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Studies on mumps virus infection, genotype identification of circulating strains among MMR vaccine recipients

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Mumps, long considered a vaccine preventable childhood infection has now caused worldwide re-emergence in vaccinated populations. Thus, a study was done to investigate the cases of vaccine failure among mumps suspect cases in Chennai, India. Results revealed an alarming 90.5 % of vaccinated cases were positive for anti-mumps IgM antibody, indicating that MMR vaccine had failed to offer protection. This is the first report to portray the high prevalence of mumps in vaccinated populations in India. Genotypic characterization of the virus revealed that the circulating strain was genotype C which is distinct from the vaccine strain of genotype N (L-Zagreb). This is also the first report in India to suggest that genotype C was responsible for the present mumps infection. Poor efficacy is a contributing factor to the failure of MMR vaccine and hence, its efficacy was analyzed by determining the seroprotective antibody level. Highest seropositivity (100%) was noticed for rubella, an intermediate number (76% & 92% who received two doses and first doses of MMR) for measles and the lowest (49% & 83%) for mumps virus. This warrants a revisit of vaccine preparation using circulating strains and optimization to improve its efficacy.

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Has rift valley fever virus evolved to increased virulence in human population after existence for a century in East Africa?

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In Kenya, of 22 RVF outbreaks from 1912 to 2007, 14 were national affecting 3-38/69 districts while 8 were localized to 1-2/69 districts. Most of these outbreaks were epizootics in early 1900s and were not accompanied by significant epidemics in human populations in East Africa. However, in 1998 and 2006-2007, RVF outbreaks in the horn of Africa led to 478 and 350 deaths. In 2008, comparable epidemics occurred in Sudan (698 human cases; 222 deaths) and Mozambique (412 human cases, 17 deaths). Comparisons between isolates from different outbreaks reveal specific genetic mutations and reassortments that have diversified RVF virus genomes over the past century. Although genetic diversity of RVF virus appears to be low (~5%), these changes in combination with the accumulation of mutational changes over the years could have influenced RVF virus host preference and virulence in human populations. Factors such as underreporting, lack of appropriate diagnostic kits, under-recognition of clinical signs, changes in case definitions could have influenced paucity of information on human RVF in early 1900s. However, the explosive nature and pronounced RVF epidemics in humans in recent decades became so overwhelming to attribute the obvious changes to improvement in the above mentioned factors. We speculate that the evolutionary diversification of the virus could have resulted in distinct lineages with increasing virulence and pathogenicity in humans. The emerging infectious disease threats posed by the modern RVF virus strains to humans increases the potential public health and socio-economic impacts of future RVF epidemics.

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