Journal of Clinical Case Reports
ISSN: 2165-7920

Matsuura et al., J Clin Case Rep 2018, 8:1
DOI: 10.4172/2165-7920.10001064

Case Report Open Access

A Case Report of Severe Potassium Hydroxide (Alkaline) Burn with Hyperkalemia

Hiroshi Matsuura[1]*, Akinori Osuka[2], Hiroshi Ogura[1], Masashi Ueyama[2] and Takeshi Shimazu[1]

[1]*Department of Traumatology and Acute Critical Medicine, Osaka University Graduate School of Medicine, Japan*
[2]*Department of Trauma, Critical Care Medicine and Burn Center, Japan Community Health Care Organization Chukyo Hospital, Japan*

Abstract

A 28-year-old man suffered intentional dermal exposure to potassium hydroxide inside a psychiatric hospital bathroom during inpatient treatment for schizophrenia. He received initial treatment at a local emergency department (ED) and was transferred to our burn unit. On arrival at 18 hours after the injury, he was diagnosed as having 60% total body surface area (TBSA) chemical burns; third-degree: 24%, second-degree: 36%. At the outside ED, his serum potassium level peaked at 8.2 mEq/L (normal range: 3.5 to 5.0 mEq/L) and decreased to 6.9 mEq/L after he received intravenous glucose and insulin therapy. At our facility his potassium had increased to 7.3 mEq/L. The patient's urine output was maintained at >100 mL/h, but his serum potassium level rose to 8.1 mEq/L and continuous hemodiafiltration was initiated within 5 hours of admission. Early debridement was performed due to extensive third-degree burns, risk of deep-tissue alkali injury, and persistent (chemical-induced) hyperkalemia. At 40 hours after the injury, fascial resection of the lower leg was performed, involving 20% TBSA third-degree burn, and his serum potassium normalized. After five operations he was able to ambulate with assistance, and on hospital day 72 he was transferred to a rehabilitation hospital without major complications.

Keywords: Chemical burn; Potassium hydroxide; Hyperkalemia; Fasciotomy

Introduction

This case report is based on a first manuscript reported in the Japanese Journal of Burn Injuries [1]. High-concentration potassium hydroxide preparations are used as metal and pipe cleaning agents and are rarely involved in human exposures. Because chemical burns from alkaline agents may produce liquefaction necrosis with deep tissue injury [2] it is necessary to rapidly develop a treatment plan that includes debridement and skin grafting.

We treated a patient with severe exposure to a highly concentrated potassium hydroxide preparation that resulted in recurrent hyperkalemia requiring multidisciplinary treatment including early surgical intervention. To our knowledge, this is the first report of a chemical burn with suspected dermal absorption of a potassium-containing agent as the cause of hyperkalemia.

Case Report

A 28-year-old man was admitted to a psychiatric hospital for treatment of schizophrenia. During his inpatient stay, he added approximately 500 g of 95% potassium hydroxide (solid) crystals (used by hospital staff for drain cleaning) to his bathroom toilet bowel (with an approximate water volume of 500 mL). He was found by staff as he was splashing the fluid onto his body in the bathroom. He was immediately washed with water for 15 minutes and transferred to a local emergency department 5 hour after the exposure, where he was diagnosed as having a 60% total body surface area (TBSA) chemical burn.

After tracheal intubation, he was decontaminated with tap water for 30 minutes while receiving intravenous fluids (IVFs; Potassium free Lactated Ringer's Solution including Na 90 mEq/L, Cl 70 mEq/L, Glucose 26 g/L) at 600 mL/h. His initial serum potassium level was 7.4 mEq/L (normal range: 3.5–5.0 mEq/L). After fluid resuscitation his serum potassium level transiently decreased to 5.9 mEq/L at 2 hours after arrival, but 8 hours later, it had risen to 8.2 mEq/L. The IVFs were increased to 800 mL/h, and intravenous glucose and insulin were started. He was then transferred to our burn center, at which time his serum potassium was 6.9 mEq/L at 18 hours after the injury.

At our facility, and after already receiving 7000 mL of IVFs, the infusion at 800 mL/h was continued, and his urine output was 200 mL/h. Repeat blood test showed the following: Na 135 mEq/L, K 7.3 mEq/L, BUN 23 mg/dL, creatinine 1.79 mg/dL, AST 41 IU/L, LDH 230 IU/L, CPK 1136 IU/L and albumin 2.0 g/dL (CPK-mb, AST and LDH were normal). His ECG showed peaked T-waves (Figure 1). We confirmed 60% TBSA chemical burns; 24% third-degree and 36% second-degree (Figures 1 and 2).

Clinical Course

Despite adequate urine output (>100 mL/h), hypoalbuminemia

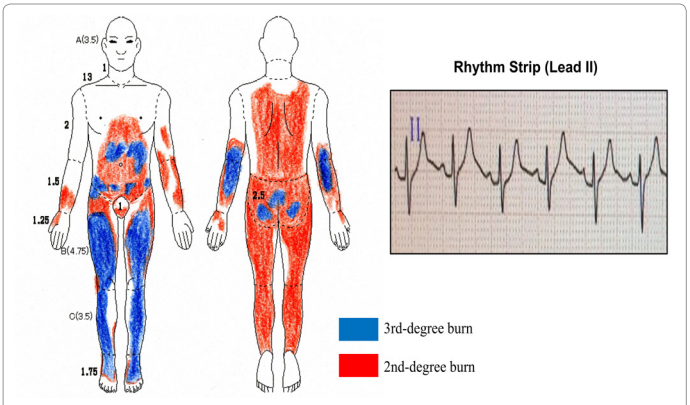

Figure 1: Distribution of partial- and full-thickness burn areas and ECG lead II upon admission to the burn unit. Blue=third-degree burn, red=second-degree burn.

***Corresponding author:** Hiroshi Matsuura, Department of Traumatology and Acute Critical Medicine, Osaka University Graduate School of Medicine, Japan, Tel: +81-6-6879-5707; Fax: +81-6-6879-5720; E-mail: matsuura@hp-emerg.med.osaka-u.ac.jp

Received January 06, 2018; **Accepted** January 25, 2018; **Published** January 31, 2018

Citation: Matsuura H, Osuka A, Ogura H, Ueyama M, Shimazu T (2018) A Case Report of Severe Potassium Hydroxide (Alkaline) Burn with Hyperkalemia. J Clin Case Rep 8: 1064. doi: 10.4172/2165-7920.10001064

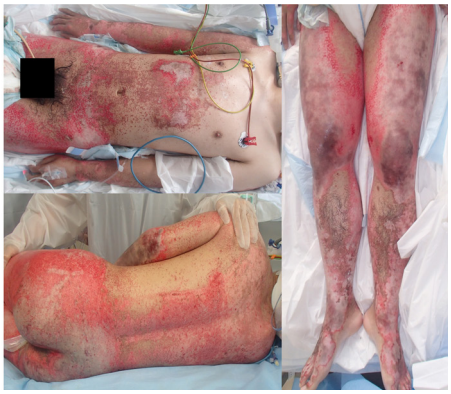

Figure 2: Photographs of the burn injury on hospital day 1.

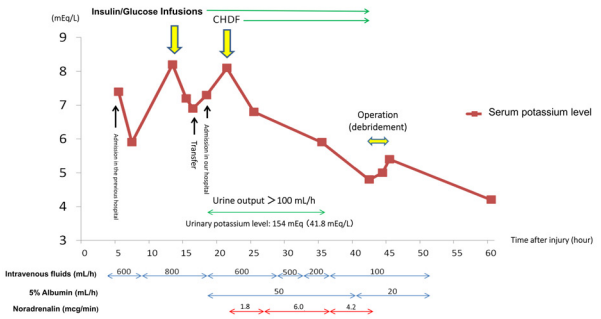

Figure 3: Schematic of changes in serum potassium concentration and therapeutic interventions.

developed to 2.0 g/dL, 5% albumin solution at 50 mL/h was started, and his wounds were re-irrigated for 30 minutes. Due to concerns about alkaline-induced coagulation necrosis into deep tissue and chemical-induced hyperkalemia, skin incisions were made to identify the depth of tissue injury on his right thigh (considered to be the most extensively injured area). The damage, however, was limited to tissue superficial to the fascia. The patient's urine output remained >100 mL/h (urine potassium concentration was 41.8 mEq/L), and IVFs (Potassium free lactated Ringers at 600 mL/h), insulin/glucose infusions, and calcium gluconate (1700 mg) were administered for the hyperkalemia. However, despite these interventions, his serum potassium level rose to 8.1 mEq/L, 4 hours after arrival to the burn unit, and continuous hemodiafiltration (CHDF) was initiated 1 hour later. The patient's plasma acid base status showed respiratory acidosis (pH=7.27, $PaCO_2$=58.7 mmHg, HCO^{3-} =26.2 mmol/L, BE=-2.0 mmol/L) on admission at our burn center and after that these statuses showed normal range. Urinary acid base status showed pH=6.0 and no remarkable change had not detected after admission. During CHDF, the patient required sedation, which resulted in hypotension (blood pressure 83/50 mmHg) that required infusion of noradrenaline at 6.0 mcg/min. Serum potassium decreased to 4.8 mEq/L and CHDF was stopped.

Due to the extensive third-degree burns, risk for worsening of coagulation necrosis in the deep tissue, and concern for alkaline-induced hyperkalemia, we decided to perform early debridement. The operation started 24 hours after burn unit admission (40 hours after injury) and involved debridement of the bilateral forearms and bilateral anterior lower legs (about 20% TBSA of third-degree burns). Intraoperative findings included damage to subcutaneous fat, but fascial excision revealed no sub-fascial injury. Considering the influence of chemical damage to the wound bed after debridement, skin grafts were not performed due to concern for ongoing alkaline injury.

During the operation, while CHDF and glucose/insulin infusions were suspended, his serum potassium gradually increased to 5.4 mEq/L but then decreased to a normal level (Figure 3). This decrease suggests that residual alkaline chemical in the tissue of the third-degree burn areas that was removed during debridement may have contributed to the hyperkalemia.

The wounds were dressing daily, and a second operation was performed on hospital day (HD) 6 involving fascial excision of both legs and anterior chest and abdomen, and skin autograft to the bilateral arms.

Artificial dermis was grafted to both thighs, and the lower legs underwent mixed skin grafting using autograft (micro skin patch) and 3:1 expanded-mesh allograft. Engraftment of each skin graft was successful without evidence of subsequent alkali damage.

On HDs 23 and 37, combination graft surgeries were performed. The cultured epidermal autograft simultaneously covered the split-thickness autograft of 4:1 expanded mesh on the bilateral lower legs, chest and abdomen. Excisions were not required on the buttocks and back. It took more than 30 days for full epithelialization.

Two more operations were required to treat an ulcer before the patient was able to ambulate with support. On HD 72 he was improved, without major complications, and was transferred to a rehabilitation hospital.

Discussion

Alkali chemical burns can cause severe injury involving liquefaction necrosis. This process involves protein denaturation and saponification of fats, which allows for further (deep-tissue) injury and burn [2]. Alkali burns can continue to penetrate into deep tissue for up to 3 days following injury until complete injury occurs [3]. Dermal absorption of other chemicals, such as chromic acid and kerosene, can also cause multiple organ dysfunction syndrome. To our knowledge, there are no other case reports of percutaneous absorption of potassium hydroxide causing hyperkalemia. Among several material safety data sheets issued by Japanese companies using potassium hydroxide, the danger of dermal absorption and systemic effects are not included. However, the data for potassium hydroxide prepared by "CHEMWATCH" mentions that it can enter the bloodstream through cuts and scratches with a potential risk of systemic injury with harmful effects [4]. And sodium and potassium hydroxides have potential risk of systemic toxicity [5]. Previous animal research has shown that the permeability of potassium ions through rabbit skin by using radioisotopes gradually increased following contact and reached a maximum 1 to 3 hours later [6]. The dermal absorption rate increased at higher potassium concentrations. In addition, dermal absorption tests with sodium and bromine in humans, showed increased blood concentrations after 200 minutes [6]. Our patient experienced an extensive dermal exposure to a highly concentrated (95%) potassium hydroxide solution for approximately 5 hours prior to first decontamination efforts. A previous case report of 85% TBSA chemical burn (mostly superficial) after the patient fell into a tank of 30% potassium hydroxide did not involve hyperkalemia but did involve the use of CHDF for acute renal failure on HD 3 [7].

Other potential etiologies for the patient's hyperkalemia are possible, including caustic-induced cellular lysis with release of intracellular potassium. However, intra-operative findings failed to suggest deep tissue (muscle) injury that would support this possibility. No other etiology for the recurrent hyperkalemia (other than dermal

absorption of the potassium hydroxide) was identified, and the patient's urine output and potassium excretion remained normal. Lastly, serum potassium gradually decreased, without specific interventions after the first surgical debridement, further suggesting that potassium hydroxide within subcutaneous tissue was a contributing cause of the hyperkalemia.

Alkali infiltrating the skin cannot be removed sufficiently by washing. Surgical intervention was very effective in the early phase of our case. This suggested that early-phase debridement of the region damaged by alkali was important to control the patient's condition. However, the buttocks and back were selected to receive conservative treatment without surgical interventions. This was very beneficial because it reduced surgical invasion and the need for grafting under a situation with few donors. The influence of alkali damage after debridement is difficult to diagnose immediately. In a patient with extensive injury, it is necessary to continually make appropriate observations and consider the treatment strategy carefully.

Conclusion

This case report illustrates the potential for significant systemic effects, including recurrent hyperkalemia, following a severe chemical burn from potassium hydroxide. Although the patient had a delayed (5 h) presentation for emergency care, repeated dermal decontamination was apparently unsuccessful in preventing deep tissue absorption. Operative treatment, including tissue debridement, was required to remove the tissue suspected to contain potassium hydroxide and prevent ongoing hyperkalemia.

References

1. Hiroshi M, Akinori O, Masashi U, Takeshi S (2017) Extensive alkaline burn with hyperkalaemia injured by potassium hydroxide. Jpn J Burn Injury 43: 157-162.
2. Palao R, Monge I, Ruíz M, Barret JP (2010) Chemical burns pathophysiology and treatment. Burns 36: 295-304.
3. Harchelroad FP, Rottinghaus DM (2004) Chemical burns. Emergency Medicine (6th edn). New York: McGraw-Hill, USA.
4. https://au.vwrcmd2.com/fl/msds/NRPD7HGDGVXF/POTASSIUM_HYDROXIDE_PELLETS_1310-58-3_MSDS.pdf.
5. Açikel C, Ulkür E, Güler MM (2001) Prolonged intermittent hydrotherapy and early tangential excision in the treatment of an extensive strong alkali burn. Burns 27: 293-296.
6. Tregear RT (1966) The permeability of mammalian skin to ions. J Invest Dermatol 46: 16-23.
7. Nakamura Y, Nakano M, Machida H, Suzuki H, Hasuike T, et al. (2012) An autopsy case of severe alkali burn. J Jpn Soc Intensive Care Med 19: 379-383.