Texas Preventable Disease NEWS

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contents:

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ORAL VIRAL LESION (HAIRY LEUKOPLAKIA) ASSOCIATED WITH ACQUIRED IMMUNODEFICIENCY SYNDROME*

From October 1981 to June 1985, 13 (11%) of 123 patients with hairy leukoplakia (HL) seen in San Francisco, California, were additionally diagnosed as having acquired immunodeficiency syndrome (AIDS). Eighty (73%) of the 110 patients who did not have AIDS at the time of HL diagnosis were followed. Twenty of these developed AIDS within 1 to 33 months (mean 7.5 months) of HL diagnosis. Seventy-nine serum specimens from the 123 patients with HL were tested for antibody to human T-lymphotropic virus type III/lymphadenopathy-associated virus (HTLV-III/LAV) by indirect immunofluorescence. Of these, 78 (99%) were positive. The one negative result was also negative by Western blot test. All cases met the CDC case definition for AIDS.

Oral viral "hairy" leukoplakia of the tongue appears as raised white areas of thickening on the tongue, usually on the lateral border. The lesions may not respond to traditional antifungal therapy and appear to have unusual virologic features. Candida has been reported on the surface of the HL lesions. A number of viruses, including papilloma, herpes, and Epstein-Barr, have been identified by electron microscopy in biopsies obtained from the HL lesions. HL was first identified in San Francisco in 1981. The lesion has also been reported in patients examined in Los Angeles, California; Baltimore, Maryland; Ann Arbor, Michigan; Paris, France; Copenhagen, Denmark; and London, England.

MMWR Editorial Note: HL may be of diagnostic value as an early indicator of HTLV-III/LAV infections, especially when observed in combination with other clinical findings. Approximately 95% of patients with AIDS and AIDS-related complex are reported to have cervical lymphadenopathy and other head and neck manifestations of disease, which may be detected by dentists or others undertaking oral or facial examination.

Health-care providers, including dental personnel, are in a unique position to identify clinical oral symptoms and their potential association with AIDS. Kaposi's sarcoma (KS), candidiasis, recurrent herpetic infections, and papillomas are oral manifestations that have been associated with AIDS. Unresolved candidiasis may be one of the earliest signs of AIDS in persons in groups at risk of acquiring AIDS. Oral KS is virtually pathognomonic of AIDS in males aged 25 to 44 years. Squamous cell carcinomas, non-Hodgkins lymphomas, and malignant melanomas have also been reported to occur in the oral cavity in association with AIDS.

While careful histories and physical examinations alone will not identify persons with AIDS or related symptoms, oral findings, including this newly reported oral lesion, are important diagnostic tools for health-care providers in early identification and treatment of AIDS.

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*Reprinted from: CDC. MMWR 1985;34:549-50.

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GENITAL HUMAN PAPILLOMAVIRUS (HPV) INFECTIONS*

Papillomavirus infections cause a wide range of diseases in humans; among these are genital warts, also referred to as condylomata acuminata. Genital warts have been recognized as a sexually transmitted disease (STD) since ancient Greek and Roman times, and are one of the most common of all STD's in this country. Among private patients in the United States, cases of genital warts have increased dramatically over the last twenty years.

Papillomavirus are a subgroup of the papovaviruses, and over 30 HPV types have been identified by DNA hybridization techniques. Genital warts are most commonly associated with virus types 6, 11, 16, and 18. Other subtypes cause common, filiform, plane, and plantar warts, epidermodysplasia verruciformis, and laryngeal papillomata. Like herpesviruses, papillomavirus is widespread throughout the animal kingdom, and many species of mammals and birds are commonly infected with this virus.

External genital wart infections are usually diagnosed by the presence of fleshy verrucae on the genitals, anus, or vaginal wall. Many HPV induced lesions, however, are subclinical epithelial proliferations on the internal genitalia that are diagnosed with colposcopic, cytologic, or histologic studies. Recently developed immunoperoxidase staining techniques may also be useful for diagnosis, although sensitivity of this test is not very high. Though this test may be available in some New Mexico laboratories, it is not generally considered a useful study. Unfortunately, there are no simple, rapid serologic tests to identify infected individuals.

Treatment of genital warts is usually difficult and often gives poor results. Many approaches have been advocated, from excision to topical applications of various preparations. Clinical trials have shown that electrocautery of external lesions was superior to cryotherapy. Results from treatment with topical podophyllin and with trichloracetic acid were less favorable. Laser therapy of internal and external lesions usually achieves satisfactory results, though prospective clinical trials have not been published on this form of therapy.

One of the most important concerns regarding HPV infection is its possible association with cervical and other genital cancers. Some investigators have suggested a direct relationship between HPV and cancer. Using DNA hybridization techniques to detect HPV-16 in cervical flat condylomata, researchers correlated the presence of this viral type with abnormal mitoses. They suggested that flat warts containing HPV-16 are precursors to invasive cancer of the cervix. Other studies have observed that flat condylomata and cervical intraepithelial neoplasia (CIN) frequently co-exist, and that lesions of the two conditions are histologically linked. Women with evidence of HPV infection of the cervix were shown to develop cervical dysplasia and neoplasia at a younger age than uninfected women. Epidemiologic studies from Mayo Clinic, furthermore, disclosed that the risk of developing cervical carcinoma-in-situ was 4 times higher in women with histories of genital HPV infection, compared to uninfected women. Malignant change in condylomatous lesions of the penis and vulva has also been observed, suggesting that genital warts are precursors to these neoplasms, as well. Laboratory and epidemiologic studies are currrently being conducted to explore the relationship between these conditions, and several researchers in New Mexico have developed an interest in HPV infections. Ongoing research here and nationwide should help with our understanding of this virus and its relationship to later development of genital concerns.

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*Reprinted from: New Mexico Health & Environment Dept. NM Epi Report, May 1985.

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PERTUSSIS ACTIVITY IN TEXAS THRU WEEK #42

During week #42, ending October 19, 1985, 69 cases of pertussis were confirmed. This report brings the total number of confirmed cases to 251: 67 clinical confirmations, 129 confirmed by DFA, and 56 culture confirmations (Table 1). The incidence (251 cases) is 318% greater than the incidence (60 cases) in all of 1984.

The Pertussis Case Register contains 362 total reports with the following dispositions:

Confirmed pertussis. . .251	Pending additional information/invest . . 91
Probable pertussis 2	Dropped from register. . 18

Geographic distribution of the confirmed cases:

Region	# Cases	Region	# Cases
1.	1	8.	6
2/12.	14	9.	61
3.	8	11.	4
4.	2	Bexar Co.	64
5.	42	Harris Co.	13
6.	7	Houston.	19
7/10.	10		

Table 1.
Age, sex, and vaccine history of reported-confirmed pertussis cases in Texas through October 19, 1985.

MALE

Age	# CASES	Doses DTP Vaccine Rec. 0	1	2	3	4+	Unk
≤1 mo.	17	16	1				
2	13	9	3				1
3	6	3	3				
4	6	3	1	2			
5	6	4	2				
6	2		2				
7	4	2	1	1			
8	3	3					
9	3	2		1			
10	1	1					
11	1			1			
1 yr.	16	7	2	2	2	2	1
2	6	1			2	2	1
3	5	1				2	2
4	3	1				1	1
5	2					1	1
6	1					1	
7-9	10			1		7	2
10-14	2					2	
15-19							
20+	8					5	3
Total	115	53	15	8	4	23	12

FEMALE

Age	# CASES	Doses DTP Vaccine Rec. 0	1	2	3	4+	Unk
≤1 mo.	19	18	1				
2	9	5	4				
3	13	6	5	1	1		
4	5	2	1	2			
5	6	4	1	1			
6	4	3	1				
7	5	1	2	1	1		
8	2	1	1				
9	2	2					
10	3	1	1		1		
11	3		2	1			
1 yr.	18	8	3	2	4		1
2	2					1	1
3	10	3	1		3	2	1
4	3	1				2	
5	4			1		3	
6	1					1	
7-9	3					2	1
10-14	2					1	1
15-19	1		1				
20+	21					6	15
Total	136	55	24	9	10	18	20

AGE SUMMARY - ALL CASES		
Age	Cases	%
<1	133	53.0
1-4	63	25.1
5-9	21	8.4
10-14	4	1.6
15-19	1	0.4
20+	29	11.5
Total	251	100.0

VACCINE HISTORY - ALL CASES		
Doses	# Cases	%
UNK	32	12.7
0	108	43.0
1	39	15.5
2	17	6.8
3	14	5.6
4+	41	16.3
Total	251	100.0

107 cases (42.6%) vaccinated, appropriately for age.

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A NOTE TO SMOKING PARENTS*

Malignant neoplasms (cancers) are the second leading cause of death in the United States (heart disease is the leading cause). . . .Cigarette smoking is now linked to a long and growing list of cancers in man. In the May 1985 issue of the American Journal of Public Health, Sandler et al** demonstrate a 50% increase in the risk of cancer (all types excluding basal cell cancer of the skin) in offspring of men who smoked cigarettes. This increased risk was independent of demographic factors, social class, or individual smoking habits and was not limited to known smoking-related sites. Mothers' and fathers' smoking were both associated with an increased risk of cancers of the blood-forming system; the risk was higher with heavier parental smoking. The risk was elevated 1.7 times when only one parent smoked but 4.6 times when both parents smoked. The authors suggest that transplacental or childhood passive exposure to cigarette smoke may be involved.

These findings should be considered tentative; other investigators will undoubtedly examine these associations in other data sets. Meanwhile, intelligent and responsible adults have another possible risk to factor into their justification for continuing cigarette smoking.

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*Reprinted from: Colorado Dept. of Health. Colorado Disease Bulletin 1985, issue no. 14.

**Sandler DP et al. Cancer Risk in Adulthood from Early Life Exposure to Parents' Smoking. AJPH 1985;75:487-92.

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