Methods: A temporary recommendation for use for infliximab in refractory TA was approved by the French National Drug Authorities (April 2014). Infliximab was administered to patients in case of disease activity with a NIH score ≥ 2 despite conventional therapy. Data regarding patient's clinical, laboratory, imaging and treatments were obtained at baseline, and at each following visit until last visit (October 2017). TA activity was evaluated according to NIH criteria and GC requirement throughout the study.

Results: Twenty-three patients were enrolled, including 19 female. The median age at inclusion was 33 years (Interquartile range, IQR: 23-44 years). At baseline, 17 (74%) patients were treated with GCs, at a median dose of 10 mg/day (IQR: 0-21) of prednisone-equivalent. After a median follow-up of 36.9 months (IQR: 10-58.7), improvement of ≥ 1 NIH criterion of TA activity was achieved for 14/22 (64%) patients. The median GC dose was 8 mg/day (IQR: 7-10) at 6 months; 5 mg/day (IQR: 0-8) at 12 months and 0 mg/day (IQR: 0-5) at 36 months of follow-up. Overall, infliximab originator had a significant GC-sparing effect between baseline and last follow-up ($p=0.009$).

Conclusion: This multicenter open-label cohort study suggests that infliximab originator is an effective GC-sparing treatment for TA refractory to conventional therapy.

Disclosure of Interests: None declared

DOI: 10.1136/annrheumdis-2020-eular.1241

AB0511

INTERNATIONAL CONSENSUS ON ANCA TESTING AND INTERPRETATION BEYOND SYSTEMIC VASCULITIS

S. Moiseev¹, J. W. Cohen Tervaert^{2,3}, Y. Arimura⁴, D. Bogdanos⁵, C. Elena⁶, J. Damoiseaux³, M. Ferrante⁷, L. F. Flores-Suárez⁸, M. Fritzler⁹, P. Invernizzi¹⁰, D. Jayne¹¹, J. C. Jennette¹², M. Little¹³, S. P. McAdoo¹⁴, P. Novikov¹, C. D. Pusey¹⁴, A. Radice¹⁵, A. D. Salama¹⁶, J. Savige¹⁷, M. Segelmark^{18,19}, Y. Shoenfeld^{1,20,21}, R. A. Sinico¹⁰, M. J. R. De Sousa²², U. Specks²³, B. Terrier²⁴, A. Tzioufas²⁵, S. Vermeire⁷, M. H. Zhao²⁶, X. Bossuyt⁷. ¹Sechenov First Moscow State Medical University, Moscow, Russian Federation; ²University of Alberta, Edmonton, Canada; ³Maastricht University Medical Center, Maastricht, Netherlands; ⁴Kyorin University School of Medicine, Tokyo, Japan; ⁵University of Thessaly, Larissa, Greece; ⁶University of Tübingen, Kirchheim-Teck, Germany; ⁷University Hospitals Leuven, Leuven, Belgium; ⁸Instituto Nacional de Enfermedades Respiratorias, Mexico City, Mexico; ⁹University of Calgary, Calgary, Canada; ¹⁰Università degli Studi di Milano-Bicocca, Monza, Italy; ¹¹University of Cambridge, Cambridge, United Kingdom; ¹²University of North Carolina, Chapel Hill, United States of America; ¹³Trinity Health Kidney Centre, Dublin, Ireland; ¹⁴Imperial College London, London, United Kingdom; ¹⁵San Carlo Borromeo Hospital, Milan, Italy; ¹⁶Royal Free Hospital, London, United Kingdom; ¹⁷University of Melbourne, Melbourne, Australia; ¹⁸Lund University, Lund, Sweden; ¹⁹Skane University Hospital, Lund, Sweden; ²⁰Sheba Medical Center, Tel Hashomer, Israel; ²¹Tel Aviv University, Tel Aviv, Israel; ²²Centro de Medicina Laboratorial Germano de Sousa, Lisbon, Portugal; ²³Mayo Clinic, Rochester, United States of America; ²⁴Hôpital Cochin, Paris, France; ²⁵National and Kapodistrian University of Athens, Athens, Greece; ²⁶Peking University First Hospital, Beijing, China

Background: ANCA can be detected in sera from patients with autoimmune, inflammatory, infectious or neoplastic diseases.

Objectives: To issue a Consensus Statement on ANCA testing and interpretation beyond systemic vasculitis.

Methods: This Statement was prepared by a group of experts, based on the results of a comprehensive search in PubMed.

Results: In certain settings beyond systemic vasculitis, ANCA may have diagnostic, clinical, and/or prognostic relevance. Testing for PR3- and MPO-ANCA by specific immunoassays should be performed in any patient with clinical features suggesting ANCA-associated vasculitis and in patients with anti-GBM disease and idiopathic interstitial pneumonia. Routine ANCA testing is not recommended in patients with connective tissue diseases (CTD), autoimmune liver diseases, inflammatory bowel diseases, infections, and/or malignancy unless there is evidence for small vessel vasculitis. ANCA testing by specific immunoassays may be useful in patients with rheumatoid arthritis, systemic sclerosis or primary Sjögren's syndrome who have kidney disease with a nephritic sediment or in patients with systemic lupus erythematosus if a kidney biopsy shows prominent necrotizing and crescentic lesions or proliferative lupus nephritis. ANCA testing may be justified in patients with suspected autoimmune hepatitis type 1, who do not have conventional disease-related autoantibodies, or in patients with inflammatory bowel diseases in case of diagnostic uncertainty to discriminate ulcerative

colitis from Crohn's disease. In these cases, ANCA should be tested by indirect immunofluorescence since target antigens are not well characterized. ANCA against bactericidal/permeability-increasing protein may be a biomarker for deteriorating lung function and a poor prognosis in patients with cystic fibrosis.

Conclusion: ANCA testing is clinically relevant not only in patients with manifestations suggesting systemic vasculitis, but also in patients with certain other disorders, particularly in patients with anti-GBM disease or idiopathic interstitial pneumonia.

Disclosure of Interests: Sergey Moiseev Grant/research support from: This work was supported by the 5-100 Project, Sechenov University, Moscow, Jan Willem Cohen Tervaert: None declared, Yoshihiro Arimura: None declared, Dimitrios Bogdanos: None declared, Cserek Elena: None declared, Jan Damoiseaux: None declared, Marc Ferrante: None declared, Luis Felipe Flores-Suárez: None declared, Marvin Fritzler: None declared, Pietro Invernizzi: None declared, David Jayne Grant/research support from: ChemoCentryx, GSK, Roche/Genentech, Sanofi-Genzyme, Consultant of: Astra-Zeneca, ChemoCentryx, GSK, InflaRx, Takeda, Inmed, Chugai, Boehringer-Ingelheim, J. Charles Jennette: None declared, Mark Little: None declared, Stephen P. McAdoo: None declared, Pavel Novikov Grant/research support from: This work was supported by the 5-100 Project, Sechenov University, Moscow, Charles D. Pusey: None declared, Antonella Radice: None declared, Alan D. Salama: None declared, Judith Savige: None declared, Mårten Segelmark: None declared, Yehuda Shoenfeld: None declared, Renato Alberto Sinico: None declared, Maria Jose Rego de Sousa: None declared, Ulrich Specks: None declared, Benjamin Terrier: None declared, Athanasios Tzioufas: None declared, Severine Vermeire: None declared, Ming-hui Zhao: None declared, Xavier Bossuyt: None declared

DOI: 10.1136/annrheumdis-2020-eular.219

AB0512

ALLERGIC PROFILE AND ALLERGEN-SPECIFIC IMMUNOTHERAPY IN EOSINOPHILIC GRANULOMATOSIS WITH POLYANGIITIS (EGPA): A SINGLE CENTER OBSERVATIONAL STUDY

L. Moroni^{1,2}, A. Cariddi¹, S. Sartorelli¹, E. Della Torre^{1,2}, T. Germano², G. A. Ramirez^{1,2}, E. Bozzolo¹, M. R. Yacoub¹, L. Dagna^{1,2}. ¹IRCCS San Raffaele Hospital, Unit of Immunology, Rheumatology, Allergy and Rare Diseases, Milano, Italy; ²Vita-Salute San Raffaele University, Milano, Italy

Background: Eosinophilic granulomatosis with polyangiitis (EGPA), previously known as Churg-Strauss syndrome, is a systemic disease characterized by late onset asthma associated with small- and/or medium-size vessel vasculitis, besides eosinophil-mediated cytotoxic organ damage. About 20-30% of patients with EGPA displays allergic manifestations related with inhalant sensitization, while prevalence of food and drug allergy is unknown in this context. Moreover, some authors in the past hypothesized in favor of a possible role of allergen-specific immunotherapy (ASIT) as a trigger of disease.

Objectives: Aim of the present study is to establish the prevalence of each category allergen sensitization and to determine whether atopy or specific immunotherapy could influence clinical expression of the disease.

Methods: Our study consisted in a retrospective demographic and clinical data collection regarding EGPA history (including age at diagnosis, organ and tissue involvement, autoantibody profile) and the presence of allergic comorbidities or previous drug hypersensitivity reactions. Patients without either proven allergic reactions or positive tests have been excluded.

Results: Fifty-three (53) patients with definitive diagnosis of EGPA have been included in the analysis among which 25 (47.2%) with chronic respiratory allergy or previous acute allergic reaction. Among allergic patients 15 (60%) resulted sensitized towards inhalants and among them 13 (86.7%) displayed multiple sensitization. Drug allergy affected 13 patients (52%), food 4 (16%). Among 15 patients with respiratory allergy, 13 were eligible to allergen-specific immunotherapy (ASIT). Seven (7) subjects underwent ASIT prior EGPA diagnosis with an average time-to-EGPA of 16.2 years. No statistically significant difference was found in terms of sex, age at diagnosis, positivity for or specificity of anti-neutrophil cytoplasm antibodies (ANCA), eosinophil count at onset, pattern of clinical manifestations comparing allergic vs. non-allergic, ASIT vs. non-ASIT, ASIT vs. allergic, ASIT vs. eligible.

Conclusion: Among patients with EGPA allergies are highly prevalent, particularly towards inhalants and drugs. In the great majority of patients multiple sensitization profile is found. Atopy doesn't seem to be associated with specific patterns of disease presentation. The absence of correlation between inhalant ASIT exposure and variation in mode and time of EGPA onset doesn't support the hypothesis of a its potential role in triggering the disease.