Type of Publication: Oral Presentation
Presented in 16th Congress of the Asian Pacific Society of Respirology. Shanghai International Convention Center, Shanghai China 03 – 06 November 2011

EFFECT OF EXON 1 CODON 52 AND 54 MANNOSE BINDING LECTIN GENE POLYMORPHISM ON THE INCIDENCE OF PULMONARY TUBERCULOSIS

K. Arrahmanda[1], Z. Ahmad[1], Sudarto[1], A. Rasyid[1] and Yuwono[2]

[1]Departement of Internal Medicine Faculty of Medicine University of Sriwijaya-Moh. Hoesin General Hospital South Sumatera Indonesia

[2]Departement of Clinical Microbiology Faculty of Medicine University of Sriwijaya-Moh. Hoesin General Hospital South Sumatera Indonesia

INTRODUCTION

Tuberculosis (TB) still world's and Indonesia problem. Approximately one third of the world population ever infected with *Mycobacterium tuberculosis* (Mtb), but only about 10% become pulmonary TB. Presumably there is influence of individual genetic factors to TB infection. Mannose binding lectin (MBL) is an acute phase protein in a non specific immune system response which will bind Mtb and activate complement system to destroy the pathogen. Polymorphisms in exon 1 codon 52 and 54 might have correlation with decreasing of MBL protein in plasma and influence to opsonin function. [1-3].

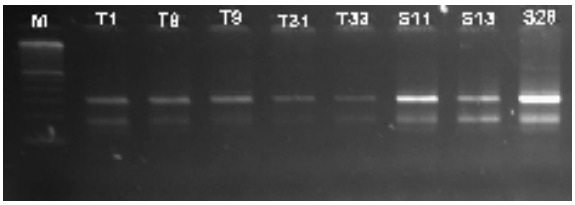
METHOD

The design of study was observational case control study. Pulmonary tuberculosis patients as case group were matched with healthy person group with tuberculin positive test as control group. The aims of the study was to determine the effect

of exon 1 MBL gene polymorphisms codon 52 and 54 on incidence of pulmonary TB in South Sumatera Indonesia. Polymorphism of codon 52 and 54 were detected by PCR-RFLP using *Ban*I enzyme and *Hha*I enzyme respectively[4].

RESULT AND DISCUSSION

We recruited 40 cases and 40 controls subject. The genotype distribution of codon 52 MBL gene were 55% allele A (wild type) and 45 allele D (mutant) in cases group, and 40% and 60% in control group respectively. Value of p 0,179, Odds Ratio 0,54 in confidence interval 95%. The genotype distribution of codon 54 MBL gene were 75% allele A (wild type) and 25 allele B (mutant) in cases group, and 65% and 35% in control group respectively. Value of p 0,329, Odds Ratio 0,61 in confidence interval 95%. These finding indicated no effect of exon 1 MBL gene polymorphisms codon 52 and 54 to incidence of tuberculosis [5].



Picture 1. PCR-RFLP result digested by *Ban*I enzyme for codon 52 exon 1 *MBL* gene. M is marker. Undigested 260 bp was homozygote wild type, digested 170 bp bp and 90 bp (not seen) was homozygote mutant and 3 band 260 bp, 170 bp, 90 bp was heterozygote.

CONCLUSION

Exon 1 MBL gene polymorphisms codon 52 and 54 have no effect on incidence of pulmonary TB in South Sumatera Indonesia

REFERENCES

1. World Health Organization. Implementing the stop TB strategy: a handbook for national tuberculosis control programmes. Geneva: WHO Press; 2008.
2. Issar Smith, Carl Nathan, and Hannah H Peavy, 2002. Progress and New Directions in Genetics of Tuberculosis An NHLBI Working Group Report. Am J Respir Crit Care Med. Vol 172. pp 1491–1496
3. Soborg C, Madson HO, Anderson AB, Lillebaek Tkok-Jensen A, Garred P. Mannose-binding lectin polymorphysms in clinical tuberculosis. *J Infect Dis* 2003;188:777-82.
4. Selvaraj P, Narayanan PR, Reetha AM. Association of functional mutant homozygotes of the mannose binding protein gene with susceptibility to pulmonary tuberculosis in India. *Tuber Lung Dis* 1999;79:221-7.
5. Selvaraj P, Jawahar MS, Rajeswari DN, Alagarasu K, Vidrayani M, et al. Role of mannose binding lectin gene variants on its protein levels and macrophage phagocytosis with live Mycobacterium tuberculosis in pulmonary tuberculosis. *FEMS Immu Med Microbiol* 2006;46(3):433-7.