Salmonella enterica Serovar Paratyphi A Infections in India

Rajni Gaind[1], Ruchi Gupta[1], Dabet Rynga[1], Bianca Paglietti[2], Manorama Deb[1], Salvatore Rubino[2] (Presented by Geetarani Purohit)

[1]Department of Microbiology, Safdarjung Hospital and Vardhman Mahavir Medical College, New Delhi, India; [2]Department of Biomedical Sciences, University of Sassari. Italy

Background: Typhoid and paratyphoid are clinically indistinguishable. Comparative data of incidence, clinical presentation, antibiograms and molecular characterization of *S.* Typhi and *S.* Paratyphi A is scarce but vital for understanding disease epidemiology and formulating therapeutic and vaccination policies.

Methods: A retrospective hospital based study was undertaken between January 1999 and September 2011. Clinical, microbiological and epidemiological profile of *S.* Typhi and *S.* Paratyphi A were investigated.

Results: The proportion of *S.* Typhi: *S.* Paratyphi A was 7.6:1 (1999) and 2.5:1 (2004) and reverted back to 8.6:1 (2011). Paratyphoid fever was significantly more frequent in older age groups and was associated with milder disease with only 11.8% patients requiring hospitalization. The incidence of multidrug resistance in *S.* Typhi was declining, but 21% of them were still MDR. All isolates of *S.* Paratyphi A were resistant to nalidixic acid since 2003, as compared to 80% resistance in *S.* Typhi in 2005. High-level fluoroquinolone resistance was also seen first in *S.* Paratyphi A in 2003. Double mutation in *gyrA* and single mutation in *parC* were identified in ciprofloxacin resistant isolates of both serovars. Interestingly nalidixic acid resistant isolates of *S.* Paratyphi A and *S.* Typhi isolates carrying same single mutations at codon 83 in *gyr*A exhibited different ciprofloxacin MIC of 1.5 and 0.5 μg/ml respectively suggesting an additional mechanism of fluoroquinolone resistance in *Salmonella* serovar Paratyphi A. Studies with efflux pump inhibitor were suggestive of efflux mediated resistance which also contributed multiple antibiotic resistance in *S.* Paratyphi A. PFGE of the isolates of the two serovars suggested that molecular epidemiology of the two serovars is significantly different.

Conclusions: The disease epidemiology clinical presentation and mechanism of resistance differ in the two serotypes. In absence of licensed vaccine for *S.* Paratyphi A this could result in increase in Paratyphoid cases and failure of preventive strategies which are focused on Typhoid fever. Vaccination and therapeutic policies need reassessment.