031 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 clinic-related experience 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, foliaceous 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 PDAD76%. Oral corticosteroids therapy prednisone was given to 113 cases, methylprednisolone to 12 cases. Adjutants therapy with azathioprine (93 cases), disulone (22 cases), cyclophosphamide (3 cases), methotrexate (11 cases), cyclosporine (3 cases), rituximab (17 cases), methotrexate and rituximab (13 cases), azathioprine and rituximab (3 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 for discontinuate 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 drugs side effects.

032 Impact of Staphylococcus aureus colonisation on barrier function and cytokine profile in atopic dermatitis skin
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Atopic dermatitis (AD) is a chronic inflammatory skin disease characterized by a dysregulated immune response associated with barrier dysfunction and dysbiosis of the skin microbiota characterized by Staphylococcus aureus colonization in lesional skin. The aim of our study was to compare non-lesional and lesional AD skin transcriptional profile depending on staphylococcal colonization. Skin biopsies were analyzed by RT-qPCR in moderate and severe adult AD patients. Staphylococcus epidermidis was detected in all lesional skin, in greater abundance than in non-lesional skin, while S. aureus colonization was found in approximately 50% of AD patients. Our results suggest a greater decrease of gene expression in lesional skin when infected by S. aureus compared to S. epidermidis. We suggest that targeting S. aureus might be more promising in AD patients than S. epidermidis.

033 Excision of halo nevus without PD-L1 expression may cure vitiligo
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Halo nevus is also called vitiligo 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. Excision of nevus is recommended to prevent progression to vitiligo vulgaris. When vitiligo may occur around malignant melanoma, hemangoma, blue nevus, nevus sebaceus, sebile, etc, it's called Sutton's nevus. Especially, vitiligo in melanoma patients is called "melanoma-associated vitiligo" and recognized to associate with favorable outcomes. It has been sometimes observed in patients who respond to immune checkpoint inhibitor (ICI) therapy as a "preferable" 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 present study. In this study, we evaluated the relationship between immune checkpoint including PD-1/PD-L1 and HDN, we performed immunomicroanatomical analysis using skin biopsies fixed in formaldehyde and/or (FFPE) samples collected from 38 HDN patients (16 males and 22 females, mean age 20.47 years) whose nevi 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 HDN excision. Immunohistochemical findings showed that PD-L1 expression was positive in 25 of 36 cases, and cases with improved vitiligo after nevus excision were predominately PD-L1 negative. PD-L1 expression in infiltrating cells was positive in 19 of 38 patients, but there was no significant correlation between PD-L1 expression and improvement of vitiligo. These results suggest that more than local immune responses are involved in vitiligo formation in PD-L1 positive HDN, and the analysis of immune responses in PD-L1 positive HDN may lead to elucidation of the mechanisms of tumor immunity and irAEs.

034 Dysfunctional Collagen XVII provokes skin inflammation
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Dysfunctional Collagen XVII provokes skin inflammation

035 Humanized anti-Desmoglein-3 antibodies as tools for research on the role of the neonatal Fc receptor in pemphigus vulgaris
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The autoimmune, blistering disease pemphigus vulgaris is caused by autoantibodies against the desmosomal cadherins, mainly desmoglein-3. Recently, it has been shown that blocking the neonatal Fc receptor (FcRn) can lead to a rapid decrease of pathogenic IgG and an improvement of various autoimmune diseases, including pemphigus, myasthenia gravis and autoimmune thymopathy. Blocking of IgG type antibodies to FcRn results in antibody recycling and increases the plasma half-life of pathogenic autoantibodies, contributing to disease suppression. Compounds that block the binding of IgG to FcRn, such as efgartimod, may be useful for the treatment of IgG-mediated autoimmune diseases. To study the pathogenic action of anti-desmoglein antibodies in vitro, mouse monoclonal anti-desmoglein-3 antibody (H11I) and human monoclonal anti-desmoglein-3 antibody (H5K23) have been developed that mimic the pathogenic effect of patient serum (PV IgG) in cultured keratinocytes. However, mouse IgG poorly binds to human FcRn. Therefore, to study the potential function of FcRn in pemphigus in human keratinocytes, we have generated recombinant anti-desmoglein-3 antibodies that contain human Fc domains. We show that these antibodies induce changes in desmoglein-3 localization and result in acantholysis in a monolayer dissociation assay in HaCaT keratinocytes. Surprisingly, the effects on keratinocyte adhesion can be inhibited by blocking IgG binding to FcRn by efgartimod. These data suggest that FcRn may not play a further role in the pathogenesis of pemphigus, beyond its known contribution to IgG recycling.

036 Identifying immune targets for atopic dermatitis relapse prevention
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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 exists, we conducted a clinical study involving more than 20 patients with recently healed AD skin. In this study, we aimed to investigate whether certain AD associated molecular markers in blood and skin indeed precede clinical relapse. By sharing our first results of the study, we hope to pave the way to a deeper understanding of flare prevention and new therapeutic interventions to delay or even prevent AD relapses in a targeted manner.