All pathology in NMO is AQP4-IgG and complement dependent

F. Paul
NeuroCure Clinical Research Center, Charité Universitätsmedizin Berlin, Berlin, Berlin, Germany, Charité Universitätsmedizin Berlin, Germany

Attacks in NMO are believed to result from binding of AQP4 to its target antigen, the astrocyte water channel AQP4, with subsequent complement activation and other mechanisms leading to tissue damage and macroscopically visible NMO lesions. Drugs targeting complement activity have shown to significantly reduce relapse activity in NMO, further underscoring the role of complement in disease pathogenesis and lesion formation. However, recently other players of the immune system such as neutrophils or T cells have been identified as contributing to lesion evolution, and not all lesions seem to be characterized by complement activation and tissue necrosis. This presentation will provide arguments against a standalone role of complement and will shed light on additional disease mechanisms we are now beginning to understand.