Cholera transmission in the measles ward?

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Two hypothetical African cities suffer from mild epidemics of El Tor cholera, with overall incidences of about 0.1% over a five-week period. The general hospitals in both cities set up cholera wards. In both of the hospitals diagnostic ability is poor, since the medical staff have never seen cholera and have received no special training in cholera diagnosis. In City A there are no laboratory facilities, and so all cases of serious diarrhoea are treated as “cholera cases”. In City B the hospital has a good laboratory, and so only those cases of diarrhoea which come to the hospital and are positive to tests for V. cholerae are considered to be “cholera cases”.

Both of the hospitals have measles wards. In examining the records for the epidemics after five weeks, the staffs of each hospital discover an alarmingly high rate of cholera among the children of the measles ward: in City A the “cholera attack rate” among the children in the measles ward is more than 50 times higher than the attack rate among children in the community, while in City B the “cholera attack rate” among the children in the measles ward is more than 200 times that of the children in the general population. In both hospitals the staffs conclude that cholera transmission took place in the measles ward of the hospital. The conclusion seems irrefutable, but is it correct?

THE BASIC INFORMATION

The first piece of information is that the infection:case ratio for El Tor cholera is extremely high. On the basis of a variety of data presented by Gangerosa & Mosley (1974), we assume this ratio to be 25:1.

The second piece of information pertains to the relative susceptibility, in poor countries, of children with and without measles to all kinds of diarrhoeal infections. As Morley (1973) has stated: “The doctor who has received his training in the industrial societies of the western world will be surprised by the prevalence of diarrhoea following measles”. Data from the Cholera Research Laboratory in Bangladesh (Koster et al. 1981) show that, of a group of children who were ill with measles, 63% had a serious bout of diarrhoea in a five-week period surrounding the measles episode. In a control group only about 4% of the children had a serious attack over a similar period. That is, apparently because of severe depression of the systemic immune response during and shortly after an attack of measles, the measles children had diarrhoea rates about 16 times higher than the children who had not recently had an attack of measles.

The final piece of information concerns the effect of depression of the systemic immune response on the infection:case ratio for cholera. Since the predominant immune response to cholera appears to be antibacterial rather than antitoxic (Mosley 1969), we expect lowered systemic immunity to result in higher rates of colonization of the small intestine by V. cholerae but not to affect the proportion of those colonized by V. cholerae who will develop clinical disease. That is, we expect the infection:case ratio to be similar both for the children who have, and for those who have not, recently had measles.

We assume that these data pertain to the population of the two African cities and consider the cholera epidemics in more detail.

City A

Recall that in the hospital of this city there are no laboratory facilities: all cases of serious diarrhoea are considered to be “cholera cases”.

In the children of the community, 0.1% of the children are genuine cholera cases, the attack rate being assumed to be the same for children and adults in this non-endemic area. All of the genuine cholera cases are assumed to come to the hospital. In addition, 4% of the children in the community suffer from serious episodes of diarrhoea during this five-week period. We assume that 25% of these cases (i.e., 1% of all the children in the community) come to hospital, where they are considered to be “cholera cases”. The hospital staff believe, therefore, that 1.1% of the children in the community contracted cholera. Of these, 91% (i.e., 1.0/1.1) are “false cases”.

We follow the same analysis for the children of the measles ward, assuming that their exposure to infection by V. cholerae is exactly the same as the exposure of the children in the community.
On the measles ward, because of the poor immune status of the children, the attack rate from cholera is 16 times higher than that in the children in the community. That is, $16 \times 0.1\% = 1.6\%$ of the measles children develop genuine clinical cases of cholera. Over the five-week period, 63% of the remaining 98.4% of the measles children (i.e., 62% of the measles children) develop serious diarrhoea and are considered to be "cholera cases". Thus, 63.6% of the measles children are considered to have contracted cholera. Of these cases, 97.5% (i.e., 62.0/63.6) are "false cases".

Thus, with exactly the same exposure to infection from *V. cholerae* in the general community and the children on the measles ward, the apparent "cholera attack rate" will be 58 times (i.e., 63.6/1.1) higher in the children of the measles ward than in the community.

City B
Recall that in this city the hospital staff is again unable to diagnose cholera correctly, but the hospital does have a laboratory capable of identifying *V. cholerae*.

In the children of the community, 0.1% are genuine cholera cases, all of which are assumed to come to the hospital. They are swabbed and correctly identified as cholera cases. In addition, 4% of the community children suffer from serious episodes of diarrhoea in the five-week period. As in City A, we assume that 25% of these cases (i.e., 1% of the children in the community) come to the hospital for treatment. At the hospital they are swabbed and their faeces tested for *V. cholerae*. Since the case rate is 0.1%, the carrier rate is $25 \times 0.1\% = 2.5\%$. That is, 2.5% of the 1% who come to the hospital with serious non-cholera diarrhoea will be diagnosed as "cholera cases". This amounts to 0.025% of the population of children. Thus, in total, 0.125% of the community children will be diagnosed as having contracted cholera. Of these, 20% (i.e., 0.025/0.125) are "false cases".

We follow the same analysis for the measles children, assuming that their exposure to cholera is exactly the same as the exposure of the children in the community.

As in City A, 1.6% of the measles children develop genuine cases of clinical cholera. Among the remaining children on the measles ward, the cholera carrier rate is 25 times the case rate, that is, $25 \times 1.6\% = 40\%$. Over the five-week period, 63% of the remaining 98.4% of the measles children develop serious diarrhoea and are swabbed for cholera tests. The 40% who are carriers of cholera will be positive on testing. That is, $63\% \times 98.4\% \times 40\% = 24.8\%$ of the measles children will be confirmed, by the laboratory, to be excreting *V. cholerae*. Although none of these are actual cases of clinical cholera, all will be considered to be "cholera cases". Thus, the hospital staff believes that 1.6% + 24.8% = 26.4% of the children on the measles ward contracted cholera. However, 24.8/26.4 = 94% of these are "false cases".

The situation in City B, then, is that, with exactly the same exposure to cholera in the general community and in the measles ward, the "laboratory-confirmed cholera attack rate" is 26.4/0.125 = 211 times higher in the children of the measles ward than in the children in the general population.

Conclusion
From the analysis of these outbreaks in hypothetical African cities we can conclude:

1. In the absence of good laboratory facilities, the number of cholera cases will be greatly over-estimated: In City A over 90% of the community "cholera cases" are not cholera cases at all, but are cases of serious non-cholera diarrhoea; in City B, with good laboratory facilities, only about 20% of the community "cholera cases" are non-cholera diarrhoeas.

2. The extremely high apparent attack rate of cholera in the measles wards has nothing to do with excessive transmission of cholera in the measles wards, but is a result of the poor immune status of the children on the ward, the high carrier:case ratio for El Tor cholera, and the difficulty of making accurate diagnoses of a disease with which the medical personnel are not familiar. Surprisingly, in the absence of good diagnostic capability, an erroneous concern with excessive cholera transmission in the measles ward is most likely where the hospital has good laboratory facilities.

REFERENCES