Lichenoid Contact Dermatitis Induced by Semen Sinapis Albae

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The clinical manifestations of contact dermatitis are varied and present as either eczematous or noneczematous eruptions [1]. Lichenoid contact dermatitis (LCD) is a rare noneczematous form of contact dermatitis. It is caused by multiple factors, including exposure to plants, drugs, and chemicals [2]. Nonetheless, to the best of our knowledge, lichenoid contact dermatitis induced by an acupoint herbal patch (AHP) with Semen Sinapis Albae (SSA) has never been reported.

A 55-year-old Chinese man was evaluated for a 4-month history of nummular eruptions on an area treated with AHP. He had a 14-year history of chronic obstructive pulmonary disease with repeated exacerbations in winter. Following a prescription from a herbalist, AHP with SSA seeds was applied twice, on June 12 and 22, 2017. Twenty-four hours after the second application, each patch was detached with no noticeable cutaneous abnormalities. One month later, pruritic erythema and pin-sized papules appeared at the site. The primary eruptions became progressively pigmented and coalesced and did not fade with discontinuation of SSA or recede with the administration of topical corticosteroids and antihistamines. The patient was otherwise in good health and denied exposure to SSA before treatment of AHP. Skin examination revealed multiple nummular lesions distributed bilaterally along the spine, each overlaying a specific Back-shu acupoint. The lesions comprised demarcated erythematous macules and plaques. Some of the lesions were entirely pigmented or overlapped with pin-sized papules (Figure, A). A skin biopsy displayed parakeratosis, acanthosis, and focal spongiosis in the epidermis. An infiltration of band-like mononuclear cells was visible along the dermoepidermal junction. There was no evidence of liquefaction of the basal cell layer or hyaline bodies (Figure, B). The patient was diagnosed with lichenoid contact dermatitis induced by SSA. The lesions did not improve with 1 month of topical application of 0.1% tacrolimus and persisted during follow-up.

The remedy of AHP was firstly introduced in the Chinese medical book Prescriptions for Fifty-two Diseases written in 350...
the Western Han Dynasty (206 BC to 25AD). This traditional approach involves applying Chinese herbs onto specific acupoints. The most common herbs applied during the AHP are Semen Sinapis Albae (白芥子), Herba Asari (细辛), Radix Euphorbiae kansui (甘遂), and Rhizoma Corydalis (延胡索) [3], which can be used individually or as a combination of several herbs. AHP has already become a prevalent treatment option for variable chronic diseases. Millions of patients in China undergo such complimentary therapy annually on the 3 dog days because of the highest potential of the Yang at these times. In traditional Chinese medicine, it is believed that AHP reverses Yang deficiency syndrome [4]. Therefore, AHP is carried out during the dog days to ensure early intervention in a disease that is quiescent in summer with a high possibility of relapse or exacerbation in winter. Chronic obstructive pulmonary disease, asthma, and chronic bronchitis all fall within the spectrum of diseases suitable for AHP [3,4].

SSA takes the form of small pungent seeds of the Brassicaceae family. It is widely used not only as a seasoning and spice, but also as a medicinal plant [5,6]. Since SSA disperses sputum and eliminates cold to invigorate the Yang, AHP may function synergistically with the herb SSA if applied on the 3 summer dog days. Acute allergic reactions have been reported during topical application of AHP with SSA. The most common involve eczematous lesions with an itching/burning sensation shortly after exposure [5]. However, noneczematous presentations, such as lichenoid contact dermatitis, are quite rare.

Lichenoid contact dermatitis is a particularly unusual type of contact dermatitis caused by exposure to amalgams and nickel [7]. Other uncommon causative allergens include methylisothiazolinone, aminoglycoside antibiotics, methacrylic acid esters, methylchloroisothiazolinone, paraphenylenediamine, and epoxy resins. Histopathology reveals hyperkeratosis, acanthosis, mild spongiosis, and exocytosis. An infiltration of band-like mononuclear cells is noted along the dermoepidermal junction, although there is no evidence of liquefaction of the basal cell layer or hyaline bodies [1,2]. Lichenoid contact dermatitis should be differentiated from other types of noneczematous contact dermatitis such as erythema multiforme, purpura, and the lymphomatoid, pigmented, pustular, dyshidrosiform, and lichenoid subtypes [8]. The clinical and histopathological data recorded in the present case made these diagnoses unlikely.

We propose that SSA may act as a major contact allergen, so that a single exposure could sensitize the predisposing individual even 1 month later. Similar reactions have been observed with epoxy resin, acrylates, some hair dyes, and black henna for tattoos [9]. However, the pathogenesis of lichenoid contact dermatitis with SSA remains to be elucidated. We present the first case of lichenoid contact dermatitis after exposure to SSA. Clinicians should include lichenoid contact dermatitis among the differential diagnoses when there is microscopic evidence of interface dermatitis.

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**Conflicts of Interest**

The authors declare that they have no conflicts of interest.

**References**

Successful Desensitization to Brentuximab After Anaphylactic Shock

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Monoclonal antibodies target specific proteins associated with pathogenesis. Brentuximab vedotin (BV) is a CD30-directed antibody-drug conjugate. It has significantly improved the management of patients who have experienced relapses after autologous stem cell transplantation (AUTO-SCT) and can induce durable remission in a subset of patients with relapsed/refractory Hodgkin lymphoma (R/R HL) [1].

As BV is an intravenous chimeric monoclonal antibody, acute infusion reactions to it are not surprising. However, few immediate-type hypersensitivity reactions (HSRs) and desensitization attempts for such reactions have been reported [2-6].

Rapid drug desensitization (RDD) was developed for the delivery of biologic agents that cause immediate-type HSRs by inducing temporary tolerance. However, desensitization protocols for monoclonal agents are seldom used [7]. We have been using RDD, as developed at the Brigham and Women's Hospital, for patients who experience immediate-type HSRs [7,8]. We report the case of a patient with relapsed HL who was successfully desensitized to BV with RDD despite having a history of BV-related grade 3 anaphylaxis.

A 34-year-old man was admitted to the hematology clinic with cervical, paratracheal, subcarinal, and preaortic lymphadenopathy in 2011. Analysis of a biopsy specimen from the cervical lymph node revealed classic HL, and 6 doses of doxorubicin, bleomycin, vinblastine, and dacarbazine (ABVD) were administered. The patient remained in remission until 2016, when he developed generalized itching and multiple lymphadenopathies; recurrence of classic HL was detected. Two cycles of cisplatin, cytarabine, and dexamethasone were given, and AUTO-SCT was planned. Four cycles of BV, etoposide, methylprednisolone, cytarabine, and cisplatin were administered without problems. AUTO-SCT was subsequently performed after high-dose conditioning treatment (BCNU/Etoposid/ARA-C/Melfelan). Unexpected early disease progression was observed at the first post-AUTO-SCT follow-up examination, prompting an immediate decision for allogeneic stem cell transplantation. BV was given again.

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