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Intra-abdominal hypertension: Incidence and association with organ dysfunction during early septic shock

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Abstract

Purpose: The objective of this article is to study the cumulative incidence of intra-abdominal hypertension (IAH) in septic shock (SS) patients during the first 72 hours of intensive care unit (ICU) admission and to determine if the presence and severity of IAH are associated with sepsis morbidity and mortality.

Materials and Methods: Eighty-one consecutive SS patients admitted to a surgical-medical ICU of an academic university hospital (January 2005 to January 2006) were included. Intra-abdominal pressure (IAP) and abdominal perfusion pressure (APP) were measured every 6 h (intermittently) for 72 h. Intra-abdominal pressure was registered as minimal, mean, and maximal values per day, during shock and throughout the study period. Intra-abdominal hypertension was diagnosed if IAP remained 12 mm Hg or higher on 2 consecutive measurements and stratified according to the most recent consensus definition (www.wsacs.org).

Results: According to maximal and mean IAP values, 67 (82.7%) and 62 (76.5%) of the patients developed IAH during the study period, respectively. Mean IAP values remained stable throughout the study period. Surgical patients had a higher incidence of IAH than medical patients (93% vs 73%, $P < .009$). Maximal IAPs were normally distributed, with nonsurvivors exhibiting significantly higher IAP levels during shock (survivors, 17.2 ± 5.3; nonsurvivors, 19.9 ± 5.6 mm Hg; $P < .04$). Patients with IAH exhibited significantly lower values of APP and diuresis, higher values of lactate and creatinine, and higher maximal norepinephrine doses, and were more frequently mechanically ventilated ($P < .05$ for all). Increasing degrees of IAH and the development of the abdominal compartment syndrome were associated with lower APP and higher maximal serum creatinine levels ($P < .03$ for both).

Conclusions: Septic shock patients have a very high incidence of IAH, which seems to be associated with the severity of shock and could be related to the development of organ dysfunctions, particularly renal dysfunction. Intra-abdominal pressure should be routinely monitored during the course of SS.

1. Introduction

Intra-abdominal hypertension (IAH) is defined by a sustained or repeated pathologic elevation of intra-abdominal pressure (IAP) higher than 12 mm Hg. The deleterious

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effects of IAH were first described more than 200 years ago [1]. Intra-abdominal hypertension may contribute to splanchnic hypoperfusion and development of multiple organ failure in critically ill patients [2-5].

Reported prevalence of IAH in critically ill patients ranges from 18% to 58.8% [6-10]. This wide variation is explained by different clinical settings (surgical or medical) and conditions (trauma, burned and postoperative patients), the IAP measurement technique used, and the cutoff IAP level defined for diagnosis of IAH (12-25 mm Hg) [10]. Using a cutoff value of 12 mm Hg to define IAH in a mixed general intensive care unit (ICU) population, Malbrain et al [6,10] reported a prevalence of 58.8% and a cumulative incidence during the first week of ICU admission of 57.7%, with maximal IAP values, but only 26.8% with mean values.

In 2004, when the World Society on the Abdominal Compartment Syndrome (WSACS) was founded, consensus definitions and grading for IAH were proposed [11,12] (www.wsacs.org). Intra-abdominal hypertension was defined as a sustained or repeated pathologic elevation of IAP 12 mm Hg or higher, and was graded into 4 levels (grade 1, 12-15 mm Hg; grade 2, 16-20 mm Hg; grade 3, 21-25 mm Hg; and grade 4, >25 mm Hg). Abdominal compartment syndrome (ACS) was defined as a sustained IAP higher than 20 mm Hg (with or without abdominal perfusion pressure [APP] <60 mm Hg), which is associated with a new organ dysfunction [13]. Primary ACS is a condition associated with injury or disease in the abdominopelvic region, which frequently requires early surgical or interventional radiologic procedures, and secondary ACS refers to conditions that do not originate from the abdominopelvic region.

Previous studies on IAH have focused mainly on trauma [14-16], surgical [17-19], burned [20], and obese [16] patients, and an elevated prevalence of IAH has also been reported in sepsis [21]. However, limited information exists about the prevalence and consequences of IAH in septic shock (SS) patients [22]. Septic shock patients usually have increased gut permeability and ileus, together with other risk factors for IAH, such as massive fluid resuscitation. Intra-abdominal hypertension could aggravate hemodynamic instability and hypoperfusion, contributing to organ dysfunction in this setting [23].

In an earlier study, our group found a 51% incidence of IAH in a small series of surgical and medical SS patients [24]. However, a higher level of IAP (≥20 mm Hg) than proposed by the consensus conference was used to define IAH, possibly underestimating the real incidence of IAH in SS patients.

Therefore, the aim of this study was to determine the cumulative incidence of IAH in SS patients during the first 72 hours of ICU admission, according to the new consensus definitions [11,12]. The secondary objective was to determine if the presence of IAH and its different levels of severity are associated with more severe forms of SS in terms of vasopressor requirements, lactate levels, severity of organ dysfunctions, and mortality.

2. Materials and methods

A prospective observational study was conducted from January 2005 to January 2006 in the Surgical and Medical Intensive Care Units of the University Hospital of the Pontificia Universidad Católica de Chile, Santiago, Chile. Although no specific intervention was made in connection with the IAP measurements, the study was approved by the ethical committee of the Pontificia Universidad Católica de Chile, and all patients or their relatives signed an informed consent form.

2.1. Patients

Eighty-one consecutive SS patients admitted to the ICU during the study period were included. Three patients died before a minimum of 3 measurements could be completed and were therefore excluded. Septic shock was defined as the state of acute circulatory failure characterized by persistent arterial hypotension unexplained by causes other than infection. Hypotension was defined as systolic arterial pressure lower than 90 mm Hg, mean arterial pressure (MAP) lower than 60 mm Hg, or reduction in systolic blood pressure greater than 40 mm Hg from baseline, requiring vasoactive support despite adequate volume resuscitation [25].

Demographic and clinical data including age, sex, date of enrollment, medical or surgical condition, and APACHE II were registered upon admission. During follow-up, the

Table 1 Mean values and cumulative incidence (expressed as maximal. mean and minimal values) of IAH during the study period (72 hours) during shock and per day

	No. of patients	Mean ± SD	Incidence (%)
During the study period (72 h)			
Maximal IAP	81	18.1 ± 5.5	82.7
Mean IAP	81	15.1 ± 4.7	76.9
Minimal IAP	81	11.2 ± 4.1	46.2
During shock			
Maximal IAP	81	17.9 ± 5.6	81.5
Mean IAP	81	15.1 ± 4.9	72.8
Minimal IAP	81	12.3 ± 4.3	58
During the first 24 h			
Maximal IAP	81	16.5 ± 4.9	76.9
Mean IAP	81	14.8 ± 4.5	72.8
Minimal IAP	81	12.8 ± 4.6	61.7
During the second day			
Maximal IAP	74	16.7 ± 4.9	79.1
Mean IAP	74	14.8 ± 4.3	70.8
Minimal IAP	74	13.0 ± 4.1	62.1
During the third day			
Maximal IAP	70	17.8 ± 5.4	88.5
Mean IAP	70	16.2 ± 5.2	78.5
Minimal IAP	70	14.1 ± 5.4	74.2

sequential organ failure assessment (SOFA) score, maximal norepinephrine (NE) infusion rates, lactate levels, and fluid balance were registered on a daily basis. The study period included the first 72 hours after ICU admission, and ICU mortality, hospital mortality, and 28-day mortality were assessed thereafter.

2.2. Hemodynamic management

All SS patients were treated with a standard hemodynamic management algorithm [26] based on NE as vasopressor, with implementation of sequential interventions designed to reach a MAP of higher than 70 mm Hg and normalization of lactate and venous oxygen saturation values. Briefly, the algorithm begins with fluid administration in which at least 30 mL/K of normal saline are infused in 1 hour and continued until central venous pressure higher than 10 mm Hg or pulmonary artery occlusion pressure in the range of 14 to 18 mm Hg is achieved. If MAP remains lower than 70 mm Hg despite fluid administration, NE infusion is started and progressively increased until the MAP goal is achieved. Other interventions, such as administration of additional fluids, dobutamine infusion or red cell transfusions, are started if necessary, with the goal of normalizing lactate and central or mixed venous oxygen saturation.

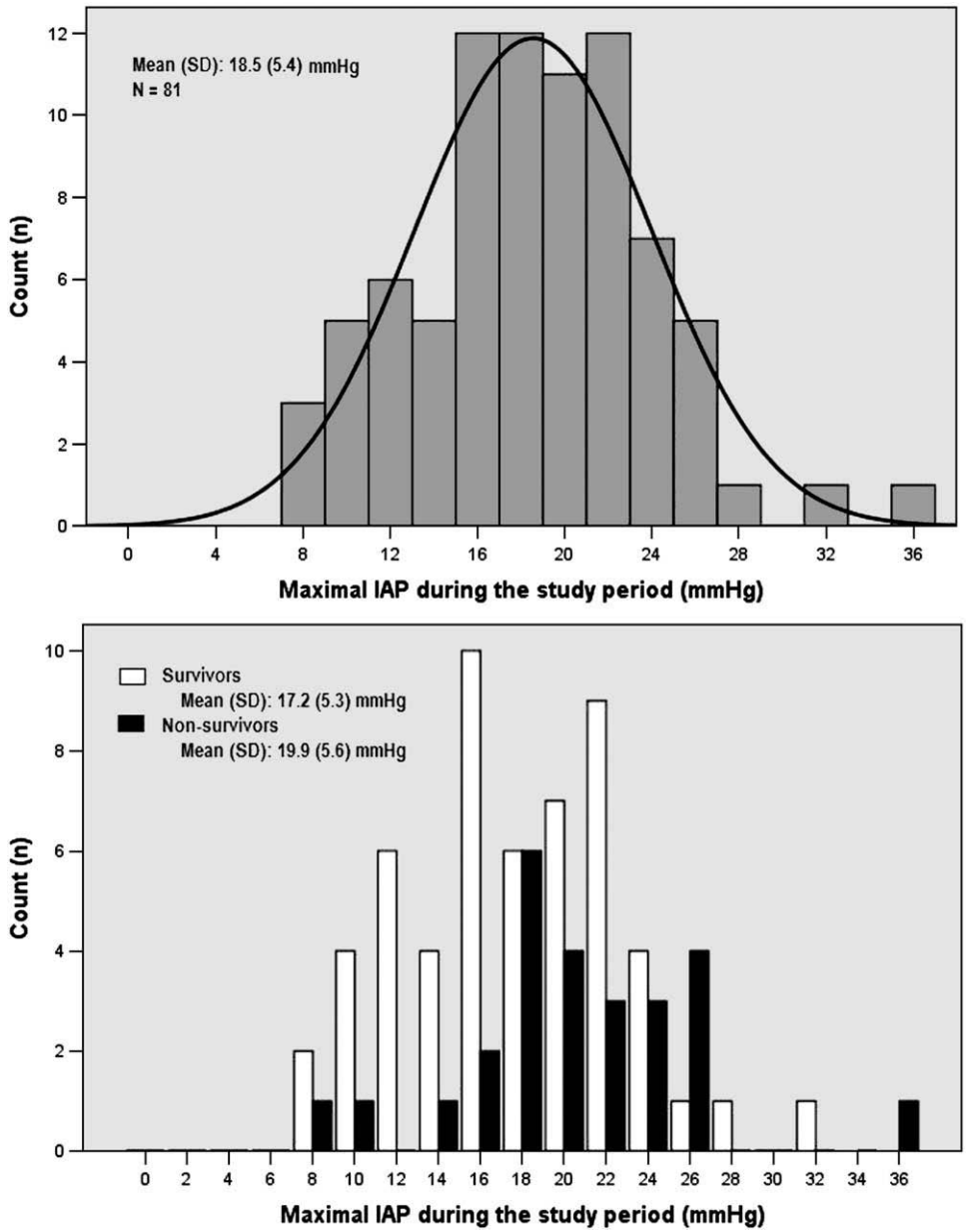

Fig. 1 Top, histogram of maximal IAP during the study period (72 hours). Bottom, histogram of maximal IAP split according to the outcome at 28 days.

Table 2 Main characteristics of the patients according to WSACS consensus definitions and grading system

Variable	Total group	IAH [1]	All IAH (+)	IAH (+)				*P**	*P***
				12-15 mm Hg	16-20 mm Hg	21-25 mm Hg	>25 mm Hg		
n	81	14 (17.3%)	67 (82.7%)	8 (11.9%)	32 (47.8%)	21 (31.3%)	6 (9%)		
Age (y)	58.6 ± 18.2	55 ± 13	58.5 ± 16.2					.48	
Male/female	54/27	9/5	45/22						
APACHE II	21.8 ± 6.8	18.9 ± 3.8	22.1 ± 9					.19	
Mechanical ventilation	72 (88.8%)	10 (71.4%)	62 (92.5%)					<.03	
Cumulative fluid balance [2]	4781 ± 2960	4619	5135					.48	
Diuresis (mL/h)	34 ± 31	63 ± 47	29.7 ± 25					.045	
Maximal IAP (mm Hg)	18.1 ± 5.5	10.4 ± 2.4	19.8 ± 4.3	14.1 ± 0.8	17.8 ± 1.5	22.5 ± 1.4	28.8 ± 4.2	<.001	<.001
APP (mm Hg)	56.8 ± 5.4	64.6 ± 2.4	55.2 ± 4.3	60.9 ± 0.8	57.2 ± 1.5	52.4 ± 1.4	46.2 ± 4.2	<.001	<.001
SOFA score (maximal)	11.1 ± 3.2	10 ± 2.8	11.3 ± 3.3	12 ± 2	10.6 ± 3.6	12.3 ± 3.8	11 ± 2	.17	.25
Maximal NE dose (μg/K per minute)	0.44 ± 0.5	0.28 ± 0.25	0.48 ± 0.54	0.26 ± 0.2	0.45 ± 0.5	0.67 ± 0.7	0.16 ± 0.2	.03	.06
Maximal lactate level (mmol/L)	5.1 ± 4	2.5 ± 1.9	5.7 ± 4.2	4.8 ± 3.5	5.9 ± 4.7	6.2 ± 3.9	3.2 ± 2.2	.008	.04
Maximal serum creatinine (mg/dL)	2.6 ± 2.3	1.8 ± 1.8	2.7 ± 2.3	1.5 ± 0.5	2.3 ± 1.4	3.3 ± 2.7	4.7 ± 4.8	.03	.03
Lowest Pao_2/FiO_2 ratio	192 ± 87	194 ± 119	192 ± 82	123 ± 56	193 ± 90	213 ± 74	196 ± 47	.95	.24
ICU mortality	26 (32%)	6 (42.8%)	20 (29.9%)	2 (25%)	9 (28.1%)	7 (33.3%)	2 (33.3%)	.46	.7
28 d of mortality	27 (33.3%)	6 (42.8%)	21 (31.3%)	2 (25%)	10 (31.3%)	7 (33.3%)	2 (33.3%)	.54	.7

Values were expressed as mean ± SD for each group.

* $P < .05$ considered significant χ^2 test and *t* test for independent samples comparing IAH [1] vs IAH (+).

** $P < .05$ considered significant. One-way ANOVA comparing increasing degrees of IAH.

2.3. Mechanical ventilation

Mechanically ventilated patients were managed using a lung protective strategy following recent guidelines [27].

2.4. Intra-abdominal pressure monitoring and management

Intra-abdominal pressure and APP (calculated as MAP-IAP) were measured every 6 hours during the first 72 hours after the diagnosis of shock using a Foley bladder catheter filled with 50 mL of normal saline, with the patient in complete supine position and adequately sedated if on mechanical ventilation [28-30]. Any IAP measurement higher than 12 mm Hg was repeated after 30 minutes to confirm the finding. The second value (after 30 minutes) was used for registration. The measured IAP values were available to the attending physician for clinical use; however, no specific management recommendations were made as part of this study.

Daily maximal, mean, and minimal IAP values were analyzed throughout the study period (see definitions at the "Introduction" section). The degree of severity was registered in patients who developed IAH.

2.5. Statistical analysis

Cumulative incidence during the 72-hour period is presented as maximal, mean, and minimal values. In addition, these values are presented for the period in shock and for every 24 hours. Maximal values of IAP during the 72-hour study period were used to classify patients to compare hemodynamic, metabolic, and prognostic variables between different levels of IAP. Thereafter, the consensus definition [11,12] was used to stratify IAP levels. Maximal values of IAP during shock period were used to compare variables between survivors and nonsurvivors. A normal distribution of data was confirmed with the Kolmogorov-Smirnov test. Data are presented as mean ± SD and are analyzed with the χ^2 test, the independent *t* test, 1-way analysis of variance (ANOVA), and Pearson correlation as appropriate. SPSS 13.0.1 statistical software was used (SPSS, Chicago, Ill). Results are expressed as mean ± SD, and a 2-tailed $P < .05$ was considered as statistically significant.

Table 3 Comparison of survivors vs nonsurvivors in all the study population (n = 81)

Variable	Survivors	Nonsurvivors	*P* *
APACHE II	21.2 ± 6	23.8 ± 7	.2
APP (mm Hg)	57.6 ± 5.3	54.8 ± 5.2	.03
Maximal IAP (mm Hg) during shock	17.2 ± 5.3	19.9 ± 5.6	.04
Mean IAP (mm Hg), 72 h	13.6 ± 3.3	19.1 ± 5.5	.04
Maximal NE dose (μg/K per minute)	0.31 ± 0.3	0.55 ± 0.3	.001
Maximal lactate level (mmol/L)	3.9 ± 2.6	7.3 ± 5.4	.003
Maximal serum creatinine (mg/dL)	2.16 ± 1.9	3.48 ± 2.6	.017
Lowest Pao_2/Fio_2 ratio	209 ± 91	155 ± 68	.012
SOFA score (maximal)	10 ± 3	13 ± 3	<.001

* $P < .05$ considered significant. *t* Test for independent samples.

Table 4 Comparison of patients with or without ACS considering only patients with IAH (n = 67)

Variable	Non-ACS	ACS	*P* *
No. of patients	40	27	
APACHE II	21.3 ± 7	24.6 ± 6	.1
APP (mm Hg)	57.9 ± 4	51.1 ± 4	<.001
Maximal IAP (mm Hg)	17 ± 2	23.9 ± 3.4	<.001
Mean IAP (mm Hg), 72 h	14.3 ± 1.3	20.1 ± 4.4	.007
Maximal NE dose (μg/K per minute)	0.38 ± 0.3	0.44 ± 0.4	.5
Maximal lactate level (mmol/L)	5.7 ± 4.5	5.5 ± 3.8	.8
Maximal serum creatinine (mg/dL)	2.1 ± 1.3	3.6 ± 3.1	.02
Lowest Pao_2/Fio_2 ratio	180 ± 87	209 ± 69	.18
SOFA score (maximal)	11 ± 3	12 ± 3	.16
ICU survival (n(%))	12 (30%)	9 (33%)	.4

* $P < .05$ considered significant. *t* Test for independent samples.

3. Results

Eighty-one consecutive SS patients were enrolled in the study. Septic shock was caused most frequently by intra-abdominal (44.5%) and pulmonary (27%) infections, followed by urinary (8.6%), soft tissue (8.6%), obstetric (4%), and oncologic (6%) infections. Surgical patients had a higher incidence of IAH (37/40 patients, 92%) than medical patients (30/41 patients, 73%) ($P < .009$).

For the 72-hour study period, the cumulative incidence was 82.7% (67/81 patients) for maximal IAP values and 76.5% (62/81 patients) for mean values. When considering only the shock period, the cumulative incidence was 81.5% for maximal IAP values and 72.8% for mean values (Table 1). A total of 980 measurements were performed. At the end of the study period, 16 (19.7%) patients still required vasoactive drugs and 11 (13.6%) had died. Accordingly, the mean duration of IAP monitoring was 64 hours when considering all patients. Maximal IAP were normally distributed and were higher in nonsurvivors (survivors, 17.2 ± 5.3, vs nonsurvivors, 19.9 ± 5.6 mm Hg; $P < .04$) (Fig. 1).

Septic shock patients with IAH had significantly lower APP and diuresis, had higher lactate and creatinine levels, required higher NE concentrations, and were more frequently mechanically ventilated (Table 2). For patients with

IAH, increasing degrees of IAH were paralleled by a progressive decline in APP and an increase in maximal serum creatinine levels. Maximal lactate levels increased significantly in parallel with IAP levels, except in patients with IAP higher than 25 mm Hg (n = 6). Increasing degrees of IAH were not associated with differences in Pa_{O_2}/Fi_{O_2} ratios, SOFA scores, or mortality (Table 2). Abdominal perfusion pressure was negatively correlated with maximal serum creatinine ($P < .001$, $r = 0.37$).

When analyzing all patients, nonsurvivors exhibited higher mean and maximal IAP, higher lactate and creatinine levels, higher SOFA scores, and lower APP and Pa/Fi_{O_2} ratios than survivors (Table 3).

Of the 67 patients with IAH, 33 (49%) had primary IAH and 34 (51%) had secondary IAH. When comparing these 2 groups, there were no differences in mortality, IAP, APP, or shock severity parameters. In addition, 12 (18%) of the 67 patients with IAH had primary ACS and 15 (22%) had secondary ACS. When comparing patients with and without ACS, only the maximal serum creatinine was significantly higher in patients with ACS (Table 4).

4. Discussion

Few studies have focused on IAH in critically ill patients, and this is the first observational study of IAH in the subgroup of patients with SS applying the new WSACS Consensus definitions [11,12].

Intra-abdominal hypertension has a high incidence during the early course of SS. Even when the mean value of IAP is considered, the incidence is still elevated. Rates of 82% based on maximal values or 77% based on a 72-hour mean IAP raise concern about a frequently underlooked problem in SS care. Furthermore, 38% of the patients fulfill criteria for ACS.

Our data represent the highest incidence reported to date for any known condition associated with a high risk of IAH. Previous studies defining IAH with an IAP 12 mm Hg or higher have reported a prevalence of 58% in sepsis [21] and 59% in a mixed population of critically ill patients [6]. The incidence found in our study, although very high, should not be completely unexpected considering the several predisposing factors commonly present in SS patients: acidosis, coagulopathy, intra-abdominal infections, bacteremia, mechanical ventilation, massive fluid resuscitation, ileus, and a high degree of interstitial edema [11].

Medical patients also exhibited a high incidence of IAH, and 21% developed ACS. Our results are consistent with previous data from a multicenter study in a mixed ICU population, where medical and surgical patients had a prevalence of 54.4% and 65%, respectively [6]. This is a relevant observation, in light of a recent survey showing that 25% of medical intensivists had never managed a patient with IAH, 23% were unaware of bladder pressure measurement procedure, and only 25% believed that physiologic compromise occurred with IAP between 12 and 19 mm Hg [31,32]. We believe that IAP should be routinely measured and active management considered in all SS patients, irrespective of whether the setting is medical or surgical.

Our findings suggest that the coexistence of SS and IAH is associated with a higher severity of shock, reflected in higher lactate values, greater NE requirements, and more severe renal dysfunction compared with SS without IAH. Higher degrees of IAH were also related to more severe renal dysfunction. Previous studies have shown that IAH is an independent predictor of mortality in a mixed population of ICU patients [10]. An association between IAH and mortality was not observed, despite the fact that IAH patients presented more severe forms of shock. We cannot explain this fact, but it certainly should be explored in future studies.

The kidneys appear to be particularly vulnerable to IAH. Renal perfusion pressure and glomerular filtration gradients are closely related to IAP [33,34]. Experimental models have shown that IAH has a profound impact on renal intrapelvic pressure, probably secondary to ureteral compression [35], although stenting of the ureter did not improve renal function in an animal study. Furthermore, some other studies indicate that direct organ compression increases resistance in renal veins and arterioles. Reduced heart function may also lead to decreased perfusion of the kidneys. These mechanisms in combination may be responsible for a decreased glomerular filtration rate. The increased release of antidiuretic hormone, renin, and aldosterone contributes to tubular necrosis and renal failure [36,37]. Intra-abdominal pressure has been found to be independently associated with the development of renal failure in postoperative abdominal surgery and liver transplant patients [5,35]. In our study, creatinine serum levels were directly proportional to an increase in IAP and inversely related to APP.

Some limitations of our study are the use of 50 mL for IAP measurements instead of 25 mL, as is currently recommended [11,30,38-40], and a brief observation period, because most of the patients still had IAH at 72 hours. It would be interesting to observe the prolonged effect of IAH in these patients. Another limitation is that the data were collected at only one center, and IAP could vary depending on the characteristics of the ICU population and the strategies used for resuscitation, mechanical ventilation, and sedation. An extended multicenter study of SS patients based on IAP measurements obtained by filling the bladder with 25 mL of saline may overcome these limitations.

In summary, patients with SS have a very high incidence of IAH, which seems to be associated with the severity of shock and could be related to the development of organ dysfunction, particularly of the kidney. Medical patients also exhibited an elevated incidence of IAH and ACS. Intra-abdominal pressure monitoring should be routinely performed during early SS.

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