Pemphigus: a moroccan retrospective study over 30 years
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Pemphigus is an autoimmune blistering disease, through this hospital series, we aim to describe our clinical experience and outcomes in pemphigus. We performed a unicentric retrospective study of all patients admitted to the university hospital Ibn Sina Rabat Morocco with the diagnosis of pemphigus from 1990 to 2020 (over 30 years). Inclusion criteria; age, gender, mean duration of the disease, mortality rate, treatment, disease course. The diagnosis was mainly clinical and confirmed by histopathology, direct immunofluorescence and indirect immunofluorescence. There were 302 cases, 26 new patients in 2020. The most common variant was pemphigus vulgaris 125 cases followed by pemphigus seborrhoeic 99 cases, foliaceus 40 and vegetans 27 cases. sex-ratio 0.75, the average age; 53 years old, the mean duration of the disease before diagnosis was 13, 36 months, severe PDAD87.6%. Oral corticosteroids therapy prednisone was given to 113 cases, methylprednisolone to 12 cases. Adjuvants treatment with azathioprine (93 cases), disulfane (22 cases), cyclophosphamide (13 cases), methotrexate (10), thalidomide (10), tacrolimus (13 cases), azathioprine and rituximab (313 cases). Clinical remission (133 cases) (mean duration 2.5 months), lost to follow-up (19 cases), relapses (89 cases) (mean duration 51 months), death (33 cases). In our series, no statistical differences have been noted concerning the duration of the remission between oral steroids only and the association with a immunosuppressive agent. For economical reasons azathioprine is the most used sparing agent. The duration of treatment is very prolonged, we suggest a progressive tapering of steroids. Our recommendations are to discontinue treatment in a patient with clinical remission on low doses of oral steroids is the absence of new lesions and circulating antibodies. Usually over than 2 years with a close monitoring of drug side effects.

Exclusion of hallo nevus without PD-L1 expression may cure vitiligo
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Haloe nevus also called as Sutton nevus, is a nevus cell nevus that is surrounded by vitiligo and is caused by a T-cell mediated immune response to the nevus antigen. Exclusion of nevus is recommended to prevent progression to vitiligo vulgaris. When vitiligo may occur around malignant melanoma, hemangoma, blue nevus, neurofibromata, seilie wart, etc. it’s called Sutton’s syndrome. Especially, vitiligo in melanoma patients is called “melanoma associated vitiligo” and recognized to associate with favorable outcomes. It’s sometimes observed in patients who respond to immune checkpoint inhibitor (ICI) therapy as a “preference” immune-related adverse event (irAE), the mechanism has attracted attention. However, there are few reports on analyses of immune checkpoint including PD-1/PD-L1 in the skin. In the present study, to evaluate the relationship between immune checkpoint including PD-1/PD-L1 and vitiligo, we examined immune checkpoint expression using skin samples fixed paraffin-embedded FFPE (FFPE) samples collected from 38 HN patients (16 males and 22 females, mean age 20·7 years) whose nevus were resected in Nagoya City University Hospital. Of the 29 cases that we were able to follow after nevus excision, 12 cases showed improvement of the surrounding vitiligo and 17 cases showed no change or expansion of vitiligo. 25 cases have vitiligo vulgaris, and 10 of them showed improvement of vitiligo vulgaris after HN excision. Immuno-histochemical findings showed that PD-L1 expression was positive in 25 of 36 cases, and cases with improved vitiligo after nevus excision were predominantly PD-L1 negative. PD-1 expression in infiltrating cells was positive in 19 of 38 patients, but there was no significant correlation between PD-1 expression and improvement of vitiligo. These results suggest that more than local immune responses are involved in vitiligo formation in PD-L1 positive HN, and the analysis of immune responses in PD-L1 positive HN may lead to elucidation of the mechanisms of tumor immunity and irAEs. Impact of Staphylococcus aureus colonisation on barrier function and cytokine profile in atopic dermatitis skin
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Atopic dermatitis (AD) is a widespread skin disease that is characterized by lesions at specific body sites, which are typically driven by a type 2 immune response. While the active state is well-researched and a broad variety of treatment options exists, lesions often recur in a matter of weeks after treatment cessation. Recent research suggests that clinically healed AD skin is still distinguishable from healthy skin through increased subclinical inflammation. To understand the pathophysiology of relapses in atopic dermatitis, a field for which hardly any research has been conducted, we have developed a murine transgenic mouse line, Dc14A mice, in which deletion of exon 18 in Col17a1 results in the absence of NC14A domain, corresponding to human NC16A domain, the main autoantigen in BP. Col17a1 levels in serum were detected by ELISA. Inflammatory cell populations in spleen and draining lymph nodes were analysed by flow cytometry. Symptomatic DC14A mice had increased proportion of effector T cells, myeloid derived suppressor cells and CD11b+ myeloid cells in both spleen and lymph nodes. In addition, proportion of B cells was increased in lymph nodes. Because anti-IL-17A-therapy has a potential as BP treatment we treated symptomatic DC14A mice with anti-IL-17A. The treatment restrained symptoms, but histologically inflammation was similar to untreated lesion skin. Our results suggest that structural modifications of col17a1 reflect the state in BP skin where autoimmune reaction results in the decrease of col17a1 and blister formation. Thus, DC14A mouse line could be further utilized as a potential BP-model.