METHODS: SLE patients fulfilling the 1982 ACR classification criteria who received RTX treatment at the Karolinska University Hospital during the years 2001–2015 were included. Storage serum samples obtained prior to and after six months from initiation of treatment were analysed for the detection of ADA using a GSK-developed and validated electrochemiluminescence assay. Disease specific screening and confirmation cut-point for SLE samples (1.44 AU/mL and 29% respectively) were used. Clinical and laboratory data were retrieved from electronic medical charts. SLE activity was measured using SLE disease activity index 2000 (SLEDAI-2K).

We defined treatment response according to the SLE responder index (SRI).2

RESULTS: Thirty-eight patients (89.5% females, median age 35.0 years; IQR: 27.7–55.0) were included in this retrospective analysis. The median disease duration was 6.2 years (IQR: 2.1–11.6) and the baseline median SLEDAI-2K was 6.9 (IQR: 7.0–16.5). The indications for RTX were active lupus nephritis (65.8%), CNS involvement (10.5%), arthritis (13.2%), haematological manifestations (7.9%), or mucocutaneous involvement (2.6%). Twenty-six patients (68.4%) received RTX according to the lymphoma regimen (375 mg/m² at day 1, 7, 14, 28) while 12 (31.6%) according to the arthritis regimen (2 infusions at a dose of 1 g, 14 days apart). Intrahepatic corticosteroids and cyclophosphamide were given in 65.8% and 63.2% of the patients, respectively.

ADA were detected in 18 patient samples (47.4%) at follow-up and stratified into reactive samples (confirmed positive but with a titer <2 AU/mL; n=3), low positive (2–10 AU/mL; n=6), medium positive (11–50 AU/mL; n=4), and high positive (>51 AU/mL; n=5).

We found no association between the occurrence of ADA and either SRI response (p=0.26, Fisher exact test) nor the concomitant use of high dose IV 6-methylprednisolone (p=0.56, c² test) or IV cyclophosphamide (p=0.11, c² test). At follow-up, patients positive for ADA had higher levels of CD19+B cells (median: 0.03 ×10⁹ cells/L; IQR: 0.01–0.13) compared to negative patients (median: 0.01 ×10⁹ cells/L; IQR: 0.005–0.01; p=0.007, Mann-Whitney test). Conclusions: ADA to RTX in SLE are more frequent than in RA and MS and occur irrespective of treatment response and cotreatments, but are associated with higher counts of CD19+B cells at follow-up. Such finding could reflect either incomplete B-cell depletion in ADA positive patients, or earlier repopulation. Further studies should address the relation between ADA titers and clinical outcomes as well as immunological consequences.

REFERENCES:

Disclosure of Interest: None declared


AB0514

THE EFFECT OF HYDROXYCHLOROQUINE ON REDUCING PROTEINURIA IN STABLE SLE PATIENTS

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Background: It is thought that hydroxychloroquine (HCQ) reduces proteinuria by preventing endothelial dysfunction in mouse models, but the effects in systemic lupus erythematosus (SLE) patients are not known.

OBJECTIVES: To investigate the effects of HCQ on proteinuria in stable SLE patients.

Methods: This was a single-centre, retrospective cohort study. We included stable SLE patients who met the updated or revised American College of Rheumatology 1997 criteria for SLE or the 2012 Systemic Lupus International Collaborating Clinics criteria, and had no active organ dysfunction that needed an increase in immunosuppressive therapy. The subjects (HCQ group) were SLE patients with proteinuria >0.2 g/gCr who started HCQ between 11/1/2015 and 8/1/2017. The controls (non-HCQ group) were SLE patients with proteinuria >0.2 g/gCr seen between January 2016 and October 2016 (non-HCQ group). The reduction in proteinuria over 6 months in the HOC and non-HCQ groups was compared. The following patients were excluded from the analysis: those who had proteinuria of other aetiologies (diabetic nephropathy, etc.), those who increased the prednisolone (PSL) dose or started immunosuppressive agents, angiotensin converting enzyme inhibitors, or betablockers, etc.), those who increased the prednisolone (PSL) dose or started immunosuppressive agents, angiotensin converting enzyme inhibitors, or angiotensin II receptor blockers beginning 1 month before the observation period started until its end. Improvement was defined as a reduction in proteinuria >0.1 g/gCr. The statistical analysis was performed with the Next and chi-square test.

RESULTS: There were no significant differences in disease duration, sex, Systemic Lupus Erythematosus Disease Activity Index, or the use of immunosuppressants at baseline between the HOC group (n=16) and non-HCQ group (n=14) groups. The respective mean PSL dose at baseline was 5.9±2.8 and 3.3±3.8 mg in the HOC and non-HCQ groups (p=0.042). Patients in the HOC group were younger (mean 47±12 vs. 63±14 years, p<0.005). The kidney pathology of the HOC group was 6.25, 6.25, 6.25, 6.25% class I to V, respectively, compared with 7.14% class IV, 14.2% class V, and 14.2% class IV-V in the non-HCQ group. The other patients were diagnosed with lupus nephritis clinically.

Proteinuria was significantly lower in the HOC group than in the non-HCQ group (p<0.001, Mann-Whitney test). The mean proteinuria at baseline and 6 months was 0.501±0.276 and 0.331±0.274 g/gCr, respectively, in the HOC group, and 0.587±0.409 and 0.717±0.720 g/gCr in the non-HCQ group. The proportion of patients who improved in the HOC and non-HCQ groups was 68.7% (11/16) and 28.5% (4/14), respectively (p=0.028).

Conclusions: HCQ may reduce proteinuria in SLE patients. This suggests that HCQ administration protects the kidneys of SLE patients.

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