Relationship of main polymorphic sites of Interleukin-13 and susceptibility to brucellosis

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Background: Brucella is an intracellular gram negative bacterium. Some previous reports have shown that gene polymorphisms of cytokines can affect on resistance or susceptibility to Brucella infection. Interleukin -13, a cytokine secreted by TH2 lymphocytes, has an important role on macrophages to induce the immune response against established infections. There are several polymorphic sites in IL-13 gene. In this study the association of three polymorphic sites of IL-13 (-1055C/T, -1512A/C and +2044G/A) and susceptibility to brucellosis was investigated.

Methods: One hundred and sixty nine patients with brucellosis and seventy one healthy controls were included in this study. DNA was extracted from the whole blood of controls and patients. All specimens were genotyped for three bi-allelic IL-13 gene polymorphisms at positions (-1055C/T, -1512A/C and +2044G/A) by polymerase chain reaction-restriction fragment length polymorphism (PCR-RFLP).

Results: None of the studied alleles and genotypes of IL-13 gene (-1055C/T, -1512A/C +2044G/A) showed significant relationship with susceptibility to brucellosis. three haplotypes of eight haplotypeTCA (P = 0.01), TCG (P = 0.002), CCA (P = 0.034) and one haplogenotype TAC/TCA (P = 0.025) were significantly higher among brucellosis patients compare to the controls.

Conclusion: In regard to no significant role of different alleles and genotypes within 3 site of interleukin-13 gene (-1055C/T, -1512A/C +2044G/A) in susceptibility to brucellosis, it seems that analysis of haplotypes and haplogenotypes of IL-13 are more logical than genotypes.

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Hospital from March to August 2011 and were followed for death or discharge from hospital stay. All variables were documented on a proforma and analyzed using SPSS version 13.0 and a P-value of <0.05 was considered statistically significant.

**Results:** The mean age of our patients was 53.7 ± 20 years. There were 53% males and 47% females. Among the co-morbidities, 81% of people with ischaemic heart disease had respiratory infection with Acinetobacter, as compared to 42% of those without ischaemic heart disease (p = 0.020). The primary diagnosis of majority of patients was sepsis (55%), followed by trauma (13%) and malignancy (10%). The most frequent source of positive culture was from Tracheal Aspirate (48%) followed by Urine culture (25%). There was a significantly higher prior admission rate in patients with respiratory Acinetobacter infection as compared to non-respiratory group (p = 0.002). The most commonly used antibiotic was Polymyxin (62%), followed by carbapenems in 18%. In 83% the Acinetobacter infection had been nosocomially acquired as opposed to 17% in whom it was community acquired. Out of 60 patients, 19 expired (32%) whereas 41 (68%) were discharged from the hospital. Although greater proportion of patients died in the respiratory group (40%) compared to non-respiratory group (23%), the difference was not statistically significant (p = 0.165). Mean length of stay was 18 days and among those who survived there was 23% readmission rate. Logistic regression did not reveal any modifier effect from age, gender, primary diagnosis, co-morbidities and use of antibiotics.

**Conclusion:** In this study, there was no significant difference in frequency of mortality between patients having respiratory versus non-respiratory acinetobacter infections.

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**Does genotypes and virulence profiles play role in classifying Staphylococcus aureus as infectious or colonizing strain?**  

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**Background:** Staphylococcus aureus is one of the well-known pathogen in human medicine. S. aureus can cause asymptomatic colonisation as well as life-threatening infection. In the present study, we address the question does the genotype and virulence factors play role in making S. aureus as a pathogen or colonizer and whether the clinical outcome of S. aureus can be predicted by genotyping and virulence profiling.

**Methods:** A total of 18 S. aureus isolates collected from bloodstream, wound and healthy nasal carriers (6 from each group) were characterised by multilocus sequence typing (MLST), accessory gene regulator (agr) typing, staphylococcal protein A (spa) typing, pulsed field gel electrophoresis (PFGE) and virulent gene profiling (sea, seb, sec, sef, seh, eta, etb, ACME, cna, fnbA, icaA, icaD, pvl, tss).

**Results:** Molecular typing revealed that majority of infectious isolates belonged to ST1 and ST239. Most of the infectious and carriage isolates shared the agr group III. Spa typing and PFGE patterns demonstrated high variance among S. aureus isolates, although with the similar clinical manifestation. On the other hand, isolates that shared similar genotype presented different clinical outcomes. Among the 18 isolates, mecA and pvl genes were only possessed by the invasive isolates, while virulence genes sea and seg enterotoxin b and g were often harbored by healthy carriage isolates. Bacteremia associated-isolates rarely harbored seh and sei when compared to wound infection and healthy carrier isolates.

**Conclusion:** Overall, the virulence genes were heterogeneously distributed among the isolates and propose an exchange of virulence genes between the S. aureus strains. Isolate which harbor most of the virulence genes (exclude pvl gene) do not mean that it can cause significant clinical manifestation in host. Further studies are needed to explain whether virulence gene expression and host factor may play a role in the clinical infection outcomes.

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**Antimicrobial susceptibility and genotypic characterization of clinical Salmonella enteritidis strains isolated from a tertiary hospital in Malaysia by using multilocus variable number of tandem repeat analysis and pulsed-field gel electrophoresis**  

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**Background:** Salmonella enterica serovar enteritidis (S. enteritidis) causes non-typhoidal salmonellosis (NTS) in humans. Twenty-eight percent of NTS serovars identified and reported to the laboratory based surveillance database of Malaysian Ministry of Health in 2005 was S. enteritidis. The increasing occurrence of multidrug resistant (MDR) S. enteritidis complicates available therapeutic options. Phage typing and pulsed-field gel electrophoresis (PFGE) are commonly used subtyping methods, but there are limitations. The advent of multi-locus variable number of tandem repeats analysis (MLVA) provided a better discrimination of S. enteritidis. This study aimed to determine the antibiograms and genotypes of clinical S. enteritidis strains isolated from a tertiary hospital in Penang, Malaysia.

**Methods:** A retrospective study involving 16 clinical S. enteritidis strains isolated from 2005 to 2006 was conducted. The resistance of the strains against 14 antimicrobial drugs was examined, and the clonality of the strains was determined by both MLVA and PFGE of XbaI digested bacterial chromosomal DNA.

**Results:** Both invasive (n = 9) and non-invasive (n = 7) S. enteritidis were examined and a high percentage of multidrug resistance was observed (66% of invasive and 43% of non-invasive strains). MLVA (D = 0.77) yielded five distinct types (M1 to M5), which correlated to antimicrobial resistance patterns of the strains. Strains of MLVA type M1 were resistant to tetracycline, type M2 resistant to nalidixic acid, types M3 and M4 resistant to both ampicillin and nalidixic acid, and type M5 resistant to sulfonamides, trimethoprim, trimethoprim-sulfamethoxazole and tetracycline. The VNTR loci were genetically homogeneous (Nei’s diversity index ≤ 0.53) among the strains. PFGE (D = 0.94) subtyped all strains into 11 pulsortypes, showing high genetic homogeneity (0.84 < F < 1.00) among