Abdominal Compartment Syndrome
Early Diagnosis and Management

An Essay

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in General Surgery

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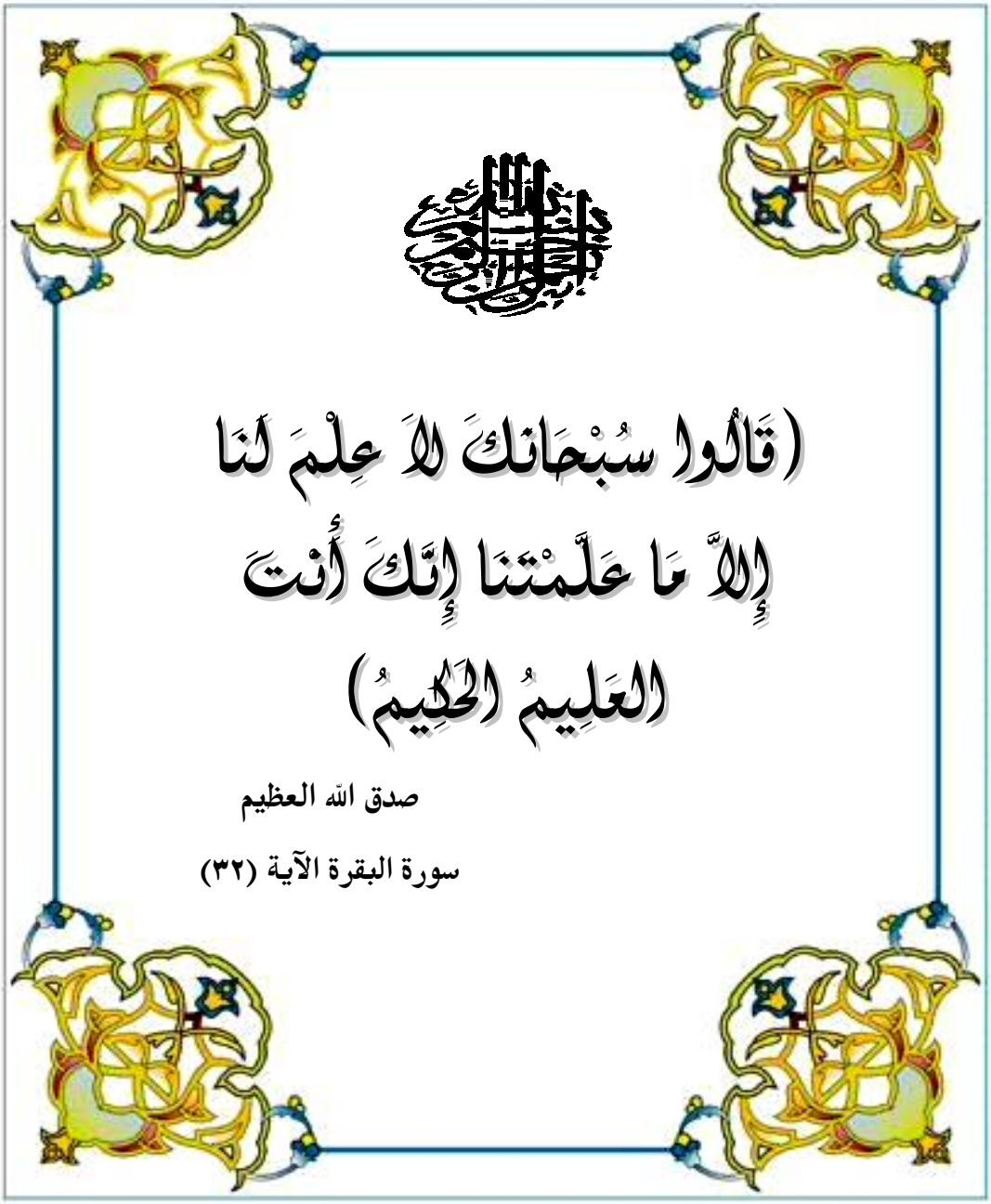

بسم الله الرحمن الرحيم

(قَالُوا سُبْحَانَكَ لاَ عِلْمَ لَنَا
إِلاَّ مَا عَلَّمْتَنَا إِنَّكَ أَنْتَ
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List of Abbreviations

Abbreviations	Meaning
ACS	Abdominal Compartment Syndrome
AKI	Acute Kidney Injury
APP	Abdominal Perfusion Pressure
ARDS	Acute Respiratory Distress Syndrome
ATP	Adenosine Tri Phosphate
COP	Cardiac Output
CPP	Cerebral Perfusion Pressure
CSF	Cerebro-Spinal Fluid
CT	Computed Tomography
CVP	Central Venus Pressure
DL	Decompressive Laparotomy
FG	Filtration Gradient
GFP	Glomeruler Filtration Pressure
IAH	Intra Abdominal Hypertension
IAP	Intra Abdominal Pressure
ICP	Intra Cranial Pressure
ICU	Intensive Care Unit
IV	Intra Venous \ Inter Vertebral
IVC	Inferior Vena Cava
MAP	Mean Arterial blood Pressure
MCFP	Mean Circulatory Filling Pressure

Abbreviations	Meaning
NPWT	Negative Pressure Wound Therapy
PAOP	Pulmonary Artery Occlusion Pressure
PTFE	Poly Tetra Fluoro Ehhylene
PTP	Proximal Tubular Pressure
SIRs	Systemic Inflammatory Response
SLAF	Sub cutaneous Linea Alba Fasciotomy
SVV	. Stroke Volume Variation
VAC	Vacuum Assisted Closure
WSACS	World Society of the Abdominal Compartment Syndrome

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INTRODUCTION

Intra-abdominal pressure (IAP) is the steady-state pressure concealed within the abdominal cavity. The values of (IAP) range from sub atmospheric to (0 mm Hg) in normal individuals, (5 7mmHg) in critically ill adults **(Malbrain et al., 2010).**

(IAP) increases with inspiration and decreases with expiration due to diaphragmatic contraction and relaxation, respectively. The (IAP) also varies with body position [higher in vertical compared with horizontal, higher in prone than in supine] and with the contraction of abdominal musculature **(Sanchez et al., 2011).**

Abdominal Compartment Syndrome (ACS) represents the natural progression of end –organ dysfunction caused by increased (IAP), and develops if Intra Abdominal Hypertension (IAH) is not recognized and treated appropriately **(Malbrain et al., 2010).**

Risk factors for (IAH /ACS) may be surgical or medical. Surgical may be postoperative [Hemorrhage, edema following Extensive dissections, reduction of diaphragmatic hernia, abdominal surgery especially with tight fascial closure, ileus, peritonitis or intra abdominal abscess] or post-traumatic [multiple trauma, burns, intra- or retroperitoneal bleeding].

Medical risk factors may be edema, ascites secondary to massive fluid resuscitation (e.g septic shock), acute pancreatitis, intra abdominal or retro peritoneal tumour **(Cheatham et al., 2009).**

Increased (IAP) has deleterious effects on end-organ function and may compromise respiratory, cardiovascular, renal, gastrointestinal, hepatic and central nervous system functions **(Shear & Ronsner, 2011).**

Because physical exam has a low sensitivity for the detection of increased (IAP), the diagnosis of (IAH /ACS) depends on the accurate & frequent measurement of (IAP) at risk patients **(Sugrue et al., 2010).**

Direct measurement of (IAP) can be obtained by an intra peritoneal catheter installed for ascites drainage or peritoneal dialysis, an intra peritoneal pressure transducer and during laparoscopic surgery. Indirect method for measuring (IAP) include intra vesical, gastric, rectal, uterine, inferior vena cava and airway pressure measurements **(Banieghbal et al., 2009).**

Because of its simplicity and low coast,(IAP) measurement by the intra vesical route has been considered as the gold standard **(Cheatham & Malbrain, 2010).**

The most effective way of preventing (ACS) is by early recognition of patients at risk and timely intervention to

minimize the possibilities for the development of (IAH). In this context, a high index of suspicion and close monitoring of (IAP) are of paramount importance **(Hunter & Damani, 2010).**

Appropriate management of (IAH/ACS) is based upon four general principles:-

1. Serial monitoring of (IAP).
2. Optimization of systemic perfusion and organ function.
3. Institution of specific medical interventions to reduce (IAP) and end organ consequences of (IAH /ACS).
4. Prompt surgical decompression for refractory (IAH) **(Kirkpatrick et al., 2012).**

Surgical decompression of the abdomen has long been the standard treatment of (IAH/ACS) and can be life saving when a patient organ dysfunction, failure, or both are refractory to medical treatment **(Chen et al., 2010).**

Delayed abdominal decompression and disregard of high (IAP) levels are associated with significant increases in patient mortalilty **(Cheatham & Malbrain, 2010).**

Prophylactic decompression and creation of temporary abdominal closure in surgical patients at risk for (IAH/ACS) significantly reduces the subsequent development of (IAH/ACS) and improves survival. Prognosis of (ACS) is

associated with 80% – 100% mortality if unrecognized and untreated. Most deaths from (ACS) are attributed to sepsis & multiple organ failure **(Cheatham, 2009).**

HISTORY

Although initially recognized over 150 years ago, the pathophysiologic implications of elevated intra-abdominal pressure (IAP) have essentially been rediscovered only within the past two decades **(Ivatury et al., 2006).**

The impact of elevated (IAP) upon respiratory function was first documented in 1863 and subsequently in 1870. It was identified in an animal model that an (IAP) between 27 and 46 cm H2O significantly elevated intra thoracic pressure, respiratory failure and death. The theory that respiratory failure is the cause of death in severe Intra Abdominal Hypertension (IAH) persisted until 1911 when it was demonstrated in cat, dog, and rabbit models that elevated (IAP) causes death by cardiovascular collapse rather than by respiratory failure **(Coombs, 1922).**

Overholt (1931) extensively studied the properties of the abdominal wall and confirmed that normal (IAP) is sub atmospheric and that procedures which restrict movement of the abdominal wall or distention of the stomach or colon all result in an increase in (IAP). Overholt postulated that (IAP) is governed by both the pressure induced, by the abdominal content and the "flexibility" (compliance) of the abdominal wall **(Overholt, 1931).**

Investigation into the physiologic effects of (IAP) on renal function in humans essentially began in 1947 with the work of **(Bradley &Bradley , 1947).**

The experiences of surgeons treating infants with gastroschisis or omphalocele further contributed to the understanding of both the concept of "loss of abdominal domain" as well as the life threatening cardiac, pulmonary and gastrointestinal complications which can occur when abdomens are primarily closed without consideration of elevated (IAP) **(Schwartz et al., 1983).**

Gross (1948), first described the use of a "staged abdominal repair" in the management of such infants unknowingly pioneering the open abdomen techniques which have now become standard in the treatment of (IAH) and Abdominal Compartment Syndrome (ACS) **(Gross, 1948).**

Although surrogate measurement of (IAP) via measurement of intra vesicular, intra gastric and intra colonic pressure in animal models was common place in the 1920's and 1930's, it was done by (Soderberg & Westin, 1970), who first described the strong correlation between (IAP) and intra vesicular pressure during laparoscopy in humans **(Soderberg & Westin, 1970),**

The introduction of laparoscopic techniques into mainstream surgical practice in the late 1980's and early 1990's