INTRODUCTION

Intra-abdominal hypertension (IAH) is a frequent occurrence in critically ill patients [1] with studies have demonstrated that it is common in these settings [2]

IAH, especially abdominal compartment syndrome (ACS), can affect organ function [3] and are one of the major causes of organ failure [4] , a syndrome which has plagued intensive care unit (ICU) patients and physicians for decades . [5]

The pathophysiology of IAH and ACS is based on the chain reaction of physiological processes generated by the increased abdominal pressure which affects almost every organ and it could be fatal without correct diagnosis and treatment. [4] as it adds to organ dysfunction in a dose-dependent manner [6], both intra-abdominal and remote organs are involved. [7]

There appears to be a close link between IAH and severe sepsis and septic shock, with fluid resuscitation as one of the major contributors to elevated Intra-abdominal pressure (IAP) [6].

After initial filling to reverse distributive shock, emphasis shifts to limitation and elimination of interstitial edema in vital organs. Indeed, a positive fluid balance resulting from third spacing is independently associated with impaired organ function and worse outcome [8] while fluid resuscitation is an important aspect of the management of septic shock patients, more attention to the problem of IAH in this setting is urgently needed.[6]

Because of the nature of the illness and injury associated with IAH or abdominal compartment syndrome (ACS), these patients retain large volumes of sodium and water, and due to capillary leak, this will exacerbate tissue oedema and third spacing triggering a vicious cycle of ongoing IAH. [9]

Increased attention to intra-abdominal pressure (IAP), along with changes in the clinical management of critically ill or injured patients, have led to an exponential growth in research relating to intra-abdominal hypertension (IAH) and abdominal compartment syndrome (ACS) in recent years [10], while appropriate and prompt management could improve survival. [3]

The significant prognostic value of elevated intra-abdominal pressure has prompted many intensive care units to adopt measurement of this physiologic parameter as a routine vital sign in patients at risk. [5], it can be measured easily and reliably in patients through the bladder using simple tools. [11]

Abdominal perfusion pressure (APP) is a novel, clinically measurable parameter that has been introduced to explain the circulatory compromise in the abdominal cavity in the presence of IAH/ ACS. [12], for IAH has been identified as a continuum of pathophysiologic changes beginning with regional blood flow disturbances and culminating in frank end-organ failure and the development of ACS [5],

APP, similar to the familiar concept of cerebral perfusion pressure, is defined as the difference between the mean arterial pressure (MAP) and the IAP, and implies that as the IAP rises, the perfusion of organs or vessels in or near the abdomen falls even in the absence of a drop in MAP. [12]

The effects of increased IAP are multiple, but the kidney is especially vulnerable to increased IAP because of its anatomic position [11], and it is the most consistently described organ dysfunctions with oliguria being one of the earliest clinical signs of IAH [13].

IAH should be considered as a potential cause of acute kidney injury in critically ill patients; [11] and can be present at relatively low levels of intra-abdominal pressure (IAP). [12]

Although the means by which kidney function is impaired in patients with ACS is incompletely elucidated, available evidence suggests that the most important factor involves alterations in renal blood flow. [11]

Acute kidney injury (AKI) is an increasingly common clinical problem faced by nephrologists and intensivists, as well as general physicians and surgeons. [14]

Critical care nephrology has recently gained an increased interest since clinicians operating in the field of critical illness realized that the dysfunction of the renal system in their patients was always associated as a victim or as a potential culprit with worse outcomes [15].

Until recently, there was a lack of consensus as to the best definition, characterization, and evaluation of acute renal failure. [16] which has been a major factor hampering clinical research and comparison of trial data. [17]

A need for clear definitions of renal injury and renal failure has led to the request for measurable criteria [18]

RIFLE is the first widely accepted AKI definition, validated in over half a million patients worldwide [19]

The Risk, Injury, Failure, Loss, and End-Stage Renal Disease (RIFLE) is a consensus-based classification system for diagnosing acute kidney insufficiency (AKI), based on serum creatinine and urine output criteria [19].

Such an acronym conventionally described 3 stages of progressively higher creatinine levels or decreased urine output flows (risk, injury, and failure) and 2 outcomes (loss of function and end stage kidney disease) [15].

This study is for addressing the link between the APP and the function of the kidney assessed by RIFLE criteria.