Mechanism of Acute Ascites Formation After Trauma Resuscitation

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Background: Severely injured patients have been observed to acutely develop ascites; however, the pathogenesis of this rare phenomenon is poorly understood.

Objectives: To report the factors common among severely injured patients developing ascites and to formulate a hypothesis regarding its origin.

Methods: Retrospective review of case series.

Results: We identified 9 injured patients between January 1, 1993, and December 31, 1998, who acutely developed significant amounts of ascites. The mean ± SD estimated ascites volume was 2.0 ± 0.8 L. All 9 patients had severe shock and were mechanically ventilated before abdominal decompression for the abdominal compartment syndrome. The mean ± SD peak inspiratory pressure was 39.0 ± 5.8 cm H2O. The mean ± SD volumes of crystalloid and blood product infusion before decompression were 16.1 ± 10.2 L and 5.2 ± 4.8 L, respectively, in a mean ± SD of 17 ± 15 hours. In comparison, the mean ± SD volumes of crystalloid and blood product transfusion among 100 contemporary, randomly selected patients undergoing trauma laparotomy were 5.1 ± 5.5 L and 1.1 ± 2.5 L, respectively (P < .001). Eight patients had only extra-abdominal injuries, while 1 patient had a combination of extra- and intra-abdominal injuries. Two patients were found to be cirrhotic by liver biopsy, but the other 7 patients had no known preexisting hepatic disease. Eight patients had absorbable mesh temporary abdominal closure, and 1 patient had primary fascial closure. There was persistent ascitic drainage in 5 patients; however, in all but 1 patient with cirrhosis, the drainage did not persist beyond 3 days. Two patients died, 1 of sepsis and the other of a closed head injury.

Conclusions: Common denominators of posttraumatic ascites include shock, massive fluid resuscitation, and elevated intrathoracic pressure. The rapid onset of ascites in the setting of elevated intrathoracic pressure suggests that the patient’s ability to clear ascitic fluid is overwhelmed.

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In severely injured patients, the development of generalized edema, including visceral edema, is a well-recognized phenomenon. When hemorrhage is controlled and as blood products and crystalloid are being transfused to restore perfusion, a remarkable phase of accumulation of edema in the interstitial space commences.1 The development of ascites following trauma resuscitations is a clinical phenomenon not as well described. Eldor and colleagues2 documented ascites in a 12-year-old girl following bilateral femur fracture fixation during which 15 L of crystalloid was used for resuscitation. Markert et al3 reported 3 cases of subcapsular liver hematoma causing hepatic outflow obstruction and consequent ascites. Three case series4-6 involving 20 patients have described the phenomenon of secondary abdominal compartment syndrome (ACS), in which resuscitated trauma patients develop massive bowel edema with or without ascites. Our goal in this article was to describe the clinical characteristics of 9 severely injured patients who acutely developed ascites during resuscitation from shock. Furthermore, we sought to understand the physiological reasons for the rapid onset of the ascites and its association with intra-abdominal hypertension (IAH).

METHODS

A retrospective review of all patients who underwent a decompressive laparotomy for primary or secondary ACS between January 1, 1993, and December 31, 1998, at our level 1 trauma center was performed. Our objective was to identify cases in which acute onset of ascites was clinically apparent or significant. Operative reports were reviewed for specific mention of ascites and the presence or ab-
sence of visceral edema, retroperitoneal edema or hematoma, and hepatic cirrhosis. We tried to ascertain the volume of ascites discovered in each case by looking for a specific measurement recorded by collection of the fluid in a suction canister or by a gross estimate of the surgeon. Further information obtained from the medical record and the trauma registry database included age, sex, trauma type, injury types and locations, presence or absence of shock, time interval from injury to abdominal decompression, crystalloid volume infusion, blood product transfusion data, presence of preexisting hepatic disease, Injury Severity Score, and Abdominal Trauma Index.7

Ascites was defined as the presence of significant amounts of intraperitoneal serous fluid. Serosanguinous fluid or cloudy fluid was also allowed. Intra-abdominal hypertension was defined as a clinically evident tight or tense abdomen or a urinary bladder pressure of 25 mm Hg or 30 cm H2O or urine.6 Abdominal compartment syndrome was defined as 1 or more signs of organ failure in the setting of IAH in which abdominal decompression resulted in clinical improvement.8 Shock was defined as clinically evident poor perfusion, systemic acidosis, or persistent hypotension. Measurements of urinary bladder pressure, systolic blood pressure, peak inspiratory pressure, central venous pressure, and pH were obtained before and after decompression, if available, and compared by means of the paired samples t test. Mortality was defined as death during admission or within 30 days. The results are presented as mean ± SD.

To compare the volumes of crystalloid and blood product transfusion in patients who developed significant ascites with the volumes used in the more typical patient with a trauma laparotomy, we randomly selected 100 contemporary patients from our trauma registry who had a trauma laparotomy. Using the Wilcoxon rank sum test, the mean volumes of crystalloid and blood product transfusion before laparotomy were compared with those of the patients who developed ascites. This test was chosen because of the small number of patients in the ascites group.

The policy on our trauma service during this period was to aggressively resuscitate patients with clinical or laboratory evidence of shock with successive liters of crystalloid and to use blood products to obtain a goal hematocrit of 30% and normal coagulation factors. We routinely used 5 to 10 cm of positive end-expiratory pressure in patients on mechanical ventilation.

RESULTS

Table 1. Physiological Factors Before and After Decompression

<table>
<thead>
<tr>
<th>Measure</th>
<th>Before Value</th>
<th>No. of Patients</th>
<th>After Value</th>
<th>No. of Patients</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Urinary bladder pressure, cm H2O/urine</td>
<td>36 ± 5</td>
<td>4</td>
<td>8 ± 1</td>
<td>2</td>
<td>Not applicable</td>
</tr>
<tr>
<td>Systolic blood pressure, mm Hg</td>
<td>100 ± 26</td>
<td>9</td>
<td>125 ± 24</td>
<td>9</td>
<td>.04</td>
</tr>
<tr>
<td>Peak inspiratory pressure, cm H2O</td>
<td>38.0 ± 5.8</td>
<td>9</td>
<td>30.0 ± 7.6</td>
<td>8</td>
<td>.02</td>
</tr>
<tr>
<td>Central venous pressure, mm Hg</td>
<td>22.0 ± 5.3</td>
<td>9</td>
<td>14.0 ± 5.3</td>
<td>9</td>
<td>.01</td>
</tr>
<tr>
<td>pH</td>
<td>7.14 ± 0.11</td>
<td>9</td>
<td>7.26 ± 0.12</td>
<td>8</td>
<td>.01</td>
</tr>
</tbody>
</table>

*Values are given as mean ± SD.

There were 9 patients in whom ascites was found at laparotomy for abdominal decompression. There were 6 men and 3 women, with a mean age of 47 years (range, 20–57 years). The cause of injury was motor vehicle crash (4 patients), pedestrian struck by an automobile (2 patients), motorcycle crash (1 patient), and extra-abdominal gunshot wound (2 patients). The mean Injury Severity Score was 24.0 ± 8.5, and the mean Abdominal Trauma Index was 6.0 ± 9.9. Injuries included extremity fractures in 6 patients, head injury in 4, rib fractures in 4, pelvic fracture in 2, chest gunshot in 1, shoulder gunshot in 1, and small-bowel mesentery tear in 1. The single patient with intra-abdominal injury had a small-bowel resection for a lacerated mesentery shortly after the injury. He developed postoperative ACS 55 hours later, with clear ascites and intact anastomotic staple lines found on reopening of his fascial closure. This was the only patient who developed ascites following a laparotomy; the other 8 patients had not had a previous trauma laparotomy. All 9 patients had clinical or laboratory evidence of shock and were mechanically ventilated.

Six patients had abdominal decompression in the operating room and 3 patients had abdominal decompression in the intensive care unit. One of these had an unsuccessful attempt at percutaneous drainage of ascites with a diagnostic peritoneal lavage kit before laparotomy. The surgeon (J.C.M.) suspected significant amounts of ascites and was trying to avoid laparotomy. Although this patient had elevated urinary bladder pressure, the ascites would not drain through the tubing. The mean time from injury to abdominal decompression was 17 ± 15 hours (range, 4–44 hours). All 9 patients had immediate improvement in pulmonary or cardiovascular factors following decompression (Table 1). The mean volumes of crystalloid and blood product transfusion before decompression were 16.1 ± 10.2 L (range, 6.2–37.0 L) and 5.2 ± 4.8 L (range, 0.4–17.4 L), respectively. In comparison, the mean volumes of crystalloid and blood product resuscitation of the 100 contemporary, randomly selected trauma patients who had laparotomy at our center but who did not have ascites were 5.1 ± 5.5 L (range, 0.2–44.0 L) and 1.1 ± 2.5 L (range, 0–15.8 L), respectively (P < .001 for both).

The mean volume of estimated ascites was 2.0 ± 0.8 L (Table 2). In 6 patients, the ascites was described as “serous” or “clear,” in 2 patients the ascites was “cloudy” or “slightly cloudy,” and in 1 patient it was “blood-tinged.” Six patients had visceral edema and 2 did not, while in 1 patient who had 3.0 L of ascites, there was no mention of the presence or absence of visceral edema. Two of the patients with visceral edema also had retroperitoneal edema or hematoma. The 2 patients without visceral edema were the same 2 patients who had cirrhotic-appearing livers and were proven by biopsy to have pre-existing hepatic cirrhosis secondary to alcohol abuse.
remaining 7 patients had no known history of preexisting hepatic disease and had noncirrhotic-appearing livers on exploration. The mean volume of crystalloid resuscitation in the 7 noncirrhotic patients was 18.6 ± 11.2 L and in the 2 cirrhotic patients was 7.6 ± 2.0 L (P = .8, Wilcoxon rank sum test).

Eight patients had absorbable mesh placed because further ascites or edema drainage was expected or because their abdomens were too tight to close. There was persistent ascitic drainage following absorbable mesh closure in 5 patients. In all but 1 patient with hepatic cirrhosis, the drainage did not persist beyond 3 days. This patient ultimately died of sepsis and pulmonary failure (see end of the “Results” section). The 6 patients with absorbable mesh closure who survived required a mean of 3 operations for ultimate abdominal wall closure in a mean of 10 days. Three patients had secondary fascial closure, and 3 required elevation of skin and subcutaneous skin flaps over an abdominal wall defect.8

One of the patients with cirrhosis had a primary fascial closure following her decompressive laparotomy. This patient was a pedestrian struck by an automobile and had extremity and rib fractures. She was initially hypotensive and received 2.0 L of crystalloid in the emergency department. An initial abdominal computed tomographic scan showed a small amount of fluid in her pelvis. She had an extensive extremity operation during which she received 4.0 L of crystalloid and 1.0 U of blood. She was initially intubated, but developed hypotension and respiratory distress and was noted to have a tensely distended abdomen. She received another 2.0 L of crystalloid before abdominal decompression in which 2.0 L of ascites and a cirrhotic liver were found. The surgeon removed the ascites, closed the fascia, and allowed the patient to be extubated immediately after surgery. She had no further abdominal complications.

Two patients died; the causes of death were pulmonary failure and sepsis (1 patient) and severe closed head injury (1 patient). The patient who died of pulmonary failure and sepsis had preexisting hepatic cirrhosis and persistent ascitic drainage after decompression until the time of death 6 weeks following the injury.

COMMENT

These 9 patients represent a distinct subset of the population of severely injured patients. They are unusual because they experienced the rapid onset of ascites during their initial resuscitation and because most had injuries remote from their abdomen. The clinical courses of these patients are similar to what other clinicians have described. Investigators have observed that severe hemorrhagic shock was the basic factor setting the stage for this scenario and emphasized the presence of an ischemia-reperfusion injury as the primary cause.4,6 Eldor et al6 concluded that ascites developed because of “overtransfusion.” Therefore, some investigators ascribe the ascites to the need for aggressive resuscitation of hypovolemia, while 1 author suggests that the ascites is a complication of overaggressive resuscitation. In our patients, the onset of ascites was associated with the presence of shock and the infusion of extraordinarily large volumes of balanced electrolyte solutions and blood products. We believe that the profound nature of these patients’ shock required such aggressive fluid resuscitation; however, we remain open to the possibility that a more directed resuscitation with slower infusion rates may be a method for avoiding this scenario.

The mechanism of ascites formation has been extensively studied.10-13 In patients with cirrhosis of the liver, intrathoracic fibrosis leads to the obstruction of blood flowing into the hepatic veins and to hepatic sinusoidal hypertension. The basement membrane of the hepatic sinusoids is highly permeable; thus, fluid can be forced into the interstitium of the liver and across the hepatic capsule to form ascites. However, most patients in this series did not have cirrhosis. Human and animal investigations indicate that, in addition to the liver, edematous bowel may be a source of ascites in these patients. Interstitial edema in patients resuscitated from shock results from the sequestration of fluid in the interstitial space of injured and noninjured tissue.1 The liters of crystalloid infused into the patient dilute the plasma protein concentrations, and the resultant fall in intravascular oncotic pressure drives water into the interstitium. Visceral ischemia and reperfusion injury with resultant edema have an additive effect.4 It is not unreasonable, therefore, to expect that a portion of this accumulating edema fluid could travel across the peritoneal surface of the liver and the bowel. This possibility is supported by animal studies14,15 in which the combination of splanchic venous congestion produced by portal vein ligation and hypoproteinemia produced by plasmapheresis readily produces ascites. Zarins et al16 produced massive ascites in baboons by reducing the plasma oncotic pressure by 76% by plasmapheresis alone.

An additional factor that may have contributed to the rapid onset of ascites in these patients is that they were intubated with positive pressure ventilation and had elevated intrathoracic pressure. Elevated intrathoracic pressure could have several physiological effects.17 An increase in the right atrial pressure could be transmitted retrograde and could increase pressure in the hepatic sinusoids. Increased hepatic hydrostatic pressure from occlusion of the vena cava above the liver readily produces ascites in dogs.18 The decreased oncotic pressure in the

Table 2. Patient Characteristics

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>Ascites Volume, L</th>
<th>Presence of Edema or Hematoma</th>
<th>Preexisting Hepatic Disease</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>1.0†</td>
<td>None</td>
<td>Cirrhotic</td>
</tr>
<tr>
<td>2</td>
<td>1.5†</td>
<td>Small-bowel edema</td>
<td>None</td>
</tr>
<tr>
<td>3</td>
<td>2.0†</td>
<td>None</td>
<td>Cirrhotic</td>
</tr>
<tr>
<td>4</td>
<td>3.0†</td>
<td>Small-bowel edema</td>
<td>None</td>
</tr>
<tr>
<td>5</td>
<td>1.0†</td>
<td>Small-bowel edema</td>
<td>None</td>
</tr>
<tr>
<td>6</td>
<td>2.0†</td>
<td>Small-bowel edema</td>
<td>None</td>
</tr>
<tr>
<td>7</td>
<td>1.5†</td>
<td>Small-bowel edema</td>
<td>None</td>
</tr>
<tr>
<td>8</td>
<td>3.0†</td>
<td>Not mentioned</td>
<td>None</td>
</tr>
<tr>
<td>9</td>
<td>2.8†</td>
<td>Small-bowel edema</td>
<td>None</td>
</tr>
<tr>
<td>10</td>
<td>2.0†</td>
<td>Retroperitoneal hematoma</td>
<td>None</td>
</tr>
<tr>
<td>11</td>
<td>1.5†</td>
<td>Retroperitoneal edema</td>
<td>None</td>
</tr>
</tbody>
</table>

*Measured.†Estimated.
sinusoids from the crystalloid resuscitation, combined with increased hydrostatic pressure, would readily favor ascites formation. Elevated intrathoracic pressure could also impede the return of visceral lymphatic fluid via thoracic pathways to the thoracic duct and the migration of ascites through tiny diaphragmatic fenestrae into the pleural space. The rapid onset of ascites in some severely injured patients may thus overwhelm the normal mechanisms for clearance of intraperitoneal fluid.

Optimal management of the patient with posttraumatic ascites is not clear. In this series, ascites was associated with the development of ACS, but because most of the patients also had bowel edema, the exact contribution that the ascites made to the IAH is uncertain. Although in this series the ACS was treated by decompressive laparotomy, an attempt at decompression of the ascitic fluid using a peritoneal catheter is an attractive concept. Unfortunately, our single attempt at percutaneous drainage was unsuccessful. Another temporary measure for reducing IAP is pharmacologic paralysis to relax the abdominal musculature. Decompressive laparotomy is effective at relieving IAH but commits the patient to a series of abdominal operations to achieve definitive closure. Following decompression, temporary abdominal closure with polyglycolic acid mesh (Dexon; Davis and Geck, Inc, Danbury, Conn) was our treatment of choice. In most patients, persistent ascitic drainage through the mesh was only a temporary problem.

In 1 patient with cirrhosis, however, persistent fluid migration through the mesh was problematic, preventing definitive abdominal wall closure and probably contributing to his death. The other patient with cirrhosis had a successful primary fascial closure following her decompression. Two factors likely contributed to the success of her primary fascial closure: her rapid recovery from only mild shock and her immediate postoperative extubation. The normalization of intrathoracic pressure promoted by the removal of positive pressure ventilation and the lack of necessity of further fluid resuscitation in this patient decreased the impetus for ascites reaccumulation. We conclude that open abdomen management of cirrhotic patients may carry a high mortality and that the optimal management is unclear.

CONCLUSIONS

Common denominators in the development of posttraumatic ascites include shock, massive fluid resuscitation, and high-pressure mechanical ventilation. Edematous liver and bowel associated with the massive resuscitation are likely origins of the fluid. Increased intrathoracic pressure associated with mechanical ventilation impedes ascitic clearance into the thoracic cavity. Hepatic cirrhosis is not necessary for the development of this phenomenon. Surgeons should recognize that even in the absence of abdominal injury, if blood loss and shock are severe and massive resuscitation is required to restore hemodynamic stability, the patient is at risk of developing a potentially lethal complication within the abdomen. Although further research may reveal methods for more directed fluid resuscitation, monitoring these patients for signs of IAH is prudent so that timely decompression of the ascites can be accomplished. If the IAH is relieved and the sequence of events that led to the shock are reversed, recovery can be expected.

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REFERENCES