Poster presentation

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The phagocyte specific protein S100A12 as a novel biomarker in Muckle-Wells-Syndrome before and during therapy with Anakinra and Canakinumab (ACZ885)

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Background

S100A12 is a member of the damage-associated molecular pattern molecules (DAMP) and expressed by activated granulocytes exhibiting its proinflammatory capacity by binding to the Receptor for Advanced Glycation End-products (RAGE). The aim of this study was to evaluate S100A12 in MWS patients before and during therapy with Anakinra (IL1Ra) and Canakinumab (a fully human monoclonal antibody against IL-1 β).

Patients and methods

Eight patients (1 male, 7 female) with documented *CIAS*1 mutations (5 E311K, 2 T348M, 1 V198M) and active disease were included in this study. After Anakinra treatment therapy was switched to Canakinumab (ACZ885). Muckle-Wells-Syndrome Disease-Activity-Score (MWS-DAS), SAA, CRP, ESR and S100A12 values were recorded one day before (baseline) and during treatment with Anakinra (day 30–120) and Canakinumab (day 8).

Results

MWS-DAS, and all inflammation markers fell significantly from baseline to follow-up in both treatment groups. S100A12 was significantly lower with Canakinumab, than with Anakinra therapy (87 ± 64 ng/ml vs. 145 \pm 58; p < 0,05). With Canakinumab, 88% of values were within normal limits within one week of treatment (<120 ng/ml) whereas only 50% of values reached normal range with Anakinra. Correlation between S100A12 and MWS-DAS as well as ESR, CRP and SAA was significant (p < 0,05).

Conclusion

S100A12 is a sensitive marker of inflammation in patients with MWS and may be a valuable parameter to monitor subclinical inflammation. Data indicates that treatment with canakinumab, in contrast to anakinra, normalized S100A12 levels in the majority of patients.