Chapter 1

Intra-Abdominal Hypertension and Abdominal Compartment Syndrome in Liver Diseases

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Abstract

Intra-abdominal hypertension (IAH) is defined as an intra-abdominal pressure (IAP) above 12 mmHg. Abdominal compartment syndrome (ACS) is defined as an IAP above 20 mmHg with evidence of organ failure. Moreover, IAH/ACS is a condition that can cause acute renal failure, respiratory failure, circulatory disease, gastrointestinal dysfunction, and liver failure due to elevated IAP. The incidence of IAH/ACS increases in the more critically ill patient and is associated with significantly increased morbidity and mortality. Ascites, blood, or tumors increase IAP. In liver cirrhosis, massive ascites is often encountered. Hence, preventing IAH/ACS conditions may improve outcomes of patients with liver disease.

Keywords: intra-abdominal hypertension (IAH), abdominal compartment syndrome (ACS), hepatorenal syndrome (HRS)

1. Introduction

The pressure within the abdominal cavity is normally a little higher than the atmospheric pressure to less than 0 mmHg. Certain physiological conditions such as morbid obesity and pregnancy may be associated with chronic IAP elevations. However, even small increases in intra-abdominal pressure can have adverse effects on renal function, cardiac output, hepatic blood flow, respiratory mechanics, splanchnic perfusion, and intracranial pressure. IAP is approximately 5–7 mmHg in critically ill adults.

Wendt et al. firstly described oliguria in the presence of elevated intra-abdominal pressure in 1876 [1]. In 1947, Bradley published a seminal study of the renal effects of elevated IAP in humans [2]. Despite these early descriptions of the adverse effects of IAH, physicians are not careful about the significance of increased abdominal pressure.

Until recently, patients with ACS were not infrequently managed in the intensive care unit and typically presented with a tense distended abdomen, increased peak inspiratory airway pressure, severe hypercapnia, hypotension, and oliguria. Abdominal ascites occurs typically at the end stage of liver failure. Massive ascites also influences IAP and causes oliguria and acute kidney injury. Commonly, we recognize this symptom and confused it with hepatorenal syndrome (HRS). In such patients, we should take into account the elevation of renal parenchymal and renal vein pressures, as they are likely the mechanisms of renal impairment. Note that IAH/ACS and HRS are occurring simultaneously. Recently, Matsumoto et al. reported that renal vein dilation predicts poor outcome in patients with refractory cirrhotic ascites [3].

2. Pathophysiology of ACS

The pathology of IAH/ACS is perfusion imbalance in multiple organs: compression of the portal system in the abdominal cavity, compression of the inferior vena cava system in retroperitoneal organs, compression of the diaphragm in the intrathoracic organ, and perfusion dysfunction of the brain circulation through increase of intrathoracic pressure [4].

The perfusion imbalance in the upper body originated from the abdominal cavity, which causes circulation impairment and further increased intrathoracic cavity pressure and retroperitoneal cavity. This imbalance presents a functional disorder that substantially affects multiple organs.

ACS is similar to the compartment syndrome in muscular diseases. It is a circulatory disease caused by internal pressure of organs sectioned in a small wall of the compliance anatomically [5]. The normal IAP ranges from sub-atmospheric level to 0 mmHg. Certain physiological conditions, such as morbid obesity and pregnancy, may be associated with chronic IAP elevations. Moreover, IAH is defined as an IAP above 12 mmHg. ACS is defined as an IAP above 20 mmHg with evidence of organ failure [6]. IAP is the steady state of pressure concealed within the abdominal cavity. The normal IAP for critically ill patients are 5–7 mmHg range. Once, IAP have increased, patients become the state of IAH. IAH is recognized sustained IAP greater than to 12 mmHg. IAH may also be subclassified according to the duration of symptoms into one of the four groups. This fulminant example of IAH commonly leads to rapid development of ACS. With its development over a protracted time course, the abdominal wall adapts and progressively distends in response to increasing IAP, allowing time for the body to adapt physiologically. The clinical consideration of IAH subtypes is useful in prescribing patients at risk for ACS (**Table 1**) [6].

Primary ACS is characterized by the presence of acute or subacute IAH of relatively brief duration occurring as a result of an intra-abdominal cause such as severe acute pancreatitis, abdominal trauma, ruptured abdominal aortic aneurysm, and liver transplantation [7].

Secondary ACS is characterized by the presence of subacute or chronic IAH that develops massive fluid resuscitation such as an extra-abdominal cause such as sepsis, capillary leak, burns [8].

Classification of IAH		
Hyperacute IAH	Elevated IAP for seconds	Secondary to physical activity, coughing, laughing, sneezing, straining, or defecation
Acute IAH	Elevated IAP that develops over hours and can lead to rapid development of ACS	Secondary to trauma or intra-abdominal hemorrhage
Subacute IAH	Elevated IAP that develops over days and can also lead to ACS	Medical patients
Chronic IAH	Elevated IAP that develops over months or years.	Pregnancy, morbid obesity, intra-abdominal tumor, ascites

IAH, intra-abdominal hypertension; ACS, abdominal compartment syndrome.

Table 1. Classification of intra-abdominal hypertension.

Grading of IAH	
Grade I	IAP 12-15 mmHg
Grade II	IAP 16-20 mmHg
Grade III	IAP 21-21 mmHg
Grade IV	IAP > 25 mmHg

Table 2. Grading of intra-abdominal hypertension.

The World Society of Abdominal Compartment Syndrome classified IAH into grade I–IV and ACS (**Table 2**) [9].

Burch et al. suggested that most patients with grade III and all patients with grade IV should undergo abdominal decompression [10].

3. ACS results in non-abdominal organ failure

3.1. Cardiovascular

Due to the increased intrathoracic pressure, indirect measures of cardiac filling such as central venous pressure and pulmonary artery occlusion pressure give inaccurate results and can be increased despite profound intravascular volume depletion. The decrease in cardiac output caused by intra-abdominal hypertension is therefore exacerbated by hypovolemia [11].

3.2. Respiratory

Respiratory distress and failure: Initial signs of ACS include elevated peak airway pressures in intubated patients with decreased tidal volumes. The ensuing increase in intrathoracic pressure and hypoxic pulmonary vasoconstriction can lead to pulmonary hypertension [12].

3.3. Neurological

Intracranial perfusion pressure is decreased by increase in intracranial pressure (ICP) caused by venal perfusion defect, including renal failure. For increased ICP, decompressive laparotomy has been shown to reduce intractable elevated ICP in patients with IAH, and compression of the ureters is not thought to contribute to renal dysfunction, as the insertion of ureteric stents does not result in an improvement in urine output [13].

4. Intra-abdominal organ failure in ACS

4.1. Renal function disorder

ACS is characterized by marked reduction in glomerular filtration rate (GFR) and renal plasma flow in the absence of other causes of renal failure. Moreover, changes in cardiac output, direct compression of the renal vessels or renal parenchyma with diminished renal blood flow, increase in renal vascular resistance, and distribution of blood from the renal cortex to the medulla are reported the mechanisms of renal dysfunction [14]. Bradley et al. are the first to report that animals become anuric with an IAP of 30 mmHg [2]. Additional factors that cause IAP to reach ACS range include reduction in cardiac output and elevated levels of catecholamines [15]. Renin, angiotensin, and inflammatory cytokines may also come into play, further worsening renal function.

4.2. Liver function disorder

Diebel et al. reported that the portal vein (PV) pressure decreased experimentally in 65% of patients with an IAP of 40 mmHg, and liver tissue microcirculation quantity decreases to 71% [16].

Liver dysfunction occurs due to decrease PV flow because of IAH. Furthermore, with cardiac dysfunction, liver ischemia becomes worse. Persistent IAP decrease the mean arterial blood pressure in the superior mesenteric artery (SMA) and PV flow by 50% [17].

Rasmussen et al. reported that an IAP of 25 mm Hg results in a 66% decrease in PV blood flow and a 6.5-fold increase in portal/hepatic vascular resistance compared to baseline levels [18].

Furthermore, in studies evaluating the effects of increased IAP on hepatocyte, the characteristics of the sinusoid should be expected to elucidate hepatic dysfunction from increased IAP.

4.3. Gastrointestinal functional disorder

To determine the possibility of bacterial translocation (BT), of which there is failure of the mucous membrane barrier mechanism caused by decline in blood circulation in mucous membranes, pH in the mucous membrane declined as well. Besides, this phenomenon is regarded as the cause of multiple organ dysfunction syndrome (MODS) after ACS, but there is no direct proof. Even if the IAP is at 20 mmHg, blood flow to the intestinal mucosa decreases to 28%

experimentally in 61% of the baseline value of 40 mmHg [16]. In MODS, there is gastrointestinal mucous membrane acidosis of which the IAP is expected to be at 10 mmHg is derived from ACS.

5. Diagnosis of ACS

There are various methods of measuring intermittent IAP, such as invasive (direct, i.e., needle puncture of the abdomen during peritoneal dialysis or laparoscopy) and noninvasive (indirect, i.e., transduction of intravesicular or "bladder," gastric, colonic or uterine pressure via the balloon catheter). Noninvasive measurement of bladder internal pressure and intragastric

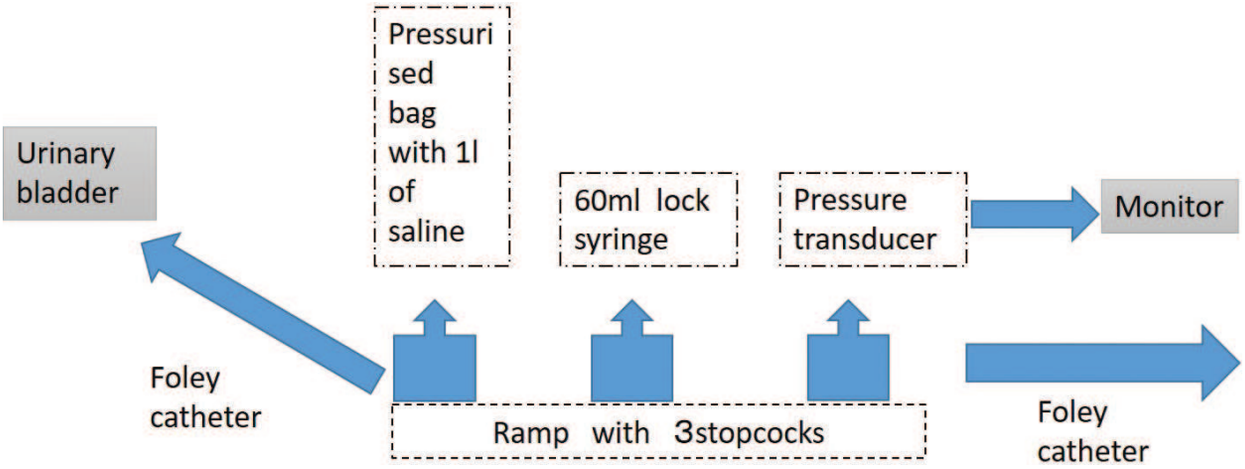

Figure 1. Measurement of intra-abdominal pressure using bladder pressure measurements.

pressure are recommended. The internal bladder pressure are commonly related to IAP measured directly in the range of 5–70 mmHg [6].

Intrabladder pressure monitoring estimated for IAP can be obtained either via a closed transducer technique or the closed Foley Manometer technique, which seems safe and does not alter the risk of UTI in patients with critical illness [19] (**Figure 1**).

6. ACS treatment

Discussions on IAP to become the adaptation standard of decompression is divided, but more than 25 mmHg is assumed to be a tentative adaptation standard clinically. However, recently, reports on gastrointestinal disorder due to impairment of IAP in the lower abdominal cavity need to be considered. The World Society of the Abdominal Compartment Syndrome suggested a management algorithm for IAH/ACS [20].

An early indication of the open abdomen technique has been shown to reduce mortality [21]. Chen et al. reported that laparoscopy can be used as a safe alternative for ACS decompression [22].

The World Society of the Abdominal Compartment Syndrome has noted that correct fluid therapy and perfusion support during resuscitation form the cornerstone of medical management in patients with abdominal hypertension [23].

Pharmacologic therapy is less effective than drainage procedures. Agustí et al. reported that dobutamine restores intestinal mucosal blood flow in a porcine model of intra-abdominal hyperpressure [24].

If a patient experiences decompensation, ACS should be re-examined as a potential cause.

7. Possible involvement of IAH/ACS and HRS

We reported an autopsy case with HRS and ACS diagnosed with a clinical and histopathological consideration of liver and kidney diseases. Further clinical studies are needed to improve the management of renal failure in patients with acute liver failure and advanced liver cirrhosis (**Figures 2** and **3**) [25].

HRS was originally described in 1863 by Flint as an association between liver disease and oliguric renal failure in the absence of significant renal histological change [26]. HRS is recognized by intense intrarenal vasospasm caused by the imbalance between vasodilatory and vasoconstrictive mediators seen in end stage of liver disease [27].

Although the precise role of IAH in HRS remains incompletely understood, it can be argued that diminished glomerular perfusion due to venous congestion results in further decline of GFR.

Cade et al. reported significant increases in urine flow rate and creatinine clearance after reduction in IAP from 22 to 10 mmHg with paracentesis in patients with cirrhosis [28]. Moreover,

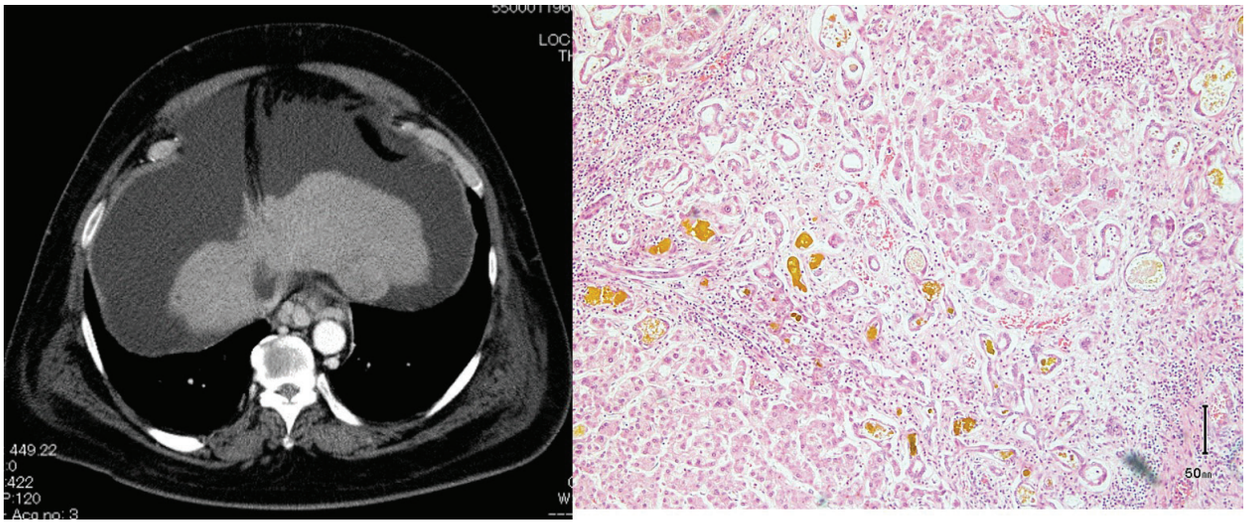

Figure 2. Liver: end-stage liver cirrhosis. Microscopic findings showed hepatic sinusoidal dilation due to portal hypertension and severe jaundice.

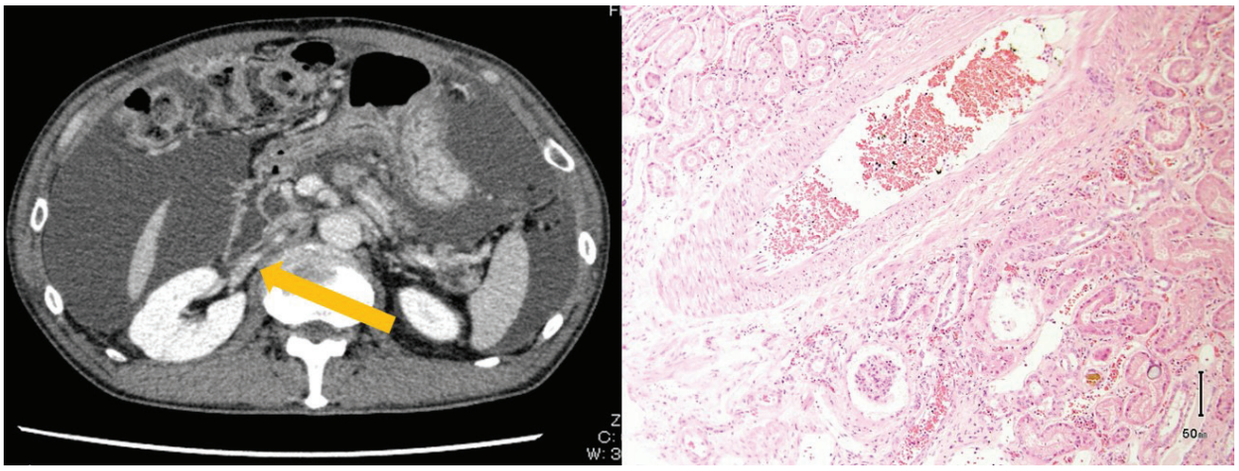

Figure 3. Kidney: microscopic findings showed swelling in the renal tubules. There was no change in the glomerulus and collecting tubule and no renal fibrosis. CT the right renal vein was compressed by massive ascites (arrow).

compression of renal vein is suggested to be vital in the development renal dysfunction. IAH is the significant pathological mechanism and independent risk factor in the occurrence and development of HRS [29]. Further, attempts should be made to decrease IAP following surgical decompression, large-volume paracentesis (LVP), and appropriate diuretic drug.

8. Drug strategy for ACS

Several methods are reported to control refractory abdominal ascites in end-stage liver cirrhosis, such as avoidance of non-steroidal anti-inflammatory drugs [30], dietary sodium restriction [31], diuretic, LVP, cell-free and concentrated ascites reinfusion therapy [32], transjugular intrahepatic portosystemic shunt [33], and peritoneovenous shunt [34].

IAH is defined as an IAP above 12 mmHg. Hence, abdominal ascites in early stage of liver cirrhosis should be treated and early stage of ascites in outpatient should be managed immediately. Outpatients with clinically apparent ascites will require diuretic therapy in addition to dietary sodium restriction. Diuretic therapy typically consists of treatment regimen for cirrhotic ascites such as combination of oral spironolactone and furosemide. Recently, aquaporin-2 is a vasopressin-regulated water channel expressed in the renal collecting duct. Urine aquaporin-2 is considered a marker of collecting duct responsiveness to tolvaptan. In Japan, on September 2013, tolvaptan was approved (in doses up to 7.5 mg/day) for treating patients with ascites who showed an inadequate response to conventional diuretics [35].

9. Conclusion

Massive ascites also influences IAP and causes oliguria and acute kidney injury (AKI). Commonly, we recognize this symptom at the stage of end stage of liver cirrhosis. This symptom has the possible involvement with HRS. In such patients, we should take into account the

elevation of renal parenchymal and renal vein pressures, as they are likely the mechanisms of renal impairment. Note that IAH/ACS and HRS are occurring simultaneously. Hepatologists should consider IAH and ACS in end-stage liver cirrhosis.

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