Objectives: Our aim was to analyze the association of HLA class II with HS in a Caucasian population from Cantabria (northern Spain).

Methods: In this study we analyzed the HLA-A,-B,-C DRB1,-DQA1 and -DQB1 allele distribution in 106 HS patients and 562 age- and sex-matched controls from a Caucasian population of Cantabria (northern Spain).

Results: HLA-A*29 and B*50 were significantly more frequent in HS patients and A*30 and B*37 in controls, but these associations disappeared after correction. On the other hand, DRB1*07, DQA1*02 and DQB1*02 were significantly more frequent in controls (p = 0.026, p < 0.0012 and p < 0.0005, respectively), and the HLA allele DQB1*0301 was significantly more frequent in HS patients (p = 0.00007) all of them after Bonferroni correction. Furthermore, the DRB1*07; DQA1*02; DQB1*02 haplotype was significantly more frequent in controls (p = 0.0005).

Conclusion: This is the first study showing an association of HLA-class II with HS. Our results suggest that HLA-II alleles (DRB1*07, DQA1*02, DQB1*02 and DQB1*02) and the DRB1*07-DQA1*02-DQB1*02 haplotype could influence on resistance or susceptibility to HS.

References:


Methods:
Objectives: To study the effect chronic generalized periodontitis on systemic methylation of TLR4 genes in comparison to only RA and RA with PD patients.

Methods: Twenty-three RA patients, among which 11 patients had chronic generalized PD, 20 patients with only PD and 15 healthy individuals recruited. DNA was isolated from PBMCs of the participants blood, then were first bisulfite converted and then methylation specific PCR were performed using DNA sequencing (Sanger) of the amplified products.

Results: The anti-CCP, DAS-28 and HAQ DI were higher in patients with both RA and PD (220±40, 5.7±0.2, 1.5±0.1 respectively, p<0.05). Control samples had shown amplification bands for methylated primers of TLR4 but not for unmethylated primers of TLR4. However, only RA, only PD and RA with PD samples, had shown amplification for unmethylated primers and not for methylated primers. These results together with DNA sequencing indicated that 4 CpG sites in the promoter of TLR4 genes were hypo-methylated in the PBMCs of patients whereas those remain methylated in healthy individuals.

Conclusion: The observations indicated that though PD is a localised disease of the gingiva there is a systemic involvement of TLR mediated pathways in them which is similar to those in RA. However, further validation in larger cohort and down-stream signalling molecules needs to be studied.

References:

Disclosure of Interests: None declared

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