EPIDEMIOLOGY SECTION

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SWIMMER'S ITCH AND OTHER JOYS OF SUMMER

Each summer, the Department of Health Services receives numerous reports of episodes of dermatitis and/or bites occurring in bathers in both fresh water lakes and ponds and marine waters along the shoreline. There are several potential causes of such occurrences: algal blooms, jelly fish, sand fleas and schistosome dermatitis. It is important to identify the specific cause of the problem in order to advise bathers of the nature of the risk, appropriate treatment and possible methods of prevention and control.

Schistosome dermatitis

Bilharziasis, a blood fluke infection caused by human schistosomes, is a serious problem in developing countries. Infection is acquired from water containing larval forms (cercariae) which have developed in snails. These invasive human schistosomes are not found in this area. Several species of Schistosoma infect mammals and birds, but cannot infect man. Many schistosome cercariae which ordinarily infect birds and semiaquatic mammals are capable of penetrating the skin of man, although they cannot produce a permanent infection. Because man is not their normal host, they will penetrate only a short distance and then die. Their presence may, however, produce an allergic response which is referred to as "swimmer's itch."

In 1928, Cort first demonstrated that cercariae of certain non-human schistosomes could cause an aggravating form of dermatitis. Since that time, swimmer's itch associated with cercariae-infected fresh water has been reported from many regions of the United States, Europe, Latin America, India, Australia and New Zealand. Dermatitis associated with salt water beaches occurs in Hawaii, and many coastal parts of the USA including Connecticut and Rhode Island. This form of schistosome dermatitis is frequently referred to as "clam-digger's itch." The clinical syndrome and the epidemiology of the disease are similar for both types of dermatitis.

Swimmers who have been exposed to the free-living cercariae may experience a prickling or nettling sensation immediately after emerging from the water. This may be accompanied by erythema of the invaded area and, in highly sensitive individuals, by local or generalized urticaria. The initial irritation soon subsides, leaving a minute macule at each site of penetration. Within a few hours, there is intense itching of the involved area with transformation of the macules into papules. The reaction reaches its maximum between the second and third day and then gradually subsides, but symptoms may be reactivated by rubbing the involved area. Because schistosome dermatitis is a sensitization phenomenon, the severity of response may be directly proportional to the number of exposures. No specific treatment is available, but the pruritis may be relieved by oral antihistamines or topical anti-pruritic agents. Topical steroids such as hydrocortisone creams may also be used and in very severe cases, a course of oral steroids may be indicated.

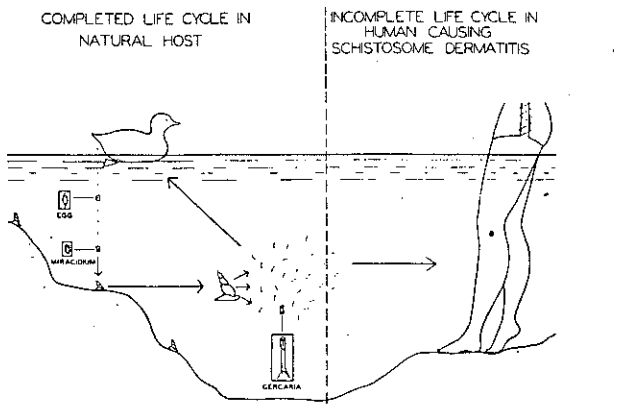

Figure 1. The life cycle of the parasite causing "swimmer's itch". Reproduced with permission from Miller MJ, Munroe E. Schistosome dermatitis in Quebec. Can Med Assoc J 1951; 65: 571.

The life cycle of all schistosomes is similar involving a specific species of snail as an intermediate host and a mammal or bird as the final host (Figure 1). Adult worms mate in the vertebrate host and lay eggs which are excreted into the water. Once in the water, the eggs hatch and release ciliated larvae (miracidia) which penetrate and infect susceptible species of snails. In the snail, they undergo the asexual phase of their life cycle finally producing numerous fork-tailed larvae (cercariae) after in four to six weeks which are released into the water in search of the final host animal. These are usually liberated at a certain period of each day and a single infected snail may shed cercariae for months. However, each cercaria can only survive for one to two days in the free state.

Swimmer's itch associated with fresh water is frequently caused by a group of schistosomes, Trichbilharzia, whose definitive hosts are birds, especially wild ducks. Snails belonging to the genera Lymnaea, Stagnicola and Physa serve as intermediate hosts for the parasite. The incidence of dermatitis caused by bird schistosomes tends to be restricted to the early part of the summer. This is related to the migratory cycle of the avian definitive host and the midsummer die-off of the snail host.

The causative agent of clam digger's itch in marine waters of New England is Austrobilharzia variglandis. A wide variety of birds are capable of harboring this parasite. The intermediate host is the mud snail, Ilyanassa obsoleta (formerly Nassarius obsoletus). The number of cercariae produced by an infected mud snail is low, less than 160 per day. Also, the prevalence of snail infection is low, about 1-3%.

Shedding of cercariae may be affected by temperature and wind currents. It is not unusual, therefore, to receive reports of dermatitis right after a very hot spell, such as occurred Labor Day weekend of 1983.

Schistosome dermatitis can only be confirmed by demonstration of cercariae in snails known to serve as intermediate hosts in the schistosome cycle. The state laboratory and some local health department laboratories can perform such tests. Because only small numbers of snails are infected, at least 100 snails (same species) must be collected to provide a sufficient sample for testing. At least one gallon of source water must accompany the specimens. These tests will only be run by special arrangement with the Epidemiology Section or the state laboratory.

Control of schistosome dermatitis is limited to host protective measures since it is neither feasible nor environmentally sound to use chemicals to control the snail population. Bathers should be advised to vigorously towel off immediately after emerging from the water. This method of mechanically removing the cercariae may be quite successful if followed. Leaving the water every 10 minutes and towel drying has also been suggested. Affected areas should be posted during periods of known activity to warn swimmers of the presence of the parasites and precautions for preventing the itch.

Jelly Fish Stings

Three types of jellyfish are found in this area: the comb jellyfish, the moon jellyfish and the Lion's Mane. Neither the comb nor the moon jellyfish have stinging cells, but they may produce a rash (due to protein enzyme agents). The Lion's Mane can grow to one foot in diameter and has stinging cells for injecting poison in plankton and small fish. The toxin is not powerful, but it may cause irritation. Over the past three to four years, Lion's mane have been more abundant in Long Island Sound.

Jelly fish stings may be treated with calamine or alcohol. One of the most effective treatments is the use of papain-containing meat tenderizers sprinkled liberally on the wetted affected areas. It is hypothesized that the papain cleaves the polypeptides of the toxin converting them into amino acids.

Algal Blooms

Algal "blooms" frequently occur in the spring and the autumn when luxuriant plant growth results from the combination of warm water, plentiful nutrients and shallow depth. They usually tend to clear themselves and affect only the aesthetic quality of a body of water. Illness in man and animals has been attributed to both marine and fresh-water algae including contact-type dermatitis and symptoms of hay fever caused by blue-green algae.

AIDS UPDATE

The Centers for Disease Control (CDC) began national surveillance for AIDS in June 1981. Since that time, 5,037 cases have been reported. The majority of these cases continue to occur in homosexual or bisexual men. The proportion of adult cases outside of population groups previously identified as being at risk of AIDS has remained constant.

Connecticut has reported 62 cases as of July 10, 1984 (Table 1). Recently, Connecticut has experienced a substantial increase of cases (1). This increase continues to reflect a national trend and may be due to both increased transmission several years ago and also the contribution of persons only now developing immunodeficiency after exposure to the putative AIDS agent more than 2-3 years ago.

Table 1. AIDS Cases, Connecticut (8/3/84)

Primary Disease*	Cases	Percent of Total
KS without PCP	6	9.2
PCP without KS	40	61.5
Both KS and PCP	5	7.7
Other OI	14	21.5
Total	65	100.0

*KS = Kaposi's sarcoma
PCP = Pneumocystis carinii pneumonia
OI = Other opportunistic infections

By Risk Group	Cases	Percent of Total
Gay/Bisexual	37	56.9
Intravenous (IV Drug User)	14	21.5
Hemophiliac	3	4.6
Haitian (recent immigrant)	3	4.6
Other	8	12.3

Cases By County of Residence	
Fairfield	26
New Haven	25
Hartford	11
Litchfield	1
Middlesex	1
Transient	1

Cases By Sex	
Males	56
Females	9

Table 2. Seropositivity of Various Groups to Core Proteins of Retroviruses (RIPA)(7)

Population	Antigen Tested LAVp25	HTLV-Ip24
AIDS Patients	51/125	7/125
Lymphadenopathy Syndrome Patients	81/113	0/113
Healthy Homosexuals*	1/100	0/100
Healthy Blood Donors	0/189	0/189

*Sera collected in 1978

Retroviruses and AIDS

Initially, suspect etiologic agents of AIDS included members of a number of virus families such as herpes viruses, retroviruses and hepatitis B, all of which were frequently found in AIDS patients. However, due to the depressed immune status in these patients, it was difficult to determine whether these viruses were the original cause of the immune deficiency or yet another opportunistic infection resulting from the immune deficiency. Recent evidence has implicated a retrovirus as the etiologic agent. Two prototypes have been described.

Lymphadenopathy-associated virus (LAV) was isolated from the lymph node cells of a homosexual man with unexplained generalized lymphadenopathy by Luc Montagnier and associates at the Institut Pasteur in France (2). This same human T-lymphotropic retrovirus was subsequently isolated from two siblings with hemophilia B, one of whom had AIDS (3). Feorino and associates have also isolated a retrovirus antigenically identical to LAV from a blood donor-recipient pair, each of whom developed AIDS (4).

A morphologically similar T-lymphotropic retrovirus (HTLV-III) was isolated by Gallo and associates at NIH from lymphocytes of 26 of 72 patients with AIDS and 18 of 21 patients with AIDS-related complex (5).

Direct comparative results suggest that they are likely to be the same virus (6). They have the same appearance by electron microscopy and are both lymphotropic and cytopathic for OKT-4 cells. Isolates from American AIDS patients are immunologically indistinguishable from LAV. Serologic tests of a large number of specimens from patients with AIDS and lymphadenopathy syndrome demonstrate similar results using either prototype as antigen. Finally, LAV and HTLV-III appear to be highly related based on competitive radioimmunoassay of their core proteins (6). More definitive studies are underway.

Three serologic procedures have been developed for the detection of antibody to HTLV-III/LAV: an enzyme-linked immunosorbent assay (ELISA) to whole disrupted virus, a radioimmunoprecipitation assay (RIPA) to the presumed major core protein (p25) of LAV, and assay of antibody to major viral antigens by the West blot technique (6). In general, these tests appear to be fairly sensitive and specific (Table 2). However, they do differ from one another. Compared to the RIPA, the experimental ELISA test appears to be the most sensitive (see below). Correspondingly, it may also have the greatest tendency to cause false positive titers. Sera from high-risk populations are being tested by the National Cancer Institute (NCI), the Institut Pasteur and CDC to determine the frequency of exposure by each of these tests to HTLV-III/LAV and to correlate seropositivity with current infection, signs and symptoms, and prognosis.

Preliminary data suggest that seropositivity is common and increasing in certain populations. Antibody prevalence to LAV (RIPA) has increased among sera samples from homosexual men attending a sexually transmitted disease clinic in San Francisco; 1% (1/100) in 1978, 25% (12/48) in 1980 and 65% (140/215) in 1984 (6). Eighty-six recent heavy IV drug users in New York City were tested for antibody response to LAV. Using the ELISA test, 87% demonstrated antibody, while 58% showed antibody by RIPA. Among a group of long-term methadone patients used as controls, fewer than 10% of 35 patients had antibody to LAV detected by RIPA.

Epidemiologic Implications

The high prevalence of antibody to HTLV-III/LAV among these groups not only supports the hypothesis that it is the etiologic agent, but also demonstrates that exposure to the virus is much more common than AIDS itself among populations with increased incidence of the disease. These data suggest that the host reponse to infection may cover a wide spectrum. It has been postulated that the primary event of T-lymphotropic retrovirus infection must be followed by immunogenic stimulation to bring about AIDS. Antigenic stimuli, such as HBV infection or frequent infusions of plasma products, may elicit T-cell proliferation including that of lymphocytes with latent infection. This activation could trigger viral expression in these lymphocytes increasing their diffusion in the population of helper T-cells (3).

While the serologic tests appear to be sensitive and specific enough to be of value in estimating the frequency of infection in certain populations and in determining information about the natural history of the disease, the implications of a positive test result are less clear for the individual. In some individuals, the result may be a false positive (i.e., cross-reaction with an antigenically related virus or non-specific test factors). The predictive value of the test must still be established, particularly for individuals who do not belong to any known AIDS risk groups (e.g., most blood donors) and in whom the prevalence of true infection with HTLV-LAV is expected to be low.

For individuals in populations at greater risk of acquiring AIDS, a positive serologic test will probably indicate infection at some time with the virus, but will not differentiate between recent or past infection (i.e., immunity)(6). Furthermore, it will not give specific information on who is viremic and capable of transmission. Carefully planned studies are required to resolve these issues.

Scientists, including Dr. Gallo, have called predictions of a vaccine within two years highly optimistic and have pointed out that they should be able to have the vaccine reagent ready in about two years. From that point, further testing, including human vaccine trials, might require from two to five more years.

What impact the new discovery may have on therapy for those who have AIDS now remains unclear. Anti-HTLV-III drug efficacy studies will be able to be performed in vitro. However, it seems unlikely that any antiviral agents identified in this manner would radically alter the course of HTLV-III infection that has resulted in AIDS. Such agents might be more likely to be effective in earlier stages of infection (e.g., pre-AIDS) in an attempt to abort the development of AIDS. It is premature, however, to speculate on modification of HTLV-III/LAV infection by any agent before we have a better understanding of the natural history of that infection. It may be that only a small percentage of such infections result in AIDS.

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REPORTED MORBIDITY - JUNE, 1984

	AMEBIASIS	BOTULISM	BRUCELLOSIS	ENCEPHALITIS (TOTAL)	Primary	Post	FOODBORNE OUTBREAKS	GONORRHEA	HEPATITIS A	HEPATITIS B	HEPATITIS NON A NON B	HEPATITIS UNSPECIFIED	LEGIONELLOSIS	LEPROSY	MALARIA	MEASLES	MENINGITIS (All Types)	Aseptic	Hemophilus influenzae	Meningococcal	Other	MUMPS	PERTUSSIS	PSITTACOSIS	RABIES IN ANIMALS	REYE'S SYNDROME	ROCKY MT. SPOTTED FEVER	RUBELLA	SALMONELLA	SHIGELLA	SYPHILIS	TUBERCULOSIS (TOTAL)	Pulmonary	Other	TYPHOID FEVER
Total for June	5	0	0	2	1	1	4	769	24	19	7	7	2	0	1	3	22	5	10	4	3	1	0	0	0	0	0	0	98	16	11	11	10	1	1
Cumulative 1984	26	0	0	8	7	1	8	5273	39	157	16	16	9	0	7	13	86	11	25	28	22	9	0	2	1	0	0	0	341	58	53	74	56	18	2
Cumulative 1983	11	1	0	6	6	0	4	4335	32	205	26	9	24	1	6	8	111	20	31	31	29	12	0	1	3	0	0	0	407	111	80	75	58	17	0

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NEW STAFF ASSIGNMENTS IN EPIDEMIOLOGY

James L. Hadler has recently been named Chief of Epidemiology and State Epidemiologist for the Department of Health Services. Dr. Hadler received his M.D. from Columbia University College of Physicians and Surgeons in 1972. He completed an Internal Medicine residency at Waterbury Hospital and was a fellow in Infectious Diseases at Yale University School of Medicine. From July 1980 to June, 1982 he did a preventive medicine residency at Yale and also earned an M.P.H. from the Department of Epidemiology and Public Health.

Jim served two years with CDC, USPHS as tuberculosis control officer on the Navajo Indian Reservation and was most recently a Yale-China Association Exchange Scholar at Hunan Medical College for a year where he studied nosocomial infections.

Ellen E. Jones, M.D., a Preventive Medicine Resident with the Centers for Disease Control, completed her Connecticut assignment in June 1984. Ellen recently married Dr. William Mangione of Denver, Colorado.

Matthew L. Cartter, M.D. has been assigned to the Department of Health Services by the CDC. Dr. Cartter, a Connecticut native, completed his undergraduate work at Wesleyan University. He received his medical degree from the University of Rochester and recently completed a medical residency at Rhode Island Hospital. Matt is a second year EIS officer. His first year was spent in Alaska doing hepatitis surveillance and directing a project to screen for hepatocellular carcinoma. His Connecticut assignment is for one year.

Drs. Hadler and Cartter may be reached at 566-2540.

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