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Paralytic poliomyelitis associated with live oral poliomyelitis vaccine in child with HIV infection in Zimbabwe: case report

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Abstract

Objective To describe a complication of oral vaccination with live, attenuated poliomyelitis virus in a child infected with HIV.
Design Case report.
Setting Teaching hospital in Harare, Zimbabwe.
Subjects A boy of 4½ years and his mother.
Main outcome measures Results of clinical and laboratory investigations.
Results Two weeks after receiving the second dose of oral poliomyelitis vaccine during national immunisation days the child developed paralysis of the right leg. He had a high titre of antibodies against poliovirus type 2, as well as antibodies against HIV-1, a low CD4 count, a ratio of CD4 to CD8 count of 0.47, and hypergammaglobulinaemia. He did not have any antibodies against diphtheria, tetanus, or poliovirus types 1 and 3, although he had been given diphtheria, tetanus, and pertussis and oral polio vaccines during his first year and a booster of the diphtheria, tetanus, and pertussis vaccine at 24 months. He had no clinical symptoms of AIDS, but his mother had AIDS and tuberculosis.
Conclusion Paralytic poliomyelitis in this child with HIV infection was caused by poliovirus type 2 after oral poliomyelitis vaccine.

Introduction

The expanded immunisation programme in Zimbabwe started in 1981 and has a coverage of around 85% in most areas of the country.[1] The vaccination schedule is three doses of trivalent oral, live attenuated, poliomyelitis vaccine and diphtheria, tetanus, and pertussis vaccine at 3, 4, and 5 months of age, with a booster of diphtheria, tetanus, and pertussis vaccine at 18 months. In line with the World Health Organisation's goal of eradicating poliomyelitis by 2000,[2] children under 5 years old in Zimbabwe received two doses of oral vaccine, regardless of their vaccination history, during the national immunisation days in 1996.[3] Most children infected with HIV live in developing countries, so the influence of HIV infection on vaccination against poliomyelitis is relevant. We describe a case of paralytic poliomyelitis in a child with HIV infection after vaccination with oral poliomyelitis vaccine.

Case history

A boy aged 4½ years who was infected with HIV had been vaccinated with diphtheria, tetanus, and pertussis vaccine and oral poliomyelitis vaccine at the ages of 3, 4, and 5 months and had received a booster of diphtheria, tetanus, and pertussis vaccine at 24 months. On the national immunisation days of 1996 (7 August and 29 September) he received oral poliomyelitis vaccine, and a few days after the second immunisation he developed diarrhoea and fever. Two weeks later he developed weakness in his right leg. He was seen at a local primary healthcare clinic, but laboratory tests were not performed.

Three months later, in January 1997, he came to Parirenyatwa Teaching Hospital in Harare because of

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Key messages

- The WHO's goal of eradicating poliomyelitis by 2000 means that children are given live, oral poliomyelitis vaccine during national immunisation days regardless of their vaccination history
- Live vaccines are contraindicated in people who are infected with HIV because of the risk of infection from attenuated micro-organisms
- The incidence of paralytic poliomyelitis associated with vaccination is low in children who are not infected with HIV
- A boy positive for HIV infection developed paralytic poliomyelitis after receiving his second dose of oral poliomyelitis vaccine during national immunisation days in Zimbabwe
- As the benefits of vaccination outweigh the risk of infection with wild poliomyelitis virus, oral poliomyelitis vaccine should continue to be used in countries where HIV infections are endemic

the persistent paralysis. On examination he was well nourished and had flaccid paralysis of his right leg, with diminished tone, power, and reflexes. Magnetic resonance imaging showed appreciable wasting of the muscles of his leg. His lymphocyte count was 2.1×10^6/l, haemoglobin concentration 108 g/l, erythrocyte sedimentation rate 61 mm in the first hour, and total IgG concentration 29.3 g/l (normal value for children aged 5-7 years in Harare 8.0 (SD 3.2) g/l[4]). A serum sample contained no antibodies to tetanus or diphtheria toxin (both <0.01 IU/ml; toxin binding inhibition assay).

Poliovirus and poliovirus antibodies—Poliovirus was not cultured from three stool specimens collected 24 hours apart. Serological tests showed a titre for poliovirus type 2 antibody of 1:1024 but no antibodies to poliovirus types 1 and 3 (both titres $<1:8$) (microneutralisation test; WHO poliomyelitis reference laboratory, Harare). These titres were confirmed by the Dutch National Institute of Health and the Environment in Bilthoven (titre of poliovirus type 2 antibody 1:512 and no antibodies against poliovirus types 1 and 2). In a second serum sample taken in December 1997 no IgM antibodies against poliovirus types 1, 2, and 3 and no IgG antibodies against poliovirus types 1 and 3 were detected; IgG titre against poliovirus type 2 was 1:16 (microneutralisation test, Dutch National Institute).

HIV antibodies and CD4 counts—Serum antibodies against HIV-1 were detected in two enzyme immunoassays (Sanofi Diagnostics Pasteur, Marnes la Coquette, France, and Abbott, Wiesbaden-Delkenheim, Germany). In January 1997 CD4 count was 733×10^6/l (normal count for children aged 1-5 years $>1000 \times 10^6$/l), CD8 count 1576×10^6/l, and the ratio of CD4 to CD8 count 0.47. In a second serum sample taken in December 1997 the persistence of HIV-1 antibodies was confirmed by enzyme immunoassay (Abbott) and western blotting (Genelabs Diagnostics, Singapore).

Maternal history—During the second half of 1997 the patient's mother became severely ill, having lost much weight. She was treated for tuberculosis for six months in hospital outside Harare; her HIV serology was positive. She also had chronic diarrhoea. According to WHO case definitions,[5] she had AIDS. She died in August 1998.

Follow up—In February 1999 the child still had severe paralysis of his leg and a pronounced limp. He was admitted because of weight loss. An *x* ray film showed bilateral hazy infiltration of the lungs and he was treated for tuberculosis. His HIV infection was progressing to AIDS.

Discussion

The results of physical examination and the presence of antibodies against poliomyelitis virus meet the criteria of the ninth revision of the international classification of diseases (ICD-9) for paralytic poliomyelitis, as the paralysis occurred within 4-30 days after vaccination.[1] Poliomyelitis was probably caused by the vaccine strain of poliovirus type 2. The presence of HIV antibodies meet the WHO's criteria for confirmed HIV infection,[5] which was probably acquired perinatally.

Since 1990 no wild type poliovirus has been isolated in Zimbabwe (WHO Poliomyelitis Reference Laboratory, Harare, personal communication).[1] In the neighbouring country of Namibia, however, an outbreak of poliomyelitis caused by wild type poliovirus type 1 occurred in 1993.[6] A review of hospital records during 1990-2 showed that three children under 15 years old developed acute flaccid paralysis in Zimbabwe.[1] Virus isolation and antibodies were not reported, so the infection may not have been caused by poliovirus. In 1995 acute flaccid paralysis was diagnosed in 39 children in Zimbabwe; poliovirus type 1 was isolated from stool samples in two children aged 5 and 6 years who did not remain paralysed. In 1996 among 20 children with acute flaccid paralysis poliovirus type 2 was isolated from one child aged 1 year who had residual paralysis (WHO, Poliomyelitis Reference Laboratory, Harare, personal communication). The HIV status of these last three children is not known.

Our patient probably had a humoral immune deficiency because after vaccination he did not develop antibodies against diphtheria toxin, tetanus toxin, or poliovirus types 1 and 3 and probably not against poliovirus type 2 after oral vaccination during infancy. This deficiency could be due to an altered immune state from his perinatally acquired HIV-1 infection. Interestingly, after infection with the vaccine strain he made antibodies against poliovirus type 2. Children infected with HIV-1 develop significantly less antibody than uninfected children in response to diphtheria toxoid, tetanus toxoid, and oral poliomyelitis vaccine.[7 8] Adults with HIV infection and low CD4 counts also form less antibody than do those with CD4 counts $>300 \times 10^6$/l in response to diphtheria toxoid, tetanus toxoid, and inactivated poliomyelitis vaccine.[9]

In our patient humoral immune deficiency, especially the absence of polio antibodies, might have contributed to the dissemination of the virus after vaccination with oral vaccine during the national immunisation days, thus causing infection of the central nervous system. The prevalence of paralytic poliomyelitis associated with oral poliomyelitis vaccine is low: one case per 2.5 million doses. In addition, 18% of those who developed this complication were immunocompromised, predominantly young children with hypogammaglobulinaemia or agammaglobulinaemia.[3 10 11] Children infected with HIV are potentially at risk of this and other complications after vaccination

with live attenuated micro-organisms.[3 7 8] We believe that only one case of paralytic paralysis associated with oral poliomyelitis vaccine has been reported in a child with HIV infection from Romania.[12] Thus oral poliomyelitis vaccine seems to be safe when given during the first year of life.[1 7 10 13] To our knowledge, our case is the first report of poliomyelitis associated with poliomyelitis vaccination in a child infected with HIV from Africa. In countries where HIV infection is endemic and the risk of infection with wild type poliomyelitis virus is high, the benefits of immunisation outweigh the apparently low risk of paralysis due to vaccination with oral poliomyelitis vaccine.

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Postcodes as useful markers of social class: population based study in 26 000 British households

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Markers of poverty or of low social class are associated with many diseases and potential causes of disease, but medical studies often fail to record sufficient information on socioeconomic status.[1] Postcodes of individuals are, however, often available in Britain, and commercial software exists that estimates household income from the postcode alone. We assessed how informative postcode income estimates are, either about reported household income or about other characteristics related to social class in a large, population based survey of British residents.

Methods and results

The family resources survey involves personal interviews with members of private households in England, Scotland, and Wales selected by stratified clustered probability sample.[2] During 1995-6, 26 445 (70%) of 37 712 eligible households gave answers to questions on socioeconomic characteristics. Reported weekly household income was taken as the sum of all sources of pretaxation income (excluding housing benefit) reported by household members. During 1985 to 1993 members of 11 million households, or about half of all households in Britain, provided information to a marketing company about annual income and gave a complete address that included a full postcode—that is, 6 or 7 characters.

This information was used to produce commercial software that estimates household incomes from postcodes. After adjustments for regional variation and for inflation in reported income levels, the pretaxation incomes of at least six households were used to calculate a weighted average income for that postcode. When there were fewer than six responses, the income information was combined with the data for respondents with neighbouring postcodes until a reliable estimate could be made. Parts of this database are updated annually. We compared household income estimates obtained by FIND (a commercially available software program) with information reported in the family resources survey. Matching of the data was carried out at the Office for National Statistics. The investigators in this study were provided with columns of numerical data without any personal identifiers.

The overall correlation coefficient between postcode estimates and reported values of weekly household income for 26 282 individuals was moderate (0.40, 99% confidence interval 0.39 to 0.42; $2P < 0.0001$). When households were ranked in three equal sized groups on the basis of postcode income estimates, there were substantial and highly significant differences in reported weekly income, duration of education, home ownership, membership of higher social classes, and access to various consumer goods ($2P < 0.0001$ for each) (see table).

Comment

Postcode income estimates are easily available in Britain and can be useful markers of social class. As UK postcodes are usually shared by only 15 to 20 households,[3] these estimates should more accurately predict the social class of individuals than can more

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