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Systemic epinephrine toxicity following topical epinephrine gauze use during burn debridement

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Abstract:

A 54-year-old male with extensive burns caused by a high-voltage alternating current electrical injury underwent three surgical debridement procedures. During the first operation, he developed intraoperative hypertension and tachycardia, followed postoperatively by hypotension, lactic acidosis, hyperglycemia, and hypokalemia, which improved with fluid resuscitation and vasopressor support. Similar hemodynamic and metabolic disturbances recurred during the second debridement. A detailed review of the anesthetic records revealed that epinephrine-soaked gauze had been applied intraoperatively for hemostasis, with vital sign changes occurring shortly thereafter. These findings suggested systemic epinephrine toxicity, followed by hypotension related to antihypertensive therapy. After discussion with the plastic surgery team, topical epinephrine was avoided during the third procedure and replaced with thrombin-soaked gauze, with no recurrence of complications. This case highlights that topical epinephrine used for hemostasis in extensive burn wounds can cause clinically significant systemic toxicity and underscores the importance of interdepartmental communication in preventing perioperative adverse events.

Keywords:

Burn injury, epinephrine, hemodynamic instability, patient safety, systemic toxicity

Introduction

Burn injuries require highly coordinated, multidisciplinary care involving multiple specialties. However, responsibility for perioperative management – including intraoperative anesthesia and postoperative intensive care – may shift rapidly between teams over a short period, and the primary service involved in patient management may change accordingly. In such settings, inadequate information sharing is common and may predispose patients to serious complications.

Epinephrine is a familiar medication widely used across many specialties via

intramuscular or intravenous administration. In contrast, topical epinephrine is used for more limited indications, such as hemostasis for oozing during burn wound care and surgery, and is therefore familiar to only a subset of clinicians. Consequently, when applied over large surface areas, it may not be widely recognized that transdermal absorption of epinephrine can lead to systemic toxicity, including tachycardia, hypertension, and metabolic disturbances such as lactic acidosis.^[1,2]

Case Report

A 54-year-old male with a history of diabetes mellitus after self-discontinuation of treatment was transported to the emergency department after accidental contact with a 20,000-V alternating current power line at

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work. On arrival, he was found to have extensive burns involving the face, left posterior neck, left lateral chest, back, left lower extremity, and the posterior aspect of the right thigh. The total burn surface area (TBSA) was estimated at 64%, including approximately 60% second-degree and 2% third-degree burns. The patient was alert, body temperature was 36.8°C, blood pressure was 128/67 mmHg, pulse rate was 95 beats per minute, respiratory rate was 20 breaths per minute, and SpO₂ was 93% on room air.

Because laryngeal edema was identified, the patient was intubated, and fluid resuscitation and vasopressor therapy were initiated. His respiratory and circulatory status subsequently stabilized, and the laryngeal edema improved, allowing successful extubation on hospital day 11.

The patient remained afebrile without signs of infection. After clear demarcation of necrotic tissue, surgical debridement of the left thigh, lateral aspect of the left lower leg, left chest, dorsum of the left hand, and back was performed by the plastic surgery team on hospital day 17. Preoperative vital signs were as follows: the patient was alert, body temperature was 37.2°C, blood pressure was 97/64 mmHg, pulse rate was 96 beats per minute, respiratory rate was 22 breaths per minute, and SpO₂ was 98% on room air. The procedure was performed under general anesthesia, induced with 1.5% sevoflurane, fentanyl 0.4 mg, and rocuronium 60 mg, and maintained with 1.5% sevoflurane and remifentanyl at 0.3 µg/kg/min. Approximately 90 min after the start of surgery, the blood pressure rapidly increased to 204/106 mmHg. Pain-related sympathetic activation was suspected, and remifentanyl infusion rate was increased to 1 µg/kg/min, and fentanyl was administered in intermittent boluses of 0.2 mg each, for a total dose of 1.5 mg; however, blood pressure remained elevated, and nicardipine was administered in intermittent boluses of 0.4 mg each, for a total dose of 1.2 mg. The procedure was completed with an estimated blood loss of 120 mL, an operative time of 2 h 44 min, and an anesthesia time of 4 h 43 min [Figure 1].

After returning to the ward, the patient developed shock vital signs, with blood pressure of 65/43 mmHg and a pulse rate of 108 beats per minute, along with hypothermia of 35.2°C. The lactate level increased to 10.6 mmol/L. Intravascular volume depletion due to excessive exudation from the debrided burn surfaces was suspected, and crystalloid infusion was initiated. Continuous norepinephrine was initiated and titrated up to a maximum dose of 0.25 µg/kg/min, taking into account peripheral vasodilation associated with rewarming. The patient's condition improved with supportive management, and norepinephrine was discontinued on postoperative day 4.

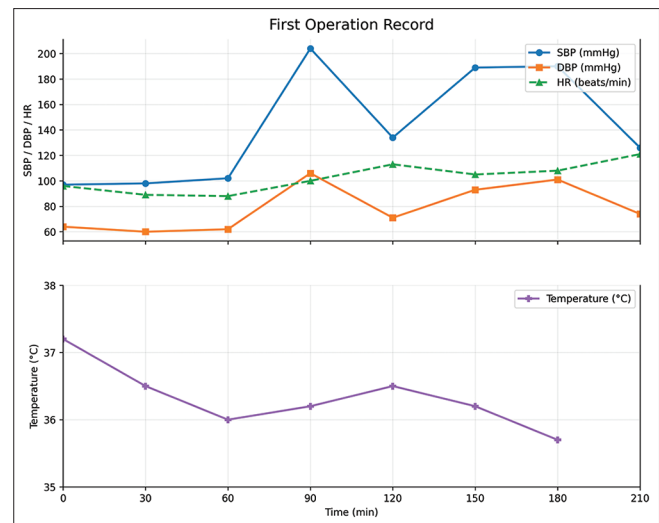


Figure 1: Anesthesia record from the first operation

A second debridement of the same areas was performed on hospital day 31. Preoperative vital signs were as follows: the patient was alert, body temperature was 38.0°C, blood pressure was 130/66 mmHg, pulse rate was 70 beats per minute, respiratory rate was 22 breaths per minute, and SpO₂ was 98% on FiO₂ of 0.21. The surgery was performed under the same anesthetic regimen as previously used. 60 min after the start of surgery, the pulse rate increased to 108 beats per minute, and the intravenous remifentanyl increased. Approximately 120 min into the procedure, the blood pressure rapidly increased to 205/95 mmHg. Despite increasing the remifentanyl infusion rate to 0.6 µg/kg/min and administering fentanyl in intermittent 0.25 mg boluses to a total dose of 0.5 mg, blood pressure control remained inadequate, and nicardipine infusion was initiated. Intraoperative blood tests revealed hyperglycemia, with a blood glucose level of 281 mg/dL, and lactic acidosis, with a lactate level of 6.7 mmol/L. The surgery was completed with minimal blood loss, an operative time of 2 h 28 min, and an anesthesia time of 4 h 3 min [Figure 2].

After returning to the ward, the patient again developed hypotension similar to that observed after the first procedure, and crystalloid resuscitation and continuous norepinephrine infusion were initiated.

Because similar perioperative courses occurred during the first two procedures, adverse effects of commonly used anesthetic agents, anaphylaxis, and bleeding were considered as potential causes. However, there were no skin rashes or gastrointestinal symptoms, and ultrasonography showed no evidence of significant intravascular volume depletion. A detailed review of the anesthetic records revealed that epinephrine-soaked gauze (1:5,000 dilution, corresponding to a maximum epinephrine dose of approximately 3.4 mg) had been

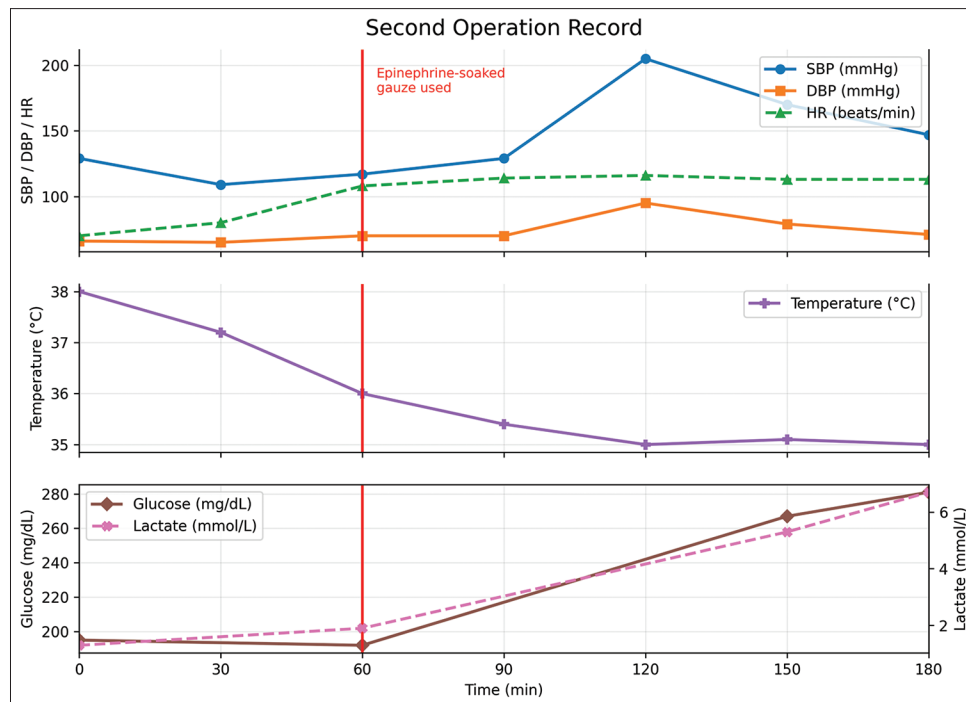


Figure 2: Anesthesia record from the second operation. Hyperglycemia and lactic acidosis progressed after the application of epinephrine-soaked gauze

applied over the entire back for hemostasis of the wound, with the timing closely corresponding to the observed changes in vital signs. No local anesthetic agents were used.

The episodic severe hypertension, tachycardia, marked elevation in serum lactate, hypokalemia, and hyperglycemia were therefore considered consistent with systemic epinephrine toxicity due to topical application to extensive burn wounds. The subsequent hypotension was attributed to the effects of antihypertensive therapy after rapid dissipation of epinephrine's pharmacologic effects.

A third debridement was scheduled on hospital day 45. After sharing these considerations and discussing intraoperative management with the anesthesiology and plastic surgery teams, topical epinephrine was avoided, and local hemostasis was achieved using thrombin-soaked gauze. No abnormal intraoperative hypertension or lactic acidosis occurred, and the procedure was completed uneventfully.

Written informed consent was obtained from the patient for publication of this case report.

Discussion

Epinephrine toxicity is most commonly reported after inadvertent intravenous administration during anaphylaxis treatment. Due to its potent α - and β -adrenergic effects, excessive exposure can result in

metabolic acidosis, lactic acidosis, hyperglycemia, and hypokalemia, and in severe cases, myocardial ischemia, arrhythmia, and pulmonary edema.^[1-4]

Topical epinephrine is widely used for hemostasis and is generally considered safe because the administered dose is usually small.^[5] However, in patients with extensive burn wounds, the total amount applied may be substantial. Furthermore, burn-related inflammation increases vascular permeability and blood flow, which may enhance transdermal absorption and lead to significant systemic exposure.

Previous studies have shown that, in burn patients, topical epinephrine can markedly elevate plasma catecholamine levels, accompanied by tachycardia and subsequent increases in serum lactate.^[1] These findings are consistent with the clinical manifestations observed in this case.

In the present case, the close temporal relationship between topical epinephrine application and abrupt hypertension, tachycardia, and metabolic abnormalities – including the parallel increase in lactate and glucose levels observed during the second procedure, a pattern strongly suggestive of adrenergic-driven metabolic effects – strongly supports adrenergic excess as the underlying mechanism.

Alternative causes were considered. Inadequate anesthesia was unlikely given sufficient opioid administration and lack of response to escalation. Sepsis,

occult bleeding, and significant exudate loss were not supported by clinical findings or imaging. Reperfusion injury was also unlikely due to the reproducibility of events and their consistent temporal association with epinephrine exposure.

Notably, recurrence during two procedures and complete resolution after discontinuation of topical epinephrine further support causality.

This case also highlights the importance of interdisciplinary communication in perioperative care. Because intraoperative management was performed by a different specialty team, recognition of the cause required integration of information across departments. The emergency team contributed to recognition of deterioration, resuscitative management, and communication that led to the prevention of recurrence.

More broadly, emergency physicians are uniquely positioned to function as integrators in complex clinical settings. Their strength lies in a comprehensive, system-wide assessment of patients with undifferentiated conditions, as well as in coordinating care across multiple specialties. In situations such as the present case, where clinical deterioration cannot be explained by a single organ system or specialty-specific framework, this holistic perspective is essential. By synthesizing information from different teams and maintaining a broad differential diagnosis, emergency physicians can play a critical role in identifying uncommon but clinically significant conditions and improving overall patient safety.

Communication failures are a recognized contributor to adverse events, particularly in complex perioperative settings involving multiple teams.^[6-8] In such environments, critical information – such as intraoperative medication use – may not be consistently shared across specialties, increasing the risk of delayed diagnosis and inappropriate management. In this case, the use of epinephrine-soaked gauze was considered routine by the surgical team but was not initially recognized by the postoperative care team, likely contributing to the delayed identification of the underlying cause. Structured communication strategies and clear documentation may help mitigate these risks and improve patient safety.

Conclusion

Although systemic toxicity from topical epinephrine is rare, it can occur. In situations such as the

present case – where procedures are performed by consulting departments or care is transitioned across teams – inadequate handover may result in otherwise unexplained clinical deterioration. Careful attention to interdepartmental information sharing is therefore essential to prevent avoidable perioperative complications.

Author contribution statement

HH: Conceptualization (lead), review of the literature (lead), writing – original draft (lead). TT: Conceptualization (lead), review the literature (lead), writing – review and editing (lead). CY, TM: Writing – review and advise (equal).

Conflicts of interest

None Declared.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient has given his consent for his images and other clinical information to be reported in the journal. The patient understands that name and initials will not be published and due efforts will be made to conceal identity, but anonymity cannot be guaranteed.

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