The importance of single nucleotide polymorphisms in interferon gamma receptor-1 gene in pulmonary patients infected with rapid grower mycobacterium

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ABSTRACT
Objective/Background: Interferon gamma (IFN-γ) plays a key role in protective immune response against Mycobacterial infection. IFN-γ excretes its antimycobacterial effectors mechanisms by activation of macrophages and dendritic cells via interaction with its receptor complex, that is, a ligand-binding subunit (IFN-γ receptor (IFNGR)1) and an accessory subunit (IFNGR2) on the cell surface. It has been shown that individuals with complete or partial IFNGR1 receptor deficiency are highly susceptible to infection by nontuberculous mycobacteria (NTM), Mycobacterium tuberculosis, and some Salmonella species. In the present study, we aimed to study the IFNGR1 T-56C single nucleotide polymorphism (SNP) in pulmonary patients that were infected with rapid grower mycobacterium.
Methods: Sputum specimens from suspected nontuberculosis pulmonary patients (n = 95) were digested and decontaminated using 4% NaOH method. Molecular identification of mycobacterium was then performed by hsp65 genes using polymerase chain reaction-restriction fragment length polymorphism (PCR-RFLP). Finally, the host genomic DNA from confirmed patients with rapid-grower mycobacterium (n = 20) and control subjects (n = 20) were screened for SNPs of IFNGR1 (T-56C) by PCR-RFLP.
Results: Out of 95 NTM patients, 20 (21.0%) were infected with rapid grower mycobacterium (RGM). The frequency of Mycobacterium chelonae (n = 12) was more than Mycobacterium fortuitum (n = 8), but the differences were not statistically significant. Interestingly, 18 patients (90%) had CC genotypes, whereas the remaining two had TC genotypes. The frequency of CC genotypes in the control group was <10% (p < 0.05).
Conclusion: There is a significant association between SNP of IFNGR1 at −56 and susceptibility to rapid grower infection.

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Conflicts of interest

The authors have no conflicts of interest to declare.