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Abdominal Compartment Syndrome in Burn Patients: Not Always a Consequence of Excess Fluid Resuscitation

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Background	A patient with burns to extremities with abdominal compartment syndrome.
Summary	A 19-year-old female presented with 25% total body surface area (TBSA) burns and grade I inhalation injury injury. The patient was intubated for airway protection and fluid resuscitation, and her wounds were dressed with silver sulfadiazine. The following morning, labs were concerning for a leukopenia thought to be secondary to silver sulfadiazine; the patient's wounds were redressed with mafenide acetate. That evening, the patient had a worsening metabolic acidosis and decreased urine output that was intermittently responsive to intravenous fluid boluses; however, her blood pressure progressively worsened, requiring vasopressor support. Despite the additional fluid, her total fluid requirement was appropriate at 0.14 liters per kilogram (L/kg). Mid-morning, her exam was concerning for a tense abdomen; upon further evaluation, her peak airway pressure was 45, and bladder pressure was 35. A diagnosis of abdominal compartment syndrome was made, and she was taken to the operating room (OR) for decompressive laparotomy. Intraoperative findings were notable for ischemia of an isolated segment of her colon, and a right colectomy was performed. It is important to note that severe burns can cause transient splanchnic vasoconstriction. If the gastrointestinal tract cannot compensate for periods of decreased blood flow, either through pressure autoregulation or increased oxygen extraction, ischemic injury will occur. If unrecognized, ischemic bowel can lead to abdominal compartment syndrome and end organ failure. The important takeaway is that this burn patient did not have a circumferential abdominal burn; however, she did develop abdominal compartment syndrome related to ischemic bowel, most likely secondary to splanchnic vasoconstriction exacerbated by vasopressor.
Conclusion	Severe burns affect every organ system, and the physiologic insult results in transient vasoconstriction that can lead to nonocclusive mesenteric ischemia, which, if there is a delay in diagnosis, can lead to secondary abdominal compartment syndrome.
Key Words	abdominal compartment syndrome; burn injury; intestinal ischemia
Abbreviations	TBSA: Total body surface area, MAP: Mean arterial pressure, IAP: Intraabdominal pressure IAH: Intraabdominal hypertension

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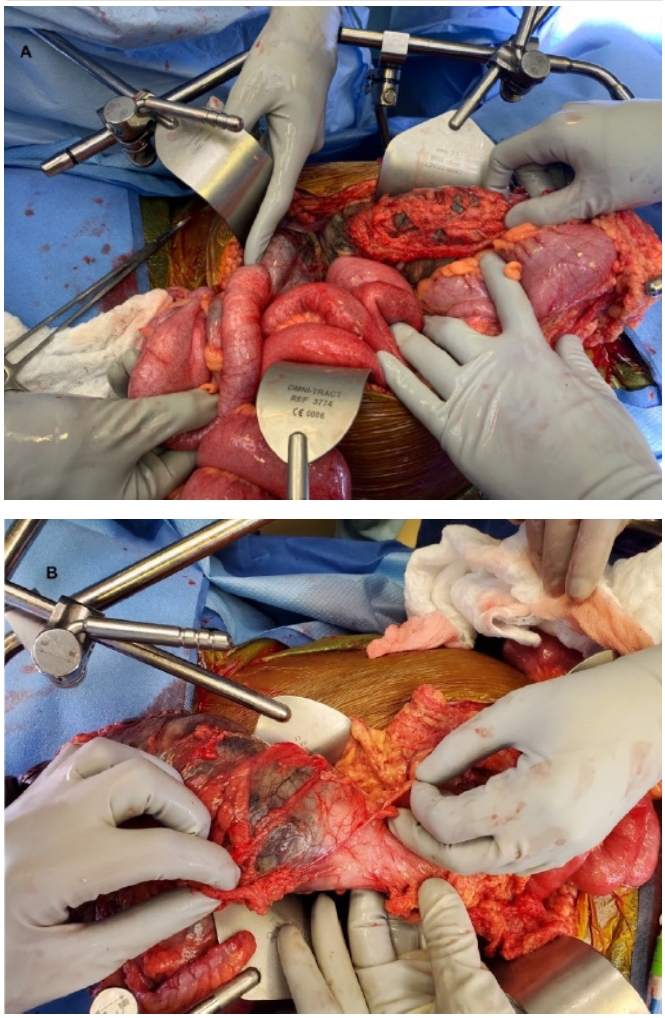
Case Description

A 19-year-old female presented with 25% total body surface area (TBSA) burns and inhalation injury after a home fire. She sustained partial thickness burns to her lower and upper extremities bilaterally. Upon arrival, she was found to have soot in the oropharynx and burns around the mouth. She was thus urgently intubated for airway protection; bronchoscopy was notable for grade I injury. Her wounds were debrided bedside and dressed with silver sulfadiazine. Fluid resuscitation was initiated using the modified Brooke formula, and adjustments were made based on her physiologic response. Judicious use of volume was used while following multiple end points, namely urine output and mean arterial pressure (MAP). In the first 24 hours, she received 10 liters of fluid (a combination of two-thirds crystalloid and one-third colloid). This patient was not over-resuscitated based on her weight and hourly tracked urine output. The following morning, labs were concerning for a leukopenia thought to be secondary to silver sulfadiazine, and her wounds were re-dressed with mafenide acetate. However, that afternoon, she experienced worsening metabolic acidosis, hypotension, and oliguria. The metabolic acidosis was attributed to the topical antimicrobial agents used for wound dressing. The oliguria and hypotension were addressed with additional fluid. Her urine output was intermittently responsive to fluid boluses; however, she required vasopressors that evening to maintain a MAP >65 mm Hg.

The following morning, her exam revealed a tense abdomen. Upon further evaluation, her peak airway pressure was 45 mm Hg, and her bladder pressure was 35 mm Hg. X-ray imaging revealed intestinal pneumatosis suggesting advanced bowel ischemia.

A diagnosis of abdominal compartment syndrome was made, and the patient was taken emergently to the OR for a decompressive laparotomy. The procedure revealed a region of ischemic right colon surrounded by hyperemic colon (Figure 1). A right hemicolectomy was performed, and the patient was left in discontinuity with a temporary abdominal closure. Twenty-four hours later, she was appropriately resuscitated and no longer required vasopressor support. She was taken back to the OR for a second look when all bowel was found to be viable without further evidence of ischemia.

Figure 1. Intraoperative Images of Right Colon Confirm Suspicions of Ischemic Bowel. Published with Permission



A) Dark, necrotic bowel is surrounded by hyperemic colon; and B) a clear demarcation of the border of ischemic colon is seen.

No anastomosis was performed due to the extensive burn surgeries and ensuing fluid shifts expected over the coming days. An end ileostomy with mucous fistula was created, and the abdomen was closed. Upon further diagnostic review, the patient had no source of emboli on imaging, and the specimen pathology showed no evidence of an embolic event. The patient returned to the OR in the following days for serial burn debridement and cadaveric skin allografts, which were later replaced with autografts. The remainder of her course was uneventful, and she was discharged home. After six months, the patient underwent an elective reversal of the stoma without complication.

Discussion

Abdominal compartment syndrome is a syndrome characterized by a new organ failure resulting from a sustained intraabdominal pressure (IAP) of >20 mm Hg. Typically, abdominal compartment syndrome in a burn patient results from either decreased abdominal wall compliance caused by circumferential torso burns or excess fluid during large-volume resuscitation (exceeding 0.3 liters per kilogram in the first 24 hours),[1] leading to visceral edema. However, this patient had no abdominal burns and was not over resuscitated (the patient only received 0.14 L/kg of fluids). Thus, given the early signs of compromised bowel, we propose that the abdominal compartment syndrome in this patient was secondary to ischemic bowel.

Severe burns affect every organ system. In the first 24 to 72 hours, both increased vascular permeability and transient splanchnic vasoconstriction occur. Vascular permeability leads to intravascular volume depletion and edema formation. In murine studies, transient splanchnic vasoconstriction has been shown to reduce superior mesenteric artery blood flow to 30% of baseline.[2] If the gastrointestinal tract is unable to compensate for periods of decreased blood flow, either through pressure autoregulation or increased oxygen extraction, ischemic injury will occur; watershed areas are particularly sensitive to ischemia.[2]

Consistent with what is reported in the literature, it was concluded that the etiology was localized nonocclusive mesenteric ischemia, likely a product of the splanchnic vasoconstriction (a combination of burn physiology and vasopressor effects) in combination with a compromised ability to adequately compensate for the reduced blood flow to the abdomen due to inhalation injury.[1,3] Leukopenia and lactic acidosis were initially attributed to the topical antimicrobial agents used in the wound dressings. However, it is more likely that the observed leukopenia was an early sign of ischemia and that the metabolic acidosis was a result of the compromised bowel. Intraabdominal hypertension (IAH) and worsening oliguria observed were secondary to the ACS the patient developed. It is possible an earlier diagnosis could have been made had a broader differential been kept in mind.

Conclusion

Severe burns have significant pathophysiological effects on all organs. Specifically, intestinal vasoconstriction can be difficult to compensate for with additional inhalation injury. If unrecognized, ischemic bowel can lead to abdominal compartment syndrome, end organ failure, sepsis, and death. This case report illustrated the interaction of volume status, shock, splanchnic vasoconstriction, non-occlusive mesenteric ischemia, and abdominal compartment syndrome.

Lessons Learned

As always in burn patients, it is critical to closely monitor the use of fluid resuscitation because over-resuscitation is one of the most common causes of abdominal compartment syndrome in burn patients. Another way to avoid this outcome is to have a nursing protocol in place for continuous bladder pressure monitoring in all major burn patients. Thus, the clinician will be alerted to this diagnosis before end organ damage occurs. Maintaining a broad differential diagnosis of burn patients whose clinical response deviates from the expected course is also critical. As made clear by this case, a burn patient can develop abdominal compartment syndrome secondary to bowel ischemia, even without excessive fluid resuscitation or circumferential abdominal burns.

References

1. Nielson CB, Duethman NC, Howard JM, Moncure M, Wood JG. Burns: Pathophysiology of Systemic Complications and Current Management. *J Burn Care Res*. 2017;38(1):e469-e481. doi:10.1097/BCR.0000000000000355
2. Tokyay R, Zeigler ST, Traber DL, et al. Postburn gastrointestinal vasoconstriction increases bacterial and endotoxin translocation. *J Appl Physiol (1985)*. 1993;74(4):1521-1527. doi:10.1152/jappl.1993.74.4.1521
3. Muschitz GK, Fochtmann A, Keck M, et al. Non-occlusive mesenteric ischaemia: the prevalent cause of gastrointestinal infarction in patients with severe burn injuries. *Injury*. 2015;46(1):124-130. doi:10.1016/j.injury.2014.08.035