THE PREVALENCE OF INTRAABDOMINAL HYPERTENSION AND ITS ASSOCIATION WITH MORTALITY IN POST EMERGENCY LAPAROTOMY AT HOSPITAL TENGKU AMPUAN AFZAN (HTAA), KUANTAN

BY

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A dissertation submitted in fulfilment of the requirement for the degree of Master of Surgery (General Surgery)

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ABSTRACT

The purpose of this study was to measure the prevalence of Intraabdominal hypertension (IAH) and its complications in relation to mortality rate. A double-blind prospective study of all post emergency laparotomy general surgical patients in ICU HTAA, Kuantan from June 2014-June 2015. Intraabdominal pressure (IAP) was measured by intravesicular technique using a manometry; taken once daily (first reading within 24 hours of admission). If IAP exceeds 10mmHg, 2 subsequent readings were taken 4 to 6 hours apart. Data included the demographics, clinical, Sequential Organ Failure Assessment (SOFA) score and Acute Physiology And Chronic Health Evaluation II (APACHE II) score . We enrolled 51 patients, age of the subjects range from 14 to 82 with mean of age at 49.22 ± 17.35. Prevalence of IAH was 73% and 18% had abdominal compartment syndrome (ACS). Mean for SOFA and APACHE II score were 8.10 ± 4.920 and 14.57 ± 6.152, respectively. IAH (p=0.067), per se was found to be not associated with mortality but was associated with Acute Kidney Injury or Renal replacement Therapy (p=0.037), duration in ICU (p=0.005), duration on ventilator (p=0.005) and Max SOFA score (p=0.006). ACS (p=0.001) was found to have significant association to mortality, Acute Kidney Injury or Renal replacement Therapy (p=0.037), duration in ICU (p=0.044), duration on ventilator (p=0.024), Max SOFA score (p=<0.001), and higher surgical experience (p=0.009). The factors associated to mortality in this study were presence of AKI or RRT (p=0.001), duration on ventilator (p=0.021), SOFA score (p=0.001), and APACHE II score (p=0.003). Post laparotomy patients have a higher probability of developing IAH and ACS in this study. This furthermore stresses the importance of monitoring of IAH/IAP, in order to provide a timely identification, treatment and management of IAH in order to reduce the complications of organ dysfunction and ACS, and thus reducing the mortality rate.

APPROVAL PAGE

I certify that I have supervised and read this study and that in my opinion, it conforms to acceptable standards of scholarly presentation and is fully adequate, in scope and quality, as a dissertation for the degree of Master of Surgery (General Surgery).

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DECLARATION

I hereby declare that this dissertation is the result of my own investigation, except where otherwise stated. I also declare that it has not been previously or concurrently submitted as a whole for any other degrees at IIUM or other institutions.

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CHAPTER ONE

INTRODUCTION

1.1 BACKGROUND OF THE STUDY

Intra abdominal hypertension (IAH) is common with a prevalence of approximately 40% in the critically ill population and is an independent risk factor for mortality (OR 1.85). Intra abdominal hypertension was first described around 150 years ago and in the year of 1989, abdominal compartment syndrome (ACS) was first mentioned by Fietsam (Sugrue and Buhkari, 2009). The World Society of the Abdominal Compartment Syndrome (WSACS) defined Intra abdominal hypertension (IAH) as sustained or repeated pathologic elevation of IAP ≥ 12 mmHg meanwhile Abdominal compartment syndrome (ACS) is defined as a sustained IAP > 20 mmHg (with or without an abdominal perfusion pressure, APP < 60 mmHg) that is associated with new organ dysfunction / failure (Kirkpatrick *et. al.*, 2013).

The incidence of IAH is around 40-80% in all critically ill patients admitted to Intensive Care Unit (ICU) and the incidence of ACS is around 2-8% (Sugrue and Buhkari, 2009; Cheatham, 2009; Mayer et. al.,2010; Luckianow *et. al.*, 2012; Malbrain *et. al.*, 2005; Scheppach, 2009; Dalfino, 2008; and Busani *et. al.*, 2006). IAH was also found to be most commonly encounted in post emergency surgery patients compared to subspecialized surgeries (Cheatham, 2009).

IAH is associated with increased mortality and morbidity rate but in the current practice there is no routine measurement of intraabdominal pressure (IAP) to detect IAH or ACS (Sugrue and Buhkari, 2009; Cheatham, 2009; Mayer et. al.,2010).

The clinical sensitivity of detecting ACS is less than 50% which makes traditional methods of surgeons diagnosing ACS less accurate (Cheatham, 2009; Luckianow et. al., 2012; Mayer et. al.,2010).

Objective assessment of IAH is not utilized by most of the ICU staff and surgical team and due to lack of awareness of the complications of untreated intra abdominal hypertension. These complications can be abdominal compartment syndrome, multisystem organ failure, and death (Sugrue and Buhkari, 2009; Cheatham, 2009).

1.2 JUSTIFICATION/RATIONALE OF THE STUDY

Post laparotomy patients have a higher risk for developing intra abdominal hypertension leading to abdominal compartment syndrome due to the intra abdominal pathology and thus have a higher mortality rate. Proper management of intra abdominal hypertension and abdominal compartment syndrome can reduce mortality rate if it is diagnosed but in practice it is not routinely done.

The rationale of this study is to determine whether the incidence and mortality rate is significant enough to recommend routine measurement of intra abdominal pressure in all patients admitted to ICU post emergency laparotomy in HTAA. Clinical diagnosis of abdominal compartment syndrome which is the current practice has a sensitivity of less than 50% and therefore a more objective measurement is needed to diagnose the condition as recommended by the World Society of Abdominal Compartment Syndrome (WSACS).

Measuring intra abdominal pressure by intravesicular technique does not add any form of investigative invasiveness to the patient as all patients in ICU are always on a Foley's catheter to measure the urine output. Repeated measurement and

instillation of 25mls of normal saline for every measurement has been proven not to cause urinary tract infection.

1.3 OBJECTIVES AND RESEARCH QUESTIONS

1.3.1 Primary objective

- To describe the prevalence of IAH and its association with ICU stay mortality rate in all post emergency laparotomy general surgical patients admitted to ICU in HTAA,Kuantan.

1.3.2 Secondary objectives

- To measure the prevalence of IAH and its complications (length of ICU stay, mechanical ventilation, renal replacement therapy, demographics) in all post emergency laparotomy general surgical patients admitted to ICU in HTAA,Kuantan.
- To describe the prevalence of Primary ACS and its association with the prevalence of IAH, ICU stay mortality rate and its complications (length of ICU stay, mechanical ventilation, renal replacement therapy, demographics) in all post emergency laparotomy general surgical patients admitted to ICU in HTAA,Kuantan.

1.3.3 Research questions

- What is the incidence of IAH and its association with mortality in all post emergency laparotomy surgical patients admitted to ICU in HTAA,Kuantan?

CHAPTER TWO

LITERATURE REVIEW

2.1 DEFINITION OF INTRAABDOMINAL HYPERTENSION

The abdomen can be compared to a box with fixed walls (pelvis, spine and costal arch) and flexible walls (diaphragm and abdominal wall). The pressure within the abdomen at any given time is determined by the nature of its contents and the elasticity of the walls. In accordance with Pascal's law, the intra abdominal pressure measured at a point is assumed to represent the intra abdominal pressure throughout the abdomen because the abdomen and its contents can be considered as relatively non-compressive and primarily fluid in character. (Surgue and Buhkari, 2009; Meyer et. a., 2010; Malbrain et. al., 2005).

Intra abdominal pressure is thus defined as a steady-state pressure concealed within the abdominal cavity. Inspiration increases intra abdominal pressure due to contraction of diaphragm and expiration decreases it due to relaxation of diaphragm. Conditions that decrease the elasticity of the abdominal wall eg. 4^{th} degree burns, space occupying lesions eg. gravid uterus or tumors, ascites, hemoperitoneum, pneumoperitoneum, increase in volume of hollow viscera (bowel edema) or solid organs (hematoma) will also increase the IAP.

In pathological conditions, intra abdominal pressure will be raised due to the increased volume of hollow viscera (eg bowel edema), increased intra abdominal contents (ascites, blood or collection) and decreased abdominal wall compliance (post laparotomy) (Cheatham, 2009; Meyer et. a., 2010; Luckianow et. al., 2012; Malbrain et. al., 2005; Scheppach, 2009).

Intra abdominal pressure is around 0-5 mmHg in healthy adults and 5-7 mmHg in critically ill patients. Intra abdominal hypertension (IAH) is defined by repeated or sustained pathological increase of the pressure of intra abdominal by more than 12 mmHg in at least 3 readings within 4 to 6 hours as recommended by the World Society for Abdominal Compartment Syndroms (WSACS). It can be graded into four grades; grade I (IAP of 12–15 mmHg), grade II (IAP 16–20 mmHg), grade III (IAP 21–25 mmHg) and grade IV(IAP >25 mmHg) (Malbrain et. al., 2005).

2.2 PATHOPHYSIOLOGY OF INTRAABDOMINAL HYPERTENSION

The cardiovascular venous return is significantly reduced by the increased intrathoracic pressure through the cephalic deviation of the diaphragm, resulting in reduced cardiac output (CO) at an IAP of only 10 mmHg. Hypovolemia may lower the threshold for organ dysfunction meanwhile fluid resuscitation shows a protective effect in hypervolaemic patients by increasing venous return in non-severe elevations in IAP. Increased intrathoracic pressure may cause direct cardiac, (systemic and pulmonary) vascular and pulmonary compression, increasing the afterload and further reducing CO. Patients with hypovolaemia or low cardiac contractility are affected in particular and may profit from preload augmentation through volume administration. Cardiac preload is reduced because venous return is compromised from the lower extremities due to IAH. The pressure in the femoral veins are increased markedly and result in the formation of peripheral limb edema and increasing the risk of developing deep vein thrombosis in patients with IAH. Clinically it manifests as inability to maintain a mean arterial pressure of >65 mmHg and increased inotropic requirements (Cheatham, 2009; Malbrain et. al., 2005; Malbrain, 2013).

Pulmonary compression of the pulmonary parenchyma through direct IAP transmission and cephalad deviation of the diaphragm (splinting of diaphragm) will lead to pulmonary dysfunction at an IAP of 16–30 mmHg. Patients with shock or hypotension are particularly affected. The pulmonary capillary membrane oxygen transport decreases and the intrapulmonary shunt fraction increases. Increased alveolar dead space and carbon dioxide retention occurs due to parenchymal compression causing decreased pulmonary capillary blood flow. Mean airway pressures and peak inspiratory pressures are increased markedly potentially leading to alveolar barotrauma. Ventilation and perfusion mismatch are further aggravated by dampened dynamic pulmonary compliance and spontaneous tidal volumes. Arterial hypoxemia and hypercarbia will eventually ensue due to a combination of these effects. Clinically it manifests as increased ventilation settings (Malbrain et. al., 2005; Malbrain, 2013; Santa-Teresa et. al., 2012; Fietsam et. al., 1989).

IAH reduces renal arterial and venous blood flow and parenchymal function resulting in oliguria when the IAP is 15 mmHg and anuria at 30 mmHg. Renal dysfunction develops more of the compression of the renal vein rather than renal artery. However the renal parenchymal cortex becomes ischaemic with decrease in glomerular and tubular function as the blood flow is shunted away from high renal vascular resistance and renal vein pressure. Reduced cardiac output further aggravates the renal dysfunction. The decreased glomerular filtration rate eventually results in a decreased renal clearance of various components such as serum creatinine and urea nitrogen in the blood. Antidiuretic hormone, aldosterone and plasma renin activity levels are increased significantly. Clinically it manifests as acute kidney injury with

higher need for renal replacement therapy (Luckianow, 2012; Malbrain, 2013; Santa-Teresa et. al., 2012; Schaeffer et. al.,1990).

The gastrointestinal organs the most sensitive of all the organ systems to any increase in IAP is the bowel. Mesenteric blood flow will be impaired with an IAP as low as 10 mmHg. At an IAP of 40 mmHg, celiac artery blood flow is only at 57% capacity and superior mesenteric artery blood flow is worse functioning at 31% capacity only. Hypovolemia or hemorrhage aggravates the already impaired mesenteric perfusion. Additionally, intestinal edema worsens IAP and malperfusion due to venous hypertension which is fueled by compression of the thin walled mesenteric veins from high IAP. Mucosal perfusion of the intestine is diminished when IAP levels reached 20 mmHg maybe resulting in the multi organ failure or dysfunction from sepsis as a consequence of persistent bacterial translocation contributed mainly by mucosal barrier atrophy. Clinically it manifests as intraabdominal sepsis (Cheatham, 2009; Mayer et.al., 2010; Dalfino, 2008; Malbrain, 2013; Kirkpatrick et. al., 2013; Santa-Teresa et. al., 2012; Fietsam et. al., 1989; Schaeffer et. al.,1990).

Hepatic IAH reduces blood flow to the portal vein, all of the hepatic veins and hepatic arteries in several ways. Decreased cardiac output directly affects hepatic artery flow. The diaphragm is passed by the hepatic veins with subsequent anatomical narrowing and liver compression leads to decreased hepatic and portal venous flow. Energy substrates productivity and function of hepatic mitochondria as well as lactic acid clearance are decreased by the impaired hepatic microcirculatory blood flow. Organ dysfunction occurs at IAP levels approximately 10 mmHg even when the mean arterial pressure and cardiac output is normal. Clinically it manifests as acute liver

dysfunction or failure (Cheatham, 2009; Unit, 2006; Fietsam et. al., 1989; Schaeffer et. al.,1990).

Central nervous system IAH directly affects cerebral perfusion and function, especially when intracranial pressure (ICP) increases acutely (e.g., in traumatic injury and acute illness). ICP may also be directly affected by elevations in intraabdominal and intrathoracic pressure and cerebral perfusion pressure as a consequent reduces.Decreased cerebral venous outflow and increased cerebral blood flow from increased PaCO2 and cerebrospinal fluid pressure due to decreased lumbar venous plexus blood flow are proposed mechanisms of disease.

Cerebral venous blood flow is decreased because of impaired venous return from the cranium from high intrathoracic pressure leads to intracerebral venous pooling. This pre-existing cerebral perfusion abnormalities can be further worsen during chronic intracranial hypertension, trauma or other causes of decreased cerebral compliance. Hypovolaemia may further aggravate already marginal cerebral perfusion. Clinically it manifests as poor Glasgow Coma Score (GCS) recovery (Cheatham, 2009; Unit, 2006; Malbrain, 2013; Santa-Teresa et. al., 2012).

Abdominal wall IAH significantly impinges on the circulatory system of the abdominal wall. Rectus sheath perfusion functions at 58% of baseline when the IAP is only 10 mmHg and at 40 mmHg it becomes 20% of baseline potentially explaining the vulnerability to the development of necrotizing fasciitis, high rate of full thickness wound dehiscence and overall impaired wound healing. Clinically it manifests as surgical site infection or burst abdomen (Cheatham; 2009; Malbrain et. al., 2005; Scheppach, 2009; Busani et. al., 2006).

It must be emphasized that before significant organ failure or dysfunction occurs, rapid recognition of IAH and treatment can potentially reverse all of these

pathophysiologic changes in the patient. (Cheatham, 2009; Luckianow et.al., 2012; Scheppach, 2009).

2.3 CLASSIFICATION OF INTRAABDOMINAL HYPERTENSION

Intra abdominal hypertension (IAH) can be classified as follow:-

1) Hyperacute – occurs in physical activity, sneezing, coughing, straining or laughing that last for a few seconds or minutes due to short bursts in elevations in IAP.

2) Acute – occurs in intra-abdominal hemorrhage, post laparotomy, peritonitis or trauma and developes over a period of hours and is seen primarily in general surgical patients. It is the pathology studied in this research.

3) Subacute- occurs for medical patients and is over a period of days.

4) Chronic- Seen in pregnancy, cirrhosis, chronic ascites, peritoneal dialysis, intra abdominal space occupying lesion or morbid obesity and develops over a period of months to years.

High intra abdominal pressure causes impairment of perfusion to the tissues which leads to organ dysfunction mainly involving the renal, cardiovascular, respiratory, gastrointestinal and neurological system (Sugrue and Buhkari, 2009; Mayer et. al., 2010; Dalfino, 2008; Malbrain, 2013). It starts at the cellular level where it impairs the function of mRNA, intracellular enzymes and nitrous oxidation (Sugrue and Buhkari, 2009; Cheatham, 2009; Mayer et. al., 2010; Dalfino, 2008; Unit, 2006; Malbrain, 2013). As IAP increases furthermore, it exerts its effect on the organs with impaired local perfusion, high vascular organ resistance and reduction in visceral organ blood flow. And if further increase in pressure, it becomes systemic where there will be increased afterload and reduced preload (Sugrue and Buhkari, 2009; Mayer et. al., 2010; Dalfino, 2008; Unit, 2006; Malbrain, 2013).

2.4 ABDOMINAL COMPARTMENT SYNDROME

Critical intra abdominal pressure that causes impairment of perfusion to the tissues occurs when the pressure exceeds 12 mmHg. Abdominal compartment syndrome (ACS) happens initially from reductions to the microcirculation of the blood vessels at this pressure. If intra abdominal hypertension is not diagnosed properly resulting in progression of end organ dysfunction or failure then it is the hallmark of abdominal compartment syndrome. Classically, abdominal compartment syndrome is defined by the triad: (a) pathologic state caused by an acute increase in intra abdominal pressure >20 mmHg, (b) presence of adverse effects on end-organ function, and (c) abdominal decompression has beneficial effects (Mayer et. al., 2010; Malbrain et. al., 2005).

Abdominal compartment syndrome (ACS) can also be classified as primary, secondary, or recurrent, based on its duration and causative mechanism as follows :- Primary ACS which is also called abdominal or surgical ACS is defined by the presence of acute or subacute IAH secondary to an intra abdominal etiology (post abdominal surgery or trauma). It is the pathology studied in this research. Secondary ACS which is also called extra-abdominal or medical ACS is defined by the presence of subacute or chronic intra abdominal hypertension as a result from pathological states requiring massive crystalloid or colloid resuscitation, such as major burns or septic shock. Recurrent ACS which is also called tertiary ACS denotes the recurrence of ACS following remission of a similar episode of ACS. The most important thing for primary prophylaxis of ACS is to recognize the etiology of IAH that will ultimately progress to ACS (Luckianow et. al., 2012). The most prominent contributor towards ACS are hemorrhage, intestinal obstruction, intra abdominal sepsis and fluid overload although 45% are usually multifactorial (Sugrue and Buhkari, 2009; Unit, 2006). Tissue edema can be minimize by reducing administration

of fluids if intraabdominal sepsis is diagnosed early and treated promptly (Sugrue and Buhkari, 2009; Unit, 2006). Excellent surgical technique with correction of bleeding tendencies or coagulopathy helps reduce post operative hemorrhage and hematoma (Sugrue and Buhkari, 2009; Unit, 2006). Prophylactic abdominal decompression is a common practice among trauma surgeons but in general surgery it has not gained as a popular entity (Sugrue and Buhkari, 2009; Unit, 2006). Abdominal compartment syndrome has a mortality of 100% without decompression and 50% with decompression. The most commonly used technique for decompression is laparostomy by application of a bogota bag (Cheatham, 2009; Malbrain, 2013; Kirkpatrick et. al., 2013).

2.5 TECHNIQUES OF MEASURING INTRA ABDOMINAL PRESSURE

The diagnosis of abdominal compartment syndrome (ACS) by serial clinical examination has been proven to be mostly unreliable due to poor sensitivity with a positive predictive value of less than 50% as mentioned earlier, confirming that it is an inaccurate diagnostic tool. Measurement of abdominal girth is also unreliable (Sugrue and Buhkari, 2009; CHeatham, 2009; Scheppach, 2009).

Radiological investigations such as chest X-ray or abdominal X-ray, ultrasound of the abdomen or computer tomography (CT) are not sensitive to detect the existence of increased intra abdominal pressure. However, they are frequently used to demonstrate the cause of intra abdominal hypertension (abscess, ascites, hematoma, collection or bleeding) and helps in determining further management (drainage of collections or paracentesis) (Cheatham, 2009; Malbrain et. al., 2005; Busani et. al., 2006).

Therefore to diagnose IAH/ACS correctly, an objectively accurate and frequent measurement of intra abdominal pressure is required. IAP monitoring helps guide the resuscitation during ACS as well as serve as an accurate, safe and cost-effective tool for the diagnosing the presence of IAH (Mayer et. al., 2010; Scheppach, 2009; Unit, 2006).

Due to the significant mortality and morbidity of IAH/ACS and the good risk-benefit profile of measurement of IAP, any patients with 2 or more risk factors for IAH should be measured at least once a day as a baseline for the intra abdominal pressure (Grade 1B) and if IAH is confirmed, the measurement of IAP should be done 4 to 6 hourly until the patient has recovered from the critical illness (Grade 1C) (Mayer et. al., 2010; Dalfino, 2008; Busani et. al., 2006; Malbrain, 2013; Kirkpatrick et. al., 2013).

All post emergency laparotomy patients have two risk factors, one is the laparotomy itself and the other one is the abdominal pathology that necessitates the emergency surgery. Given the presence of the two risk factors, it is recommended that all post emergency laparotomy patients should have their intra abdominal pressure routinely assessed objectively to look for abdominal compartment syndrome or intra abdominal hypertension (Luckianow et. al., 2013; Dalfino, 2008; Santa-Teresa et. al., 2012; .Fietsam et. al., 1989).

The methods for IAP assessment can be direct or indirect, continuous or intermittent. Direct assessment are not usually done but can be accomplished by placement of an intraperitoneal catheter for peritoneal dialysis or ascites drainage, placement of a transducer in the peritoneum and when performing laparoscopic surgery. It is an invasive procedure and adds to the critically ill patient more stress (Cheatham, 2009; Luckianow et. al., 2012; Unit, 2006).

Indirect methods for measurement of IAP includes intravesicular, inferior vena cava, uterine, rectal, gastric and also during airway pressure measurements. Because it is relatively cheap and technically easier to perform, the gold standard for IAP measurement is currently by the intravesicular route. Due to the anatomy and physiology of the bladder which can act as a passive reservoir, it can act as a transducer of IAP when a small amount of normal saline is infused owing to the excellent bladder wall compliance. This translates any change in intravesicular pressure as a change in IAP. The World Society for Abdominal Compartment Syndroms (WSACS) recommends that the specific technique in measuring IAP includes an instillation volume of 25 ml of normal saline, zeroing of the transducer at the level of mid-axillary line and taken at end-expiration in supine position (Sugrue and Buhkari, 2009; Luckianow et. al., 2012; Kirkpatrick et. al., 2013; Santa-Teresa et. al., 2012; .Fietsam et. al., 1989). In the ICU setting, intravesicular measurement is the most feasible as all patients admitted to ICU will have a Foley's catheter inserted to the bladder for hourly urine output measurement. So a probe needs only to be inserted at a port on the Foley's catheter minimizing the invasiveness. Traditionally the measurement of IAP is done intermittently using a manometer but the advent of the manometry permits continuous measurement of IAP (Cheatham, 2009; Mayer et. al., 2010; Luckianow et al., 2012; Malbrain et. al., 2005; Dalfino, 2008; Kirkpatrick et. al., 2013; Malbrain 2013).

Not all patients are suitable for measurement of bladder pressure. The patients with neurogenic bladder, bladder surgery, pelvic fracture or gross hematuria are contraindicated and IAP has to be obtained by other methods (Cheatham, 2009; Mayer et. al., 2010; Malbrain et. al., 2005; Santa-Teresa et. al., 2012).

This leads to the development of the nasogastric technique to monitor IAP. This technique permits continuous measurement easily as well as avoiding the complications of hydrostatic fluid column creation in the bladder offered as an advantage. However, not all ICU patients have a nasogastric tube inserted and it is contraindicated in patients with paralytic ileus which is common in post laparotomy patients (Mayer et al., 2010; Unit, 2006; Kirkpatrick Et. al., 2013).

Measurement through rectum, uterine and inferior vena cava is invasive and not feasible. Airway pressure measurements is not as accurate as other methods and is contraindicated if there is lung pathology or infection which is common in all ICU patients (Cheatham, 2009; Malbrain et. al., 2005; Santa-Teresa et. al., 2012; Schaeffer et. al., 1990).

CHAPTER THREE

METHODOLOGY

3.1 STUDY DESIGN AND SAMPLING

3.1.1 Study Design

Prospective study

3.1.2 Study Period

June 2014 till June 2015

3.1.3 Study Location

Intensive Care Unit, Hospital Tengku Ampuan Afzan (HTAA), Kuantan

3.1.4 Reference Population

All post emergency laparotomy general surgical patients in HTAA, Kuantan

3.1.5 Study Population

All post emergency laparotomy general surgical patients in ICU HTAA, Kuantan.

3.1.6 Inclusion Criteria

- Age more than 12 years old on day of admission to ICU in HTAA,Kuantan.
- Admitted to ICU in HTAA, Kuantan
- Admitted to ICU in HTAA,Kuantan due to Post emergency laparotomy general surgery
- Patient was admitted to the ICU in HTAA,Kuantan within the same hospital admission