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Contents:

VIRUS REPORTING SCHEME: A total of 1657 reports were processed for this period.

Five cases of Q fever were reported, 3 from New South Wales, 1 from South Australia and 1 from Queensland. No occupational exposure data was available for the reported cases. However none of the five patients was involved in the Q fever vaccine field trial conducted in South Australia.

Cytomegalovirus (CMV) was isolated from :-

- the leucocytes of a 30 year old, HIV antibody positive male with CMV retinitis. The patient was treated with DHPG (ganciclovir) and AZT (azidothymidine).
- the saliva of two HIV antibody positive males, a 40 year old with CMV colitis and a 44 year old patient with CMV retinitis who was treated with DHPG.
- the urine of two HIV antibody positive males, a 56 year old who presented with abdominal pain, and a 32 year old patient with Pneumocystis carinii pneumonia.
- the bronchial washings of two HIV antibody male patients, with P. carinii pneumonia, aged 30 and 32 respectively.
- the cervical swab of a 22 year old female intravenous drug abuser, in her eighth week of pregnancy, who had had a cervical lesion for one week.

Herpes simplex virus type 1 was isolated from the oesophageal biopsies of two males, aged 19 and 30 respectively who had immunosuppression and progressive oesophageal ulceration.

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One hundred and nineteen cases of rotavirus associated diarrhoea have been reported from this period. The demographic characteristics of these cases are detailed below:-

	SEX			
AGE	Male	Female	Unknown	TOTAL
0-1	37	42	-	79
2-5	11	14	-	25
6-10	-	-	-	-
Adults	3	-	-	3
unknown	8	3	1	12
TOTAL	59	59	1	119

Rotavirus infections are expected to predominate during the winter months with an incubation period of 2-4 days. Symptomatic infections, including diarrhoea, fever, abdominal pain and vomiting, leading to dehydration are most commonly seen in children between ages 6 months to 10 years, and transmission appears to be by faecal-oral route. Nocosomial infections are also frequent.

AIDS - WHO MEETING

(based on WER NO. 30, 24 July 1987)

At the third meeting of the WHO Collaborating Centres on AIDS, held in Washington on 6 June 1987 in conjunction with the Third International Conference on AIDS, technical issues of international concern were discussed, including questions regarding HIV infection and the possible revision of the Centers for Disease Control (CDC)/WHO AIDS case definition. A consensus was reached regarding:

A. transmission of HIV,
B. HIV infection and health workers, and
C. the present status and future developments in laboratory testing.

Further evaluations of the CDC/WHO AIDS case definition and the case definition based on clinical criteria currently in use are required before a decision can be taken regarding a revision of the case definitions to be recommended by WHO.

A. Transmission of HIV

Epidemiological studies in Europe, the Americas, Africa and Australia repeatedly have documented only 3 modes of HIV transmission:

1. sexual intercourse (heterosexual or homosexual);

2. contact with blood, blood products, or donated organs and semen; the vast majority of contacts with blood involve transfusion of unscreened blood or the use of unsterilized syringes and needles by IV drug abusers or in other settings;

3. mother to child-mostly before, and perhaps during or shortly after birth (perinatal transmission).

There is no evidence to suggest that HIV can be transmitted by the respiratory or enteric routes or by casual, person-to-person, contact in any setting, including household, social, work, school or prison settings.

Epidemiological and laboratory studies have established that of the "body fluids", transmission seems limited to blood, semen, and vaginal/cervical secretions. Kissing has not been documented to pose a risk of HIV transmission. While unproven, some theoretical risk from vigorous "wet" kissing (deep kissing or tongue kissing) may exist.

There is no evidence to suggest that HIV transmission involves insects, food, water, toilets, swimming pools, sweat, tears, shared eating and drinking utensils or other items such as second-hand clothing or telephones.

B. HIV Infection and Health Care Workers

Reports of HIV infection of a small number of health workers have emphasized the need to adhere to existing guidelines for the prevention of blood-borne infections. Such existing guidelines refer to situations in which there is a possibility of exposure to blood or any body fluid regardless of their source.

Available information indicates that health workers are normally at very low occupational risk of HIV infection. This very low risk can be further minimised if existing guidelines for avoiding any blood-borne infection are rigorously implemented and strictly enforced.

Routine HIV screening of patients to protect health workers should not be implemented without careful and detailed consideration of all of the HIV screening criteria developed by the World Health Organization.

C. Present status and future developments in laboratory testing for HIV

1. INTRODUCTION

The following types of tests are available or under development:

- measurement of antibodies against viral antigens;
- measurement of neutralising antibodies;
- detection of viral antigens;
- detection of viral RNA or cDNA;
- virus isolation and characterisation of virus isolates from various geographical regions.

2. MEASUREMENT OF ANTIBODIES AGAINST VIRAL ANTIGENS (ANTI-HIV)

Determination of anti-HIV should consist of a primary screening test to be followed by confirmation with a second supplemental assay based on a different test principle. Current antigen-antibody binding assays have a high degree of specificity and sensitivity. Second generation tests using recombinant antigens or future use of synthetic peptides promise to improve sensitivity and particularly specificity. Generally these test systems measure antibodies of the IgG class, but test systems measuring specific IgA and IgM antibodies are needed also and should be developed further.

Although more specific ELISA or other antigen-binding assays may in the future make supplemental (confirmatory) tests unnecessary, reactivities indicating presence of anti-HIV obtained with any of the currently available screening tests should be confirmed by another test method. Western-blots (immunoblots) are the most widely used and reliable tests, but radioimmunoprecipitation (RIPA) or immunofluorescence may be used. The latter should, however, only be used by laboratories with extensive experience with this test system.

Test systems should be developed which detect antibodies to HIV1 and HIV2 either together in one test or individually. The antigenic specificities of HIV isolates from different parts of the world should be continuously characterised to assure that the diagnostic method covers the antigens of the viruses prevalent in a given region. Simplified, less expensive tests should be developed further. These test systems should have at least the same sensitivity as currently used test systems, but a slight decrease in specificity might be acceptable.

3. MEASUREMENT OF NEUTRALISING ANTIBODIES

Neutralisation tests are used for research purposes and for evaluation of antibody responses following vaccination. The biological relevance of the antibodies measured by the various test systems needs further study and all test systems must be standardised, so that results obtained in different laboratories can be compared.

4. DETECTION OF VIRAL ANTIGENS

The tests available today need further clinical and technical evaluation. They are not recommended for routine diagnosis or screening of blood donors. Increase of HIV p24 antigen in serum has been associated with progression of disease but this does not occur in all cases. Decrease of HIV p24 in serum has been taken as an indication of a decrease of HIV replication and is used for evaluation of the effectiveness of antiviral therapy. These preliminary observations require additional studies. Absence of detectable antigen does not guarantee lack of infectiousness of a given serum, semen, body fluid or organ.

5. DETECTION OF VIRAL RNA OR CDNA

Methods for detection of viral RNA or cDNA in routine diagnostic laboratories are under development and may offer the most sensitive test systems for direct demonstration of HIV in fluids or tissues.

6. VIRUS ISOLATION AND CHARACTERISATION OF VIRUS ISOLATES FROM VARIOUS GEOGRAPHICAL REGIONS

Techniques are still cumbersome and time-consuming but have been considerably improved, so that an almost 100% isolation rate can be achieved if multiple blood samples are examined. An optimised standard protocol should be worked out and made available to laboratories using this technique for basic or clinical studies. Virus isolates should be characterised to monitor the emergence of variant or new antigenic types.

7. STANDARDISATION AND REFERENCE REAGENTS

All of the above-mentioned test systems need further standardisation. International antibody units should be established and appropriate reference reagents (both antigens and antibodies) should be prepared. The WHO Collaborating Centres on AIDS should play an active role in the preparation and evaluation of these reference reagents and WHO standards should eventually be established. WHO should also establish a repository of HIV1 and HIV2 as well as SIV isolates. In addition it would be desirable to prepare a list of available clones of human and simian retroviruses.

8. HTLV-I AND HTLV-II

The prevalence of HTLV-I and HTLV-II in various population groups should be monitored, but there seems to be no current need for general screening of blood or organ donors for HTLV-I and HTLV-II.

AIDS - NOT TRANSMITTED BY INSECTS

Detailed study of AIDS patients not belonging to recognised high risk groups have not revealed the existence of new modes of HIV transmission such as casual contact; insect bites; or food borne, waterborne, or environmental spread. Concerns persist about the vector transmission of AIDS. However currently available epidemiological and experimental data argue against such a proposal.

Epidemiological Data

Currently available data do not indicate that the AIDS virus is transmitted by insects.

- In Africa, where the disease afflicts men and women almost equally, AIDS remains a disease of the sexually active.

- In the United States, the disease continues to afflict traditional risk groups: homosexuals and intravenous drug users.

  - Epidemiologic analysis of AIDS data in the US indicates that the situation is clearer in the five to 15 year old age group, which lies between the youngest children for whom perinatal transmission is the most important and the adult age groups where sexual and drug related

transmission predominate. Five to 15 year olds, who include the majority of school children, comprise 16% of the US population. However only 62 AIDS cases (0.2% of total cases) have occurred in this large group, which is exposed like other groups to casual contact with HIV-infected persons, insects, and environmental factors. Of these, 61 (98%) fit into established risk categories; the risk factor investigation is incomplete on the remaining case(1). If insects were a factor, significant numbers of children would also be infected with the virus; but they are not, even though the children get their fair share of insect bites.

- Despite claims by two activists in North Miami who maintain that environmental factors contributed greatly to the spread of AIDS, to explain the unusually high rate of AIDS infection in the town of Belle Glade, Florida, where many residents live in crowded, squalid conditions - (where approximately 100 insect bites a day are not unusual) - the Centers for Disease Control (CDC, Atlanta, USA) has in 1986 concluded that sexual contact and shared needles are responsible for the high incidence of AIDS in Belle Glade, not swarms of mosquitoes(2).

Experimental Data

There are two ways in which a blood-sucking insect can spread disease: biologically and mechanically.

. Biological transmission occurs when an insect ingests virus-infected blood, the virus replicates inside the host, eventually finding its way to the insect's salivary glands. Saliva may then be secreted by insects during feeding to keep blood from coagulating. Unlike some particularly virulent viruses - yellow fever, dengue, and encephalitis - there is no indication to suggest that the AIDS virus replicates inside insects, and thus biological transmission of AIDS is impossible.

  - Experimental studies conducted at CDC (Atlanta, USA) could detect no evidence of viral replication in the cells from ticks, moths and fruitflies infected with genetic material from HIV isolated from an African AIDS patient(3).

  - Studies conducted at the Institut Pasteur (Paris) using nucleic acid probes, detected HIV-1 related sequences in a number of insects collected in Zaire and the Central African Republic. The genetic material is found in cells in tissue throughout the insect's bodies, in male mosquitoes as well as females (although male mosquitoes do not feed on blood), and even in insects that are not blood-sucking species such as cockroaches and antlions. Although no explanation was given to account for the presence of the genetic sequences, no free virus, no HIV-related proteins or RNA can be found in the insects, indicating that the virus is not replicating in the insects(4).

. The possibility of mechanical transmission by bedbugs, mosquitoes or other insects is more difficult to dismiss,

given that mechanical transmission of the virus could theoretically occur if a mosquito, for instance, was interrupted while feeding on an infected host, then flew to another person and injected a miniscule portion of tainted blood. The popular question remains that if one can get AIDS from sharing needles, why not from mosquitoes which are likened to small needles. The answer is volume, a realistic biological quantity:

The proboscis of a mosquito or the mouthparts of a bedbud apparently do not hold enough residual blood to give an infectious dose. A mathematical model assuming a person infected with AIDS virus had a concentration of 1000 units of free virus per mL of blood, estimates that a mosquito interrupted during feeding on this host could inoculate a second person with approximately 1×10^{-10} mL of infected blood. Thus the chances of inoculating a single unit of virus would be 1×10^{-7} (ie 1 in 10 million) - too remote for a possibility.

Expert scientific opinions expressed at a recent workshop held by the office of Technology Assessment (USA) in July this year have concluded that no further research on AIDS transmission by insects is warranted at this stage since there is:

- no evidence that the virus replicates in insects,
- no evidence that it is transmitted mechanically, and
- no evidence that it is transmitted biologically[5].

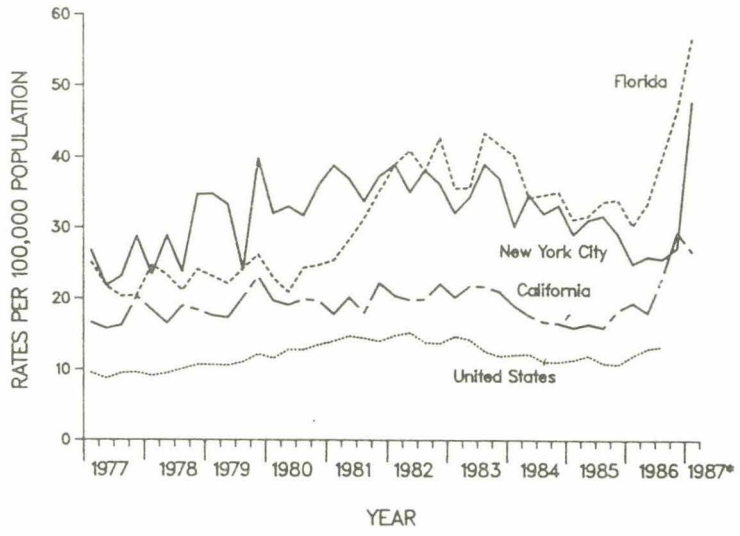
REFERENCES

1. MMWR (1986) 35 (49): 757-66
2. SCIENCE (1986) 24 Oct: 415
3. Lancet (1987) i:1094-5
4. AIDS International Conference - Abstracts MP. 37
5. Science (1987) 237:355

INCREASES IN PRIMARY AND SECONDARY SYPHILIS - UNITED STATES
(based on MMWR Vol.36/No.25, 3 July 1987)

After a five-year trend of decreasing incidence of primary and secondary syphilis in the United States, 8 274 cases were reported during the first quarter of 1986. The estimated annual rate per 100,000 population rose from 10.9 cases to 13.3 cases (Figure 1). An increase of this magnitude has not been observed in over ten years.

Figure 1: Incidence rates of primary and secondary syphilis, by quarter - United States and selected areas, 1977 - 1st quarter 1987.



*1987 data are estimated.

Increases of 20 or more cases over the number reported during the first quarter of 1986 were observed in eight states, four major metropolitan areas, and the Commonwealth of Puerto Rico (Table 1).

Table 1: States and metropolitan areas reporting increases of ≥ 20 cases of primary and secondary syphilis - United States, 1st quarter 1986 and 1st quarter 1987

Reporting area	Number of cases		
	1st quarter 1986	1st quarter 1987	Increase
California			
. Los Angeles	508	970	91%
. Other	784	847	8%
. Total	1 292	1 817	41%
Florida	877	1 679	92%
New York			
. New York City	510	875	72%
. Other	55	90	64%
. Total	565	965	71%
Georgia	383	417	9%
C'wlth of Puerto Rico	207	229	11%
Pensylvania			
. Philadelphia	118	187	58%
. Other	33	38	15%
. Total	151	225	49%

Table 1: Cont'd States and metropolitan areas reporting increases of ⩾ 20 cases of primary and secondary syphilis - United States, 1st quarter 1986 and 1st quarter 1987

Reporting area	Number of cases		
	1st quarter 1986	1st quarter 1987	Increase
Mississippi	131	162	24%
Maryland			
. Baltimore	56	95	70%
. Other	40	53	33%
. Total	96	148	54%
Arizona	63	95	51%
Oregon	27	48	78%
Nevada	20	41	105%

The three areas reporting the largest increases were:

. California where increases of 10 or more cases occurred in Los Angeles, Long Beach, and seven smaller counties (San Francisco continued a 5-year trend of decreasing incidence),
. Florida where ten counties experienced increases, the largest being in Dade, Orange, and Palm Beach counties,
. New York City where all boroughs except Richmond experienced substantial increases.

All three areas with the largest increases had collected demographic data and information on the sexual preferences of patients with cases reported during the period January-March 1986 and January-March 1987 (Table 2).

. In California and New York City, increases in primary and secondary syphilis occurred exclusively among heterosexuals. In addition, blacks experiences greater increases than whites in these two areas.
. In Florida, the increase occurred in each demographic group and in each group with similar sexual preferences.

The ratio of cases among males to cases among females in the three areas fell from 2.6:1 to 2.1:1. For several other areas experiencing increase in total cases, the incidence declined for white men citing at least one male sexual partner.

Table 2: Cases of primary and secondary syphilis by patient characteristics - United States, 1st quarter 1986 and 1st quarter 1987

Reporting area/Patient characteristic*	Number of cases 1st quarter 1986	1st quarter 1987	Increase
Florida			
. Sex - male	565	1078	91%
- female	314	595	90%
. Race - black	694	1331	92%
- white and other	185	342	85%
. Sexual preference (male)+			
- heterosexual	347	521	50%
- homo-/bi-sexual	38	79	108%
California			
. Sex - male	953	1296	36%
. - female	260	507	95%
. Race - black	368	849	131%
. - white and other	845	954	13%
. Sexual preference (male)+			
- heterosexual	643	1130	76%
- homo-/bi-sexual	277	148	-47%
New York City			
. Sex - male	349	585	68%
. - female	158	290	84%
. Race - black	246	475	93%
- white and other	261	400	53%
. Sexual preference (male)+			
- heterosexual	125	250	100%
- homo/bi-sexual	45	22	-51%

* Demographic data were available for 99% of patients with reported cases.

\+ Excludes men whose sexual preferences was not determined;

. 2% of men with syphilis in California,
. 40% of men with syphilis in Florida, and
. 53% of men with syphilis in New York City.

MMWR Editorial Note

Although primary and secondary syphilis had been declining since 1982[1], they now appear to be on the upsurge in some areas. While 70% of cases among males occurred among homosexual and bisexual men during the 1970s[2], cases among these groups now appear to be on the decline in some areas (Table 2). As with two smaller outbreaks in the 1980s[3, 4], the current increase appears to be largely among heterosexuals.

The increases in primary and secondary syphilis have prompted two major concerns:

1. This trend is likely to have a severe adverse effect on efforts to control congenital syphilis. While sexually acquired syphilis that is diagnosed in its early stages can be effectively treated with long-acting penicillin preparations, congenitally acquired syphilis is responsible for high rates of infant morbidity and mortality[5]. After an 8-year decline, the incidence of congenital syphilis among infants began rising in 1983[6]. The areas with the largest increase in primary and secondary syphilis already have some of the highest rates of congenital syphilis in the nation[6].

   Any increases in acquired syphilis among heterosexual adults in these areas are certain to be followed by further increases in congenital syphilis.

2. A history of sexually transmitted disease is associated with increased risk for human immunodeficiency virus (HIV) infection and acquired immunodeficiency syndrome (AIDS) among both homosexuals[7, 8] and heterosexuals[9]. New York City and Florida have increased incidences of syphilis as well as high rates of AIDS among heterosexuals, particularly among those who abuse intravenous drugs[10]. Because genital ulceration is associated with higher rates of HIV infection[11, 12], the increases in primary and secondary syphilis in these areas may be the forerunner of case reports of treatment failures[13] and an atypical course in one patient[14], and response to treatment of syphilis. These reports suggest the potential for problems in the management of patients with both infections.

CDI Editorial Comment:

An epidemiologic analysis of primary and secondary syphilis in the three areas described above, reveals that attack rates are disproportionately high for blacks in general. Should these data be extrapolated to the overall cumulative incidences of treponemal infections in the United States, the situation resembles that of AIDS where cumulative incidences of AIDS among blacks and Hispanics are over three times the rate for whites[14].

Several factors may contribute to the elevated incidence of AIDS and HIV infection among these racial/ethnic groups. The racial/ethnic distribution of AIDS cases may reflect, to some degree, the racial/ethnic distribution of the populations at risk in the high-prevalence areas. Persons at risk become so as a result of underlying risk factors, not because of their race/ethnicity. Despite the recommendations to intensify the search for factors to explain the increased susceptibility of blacks to HIV infection, established risk factors (needle sharing, promiscuity, and receptive anal/genital contact) failed to account for significant differences in human immunodeficiency virus (HIV) seroprevalence between black and white patients in a cohort of homo-/bi-sexual men[15].

It is currently proposed that syphilis may qualify as an important factor[16] since there is substantial indirect evidence to support the hypothesis that syphilis lesions facilitate transmission of HIV[17]. Several studies have noted statistically significant differences in the proportion of HIV-infected patients bearing markers of treponemal infection, compared with their uninfected counterparts[17-19].

The strength and consistency of this association, commonly thought to reflect the degree of promiscuous behaviour by individuals who develop AIDS[19], more plausibly suggest a facilitating role for syphilis in the transmission of HIV. The idea is intuitively appealing. Loss of epithelial integrity, frequently observed in syphilis, can provide the entry portal apparently needed by HIV.

The independent association of syphilis with HIV infection, has also been evaluated:

- in the San Francisco men's health study where a higher prevalence of HIV was recorded among homo-/bi-sexual blacks (66%) than whites (49%). With respect to the occurrence of syphilis in the study population, indeed a higher percentage of blacks than white (38% vs 28%) gave a history of syphilis. However, syphilis was approximately equally reported by HIV-infected blacks and whites (42% vs 41%)[20].

- by performing a logistic regression analysis that included the following variables: syphilis, race (black or white), and the most important established risk factors for HIV infection (member of male sexual partners in the previous two years, number of partners with whom anal receptive intercourse had been carried out in the previous two years, needle sharing in the previous five years, and frequency of douching before anal intercourse)[21].

  The results indicated significant independent association between both:
  . syphilis and HIV infection (odds ratio = 2.64; P .001), and
  . race and HIV infection (odds ratio = 2.61; p = .05)

  The interaction term for syphilis and race was not significant (P = .31).

The prevailing hypothesis that syphilis is associated with HIV infection is supported by these data, but other factors must also contribute to the differences in HIV infection rates between blacks and whites. This hypothesis, although contributing to the understanding of HIV transmission by conventional (ie penile-vaginal) intercourse, can only partly account for the observations that blacks have the highest rates of early syphilis and that blacks have the highest rates of heterosexual AIDS cases[22] may be closely linked; blacks make up only 12% of the United States population, yet account for an astonishing 73% of all AIDS cases classified as heterosexually transmitted[22].

REFERENCES

1. MMWR (1986) 33 (54):57-9
2. Sex Transm Dis (1983) 10:77-80

3. MMWR (1987) 36:221-4
4. Am J Epidemiol (1987) 125: 277-83
5. Sexually Transmitted Diseases. New York: McGraw Hill Co, (1984): 352
6. MMWR (1986) 35:625-9
7. Ann Intern Med (1983) 99:145-51
8. Am J Epidemiol (1987) 125:1035-47
9. International Conference on AIDS, April 14-17, (1985):45
10. MMWR (1986) 35:757-60, 765-6
11. International Conference on AIDS, June 1-5 (1987). Abstract MP91:25
12. International Conference on AIDS, June 1-5 (1987). Abstract Th P68:174
13. N Engl J Med (1987) 316:1587-9
14. MMWR (1986) 35(42): 655-666
15. JAMA (1987) 257:1901-02
16. JAMA (1987) 258:473-74
17. N Engl J Med (1986) 314:414-18
18. Genitourin Med (1986) 62:384-89
19. JAMA (1987) 257:635-639
20. JAMA (1987) 257:321-325
21. JAMA (1987) 258:473-474
22. AIDS Weekly Surveillance Rep, March 30, 1987.

TERTIARY SYPHILIS DEATHS - SOUTH FLORIDA (USA)

(based on MMWR vol. 36/No. 29, 31 July 1987)

From January 1984 through July 1986, the Centers for Disease Control (CDC) received reports from three counties in South Florida of 18 persons considered to have evidence of tertiary syphilis at autopsy. Based on histologic review at CDC:

- 8 had evidence strongly suggestive of syphilis aortitis,
- 3 showed cerebral chronic perivascular inflammation consistent with central nervous syphilitic involvement, and
- 7 were not confirmed on histologic review.

Of the 11 cases consistent with tertiary syphilis:

- 9 were reported by the medical examiners of Broward County, (the overall proportion of tertiary syphilis among persons autopsied by the medical examiners was 4 per 1,000),
- 1 was reported by the medical examiner of Collier County, and
- 1 was reported by a pathologist in Dade County.

The 11 decedents with evidence of tertiary syphilis ranged from 32 to 69 years of age at the time of death:

- sex: 2 males and 9 females
- race: 6 white and 5 of other races
- 7 of the 11 decedents had reactive post-mortem microhaemagglutination - Treponema pallidum (MHATP) serologic tests, and

- 4 had positive post-portem ELISA and Western blot tests for antibody to the human immunodeficiency virus (HIV), (No post-mortem blood was tested for one of the decedents).

To determine which factors may have been associated with evidence of tertiary syphilis at autopsy, a case-control study was performed. Data on the 11 reported decedents were compared with data on 29 autopsied decedents with positive post-mortem MHATP tests but no evidence of tertiary syphilis (TS) (Table 1).

Table 1. Risk factors for tertiary syphilis (TS) evaluated in a case-control study - South Florida, 1984-1986.

Risk Factors	Decedents: Evidence of TS (n=11)	Decedents: No Evidence of TS (N-29)	Odds Ratio	95% Conf. Int.	Fisher's Exact Test
HIV antibody					
. positive	4+	10	1.3	(0.2, 6.9)	p=1.0
. negative	6	19			
Age (years)					
. 50	6	11	2.0	(0.4, 10.2)	p=0.5
. 50	5	18			
Race					
. White	6	17	1.2	(0.2, 7.4)	p=1.0
. non-white	5	12			
*Sex					
. female	9	8	11.8		
. male	2	21			
Drug abuse					
. evidence	1	2	1.4	(0.02, 28.5)	p=1.0
. no evidence	10	27			

\+ one case omitted due to unavailability of post-mortem blood for analysis.

* sex, as a risk factor, has been re-analysed because of error detected in the original article. Although odds ratio (OR=11.8) has been calculated, no value was available for the 95% confidence interval limits and no p value was available for the 2 tailed Fisher's exact test.

The names of persons in both groups were cross-referenced with the state syphilis registry; only:

. 3 with tertiary syphilis, and
. 2 in the control group

were known to have received treatment in Florida with late syphilis (late latent in two and cardiovascular syphilis in one - these three also had HIV infection).

MMWR Editorial Note

This study does not support the hypothesis that HIV infection modifies syphilis infection[1], as it appears to modify clinical manifestations of tuberculosis[2]. While severe manifestations of late syphilis in persons with HIV infection have been observed previously [3, 4], such manifestations have also been observed among other persons[5]. Moreover, while iatrogenic and other non-HIV-related causes of immunosuppression often reactivate tuberculosis[6], rapid progression to and early mortality from tertiary syphilis have not been demonstrated in similar clinical circumstances. Animal experimentation and anecdotal case reports, however, suggest that suppression of cell-mediated immunity may result in an unusual distribution of syphilitic lesions[7] and possibly other unusual manifestations of syphilis[1,4].

A history of syphilis infection is common among persons with HIV infection. Homosexual men with AIDS have been shown to be significantly more likely to have a history of syphilis than are homosexual men without AIDS[8]. This association has been interpreted to reflect behaviours that are likely to expose patients to HIV infections[9], although excess risk independent of such behaviours has been reported[8]. Since these infections are common in the same populations, evidence of both at death, as found in the study presented here, would be expected to be a common event.

It is not unusual, particularly among persons autopsied by medical examiners and even in areas with a low prevalence of syphilis, to find evidence of tertiary syphilis at autopsy despite its being unsuspected during the decedents life[10]. In one study, 1% of a series of decedents autopsied by Danish medical examiners had evidence of active syphilitic aortitis[10]. Cardiovascular syphilis diagnosed on autopsy may occur among relatively young persons (in two series of autopsied, the mean ages were 36[11] and 52[12]. However, as appreciated in the pre-antibiotic era[12] and noted in this series, the diagnosis may be difficult to confirm.

The possibility that penicillin treatment for syphilis may have failed in 2 HIV-seropositive patients during latency is disturbing. Failures of penicillin treatment to arrest syphilis infection are considered rare in early disease, though such failures have been reported[4,13]. They have also been reported in treatment of late infection[14], when treatment failure is probably more common. Studies are currently being conducted to:

1. identify risk factors associated with treatment failure of syphilis to prevent or effectively treat tertiary syphilis, and
2. evaluate the clinical and serologic responses to treatment for syphilis of persons with HIV infection.

Physicians who have diagnosed central nervous system, cardiovascular, or other unusual manifestations of syphilis in persons less than 55 years of age are encouraged to report these findings through their state and local health departments to the Division of Sexually Transmitted Diseases, Center for Prevention Services, CDC. Pathologists diagnosing tertiary syphilis on autopsy are also encouraged to report such cases.

CDI Editorial Comment

Based on the original Table, the case-control study concluded that the two groups were not significantly different in terms of age, race, sex, or intravenous drug use. HIV infection was not significantly associated with tertiary syphilis - with four of the decedents with tertiary syphilis and 10 of those in the comparison group had serologic evidence of HIV infection confirmed by Western blot (odds ratio [OR]=1.3, 95% confidence interval = 0.2-6.9).

Revision of data by the CDI bulletin, in relation to the sex distribution of cases, seems to indicate that the two groups differ in terms of sex; with females being at much higher risk for tertiary syphilis than males (odds ratio [OR] = 11.8). Also, although other variables studied such as HIV antibody status, age and race may not qualify as risk factors, drug abuse, as a variable cannot be adequately measured in such a case-control study involving post-mortem examinations.

Johns et al[1] have suggested that not only may HIV infection impair immune defences and facilitate the progression of syphilis, but also suggests that syphilitic immunosuppression may impair host defences against HIV. The present case-control study of autopsied persons produces results of little relevance to this debate.

REFERENCES

1. NEJM (1987) 316: 1569-72
2. Chest (1986) 90: 469-70
3. Ann Opthalmol (1986) 18: 260-1
4. NEJM (1987) 316: 1587-9
5. Can J Psychiatry (1981) 26: 68-70
6. Br J Dis Chest (1976) 70: 195-205
7. Arch Immunol Ther Exp (1979) 27: 45-51
8. Am J Epidemiol (1987) 125: 1035-47
9. Am J Public Health (1984) 74: 259-60
10. Forensic Sci Int (1980) 15: 3-17
11. Indian Heart J (1967) 19: 86-95
12. US Public Health Service, CDC, (1950)
13. G. Arch Neurol (1986) 43:137-8
14. Genitourin Med (1986) 62: 129-31

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

REPORTING PERIOD - 27-7-87 to 9-8-87 BULLETIN NUMBER 87/16
VIRAL IDENTIFICATIONS FROM CONTRIBUTING LABORATORIES

VIRUS OR VIRAL ANTIGEN	ICPMR (NSW)/ WVH (ACT)	RAHC (NSW)	PHH/ POW (NSW)	FAIR-FIELD (VIC)	RCH (VIC)	IMVS (SA)	STATE LAB (QLD)	STATE LAB (WA)	Total
0100 ADENOVIRUS NOT TYPED	6	4	2		6	4	17	1	40
0101 ADENOVIRUS TYPE 1						2			2
0102 ADENOVIRUS TYPE 2				3	2				5
0103 ADENOVIRUS TYPE 3			1		4	1			6
0104 ADENOVIRUS TYPE 4				1					1
0105 ADENOVIRUS TYPE 5						1		2	3
0130 ADENOVIRUS TYPE 30				1					1
0199 ADENOVIRUS TYPING PENDING		1	1						2
0201 INFLUENZA A VIRUS	4			3					7
0202 INFLUENZA A VIRUS SUBTYPE H3N2						1			1
0203 INFLUENZA B VIRUS	1			1	8	2			12
0301 PARAINFLUENZA VIRUS TYPE 1						6			6
0302 PARAINFLUENZA VIRUS TYPE 2							3		3
0303 PARAINFLUENZA VIRUS TYPE 3				2	2	9	6	1	20
0400 RESPIRATORY SYNCYTIAL VIRUS (RS)	36	18	10	27	38	46	43	69	287
0500 RHINOVIRUS (ALL TYPES)	1		1	4	17	3	1		27
0600 MYCOPLASMA PNEUMONIAE	18		8	6	1	4		9	46
0700 ORNITHOSIS-PSITTACOSIS	1			1					2
0816 COXSACKIEVIRUS A16	2								2
0901 COXSACKIEVIRUS B1								2	2
0902 COXSACKIEVIRUS B2				1					1
0903 COXSACKIEVIRUS B3	1			3	7				11
0904 COXSACKIEVIRUS B4						1			1
1003 ECHOVIRUS TYPE 3		1							1
1005 ECHOVIRUS TYPE 5				1					1
1009 ECHOVIRUS TYPE 9			1						1
1011 ECHOVIRUS TYPE 11				3					3
1018 ECHOVIRUS TYPE 18				2	2				4
1020 ECHOVIRUS TYPE 20				1		1			2
1100 POLIOVIRUS NOT TYPED			1		3		1		5
1101 POLIOVIRUS TYPE 1	1					2			3
1102 POLIOVIRUS TYPE 2								1	1
1103 POLIOVIRUS TYPE 3	1								1
1200 MUMPS VIRUS			1						1
1300 HERPES VIRUS GROUP-NOT TYPED	17		2					1	20
1301 HERPES SIMPLEX VIRUS NOT-TYPED		2					1	2	5
1302 EPSTEIN-BARR VIRUS (EB VIRUS)	16		1	2		1		6	26
1303 VARICELLA-ZOSTER VIRUS	3	1	4	4		1	1	6	20
1306 HERPES SIMPLEX TYPE 1	34	1		49	2	37	40	18	181
1307 HERPES SIMPLEX TYPE 2	71			52		4	63	40	230
1401 COXIELLA BURNETI	3					1	1		5
1502 PICORNA VIRUS-NOT TYPED	6		12				8	1	27
1515 CONTAGIOUS PUSTULAR DERMATITIS (ORF VIRUS)			1						1
1521 MEASLES VIRUS		1		3	1			1	6
1522 RUBELLA VIRUS	4	1		4		2			11
1532 HEPATITIS B ANTIGEN	95	6	3	33		22	33	22	214
1535 HEPATITIS A ANTIBODY	7	2		4		6	3	1	23
1541 CHLAMYDIA A - C TRACHOMATIS	44		3	19	1	44	13	45	169
1556 CMV - CYTOMEGALOVIRUS	9	1	8	29	7	4	3	19	80
1564 ROTAVIRUS	19	6	10	3	10	34	25	11	118
1566 NORWALK AGENT				1					1
1599 ENTEROVIRUS TYPING PENDING		1	1		4				6
9990 AUSTRALIAN ENCEPHALITIS								1	1
9992 ROSS RIVER VIRUS				1					1
9994 SMALL VIRUS (LIKE) PARTICLE		1							1
Total	400	47	71	264	115	239	262	259	1,657

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

PERIOD : 27-7-87 to 9-8-87 BULLETIN NO 87/16
Viral Identifications by Clinical Information Table 1.
Code 00,99 -No ill or data; 01,02,11,12 -Respiratory; E3 -Encephalitis; M3 -Meningitis; 04 -Paralysis; 05,13 -CNS other unspec.; 07,49 -GI; 17,47 -Hepatic; 19 -CVS; 89 -Urinary; 06 -Skin/mucous.

VIRUS OR VIRAL ANTIGEN	No-ill or data	Respir -atory	Enceph -alitis	Mening -itis	Para- lysis	CNS other unspec	GI	Hepa -tic	CVS	Urin -ary	Skin/ mucs memb
0101 ADENOVIRUS TYPE 1.....		1									
0102 ADENOVIRUS TYPE 2.....		5					1				
0103 ADENOVIRUS TYPE 3.....		4									2
0105 ADENOVIRUS TYPE 5.....		2									
0130 ADENOVIRUS TYPE 30.....							1				
0201 INFLUENZA A VIRUS.....		4							1		
0202 INFLUENZA A VIRUS SUBTYPE H3N2		1									
0203 INFLUENZA B VIRUS.....		10				1	1				
0301 PARAINFLUENZA VIRUS TYPE 1....	1	5									
0302 PARAINFLUENZA VIRUS TYPE 2....		3									
0303 PARAINFLUENZA VIRUS TYPE 3....	1	19									
0400 RESPIRATORY SYNCYTIAL VIRUS (RS).....	5	279									
0600 MYCOPLASMA PNEUMONIAE.....	10	25				1			1		2
0700 ORNITHOSIS-PSITTACOSIS.....		1									
0816 COXSACKIEVIRUS A16.....											2
0901 COXSACKIEVIRUS B1.....		1					1				
0902 COXSACKIEVIRUS B2.....				1							
0903 COXSACKIEVIRUS B3.....		7		1			2		1		
0904 COXSACKIEVIRUS B4.....		1									
1003 ECHOVIRUS TYPE 3.....		1									
1005 ECHOVIRUS TYPE 5.....		1									
1009 ECHOVIRUS TYPE 9.....				1							
1011 ECHOVIRUS TYPE 11.....				3							
1018 ECHOVIRUS TYPE 18.....			1	2							
1020 ECHOVIRUS TYPE 20.....		1									
1101 POLIOVIRUS TYPE 1.....		1					1				
1103 POLIOVIRUS TYPE 3.....							1				
1301 HERPES SIMPLEX VIRUS NOT-TYPED		1						1			4
1302 EPSTEIN-BARR VIRUS (EB VIRUS).	4	2						2	1		1
1303 VARICELLA-ZOSTER VIRUS.....	7										10
1306 HERPES SIMPLEX TYPE 1.....	9	12					2			2	86
1307 HERPES SIMPLEX TYPE 2.....	9										7
1401 COXIELLA BURNETI.....	1							1			
1502 PICORNA VIRUS-NOT TYPED.....		1									
1515 CONTAGIOUS PUSTULAR DERMATITIS (ORF VIRUS).....											1
1521 MEASLES VIRUS.....		1				1					4
1522 RUBELLA VIRUS.....											9
1532 HEPATITIS B ANTIGEN.....	69							132			1
1535 HEPATITIS A ANTIBODY.....	4							15			
1541 CHLAMYDIA A - C.TRACHOMATIS...	11	1									
1556 CMV - CYTOMEGALOVIRUS.....	7	21	1		1	2	1	1		7	1
1564 ROTAVIRUS.....							118				
1566 NORWALK AGENT.....							1				
9990 AUSTRALIAN ENCEPHALITIS.....			1	1							
9994 SMALL VIRUS (LIKE) PARTICLE...							1				
Total.....	138	411	3	9	1	5	131	152	4	9	196

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

PERIOD : 27-7-87 to 9-8-87 BULLETIN NO 87/16
Viral Identifications by Clinical Information Table 2.
Code 10 -Eye; 59 -Genital; 39 -Endo/sal gland;
38 -RES; 29 -Muscle/joint; 69 -Congenital; P8 -PUO;
G8 -Fever/malaise; 09 -Other; A1 -SIDS ...

VIRUS OR VIRAL ANTIGEN	Eye	Genital	Endo/sal gland	RES	Muscle/joint	Congenital	PUO	Fever/malaise	Other	SIDS
0101 ADENOVIRUS TYPE 1.....										1
0102 ADENOVIRUS TYPE 2.....								1		
0103 ADENOVIRUS TYPE 3.....	1									
0104 ADENOVIRUS TYPE 4.....	1									
0105 ADENOVIRUS TYPE 5.....	1									
0201 INFLUENZA A VIRUS.....								3	1	
0203 INFLUENZA B VIRUS.....								4		
0400 RESPIRATORY SYNCYTIAL VIRUS (RS).....	1		1					1	8	
0600 MYCOPLASMA PNEUMONIAE.....					1		2	3	5	
0700 ORNITHOSIS-PSITTACOSIS.....								2		
0901 COXSACKIEVIRUS B1.....								1		
0902 COXSACKIEVIRUS B2.....								1		
0903 COXSACKIEVIRUS B3.....					1			1		
1003 ECHOVIRUS TYPE 3.....									1	
1018 ECHOVIRUS TYPE 18.....								2		1
1020 ECHOVIRUS TYPE 20.....								1		
1101 POLIOVIRUS TYPE 1.....									1	
1102 POLIOVIRUS TYPE 2.....										1
1302 EPSTEIN-BARR VIRUS (EB VIRUS).		1	5	4				4	6	
1303 VARICELLA-ZOSTER VIRUS.....								2	1	
1306 HERPES SIMPLEX TYPE 1.....	6	60	1				2		3	
1307 HERPES SIMPLEX TYPE 2.....		148								
1401 COXIELLA BURNETI.....							2	1	1	
1521 MEASLES VIRUS.....									1	
1522 RUBELLA VIRUS.....					1				2	
1532 HEPATITIS B ANTIGEN.....						1			11	
1535 HEPATITIS A ANTIBODY.....									4	
1541 CHLAMYDIA A - C.TRACHOMATIS...	3	153							1	
1556 CMV - CYTOMEGALOVIRUS.....	4	7	1			3	2	9	11	1
9992 ROSS RIVER VIRUS.....					1					
Total.....	17	369	8	4	4	4	8	36	57	4

NOTIFIABLE DISEASES REPORTED IN AUSTRALIA

Period 4 - 22 March 1987 to 18 April 1987 Bulletin...87/16...

Disease	N.S.W.	VIC.	QLD.	S.A.	W.A.	TAS.	N.T.	A.C.T.	Total	Cumulative Total to Date for Year
Amoebiasis	1		3	3					7	21
Ankylostomiasis				2	1		NN		3	12
Anthrax									-	1
Arbovirus infection	16		146		6				168	362
Brucellosis			1						1	7
Campylobacter infections	114		NN	88	8	NN	5	NN	215	999
Chancroid				NN	1				1	4
Cholera									-	1
Congenital rubella syndrome			NN			NN		NN	-	-
Diphtheria							4		4	9
Donovanosis			2	NN	3		6		11	32
Giardiasis	26		NN	87	9	NN	NN	NN	122	484
Genital herpes	61		53	20	NN	NN		5	139	666
Gonococcal ophthalmia neonatorum		NN			NN	NN	2	NN	2	3
Gonorrhoea	61		74	57	115	7	150	4	468	1 889
Hepatitis A (infectious)	9	3	4	8	7	2	5		38	210
Hepatitis B (serum)	55	13	57	6	34			2	167	532
Hepatitis – unspecified	1		6	2	NN	NN	2		11	66
Hydatid disease	1								1	2
Lassa fever			NN			NN		NN	-	-
Legionnaires disease	4		NN			NN		NN	4	6
Leprosy									-	5
Leptospirosis	2	3	6			1			12	58
Lymphogranuloma venereum				NN	NN	NN		NN	-	-
Marburg disease			NN			NN		NN	-	-
Malaria	8	6	33	7	2		1	2	59	219
									-	-
Meningococcal infections	2					NN			2	19

Disease	N.S.W.	VIC.	QLD.	S.A.	W.A.	TAS.	N.T.	A.C.T.	Total	Cumulative Total to Date for Year
Non-specific urethritis	264		NN	38	NN	NN	NN	NN	302	1 264
Ornithosis									-	2
Pertussis (whooping cough)	3		NN	13	12	NN	4	NN	32	168
Plague									-	-
Poliomyelitis									-	-
Q. fever	13		18	3					34	135
[illegible]ies				NN		NN		NN	-	-
Salmonella infections	93	9	75	39	25	35	17	7	300	1 044
Shigella infections	3	1	8	5	12		16		47	202
Smallpox									-	-
Syphilis	28		70	9	16	1	61		185	621
Tetanus		1			1				2	3
Trachoma		NN			5	NN	NN		5	21
Tuberculosis (all forms)	33	23	22	2	9		3	3	95	302
Typhoid fever	4								4	22
Typhus (all forms)									-	-
[illegible]ibrio parahaemolyticus infections			NN			NN		NN	-	1
Yellow fever									-	-
Yersinia infections	11		NN			NN		NN	11	33

NN - Not Notifiable

(Note: Data collected under the Notifiable Diseases Returns may bear little or no correlation to that collected under the CDI laboratory scheme. Whilst the latter is a sampling program, the Notifiable Diseases data is dependent upon voluntary reporting by medical practitioners etc.)