P165

신이식후 인터루킨 17A와 17RA 유전자 다형성과 급성거부반응의 연관성

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Association between Interleukin 17A and IL17RA Gene Polymorphisms and Acute Rejection Following Kidney Transplantation

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Introduction and Aims: Kidney transplantation is the best treatment for the patients with end stage renal disease compared to other renal replacement therapies. But the development of acute rejection episodes (ARE) after kidney transplantation had a negative effect to the renal allograft and shortened the length of allograft survival. T cell immune reaction is reported to be the main cause of ARE. Th17 cells, the newly discovered T cells, may be associated with the occurrence of ARE in addition to Th1 and Th2 cells. Many studies showed that the level of IL17A had increased during ARE and IL17A mRNA and protein had detected in rejecting renal allograft. IL17A binds to IL17 receptor (composed with IL17RA and IL17RC), which induces many signal transductions and activates many inflammatory pathways. Single nucleotide polymorphisms (SNPs) of many cytokines could affect the occurrence of ARE but there is no report about the relation of the SNP of IL17A and IL17RA with ARE. Therefore this study aimed to investigate the association between SNPs of IL17A complexes and the occurrence of ARE.
Methods: We analyzed 2 SNPs of IL17A (rs3819024, rs22795913) and 4 SNPs of IL17RA (rs879759, rs979577, rs2229151, rs4819554) among 332 renal recipients, 58 of whom had developed AR. SNPs of IL17A are all regulatory SNPs (thought to be the promoter) and SNPs of IL17RA are three exonic SNPs (rs879575, rs879577, and rs2229151) and one regulatory SNP (thought to be the promoters, rs4819554).
Results: The genotyping of the 58 AR patients and the 274 patients without AR demonstrated a significant relationship between genotype frequencies and the SNPs. The occurrence of ARE was associated with rs879577 ($p=0.004$, Codominant model). Among haplotype no haplotype showed significant association with AR.
Conclusion: Our results suggest that IL17RA gene polymorphisms are associated with the development of AR.

Key Words: 다형성, 인터루킨 17A, 급성거부반응
Polymorphism, Interleukin 17A, Acute rejection