EPIDEMIOLOGY SECTION

Connecticut Epidemiologist

STATE OF CONNECTICUT DEPARTMENT OF HEALTH SERVICES
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Vol. 2, No. 6
June, 1983

LEGIONNARIES' DISEASE: WHAT HAVE WE LEARNED?

It's been seven years since the outbreak of pneumonia among American Legionnaires in Philadelphia in July 1976 and the subsequent isolation of an etiologic agent. During this time, an impressive body of knowledge has been developed regarding Legionella species and the various manifestations of the disease called legionellosis or Legionnaires' disease (LD).

Surveillance of legionellosis has been conducted by the State of Connecticut Department of Health Services and by the Centers for Disease Control (CDC) since that time. Availability of diagnostic tests in 1977 and the decision of the Conference of State and Territorial Epidemiologists to make legionellosis a nationally reportable disease in 1978 promoted surveillance efforts. Retesting of sera from previously reported outbreaks which had unidentified etiologies has demonstrated that Legionella pneumophila was the cause of many of these outbreaks. Additionally, continuing investigations of other outbreaks and sporadic cases have contributed to our understanding of the natural history of LD.

The etiologic agent of LD is a fastidious gram-negative rod which belongs to a newly described genus and family of microorganisms. Six serotypes of Legionella pneumophila, lso referred to as Legionnaires' Disease Bacillus (LDB) are currently recognized (Table 1). Serogroup 1 appears to be the most common. Several other related species have also been identified and have been included within the family Legionellaceae. Of these, L. micdadei, the name given to the Pittsburgh pneumonia agent (PPA), has also been identified as a common cause of nosocomial pneumonia. To date, 23 different antigens have been demonstrated.

Laboratory experiments have demonstrated that cell-mediated immunity is the most important host-defense system in LD is the disease transmitted by the airborne route. The primary site of infection is the lower respiratory tract. Initial pulmonary symptoms of nonproductive cough and dyspnea without upper respiratory symptoms of pharyngitis or tracheitis, and the multilobar distribution of the pneumonia without preferential involvement of the dependent lower lobes, all suggest the organisms are inhaled rather than aspirated. To date, attempts to demonstrate colonization with Legionella pneumophila have been unsuccessful.

CLINICAL FEATURES

LD is recognized in three distinctive clinical-epidemiologic patterns: nonpneumonic (Pontiac fever), community-acquired pneumonia, and nosocomial pneumonia. Characteristics of these various manifestations are summarized in Table 2.

Pontiac fever is a self-limiting, nonpneumonic, febrile illness characterized by a short incubation period and a high attack rate (almost 100%). No distinctive clinical findings differentiate Pontiac fever from a group of epidemic diseases called inhalation fever. Of the known inhalation fevers, it most closely resembles humidifier fever. Both illnesses occur as common-source outbreaks after short incubation periods and reexposure can produce recurrent symptoms. While humidifier fever is believed to be caused by immunologic mechanisms, it is not clear whether symptoms in Pontiac fever represent infection with Legionella pneumophila or an immune mediated-mechanism (1).

Table 1: Recognized Members of Family Legionellaceae

Legionella pneumophila		Non-pneumophila Legionellae
Serogroup 1	Philadelphia	L. bozemanii
Serogroup 2	Togus	L. dumoffi
Serogroup 3	Bloomington	L. gormanii
Serogroup 4	Los Angeles	L. jordanis
Serogroup 5	Dallas	L. longbeachae
Serogroup 6	Chicago	L. micdadei
		L. wadsworthii

Table 2: Comparsion of Clinical and Epidemiologi Features of Legionnaires' Disease Outbreaks (1)

Feature	Pontiac Fever	Community-Acquired Legionnaires' Disease	Nosocomial Legionnaires' Disease
Outbreak pattern	Common source	Common source	Common source in hospital
Source	Air conditioning Steam turbine Whirlpool	Air conditioning Chimney aerosols Evaporative condensor	Cooling tower Contaminated potable water Respiratory therapy equipment
Incubation period	5-66 hrs. (average 36)	2-11 days (average 7)	1-28 days
Attack rate	95-100%	4%	0.5-1.5%
Underlying disease	None reported	66%	90%
CFR	0%	15-25%	25-50%
CFR related to underlying disease or immunosuppression	No	No	Yes
Recurrent disease with reexposure	Yes	Unknown	Unknown

Patients with LD generally have pneumonia. Infection is most commonly manifested as atypical pneumonia with cough but little or no sputum production early in the illness. Early symptoms may be nonspecific and may include headache, lethargy, anorexia, malaise, and profound weakness. Diarrhea occurs in about 50% of cases and may precede or follow onset of respiratory symptoms. Fever usually increases during the first several days of illness to 38.9°C and is unremitting unless appropriate therapy is given. Antipyretic agents and corticosteroids have little effect on the fever (2). Changes in mental status and other central nervous system indicators occur in about one-third of cases and range from lethargy and confusion to grand mal seizure and coma.

Unless appropriate therapy is initiated, the illness usually worsens during the first week. The pneumonia extends to involve adjacent or other lobes of the lung. Involvement of the entire lobe or both lobes with adult respiratory distress syndrome (ARDS) may result.

Clinically, nosocomial LD differs from community-acquired disease in many respects. It occurs primarily in compromised patients, including by order of frequency, patients with severe immunosuppresion (such as organ transplant recipients), cancer, cardiac, pulmonary, or renal diseases. Nosocomial LD also tends to be more severe than community-acquired LD. The case fatality ratio of nosocomial LD (25-50%) is significantly higher than in community-acquired cases even when effective antimicrobial therapy, i.e. erythromycin, has been initiated

(1,2,3). Outbreaks of LD have been commonly noted in hospitals. Nosocomial acquisition by immunosuppressed patients has been noteworthy in Los Angeles, Chicago, Pittsburgh, and Vermont.

EPIDEMIOLOGIC FEATURES

Sporadic, community-acquired LD is estimated to account for 5-15% of bacterial pneumonias (30,000-100,000 cases per year). Several serosurveys have been conducted in the United States to determine the prevalence of infection with Legionella pneumophila. Reciprocal indirect fluorescent antibody (IFA) titers of $\geq$ 128 to Legionella pneumophila serogroup were observed in 1.3% of normal controls to 24.8% of control volunteers in areas experiencing outbreaks (4,5). However, among 113 patients with chronic pulmonary disease only one patient had a titer 1:128 (0.9%)(6). A similarly low prevalence was noted in middle-aged and elderly Americans from four large cities. Only 15 of 1,143 sera (1.3%) demonstrated reciprocal titers of $\geq$128 (7).

In Connecticut a serosurvey was conducted as part of an investigation of an outbreak of nosocomial pneumonias. Three groups were tested: hospital employees, nursing personnel from the affected wing and industry employees from a company approximately five miles away from the affected hospital. Results of the survey are shown in Table 3. The proportion of seroreactive employees with titers $\geq$128 or $\geq$256 was not significantly different for the three population groups (8).

It must be noted that rates of seropositivity are affected not only by the prevalence of infection in a geographic area but also by the number of antigens used and the method of antigen preparation employed (i.e. heat-killed vs. ether-killed vs. formalin-killed).

The incidence of LD pneumonia in the general population has been estimated from a prospective study of pneumonia in a prepaid medical group in Seattle. To date this is the only population based study conducted. Extrapolation from this study suggests the annual incidence is about 12/100,000 population or about 25,000 cases per year (3).

However, this must be considered as a minimum figure because it does not take into account hospitalized cases or areas of hyperendemic activity.

The incidence of fatal nosocomial LD pneumonia was estimated by a retrospective study of lung tissue specimens from 263 fatal cases of nosocomial pneumonia submitted to the CDC by hospitals participating in the National Nosocomial Infection Study (NNIS). In 3.8% of these cases, Direct Fluorescent Antibody (DFA) tests were positive for Legionella pneumophila. Extrapolation of this data suggests that approximately 950 fatal nosocomial cases occur in U.S. hospitals each year (9).

More recent studies have attempted to estimate the incidence of nosocomial pneumonia due to Legionella species. In a prospective study of 1,658 patients admitted to Wadsworth Hospital, fourfold or higher titers developed in 15 (1.5%) of 1,018 paired sera. Seven of these patients developed clinical pneumonia; however, 8 (53%) did not develop pneumonia and presumably represent subclinical legionellosis (10). An eighth patient was diagnosed at autopsy by DFA. Based on patients with clinical disease only, an attack rate of 0.5% can be estimated. In the Connecticut outbreak, 0.7% of patients admitted during August and September 1978 acquired nosocomial LD (3).

Muder et al. conducted a three month prospective study in two hospitals in Pittsburgh -- the Pittsburgh VA Hospital where endemic Legionnaires' has been well documented and a 607-bed community hospital where LD had neven been documented. During the three month period a total of 57 cases of pneumonia were identified at the VA, of which 32 (56%) were nosocomial. Of the nosocomial cases, 14 (44%) were caused by L. pneumophila serogroup 1 and three by L. micdadei (9.4%). (In two patients both LDB and PPA were identified). At the community hospital 73 cases of pneumonia were diagnosed, 28 (38%) of which were nosocomial. Four of these (14.3%) were due to L. pneumophila (11). No denominator data is available to determine incidence. However it is noteworthy that in hospitals previously reported to be hyperendemic foci of LD, the proportion of nosocomial pneumonias caused by L. pneumophila has been reported as 11% to 29%. These results strongly suggest that Legionnellae are an important cause of nosocomial pneumonia and that a substantial number of pneumonias of unknown etiology may be due to these microorganisms.

Prognosis and case-fatality ratios (CFRs) are affected by the use of appropriate antimicrobial agents and the presence of underlying diseases. The CFR is approximately 15-20% overall and has been approximately 25% at Wadsworth VA Hospital where underlying illness has been common in affected patients. Rates are highest (80%) among immunosuppressed patients who do not receive erythromycin and lowest (7%) among patients who receive erythromycin and are not immunosuppressed (2).

LABORATORY DIAGNOSIS

Currently, the following three methods are used for the diagnosis of LD; isolation of the organism, indirect immunofluorescence studies of sera to detect antibody (IFA), and direct immunofluorescence examinations of specimens to detect the antigen (DFA). Other methods designed to detect the presence of the organism in urine are still experimental.

The most widely used test is the Indirect Fluorescent Antibody (IFA) technique for serologic diagnosis. An increase in titer of at least fourfold (to at least 1:128) in acute and convalescent serum specimens is considered confirmatory. The sensitivity of the IFA test is influenced by the antigen(s) used, the methods used to inactivate them, the suspending solution, and by the prevalence of the disease in a given population. In various studies, the sensitivity of the IFA for Legionnaires' has been estimated to be about 75% (12,13,14).

The diagnostic specificity of a given test is lower in a population with a higher background prevalence of antibody.

For diagnostic and epidemiologic purposes, a fourfold rise in titer is required. However, only about three-quarters of patients with clinical disease proven by isolation of the organism develop a fourfold rise in titer early in the course of their illness. As many as 25% of the seroconversions that do occur are not detected until 4-8 weeks after the onset of illness and require use of multiple antigens for testing (12). While single specimens with high titers ($\geq$ 1:256) together with clinical illness have been considered presumptive evidence of LD their significance is actually difficult to interpret. The high prevalence of antibody to LDB among Connecticut residents must be taken into consideration in attempting to evaluate the signifiance of a single titer. The State Laboratory will test single specimens but encourages physicians and laboratories to submit both acute and convalescent sera at the same time for simultaneous testing. Serum from the acute blood specimen may be frozen until the convalescent serum is obtained.

Table 3: Seroactivity of Selected Populations to L. pneumophila Serogroup 1, Connecticut, 1978 (8)

	Total Tested	IFA Titer $\geq$ 128		IFA Titer $\geq$ 256	
		#	%	#	%
Hospital Employees	293	71	(24.2)	32	(10.9)
Nursing Personnel Wing "X"	94	22	(23.4)	11	(11.7)
Industry Employees	154	32	(20.8)	17	(11.0)

The major drawbacks of the IFA test for LDB are 1) it is retrospective, 2) it does not usually influence the choice of therapy, 3) it may entail close reactions with other organisms, 4) it requires a battery of antigens from all serogroups, 5) the results of a single serum are equivocal and 6) at least 23 different antigens have been recognized, many of which are not available.

Direct fluorescent antibody (DFA) testing of lung tissue, respiratory tract secretions, pleural fluid, pus and other tissue specimens provides the most rapid method of diagnosis. This technique requires multiple reagents with serogroup conjugates for the six serogroups of L. pneumophila and the other recognized species. The DFA has been most successful with lung tissue, expectorated sputum, endotrachael suction aspirates and tracheal aspirates. If positive the DFA is an excellent indicator of infection with Legionnella. However, if negative it does not rule out LD. The sensitivity of the DFA test for sputum is approximatey 70%; therefore, therapeutic and diagnostic decisions should not be based on a single negative result. Presence and duration of antimicrobial therapy affect not only the quantity of organisms likely to be seen on a smear but also qualitative differences in the gross character of the sputum and the microscopic appearance of the DFA positive bacilli (2).

Problems with this method involve the high level of skill needed for performance, the lack of availability of reagents, and (although uncommon), the complication of cross-reactions with strains of B. fragilis, Pseudomonas fluorecens, and other Pseudomonas sp.

Culture methods for isolation of Legionella species from clinical specimens are now within the capabilities of most clinical microbiology laboratories. Selective media suitable for growth of Legionella are easily prepared and are also available commercially. These all contain cysteine, a growth requirement for Legionnellae. The average incubation period for growth on the buffered, supplemental agar is about three days with a range of 1 to 10 days.

Because there is no single highly specific and sensitive test, we recommend that all three methods be used in the diagnosis of LD. The State Laboratory provides all three diagnostic services and also receives organisms isolated from clinical specimens for confirmation. Questions regarding diagnostic testing should be directed to the appropriate section of the laboratory (Legionella Serology: 566-2872, Legionella cultures and DFA: 566-4340).

RISK FACTORS

Cigarette smoking has been implicated as a risk factor in both sporadic and epidemic LD. In a case-control study of sporadic cases, persons smoking one or more packs of cigarettes per day had a risk 4.2 times higher than that of nonsmokers. Alcohol consumption, excavation sites near the home, and the presence of underlying disease have all been associated with increased risk of legionellosis. A male predominance among LD cases (2.5:1) is unexplained but may be a reflection of cigarette smoking and alcohol consumption among men.

Risk factors have been more consistently identified with nosocomial LD than with sporadic cases and community-associated outbreaks.

Major underlying predispositions include immunosuppression due to disease or therapy, cardiac disease, pulmonary disease, malignancy, and renal disease. Duration of hospitalization has also been identified as a risk factor in patients who are not immunosuppressed (2).

THE ROLE OF POTABLE WATER

Evidence has accumulated to suggest that L. pneumophila is widely distributed in nature and in man made environments. It has been isolated from thermal effluent water, creek water, mud, water from cooling towers, and evaporative condensors as well as from fresh-water lakes in diverse geographic locations. The frequency of its isolation suggests it is part of the natural aquatic environment. The organism has been associated with blue-green algae and amoeba in water and has been shown to survive for over a year in tap water. Although L. pneumophila is not thermophilic, it can survive in water temperature of 6°C to 67°C.

Implication of potable water as a source of nosocomial LD has now been raised by investigators in the United States and the United Kingdom. In virtually all these studies, the same serotype of L. pneumophila was isolated from patients and various potable water sites, such as hot or cold water taps, shower heads, and water storage tanks. However, these organisms were almost always found in areas of the hospital not associated with the disease. Stout et al. demonstrated that L. pneumophila is ubiquitous within the water system of a hospital with endemic LD (15). They also showed that the organism is present in highest concentration in the sediment of various water supplies, such as faucets, showers, and hot water tanks and that it has a predisposition for the thermal environment of the hot water distribution system. Aerosolization of contaminated potable water may, therefore, serve as the mechanism for the inhalation of infected droplets. Recently, Meenhorst et al. demonstrated that inhalation of aerosols of naturally contaminated potable water could produce legionella pneumonia in guinea pigs (16). These results add support to this mode of transmission as a possible mechanism for human disease.

Arnow et al. implicated aerosols from respiratory devices as the source of a nosocomial outbreak involving five patients who were being treated with corticosteroids at the same time. Tap water was used to fill nebulizers for oxygen masks and room humidifiers (17). L. micdadei has been isolated from nebulizers in use by patients, however, none were from rooms of patients who contracted pneumonia (18).

More recently we investigated 37 cases of nosocomial LD which occurred over a 2-year period. Significant risk factors of exposure were use of a nebulizer and/or IPPB apparatus and exposure to a portable room humidifier (19). This equipment was rinsed between uses with potable water. The humidifiers were filled with potable water. Respiratory devices with resevoirs such as aerosol masks, nasal cannulae and mechanical ventilators, which are both filled and rinsed with sterile water, could not be implicated. Cases, whether they were on steroids or not, were more likely to have been exposed to the implicated respiratory devices. These results suggest that rinsing respiratory equipment with sterile rather than potable water may prevent some nosocomial cases of LD.

Where decontamination of hospital water supplies has been undertaken, two appraoches have been used. Chlorination (3300 ppm free chlorine) of

cooling tower water may be effective, but may have long term corrosive effects. Furthermore, such high levels in potable water would give a bad taste to the water and may have long term affects on the metals involved in the plumbing circuit. Hot water systems also tend to vaporize and drive off the chlorine leaving levels that are ineffective.

A second approach has been superheating of the hot water system. Several investigations have shown that periodic raising of the hot water storage tank temperature to 77°C for varying periods (usually 48 to 72 hours) combined with flushing of faucets and showers (to get rid of the sediment) results in removal of culturable Legionella for varying periods of time. In certain hyperendemic settings, new cases of LD stopped for periods of several weeks until the tanks became recontaminated with high concentrations of Legionellae. One disadvantage of this approach is the risk of scalding individuals using taps fed from these storage tanks during the period of superheating.

SUMMARY

Legionnella is now well recognized as a cause of acute community-acquired pneumonia. It is also responsible for a significant percentage of nosocomial pneumonias especially when associated with outbreaks. Clinical and laboratory features are helpful but diagnosis must be confirmed by bacteriologic or serologic methods. All available methods should be used to confirm diagnosis since no one test has high sensitivity and specificity.

Legionella pneumophila has been shown to be ubiquitous in many water supplies. Contamination of hospital potable water supplies is probably responsible for most outbreaks of nosocomial pneumonia due to this organism. The finding of nosocomial cases of L. pneumophila should prompt an epidemiologic investigation to identify other cases in current patients and retrospectively. If a number of other cases are found, a case control study may be indicated. Because of the ubiquitous nature of the organism, environmental sampling should only be undertaken if a problem is documented and an epidemiologic association has been established. The Epidemiology Section can be contacted for consulation regarding investigation of nosocomial cases and environmental sampling.

Until we better understand the pathogenesis of legionellosis, we need to avoid pressures to modify both the hospital environment or the community environment in the absence of well-documented disease and associated exposures to specific environmental sources.

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REPORTED MORBIDITY - MAY, 1983

	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 MAY -1983	2	0	0	2	2	0	0	705	6	61	6	2	6	1	0	0	14	3	2	4	5	0	0	0	0	0	0	0	81	10	10	9	5	4	0
CUMULATIVE-1983	6	0	0	6	6	0	3	3456	22	165	23	4	21	1	4	2	82	10	22	28	22	11	0	0	0	0	0	0	290	104	80	55	42	13	0
CUMULATIVE-1982	14	1	3	13	10	3	3	3275	28	168	10	13	19	1	6	4	87	14	19	30	24	12	2	0	1	0	0	1	241	133	58	44	34	10	1

AIDS UPDATE

As of June 20, there have been 1,641 cases of AIDS reported in the United States. Seventeen of these cases have been reported from Connecticut. Since the March 1983 issue of the Connecticut Epidemiologist, many hotline numbers and publications have became available as good sources of information about AIDS. The U.S. Public Health Service is providing a hotline number and a new leaflet, called "FACTS ABOUT AIDS" (available at the Department of Health Services). The hotline number is 800-342-AIDS and will be open weekdays, 8:30-5:30.

AIDS: NATIONALLY VS. CONNECTICUT (as of June 20, 1983)

	Total # Cases	Deaths	Year of Diagnosis (Before) 1981	1981	1982	1983
United States	1641	644 (39%)	55 (3%)	224 (14%)	832 (51%)	529 (32%)
Connecticut	17	9 (53%)	0 (0%)	3 (18%)	7 (41%)	7 (41%)

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