Implications of the Human Immunodeficiency Virus Epidemic for Control and Eradication of Measles

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Human immunodeficiency virus (HIV)-infected persons may be important, unrecognized transmitters of measles virus, thwarting eradication efforts. We reviewed the published English-language literature on measles and measles immunization in HIV-infected persons to investigate the clinical features of measles, the responses to measles immunization, and the safety of measles vaccine in HIV-infected persons and, conversely, the effect of measles and measles immunization on HIV infection. HIV-infected persons with measles are likely to have uncharacteristic clinical findings and severe illness, with high rates of pneumonitis and death. Primary and secondary failure of measles vaccine in HIV-infected children may permit transmission of measles virus in spite of high rates of immunization coverage. A factor that complicates measles-control efforts in areas of high prevalence of HIV is the potential for fatal infection with measles vaccine virus. Further research on the impact of the HIV epidemic on measles and measles immunization is necessary to guide strategies for the eradication of measles.

The World Health Organization considers eradication of measles to be technically feasible with available measles vaccines, and the years 2005–2010 have been proposed as a target date [1, 2]. The major obstacles to eradication of measles are claimed to be perceptual, political, and financial. Although HIV infection is recognized to be a major factor in the transmission of tuberculosis and sexually transmitted infections, the role of the HIV epidemic in enhancing transmission of measles virus has not been addressed. HIV-infected persons may be important, unrecognized transmitters of measles virus, thwarting eradication efforts.

Several factors suggest that the HIV epidemic may enhance transmission of measles virus and impede the eradication of measles. HIV-infected mothers may have defective transfer of IgG antibodies across the placenta, which results in lower titers of protective antibodies in the infant and enlarges the window of susceptibility to infection before the infant receives routine immunization. HIV-infected children may not respond adequately to measles immunization and thus remain susceptible to measles infection. Enders and colleagues [3, 4] recognized in 1959 that immunocompromised children develop unusual and severe clinical manifestations following infection with measles virus, including fatal giant cell pneumonitis and prolonged shedding of virus, often in the absence of rash. Studies of measles in severely malnourished children also demonstrated prolonged excretion of virus [5, 6]. Similarly, HIV-infected children and adults with measles may have prolonged excretion of measles virus and unusual clinical manifestations, resulting in misdiagnosis.

Methods

We reviewed the published English-language literature about measles and measles immunization in HIV-infected persons identified through a search of the MEDLINE database by use of the keywords “measles,” “human immunodeficiency virus,” and “acquired immunodeficiency syndrome.” References cited in the retrieved publications were used to identify additional published studies about measles and measles immunization in HIV-infected persons. This body of literature was used to investigate the clinical features of measles, the responses to measles immunization, and the safety of measles vaccine in HIV-infected persons and, conversely, the effect of measles and measles immunization on HIV infection. The purpose of the investigation was to assess the implications of the HIV pandemic for control and eradication of measles.

Results

Clinical features of measles in HIV-infected children and adults. A report from the Centers for Disease Control (CDC; now called Centers for Disease Control and Prevention) first brought to medical attention the unusual and severe clinical manifestations of measles in five HIV-infected children (a sixth child was reported but subsequently found not to be HIV-
infected) [7]. Two of the five children did not have a characteristic measles rash. One of these children had only a transient rash but developed severe measles pneumonitis, and the other died of measles giant cell pneumonitis but never developed a rash. These children had received either varicella immune globulin or intravenous immune globulin [8]. Although the administration of varicella immune globulin or intravenous immune globulin may have altered the character of the rash in these children, additional case reports from the United States confirmed the unusual clinical manifestations and severity of measles in HIV-infected persons. In particular, several case reports documented a delayed, uncharacteristic, or absent rash and the frequent occurrence of pneumonitis in HIV-infected children [9–14] and adults [14–17] with measles.

Because of the uncharacteristic, delayed, or absent rash, the diagnosis of measles can be overlooked in HIV-infected persons, increasing the potential for nosocomial transmission of measles virus. HIV-infected persons also may be at increased risk of nosocomial acquisition of measles because of frequent clinic visits and hospitalizations. Four of the five HIV-infected children reported by the CDC acquired measles within a health care setting [7]. One of the children was the index case for further nosocomial transmission to two primary and one secondary contacts [8]. In subsequent reports, nosocomial acquisition of measles was documented for five of seven HIV-infected children [11, 12].

Although the most commonly reported complication of measles in HIV-infected persons is pneumonitis, measles-associated neurological disorders in HIV-infected children and young adults have been described, including encephalitis [15, 18, 19], subacute encephalitis [20–22], subacute scarring pneumonia encephalitis [23, 24], and myelopathy [19, 25]. The descriptions of subacute measles encephalitis [20–22] are consistent with measles inclusion body encephalitis, a progressive neurological disease that typically occurs in immunocompromised persons 1–6 months after measles virus infection. The two reported HIV-infected children with subacute scarring pneumonia encephalitis are unusual because of their young ages (18 and 21 months) [23, 24]; these children may have had measles inclusion body encephalitis. Both measles inclusion body encephalitis and subacute scarring pneumonia encephalitis are characterized pathologically by intranuclear and intracytoplasmic inclusions; however, more rapid neurological deterioration occurs in measles inclusion body encephalitis because of the impaired immune response. Involvement of the spinal cord by measles virus is unusual; the one reported child developed a progressive gait disturbance 1 month after having clinical measles [25]. Pathological examination of the spinal cord showed vacuolar myelopathy and intranuclear inclusion bodies. An in situ nucleic acid probe for measles virus in the grey matter of the spinal cord yielded positive results.

The relationship between the severity of measles and the degree of HIV-induced immunosuppression is not well characterized. CD4⁺ T lymphocyte counts in HIV-infected children and adults with measles ranged from 2 to 1,921/mm³ [8, 10, 12, 14, 15, 17]. The ratios of CD4⁺ to CD8⁺ T lymphocytes ranged from 0.17 to 2.6 in four HIV-infected children with measles [9]. Although there are few HIV-infected persons with measles for whom CD4⁺ T lymphocyte counts are available, severe measles did occur in eight persons with CD4⁺ T lymphocyte counts of >200/mm³.

The effectiveness of antiviral therapy in HIV-infected persons with measles is unclear, because the numbers of persons studied are few. Ribavirin alone and in combination with intravenous immune globulin was used to treat measles in HIV-infected persons on the basis of limited efficacy data in immunocompetent hosts [26]. Three children [7, 9] and three adults [14, 17] with severe measles pneumonitis were treated with ribavirin; one child and one adult died. Two adults treated with the combination of ribavirin and intravenous immune globulin survived [15, 16]. Ribavirin also was administered to an HIV-infected man with fatal subacute measles encephalitis [21].

In the United States, the case-fatality rate (CFR) in HIV-infected persons with measles is much higher than the baseline rate of 0.1%. Six of the 19 reported HIV-infected children with measles died (almost one-third), and one of five HIV-infected adults died. Seven of the 19 persons who died from complications of measles in New York City between 1990 and 1991 were HIV-seropositive; six persons were HIV-seronegative and the HIV status of the remaining six was unknown, although HIV infection was suspected in four [27]. Of the 12 children who died during an epidemic of measles in Puerto Rico in 1990, 3 were HIV-seropositive [28].

The data comparing the severity of measles in HIV-infected and -uninfected children in developing countries are less consistent. In former Zaire, 5% of 314 hospitalized children with measles were HIV-seropositive [29]. Overall, no difference in CFR between HIV-seropositive and HIV-seronegative children was found. However, there was a suggestive, but not statistically significant, difference in CFR for children >9 months of age: 50% of 8 HIV-seropositive children died, compared with 29% of 208 HIV-seronegative children. In Zambia, the CFR for measles was significantly higher in HIV-seropositive children (28% of 68) than in HIV-seronegative children (8% of 356) [30]. The CFR for measles was lower in vaccinated than in unvaccinated HIV-seronegative boys, whereas no difference in CFR was found between vaccinated and unvaccinated HIV-seropositive boys and HIV-seronegative girls. Interestingly, the measles CFR was higher in vaccinated (42% of 12) than in unvaccinated (17% of 12) HIV-seropositive girls.

**Measles immunization of HIV-infected persons.** At the time of the CDC report on measles in HIV-infected children, standard practice proscribed administration of live, attenuated measles vaccine to HIV-infected children. After this report, the Advisory Committee on Immunization Practices recommended that asymptomatic HIV-infected children be immunized with measles-
mumps-rubella (MMR) vaccine and consideration be given to vaccinating symptomatic HIV-infected children [31]. However, prospective and cross-sectional studies suggest that HIV-infected children may not develop adequate protective immunity following measles immunization. We found two published prospective studies of the response to a single dose of standard-titer measles vaccine [9, 12] and five studies of the response to two doses of vaccine [12, 32–35]. About one-quarter to one-third of the HIV-infected children responded to measles vaccine in the prospective studies (table 1). The response to a second dose of vaccine was variable but generally poor.

The prevalence of antibody to measles virus among vaccinated HIV-infected children was reported in several cross-sectional studies [9, 33–41]. The study populations varied widely in age at measles immunization, the number of doses of vaccine received, the interval between immunization and assay, the method used to assay antibody to measles, and the degree of immunosuppression at the time of assessment (table 2). Several studies included both children with perinatally acquired and children with transfusion-acquired HIV infection. The median prevalence of antibody among vaccine recipients was 60%, but there was a wide range (17%–100%). Compared with a control group of vaccinated HIV-uninfected children, HIV-infected children were found in several studies to have a significantly lower prevalence of measles antibody [32, 40, 41]. HIV-infected children appear to have a more rapid decline in measles antibody than do immunocompetent children [13, 32, 34, 41], with resultant secondary vaccine failure.

<table>
<thead>
<tr>
<th>Reference</th>
<th>Year</th>
<th>No. of children</th>
<th>Mean age at immunization, mo (range)</th>
<th>Mean interval between immunization and assay, mo (range)</th>
<th>Assay</th>
<th>Response to primary immunization</th>
<th>Response to repeat immunization</th>
</tr>
</thead>
<tbody>
<tr>
<td>[12]</td>
<td>1992</td>
<td>35</td>
<td>81 (12–194)</td>
<td>3.3 (1–8.8)</td>
<td>EIA</td>
<td>37%</td>
<td>0*</td>
</tr>
<tr>
<td>[32]</td>
<td>1993</td>
<td>2</td>
<td>. . .</td>
<td>. . .</td>
<td>EIA</td>
<td>. . .</td>
<td>50%</td>
</tr>
<tr>
<td>[33]</td>
<td>1994</td>
<td>4</td>
<td>52 (22–121)</td>
<td>6 (1–14)</td>
<td>IFA</td>
<td>. . .</td>
<td>0</td>
</tr>
<tr>
<td>[34]</td>
<td>1995</td>
<td>3</td>
<td>(72–120)</td>
<td>. . .</td>
<td>EIA</td>
<td></td>
<td>66%</td>
</tr>
</tbody>
</table>

NOTE. EIA = enzyme immunoassay; IFA = indirect fluorescence assay; MN = microneutralization EIA.

* Four children received repeat immunization.

Table 2. Seroprevalence of antibody to measles virus in HIV-infected children.

<table>
<thead>
<tr>
<th>Reference</th>
<th>Year</th>
<th>Country</th>
<th>No. of children</th>
<th>Mean age at immunization, mo (range)</th>
<th>Mean interval between immunization and assay, mo (range)</th>
<th>Assay</th>
<th>Measles seroprevalence (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>[36]</td>
<td>1987</td>
<td>Rwanda</td>
<td>3*</td>
<td>. . .</td>
<td>1.3</td>
<td>. . .</td>
<td>100</td>
</tr>
<tr>
<td>[37]</td>
<td>1990</td>
<td>Romania</td>
<td>16</td>
<td>. . .</td>
<td>. . .</td>
<td>HIA</td>
<td>25</td>
</tr>
<tr>
<td>[12]</td>
<td>1992</td>
<td>United States</td>
<td>80</td>
<td>. . .</td>
<td>. . .</td>
<td>EIA</td>
<td>40</td>
</tr>
<tr>
<td>[38]</td>
<td>1993</td>
<td>United Kingdom</td>
<td>9</td>
<td>. . .</td>
<td>(3–9)</td>
<td>EIA</td>
<td>100</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>12</td>
<td>(12–15)</td>
<td>1–3</td>
<td>EIA</td>
<td>50</td>
</tr>
<tr>
<td>[33]</td>
<td>1994</td>
<td>United States</td>
<td>10</td>
<td>24 (12–95)</td>
<td>44 (1–118)</td>
<td>IFA</td>
<td>20</td>
</tr>
<tr>
<td>[39]</td>
<td>1994</td>
<td>United States</td>
<td>17</td>
<td>. . .</td>
<td>3.1</td>
<td>EIA/MN</td>
<td>77</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>26</td>
<td>. . .</td>
<td>13.3</td>
<td></td>
<td>80</td>
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<td></td>
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<td></td>
<td></td>
<td>26</td>
<td>44</td>
<td></td>
<td>58</td>
</tr>
<tr>
<td>[40]</td>
<td>1994</td>
<td>United States</td>
<td>199†</td>
<td>. . .</td>
<td>158 (84–228)‡</td>
<td>EIA</td>
<td>41</td>
</tr>
<tr>
<td>[41]</td>
<td>1995</td>
<td>United States</td>
<td>37</td>
<td>16 (14–28)</td>
<td>19 (1–80)</td>
<td>EIA</td>
<td>60</td>
</tr>
<tr>
<td>[34]</td>
<td>1995</td>
<td>United States</td>
<td>9</td>
<td>16 (8–26)</td>
<td>11.4 (3–29)</td>
<td>EIA</td>
<td>78</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>17</td>
<td>(14–42)</td>
<td>29 (1–127)</td>
<td>EIA</td>
<td>82</td>
</tr>
<tr>
<td>[35]</td>
<td>1996</td>
<td>United States</td>
<td>81§</td>
<td>14 (6–123)</td>
<td>6 (1–155)</td>
<td>MN</td>
<td>72</td>
</tr>
</tbody>
</table>

NOTE. EIA = enzyme immunoassay; HAI = hemagglutination inhibition assay; IFA = indirect fluorescence assay; MN = microneutralization EIA.

* HIV-seropositive (HIV infection not confirmed).

† Children and adolescents with hemophilia.

‡ Age at time of assay; age at immunization was not available.

§ Twenty-seven children received two or more doses of measles vaccine.
titer of antibody to measles virus, as determined by EIA, decreased from 81 EU/mL to 41 EU/mL 9–15 months after immunization in HIV-infected children, compared with a decline from 133 EU/mL to 104 EU/mL 13–22 months after immunization in HIV-uninfected children [32]. The median half-life of IgG antibody to measles virus was 18 months in 17 HIV-infected children, and the median time to loss of EIA-detectable antibody was 30 months after immunization [41]. No correlation was demonstrated between CD4 T lymphocyte count and slope of antibody decline. With a cross-sectional study design, children studied <1 year after immunization were more likely to have measles antibody (47% [47%] of 15) than were children studied >2 years after immunization (7 [47%] of 15) [35].

An association between lack of measles virus–specific antibodies after vaccination and low CD4 T lymphocyte count was documented in one prospective study [12] and two cross-sectional studies [35, 41]. A third cross-sectional study failed to demonstrate this association [32], and another cross-sectional study showed no association between the presence of antibody to measles virus and symptomatic HIV infection [39].

Placental transfer of maternal IgG antibodies is crucial for the protection of young infants against measles, but maternal antibodies can inhibit the response to measles vaccine. Placental transfer of maternal antibodies, including antibody to measles virus, may be impaired in HIV-infected women despite maternal hypergammaglobulinemia [42–44]. Children born to HIV-infected women, whether the children are HIV-infected or not, may be at risk of acquiring measles at an early age before routine measles immunization. In Kenya, 9% of 109 children born to HIV-infected mothers and only 3% of 194 children born to HIV-uninfected mothers acquired measles before 9 months of age (P = .02; OR = 3.8; 95% CI, 1.2–13.2) [42]. The incidence of measles after 9 months of age was not different between the two groups. In Rwanda, 15% of 22 HIV-infected children and 10% of 83 HIV-uninfected children born to HIV-infected mothers had antibody to measles virus that was detectable by EIA at 6 months of age, compared with 25% of 97 children born to uninfected mothers (P = .02) [45]. However, the geometric mean titer was not statistically different between the three groups. No significant difference in the presence of measles antibody measured by EIA at 6 months of age in children born to HIV-infected and -uninfected mothers was demonstrated in former Zaire [46]. In the United States, only 1 of 23 HIV-infected infants <6 months of age had maternally acquired measles antibody [13].

There appears to be a better response to measles vaccine administered at 6 months of age than to that administered at 12–15 months of age, perhaps because the younger HIV-infected infants are not yet immunocompromised [13, 35]. During a measles epidemic in Philadelphia, EIA was used 1–3 months after immunization to evaluate the responses to measles immunization in HIV-seropositive infants who received standard-titer, single-antigen measles vaccine between 6 and 12 months of age [13]. No statistically significant difference was found between 13 HIV-infected children and 22 who were initially HIV-seropositive but then tested HIV-seronegative. However, of 12 HIV-infected children vaccinated at the age of ≥12 months, only 50% had detectable measles antibody, compared with 93% of 14 children without HIV infection (P = .02).

Two studies examined the immune responses of HIV-infected children to high-titer Edmonston-Zagreb measles vaccine administered at 6 months of age. In prospective studies in Rwanda [45] and the former Zaire [46], high-titer Edmonston-Zagreb vaccine was administered to HIV-infected children, HIV-uninfected children born to seropositive mothers, and children born to HIV-seronegative mothers. The antibody responses to measles virus at 9 months of age were similar in the three groups, with 76%–94% of children responding to the vaccine. The major predictor of seroconversion was the absence of detectable maternal measles antibody at the time of vaccination. These studies were initially encouraging and suggested that measles immunization with high-titer vaccine was efficacious at 6 months of age in both HIV-infected and HIV-uninfected children. However, the high-titer vaccine was associated subsequently with increased mortality in girls, prompting withdrawal of the vaccine [47].

In adults, the response to measles vaccine has been reported in two prospective studies. None of the three HIV-infected inmates who were seronegative for measles virus developed detectable antibody after vaccination [48]. Only two of six HIV-infected U.S. Navy and Marine Corps personnel who were seronegative for measles virus had detectable measles antibody 1 year after measles vaccination [49].

Seroprevalence studies of measles in HIV-infected adults provide different information than do those of measles in children, because measles vaccination or natural infection in most adults would have occurred before subjects became HIV-infected. Measles antibody was detected in 79%–99% of HIV-infected adults (table 3) [48–56], showing that in most adults with preexisting measles immunity, antibody is not lost despite progressive HIV-induced immune suppression. Loss of antibody has, however, been reported occasionally. Among 145 HIV-infected and 50 HIV-uninfected gay men, two HIV-infected men but none of the controls lost detectable measles antibody over a period of 4–5 years (range, 1.6–5.8 years) [55]. In aggregate, there was no correlation between changes in titers of antibody to measles virus and either CD4 T lymphocyte count or development of AIDS. A lack of correlation between CD4 cell count and measles seropositivity in HIV-infected adults was also found in other studies [34, 48–51, 56].

Birth after 1957 was identified as a risk factor for lack of measles antibodies in different populations of HIV-infected adults [51–53, 56]. Persons born before 1957 are assumed to have acquired immunity from natural measles virus infection, whereas those born after 1957 are considered susceptible to
measles unless immunized. These results suggest that natural immunity to measles is more robust than it is immunity following immunization. Few studies have determined measles immunity among HIV-infected women. Measles antibody was found in 79% of 34 HIV-infected women born between 1957 and 1975 attending an HIV treatment program in New Orleans [54]. In northern California, 90% of 61 HIV-infected women had antibodies to measles [56].

Safety of standard-titer measles vaccine in HIV-infected persons. Despite initial concerns, immunization of HIV-infected children with standard-titer measles vaccine has long been considered safe [57–59]. Decision analysis suggested that exclusion of HIV-seropositive children from measles immunization would decrease vaccine-associated adverse events without greatly increasing the rate of complications of measles only under the most extreme estimates of HIV prevalence, measles vaccine efficacy, frequency of vaccine adverse events, and frequency of measles complications [60].

Published reports have supported this conclusion. In only one of the many studies of measles immunization in HIV-infected persons was a potential adverse reaction reported. One HIV-infected vaccine recipient in a prospective study of prison inmates developed fever, rash, coryza, and conjunctivitis 12 days after measles immunization [48]. However, infection with wild-type or vaccine-strain measles virus was not confirmed. A retrospective survey conducted by the New York City Department of Health found no complications following administration of 538 doses of live virus vaccines (oral poliovirus vaccine and MMR) to 221 HIV-infected persons [61]. Persistent excretion of measles vaccine virus was not demonstrated in 10 HIV-infected children immunized with MMR [33]. Cultures of plasma, peripheral blood mononuclear cells, and polymophonuclear leukocytes obtained 1–130 months after immunization were negative for measles, mumps, and rubella viruses.

The first documented serious adverse event following measles immunization in an HIV-infected person was reported by the CDC in 1996, when a 20-year-old man with hemophilia A and HIV infection died 10 months after receiving his second dose of measles vaccine [62, 63]. He had a very low CD4+ T lymphocyte count but no HIV-related symptoms and was not receiving antiretroviral therapy at the time of vaccination. Ten months after immunization, he developed cough and pulmonary infiltrates. An open lung biopsy showed giant cell pneumonitis, and culture yielded measles virus. He was treated with ribavirin but died 15 months after immunization. Complete sequencing of the isolate showed a difference of only two nucleotides, each encoding an amino acid change, between it and the Moraten strain of measles vaccine virus [63]. Recommendations for measles immunization of severely immunocompromised persons with HIV infection were altered after this report [64]. Measles vaccine is no longer recommended for HIV-infected children or adults with severe immunosuppression, defined by age-specific CD4+ T lymphocyte counts.

Effect of measles and measles immunization on progression of HIV infection. Few studies have examined the effect of measles or measles immunization on the progression of HIV infection and the potential for enhanced immune dysregulation. In a study of HIV-infected children in New York City, the level of circulating HIV p24 antigen increased following measles immunization in three children with preexisting p24 antigen but not in eight HIV-infected children after measles immunization [9]. The effect of measles and rubella vaccination on lymphocyte subpopulations was studied among 39 HIV-infected adults and 17 hospital workers who served as controls [48, 65]. The percentage of CD8+ T lymphocytes decreased in both groups 3 weeks after immunization, but the percentage of CD4+ T lymphocytes did not change [65]. HIV p24 antigen was detected in three persons before and after measles immunization but was undetectable in the remaining vaccine recipients [48]. Measles virus infection, but not measles immunization, can result in the production of antibodies that cross-react

#### Table 3. Seroprevalence of antibody to measles virus in HIV-infected adults in the United States.

<table>
<thead>
<tr>
<th>Reference</th>
<th>Year</th>
<th>Study population</th>
<th>No. of adults</th>
<th>Mean age, y (range)</th>
<th>Mean CD4+ cell count/mm² (range)</th>
<th>Measles seroprevalence (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>[50]</td>
<td>1991</td>
<td>Outpatients</td>
<td>105</td>
<td>36 (21–59)</td>
<td>436 (76–1,137)</td>
<td>99</td>
</tr>
<tr>
<td>[51]</td>
<td>1992</td>
<td>Outpatients</td>
<td>262</td>
<td>35 (18–73)</td>
<td>374 (4–1,462)</td>
<td>95</td>
</tr>
<tr>
<td>[52, 53]</td>
<td>1992</td>
<td>Prison inmates</td>
<td>51</td>
<td>. . .</td>
<td>. . .</td>
<td>88</td>
</tr>
<tr>
<td>[54]</td>
<td>1992</td>
<td>Outpatients</td>
<td>34</td>
<td>25 (18–35)*</td>
<td>636 (317–1,040)*</td>
<td>79</td>
</tr>
<tr>
<td>[48]</td>
<td>1993</td>
<td>Prison inmates</td>
<td>39</td>
<td>(20–41)</td>
<td>&gt;200 in 97%</td>
<td>92</td>
</tr>
<tr>
<td>[49]</td>
<td>1994</td>
<td>U.S. Navy and Marine Corps personnel</td>
<td>210</td>
<td>(18–45)</td>
<td>&gt;200 in 75%</td>
<td>95</td>
</tr>
<tr>
<td>[34]</td>
<td>1995</td>
<td>Outpatients</td>
<td>25</td>
<td>36</td>
<td>. . .</td>
<td>96</td>
</tr>
<tr>
<td>[56]</td>
<td>1998</td>
<td>Outpatients</td>
<td>619</td>
<td>35 (18–74)</td>
<td>280</td>
<td>90</td>
</tr>
</tbody>
</table>

NOTE. Antibody was assayed by EIA.
* Information provided for 11 women lacking measles antibody.
with HIV-1 antigens, visible as one or more bands on immunoblots [66].

Conclusion

The global HIV epidemic has important implications for control and eradication of measles, although much remains to be learned about the clinical, epidemiological, and immunologic interactions of measles virus and HIV. HIV-infected persons with measles are likely to have uncharacteristic clinical findings and severe illness, with high rates of pneumonitis and death. Delayed or absent rash may lead to misdiagnosis and nosocomial transmission of measles virus. High rates of primary and secondary failure of measles vaccine in HIV-infected children may permit transmission of measles virus in spite of high coverage rates of immunization. Two factors that complicate measles-control efforts in areas of high HIV prevalence are the potential for fatal infection with measles vaccine virus and acceleration of HIV disease progression following measles immunization. Global eradication of measles is a worthy goal; however, further research on the impact of the HIV epidemic on measles and measles immunization is necessary to guide strategies for the eradication of measles.

References


