Abstracts

Results She showed complete recovery after rituximab.

Conclusions Rituximab can be considered as an effective treatment modality in APLA related chorea where conventional measures have failed.

64 DYSBIOSIS AND GUT BARRIER DYSFUNCTION IN ANTIPHOSPHOLIPID SYNDROME AS REVEALED BY IGA-SEQ PROFILING

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Background and aims The antiphospholipid syndrome (APS) is an autoimmune thrombophilic non-gut disorder with high mortality. Various pathogens have been associated with transient antiphospholipid antibody production. We hypothesised that members of the gut microbiota in APS patients could represent a chronic trigger and exhibit heightened adaptive immune responses to the microbiota. The purpose of this study was to explore gut barrier function and faecal IgA-coated microbial composition in APS patients.

Methods Stool from 15 APS patients, 5 non-autoimmune thrombotic states, and 12 normal donors (total of 17 controls) was collected. Faecal homogenates were analysed for the gut permeability marker calprotectin and, in parallel, stained stained with PE-conjugated anti-human IgA prior to cell sorting. Faecal DNA was isolated and PCR-amplified targeting the V4 region of the 16S rRNA gene. Samples were sequenced on the Illumina MiSeq platform.

Results Faecal calprotectin and IgA-coated faecal bacterial levels were significantly higher in APS patients compared to controls (p<0.003; p<0.05). LEfSe analysis of IgA+ fractions showed that the strongest IgA-coated genus is Blautia in APS.

Conclusions These data suggest gut barrier dysfunction and aberrant IgA coating of commensals in APS. Markedly enhanced bacterial IgA coating in several APS patients supported a stronger adaptive immune response to the microbiota. Increased IgA coating of Blautia might reflect altered gut homeostasis as a Blautia species was shown to be part of proinflammatory IgA+ consortium in a recent study in IBD. To our knowledge, this study represents the first 16S rRNA profiling of IgA-coated gut commensals in patients with non-gut autoimmunity.

66 ANTIPHOSPHOLIPID SYNDROME – CLINICAL AND IMMUNOLOGICAL CORRELATIONS AND ATHEROSCLEROSIS

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Background and aims In recent years, it is found that role in atherogenesis plays inflammatory component of immune response. In recent years found unarguably data for accelerated development of atherosclerotic plaques in patients with APS.

Our aim is to investigate the frequency of cardiovascular events and atherosclerosis in patients with primary and secondary antiphospholipid syndrome compared to healthy individuals and patients with systemic lupus erythematosus without antiphospholipid antibodies.

Methods We studied 99 patients with APS, 13 SLE and 32 healthy controls. They were tested for aPL antibodies (aCL, anti-h2gp1, anti-prothrombin), ANA-screen, ANA - profile and standart laboratory.

We examine Intima-Media thickness of carotit arteries and Calcium score of: a. coronaria sinister, a. anterior descendens, a, circumflexa sinister, a. coronaria dexter, Aorta, Vagina to validate the atherosclerosis.

Results We found strong, statistically significant correlation between aCL antibodies and the presence of plaques in the left common carotid artery (p=0.041) and absent a correlation between aPL titers and presence of carotid plaques. In the group with APS, 33,3% (14) establishes a positive calcium score of coronary arteries, 11.9% (5) positive for aorta, Aortic valve Absent deposits, In the control group positive calcium score is one person (5.88%).