

Oral Steroids for Dermatitis

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ABSTRACT

Contact/allergic dermatitis is frequently treated inappropriately with lower-than-recommended doses or inadequate duration of treatment with oral and intramuscular glucocorticoids. This article highlights a case of dermatitis in a Ranger Assessment and Selection Program student who was improperly treated over 2 weeks with oral steroids after being bit by *Cimex lectularius*, commonly known as bed bugs. The article also highlights the pitfalls of improper oral steroid dosing and provides reasoning for longer-duration oral steroid treatment.

KEYWORDS: *dermatitis; steroids; bed bugs; military; Cimex lectularius*

Introduction

Insect infestations in the US military have been a problem since World War I.¹ Presenting symptoms for bed bug (*Cimex lectularius*) bites can include pain and itching, and at times, are psychologically bothersome.²⁻⁴ In the 1950s, there was a considerable reduction in bedbug occurrences in the United States.² Whether from globalization, increased world travel, or resistance to insecticides, within the past 15 years, there has been a resurgence of bedbug infestations.^{2,5,6} Bed bug bites cause a variety of dermatologic conditions ranging from purpuric, vesicular, and bullous lesions, to pruritic erythematous maculopapular lesions, and to a contact/allergic dermatitis.^{3,4} Dermatitis is one of the most common medical complaints within the military.^{1,6} It is not uncommon to treat contact/allergic dermatitis secondary to bed bug infestations with oral and/or parenteral glucocorticoids.⁷ While, the literature does not recommend the use of steroids for these types of reactions, if it is used, it should be a long tapering dose.^{2,4,8} Here, we will highlight the improper treatment of contact/allergic dermatitis due to improper administration of oral and parenteral steroids.

Case Report

A 21-year-old active duty Servicemember initially presents to the Battalion Aid Station (BAS) and his primary

care provider with a complaint of erythematous maculopapular rash across his entire posterior thorax and axillary fold. The patient complains that the rash is extremely pruritic and the areas affected have a constant burning sensation. He first noticed the rash about 3 weeks before his visit to the BAS. The rash began with a group of 12 to 18 small papules localized to the patient's right scapula region and has since spread across his entire posterior thorax and axillary folds bilaterally. The patient denied recent contact with known infectious disease or recent travel but endorses contact with a new domesticated pet dog that correlates with the onset of his symptoms. The patient's past medical, surgical, and family histories were noncontributory. He also denied any recent viral infections or immunizations. The patient's medical records indicate that all immunization were up to date. The patient was seen at two other clinics, neither of which provided him a definitive diagnosis; both treated him symptomatically with oral steroids and antihistamines. The patient states he has short-term relief (1 to 2 days) but denies any significant benefit from previous medications prescribed by the clinics.

Examination of the patient's skin reveals an asymmetrically distributed erythematous maculopapular rash with several papules varying in size from 0.1cm to 1cm. The patient has many excoriated papules that have hemorrhagic crust without purulence. The erythematous maculopapular rash is distributed across the patient's entire posterior thorax, axillary folds bilaterally, and on the patient's right side near the pectoral region. No rash appears on any portions superior to the superior portion of the shoulders bilaterally, any portion of the arms distal to the patient's elbows bilaterally, or any portion inferior to the patient's waistline. Diagnostic laboratory evaluation with complete blood count revealed an elevated white blood cell count, which could have been explained by the oral steroid use. Additionally, 5mm punch biopsy specimens were obtained at four symptomatic locations. The rest of the findings on physical examination, which included head, eyes, ears, nose, and throat; and his respiratory, cardiovascular, gastrointestinal, neurologic, and musculoskeletal systems, were normal or unremarkable.

Treatment

The patient's initial treatment at the Troop Medical Clinic included injection of 12mg dexamethasone intramuscularly (IM) in the clinic, and he was prescribed 60mg prednisone by mouth (PO) once a day for 5 days. He was also given hydroxyzine PO as needed for the itching. Within days, the rash reappeared and presented with a larger distribution with more severe itching. Due to this, the patient visited the Army Community Hospital emergency department 1 week later. There he was treated with 100mg doxycycline PO twice a day for 10 days, 50mg prednisone PO once a day for 6 days, 50mg diphenhydramine PO as needed for 10 days, and 150mg ranitidine PO once a day for 10 days. Both courses of medications proved to be inadequate and worsened his symptoms.

Six days after being seen in the emergency department, he came to his BAS for worsening symptoms. There, he was treated with a 20-day tapering course of oral prednisone (60mg once a day for 5 days, 40mg once a day for 5 days, 20mg once a day for 5 days, and 10mg once a day for 5 days). This 20-day tapered course gave the patient complete relief of the pruritus and rash. Evaluation of the biopsy specimens revealed spongiotic dermatitis.

Discussion

It was determined that the cause of the rash and pruritus was *C. lectularius*, commonly known as "bed bugs" (Figure 1). Bed bugs have been problematic for humans for more than 3,550 years.³ They are small (4 to 7mm), reddish-brown, flat, wingless, oval insects that emit an offensive odor.³ There are three salivary proteins of bed bugs that may be important: a nitric oxide-liberating heme protein, an anticoagulant, and a pyrase-like nucleotide-binding enzyme.⁹

Bed bugs hide in cracks and crevices in beds and are found in overcrowded areas with high turnover.^{3,10} Transient military housing for students are prime areas for the transmission of several diseases and for insect infestations, including bed bugs. Although the transient housing is often treated for insects and the students are taught proper hygiene, purchasing a new mattress for each new student is not feasible. There have been several military installations with bed bug infestations.¹¹

Spongiotic dermatitis is defined by the presence of epithelial intercellular edema.¹² It can be further classified as acute, subacute, and chronic.¹² When vesicles form, they typically contain a protein-based fluid along with lymphocytes and histocytes. Spongiotic dermatitis is caused by several conditions, with subacute spongiotic

Figure 1 Patient's asymmetrically distributed erythematous maculopapular rash.



dermatitis being the most common presentation. Typically, these would be seen in the BAS, due to allergic and contact dermatitis, atopic dermatitis, seborrheic dermatitis, and drug reactions.¹² Interestingly, the diagnosis of spongiotic dermatitis does not rule out a fungal infection¹³; therefore, proper testing should be completed to ensure the absence of fungal infection.

Although the actual immunologic pathology is still uncertain, what some postulate is an allergic dermatitis with IgE and IgG being isolated in other studies bed bug can cause various skin reactions.^{3-5,10,14} Allergic dermatitis is a type IV hypersensitivity reaction mediated by the T cell and can be of slow onset.^{8,15} Allergic contact dermatitis can be distinguished from irritant dermatitis by the dominant symptom of pruritus, exposed areas of skin, vesicles and bullae, and distinct borders.¹⁶ Typically, bed bugs do not cause severe reactions and require the use of only topical corticosteroids. In rare cases, a reaction can occur that would require oral glucocorticoids and other medications.⁷ According to Usatine and Riojas, there is "no evidence to support the use of long-acting injectable steroids in the treatment of contact dermatitis."¹⁶

The organ and glands that are pivotal in the area of concentration for treatment with glucocorticoids are the

hypothalamus, adrenal gland, and pituitary gland. The hypothalamus is located inferior to the thalamus and superior to the brainstem. The pituitary gland is located inferior to the hypothalamus and connected to it via the infundibulum. The pituitary gland has a posterior lobe and an anterior lobe. The posterior lobe is composed of the infundibulum and pars nervosa, and the anterior lobe is composed of the pars tuberalis in the superior region and the pars distalis in the inferior portion. The adrenal glands are located just superior to the kidneys and measure approximately 5cm vertically, 3cm wide, and 1cm from anterior to posterior. They also are composed of two parts: the adrenal cortex and the adrenal medulla. The adrenal cortex comprises three regions: the zona glomerulosa, the zona fasciculata, and the zona reticularis.¹⁷

The hypothalamic-pituitary-adrenal (HPA) (Figure 2) axis is the mechanism by which the hypothalamus, pituitary gland, and adrenal gland communicate. Within the hypothalamus, corticotropin-releasing factor (CRF) is secreted by the hypophysiotropic neurons localized in the medial parvocellular subdivision of the paraventricular nucleus (PVN).¹⁸ CRF is a 41-amino-acid peptide and is the primary regulator of the HPA.¹⁸ CRF binds to receptors in the pituitary gland and releases adrenocorticotrophic hormone (ACTH) into systemic circulation.^{17,18} ACTH enters the systemic circulation by the anterior lobe. ACTH travels by the bloodstream to the adrenal cortex to trigger the production of the corticosteroid hormone cortisol from cholesterol into δ 5-pregnenolone, the initial step in glucocorticoid synthesis.^{17,18} Cortisol is

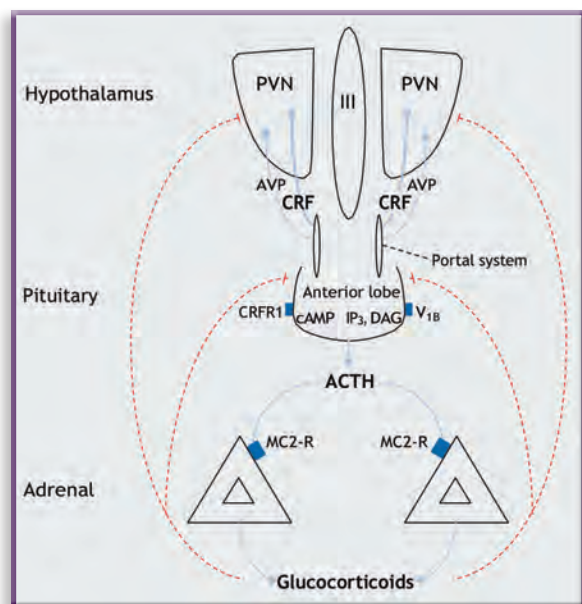
released into the bloodstream to regulate metabolic, cardiovascular, immune, and behavioral processes.¹⁸ When sufficient glucocorticoids are detected in the body, the hypothalamus inhibits the production of CRF through two mechanisms. The first is not fully understood, but regulation may occur through sensing the rate at which glucocorticoids are secreted.¹⁸ The second system is the traditional HPA feedback, which is well known. Glucocorticoids bind with receptors in the PVN and possibly in the hippocampus, allowing for modification of the HPA.¹⁸ Thus, the HPA axis is a negative feedback system.

The body naturally produces cortisol as a stress response to inflammation. In some cases, steroidal anti-inflammatory medications are given to alleviate swelling. They alleviate the inflammation by inhibiting the release of arachidonic acid from the plasma membrane, which limits the synthesis of all eicosanoids, thereby alleviating inflammation.¹⁷

Oral glucocorticoids have been used since the early 20th century. They are effective in treating a variety of inflammatory processes. In 1935, two American physicians, Dr Philip S. Hench and Dr Edward Kendall, began researching the adrenal glands. By 1940, with the collaboration of Dr Tadeus Reichstein from Switzerland, they had isolated 28 compounds from the adrenal glands.¹⁹ On 21 September 1948, the compound known at the time as compound E (cortisone) was given to a patient, with positive results.¹⁹ In 1950, Drs Hench, Kendall, and Reichstein were awarded the Nobel Prize in Physiology and Medicine for the discovery of cortisone.¹⁹ The discovery of cortisone provided the required research and base structure needed for the development of the oral glucocorticoids used today.

Oral glucocorticoids are used to treat various types of dermatitis, but they are generally only given in severe cases of dermatitis involving more than 20% of body surface area.¹⁶ The first-line treatment option for glucocorticoids given for severe cases of dermatitis is prednisone, 0.5 to 1mg/kg, up to a maximum single dose of 60mg PO daily for 5 to 7 days.¹⁶ After that time, if the patient is doing well on the initial dose, then the dose can be reduced by one-half for the next 5 to 7 days.¹⁶ This tapering can continue until the patient is at a low enough dose that withdrawal symptoms will not be severe. Tapering of steroids is of extreme importance, especially when dealing with oral glucocorticoids. If a course of steroids is not properly tapered, the patient can develop various complications, such as HPA axis suppression and rebound dermatitis, due to rapid discontinuation of steroids. A steroid “dose pack” is insufficient to treat a topical allergic dermatitis and should not be used.^{8,16} The American Academy of Dermatology guidelines for the treatment of contact dermatitis

Figure 2 HPA axis schematic.



Source: Used with permission from Smith SM, Vale WW. The role of the hypothalamic-pituitary-adrenal axis in neuroendocrine responses to stress. *Dialogues Clin Neurosci*. 2006;8:383–395.

recommend topical treatment alone for mild cases of contact dermatitis, defined as a “limited site of involvement, acute contact dermatitis when the offending agent has been removed, or chronic contact dermatitis with limited symptoms.”²⁰ The guidelines state that systemic treatment may be indicated to control itching or edema, for moderate to severe cases.²⁰ The systemic treatments listed by the American Dermatological Association include oral or intramuscular glucocorticoids but do not indicate duration of treatment.

Conclusion

Few studies exist that define best practices for oral steroids with allergic dermatologic conditions. Some of the more-cited publications report on exposure to *Toxicodendron* (poison ivy, sumac, oak), and even those do not provide consensus for the provider on when the condition is “severe” enough to warrant oral steroids.^{15,16,21,22} Based on the literature and cited publications, it is recommended that the use of oral glucocorticoids for dermatitis-related illnesses should be reserved for only severe cases (greater than 20% body surface area) and should be prescribed in a tapering dose to be taken over 15 to 21 days. It is the opinion of the authors that the liberal use of oral and IM glucocorticoids for contact/allergic dermatitis is unwarranted in most cases and these patients should be treated with topical glucocorticoids and/or an antihistamine. In the case of the 21-year-old patient with allergic dermatitis secondary to bed bug bites, we concluded that there was a high probability that the patient had developed rebound dermatitis due to the improper administration of oral glucocorticoids administered at the previous two clinics the patient attended before attending his BAS.

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Disclosures

The authors have nothing to disclose.

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