number of micro vascular in the dermis, and neutrophilic micro-abscesses (Figure 3).

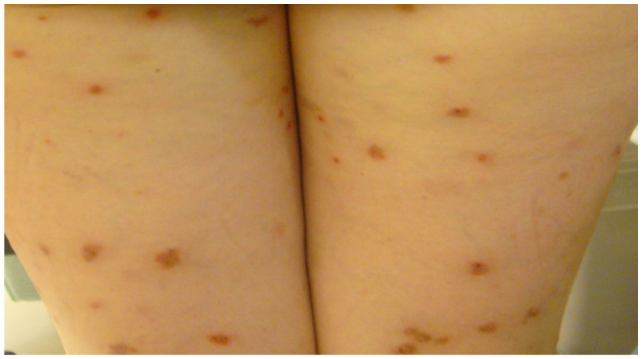

Figure 2: Clinical findings of new plaque on her thighs.

Laboratory data were immunoglobulin E (IgE) RIST, 384 IU/ml; eosinophils 2.5% (1-5%); thymus-and activation-regulated chemokine (TARC), 1359; and lactate dehydrogenase (LDH), 192 (120-240) IU/l; and the patient's biochemical data were within normal range. We started to treat her using cyclosporine 200 mg/day, second-generation anti-histamine tablets, steroid ointments, and Dovobet® ointment (calcipotriol hydrate and betamethasone dipropionate compounding ointmentunder a diagnosis of AD with psoriasis.

Her skin condition gradually improved, and now we treat her using narrow-band UVB phototherapy and steroid ointments. Her skin condition is under good control (Figure 4).

Discussion

The present report may provide critical evidence against the mainstream view that AD and psoriasis do no coexist. Although the non-coexistence of AD and psoriasis is theoretically correct, unexpected events may occur in the human body, leading to such a co-existence.

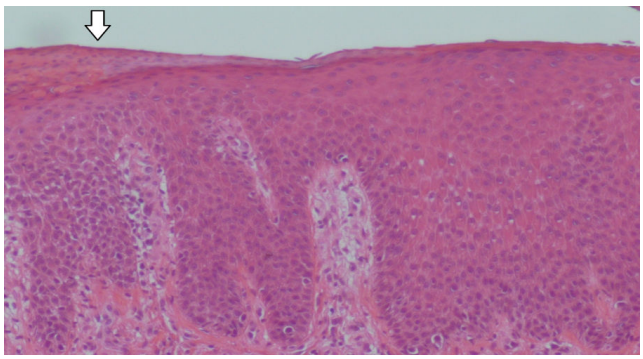

Figure 3: Histopathological findings of her new plaque. Allow indicate neutrophilic micro-abscesses.

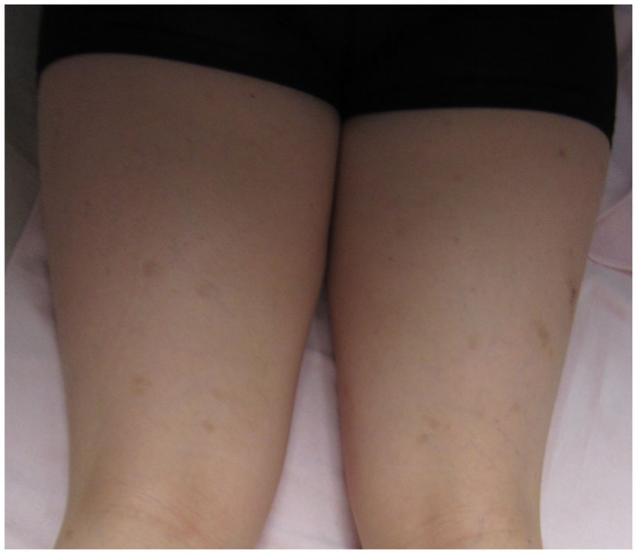

Figure 4: Clinical findings of her improved skin on the thighs.

Many physicians have investigated the genetic relationships between AD and psoriasis, and genomic imprinting in these two conditions shows different patterns [10]. These dermatoses are not hereditary disorders, but the incidence of children with AD from parents with atopic (allergic) disease are higher than that from parents or one parent without atopic (allergic) disease [11,12]. Stefanie [6] suggests that antigen-specific T-cell subsets determine the pathogenesis of psoriasis and atopic eczema. The immunological status of AD and psoriasis has been previously described. AD is a Th2/Th22-dominant [1] disease, and psoriasis is a Th1 and/or Th17 disease [1,13]. AD causes Th2-dominated immune reactions through the elevation of allergen-specific IgE [2], while psoriasis is characterized by Th1 immune reactions through the elevation of interleukin-17A, -17F and -22 [3]. The results of tumor necrosis factor α (TNF-α) antibody injections indicate that blocking with a specifically targeted agent against Th1-mediated and Th17-mediated immune disease may result in a flare of Th2-mediated disease [6]. Stefanie [6] reports that distinct T-cell subpopulations were found to infiltrate the same organ: predominantly Th1 and Th17 cells in psoriasis, and Th2 cells and Th22 cells in atopic eczema. Previous reports have also shown that acute AD has a largely Th2-mediated action, but the chronic phase of AD is more Th1-mediated [4,14,15]. Our speculation is that psoriasis might be a transition of the immunological skin situation due to allergens or irritant reactions, because we confirmed by patch test in psoriasis patients that eczema reactions are positive reactions.

Previous studies on chemokines and these dermatoses have shown that C-C motif chemokine ligand 1 (CCL1) is associated with AD, and that both CCL1 and CCL18 may play important roles in the initiation and progression of atopic skin inflammation [16]. Additionally, C-C motif chemokine receptor 4 (CCR4) and CXCR3 were found to be preferentially expressed on CD4+ T cells from AD and psoriasis vulgaris, respectively [17]. Uchida [17] showed that CCR4 and CXCR3 expression on peripheral blood CD4+ and CD8+ T cells in adult AD is greater than that in patients with psoriasis vulgaris and healthy controls.

Th2 cells selectively express CCR3, CCR4, and CCR8, while Th1 cells selectively express CXCR3 and CCR5 on their surface [18-21]. The chemokine receptor predominantly expressed on Th1 cells is CCR5 [21]. Uchida [17] demonstrated that the expression of CCR5+ cells increases in the affected areas of AD and psoriasis patients, but psoriasis patients possess cells that express CCR5 rather than CCR4. Not all chemokines can be completely classified based on Th1 or Th2 reactions, and each chemokine overlaps with Th1 and Th2 cells in affected areas.

Generally, dry skin (xerosis cutis) is more likely to lead to contact dermatitis than normal skin. Psoriasis areas show a lot of scale, and it may therefore be easy for allergic contact dermatitis (ACD) to arise via psoriasis affected areas. Our impression is that the number of people with psoriasis and ACD is smaller than that of those with AD and ACD, and that this may depend not only on Langerhans and other dendritic cells in AD and psoriasis and other immune skin conditions, but also on Th valance and chemokines.

The ligand of Th2-type CCR (CCR4) is TARC [22]. TARC are produced by skin cells in AD, and CCR-4 expressed cells are attracted to the AD lesions. Serum TARC levels are markedly elevated and epidermal keratinocytes strongly express TARC in AD [23]. These previous reports indicate that TARC level could be reflected as inflammation levels (markers) of AD. In the present study, our patients' TARC levels were not high, but our findings show a transition from acute to chronic stage as TARC and IgE RIST increase. These findings support the diagnosis of AD.

The pathogenesis of AD and psoriasis expressed in terms of Th cells remains to be fully confirmed, and additional clarification is required regarding unexpected immunological events that could develop in the human body. Further investigation is needed on the immunological situation of the coexistence of AD with psoriasis.

Conclusion

We reported a case of AD coexisting with psoriasis vulgaris in adult female. The pathogenesis and immunopathology of comorbidity in adult with AD and psoriasis are not classified completely. It needs further investigations.

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