Methods.— Five thousand sera of 3095 patients from three French hospitals were found IIF-ANCA positive from 2004 to 2012. PR3 and MPO specificities were assessed by ELISA, immunodot or ALBIA.

Results.— Twenty-eight IIF-ANCA positive patients had ANCA specific for both PR3 and MPO by at least one of the solid phase test (0.7% of serum, 0.9% of patients). None of the 23 patients with an available file had a necrotizing systemic vasculitis. No relevant clinical association was noticed.

Discussion.— We described 23 documented patients with both PR3 and MPO-ANCA. None of them had a SV, neither granulomatosis with polyangiitis nor microscopic polyangiitis nor Churg and Strauss syndrome. To the best of our knowledge, no cohort studies focused on such PR3 and MPO-ANCA. Only case reports or incidentally reported cases in serological studies appear in the literature [1,2,3].

Conclusion.— This cohort study demonstrates that, unlike the mono-specific PR3- or MPO-ANCA, ANCA with both anti-PR3 and anti-MPO activity, are not associated with SV, unlike few reports of sparse MPO and PR3-ANCA cases ascertained as SV with old laboratory tests.

References

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Synergistic effect of GCSF and LPS in ANCA Vasculitis
R. Popat, S. Freeley, M. Robson
King’s College London, London, United Kingdom

Introduction.— Granulocyte colony stimulating factor (GCSF) is a cytokine that is important in mobilizing neutrophils from the bone marrow and has proinflammatory effects. We have previously shown that serum GCSF is raised in patients with ANCA vasculitis and exacerbates disease in an established murine model [1]. LPS was given to all mice in the previous study, though it was not known if this was required with GCSF. We set out to investigate the relative role of GCSF and LPS in this model.

Methods.— Purified murine MPO was used to immunise MPO knockout mice to generate anti-MPO antibody. Four groups of wild type C57BL/6 mice were used (n = 5–6/group). They were given GCSF or control subcutaneously starting 8 days before the disease induction with anti-MPO (day 0). LPS or control was administered intraperitoneally on day 0 and 3. Serum, urine and histology was assessed on day 7.

Results.— The group which received both LPS and GCSF had significantly higher serum creatinine levels compared to mice with administration of neither LPS nor GCSF, administration of LPS alone or GCSF alone (12.3 ± 0.8 compared to 9.0 ± 0.5, 8.3 ± 0.6, 9.5 ± 0.4 umol/L respectively, P = 0.001). This group also had significantly more albuminuria compared to the other three groups (106 ± 18.24 compared to 15.7 ± 1.43, 27.21 ± 4.33, 21.75 ± 2.75 mcg in 24 hrs respectively, P < 0.0001). Furthermore, this group had significantly more glomerular crescents compared to the other three groups (31.6 ± 5.2 compared to 0 ± 0.3, 0.3 ± 0.3, 0.2 ± 0.2 per 100 glomeruli respectively, P < 0.0001) and glomerular macrophages (15.3 ± 1.3 compared to 0.5 ± 0.1, 2.2 ± 0.3, 4.1 ± 0.8 cells per glomerulus respectively, P < 0.0001).

Discussion.— This study shows that both LPS and GCSF are required to obtain robust disease in this model, with a strong synergistic effect on both histological and biochemical disease parameters.

Conclusion.— These findings have implications for investigators using this model and for our understanding of disease pathogenesis. They suggest that endogenous GCSF may be a therapeutic target.