Cholera Pattern in Children of Delhi

Cholera is a prototype of toxigenic diarrhea. The epidemiology of cholera took a dramatic turn in the later months of 1992, *V. cholerae* 0139 strain emerged in Madras (Chennai), which then spread far and wide(1). The present study was conduced to elucidate the type of cholera organism prevalent and its antibiotic sensitivity pattern in east Delhi.

Study period was July 2001 -Nov 2002, in pediatric emergency of G.T.B. hospital and University College of Medical Sciences, Delhi. All the patients satisfying the criteria were studied.

Inclusion criteria: All pediatric patients admitted with acute onset, profuse, rice water like diarrhea of less than 24 hrs duration(2).

Exclusion criteria: Dysentery, mucoid diarrhea and history of receiving any antibiotic drug 48 hours prior to admission. Stool samples were immediately transported in bile alkaline peptone water and processed. Hanging drop/dark ground microscopy was done to look for the motility. The stool samples were cultured using macConkey agar, Deoxycholate citrate agar (DCA), and Thiosulphate citrate bile salt sucrose agar. Plates were incubated at 37ºC for 24 hours.

Information about age, sex, caste, place of residence, socio economic status and source of water supply was taken from parents/ guardians of all patients. Susceptibility pattern of all the isolates were tested for the following antibiotics: chloramphenicol, gentamicin, cotrimoxazole, cefotaxime, furazolidine and ciprofloxacin(3).

Out of total 1324 stool samples, 133 children (10%) grew *Vibrio cholerae*. Out of these 133,70 were boys and 63 girls; 125 (94%) grew 01 Ogawa, one grew 0139 (0.8%) and 7(5.2%) were non 01-non 0139. Hanging drop was positive in 59 cases only. Maximum number of cases were from the local areas, during June to October months *i.e*., 15, 30, 20, 16 and 12 children respectively. Distribution of parents were in <1, 1-5, 5-10 and 10-12 years of age *i.e*., 13, 66, 37 and 17 respectively. 01 Ogawa was sensitive to chloramphenicol, gentamicin, cefotaxime and ciprofloxacin, but showed resistance to cotrimoxozole and furazolidine. Non 01- Non 0139 were also sensitive to the above anti-biotics except furazolidine and cotrimoxazole (moderately). Furazolidine and cotrimoxazole resistance in *V. cholerae* was seen in this study. This report addresses the fact that proper monitoring of usage of antibiotics is essential. Similar finding of growing antibiotic resistance was found by Avasthi, *et al*.(5).

The sudden emergence of *V. cholerae* 0139 on late 1992 and its quick dissemination in many countries was initially considered as the beginning of 8th pandemic of cholera. It was also thought that it might replace *V. cholerae* 01 Eltor, as the latter had replaced *V. cholerae* 01 classical in, 1960s. However, *V. cholerae* 01 biotype remained firmly established in Delhi during and after the emergency of *V. cholerae* 0139 strain(4). Why *V. cholerae* 0139 has not been able to replace *V. cholerae* 01 biotype, or has it lost its epidemic potential is still not clear.

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Antibody Response to Pulse Polio Immunization in Aligarh

The report by AS Hasan and colleagues is a timely reminder of the problems with OPV in India(1), first described in 1972(2). They measured seroconversion rates in three subgroups - vaccinated exclusively through NIP or pulse campaigns and vaccinated both ways(1). The highest response was in the last subgroup that received the highest number of doses (mean 8.4) of OPV. Seroconversion rate increases with increasing doses(3). This correlation is illustrated by Hasan's data, presented differently. For simplifying inter-group comparison, 'seroconversion index', a single variable, is useful in place of three variables of type-specific seroconversion rates(3). The mean of seroconversion rates will suffice as surrogate for seroconversion index(3). The *Table I* gives the results.

A similar gradation is also present with geometric mean antibody titres (GMT) - lowest in the NIP subgroup, and highest in the subgroup of NIP and pulse(1). Repeated infections do result in rise in GMT(3). Thus, without controlling for the number of doses the study is inadequate to compare differential responses of pulse versus NIP.

Loss of vaccine potency could not have been the reason for poor antibody response, since vaccine vial monitors were mandatory in 1999-2002(1). Their recommendation to investigate the cause of poor response is untimely in 2004, but what is urgently needed is to explore methods of improving immune responses for achieving interruption of wild poliovirus transmission. In spite of 96.5% coverage with 2-18 doses of OPV, gaps in immunity remained in the vaccinated and

TABLE I–*Relationship Between Number of Doses and Seroconversion Rate.*

Group/subgroup	Mean No. of doses	Mean sero conversion rate
NIP subgroup	3.8	80.8
Pulse subgroup	6.3	84.8
Vaccinated group	7.8	87.5
NIP and pulse subgroups	8.4	88.5