— F-335 —

The significance of serum total cholesterol in systemic lupus erythematosus associated protein-losing enteropathy

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The characteristics of protein-losing enteropathy (PLE) were evaluated in patients with systemic lupus erythematosus (SLE). Among the SLE patients (n=380) in a tertiary hospital, we reviewed the records of 7 patients who had generalized edema, hypoalbuminemia without proteinuria and positive results of 99mTc-labeled human serum albumin scintigram. The characteristics and laboratory findings were compared between the 7 patients and patients with lupus enteritis (n=15) or idiopathic PLE (n=11). Compared to lupus enteritis, the level of ESR and serum total cholesterol was elevated significantly in SLE- related PLE. Compared to idiopathic PLE, the level of serum total cholesterol was still higher, but the level of serum albumin decreased significantly in SLE- related PLE. Among patients with SLE-related PLE, 4 patients had high serum total cholesterol ($\geq$ 248 mg/dl) and achieved complete remission after receiving high-doses of steroid treatment alone. However, 3 patients who had lower serum total cholesterol level ($\leq$ 219 mg/dl) responded poorly to the steroid-only treatment. They could achieve complete remission only after 3 months of cyclophosphamide pulse with concurrent corticosteroid therapy. In the patients with SLE-related PLE, the elevation of serum total cholesterol level is a distinctive characteristic, which might serve as a marker for favorable steroid response.

— F-336 —

Association of programmed cell death 1 (PDCD1) polymorphisms and systemic lupus erythematosus: a meta-analysis

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Objective : Programmed cell death 1 (PDCD1 or PD1) polymorphisms have been inconsistently reported to be associated with systemic lupus erythematosus (SLE). The aim of this study was to explore whether the PDCD1 polymorphisms confer a susceptibility to SLE and lupus nephritis (LN). **Methods :** We conducted a random effect meta-analysis on the association of PDCD1 polymorphisms with SLE in overall and specific ethnic populations. Subgroup analysis was performed based on renal involvement and sex. When publication bias was suggested, we adjusted the bias using the trim and fill method. **Results :** A total of 15 separate comparisons were included in this meta-analysis consisting of nine European, two Latin American, two African, one Asian, and one unknown participant. An association between SLE and the PD1.3A allele was found in the overall population, but the OR after publication bias adjustment did not show a significant association with SLE (OR = 1.226, 95% CI = 0.911-1.651). In subgroup analysis, the PD1.3A allele was significantly associated with SLE in Latin Americans (OR = 3.073, 95% CI = 1.416-6.461, p = 0.003), but not in patients of European and African decent. There was no association between the PD1.3A allele and female SLE patients, but the PD1.3A allele was a risk factor for LN in European descendants (OR = 2.207, 95% CI = 1.488-3.467, p < 0.001). The PD1.5C allele was a risk factor for SLE in Europeans (OR = 1.297, 95% CI = 1.024-1.643, p = 0.031), but not in Latin Americans and Africans. The ORs for the PD1.6A allele overall and for each ethnic group showed no association with SLE. **Conclusion :** Although a significant association of the PD1.3A allele and SLE in Europeans was not found, this meta-analysis demonstrated an association of the PD1.3A allele with LN in European and SLE in Latin American populations. Furthermore, the PD1.5C allele was associated with SLE susceptibility in Europeans. The meta-analysis supports a role of PDCD1 in the pathogenesis of SLE and LN. More studies are needed to clarify the role of PDCD1 in SLE and LN within various ethnic groups.