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JSM Burns and Trauma

Case Report

Burn-Like Skin Necrosis in a Patient following Infusion of Sodium Bicarbonate

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Submitted: 13 June 2016

Accepted: 22 June 2016

Published: 24 June 2016

Keywords

- Burn-like
- Skin necrosis
- Leakage
- Sodium bicarbonate
- Systemic capillary leak syndrome
- Multiple organ dysfunction syndrome

Abstract

This is a report of skin necrosis following a leakage of intravenous infusion of 5% sodium bicarbonate injection during the treatment of continuous veno-venous hemodialysis (CVVHD) in a female patient. The patient sequentially suffered from systemic capillary leak syndrome (SCLS), severe sepsis, and multiple organ dysfunction syndromes (MODS). She died due to refractory septic shock and MODS. Burn-like skin necrosis, although rare, is a recognized complication in infusing hyperosmotic solutions through phleboclysis. It is important to discriminate the solution infused via phleboclysis, or to infuse a hyperosmolar solution through a central vein.

ABBREVIATIONS

ARDS: Acute Respiratory Distress Syndrome; COP: Colloid Osmotic Pressure; CVVHD: Continuous Veno-Venous Hemodialysis; ICU: Intensive Care Unit; MODS: Multiple Organ Dysfunction Syndrome; rhEGF: Recombinant Human Epidermal Growth Factor; rhFGF: Recombinant Human Basic Fibroblast Growth Factor; SCLS: Systemic Capillary Leak Syndrome

INTRODUCTION

Increased permeability of small vessels to proteins and other macromolecules is a well recognized feature of critical illness. Increased permeability leads to an increased escape of serum proteins from the vessels, especially albumin, and a decrease in plasma colloid osmotic pressure (COP). This in turn allows fluid to shift from the intravascular into the interstitial compartment and the subsequent hypovolaemia contributes to the hypotension seen in septic shock [1,2]. We report a case of severe burn-like skin necrosis in a patient with systemic capillary leak syndrome (SCLS) after administration of 5% sodium bicarbonate solution through a peripheral vein during continuous veno-venous hemodialysis (CVVHD). It shows the importance of selection for a suitable venous channel in infusing a hyperosmolar solution as well as the importance of monitoring for transfusion management in ICU patients.

CASE PRESENTATION

An 90-year-old female was hospitalized for hypoalbuminemia and systemic edema at Department of General Surgery in our hospital in January 2016. Physical examination on admission revealed: height, 162 cm; body weight, 50.5 kg (an increase in body weight by 5.5 kg during the previous 10 days); pulse rate, 102 beats/min; blood pressure, 80/45 mmHg; respiratory rate, 25/min; and body temperature, 37.5°C. Laboratory data indicated severe hemoconcentration (hemoglobin 22.6 g/dL, hematocrit 62.2%), hypoproteinemia (serum total protein 2.4 g/dL), and acute renal failure (creatine 1.8 mg/dL, blood urea nitrogen 35 mg/dL). Symptoms and signs of acute inflammation were only mildly manifested (white blood cell count 13100/μL, C-reactive protein 1.0 mg/dL, procalcitonin <0.5 μg/L), and features of sepsis were not observed. Platelet count was in normal range. Differential blood count indicated no sign of hematologic disorders. Electrolytes were in normal range (sodium 134 mmol/L, potassium 4.4 mmol/L). Parameters of cholestasis and aminotransferases were not markedly altered (bilirubin 0.6 mg/dL, alkaline phosphatase 62 U/L, gamma-glutamyl transferase 64 U/L, aspartate aminotransferase 30 U/L, and alanine aminotransferase 38 U/L). Serum IgG was 668 mg/dL, IgA 85.4 mg/dL, IgM 27.0 mg/dL, and IgE 75 mg/dL. Arterial blood gas analysis showed the following data: pH 7.29, PCO_2 43 mmHg, PO_2 91 mmHg, bicarbonate 19.5 mmol/L, anion gap 5.6 mmo/L. Creatine kinase value was normal (126 U/L) on hospital day 1 and increased to over 7000 U/L on day 5 (day of admission to our ICU). Ultrasonography and computed tomography showed bilateral pleural effusion and ascites. Further examinations, including bone marrow puncture, colonoscopy, and cytological

analysis of ascites as well as pleural effusion did not reveal cardiac ailment, infection, or malignancy. After extensive diagnostic procedures, the diagnosis of SCLS was made.

On hospital day 5, the patient began to complain of dyspnea and she subsequently developed acute respiratory distress syndrome (ARDS) and severe sepsis. Her chest radiograph showed evidences of definite pneumonic consolidation. After the preliminary assessment of hospital-acquired infection and a blood culture examination, the patient was treated with sulperazone, ciprofloxacin and intravenous fluid in ICU. Soon, the central venous pressure rose to 10 mmHg, but the systolic blood pressure remained at 80 mmHg. Therefore, we started an infusion of dobutamine (15 μg/kg/min), dopamine (25 μg/kg/min) and norepinephrine (100 μg/min) through the central venous catheter in the right subclavian vein. With the appearance of respiratory distress and anuria with exacerbation of azotemia caused by septic shock, we began to institute mechanical ventilation and CVVHD. To correct acidosis occurring during CVVHD, 5% sodium bicarbonate (brand name: Tansuanqingna; specification: 250 ml, 12.5g; CR Double-Crane Pharmaceuticals Co., Ltd, China) was given in adequate amount of fluid to replenish fluid through a peripheral vein on the patient's right ankle. Two hours after the first sodium bicarbonate infusion (180 ml/hour), erythematous rash around the injection site appeared (Figure 1A), necessitating discontinuation of the intravenous sodium bicarbonate infusion. Meanwhile, the region of leakage expanded to both the whole ankle and the right lower legs with variably sized bullous lesions (Figure 1A). This area of drug leakage subsequently progressed to extensive ulceration one week after the leakage (Figure 1B). 33% magnesium sulfate solution and compress topical treatment with potent recombinant human epidermal growth factor (rhEGF) and recombinant human basic fibroblast growth factor (rhFGF) were initiated, but the skin lesions accompanying embolismed vasoganglion sequently extended to full-thickness (Figure 1C) and developed into a burn-like necrotic lesion measuring 30 × 10 cm^2 with a adamant black eschar surface (Figure 1D) over the next two weeks. An attempt at accepting a skin graft failed because of the patient's aggravated pathogenetic condition. Despite received the continuous anti-sepsis bundle therapies, the patient eventually died due to refractory septic shock and MODS. Because without the permission of the next of kin of the deceased, the biopsy of this skin lesion or the necropsy was not performed. However, medical experts have testified that the nurse's negligence caused the injury.

The case report was approved by the Institutional Review Board of Beijing Chao-Yang Hospital (approval number 2016–25) and in accordance with the Declaration of Helsinki principles. Prior to the report, an informed consent was signed by her next of kin.

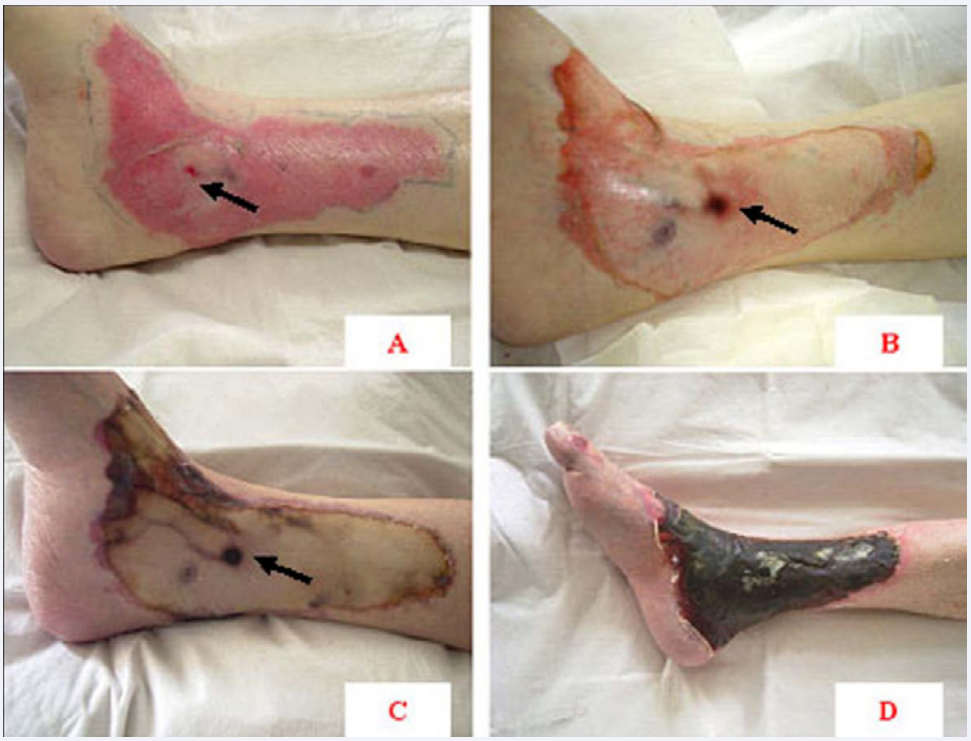

Figure 1 Burn-like skin necrosis following a leakage of intravenous infusion of 5% sodium bicarbonate injection in a female patient. Two hours after the first sodium bicarbonate infusion, erythematous rash around the injection site appeared, necessitating discontinuation of the intravenous sodium bicarbonate infusion. Meanwhile, the region of leakage expanded to both the whole ankle and the right lower legs with variably sized bullous lesions (Figure 1A). This area of drug leakage subsequently progressed to extensive ulceration one week after the leakage (Figure 1B). 33% magnesium sulfate solution and compress topical treatment with potent recombinant human epidermal growth factor (rhEGF) and recombinant human basic fibroblast growth factor (rhFGF) were initiated, but the skin lesions accompanying embolismed vasoganglion sequently extended to full-thickness (Figure 1C) and developed into a burn-like necrotic lesion measuring 30 × 10 cm2 with a adamant black eschar surface (Figure 1D) over the next two weeks.

DISCUSSION

SCLS is a rare disorder characterized by unexplained, often recurrent, non sepsis-related episodes of increased capillary hyperpermeability leading to hypovolemic shock due to a markedly increased shift of fluid and protein from the intravascular to the interstitial space. Hemoconcentration, hypoalbuminemia and a monoclonal gammopathy (IgG class monoclonal gammopathy predominates, with either kappa or lambda light chains) are the characteristic laboratory findings [1-3]. SCLS was first described in 1960 by Clarkson et al [4]. Common clinical manifestations of SCLS are diffuse swelling, weight gain, renal shut-down and hypovolemic shock. Here we present a patient who suffered from SCLS with hypovolemic shock and MODS. This shift of plasma and protein was reported to cause multiple organ failure and lead to death during the initial capillary leak phase [5-7]. Analogically, it might also result in the leakage of drug solutions from blood vessels into the interstitial space.

5% sodium bicarbonate injection is a sterile, nonpyrogenic solution of sodium bicarbonate in water for injection. Intravenous sodium bicarbonate therapy has been widely used to treat metabolic acidosis and to alkalinize the urine. It is also used as adjunctive therapy in treating hypercalcemic or hyperkalemia crises. When used in low amount of fluid, no common complications have been reported with sodium bicarbonate. Overly aggressive therapy with sodium bicarbonate injection, USP can result in metabolic alkalosis and hypernatremia. Rapid infusion of hyperosmolar sodium bicarbonate has been associated with intraventricular hemorrhage in the pediatric literature [8]. Other main side effects of sodium bicarbonate include: (1) pain, burning or swelling at the site of injection; (2) extreme irritability; (3) muscle spasms or cramps; (4) breathing difficulties; and (5) symptoms caused by low levels of potassium in the blood: drowsiness, loss of appetite, muscle twitching or trembling, nausea or vomiting, unusual tiredness or weakness. However, the local adverse effects resulting in a full-thickness skin necrosis similar to burn eschar are very rare, or at least rarely reported.

This report described one case of a severe, extensive inadvertent extravasation that occurred approximately two hours after intravenous sodium bicarbonate infusion for acid-intoxication. In this case, the clinical presentation was characteristic, with chemical cellulitis because of the alkalinity of hypertonic solutions, followed by the development of an extensive burn-like skin necrosis, ulceration or sloughing at the site of infiltration. The occurrence within hours of intravenous hypertonic solutions infusion, characteristic changes at onset, and the clinical course should be aware by all medical staff. A slow rate of administration of a properly diluted solution into a large bore needle and vein is essential if intravenous administration is necessary. Prompt elevation of the part, warmth and local injection of lidocaine or hyaluronidase are also recommended to reduce the likelihood of tissue sloughing from extravasated intravenous solutions [9].

ACKNOWLEDGEMENT

Subsequent clinical researches about SCLS were supported, in part, by grants from the National Natural Science Foundation (nos. 81372042 30901561), Beijing Nova program of Science and Technology (no. Z141107001814043) and High level health technical personnel training plan of Beijing health system (no. 2014-2-022).

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Cite this article

Huang L, Li J, Yao Y, Zhang Z, Li W (2016) Burn-Like Skin Necrosis in a Patient following Infusion of Sodium Bicarbonate. JSM Burns Trauma 1(1): 1002.