019 Herpes simplex virus infection in pemphigus patients: a prospective study
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This study aims to consider herpes simplex virus (HSV) infection in front of any severe, recalcitrant pemphigus lesions and to remove the possible long-term effect associated to their broad use of immunosuppressive agents. We performed a prospective study over 15 months (September 2019 - November 2020), 8 cases of PV associated with HSV infection were collected. 1 case of seborrheic, 3 cases of folliculosis, 4 cases of vulgans. PDIA score ranged between 23-102. Sex ratio F/M = 1, median age 48.8 years. The mean duration of PV was 5.5 months (2 patients were hospitalized for relapses and 6 had PV de novo). PV was confirmed on skin biopsy, direct and indirect immunofluorescence. The treatment was based on oralsteroids with azathioprine in 4 cases, rituximab in 3 cases. 3 of the patients had a history of VZV infection. HSV infection occurred with an average of 27.5 days after the onset of the treatment. Clinically herpetic lesions appeared as fissures, erosions and haemorrhagic crusts. The transcutaneous test was positive. Recovery was rapid in all patients treated with acyclovir 5% valaciclovir. Association between HSV and PV may result from immunosuppressive therapies and/or a causative factor associated with treatment-resistant lesions. The mechanism of viral induction of autoimmunity can be explained in several ways including molecular mimicry. Upregulation of production of interferon and interleukins, high level of IFN leads to increased expression of IL-12 on keratinocytes making the herpesvirus antigenic structure site active. Over production of IL-4 and IL-10 causes a shift of TH1 to TH2 response which potentiates antibody-dependent cell lysis. Pemphigus is characterized by antibodies against desmosomal proteins/E-cadherin, the formation of functional tight junctions and the stratum corneum are a major physical barrier for HSV invasion into tissue. These observations suggested the recognition of herpetic infection helps to avoid unnecessary changes of immunosuppressive treatment for presumed refractory pemphigus.

021 A retrospective analysis of the clinical, biomedical, immunological, histopathological and radiological spectrum of Systemic Lupus Erythematosus at a tertiary care centre in North India
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The objective of our study was to retrospectively analyse the clinical, biomedical, immunological, histopathological and radiological spectrum of Systemic Lupus Erythematosus (SLE) in North India. We retrospectively analysed the medical records of all patients who presented with SLE to a tertiary care centre in North India from January 2011 to August 2021. We included only records of patients with detailed information on history, examination, biomedical, immunological, histopathological and radiological investigations. We analysed the medhist of 531 patients, 345 (64.7%) patients were positive for anti-nuclear antibodies, 61 patients (11.5%) patients were positive for anti-dsDNA antibodies. The mean age of the patients with SLE was 29 years, of whom 382 (71.8%) were females. 226 (42.5%) patients had serositis, 26 (4.9%) patients had pericardial effusion, and three had pleural effusion. One patient had antibodies to lupus anticoagulant. The mean age of the patients with SLE was 31 years, of whom 391 (73.7%) were females. 226 (42.5%) patients had serositis, 26 (4.9%) patients had pericardial effusion, and three had pleural effusion. Seventy-eight (11.9%) patients were male, and the rest were female. Of the patients presented with fever, joint pain and photosensitivity. Nearly all patients had lesions suggestive of cutaneous lupus erythematosus. Thirty-two (6.0%) patients had a positive Direct Coomb’s test, and twenty-five (3.7%) patients presented with leukopenia. Sixteen patients had thrombocytopenia. Forty-four (4% 94%) patients had an elevated erythrocyte sedimentation rate. Thirty-two (6% 94%) patients had a positive anti-dsDNA antibody test. Seventeen patients had proteinuria. None of our patients had lupus nephritis. Two patients had paracardial effusion, and three had pleural effusion. One patient had antibodies to lupus anticoagulant. Twelve patients had low C3 levels, and seven patients had low C4 levels. This retrospective analysis presents an insight into the manifestations of systemic lupus erythematosus in North India and sheds light on the disease status and treatment response.

022 Exploring the potential of the novel IFN gamma antagonist TACG-X003 as a treatment for alopecia areata in pre-clinical models
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Alopecia areata (AA) is an autoimmune hair loss disorder in which hair follicles (HF) that have lost their protective immune privilege (IP) are attacked by an inflammatory cell infiltrate including key effector CD8+ /CD28+ cells rapidly turn into catagen. IFN gamma is recognized as a key pathogenic cytokine in driving IP collapse and AA pathology. Recently several IFN induced inhibitors are used off-label, despite the possible long-term effect associated to their broad use. We investigated the potential of the novel IFN gamma antagonist TACG-X003 as a treatment for alopecia areata in pre-clinical models aiming at identifying Desmoglein 3 (Dsg3) reactive CD4+ T cells by using fluorochrome-labeled HLA-DP, -DQ, -DR1, and -DQ5 tetramers loaded with Dsg3 peptides. Proliferation of CD4+ T cells was studied by CFSE-dye dilution in response to in vitro stimulation with Dsg3 peptide or peptides. Flow cytometric analysis enabled phenotyping of CD4+ T cells and identification of Dsg3-reactive CD4+ T cells by using fluorochrome-labeled HLA-DP, -DQ, -DR1, and -DQ5 tetramers loaded with immunogenic Dsg3 peptides. TACG-X003 did not affect proliferation of Dsg3-reactive CD4+ T cells in response to Dsg3 stimulation. In contrast to healthy controls, PV patients showed increased number of activated CD4+ T cells with a stimulatory capacity. Dsg3-reactive CD4+ T cells showed increased proliferation in response to Dsg3 peptides and showed IL-6 secretion was enhanced in PV even correlating with the clinical course. Our findings characterize Dsg3 reactive CD4+ T cells contributing to PV pathogenesis and thus serve as basis for the development of therapeutic targets in the future.