202726300

• **Mtafiti: Alex Muturi Mwhiki,**

Idara ya Upasuaji ya Shule ya Afya – Chuokikuucha Nairobi,

Sanduku la Posta 2678 KNH Nairobi 00202. Nambari ya simu ya rununu
0721811483.

(ii) Sehemu ya pili – Idhini ya mgonjwa.

Mimi (Jina).....kwa niaba ya mgonjwa wangu
(Jina la Mgonjwa

.....) nimekubali kushiriki katika utafiti huu unaofanywa na
Daktari Alex Muturi Mwhiki kutokana na hali ambayo nimeelezwa na sio kwa malipo ama
shurutisho lolote.

Nimeelewa kwamba ninaweza kujiondoa wakati wowote nitakapo na hatua hii haitahatarisha
matibabu anayo yapata mgonjwa wangu. Matokeo ya utafiti ya wezakuwa ya
manufaakwangu ama kwa wagonjwa wengine kwa jumla na hata madaktari wenyewe, kwa
kuendeleza elimu, na hata kupunguza vifo vinavyo epukika.

Sahihi /ama alama ya kidole cha gumba katika sanduku →

Tarehe.....

Siku/Mwezi/Mwaka

Jina la shahidi.....

Sahihi.....

Tarehe.....

(Siku/Mwezi/Mwaka)

(iii) Sehemu ya tatu – Dhibitisho la mtafiti

Hii ni kuidhinisha ya kwamba nime mueleza msimamizi wa mshiriki (mgonjwa) kwenye utafiti kuhusu utafiti huu na pia nimempa nafasi ya kuuliza maswali. Nimemueleza yafuatayo;

- Kwamba kushiriki ni kwa hiari yake mwenyewe bila malipo.
- Kushiriki hakutasababisha madhara ama kuhatarisha maisha kamwe.
- Anaweza kujiondoa kutoka kwa utafiti huu wakati wowote bila kuhatarisha matibabu anayoyapata katika hospital kuu ya Kenyatta.
- Habari ambazo atapeana hazitatangazwa hadharani bila ruhusa kutoka kwake (mshiriki) na pia kutoka kwa mdhamini mkuu wa utafiti wa hospital kuu ya Kenyatta na chuo kikuu cha matibabu.

Jina la anayesimamia mshiriki

Sahihi.....Tarehe.....

Kidole cha gumba kwa

yule asiyelewa

kuandika

AppendixII: Data Collection Sheet

Intra-abdominal hypertension and abdominal compartment syndrome among surgical patients in the critical care units at kenyatta National Hospital.

Date_____

Patient identification code _____

Socio-demographic data

Admission date _____

Age (yrs) _____ sex _____

Diagnosis at admission:

Abdominal pelvic surgical condition_____ Non abdominal pelvic
diagnosis_____

Reason for admission to critical care unit_____

Fluids administration

Amount of IV fluids given over 24 hours in ML_____

Urine output in 24 hours_____

Fluid balance over 24 hours_____

Number of pints of blood transfused over 24 hours_____

Vital signs

Pulse 0hrs_____ 12hrs_____ 12hrs_____ 24hrs_____

Systolic Blood pressure 0hrs_____ 12hrs_____ 24hrs_____

Respiratory rate ohrs_____ 12hrs_____ 24hrs_____

Peak airway pressure 0hrs_____ 12hrs_____ 24hrs_____

Temperature 0 hr_____ 12hrs_____ 24hrs_____ -

Glasgow coma scale at admission_____

Intra-abdominal pressure readings:

0hr _____ 12hrs _____ 24hrs _____

Laboratory investigations

Total blood count at _____

Serum creatinine _____

Serum urea _____

Serum bilirubin _____

Base excess _____

Appendix III: Letter of Approval from KNH/ UoN-ERC Letter of Approval



UNIVERSITY OF NAIROBI
COLLEGE OF HEALTH SCIENCES
P O BOX 19676 Code 00202
Telegrams: varsity
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Facebook: <https://www.facebook.com/uonknh.erc>
Twitter: @UONKNH_ERC https://twitter.com/UONKNH_ERC



KENYATTA NATIONAL HOSPITAL
P O BOX 20723 Code 00202
Tel: 726300-9
Fax: 725272
Telegrams: MEDSUP, Nairobi

Ref: KNH-ERC/ Mod&SAE/132

28th April, 2016

Dr. Alex Muturi
Dept. of Surgery
School of Medicine
University of Nairobi

Dear Dr. Muturi

Re: Approval of change of study title " Prevalence and predictors of intra-abdominal hypertension and compartment syndrome in surgical patients in Critical Care Units at KNH (P570/09/2014)

Your communication of March 22, 2016 refers.

The KNH- UoN Ethics and Research Committee has reviewed and approved change of study title from 'Incidence and Early Predictors of intra-abdominal hypertension and Compartment Syndrome in Surgical patients admitted in Critical Care Units at Kenyatta National Hospital' to ' Prevalence and predictors of intra-abdominal hypertension and compartment syndrome in surgical patients in Critical Care Units at KNH'.

Yours sincerely,

PROF. M.L. CHINDIA
SECRETARY, KNH-UON ERC

c.c. The Principal, College of Health Sciences, UoN
The Deputy Director, CS, KNH
The Chair, KNH- UoN ERC
The Dean, School of Medicine, UoN
The Chair, Dept. of Surgery, UoN

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