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Case Report

Postoperative Bromoderma Due to Rocuronium Bromide Exposure: A Case Report

Roküronyum Bromür Maruziyetine Bağlı Postoperatif Bromoderma: Olgu Sunumu

[1]Emre Sünbüloğlu, [1]Cahit Yavuz, [1]Fatma Tuncez Akyurek, [1]Gülcan Saylam Kurtipek, [2]Dilara Yapıcı, [2]Pınar Karabağlı

[1]Selcuk University School of Medicine Department of Dermatology,Konya,Türkiye
[2]Selcuk University School of Medicine Department of Clinical Pathology,Konya,Türkiye

Correspondence

Cahit Yavuz, Assoc. Prof.
Selcuk University, School of Medicine, Department of Dermatology, Konya, Türkiye

E-Mail: cyavuz@hotmail.com,

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ABSTRACT

Bromoderma is a dermatological condition characterized by skin eruptions resulting from oral intake, inhalation, or direct contact with substances containing bromide. Although several bromide-containing medications are available in our country, bromoderma remains a rare clinical presentation. We present a case of bromoderma that developed as a secondary reaction to the use of an anesthetic agent during surgery for decubitus ulcers in the Department of Plastic and Reconstructive Surgery.

Keywords: Bromoderma, Rocuronium bromide, Skin eruption

ÖZ

Bromoderma, bromür içeren maddelerin oral alımı, inhalasyonu ve/veya doğrudan temas sonucu gelişen deri döküntüleri ile karakterizedir. Ülkemizde birçok bromür içeren ilaç bulunmasına rağmen, bu durum nadir görülen bir klinik durumdur. Plastik ve Rekonstrüktif Cerrahi bölümünde dekübit ülserleri için yapılan bir operasyonda kullanılan anestezik ajan nedeniyle gelişen bromoderma vakasını sunmak istiyoruz.

Anahtar Kelimeler: Bromoderma, Roküronyum bromür, Deri döküntüsü

Introduction

Bromoderma is a dermatological condition characterized by skin eruptions resulting from oral intake, inhalation, or direct contact with substances containing bromide. Bromide, a halogen element, was widely used in the early 20th century, particularly in pediatric medicine, due to its antitussive, antispasmodic, anticonvulsant, and sedative properties. However, the advent of newer anticonvulsants and the subsequent discontinuation of bromide-containing solutions, such as calcium and potassium bromide, in many countries has significantly reduced the incidence of bromoderma, making such cases increasingly rare. Verbal consent was granted from the patient.

Case

A 31-year-old male patient was referred to our clinic approximately 15 days following surgery for decubitus ulcers in the department of plastic and reconstructive surgery due to the onset of redness and exudative lesions on his legs. His medical history revealed that he had become paraplegic as a result of a gunshot wound 9 years prior and had undergone four separate surgeries for decubitus ulcers at different intervals. The patient had no known chronic conditions and was not on any regular medications. The case also reported that the eruptions on his legs began two weeks after the most recent surgery and were evaluated in our clinic during the third postoperative week.

On dermatological examination, multiple, scattered vegetative plaques with intact bullae, surrounded by erythema, were observed on the distal two-thirds of the anterior aspect of both tibias, along with 2-3 erosive areas (Figure 1). A biopsy was performed from the bullous vegetative plaque on the distal right tibia, with initial differential diagnoses of pyoderma gangrenosum and bromoderma. Histopathological examination of the punch biopsy revealed orthokeratosis on the surface, areas of necrotic epidermis, and parakeratosis in other regions, accompanied by subcorneal pustules. Nuclear

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Complaints: geneltip@selcuk.edu.tr

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debris and neutrophil aggregates were observed within the pustules, and widespread extravasation of erythrocytes, particularly in the superficial dermis, was noted. Endothelial prominence in vascular structures around the lesion, along with inflammation composed of perivascular neutrophils, eosinophils, leukocytes, and lymphocytes, was also observed (Figure 2). These histopathological findings were consistent with bromoderma.

Following confirmation of the diagnosis, the patient's medical history was reviewed in detail. Anesthesia records revealed that rocuronium bromide had been used as the anesthetic agent during the surgery, with the patient receiving a 50 mg intravenous dose of rocuronium bromide as a push. After the diagnosis was established, the patient was placed on follow-up care without specific treatment, and the lesions were monitored under local care. At the two-month follow-up, the lesions had nearly resolved, leaving only post-inflammatory hyperpigmentation (Figure 3).

Discussion

Bromoderma is a dermatological condition characterized by skin eruptions resulting from the intake, inhalation, or direct contact with bromide-containing agents. Bromide, a halogen element, was historically widely used in pediatrics during the early 20th century for its antitussive, sedative, antispasmodic, and anticonvulsant properties. However, with the development of modern anticonvulsants and the banning of calcium and potassium bromide solutions in many countries, cases of bromoderma have become increasingly rare (1).

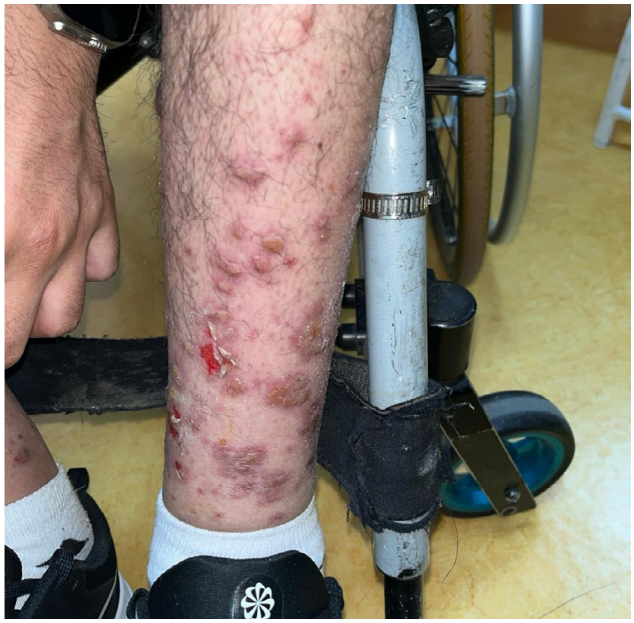

Figure1. Multiple, scattered vegetative plaques with intact bullae, surrounded by erythema.

While the pathogenesis of bromoderma remains poorly understood, three primary theories have been proposed to explain skin involvement in bromide toxicity:

1.Toxic Theory: This theory posits that bromide ions are excreted through eccrine and sebaceous glands, triggering an inflammatory response.

2.Biotropic Theory: This hypothesis suggests that bromide enhances the pathogenicity of skin saprophytes, thereby inducing skin eruptions.

3.Immunological Theory: Halogens, including bromide, may elicit delayed hypersensitivity reactions (2).

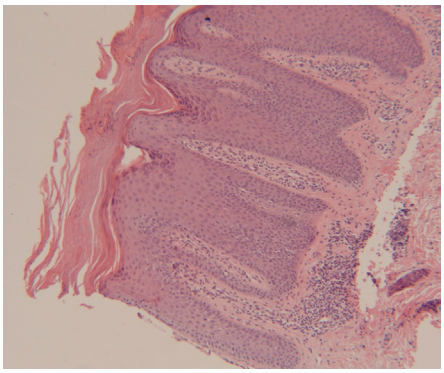

Figure2. Orthokeratosis on the surface areas of necrotic epidermis, and parakeratosis in other regions, accompanied by subcorneal pustules.

Due to the slow renal excretion of bromide, with a half-life of 10–14 days, serum bromide levels can accumulate to toxic concentrations over time (3).

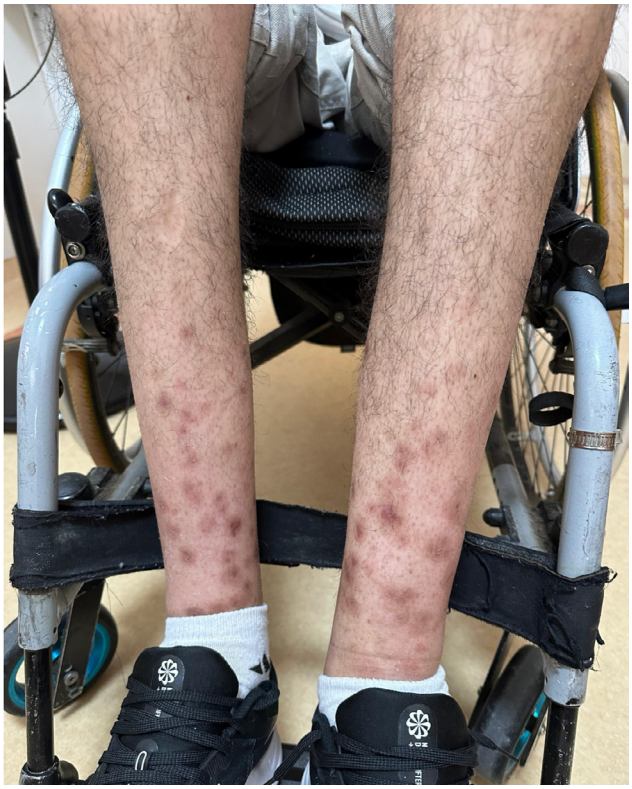

Figure 3. Resolved lesions with postinflammatory hyperpigmentation.

The clinical presentation of bromoderma can vary from mild acneiform eruptions with papules and pustules to more severe forms, including panniculitis, ulcers, and vegetative nodules. Lesions commonly affect the face, scalp, and lower extremities. Histopathological findings often reveal pseudoepitheliomatous hyperplasia, intraepidermal abscesses, and perifollicular and peri adnexal neutrophilic infiltrates. Notably, a diagnosis of bromoderma can be made even in the presence of normal serum bromide levels, particularly when systemic bromism is absent (1).

The diagnosis of bromoderma is primarily clinical and relies on a detailed history of bromide exposure. Histopathological analysis, though not highly specific, is instrumental in ruling out other conditions. Differential diagnoses include:

•Deep mycoses (e.g., sporotrichosis, cryptococcosis, chromomycosis)

•Acneiform eruptions

•Pyoderma gangrenosum

•Pyoderma vegetans

•Sweet's syndrome

•Eosinophilic folliculitis

•Atypical mycobacterial infections

•Cutaneous tuberculosis

•Congenital syphilis (1,4).

The cornerstone of bromoderma management is discontinuing the offending agent. Adjunctive therapies may include diuretics, such as furosemide and ethacrynic acid, to enhance bromide elimination via renal excretion. Additionally, topical corticosteroids and antiseptic solutions can be used as local treatments (4). A review of the literature identified an infant case with the shortest onset of bromoderma following bromide exposure. The infant, with a history of partial epileptic seizures, was treated with phenobarbital and potassium bromide (260 mg twice daily). Skin lesions appeared by the end of the first treatment week. The lesions resolved gradually after potassium bromide was tapered and discontinued over two weeks (5). In the present case, bromoderma lesions developed on the 10th day following exposure to a bromide-containing agent. Despite the short exposure period, this case highlights the potential for bromoderma to arise even with anesthetic agents. We underscore the importance of considering bromoderma in patients presenting with infiltrative papules and plaques in the postoperative period. Further large-scale, controlled studies are warranted to better elucidate this association.

Conflict of interest

None

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