

# Study on the correlation between the changes in intra-abdominal pressure and renal functional in the patients with abdominal compartment syndrome

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**Abstract. – OBJECTIVE:** To observe the effect on renal function from increased intra-abdominal pressure.

**PATIENTS AND METHODS:** Sixty patients with abdominal compartment syndrome (ACS) were included in this study. Intra-abdominal pressure, mean arterial pressure (MAP) and central venous pressure (CVP) were recorded three times per day at a fixed time. Meanwhile, blood samples were collected for serum creatinine measurement and urine volume per hour was recorded.

**RESULTS:** The urine volume gradually decreased with the increasing intra-abdominal pressure, from  $92.6 \pm 20$  ml/h to  $27.9 \pm 20$  ml/h ( $p < 0.05$ ), and the serum creatinine increased from  $68.4 \pm 39.9$  mol/L to  $249.4 \pm 111.5$  mol/L ( $p < 0.05$ ). The CVP increased from  $0.98 \pm 0.19$  kPa to  $1.56 \pm 0.31$  kPa with the increase or decrease of the MAP. The increase in intra-abdominal pressure was negatively related to the urine volume ( $r = -0.193$ ,  $p < 0.05$ ), and positively related to the serum creatinine ( $r = 0.162$ ,  $p < 0.05$ ), but not related to the MAP.

**CONCLUSIONS:** The increase of intra-abdominal pressure, was closely related to oliguria and increasing serum creatinine. The use of fluid resuscitation and diuretics had few effects on the recovery of the renal function. When the intra-abdominal pressure had decreased, the urine volume increased, and the serum creatinine decreased.

*Key Words:*

Abdominal compartment syndrome, Renal function, Intra-abdominal pressure, Mean arterial pressure, Central venous pressure.

## Introduction

Abdominal compartment syndrome (ACS)<sup>1-4</sup> is a syndrome of cardiovascular, renal, liver, gastrointestinal and craniocerebral dysfunction caused by a certain level of intra-abdominal pressure (IAP) due to multiple reasons. When IAP increased for more than 1.6 Kpa, it can be diagnosed as intra-abdominal hypertension (IAH), IAH can be divided into four levels by IAP. Grade I IAP is 1.6-2.0 kPa. The grade II IAP is 2.1-2.7 kPa. Grade III IAP is 2.8-3.3 kPa. Grade IV IAP is more than 3.3 Kpa. When persistent intra-abdominal pressure is greater than 2.7 kPa and also accompanied by IAH related new organ dysfunction/failure, then it can be diagnosed as ACS. The change of IAP is positively related to the degree of the disease. The effects on the functions of various organs are exacerbated by the increasing IAP and the change of renal function is directly related to the change of IAP. Various lowering-IAP measurements are essential for the improvement of the renal function. We monitored the IAP and renal function dynamically for 60 ACS patients from July 2002 to July 2006. Good efficacy was achieved finally.

## Patients and Methods

### General Information

Sixty patients were included in this study, including 38 male and 18 female, with an age

range of 15-76 years and a mean age of 42.9 years. The primary diseases included 12 cases of traumatic shock with intraperitoneal hemorrhage, 10 cases of sepsis, 2 cases of extensive burn, 12 cases of severe pancreatitis, 6 cases of abdominal infection, 7 cases of ileus after abdominal surgery, 5 cases of retroperitoneal hematoma, 3 cases of cirrhotic ascites, 3 cases of after liver transplantation. The APACHE score was  $16.6 \pm 3.8$ , the Intensive Care Unit (ICU) duration was  $11 \pm 6$  d, the mechanical ventilation duration was  $6 \pm 3$  d. The diuretic duration was  $6.0 \pm 2.3$  d. All patients had increased intrabdominal pressure (IAP) with the signs of acute renal insufficiency such as oliguria and rapidly increasing serum creatinine. The urine volume increased and the serum creatinine decreased when the IAP decreased. The treatments included decompression by laparotomy or laparoscopy, ascites draw, and the application of gastrointestinal motility drugs. External use of Glauber's salt on the abdomen and regular diuretic and low dose of dopamine therapy was used for oliguria patients. Primary renal insufficiency was excluded in all patients.

### **Monitoring Measurements**

(1) Measured the arterial pressure via radial artery intubation; (2) Measured the central vein pressure (CVP) via internal jugular vein intubation or subclavian vein intubation; (3) Measured the IAP by bladder measurement. The patient was asked to lie in the dorsal position. Then 100 ml normal saline was injected after emptying the bladder. The IAP was measured with the symphysis pubis as the baseline. (4) Measured the patient's IAP three times per day at a fixed time, and recorded the mean arterial pressure (MAP), CVP, urine volume, and collected blood samples for the detection of serum creatinine.

### **Diagnosis Criteria**

(1) High IAP (IAP > 2.00 kPa); (2) The occurrence of organ dysfunction, including renal insufficiency and urine volume < 40 ml/h; (3) The patient with mechanical ventilation had increased airway pressure. The airway pressure > 4.41 kPa indicated ACS; (4) Unstable hemodynamics with low blood pressure and decreased cardiac output, catecholamine drugs were required; (5) Lower IAP lead to rapid improvement of the organ functions<sup>5-8</sup>.

### **Statistical Analysis**

SPSS 11.5 software (SPSS Inc., Chicago, IL, USA) was used for statistical analysis. The dif-

ference and correlation were analyzed by *t*-test and linear correlation analysis.  $p < 0.05$  was considered statistically significant.

## **Results**

Thirty patients with ACS had increased IAP to varying extent, with a range from 1.62-5.32 kPa and a mean IAP of 3.40 kPa. This was consistent with the change in renal function measurements. With the increasing IAP, the urine volume decreased gradually from  $92.6 \pm 20$  ml/h to  $27.9 \pm 11.7$  ml/h ( $p < 0.05$ ), and serum creatinine increased from  $68.4 \pm 39.3$  mmol/L to  $249.4 \pm 111.5$  mmol/L ( $p < 0.05$ ). With the increased CVP (from  $1.34 \pm 0.25$  kPa to  $2.12 \pm 0.22$  kPa,  $p < 0.05$ ) and increased or decreased MAP, the increase of IAP was negatively related to the urine volume ( $r = -0.193$ ,  $p < 0.05$ ), positively related to serum creatinine ( $r = 0.162$ ,  $p < 0.05$ ), but not related to the MAP (Table I).

## **Discussion**

### **The Correlation Between the Increase of IAP and Renal Function Measurements, MAP, and CVP**

ACS is a clinical syndrome common in ICU, caused by high IAP with multiple organ dysfunctions. The value and duration of IAP are the most significant and sensitive indicators for determining the severity of ACS, and the change of IAP is positively related to the seriousness of the disease. It was shown that intravesical pressure (IVP) was related to IAP, and the measurement of IVP was non-invasive, convenient and precise. Thus, it was the best method for monitoring IAP<sup>9-13</sup>. The hospital has been following IVP to measure IAP. The normal range of IAP was 0-0.933 kPa, when the IAP increased to 2.00-2.67 kPa, oliguria may occur. When the IAP rises to 4.00 kPa, anuria may occur. The change of IAP was well related to the change of renal function measurements, with the increasing IAP. The urine volume decreased gradually, and the serum creatinine increased gradually, with increasing CVP and increased or decreased MAP. In this study, all 60 patients with ACS had increased IAP to varying extent. When IAP was  $2.31 \pm 0.4$  kPa, oliguria could occur. When IAP was  $3.11 \pm 0.5$  kPa, the urine volume further decreased or even anuria

**Table I.** The correlation between the increase of IAP and the changes of renal function measurements, MAP, and CVP.

IAP (kPa)	MAP (kPa)	CVP (kPa)	Measurements	
			Creatinine (mmol/L)	Urine volume (ml/h)
2.02 ± 0.43	10.76 ± 0.9	1.34 ± 0.25	68.4 ± 39.3	92.6 ± 20.0
2.58 ± 0.4	11.76 ± 0.65	1.75 ± 0.21*	121.9 ± 35.6*	64.3 ± 18.3*
3.38 ± 0.49	9.07 ± 1.02	2.12 ± 0.22 <sup>§</sup>	249.4 ± 11.5 <sup>§</sup>	27.9 ± 11.7 <sup>§</sup>
<i>r</i> value	0.311	0.288	0.162	-0.193

\*Indicated that IAP was 2.58±0.40 kPa, and  $p < 0.05$  in comparison with 2.02±0.43 kPa; <sup>§</sup>Indicated that IAP was 3.38±0.49 kPa, and  $p < 0.05$  in comparison with 2.58±0.4 kPa.

could occur with increased serum creatinine. The increase of IAP was negatively related to the urine volume ( $p < 0.05$ ), and positively related to the serum creatinine ( $p < 0.05$ ). It was accompanied by an increase in CVP ( $p < 0.05$ ) and an increase or decrease of MAP. The MAP was not related to the change of IAP. In clinical monitoring, we should differentiate the false increase in CVP and the false increase of MAP due to high IAP. It was shown that when IAP was 2.67 kPa, glomerular filtration rate may decrease to less than 25% of normal level, renal vascular resistance may increase to 5.5 fold of normal level whereas peripheral vascular resistance did not increase significantly. When IAP was 5.33 kPa, renal artery blood flow may decrease by 70%. At this time, when neither the effect on renal function by increased IAP was noted no abdominal decompression was performed, and irreversible renal failure may occur. Abdominal decompression could not only increase renal blood flow effectively, but also decrease the level of angiotensin-aldosterone in circulation. When IAP was decreased, urine volume could increase significantly with permeable diuretic, and renal function could be improved considerably.

#### **The Mechanism Underlying the Change of Renal Function due to High IAP**

The change of renal function due to high IAP is attributed to multiple factors. It can cause increased inferior vena cava pressure and intrathoracic pressure, decreased cardiac output, and subsequently decreased renal blood perfusion<sup>14-16</sup>. Moreover, high IAP has direct compression of renal parenchyma and renal artery and vein, which will cause decreased renal blood flow and increased renal vascular resistance, leading to decreased glomerular filtration rate and retention of sodium and water in the

renal tubule<sup>17-20</sup>. Additionally, the increased activity of renin, angiotensin, and aldosterone hormone system can cause renal vasoconstriction, retention of sodium and water, and further decrease of urine volume and the excretion of nitrogen-containing material. However, we found that for some patients who needed large amount of fluid resuscitation, renin, angiotensin and aldosterone hormone system did not change significantly, as venous transfusion and the re-absorption of Ringer-Locke liquor by peritoneum increased the blood volume and returned blood volume, which compensated the effect on cardiac output by high IAP.

The characteristics of renal insufficiency due to ACS is that oliguria progresses into anuria and diuretic has no significant impact. The supplementation of blood volume and the application of dopamine and diuretic have no significant effect. However, urine volume will increase, and renal function will improve significantly after abdominal decompression. Although total blood volume and blood pressure are normal, increased IAP will cause the oliguria or even anuria. In this study, we found that only with decreased IAP, renal function would be improved<sup>21-26</sup>.

#### **ACS Treatment**

We used different procedures based on the value of IAP. When IAP was 2.02 ± 0.43 kPa, we maintained normal fluid volume or increased fluid infusion to increase renal perfusion pressure. When IAP was 2.58 ± 0.40 kPa, we used non-surgical decompression of IAP. When IAP was 3.38 ± 0.49 kPa, we used laparoscopy or open abdominal decompression. Most people set IAP > 3.33 kPa as the indicator for open abdominal decompression. In this study, 38 patients with an IAP > 3.33 kPa underwent laparoscopy (20 cases) or open abdominal decompression (18 cases). As a result, their

oliguria recovered rapidly, and the renal functions were improved. Three other patients with cirrhotic ascites had significantly decreased IAP after continuous drainage of the ascites. The rest of the patients had relieved flatulence, intestinal edema, and their IAP decreased after continuous renal replacement treatment, external application of Glauber's salt on the abdomen. Limited fluid resuscitation was performed for patients with traumatic hemorrhagic shock to maintain the systolic pressure at about 9.31 kPa. The crystal/colloid ratio was set at 1:1.2 and the plasma proteins above 45 g/L, so as to relieve the accumulation of fluid in the 3<sup>rd</sup> space and the edema in the abdominal organs, and decrease the IAP. If necessary, low dose of dopamine (2-5 µg/kg\*min) may be used to improve the renal blood perfusion and maintain the urine volume > 1 ml/(kg\*h). In this study, although the patients had increased urine volume and recovered renal function soon after treatment, 21 patients died, accounting for 35% of the reported patients. The cause of death was not ACS. However, it was indicated that the patients with ACS may have worse condition and probable increased death rate.

### ***The Nursing of ACS***

#### ***Close Monitoring of the Condition Change***

The patients' clinical symptoms, such as abdominal distension, abdominal muscle tension, decreased abdominal wall compliance, increased heart rate, decreased blood pressure, increased peak inspiratory pressure under mechanical ventilation, oliguria or anuria, should be evaluated and treated timely to prevent further progress into multiple organ failure<sup>27</sup>.

#### ***Continuous Dynamic Monitoring of the Change of IAP***

Routinely, ICU patient should be monitored of IAP, early identification of the patient with high risk of ACS and timely treatment could prevent further damage to organ functions by high IAP. Intravesical pressure is usually used to determine IAP in ICU indirectly. It has been the gold standard of IAP measurement currently. Specifically, infused 100 ml normal saline into the empty bladder with a Foley catheter, occluded the distal terminal, inserted a syringe needle into the catheter and connected it to the pressure measurement device. The zero position was set to be the symphysis pubis level when the patient was

in the dorsal position. The normal range of IVP was 0-0.93 kPa. In order to ensure accurate measurement, the patient should be asked to empty the bladder before measurement and be in appropriate position (dorsal position). Calibrated the zero position and read the pressure value at the end of the expiration. For patients with mechanical ventilation, the IAP should be read a while after removal of the respirator so as to exclude the effect of IAP by positive pressure ventilation and the positive pressure ventilation at the end of the respiration.

## **Conclusions**

### ***Maintaining Stable Hemodynamics***

Corrected coagulation disorders and acidosis, maintained adequate respiratory support, carefully monitored the change in vital signs and prevented further worsening condition<sup>28</sup>.

### ***Ensuring Effective Renal Perfusion***

Recorded 24h input and output, maintained adequate blood volume, used low dose of dopamine (2-5 µg/kg\*min) if necessary to improve renal blood perfusion and maintain urine volume > 1 ml/kg\*h. Monitored the change of renal function measurements periodically, identified and treated the change timely.

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### **Conflict of Interest**

The Authors declare that they have no conflict of interests.

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