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STAPHYLOCOCCAL TOXIC-SHOCK-SYNDROME-TOXIN-1 (TSST-1) IS A RISK FACTOR FOR DISEASE RELAPSE IN WEGENER'S GRANULOMATOSIS

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Nasal carriage of *Staphylococcus aureus* has been identified as a risk factor for disease relapse in Wegener's granulomatosis (WG). We hypothesized that local immunostimulation by secreted staphylococcal superantigens (SAg) may be responsible for this association.

We investigated the presence of *S aureus* and staphylococcal SAg in relation to the occurrence of relapses in a cohort of 63 patients with WG followed at our clinic (follow-up 53 ± 25.2 months). Patients were seen every 6 weeks and evaluated for signs and symptoms of active WG. A nasal swab culture was performed at every visit to detect *S aureus*. *S aureus* was identified by standard techniques. *S aureus* DNA was extracted and analyzed by multiplex PCR for the presence of genes encoding for staphylococcal exotoxin A to E (sea-see), exfoliative toxin-A (eta), and toxic-shock-syndrome-toxin 1 (tsst-1). Cox proportional hazards analysis of time to first relapse as dependent variable and presence or absence of *S aureus* and SAg as time-dependent covariates was used to analyse the association between *S aureus* and SAg and disease activity of WG. Results are expressed as relative risk (RR) with 95% confidence interval.

Of 1711 nasal cultures taken (mean 14, range 4 to 51 per patient), 709 were positive for *S aureus* (41%). Of these 709 *S aureus* isolates, 326 (46%) were positive for ≥1 SAg gene, most frequently sea (48%), followed by tsst-1 (37%), and sec (19%). Relapse of WG occurred in 35 patients (renal involvement in 19). In 27 of the 35 first relapse episodes, *S aureus* had been cultured in the 3 months preceding the relapse. Compared to the absence of *S aureus*, presence of a SAg-negative and SAg-positive *S aureus* were associated with a RR of 2.26 (0.99-5.14; p=.054) and 2.88 (1.17-7.07; p=.021) for relapse within 3 months, respectively. Analysis of individual SAg genes showed that only tsst-1 was associated with a significant risk for relapse (RR 13.36, 4.19-42.62; p<.001). The results were not different when the analysis was restricted to the 52 patients with at least 1 nasal culture positive for *S aureus*.

Conclusion: The association of nasal carriage of *S aureus* and relapse of WG was confirmed. Furthermore, the risk for relapse is modulated by the presence or absence of the staphylococcal tsst-1 gene, suggesting a possible pathogenic role for this superantigen in disease activation of WG.

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CHLAMYDIA PNEUMONIAE AND GIANT-CELL ARTERITIS: FAILURE TO DETECT *CHLAMYDIA PNEUMONIAE* IN TEMPORAL ARTERY BIOPSIES BY POLYMERASE CHAIN REACTION IN 90 CASES AND 90 CONTROLS

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Purpose: To examine the reported correlation between the presence of *Chlamydia pneumoniae* in temporal artery biopsy specimens and the diagnosis of temporal arteritis (TA).

Methods: We reviewed reports of all the temporal artery biopsies performed at our institution between 1968 and 2000, identifying 90 possible cases of TA. Seventy-nine of the biopsy specimens (88%) demonstrated giant cells. The other 11 cases (12%) had other histopathological features compatible with TA. Through a rigorous chart review, we confirmed that all 90 patients with positive biopsies met the 1990 American College of Rheumatology classification criteria for TA. We chose controls from the group of individuals who had negative temporal artery biopsies during the same time 32-year period. We reviewed the charts of potential controls to ensure that their post-biopsy courses were not compatible with TA, and matched one control to each case on 3 variables: gender, year of biopsy, and age within 10 years. The biopsies of all cases and controls were re-evaluated in a masked fashion by an experienced eye pathologist; all of the original readings were confirmed. Following de-paraffinization of the samples and DNA extraction, PCR analyses were performed for C *pneumoniae* on the 180 samples. We used 2 CDC-recommended sets of PCR primers (which target 2 different genes) for C *pneumoniae*. A primer set targeting the *ompA* gene (CP1-CP2/CPC-CPD) was used to perform a nested PCR, followed by confirmation of the findings with primers targeting the 16S rRNA gene in a touchdown enzyme, time-released PCR (CPN90/CPN91). We used positive and negative controls as well as controls made from infected and non-infected Hep-2 cells, suspended in a formalin-fixed, paraffin-embedded matrix.

Results: The results of PCR analyses are shown in the table below.

PCR primer set	Cases (positive TA bx) N = 90	Controls (negative TA bx) N = 90
ompA gene	1 (1.1%)	1 (1.1%)
16S rRNA gene	0	0

Conclusions: The results of this comprehensive study, which involved a large number of biopsy-proven cases of TA and matched controls and employed sensitive and specific PCR analyses, do not support an association of C *pneumoniae* in the pathogenesis of TA.