Parallel Session 10: APS

ANTIPHOSPHOLIPID SCORE IS A NOVEL RISK FACTOR FOR IDIOPATHIC OSTEONECROSIS

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Background and Aims Our group introduced a quantitative marker of antiphospholipid antibodies (aPL) “antiphospholipid score (aPL-S)”, which well reflected the risk of developing thrombosis (Otomo K, et al. Arthritis Rheum 2012). Idiopathic osteonecrosis (ION) has been shown to occur as a result of ischemia, however, the involvement of aPL in its pathophysiology remains to be elucidated. In this study, we aimed to identify the impact of aPL on the development of ION using aPL-S.

Methods A single centre retrospective study comprising 75 consecutive patients with systemic lupus erythematosus who underwent magnetic resonance imaging of hip joints from January 2000 to March 2016. aPL-S, as well as classical risk factors for ION, were evaluated in all the enrolled patients.

Results ION of the femoral head was observed in 33 out of 75 patients (44%). High aPL-S (p=0.009), aPL positivity (p=0.009), male (p=0.007), malar rash (p=0.010) and high dose (>0.8 mg/kg/day) glucocorticoids (p<0.001) were identified as risk factors for ION in univariate analysis. Multivariate analysis confirmed high aPL-S, male, malar rash and high dose glucocorticoids as independent variables. Six out of 8 patients (75%) with very high aPL-S (>30), developed ION. Conversely, systemic lupus erythematosus disease activity index and pulse methylprednisolone therapy were not identified as risk factors for ION.

Conclusions We newly identified aPL-S as a risk factor for ION. ION should be considered as one of the antiphospholipid antibody-associated-disease.

Parallel Session 11: Effector T cells in SLE

METABOLIC REPROGRAMMING IN CD4+CD28- CXCR3INT-T-BETHI CELLS AND ITS RELEVANCE TO PATHOGENESIS IN PATIENTS WITH SLE

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Background and Aims CD4+ T cells play a crucial role in pathological process of Systemic Lupus Erythematosus (SLE). Recently, we found that T-bet is an important factor for shift to glycolysis in activated CD4+ T cells in vitro. In this study, we examined the mechanism by which T-bet in CD4+ T cells involved in pathogenesis of SLE.

Methods Peripheral blood mononuclear cells were obtained from 19 healthy controls (HCs), 30 patients with bio-naive active RA and 60 patients with SLE. The expression of CXCR3, T-bet, mTORC1 phosphorylation and IFN-γ production in CD4+ T cells were measured by flow cytometry, and assessed the association with clinical characteristics.

Results We found that the ratio of CD28+CXCR3intT-bethi cells in patients with SLE was significantly higher compared to HCs. CD4+CD28+CXCR3intT-bethi cells mainly consisted of CD45RACCR7 effector memory cells and were significantly activated with pronounced IFN-γ production. Interestingly, the ratio of CD4+CD28+CXCR3intT-bethi cells was significantly correlated with the number of immunosuppressants previously used for the SLE patients, that is treatment-resistant. Phosphorylation of mTORC1, which is important for shift to glycolysis, in CD4+ T cells from patients with SLE was significantly increased compared to HCs. T-bet expression was significantly correlated with mTORC1 phosphorylation and IFN-γ production in CD4+ T cells from patients with SLE.

Conclusions These results indicated that CD4+CD28+CXCR3intT-bethi cells might be related to refractory to established therapies in patients with SLE. In addition, these cells are constitutively activated accompanied with shift to glycolysis through IFN-γ-mTORC1-T-bet pathway, which is a potential target for patients with SLE.
Parallel Session 16: Quality of care and patient reported outcomes

DISEASE OUTCOMES AND CARE FRAGMENTATION IN SYSTEMIC LUPUS ERYTHEMATOSUS (SLE)

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Background and Aims To examine the impact of care fragmentation across multiple healthcare institutions on disease outcomes in patients with systemic lupus erythematosus (SLE).

Methods Methods: Using the Chicago HealthLNK Data Repository (HDR), an assembly of electronic health records from six institutions, we identified patients with SLE, using ICD-9 codes, whose care was delivered at more than one organisation. We examined whether patients had severe infections or comorbidities (ICD-9 code defined) that indicate SLE-induced damage. T-tests and chi-squared tests were used to examine differences between fragmentation groups. Logistic regression was used to assess factors contributing to the occurrence of disease outcomes.

Results We identified 4276 patients with SLE. 856 (20%) received care from more than one healthcare institution. African American patients and patients with public insurance were more likely to experience care fragmentation compared to white and private insurance patients (OR 1.66; 95% CI 1.44, 1.97 and OR 1.63; 95% CI 1.42, 1.95). We identified increased risk of infections (OR 1.57; 95% CI 1.30, 1.88), cardiovascular disease (OR 1.51; 95% CI 1.23, 1.86), end stage renal disease (OR 1.34; 95% CI 1.05, 1.70), nephritis (OR 1.28; 95% CI 1.07, 1.54) and stroke (OR 1.28; 95% CI 1.01, 1.62) among patients with fragmented care, adjusted for age, sex, race, insurance status, length of follow-up time, and total visit count.

Conclusion In this cross-site cohort of SLE patients, care fragmentation is associated with increased risk of severe infection and comorbidities. These results suggest that improved health information exchange could positively impact outcomes for SLE patients.