Bullous Dermatitis and Skin Necrosis Developing after Adrenalin Extravasation

Adrenalin Ektravazasyonu Sonrası Gelişen Büllöz Dermatit ve Cilt Nekrozu

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Abstract

Extravasation of vasopressors can have serious complications varying from simple local reactions to skin necrosis and compartment syndrome. Here, we presented bullous dermatitis and skin necrosis which developed due to extravasation of adrenalin infusion in a Hodgkin lymphoma patient with septic shock who was admitted due febrile neutropenia.

Keywords: Adrenalin, bullous dermatitis, child, extravasation, necrosis

Introduction

Extravasation is a frequent and potentially dangerous complication in intensive care units. Extravasation of vasopressors can have serious complications varying from simple local reactions to skin necrosis and compartment syndrome [1]. Adrenalin is a catecholamine with potent vasoconstrictor effect that is preferred in the treatment of cardiopulmonary resuscitation and shock. In this study, we presented bullous dermatitis and skin necrosis that developed due to extravasation of adrenalin infusion in a Hodgkin lymphoma patient with septic shock who was admitted due to febrile neutropenia.

Case Report

A 14 years old girl with Hodgkin lymphoma was admitted to intensive care unit, as she had convulsion during follow-up for neutropenic fever after the completion of COPP-ABV chemotherapy regimen. The patient was unconscious and there was respiratory distress when admitted to intensive care unit (ICU). Physical examination findings were as follows: body temperature, 38°C; respiration rate, 16/min; heart rate, 110/min; blood pressure, 60/30 mmHg; Glasgow coma score, 6. In the laboratory evaluations, findings were as follows: WBC, 1500/100 (5-13 10³/uL); Hgb, 6 (11-13 gr/dL); Plt, 12,000 (180-510 10³/uL); PT, 19.5 (9-14.5 seconds) APTT, 88.5 (19-39 seconds); BUN, 40 (7-26 mg/dL); creatinine, 1.2 (0.3-08 mgr/dL); AST, 60 (0-41 U/L); ALT, 75 (0-45 U/L); CRP, 120 (0-5 mg/L). During the follow-up, mechanical ventilation was initiated due to increased respiratory distress and carbon dioxide retention. Dopamine and dobutamine infusions were initiated after loading doses of 20 cc/kg, as the patient was considered in septic shock. In addition, teikoplanin, meropenem and amikacin were were initiated because of neutropenia. No catheterization was performed at early period because of prolonged PT and PTT values and thrombocytopenia. Adrenalin infusion (0.1 µg/kg/min) was initiated from a peripheral vein in the patient with undetectable blood
pressures despite dopamine and dobutamine infusion. The infusion rate was escalated up to 0.3 µg/kg/minute. On day one after the infusion, circulatory dysfunction, discoloration and hypothermia were observed on the dorsal side of the left foot. Fluid infusion was replaced by femoral catheter, which was inserted with the support of platelet and TDP, as the patient was considered to have adrenalin extravasation. On day 2, increased hyperaemia and multiple bullous reactions (as the largest being 10 x 5 cm in size) developed on the haemorrhagic, necrotic ground (Figure 1). The patient was consulted by plastic surgery and dermatology departments, which considered the patient as bullous dermatitis on necrotic ground. No debridement was performed because of bleeding diathesis secondary to septic shock. Therapy including elevation, local antibiotic ointment and pentoxyphilline was used at the reaction site to improve the circulation. The patient died due to septic shock 2 days after the onset of bullous reaction.

Discussion

Infusions and treatments via peripheral veins are routine procedures in hospitals. Extravasation injury denotes the leakage of fluid from vein to tissues surrounding the vein. Extravasation injury is most commonly seen at dorsal sides of hands and feet, and antecubital fossa [1, 2].

Incidence of extravasation has been reported as 0.1-6.5% after intravenous therapies. Decreased peripheral flow, fragile veins, capillary leakage, flexible subcutaneous tissues, and drugs and fluids used intravenously may enhance the damage. In clinical practice, it may present as mild erythema to skin necrosis with oedema [1, 3]. Extravasation risk varies depending on age in children. It is more common in children than adolescents. It is facilitated by poor venous integrity, decreased peripheral circulation, and flexible subcutaneous tissue in new-borns. In children, even small volumes may lead to extravasation injury, local compression effect, and consequently compartment syndrome [2]. Extravasation injury may remain localized and/or progress to extensive tissue necrosis. In our case, extravasation injury progressed to necrosis and bullous dermatitis due to immune deficiency, which was localized at the beginning. Signs and symptoms of extravasation include pain, erythema, swelling, tenderness, local blistering, mottling, firmness, induration, ulceration and lack of capillary filling [4]. In our case, neutropenia and septic shock secondary to chemotherapy regimen given due to Hodgkin lymphoma were the underlying reasons of aggressive tissue reaction. We failed to observe the course of necrosis and bullous reaction as the patient died due to septic shock. Total parenteral nutrition, cytotoxic drugs, vasopressors, inotropic agents, and electrolyte and hyperosmolar therapies cause the extravasation injury [5]. Adrenalin is an inotrope agent with local and systemic vasoconstrictor effects. Adrenalin should be given via catheter because of its local effect, if possible.

Specific antidote is used in the animal studies and there are only case reports in humans. Hyaluronidase, terbutalin, phentolamin and pentoxyphilline have been used in extravasation. Local debridement or surgical interventions are performed in some cases [2, 6-8]. In our case, pentoxyphilline was preferred as it is readily available. Treatment of extravasation injury includes inserting catheter to an intact site, monitoring the infusion, infusion volume and rate, and training the nurses about extravasation injury.

Informed Consent: Written informed consent was obtained from the patient/patients.

Peer-review: Externally peer-reviewed.

Conflict of Interest: No conflict of interest was declared by the authors.

Financial Disclosure: The authors declared that this study has received no financial support.

References