

Study of the Genes Involved in the Resistance of Nosocomial *Pseudomonas aeruginosa* to Fluoroquinolone

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Abstract : The major mechanism of *Pseudomonas aeruginosa* resistance to fluoroquinolones is the alteration of target enzymes, type II and IV topoisomerases due to mutations in the quinolone resistance-determining regions (QRDR) of the *gyrA* and *parC* genes coding A subunits of these enzymes. 37 isolates from patients with burn wounds and 20 isolates from blood, urine and sputum specimen were selected to evaluate mutations involved in antibiotic resistance and were subsequently verified for their resistance to ciprofloxacin. QRDRs regions of *gyrA* and *parC* were amplified by polymerase chain reaction (PCR) and were subsequently sequenced. 90% of isolates with MIC \geq 8 μ g/ml to ciprofloxacin had a mutation in *gyrA* gene in which threonine at position 83 changed to isoleucine. 87.5% of isolates had mutation in *parC*, Serine 87 changed. 75% had Ser87Leu and 12.5% possessed Serin87Trp. Various silent mutations were also detected such as Val103Val, Ala118Ala, Ala136Ala, His132His in *gyrA* and Ala115Ala in *parC*. The data indicates that the common mutation in *gyrA* is Thr83Ile and in *parC* is Ser87Leu/Trp. No individual *parC* mutation was observed while mutations in *gyrA* and *parC* occurred simultaneously and appears to be the main reason of high-level resistance to fluoroquinolones in patients with burn wounds and urine infection. The vast majority of *P.aeruginosa* isolates had mutation in *parC* which can play a crucial role in increased resistance of these isolates. This is a report of *parC* mutations from resistant *P. aeruginosa* isolates from Iran, Tehran.

Keywords : *P. aeruginosa*, fluoroquinolones, *gyrA*, *parC*, antibiotic resistance

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