

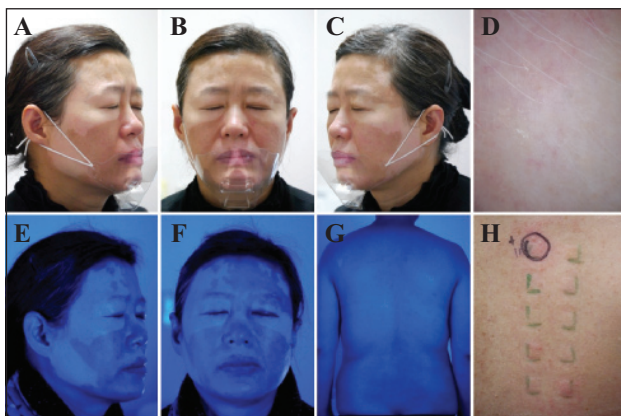


Figure 1. Contact vitiligo induced by daily contact with rubber ear loops of disposable sanitary masks. **A-C)** Depigmented patches on both mandibular areas, the preauricular and periorbital areas, and forehead. **D)** Poliosis is evident in the depigmented patches. **E-G)** Wood's lamp examination accentuated the depigmented areas. **H)** The patch test was positive for IPPD at 48 hours.

ical leukoderma syndrome" has been coined to define this phenomenon, which is classified into four stages. Stage 1 refers to depigmentation at only the site of contact, Stage 2 to local spread via the lymphatic system, Stage 3 to distant hematogenous spread, and Stage 4 to distant spread even after a year without exposure to the causative chemical [2]. The term "contact vitiligo" would best describe this case, because depigmentation progressed from the contact area, due to a suspected causative material, to distant areas, and poliosis was widely observed in the depigmented skin. In our case, contact dermatitis did not precede and the patch test result was not sufficiently strong, however, we feel that the positive patch test result for IPPD is worthy of mention, despite the unclear relevance. We hope that our case contributes to our understanding of the aetiology of contact vitiligo. ■

Disclosure. Financial support: none. Conflicts of interest: none.

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doi:10.1684/ejd.2018.3350

A case of chronic prurigo successfully treated with omalizumab

Chronic prurigo is characterized by the presence of chronic pruritus and multiple pruritic lesions accompanied by signs of repeated scratching [1]; it can be classified into several subtypes: papular, nodular, plaque, or umbilicated [1]. The pathological mechanism of each subtype has not been fully elucidated. Although multiple therapies (e.g. topical steroids, UV phototherapy, antihistamines) have been used to reduce inflammation and itch [2], some patients are difficult to treat. We have previously shown that basophils are frequently observed in the lesions of patients with chronic prurigo [3], and basophil activation status in blood, as determined by cell surface CD203c expression levels [3], is higher in patients with chronic prurigo than in healthy donors. Therefore, basophils could be involved in the pathogenesis of prurigo. Here, we report a patient with chronic papular-plaque-type prurigo who was successfully treated with the anti-IgE agent, omalizumab, which affected basophil function.

A 47-year-old Japanese man presented with a five-month history of itchy rashes on his trunk and extremities that had been unsuccessfully treated with low-dose prednisolone and topical steroids. Severe itching induced insomnia. Physical examination revealed annular urticarial erythematous macules, papules, and nodules, accompanied by scratch marks over his trunk and extremities (figure 1A-C). Biopsies of both a plaque of the arm (figure 1C) and a papule of the trunk (figure 1A) were taken. Microscopic examination of the biopsy from the plaque showed slight subepidermal oedema with lymphocytic and eosinophilic infiltration around blood vessels of the upper dermis (figure 1D), whereas the papule showed slight hyperkeratosis, parakeratosis, and acanthosis with lymphocytic and eosinophilic infiltration around superficial blood vessels and between collagen fibres in the upper dermis (figure 1K). To investigate the presence of basophils in these lesions, we performed immunohistochemical staining for basophils using BB1, a basophil-specific monoclonal antibody [4]. Although such infiltration was detected in both lesions, considerably more basophils were detected in the plaque than in the papule. In the plaque, BB1+ basophils were observed in a perivascular location and infiltrated among collagen fibres of the upper dermis (figure 1J). The number of dermal basophils reached 6.33 ± 0.94 cells/high-power field (hpf). In the papule, a slight infiltration of basophils was detected between collagen fibres in the upper dermis (figure 1L), with 2.00 ± 0.82 dermal basophils/hpf. Laboratory tests revealed blood leukocytopenia without concomitant eosinophilia or basophilia. Although serum levels of total IgE were normal, IgE antibodies to house dust mites were detected.

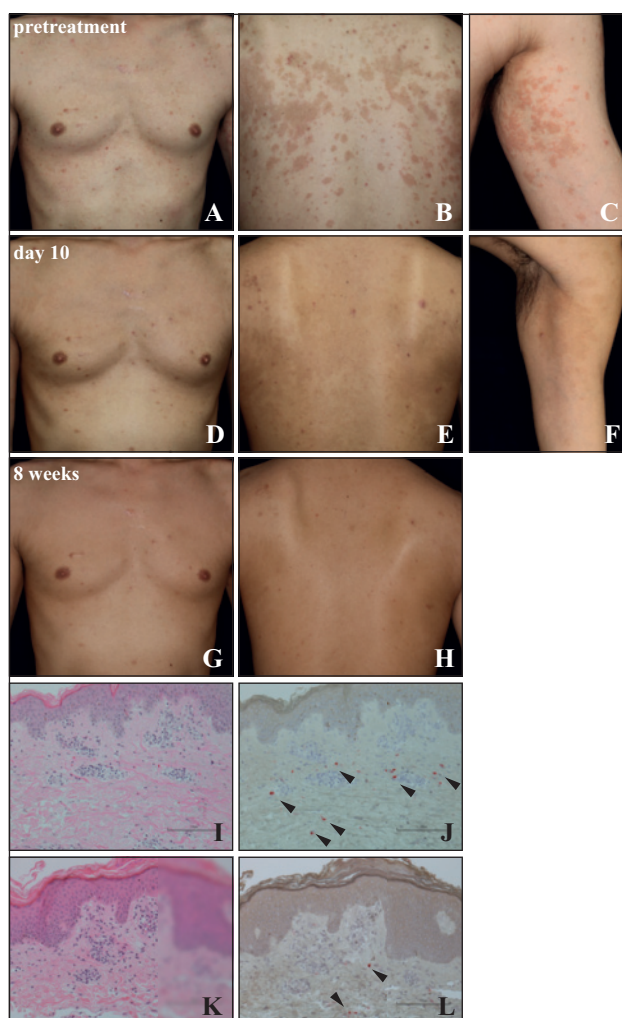


Figure 1. A-C) Cutaneous manifestations at the first visit showing erythematous macules and papules over the trunk (A, B) and arm (C). D-F) Erythematous macules and papules on the trunk (D, E) and arm (F) significantly diminished 10 days after the first injection of omalizumab. G, H) The eruptions disappeared two months after initiation of omalizumab therapy. I) Lymphocytic and eosinophilic infiltration around dermal capillaries (hematoxylin-eosin [H&E]; $\times 200$). J) Basophils (arrowheads) around the dermal capillaries and between collagen fibres (reaction products visualized using New fuchsin; $\times 200$). K) Slight hyperkeratosis, parakeratosis, and acanthosis with lymphocytic and eosinophilic infiltration around superficial blood vessels and between collagen fibres (H&E; $\times 200$). L) Basophils (arrowheads) between collagen fibres in the upper dermis (reaction products visualized using New fuchsin; $\times 200$). Scale bar: 100 μm .

After the diagnosis of chronic prurigo was made, the patient was treated with narrow-band ultraviolet B phototherapy, antihistamines, and topical steroids, but the eruptions continued to flare up periodically. Subsequently, 300 mg of omalizumab was administered once every four weeks. The eruptions significantly improved within 10 days after the first omalizumab treatment (figure 1D-F). Two months after the initiation of omalizumab therapy, the eruptions had almost disappeared (figure 1G, H). Therefore, omalizumab therapy was terminated after the third injection. There-

after, the patient was treated with antihistamines and topical steroid ointment without recurrence of either cutaneous lesions or itching.

Chronic prurigo is difficult to treat. In our patient, chronic papular-plaque-type prurigo was successfully treated with omalizumab, a recombinant, humanized, monoclonal anti-IgE antibody which is specific to the Fc region domain of IgE molecule that binds to Fc ϵ RI [5]. Omalizumab binds to free serum IgE and interferes with IgE binding to Fc ϵ RI on mast cells and basophils; this interference downregulates Fc ϵ RI and suppresses mast cell and basophil activation [5]. In our patient, omalizumab exerted sufficient therapeutic effects within 10 days after treatment initiation, which is consistent with the life span of basophils [6]. These findings suggest that basophils play an important role in the pathophysiology of chronic papular-plaque-type prurigo. Previous reports in mice [7, 8] have shown that basophils act as initiators of IgE-mediated chronic allergic inflammation, with clinical features similar to those of chronic plaque- [7] or papular-type [8] prurigo. Therefore, the therapeutic effect achieved in our patient was likely due to the inhibition of basophil activation. Thus, omalizumab could be a good therapeutic option for the treatment of chronic prurigo. ■

Disclosure. *Acknowledgements.* We thank Dr. Andrew F. Walls of Southampton General Hospital, UK, for kindly providing the BB1 antibody and Chiyako Miyagishi for technical assistance. *Financial support:* this work was supported by AMED under Grant Number 17ek0410017h0003 (HY). *Conflicts of interest:* none.

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doi:10.1684/ejd.2018.3351