Travel Report on the 18th International Vasculitis and ANCA Workshop; Tokyo, Japan, March 25-28

Receiving the travel grant of the NVVI helped me to attend the biannual Vasculitis and ANCA Workshop in March 2017 in Tokyo. During this congress I was given the opportunity to give an oral presentation on my work about plasmablast frequencies as a potential new biomarker of disease flares in granulomatosis with polyangiitis. This enabled me to come into contact with fellow researchers whom gave me valuable input for my ongoing research.

The congress was opened on March 25th with a very interesting keynote lecture by prof. Loïc Guillevin from Cochin Hospital, University of Paris Descartes (France) on future therapeutic strategies for ANCA-associated vasculitides. He emphasized that treatment should not only induce remission but also be able to prevent relapses since it greatly contributes to morbidity of patients. Another important goal should be to decrease treatment-related side-effects of drugs.

The congress consisted of plenary and workshop sessions. Topics of the plenary sessions gave us updates on disease classification, case discussions, treatment outcomes and updates on different vasculitic diseases. The workshop sessions were divided into several different subjects and PhD students and postdocs gave interesting presentations about their research in these sessions. In the workshop entitled "ANCA and other biomarkers" several interesting presentations were given. Divi Cornec from Mayo Clinic (USA) presented his work on detection of PR3-specific B cells in patients with AAV in the circulation using recombinant PR3 conjugated to FITC. Interestingly, circulating PR3-specific B cells were associated with disease activity and therefore might be a new biomarker to predict relapse risk in individual patients. Benjamin Wilde from the University Hospital Essen (Germany) showed his work on granzyme B producing B cells in AAV. He showed that granzyme B+ B cells could be induced *in vitro* and that these cells were able to suppress T cell proliferation. Moreover, in patients with AAV this granzyme B+ B cells were diminished suggesting that these cells might contribute to disease pathogenesis.

I thank the NVVI for supporting me to attend this congress, where I was updated on research in the field of ANCA-associated vasculitis, was able to present my own research and met fellow immunologists.

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