PLASMABLAST PROLIFERATION IS ASSOCIATED WITH WHY DO NOT ALL CHRONIC KIDNEY DISEASE HYPERECHOIC DEPOSITS IN THE RENAL MEDULLA

Abstract OP0355 – Figure 1 Kaplan-Meier survival curves of APS patients associated CTEPH after treatment (anticoagulation and/or PTE).

Conclusions: After a full specialised and multidisciplinary risk-benefit evaluation to limit the risk of thrombosis or bleeding and to manage possible thrombocytopenia, for those CTEPH developed in APS patients, PTE is a curative resolution.

Disclosure of Interest: None declared.


OP0356
PLASMA Blast PROLIFERATION IS ASSOCIATED WITH TOLL LIKE RECEPTOR 7 POLYMORPHISMS AND SUPPRESSION OF TYPE 1 INTERFERON, CONTRIBUTING TO THE ANTIBODY PRODUCTION IN ANTIPHOSPHOLIPID SYNDROME

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Background: Antiphospholipid antibodies (aPL) as pathogenic autoantibodies in systemic lupus erythematosus (SLE) and antiphospholipid syndrome (APS) are supported by a number of clinical, ex vivo and animal studies. Nevertheless, aPL are not eliminated by corticosteroid administration or immunosuppression. Novel therapy targeting aPL production is currently unmet needs, in contrast, little is known on its pathological mechanism.

Objectives: This study aimed to clarify the mechanism of aPL production by lymphocyte subset analysis, genomic analysis and ex vivo experiments.

Methods: B cell and T cell subsets, a total of 21 subsets, were evaluated in peripheral blood mononuclear cells (PBMC) of 26 primary APS (PAPS), 18 SLE-APS patients and 10 healthy controls by flow cytometry. Twenty-one single nucleotide polymorphisms (SNP), which were shown to be associated APS (SLE/APS) patients and 10 healthy controls by flow cytometry. Twenty-one single nucleotide polymorphisms (SNP), which were shown to be associated with autoimmune or thrombotic diseases, were analysed in genomic experiments.

Results: aPL producing capability of plasmablasts, PBMC from APS patients, but not with features of uric acid lithiasis.

Conclusions: Our in gout population, HERM was observed in 36% of patients, correlated with decreased renal function, and clearly associated with severe gout, but not with features of uric acid lithiasis.

Disclosure of Interest: None declared.


SATURDAY, 16 JUNE 2018

The links between gout and kidney function

OP0357
HYPERECHOIC DEPOSITS IN THE RENAL MEDULLA ARE ASSOCIATED WITH SEVERE GOUT AND DECREASED EGF: A TRANSVERSAL STUDY IN 503 VIETNAMESE PATIENTS

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Background: Renal medulla crystal deposits have been demonstrated by pathology in severe gout but little studied by ultrasound (US) scan.

Objectives: To assess the frequency of hyperechoic renal medulla (HERM) in gouty patients and factors associated with their development.

Methods: Renal US scan using a Ecube 9 echograph (Alpinion S. Korea) was performed in gout patients (ACR/EULAR criteria) consecutively seen at the Vien Gut general clinic, Ho Chi Minh City, Vietnam, and receiving no ULT at presentation. Age and sex of patients, gout features, associated diseases, serum (S) uric acid (UA), eGFR (MDRD), urinary lab stick parameters, uric UA/creatinine ratio, and fractional clearance of urate (FCU) were recorded. Patients with HERM were counted and compared with those who had no medullary deposits by the Wilcoxon rank sum test for continuous variables and the Fischer exact test for categorical variables.

Results: 503 consecutive patients (500 males) were included. They had a median age of 46 years, median BMI of 25 kg/m2, median gout duration of 4 years. 280 (56%) had clinical tophi, 154 (31%) urate arthropathy, 28;615) urolithiasis, 112 (22%) hypertension, 58 (11.5%) type 2 diabetes, 5 (1%) coronary heart disease. Their median eGFR was 78 ml/min, SUA 423 micromol/L, FCU 0.063, uric UA/creatinine ratio 0.253, urinary pH 6. Diffuse and bilateral HERM on the B mode with frequent twinkling artefacts on the Doppler mode was identified in 181 (36%) of the 503 patients. Univariate analysis showed that HERM associated with higher age, longer duration of gout, clinical tophi, urate arthropathy (p<0.0001 for each of the variables), higher uricemia (p<0.001), hypertension (p<0.0008), CHD (p<0.0006), lower eGFR (p<0.0001), leucocyturia (p<0.02), proteinuria (p<0.02). No association with US-diagnosed urolithiasis, hematuria, uric UA/creatinine ratio, FCU and urinary pH was found.

Multivariate analysis, log of the duration of gout (OR: 2.22 (CI: 1.63–3.08), p<0.001), clinical tophi (OR: 8.21 (4.23–16.91) p<0.001), urate arthropathy (OR: 3.74 (2.18–6.52, p<0.001), and lower eGFR (OR: 0.86 (0.75–0.99) for each 10 ml/min decrease, p=0.04) were significantly associated with HERM.

Conclusions: In our gout population, HERM was observed in 36% of patients, correlated with decreased renal function, and clearly associated with severe gout, but not with features of uric acid lithiasis.


WHY DO NOT ALL CHRONIC KIDNEY DISEASE PATIENTS GET GOUT? IMPAIRED NEUTROPHIL CHEMOTAXIS IN HYPERURICEMIA-RELATED CKD

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Background: One characteristic feature of acute gout is the infiltration of neutrophils into the inflamed joints, where they recognise monosodium urate (MSU) crystals leading to an acute inflammatory response. The development of chronic kidney disease (CKD) is associated with increased serum uric acid (UA) levels also known as hyperuricemia, a major risk factor for gout. Despite hyperuricemia, acute gout is less frequent in CKD patients. However, the effects of hyperuricemia on leukocyte chemotaxis in CKD are not fully understood.

Disclosure of Interest: None declared.


References: