Serum RIP1 level as a severity-predictive marker for Stevens-Johnson syndrome and toxic epidermal necrolysis
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Stevens-Johnson syndrome (SJS) or toxic epidermal necrolysis (TEN) are life-threatening diseases. It is often difficult to distinguish SJS/TEN and other types of generalized skin eruptions, such as maculopapular exanthema (ME) and erythema multiforme (EM) at the early stage. Although keratinocytes have been suggested to die as apoptosis in SJS/TEN, our recent study revealed that necroptosis, programmed necrosis, also contribute to keratinocyte death in SJS/TEN. Necroptosis is mediated by receptor-interacting kinase-3 (RIP3) and mixed lineage kinase domain-like pseudokinase phosphorylation. We aim to investigate whether serum RIP1 levels serve as a predictive biomarker for SJS/TEN severity. The serum samples were obtained from the patients with SJS/TEN (n=18), EM major (n=19), EM minor (n=5) and ME (n=6) in the acute phase. SJS/TEN group have significantly higher serum RIP1 levels than the other groups (EM major: P=0.002, EM minor: P<0.003, ME: P<0.003, healthy controls: P=0.0022). Also in the SJS/TEN major group, serum RIP1 levels are significantly higher than in the healthy controls (P<0.006). In addition, positive correlations were found between serum RIP1 levels and the frequency of keratinocyte death in histopathological examination, body temperature, mucosal involvement, and organ dysfunction. We also analyzed changes in serum RIP1 levels after initiation of treatment (SJS: n=4, EM: n=4) and after clinical recovery (SJS: n=1, EM: n=3). In all these samples, serum RIP1 levels decreased after treatment. We showed the clinical usefulness of serum RIP1 levels as a differential or prognostic marker for ME, EM or SJS/TEN. By predicting of the severity at the early stage, we can start appropriate treatment earlier.

Cervical Vaccine Based on E7-HSP110 or E7-HSP110-EGFP Promotes Exogenous CD8+ T Cells via RGD-Targeted Nanoparticles
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Background: Bullous pemphigoid (BP) is an autoimmune blistering disease characterized by autoantibodies against BP180 and BP230. There is emerging evidence that IgE autoantibodies play an important role in the pathogenesis of BP. Objective: To determine the rate of anti-BP180 and anti-BP230 IgE in BP, and to evaluate their diagnostic relevance in BP sera.
Methods: We collected serum samples from 156 patients who met the clinical and immunologic, and histologic features consistent with BP. In addition, 10 control individuals were included for comparison. We used commercially available IgE enzyme-linked immunosorbent assay (ELISA) to detect IgG and IgE ELISAs for both BP180 and BP230. A receiver operating characteristic (ROC) curve was used to evaluate the ability of the ELISA to detect anti-BP180 and anti-BP230 IgE. Various diagnostic parameters including test sensitivity, specificity, positive predictive value (PPV) and negative predictive value (NPV) were calculated. Results: The results of IgE ELISAs were statistically compared among various ELISAs. Results: In 156 BP patients, 96 BP patients with elevated total level of IgE (>165.3 IU/ml) and 60 BP patients with normal level of total IgE. IgG autoantibodies against BP180 and BP230 were detected in 100% (156/156) and 25.6% (39/156) patients, respectively. Anti-BP180 IgG autoantibodies, but not anti-BP230 IgE autoantibodies, correlated with total level of IgE (r=0.6914, P<0.0001). The level of BP230 IgE was significantly higher than the level of BP180 IgE (P=0.0357). Conclusion: The results of this study indicated that most BP patients exhibit elevated IgG levels in the serum. IgE autoantibodies to both BP180 and BP230 can be detected in BP sera. The results of total IgE and anti-BP180 IgE ELISAs are well correlated. In BP patients, the positivity rate of anti-BP230 IgE is higher than anti-BP180 IgE. IgE anti-BP230 autoantibodies seemed to be pathogenic in BP.

Profile of BP180 and BP230-specific IgG autoantibodies in bullous pemphigoid
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