with oral acyclovir. Secondary bacterial infection occurred in 9% of the episodes. Disseminated disease or mortality was not reported in any patients. Significant post-herpetic neuralgia developed in 9% of the episodes. Patients with HZ reactivation were more likely to have first-time renal disease (78% vs 58%; p=0.02) and a shorter SLE duration at LN (31.4±50 vs 62.7±72 months; p=0.02) than those without HZ. A trend of higher SLEDAI score, higher anti-dsDNA titer, lower C3 and albumin level but higher rate of refractory renal disease was also observed in HZ-infected patients. Other clinical parameters such as histological classes of LN, neutrophil, lymphocyte counts and immunoglobulin levels at baseline and 6 months post-treatment were not significantly different between HZ-infected and control patients. HZ-infected patients had been treated with a significantly higher dose of prednisolone (0.72±0.40 vs 0.63±0.24 mg/kg/day) as induction therapy. Dosages of other immunosuppressive drugs were not associated with HZ reactivation. Logistic regression revealed first-time renal disease (OR 2.25[1.08-4.71]; p=0.003), peak MMA daily dose (OR 1.24[1.10-3.07]; p=0.02) and cumulative CYC dose (OR 1.14[1.01-12.8]; p=0.04) during induction therapy were significantly associated with HZ within 2 years.

**Conclusion:** HZ reactivation is fairly common in LN patients undergoing immunosuppressive therapies but unpredictable from histological and laboratory parameters. Higher doses of prednisolone, MMF and CYC were associated with a higher risk of HZ reactivation within 2 years.

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**Background:** Infective factors play a central role in autoimmune diseases pathogenesis. It is possible to speculate that the host genotype could interact with genetic background of infective agents. We previously evaluated a large SLE cohort, observing the association between the S. Aureus (SA) carriage status and presence of a more active disease in terms of autoantibodies positivity.

**Objectives:** We evaluated epidemiological, molecular characterization, genetic diversity and evolution of SA isolated from SLE patients by means of phylogenetic analysis.

**Methods:** Consecutive SLE patients (ACR 1997 criteria) were enrolled: clinical/laboratory data were collected and nasal swab for SA identification was performed. On the basis of translation elongation factor (tuf) gene, a phylogenetic analysis was performed to investigate phylogenetic relationships and to assess clades in patients with persistent carriage status (nasal swab positive in two consecutive evaluation, performed 1 week apart). The first dataset was composed by seven SA tuf gene isolated from different countries (downloaded from the GenBank database, https://www.ncbi.nlm.nih.gov/nucleotide/) and 51 tuf gene SA collected from SLE patients enrolled in the present study.

**Results:** We enrolled 118 patients (MF 10/198; median age 45.5 years, IQR 13.2; median disease duration 120 months, IQR 144). Skin involvement is the most frequent disease manifestation (86 patients, 72.9%), followed by joint involvement (78 patients, 66.1%). Twenty-four patients (20.3%) were SA carriers (SA+), three of them resulted MRSA. SA+ patients showed a significantly higher prevalence of joint involvement (79.2% vs 62.7%, P=0.01) and anti-dsDNA positivity (75.0% vs 55.3%, P=0.004). Moreover, SA+ SLE showed a more active disease, in terms of SLEDAI-2k values (SA+: median 2 [IQR 3.75] versus SA−: median 0 [IQR 2], P=0.04). The phylogenetic analysis has been restricted on the 21 non-MRSA SA+ patients. The maximum likelihood phylogenetic tree of the first dataset revealed a statistically supported larger clade (A, N=17) and a smaller one (B, N=4; figure 1A). SLE patients located in the clade A showed a significantly higher prevalence of joint involvement (88.2%) in comparison with clade B (50.0%, P=0.001) and SA− (62.7%, P<0.001, figure 2B). Moreover, haematological manifestations were significantly more frequent in clade A patients (64.7%) compared with B (50.0%, P=0.004, figure 2C).

**Conclusion:** The results of the present study confirmed the association between SA carriage status and disease activity, in terms of SLEDAI-2k values and anti-dsDNA positivity. The phylogenetic analysis on tuf gene show a clustering of SA+ patients in two major clade (A and B). Interestingly the tuf genotype of clade A is significantly associated with a specific disease phenotype, characterized by joint involvement and positivity for anti-dsDNA. These findings support the hypothesis that bacterial genetic variants may be associated with specific disease features.

**References:**