

Release of Abdominal Compartment Syndrome Improves Survival in Patients with Burn Injury

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Background: Abdominal compartment syndrome (ACS) has rarely been described as a complication of burn injury. This study describes cases of ACS in patients with burn injury and the physiologic results of abdominal release.

Methods: Charts for all patients admitted to two major burn center intensive care units from January 1998 through August 2000 were reviewed for ACS. Physiologic parameters were compared before and after abdominal release.

Results: Ten of 1,014 patients developed ACS. Abdominal release improved peak inspiratory pressures and Acute Physiology and Chronic Health Evaluation II scores ($p < 0.03$). The amount of fluid required to maintain adequate urine output also decreased substantially. Forty percent of patients with ACS survived to discharge.

Conclusion: Abdominal release for patients with ACS and severe burn injury results in physiologic improvement and a

40% survival rate. We recommend bladder pressure monitoring for all patients with severe burn injuries and abdominal decompression in any patient who develops pressures greater than 30 mm Hg if they have signs of physiologic compromise. Aggressive expectant management can effect a 40% survival rate in this group of severely injured patients.

Key Words: Abdominal compartment syndrome, Burns, Abdominal compartment release.

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Abdominal compartment syndrome (ACS) is a well-documented complication of major abdominal surgery and abdominal trauma. Numerous studies have described adverse physiologic effects of elevated intra-abdominal pressure including impaired cardiac function, decreased pulmonary compliance, and decreased renal perfusion.^{1–3} As early as 1983, surgeons identified a syndrome that occurred in patients who had undergone major abdominal surgery or had major abdominal trauma. Richards et al. provided one of the earliest descriptions of a small series of patients who developed abdominal distention and anuria after major abdominal trauma with subsequent polyuria after abdominal compartment release by laparotomy.⁴ Kron et al., soon thereafter, proposed monitoring intra-abdominal pressure in patients by means of the bladder catheter, and were the first to recommend laparotomy for patients with anuria and intra-abdominal pressures greater than 30 mm Hg.⁵ Burn victims, however, have no primary intra-abdominal disease. As a result, the incidence of ACS in burn patients has, until recently, been relatively ignored.

Two studies of ACS resulting from severe burn injuries have been published to date.^{6,7} Together, these two studies include 40 patients with burn injuries. Eighteen of these patients developed intra-abdominal hypertension, but only a handful developed signs of physiologic derangements that might suggest the presence of ACS. Although these studies confirm ACS does indeed occur in the burn population, the number of patients studied was far too small to determine the incidence or survivability of ACS in patients suffering from burn injury. Furthermore, those few patients who required abdominal decompression had a very poor survival rate. It could therefore be argued that decompressive laparotomy may not be the best treatment for patients suffering from ACS as a result of massive burn injury. Burn patients are unique from the patients in whom ACS has historically been described, as no primary intra-abdominal disease exists. Furthermore, burn patients have no preexisting laparotomy incision that can be easily opened with little additional morbidity. Many argue that a laparotomy in burned patients, patients who have already lost the protective barrier of normal skin, induces unacceptable additional morbidity.

The goal of this study was to review the experience of ACS in patients suffering from severe burn injuries in two regional burn units. We also hoped to determine whether, despite the lack of primary intra-abdominal disease, abdominal compartment release would lead to resolution of the physiologic abnormalities that accompany the syndrome, thus improving patient survival.

MATERIALS AND METHODS

The burn center at the University of California Davis Medical Center is a Level I referral center that treats over 200

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adult burn victims per year from a referral area that includes a population of 7 million people distributed over 30,000 square miles. The Shriners Hospital for Children Northern California treats over 190 pediatric patients a year for acute burn injury. The Shriners Hospital receives referrals from throughout the western United States, Mexico, Canada, and the Pacific Islands. All burn patients presenting to the Sacramento region are admitted to the two hospitals. All patients older than 17 years of age are admitted to University of California Davis and all patients younger than 18 are admitted to Shriners Hospital. This review represents a spectrum of all patients presenting with burns. Over a 32-month period extending from January 1998 through August 2000, 1,014 patients with acute burn injuries were treated in the burn intensive care units (ICUs) of these two hospitals. Charts for these patients were screened for a diagnosis of ACS. The records for those patients who developed ACS and required abdominal decompression were then reviewed.

Diagnosis of compartment syndrome was first ascertained by clinical impression. ACS was suspected in any patient who presented with a clinically tense abdomen in combination with either exceedingly high peak inspiratory pressures that compromised the ability to ventilate the patient, or oliguria despite aggressive fluid resuscitation. Clinical suspicion was then supported by assessing intra-abdominal pressure. Intra-abdominal pressure was estimated by measuring bladder pressure, in the manner described by Kron et al.⁵ and validated in a study by Iberti et al.,⁸ in which 30 to 50 mL of sterile saline is injected into the urinary bladder through an indwelling Foley catheter. Injected fluid is then allowed to flow out of the bladder until a constant column of water fills the catheter tubing with no interfering air. The catheter is subsequently clamped immediately distal to an aspiration port. An 18-gauge needle is inserted into the aspiration port and connected to a pressure transducer, which is the same type of system used for arterial line pressure monitoring and which is readily available in the ICU setting. The transducer is then normalized to zero at the level of the pubic symphysis, and the bladder pressure is transduced. Only end-expiratory values are used in an effort to avoid interference from respiratory excursion of the diaphragm into the intra-abdominal space. Multiple investigators have confirmed a high correlation of bladder pressure measured in this fashion with actual intra-abdominal pressure.^{5,8,9}

All patients with a diagnosis of ACS were initially managed with nonoperative measures. In an attempt to decrease the elevated intra-abdominal pressure, nasogastric decompression, bladder catheter irrigation (to ensure patency), bowel care, sedation, pharmacologic paralysis and, if circumferential wounds to the abdomen existed, abdominal escharotomies were performed. Those patients who failed to improve rapidly after institution of these conservative measures underwent operative abdominal compartment release. Intra-abdominal hypertension was relieved in one of two ways. The majority of patients underwent exploratory laparotomy, either



Fig. 1. Example of a typical open abdomen with a silastic bag sewn to the fascia to protect the underlying abdominal contents from injury and desiccation, and to prevent excessive amounts of fluid from leaking onto surrounding bandages. Removal of silastic sheet and closure of abdominal wall should be attempted as soon as possible.

in the intensive care unit or in the operating suite, depending on the patient's clinical stability. Temporary closure was achieved with a silastic sheet, and the abdomen was closed primarily at the earliest time possible (Fig. 1). Two of the patients had their abdomens decompressed by percutaneous placement of a large-bore hemodialysis catheter into the intra-abdominal space, with drainage of intra-abdominal fluid. The procedure is performed without ultrasound guidance. A needle is placed into the abdominal cavity. A wire is then placed and a catheter is placed over the wire. No complications of the procedure occurred. As is typical of patients with burn injury, none of the patients studied presented with mechanisms of injury that would suggest the possibility of primary intra-abdominal disease.

Records for patients with documented ACS were analyzed for percentage total body surface area (TBSA) burned, the distribution of the burns (specifically, the presence or

Table 1 Demographics of Patients with Abdominal Compartment Syndrome

	Adult	Pediatric	Combined
No. of patients	4	6	10
Age (yr)	32	6	16
% Male	100	83	90
% TBSA burned	75	68	71
% Third-degree burns	75	67	70
ACS caused by sepsis (%)	0	4 (67)	4 (40)
Catheter decompressed	0	2	2
IVF/kg/% TBSA burned	3.1	3.1	3.1
Survived (%)	1 (25)	3 (50)	4 (40)

TBSA, total body surface area; ACS, abdominal compartment syndrome; IVF, intravenous fluid.

absence of circumferential abdominal burns), age, and mechanism of abdominal release. Timing of the development of ACS was also recorded, as was the presence or absence of sepsis (defined as bacteremia with signs of cardiopulmonary compromise). Intra-abdominal pressures were recorded both before and after the decompressive procedure. Likewise, multiple pre- and postdecompression physiologic parameters such as peak inspiratory pressure, central venous pressure, and mean arterial pressure were also compared. Initial volume of fluid resuscitation was noted, as was the total intravenous fluid (IVF) administered and the amount of urine produced for both the 6 hours before and the 6 hours after abdominal release. Acute Physiology and Chronic Health Evaluation II (APACHE II) scores were calculated for each patient as described by Knaus et al.¹⁰ APACHE II scores were used as a standardized indicator of severity of injury and have been shown to accurately predict patient survival.^{10,11} Survival to discharge was recorded as a primary endpoint.

All results are expressed as the mean $\pm$ SEM. Pre- and postrelease values were analyzed for statistical significance by a paired Student's *t* test. Results with *p* < 0.05 were considered significant.

RESULTS

A total of 10 of 1,014 patients (1%) of all acutely burned patients were diagnosed with and treated for ACS in the absence of any preexisting abdominal abnormality between January 1998 and August 2000 (Table 1). Six of the cases of ACS occurred in children, with a mean age of 5.5 years (range, 1 month–8 years). The remaining four cases occurred in adults, with a mean age of 32 years (range, 30–35 years). Eight patients underwent laparotomy for abdominal compartment release, and two patients had bedside percutaneous placement of a peritoneal catheter for fluid removal and intra-abdominal pressure release. Patients who developed ACS had extensive, severe burns, with a mean TBSA burn of 70%.

The patients studied fell into one of two distinct groups on the basis of the timing of their ACS and its cause. Six patients developed signs of ACS within the first 24 hours of

Table 2 Physiologic Response to Abdominal Compartment Decompression^a

	Prerelease	Postrelease	<i>p</i> Value
Bladder pressure (mm Hg)	40 $\pm$ 10	26 $\pm$ 5	0.15 ^c
Peak inspiratory pressure (cm H ₂ O)	37 $\pm$ 3	32 $\pm$ 4 ^b	0.02
PaO ₂	110 $\pm$ 13	115 $\pm$ 15	0.82
pH	7.27 $\pm$ 0.03	7.29 $\pm$ 0.04	0.50
Base deficit	5.2 $\pm$ 0.9	5.3 $\pm$ 0.9	0.71
CVP	24 $\pm$ 2	21 $\pm$ 3	0.11
MAP	75 $\pm$ 4	77 $\pm$ 4	0.42
IVF/UOP (6 h)	78 $\pm$ 32	26 $\pm$ 10	0.08
APACHE II score	25 $\pm$ 1	21 $\pm$ 1 ^b	0.01

PaO₂, partial pressure of oxygen; CVP, central venous pressure; MAP, mean arterial pressure; IVF/UOP, ratio of intravenous fluid given to urine output produced; APACHE, Acute Physiology and Chronic Health Evaluation.

^a Results expressed as mean $\pm$ SEM.

^b Improved significantly with abdominal compartment release.

^c Only three patients had bladder pressure measurements repeated after abdominal compartment release; consequently, the marked improvement does not achieve statistical significance.

hospitalization. These patients received a total average of 237 mL/kg of IVF in the first 12 hours of resuscitation. This equates to 3.1 mL/kg/%TBSA for the first 12 hours. Two of the six patients who developed ACS during the course of acute resuscitation survived to discharge. Four patients developed symptoms of ACS later in their hospital course, ranging from 11 days to several months after initial burn shock and resuscitation. None of these patients developed ACS with their initial resuscitation. Rather, ACS developed later, in conjunction with a septic episode, which was accompanied by a severe clinical decline. Two of these four patients with late-onset ACS survived.

Several physiologic parameters improved after abdominal compartment release (Table 2). Preoperative bladder pressure was significantly elevated, with a mean pressure of 40 $\pm$ 10 mm Hg. Abdominal compartment release resulted in a dramatic decrease of intra-abdominal pressure to 26 $\pm$ 5 mm Hg in those patients in whom it was measured (*p* = 0.15). Unfortunately, only 3 of the 10 patients had documented bladder pressures postoperatively, which likely prevented this impressive 35% decrease in intra-abdominal pressure from becoming statistically significant. Peak inspiratory pressures improved significantly from 37 cm H₂O prerelease to 32 cm H₂O postrelease (*p* = 0.02). Overall general physiologic condition also improved significantly with decompression, as evidenced by a significant improvement in APACHE II scores from 25 points preoperatively to 21 points after abdominal release (*p* = 0.008). Patients also tended to require less intravenous fluid after abdominal decompression. Preoperatively, patients required 78 $\pm$ 32 mL of IVF to produce 1 mL of urine. This improved dramatically to 26 $\pm$ 10 mL of IVF per milliliter of urine produced with abdominal decompression, although the improvement was not statistically sig-

nificant ($p = 0.08$). Several parameters were not affected by decompression of the ACS. These included serum pH, base deficit, partial pressure of oxygen or carbon dioxide, central venous pressure, and mean arterial pressure.

Finally, 40% of burn victims diagnosed with ACS survived to discharge, with mean hospital stays of 137 ± 31 days and 94 ± 24 days in the intensive care unit. As alluded to previously, although the numbers are small, 33% of patients who developed ACS as a result of acute resuscitation survived to discharge, compared with 50% of those patients with ACS that developed as a result of late-onset sepsis. Children had 50% overall survival compared with 25% survival in the adult population. Survival rates were not affected by method of decompression. Patients with open exploration had a 38% survival rate. Only two patients were decompressed with a percutaneous catheter, and one of these patients survived to discharge. The second patient died of sepsis. Because of the low number of patients, comparisons between groups cannot be used to determine predictors of survival. The results do suggest, however, that ACS can be successfully treated in the young and old and during early or late stages after burn injury.

DISCUSSION

ACS is a rare complication of severe burn injury. Of over 1,000 acute burn victims cared for in our ICUs over a 2.5-year period, only 1% developed the complex identified as ACS that required decompression. This is a significantly lower incidence than either of the previous series in burn victims would suggest. In the study by Greenhalgh and Warden of 30 pediatric burn victims, 11 patients developed intra-abdominal pressures greater than 30 mm Hg, for a 37% incidence.⁶ However, concurrent physiologic results of the elevated abdominal pressure were not documented, and therefore it cannot be assumed that all of these patients actually had the syndrome. Therefore, this study suggested only that intra-abdominal hypertension, but not necessarily compartment syndrome, occurred in 37% of the patients studied. Ivy et al. published a series of only 10 patients, 7 of whom developed intra-abdominal hypertension, a higher rate than published by Greenhalgh and Warden.⁷ However, only two of the seven went on to develop complications suggestive of ACS.

We did not assess bladder pressures in every patient treated for burn injury, and therefore we cannot infer the relative incidence of intra-abdominal hypertension in the absence of compartment syndrome. However, bladder pressures are routinely measured for patients with large burns who develop signs of intra-abdominal hypertension such as elevated peak pressures, decreased urine output refractory to aggressive fluid management, or a tense abdomen. Furthermore, we did not evaluate the population of burn patients that did not develop signs of ACS. It is possible the overall severity of injury in our ICU population was not as severe as that of the patients in previous studies. A lower average

severity of injury would tend to decrease the incidence of ACS in our patients. This is especially possible when we consider that our patients with ACS had on average 70% TBSA burns, which is indicative of a fairly severe injury. Therefore, it is likely the incidence of ACS is significantly higher than 1% in patients with greater than 50% TBSA burns.

Previous studies of ACS in burn victims report a total of four patients undergoing abdominal decompression for ACS. Of these, only two are reported to have survived.^{6,7} This is far too small a sample size to determine whether the 50% mortality is a true indication of the mortality expected of all burned patients undergoing abdominal decompressive procedures for ACS. A study of trauma patients with ACS without intra-abdominal injury documented a 50% survival rate after abdominal decompression.¹² This is consistent with our findings of a 40% survival rate for patients who underwent urgent abdominal decompression. Although 40% survival seems low, it represents significant improvement over the suggested 100% mortality of this syndrome if left untreated.⁵ Nonetheless, the syndrome fortunately occurs so infrequently, our total number of subjects, although much larger than in previous studies of burned patients, may still be too small to accurately determine the survival of ACS in the severely burned patient population. The results do suggest, however, that ACS is treatable in the burn patient.

The ACS documented in this study appeared to result from two separate causes. Like most cases of ACS described in the literature, the majority of patients in this study developed excessive third-spacing in the early resuscitative period, presumably because of the initial postinjury inflammatory response. However, several patients survived the initial resuscitation only to develop ACS as late as 95 days into the hospital course. This late-onset ACS was thought to be the result of a secondary inflammatory response triggered by bacteremia and sepsis. Interestingly, this group of patients with late-onset ACS had a higher survival rate than those who developed ACS during the acute resuscitative stage, although the absolute numbers are small. Significantly larger studies will need to be performed to determine whether this apparent difference in survival is real. The reasons for this potentially increased survival are unclear.

Considering the overall 40% survival rate demonstrated in this study, we recommend routine measurements of bladder pressures in all patients with severe burn injuries over a large body surface area. Assessment of intra-abdominal pressure before the physiologic compromise becomes severe may prevent the development of ACS. Early detection and treatment with noninvasive measures is important, because complications such as impaired cardiac output and decreased mesenteric blood flow occur long before the syndrome becomes clinically obvious.^{1,13} Increasing bladder pressures should raise significant concern, and conservative measures should be instituted. These measures can include flushing the bladder catheter to ensure patency, nasogastric tube decom-

pression, bowel care, and escharotomies of any existing circumferential abdominal wounds. Sedation and even pharmacologic paralysis can also be very effective tools for lowering intra-abdominal pressure. However, if these conservative measures are implemented and the patient nonetheless proceeds to develop intra-abdominal pressures greater than 30 mm Hg in conjunction with signs of overt ACS including high peak inspiratory pressures or oliguria refractory to aggressive resuscitation, operative abdominal decompression should be performed immediately. In the burned patient, this can be accomplished by either catheter decompression with a large-bore catheter or by formal laparotomy. Failure of catheter decompression to result in rapid resolution of the signs of physiologic compromise should result in formal laparotomy. The abdomen should be subsequently closed primarily as soon as is possible after the acute episode resolves and the edema reduces significantly. With an increased awareness of the signs of ACS, early conservative treatment of intra-abdominal hypertension, and rapid abdominal decompression when the syndrome manifests itself, clinicians can expect a 40% survival in this group of critically ill, severely injured patients.

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EDITORIAL COMMENT

One of the surgical advances that resulted from World War II was a better understanding of the fluid requirements of burn patients, and as such, the first burn resuscitation formula was widely promulgated.¹ Around the same time, Drs. Lund, Davidson, and Levenson at the Boston City Hospital and Drs. Cope and Moore at the Massachusetts General Hospital, based upon their experience in treating the victims of the Cocoanut Grove Nightclub fire, had defined the magnitude of fluid and electrolyte shifts during the resuscitation period of a thermally injured patient.¹ Since that time we have developed a sophisticated understanding of burn resuscitation. This better understanding of pathophysiology coupled with surgical and technical improvements in caring for massively burned patients has resulted in our ability to successfully manage very large burns in all age groups. The end result of these factors is that we now have severely thermally injured patients surviving not only their resuscitation, but also, ultimately, their hospitalization. This is certainly a major advancement, however this advancement comes at a definite price.

Specifically, regarding resuscitation, severely burned patients are being resuscitated with volumes of fluid proportionate to their burned surface area. This then results in obligatory intercompartmental fluid shifts. Clinical experience demonstrates, in deep large surface area burns, patients frequently require increasing volumes of fluid to maintain the current gold-standard end point of burn resuscitation—urine output. This vicious cycle is what has been termed “fluid creep” by Dr. Pruitt.² At the same time, we have gained a body of information regarding intra-abdominal hypertension and, in its worst form, the abdominal compartment syndrome. There is growing evidence from the non-burn trauma literature about the deleterious effects of acutely raised intra-abdominal pressure.^{3–5} This has recently been infrequently recognized by burn surgeons as a lethal sequelae of burn resuscitation. In fact, in a recent survey of Burn Surgeons, most admit it exists, but only a minority of surgeons are willing to treat it aggressively.⁶

In this issue of *The Journal of Trauma*, Dr. Hobson and colleagues from the University of California at Davis have reported the largest experience of burn patients with intra-abdominal hypertension.⁷ Additionally, to their credit, they have demonstrated that an aggressive surgical approach to lower abdominal pressure can result in survival. The literature, albeit scarce, has suggested that laparotomy in a severely burned patient universally resulted in death.⁸ This concept has not only been challenged by this current report but, in fact, refuted.

Despite this current descriptive study, several questions remain unanswered. To date, no group has prospectively described routine abdominal pressure monitoring of

all patients undergoing burn resuscitation. This would help us understand the incidence of this complication. Specific questions regarding elevated urinary bladder pressures in burn patients also remain to be answered. What pressure should be used to institute both non-operative and operative interventions to reduce pressure? Also, what is the ultimate operative intervention? If laparotomy is the answer, how should the open abdomen be handled and when is it safe to definitely close. These are just a few questions among several others.

Finally, the essence of the problem is if intra-abdominal hypertension can be avoided. This then gets us back to resuscitation regimens. The questions are what, when, and how. What should be the appropriate fluid? When is enough, enough? When should that fluid be changed? Finally, how do we appropriately monitor the resuscitation? Currently, as we resuscitate, usually with crystalloid solutions, there must be some critical value of oncotic pressure that is reached in the intravascular compartment after which continued dilution then results in a more rapid shift of fluid out of the vascular space. This, I believe, is the set-up for compartment syndromes throughout the body; of which, the abdomen is one. If one believes this hypothesis, then we are obligated to change our monitoring of burn resuscitation. Maybe urine output is not the ultimate parameter to follow. Additionally, maybe we should add an "oncometer" to our armamentarium during the resuscitation phase.

We have certainly made significant strides in the management of the severely burned patient since the 1942 Co-coanut Grove fire. However, now we must confront a new myriad of problems for which we need answers. By no means

do I suggest that we return to the days of routine renal failure following burn injury, but I think we must now try to find the answers of some of the questions posed above. I congratulate the authors for adding this important manuscript to the growing body of literature on intraabdominal hypertension in burn patients.

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