

Challenges of Transport and Resuscitation of a Patient With Severe Acidosis and Hypothermia in Afghanistan

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ABSTRACT

We present the case of a patient with new-onset diabetes, severe acidosis, hypothermia, and shock who presented to a Role 1 Battalion Aid Station (BAS) in Afghanistan. The case is unique because the patient made a rapid and full recovery without needing hemodialysis. We review the literature to explain how such a rapid recovery is possible and propose that hypothermia in the setting of his severe acidosis was protective.

KEYWORDS: *new-onset diabetes; severe acidosis; hypothermia; shock; hemodialysis*

Introduction

We present the case of a patient with new-onset diabetes, severe acidosis, hypothermia, and shock who presented to a Role 1 BAS in Afghanistan. The case is unique in that, despite arterial pH of 6.681 and base deficit of -30mEq, core temperature of 31°C (88°F), and hypotension requiring vasopressors, the patient made a rapid and full recovery without needing hemodialysis.

We review the literature to explain how such a rapid recovery is possible and propose that hypothermia in the setting of his severe acidosis was protective. The challenges unique to expeditionary and en route care of this critically ill patient are presented, including a discussion of the environmental and nonenvironmental causes of hypothermia, expeditionary re-warming techniques, and iatrogenic hypothermia-avoidance techniques. The lessons learned from the recognition and management of ketoacidosis and hypothermia are reviewed.

Case Presentation

A 38-year-old civilian contractor from Uganda presented to the Role 1 BAS in a remote forward operating base in Afghanistan with a chief complaint of weakness and dizziness on 5 February 2017. He was previously healthy, had no prior surgeries, took no medications, and had no drug allergies. He did not smoke or drink alcohol; he worked as a security advisor.

On review of systems, the patient endorsed 2 weeks of polyuria, polydipsia, decreased appetite, weight loss, fatigue, and

shortness of breath. He denied fevers, cough, or hemoptysis. On examination at the Role 1 facility, he was normothermic (oral temperature, 36.1°C [97°F]), mildly hypertensive (145/90mmHg), and tachypneic (respiratory rate [RR], 30–35/min); his heart rate (HR) was tachycardic (130–140 bpm), his oxygen saturation (Sao₂) level was 97%. He was well developed and well nourished; alert and oriented to self, place, date, and situation; and spoke in complete sentences while in mild respiratory distress. Rales were heard in the left posterior thorax on auscultation. The patient was tachypneic without accessory muscle use; heart rhythm was regular and rate was tachycardic without abnormal heart sounds; skin was warm and dry without rash; and the remainder of his examination was unremarkable.

Telemetry demonstrated sinus tachycardia. Point-of-care malaria and HIV tests were negative. Urinalysis was positive for ketones and glucose. No glucometer or other studies were available. Sepsis from pneumonia was suspected, given the patient met systemic inflammatory response syndrome criteria (HR, >90 bpm; RR, >20/min) and presented with primarily pulmonary signs and symptoms (i.e., dyspnea and tachypnea). A 1L intravenous (IV) bolus of normal saline (NS) was given, as well as 1g of ceftriaxone IV.

The Role 1 facility requested urgent, nonsurgical medical evacuation (medevac) of the patient to a Role 3 facility. Awaiting medevac, the patient received 2L of NS. The casualty evacuation, or dustoff, crew arrived at approximately 21:30 local time, and the patient was prepared for transport in a hypothermia prevention/management kit (HPMK) in stable condition. The planned medevac route was expected to take 90 minutes to the Role 3 facility and included a tail-to-tail handoff at a Role 2 facility, where a forward surgical team (FST) was located.

At the Role 2 facility, the dustoff crew was unable to proceed with the tail-to-tail handoff because of operational security and returned to the Role 1 facility at 2330 local time. The patient worsened over the next hours and received 750mg of levofloxacin IV. Although he was normotensive with stable oxygen saturation, the patient had become obtunded, his gag reflex was absent, and his work of breathing had increased.

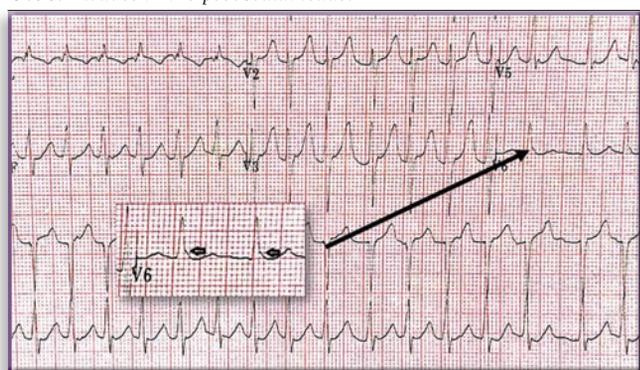
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The decision was made to intubate, using etomidate for induction and succinylcholine for paralysis. Despite three attempts to intubate with direct laryngoscopy, the vocal cords were not well visualized. The fourth attempt with a bougie adjunct was successful, and the patient proceeded at 07:00 local time with the Dustoff crew to an alternate Role 2 facility, where a tail-to-tail handoff was planned. A mechanical malfunction of the patient's endotracheal tube (ETT) occurred en route to the Role 2 facility; the ETT endcap was dislodged. Dustoff Medics were able to oxygenate the patient through a poorly seated bag-valve-mask on the uncapped ETT, with tape used as a field-expedient connector. The FST performed a tube exchange in one successful attempt. During the procedure, the patient was paralyzed with 10mg of vecuronium IV, sedated with 2mg of versed IV, and given 100mg of ketamine IV. Once the airway and invasive mechanical ventilation were secured, the Dustoff crew proceeded with the patient to the Role 3 facility, arriving at 10:10 local time (almost 12 hours after initial medevac).

In the resuscitation bay at the Role 3 facility, the patient was on a litter in a Blizzard Rescue Blanket (Blizzard Protection Systems, <http://www.blizzardsurvival.com>) without a head cover and lying supine on a cold Ready-Heat Blanket (Tech-Trade, <http://www.ready-heat.com/>). On examination, he was intubated with a 7.0F ETT, 19cm at the teeth. The Glasgow Coma Scale score was 3T; the patient had sluggishly reactive bilateral pupils, was breathing passively on the ventilator set at assist control, fraction of inspired oxygen (FiO₂) was 100%, RR was 10/min, total lung volume was 450mL, and positive end-expiratory pressure (PEEP) was 5cmH₂O. Other invasive devices included two peripheral intravenous lines, an oral-gastric tube (OGT), and a Foley catheter drain containing 150mL of light yellow urine. The patient's initial rectal temperature was 31°C (88°F), blood pressure was 153/94mmHg (mean arterial pressure [MAP], 92mmHg), HR was 165 bpm, RR was 10/min, and SaO₂ was 99% on 100% FiO₂. His examination was remarkable for tachycardia, obtundation, paralysis, and cold extremities. His 12-lead electrocardiogram demonstrated atrial fibrillation with a ventricular rate of 161 bpm and prominent Osborn waves in the precordial leads (Figure 1).

FIGURE 1 Twelve-lead electrocardiogram demonstrating atrial fibrillation with a ventricular rate of 161 bpm and prominent Osborn waves in the precordial leads.



The Role 3 team recognized the patient's extremis, and re-warming measures were initiated at time 0 minutes (T+0). The patient received a 500mL IV bolus of warmed NS, a Bair Hugger (3M, <https://www.bairhugger.com>) was applied, and his head was covered. His initial blood glucose level was 552mg/dL. In the first 30 minutes, fentanyl 50µg, midazolam, 2mg, diltiazem 10mg, regular insulin 10 units IV, and the remainder

of the warmed 1L NS bolus were given. The patient's HR responded to these interventions. Telemetry showed normal sinus rhythm of 95 bpm and MAP of 75mmHg; however, within 5 minutes, his rhythm reverted to atrial fibrillation at a rate of 130 bpm. An additional diltiazem 10mg IV bolus resulted in normal sinus rhythm at 75 bpm and MAP of 60mmHg.

Initial arterial blood gas (ABG) sample results were as follows: pH, 6.681; partial pressure of carbon dioxide (PCO₂), 42mmHg; partial pressure of oxygen (PO₂), 145mmHg; base deficit, -30mEq; sodium (Na), 132mEq/L; potassium (K), 6.4mEq/L; chloride (Cl), 111mEq/L; bicarbonate (HCO₃⁻), <5mEq/L, ionized calcium (iCa), 1.06mmol/L, hemoglobin, 11.9g/dL; and lactate, 2.8mmol/L, consistent with severe anion-gap metabolic acidosis (AGMA), respiratory acidosis, and nonanion-gap metabolic acidosis (NAGMA; Table 1). Initial diagnoses were moderately severe hypothermia, diabetic ketoacidosis (DKA), and atrial fibrillation with rapid ventricular response complicated by hypovolemic shock. The renal panel at T+40 minutes confirmed mild acute kidney injury (blood urea nitrogen [BUN]/creatinine [Cr] ratio, 161.3), hyperkalemia, and hyperchloremic acidosis.

TABLE 1 Interpretation of Acid-Base Disorders With Brief Discussion of Arterial Blood Gas Analysis

Arterial blood gas values	pH, 6.682; PCO ₂ , 42.3mmHg; PO ₂ , 145mmHg; base excess, -30mEq; SaO ₂ , 94%
Basic metabolic panel	Na, 132mEq/L; K, 6.4mEq/L; Cl, 110mEq/L; HCO ₃ ⁻ , <5mEq/L; BUN, 16mg/dL; Cr, 1.3mg/dL; blood glucose, 552mg/dL; anion gap, 22mEq/L; lactate, 2.8mmol/L
Step 1: Determine the primary acid-base disorder	The patient has a primary metabolic acidosis, indicated by the low pH and low HCO ₃ ⁻ level. The patient's acidosis is an AGMA, because the anion gap is 22mEq/L (normal, 12mEq/L). Given the mildly elevated lactate level, the cause of the patient's AGMA is ketoacidosis.
Step 2: Is the compensation for the primary disorder appropriate?	Because the patient's primary disorder is AGMA, the appropriate compensation is respiratory alkalosis. To determine the expected PCO ₂ , use the formula PCO ₂ = 1.5 × HCO ₃ ⁻ + 8. In our patient, the expected PCO ₂ for appropriately compensated AGMA is 8mmHg. His PCO ₂ is 42mmHg, which means, in addition to AGMA, he has severe respiratory acidosis that likely is iatrogenic, because he was paralyzed and, without any respiratory drive, his minute ventilation was fully ventilator dependent.
Step 3: Is there a Δ gap?	Because the patient has AGMA, it is necessary to rule out a mixed acid-base disorder such as metabolic alkalosis or NAGMA. First, determine the ΔAG, then determine the ΔHCO ₃ ⁻ . The ΔAG (aka, excess AG) is the patient's actual AG minus the normal AG (22 - 12 = 10). The ΔHCO ₃ ⁻ (aka, HCO ₃ ⁻ deficit) is the normal HCO ₃ ⁻ minus the measured HCO ₃ ⁻ (24 - 0 = 24). The ratio is 10/24 = 0.41. This indicates the patient has a "hidden" NAGMA in addition to a primary AGMA and a severe respiratory acidosis. An alternate, simpler calculation to determine the Δ gap is Na - Cl - 36. If the result is < -6, NAGMA exists. For the listed values, Δ gap calculated by this simpler equation is 132 - 110 - 36 = -14.

Δ, change in; AG, anion gap; AGMA, anion-gap metabolic acidosis; BUN, blood urea nitrogen; Cr, creatinine; HCO₃⁻, bicarbonate; NAGMA, nonanion-gap metabolic acidosis; PCO₂, partial pressure of carbon dioxide; PO₂, partial pressure of oxygen; SaO₂, oxygen saturation.

A portable chest radiograph showed the ETT was in the trachea approximately 10cm above the carina with no evidence of acute cardiopulmonary abnormalities. The ventilator respiratory rate was increased to 18/min to reduce the respiratory acidosis. Deep tracheal suctioning was performed and the ETT was advanced 2cm. During this intervention, telemetry showed a 6-beat run of ventricular tachycardia then an 8-beat run followed by sinus bradycardia with ventricular rate 51 bpm. The crash cart was opened. An IV push of 1g of calcium chloride and sodium bicarbonate 50mEq was given and the patient's hemodynamics stabilized. With a MAP of 60mmHg and HR of 106 bpm, the patient's repeated 12-lead electrocardiogram showed sinus tachycardia.

Once cardiac arrest was averted, a 7.5F, multilumen, central venous catheter was placed in the patient's left internal jugular vein, and a left femoral arterial line was placed. Repeated rectal temperature was 31.2°C despite warmed IV fluid by Belmont infuser at 50mL/min. Additional rewarming measures were initiated: The environmental controls in the resuscitation bay were increased to maximum (31°C), sterile water lavage was performed via the OGT, the urinary bladder was irrigated with 300mL of warmed sterile water, and warmed air via a heat-moisture exchanger was applied to the bronchial tree. As the patient's temperature approached 34°C, his MAP decreased to 55–60mmHg, responding well to norepinephrine bitartrate IV infusion. Repeated laboratory test results were improved: pH, 7.0; HCO₃⁻, 4.9 mEq/L; K, 5.2mEq/L; blood glucose, 457mg/dL. Severe acidosis persisted; therefore, the patient was given additional IV therapy with a sodium bicarbonate 100mEq bolus, insulin infusion at 0.15 unit/kg/h, and sodium bicarbonate infusion (D5W with 150mEq/L sodium bicarbonate at 100mL/h).

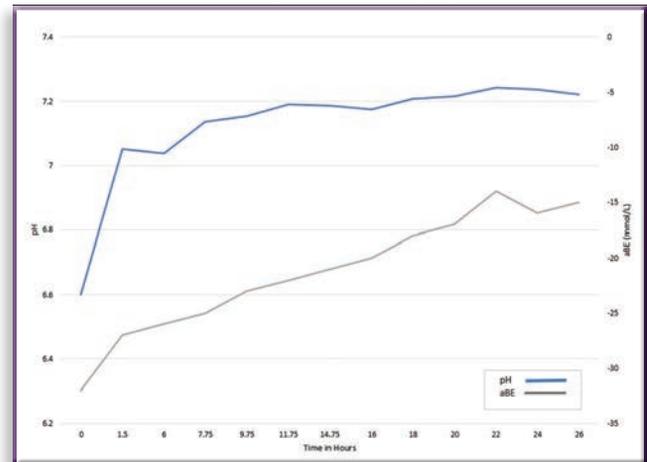
After 4 hours in the resuscitation bay, a total of 8L of warmed NS, bladder and gastric irrigation, and multiple external rewarming interventions, the patient's rectal temperature was 34.8°C (94.6°F) and his neurologic examination improved. The patient opened his eyes spontaneously, tracked, and reached for his ETT to self-extubate. A bolus of fentanyl, a propofol infusion, and wrist restraints permitted safe transport to computed tomography (CT) for whole-body imaging. CT scan of the brain and CT angiography of the chest showed no acute intracranial findings and no pulmonary embolus, respectively. Significant findings included nonspecific peribronchial edema, mesenteric edema, intra-abdominal ascites, peripancreatic edema, gallbladder wall edema, and periportal edema—all consistent with shock bowel, shock pancreas, and systemic inflammatory response. Similar CT findings are seen when a large amount of chloride-liberal IV crystalloid fluids are given during resuscitative efforts.

The patient was transported to the intensive care unit (ICU) at approximately 14:00 local time for further management of DKA with associated mixed acid-base disorder and ventilator-dependent respiratory failure. Insulin and bicarbonate drips were continued. Blood glucose level was checked each hour and blood gas measurements were obtained every 2 hours per local ICU DKA protocol. The patient's pressor requirement continued in the ICU; however, his norepinephrine bitartrate drip was minimal and titrated down to maintain MAP >65mmHg. Maintenance IV fluids were changed to half-NS. The total IV fluid infusion rate of 400mL/h was subsequently decreased to 250mL/h because of concern for pulmonary

edema secondary to volume overload. The patient tolerated a spontaneous ventilator mode with FiO₂ of 40%, pressure support of 10cmH₂O, and PEEP of 5cmH₂O. He maintained stable Sao₂ at 99%–100%, with RR of 18–20 bpm.

The norepinephrine bitartrate drip was stopped at 01:00, 11 hours after ICU admission. The bicarbonate drip was stopped at 07:00, when arterial pH consistently was >7.1 (Figure 2). Serial laboratory test results were significant for resolution of DKA (blood glucose, 180mg/dL; anion gap, 10mmol/L), resolution of hyperkalemia, and normal iCa. No infectious source of the patient's DKA was identified, either on imaging or by laboratory studies, and antibiotics were discontinued. The patient was transferred; receiving an insulin drip and minimal, invasive mechanical ventilation settings, via air ambulance to definitive care in a neighboring country.

FIGURE 2 Patient's arterial pH and base deficit over time.



He was extubated several days after transfer to the outside hospital and returned to his home for chronic management of diabetes mellitus without the need for hemodialysis or evidence of prolonged morbidity. As of this writing, the patient suffered no neurologic deficit or continued laboratory abnormalities as a result of his critical condition.

Discussion

Hypothermia

Humans maintain a normal temperature between 36.6°C (97.9°F) and 37.7°C (99.9°F). Within this “thermal neutral zone,” the basic metabolic rate maintains core temperature. Clinically significant hypothermia occurs when core temperature drops lower than 35°C (95°F). Mild, moderate, and severe hypothermia result in a continuum of pathophysiology from stage 1 (conscious with shivering) to stage 4 (no obtainable vital signs; Table 2) The patient had a core temperature of 31°C (88°F; moderate hypothermia) and clinical stage 3 hypothermia (i.e., unconscious, not shivering, vital signs present).

Measuring temperature is most often initially performed using an intermediate method such as the sublingual or forehead routes. When a temperature <36.1°C (97.0°F) is obtained by an intermediate route, a core temperature must be obtained, most commonly by distal esophageal or rectal routes. Debate among experts exists regarding measurement methods of core temperature: Rectal and urinary bladder temperatures may be categorized as intermediate routes. Most agree that

TABLE 2 Stages of Hypothermia

Measured Core Temperature	Clinical Assessment
Mild: 32°C–35°C (89.6°F–95°F)	Stage 1: Conscious, shivering
Moderate: 28°C–31.9°C (82.4°F–89.6°F)	Stage 2: Confused, shivering
Severe: <28°C (82.4°F)	Stage 3: Unconscious, not shivering, vital signs present
	Stage 4: Unconscious, vital signs absent

a temperature probe in the distal esophagus or specialized instruments placed in the nasopharyngeal space or snugly against the tympanic membrane can achieve accurate core temperatures. In expeditionary settings, an initial intermediate measurement by forehead Tempa Dot thermometer (3M) or the sublingual route followed by core measurement by the rectal or esophageal route are accepted methods.¹

In contrast to induced hypothermia in patients after cardiac arrest or who are undergoing cardiac bypass, accidental and iatrogenic hypothermia can be life threatening owing to multiple pathophysiologic derangements. Hypothermia, not intended as medical therapy, can be caused by environmental and non-environmental factors. Table 3 lists causes of nonenvironmental hypothermia. In Afghanistan in the winter, as demonstrated in the case reported here, it is challenging to maintain the thermal neutral zone during care in prolonged field settings and en route. The outside temperature on the ground on the day of admission of this patient was –5°C (23°F). Convective cooling was exacerbated during the almost 12-hour combined rotary wing and ground transportation. His prolonged medevac, interrupted by heat-losing interventions such as intubations and medicine administration, ultimately brought his core temperature to 31°C (87.8°F).

TABLE 3 Nonenvironmental Causes of Hypothermia

Cold intravenous fluids
Paralytic medications
Endocrine
Hypothyroidism
Hypopituitarism
Hypoglycemia
Diabetic ketoacidosis
Neurologic
Cerebrovascular accidents
Infections
Antipsychotic medications
Alcohol

Although the patient was normothermic initially at the Role 1 facility, he was near the threshold for hypothermia, likely as a result of his DKA. Furthermore, his ability to shiver was reduced from cold IV fluids and the use of paralytics. Before paralysis, his core temperature likely dropped below the point when shivering was possible: stage 3 hypothermia. In addition, the patient was subjected to nonenvironmental causes of hypothermia. DKA is a known cause of nonenvironmental hypothermia and results from the inability of adenosine triphosphate to use glucose in the patient with highly insulin-resistant DKA. Impaired glucose use leads to a lack of substrate for heat production. In one case series spanning 7 years, DKA was the most common cause of nonenvironmental hypothermia.² Non-environmental causes of hypothermia are important factors to consider when transporting patients in expeditionary settings. The winter environment in Afghanistan and prolonged

medevac times, including tail-to-tail handoffs, as well as iatrogenic interventions such as cold IV fluids and paralytics, and DKA are powerful contributors to hypothermia.

This patient exhibited multiple pathophysiologic manifestations of hypothermia, including life-threatening arrhythmias, electrolyte abnormalities, and decreased oxygen delivery. During the rewarming phase of resuscitation, the patient exhibited rewarming shock, hyperkalemia, and difficult-to-control blood glucose levels.

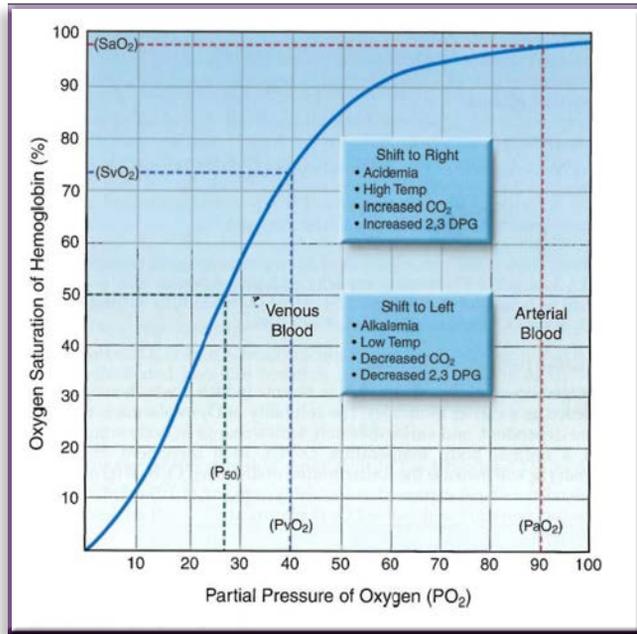
The cardiopulmonary effects of hypothermia vary. Blood pressure can remain stable due to peripheral vasoconstriction shunting intravascular volume to the core. However, hypotension can occur secondary to hypothermia-induced arrhythmias. The most common arrhythmia in hypothermic patients is atrial fibrillation, followed by ventricular tachycardia (including ventricular fibrillation). Bradycardia can be seen. The patient in this report had a combination of these arrhythmias. Rewarming shock, a phrase coined to describe hypotension and hemodynamic instability due to acidosis from oxygen consumption (Vo₂) and oxygen delivery (Do₂) mismatch, was exhibited by our patient. Vo₂/Do₂ mismatch during reperfusion is worsened by shivering, which increases oxygen demand. Reperfusion during rewarming also contributes to a systemic inflammatory response syndrome–induced vasodilatory response. This combination of decreased cardiac output from acidosis in the setting of vasoplegia leads to cardiogenic shock combined with distributive shock.

Hypothermia affects renal and metabolic systems, leading to electrolyte disturbances such as hypokalemia (hypothermia shifts potassium into cells), hypocalcemia, hypomagnesemia, and hypophosphatemia.³ In addition, cold diuresis can worsen hypokalemia. Because our patient had concomitant DKA and acute kidney injury, he was hyperkalemic. Potassium stasis was challenging during his rewarming and resuscitation, and vigilance for hypokalemia was required while he was undergoing insulin infusion therapy.

A left shift of the oxyhemoglobin dissociation curve is caused by hypothermia, whereas a right shift occurs in acidosis from DKA and increased lactate (Figure 3). This discordance likely contributed to our patient’s survival. A left shift leads to decreased oxygen delivery, which is deleterious in the setting of already-hypoxic tissues. Our patient’s pH was 6.681 as a result of ketoacidosis, respiratory acidosis, and chloride-liberal fluid resuscitation, which resulted in a right shift. Just as hypothermia increases hemoglobin’s oxygen affinity (and decreases oxygen delivery), his acidosis decreased hemoglobin’s oxygen affinity (and increased oxygen delivery). This patient had mildly increased lactate level (2.8mmol/L), which also contributed to his acidosis. His base deficit (–30mEq/L) was the lowest the authors have ever calculated. In large case series, when a discordance between initial measured lactic acid and base deficit exists in critical care patients, the base deficit does not predict mortality.⁴

Hypothermia deserves respect when considering use of paralytics. In addition to the reduction in our patient’s ability to maintain core temperature through shivering, paralytics have altered pharmacokinetics and pharmacodynamics in hypothermia. Depolarizing agents such as succinylcholine are more potent and have prolonged duration in hypothermic patients

FIGURE 3 Left shift of the oxyhemoglobin dissociation curve caused by hypothermia, whereas a right shift occurs in acidosis from DKA and increased lactate.



because of slower elimination rates,⁵ making them relatively contraindicated due to prolonged paralysis.

The hematologic system is altered when cold. Coagulation disturbances, including platelet dysfunction and inhibition of the coagulation cascade, are well known to contribute to the lethal triad of hypothermia, coagulopathy, and acidosis.⁶

Rewarming techniques usually start with external methods. Application of the HPMK, Blizzard Heat Blanket, or Ready-Heat Blanket, and warming the resuscitation bay or operating room (temperature >29.5°C–32.2°C [85.1°F–90.0°F]) are recommended. Use of forced-air convective warming devices (e.g., Bair Hugger) can rewarm at 1°C–2.5°C per hour. Though impractical, warm-water immersion can heat the patient by 2°C–4°C per hour. Internal rewarming through humidified inspired air (0.5°C–1.2°C per hour), warmed IV fluids, and body cavity lavage are advised. However, the fastest way to rewarm patients is by intravascular warming through specialized arterial and venous catheters or extracorporeally. Our patient underwent all interventions with the exception of warm-water immersion and extracorporeal rewarming.

Diabetic ketoacidosis

The causes of the patient's severe acidosis (pH, 6.682) were discovered through careful review of his clinical presentation and analysis of the calculations for mixed acid-base disorders. Although prehospital interventions and ability to work up DKA are limited, his chief complaints of weight loss, polydipsia, polyuria, and tachypnea in the setting of urine ketones and glucose were consistent with DKA. Patients with DKA are often volume depleted, which can mask an underlying pneumonia, with positive findings of pneumonia emerging only after patient resuscitation.⁷ On evaluation at the Role 3 facility, his ketoacidosis from diabetes mellitus was definitively diagnosed. Commonly, patients with DKA are severely tachypneic (i.e., have Kussmaul respiratory pattern) to compensate for their AGMA by inducing respiratory alkalosis. In our patient, his compensatory mechanism was impeded by the use of

paralytics as well as inappropriately low minute ventilation. In addition, his mixed AGMA with iatrogenic respiratory acidosis was exacerbated by crystalloid resuscitation using 0.9% sodium chloride (i.e., NS), which resulted in hyperchloremic NAGMA. Our patient's triple acidosis resulted in the severity of his pH on initial presentation.

DKA is a challenging complication of diabetes mellitus, with variable rates of occurrence across population groups.⁸ Although universally fatal before insulin therapy was developed, DKA now carries a mortality rate of 1%–4%. The patient in this report had a pH of 6.6, Acute Physiologic Assessment and Chronic Health Evaluation (APACHE) II score, 18, and lactate level of 2.8mmol/L, which indicate an exquisitely poor prognosis in ICU populations.⁹ A recent study associated mechanical ventilation, pressor support, base excess <–2mEq, elevated lactate level, and pH <7.2 with mortality rates of 81.8%, 91.8%, 79.4%, 80.2%, and 70%, respectively.⁹ Given this patient's poor prognosis yet relatively quick and uncomplicated recovery, we hypothesize that hypothermia mitigated the morbidity of his severe acidosis by normalizing his oxyhemoglobin dissociation curve. This mitigation of tissue hypoxia in the setting of reversible causes for his severe acidosis provided protective factors that contributed to a positive outcome for this patient. Though it remains the goal of providers to keep critically ill and injured patients normothermic en route and in expeditionary settings, the complex pathophysiology of this patient's acidosis and hypothermia uniquely contributed to his survival.

Conclusion

The patient made a remarkable recovery, given his initial presentation and laboratory findings significant for moderate hypothermia, severe metabolic acidosis, lactic acidosis, high base deficit, APACHE II score of 18, dependence on mechanical ventilation, and need for pressor support owing to hemodynamic instability. He had a positive outcome, which we hypothesize was influenced by the effects of hypothermia as well as the reversible nature of his acidosis. Challenges in resuscitation of critically ill patients with DKA include recognition of mixed acid-base disorders, associated respiratory pathophysiology, and fluid/electrolyte derangements, which can lead to fatal cerebral edema and cardiac arrest. These challenges are increased in deployed settings where prolonged transportation times in harsh environments exist and where equipment is often unsophisticated and unreliable.

This case report identified hypothermia in a patient that was caused by his prolonged exposure to a cold environment, the use of paralytics and cold IV fluids in his treatment, as well as his underlying disease process. The reevaluation and documentation of patients' vital signs, including core temperature, are valuable during patient movement. Providers need to acknowledge and intervene when nonenvironmental causes of hypothermia such as iatrogenic medical interventions and underlying medical diseases occur. Implementation of techniques to maintain patients' thermal neutral zone during en route and expeditionary care of the critically ill are memorable lessons and remain challenging patient factors in Afghanistan.

Disclosures

The authors have nothing to disclose.

Author Contributions

All authors approved the final version of the manuscript.

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