Therefore, the maintenance of aTreg levels might prevent the disease progression and be a treatment modality for port wine stains. Combined topical RPM regimens. In conclusion, topical RPM does not seem to be effective as a treatment modality for port wine stains.

Activated regulatory T cells in patients with alopecia areata for suppressing disease activity

Alopecia areata is one of refractory inflammatory skin disorders. However the precise mechanism remains obscure and appropriate treatment for severe cases should be developed even patients experience self regression. We previously evaluated the number of circulating CD4+ CD25+ regulatory T cells were increased substantially, but not stastically in patients with alopecia areata (1.11 ± 1.24% vs. 3.81 ± 1.12%, p = 0.074). In the present study, we examined three distinct Treg subsets: activated Treg (aTreg), resting Treg (rTreg), and non-suppressive T cells (mTreg). aTreg have the strongest suppressive activity among the three subsets and rTreg are moderately suppressive. We examined these subsets from the peripheral blood in 12 alopecia patients. aTreg levels and rTreg + mTreg levels were significantly higher than those of healthy controls (0.15 ± 0.18% vs. 0.92 ± 0.44%, p = 0.022; aTreg + rTreg + mTreg: 3.22 ± 1.55% vs. 2.38 ± 0.44%, p = 0.045).

In the three-group comparison, alopecia, psoriasis (n = 15) and healthy controls, aTreg + rTreg + mTreg levels in alopecia were higher than those in psoriasis (3.22 ± 1.55% vs. 1.94 ±1.38%, p < 0.05). aTreg levels in patients not having alopecia totally were significantly higher than that of healthy controls (2.02 ± 0.95% vs. 0.92 ± 0.44%, p = 0.047). aTreg and rTreg levels were negatively correlated with the disease duration (aTreg: r = -0.308; aTreg + rTreg = -0.544). These results indicated that increased aTreg + rTreg would suppress the disease activity at early phase and induce spontaneous remission to some extent in alopecia patients. Therefore, the maintenance of aTreg levels might prevent the disease progression and be a target of new treatment modality.

Monogenic type I interferonopathies: from diagnosis to treatment

Mutations in genes involved in nucleic acid metabolism, sensing or associated signalling cascades can cause constitutive and sustained activation of type I interferon (IFN). In such monogenic conditions, the skin has emerged as one of the first affected organs, where cutaneous pathology, encompassing severe vasculopathy and ‘chilblains’, frequently results in extensive tissue damage and can provide a major clue to the diagnosis of this novel group of disorders. Recognition of the fundamental role of IFN in the pathogenesis of the type I interferonopathies led us to consider the blocking of IFN signalling as a logical therapeutic strategy. On this basis, we have treated patients mutated in either gain-of-function mutations between the CARD and coiled-coil domain; it was not observed during the study period. Therefore, JHT is seemingly effective against PPP.

Palmoplantar pustulosis (PPP) is a chronic skin disease characterized by sterile intraepidermal pustules associated with erythematous scaling on the palms and soles. The standard therapy for PPP patients includes topical corticosteroids, topical vitamin D3 analogs, oral cyclosporine A, pсорalen plus ultraviolet A therapy (PUVA) and narrowband ultraviolet (UV-B). However, clinicians often experience PP that is refractory to these treatments. Jhumaiadoko (JHT) is a traditional herbal medicine composed of ten medical plants and has been administered to patients with supplicative skin disease in Japan. This study investigated the effect of JHT on the disease activity in PPP patients (n = 10). PPP patients were administered JHT (20 drops, 6.0 mg per day; Krasse Holdings Ltd., Tokyo, Japan) for 4 to 8 weeks in addition to their prescribed medications. The disease severity of PPP was evaluated using the palmoplantar pustulosis index and severity index (P PPPAI). The PPPSI score was calculated using the following equation: PPPAI = P + S. Exacerbation, pustules and desquamation were evaluated on a scale of 0 to 3, while the area was evaluated on a scale of 0 to 4. The results showed that the average PPPAI of all patients was 8.34 ± 9.00 before JHT treatment. Four or 8 weeks after the beginning of JHT, the average PPPAI values significantly decreased to 3.84 ± 2.44 (p < 0.01). Seven out of 10 PPP patients showed an improvement in their clinical findings. In most of these patients, the number of pustules on the palms and soles markedly decreased. In addition, some patients showed a disappearance of hyperkeratotic lesions. No adverse event was observed during the study period.