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Swine Influenza A at Fort Dix, New Jersey (January–February 1976). I. Case Finding and Clinical Study of Cases

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After the isolation of A/New Jersey/76 (Hsw1N1) influenza virus from five soldiers at Fort Dix, New Jersey, case finding was initiated by obtaining specimens for viral isolation from 95 patients with acute respiratory disease and determining antibody to influenza A/Mayo Clinic/103/74 (Hsw1N1) antigen in paired sera from 74 soldiers who had been hospitalized with acute respiratory disease. Influenza A/New Jersey virus was not isolated, but serologic studies identified eight additional soldiers as A/New Jersey influenza patients. Development of heterotypic antibody to A/Mayo Clinic antigen following infection and/or immunization with influenza A (H3N2) strains was studied and was found to occur infrequently. One of the 13 identified patients had died, and postmortem findings were consistent with viral pneumonia. Four of the 12 surviving patients had radiologic evidence of pneumonia, but clinical syndromes in all 12 were similar to those described for other influenza A infections.

In January 1976 an increase in acute respiratory disease (ARD) occurred among soldiers at Fort Dix, N.J. As part of an epidemiologic investigation by the preventive medicine officer at the post, throat washings for the isolation of influenza virus were collected from 19 hospitalized patients with ARD on January 29 and 30. Specimens for viral isolation also were obtained at autopsy from a soldier who died with ARD on February 4. All specimens were sent to Dr. M. Goldfield of the New Jersey State Department of Health (Trenton), and influenza A/Victoria/75 (H3N2) virus was identified in throat washings from seven patients. Specimens from five others, including the individual who died, contained a different strain of influenza A. On February 13 this strain was characterized by the World Health Organization Collaborating Center for Influenza, Center for Disease Control (CDC; Atlanta, Ga.) as having hemagglutinin and neuraminidase antigens similar to those of swine influenza viruses and was designated influenza A/New Jersey/76 (Hsw1N1) virus [1–3]. The discovery of human infection with an influenza virus radically different from contemporary strains required an investigation to determine the type of illness caused by the A/New Jersey virus and the nature and extent of the outbreak. This paper details case finding efforts and the clinical study of cases.

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Materials and Methods

Case finding. Two case-finding methods were initiated in mid-February 1976. In the first, a prospective search for cases, throat washings

were collected from patients with febrile ARD (temperature, ≥101 F) during the periods February 14–16 and 22–24. Soldiers admitted to the Fort Dix hospital and military and civilian patients presenting at the hospital emergency room from 7 A.M. to 7 P.M. were studied. (During four of the six collection days, the emergency room was the only operating outpatient facility on the post.) A total of 95 throat washings were obtained: 60 from soldiers in basic combat training (BCT), 13 from other military personnel, and 22 from civilians. In addition, attempts were made to obtain acute- and convalescent-phase (two weeks) sera from all persons studied during February 14–16.

The second method, a retrospective search for cases, was conducted through the Adenovirus Surveillance Program [4], which monitored ARD in the BCT trainee population, a group that consisted of ~50% of all soldiers on the post. Each week pharyngeal swabs for viral isolation were obtained from a sample of trainees hospitalized for ARD. Acute and two-week convalescent sera also were obtained from those admitted before the fifth week of the seven-week BCT program. All specimens were sent to regional laboratories, and paired sera not depleted by routine studies were stored. The U.S. Army Medical Laboratory at Fort Meade, Md., received 80% of the paired sera collected from Fort Dix trainees hospitalized between November 1, 1975 and February 14, 1976 and forwarded sera from 74 trainees (all available specimens) to the Walter Reed Army Institute of Research (WRAIR; Washington, D.C.). Thirty-nine of these trainees had been admitted after January 1, 1976. The percentages of all trainee admissions for ARD from January 5 to February 14 that were studied serologically are shown in table 1. Total ARD admissions were obtained from the Fort Dix hospital admission log and include hospital admissions for acute pharyngitis, ARD, upper respiratory tract infection, influenza and influenza syndrome, pneumonia (unspecified), and viral pneumonia. (Admissions for otitis, sinusitis, specified bacterial pharyngitis, and specified bacterial pneumonia were not counted.)

In the study of this outbreak all HAI tests were performed by standard techniques using adult chicken cells in sera pretreated with periodate [5]. Initially, only inactivated influenza A/Mayo Clinic/103/74 (Hsw1N1) antigen, supplied by the CDC, was available. Later, smaller amounts of influenza A/New Jersey/8/76 (Hsw1N1) antigen were supplied. Sera from the prospective search for cases and the Adenovirus Surveillance Program sera were tested against A/Mayo Clinic antigen. Paired sera demonstrating a fourfold or greater rise in titer were further tested against A/New Jersey and A/Victoria/3/75 (H3N2) viral antigens. Attempts also were made to obtain sera from patients with influenza A/New Jersey isolates for determination of HAI titers to all three influenza antigens mentioned above.

Table 1. Hospital admissions studied serologically among soldiers in basic training with acute respiratory disease (ARD) and number of serum pairs with fourfold or greater rises in titer of HAI antibody to influenza A/Mayo Clinic/103/74 (Hsw1N1) viral antigen.

Period of hospital admission (1976)	Total no. of trainee ARD admissions	No. of serum pairs tested (percentage of total admissions)	No. of pairs with ≥4-fold rise in titer of antibody to A/Mayo Clinic antigen
January 5–17	105	6 (6)	0
January 18–31	417	22 (5)	6
February 1–14	317	11 (3)	2

NOTE. Almost all trainees were on holiday leave from December 21, 1975 to January 4, 1976.

Throat washings, obtained with Hanks' balanced salt solution containing 0.4% bovine plasma albumin, were frozen and shipped on dry ice to the WRAIR. Aliquots (0.1 ml) of each throat washing were inoculated into the amniotic and allantoic sacs of three nine- to 10-day-old embryonated eggs by a standard procedure [5] and were incubated for 72 hr. The presence of viruses in amniotic and allantoic fluids was sought by HA with chicken, guinea pig, and human O erythrocytes. At least one blind passage was made on specimens giving no detectable HA on initial passage. Isolates were identified in HAI tests using hyperimmune antisera to influenza A/Port Chalmers/1/73 (H3N2), A/Victoria/3/75 (H3N2), and A/Mayo Clinic/103/74 (Hsw1N1) viral antigens supplied by the CDC.

Heterotypic antibody development. The pos-

sibility that infection and/or immunization with influenza A (H3N2) strains might stimulate development of antibody to A/Mayo Clinic antigen was considered. HAI titers to A/Mayo Clinic antigen were determined in acute and two-week convalescent sera from patients who had been ill with ARD in January or February 1976 and had either an influenza A/Victoria isolate or a fourfold or greater rise in CF antibody to influenza A antigen. Patients with an A/Victoria isolate included 19 people from Fort Dix and 21 soldiers from other posts. Patients with a rise in CF antibody were identified through regional laboratories and consisted of 27 soldiers from three posts other than Fort Dix. Influenza A/Victoria virus had been isolated at all three of these posts in early 1976. Ages and histories of influenza immunization for these 67 people were not known. However, most members of each group were BCT trainees who were ill within approximately two weeks of their arrival at a training post. Since trainees routinely received influenza vaccine one to three days after arrival, these recently arrived trainees were probably exposed to influenza antigens both by natural infection and by immunization.

The effect of immunization with influenza vaccine was studied in a random sample of Fort Dix trainees who received Wyeth subunit vaccine (Wyeth Laboratories, Philadelphia, Pa.) in February 1976. This vaccine was the only influenza vaccine administered to Fort Dix BCT trainees from October 1975 through March 1976 and contained 350 chick cell-agglutinating (CCA) units each of influenza A/Port Chalmers/1/73 (H3N2) and A/Scotland/840/74 (H3N2) antigens and 500 CCA units of B/Hong Kong/5/72 antigen. Over a four-day period, 49 of 550 men were identified by Social Security number for the collection of sera before and five weeks after immunization. Thirty-nine soldiers, 26 of whom were 19 years old or younger, provided both specimens. HAI antibody titers to A/Mayo Clinic, B/Hong Kong/5/72, A/Port Chalmers/1/73 (H3N2), and A/Victoria viral antigens were determined.

Clinical and pathologic data. BCT trainees with ARD and a temperature of ≥100 F were norally hospitalized on special ARD wards adapted to the large case load generated by this admission policy. The routine work-up included complete blood count, throat culture for β-hemolytic streptococci, urinalysis, and posteroanterior chest X ray (14 × 17 inches). A check list of ARD symptoms was the basic history form in an abbreviated clinical record on these wards; paramedics, nurses, or physicians recorded symptoms. Physicians recorded the physical examinations, listing only abnormal findings. Medical records of all identified A/New Jersey influenza patients were reviewed, and independent consultants confirmed interpretations of chest X rays and histopathologic slides.

Results

Case finding. Fifty specimens for viral isolation were collected during February 14–16, and 45 were obtained during February 22–24. Influenza A/New Jersey virus was not isolated from any of the specimens. A/Victoria virus was isolated from the specimens of 34 people (68%) from the earlier period and 21 people (47%) from the later period. Only one of 34 people who provided paired sera developed a fourfold or greater rise in titer of antibody to influenza A/Mayo Clinic antigen, with an acute-phase titer of <1:10 increasing to 1:20. This patient was a 29-year-old soldier from whom A/Victoria virus was isolated. Her HAI antibody titer to A/New Jersey antigen went from <1:10 to 1:10, and her antibody titer to A/Victoria antigen remained unchanged at 1:20.

Eight of the 74 serum pairs tested in the retrospective search for cases (Adenovirus Surveillance Program) had a fourfold or greater rise in titer of antibody to A/Mayo Clinic antigen. All eight of these serum pairs were from trainees who had been hospitalized after January 17, 1976 (table 1). Influenza viruses had not been isolated from any of these eight patients by the regional laboratory, but specimen storage (ambient temperature in charcoal viral transport media) was not ideal for influenza viruses, and the isolation system did not include embryonated eggs. Serologic results for these patients (patients no. S1–S8) are given in table 2. Since several of them were admitted shortly after arrival at Fort Dix (and shortly after receiving influenza vaccine), titer rises to influenza A/Victoria antigen were expected. Patients no. S1–S4 and no. S7 all were

Table 2. Titers of HAI antibody to influenza antigens in patients with an A/New Jersey isolate (V1, V2, and V4) and patients identified retrospectively through the study of paired sera (S1-S8).

Patient no., date bled (1976)	HAI antibody titer to indicated antigen		
	A/Mayo Clinic (HswlN1)	A/New Jersey (HswlN1)	A/Victoria (H3N2)
V1, 2/17*	1:80	1:80	1:10
V2, 2/24	<1:10	<1:10	1:40
V4, 2/17	1:40	1:80	1:20
S1			
1/20	<1:10	<1:10	1:20
2/3*	1:40	1:40	1:40
S2			
1/20	<1:10	<1:10	<1:10
2/3*	1:40	1:40	1:40
S3			
1/21	<1:10	<1:10	1:20
2/4*	1:20	1:40	1:80
S4			
1/21	<1:10	<1:10	1:10
2/4*	1:40	1:40	1:20
S5			
1/28	<1:10	QNS†	QNS
2/11*	1:40	QNS	QNS
2/24	1:40	1:40	<1:10
S6			
1/28	<1:10	<1:10	1:80
2/11*	1:80	1:80	1:80
S7			
2/9	<1:10	<1:10	<1:10
2/23*	1:40	1:40	1:20
S8			
2/11	1:10	1:10	1:80
2/24*	1:40	1:40	1:40

*Approximate date; the exact date may differ by a few days.
†QNS = insufficient quantity of serum.

hospitalized eight days or less after their arrival on the post.

Also in table 2 are the serologic results for three of the five patients (patients no. V1–V5) with influenza A/New Jersey viral isolates. Serum was not available from patient no. V5 who had died, and attempts to obtain a specimen from patient no. V3 were unsuccessful. Patients no. V1 and V4 had antibody to both A/Mayo Clinic and A/New Jersey antigens. Patient no. V2 was reassigned prior to this investigation and had blood drawn at his new station. This serum had been erroneously sent to another laboratory and, after several weeks' delay, was located and forwarded to the WRAIR. Although the specimen was drawn 26 days after his admission, HAI antibody titers to both A/Mayo Clinic and A/New Jersey antigens were <1:10. This patient was discharged from the Army before another specimen could be obtained.

Table 3. HAI antibody rises to influenza A/Mayo Clinic/103/74 (HswlN1) viral antigen in people infected or immunized with influenza A (H3N2) strains.

Group	No. with ≥ 4-fold rise in titer of antibody to A/Mayo Clinic antigen/no. studied (%)
Patients with A/Victoria isolates (Fort Dix and other posts)	3/40 (8)
Patients with ≥ 4-fold rise in CF antibody to influenza A (posts other than Fort Dix)	0/27
Soldiers immunized with influenza vaccine (Fort Dix)	0/39

Heterotypic antibody development. Serologic results with influenza A/Mayo Clinic antigen are shown in table 3 for three groups with influenza A (H3N2) infection and/or recent immunization with influenza vaccine. Three (8%) of 40 patients with A/Victoria isolates developed a fourfold or greater rise in HAI titer. Two of these three individuals were ill in their first week of BCT at posts other than Fort Dix. None of 27 patients with influenza diagnosed by serology alone and none of 39 trainees immunized with influenza vaccine developed a fourfold or greater rise. HAI antibody titers to B/Hong Kong, A/Port Chalmers, and A/Victoria antigens in the immunized trainees are summarized in table 4.

Clinical and pathologic data. All 13 patients with A/New Jersey influenza were male enlisted soldiers aged 17–21 years. Selected clinical data for the 12 surviving patients are summarized in table 5. Patient no. V5, the individual who died, visited the outpatient clinic on February 3 complaining of "a cold." He was thought to have an upper respiratory tract infection and was admitted to quarters for 48 hr. However, on February 4 he participated in a training march, during which he became severely ill. He was transported to the emergency room where he was pronounced dead after an unsuccessful attempt at resuscitation.

An autopsy was performed the following day. On gross examination both lungs were firm and red. The right lung weighed 900 g, and large

Table 4. HAI antibody responses to influenza antigens in basic combat trainees immunized with Wyeth subunit influenza vaccine.

Antigen	Before immunization (n = 39)		After immunization (n = 39)		
	No. with titer of ≥1:10	Geometric mean of titers ≥1:10	No. with titer of ≥1:10	Geometric mean of titers ≥1:10	No. developing ≥4-fold titer rise (%)
B/Hong Kong	39	1:67	39	1:181	19 (49)
A/Port Chalmers (H3N2)	39	1:41	39	1:81	14 (36)
A/Victoria (H3N2)	28	1:19	38	1:52	23 (59)
A/Mayo Clinic (HswlN1)	0	...	0	...	0

quantities of serosanguinous fluid drained from the parenchyma when it was sectioned. The left lung weighed 500 g and presented a similar gross appearance. All bronchi and arteries were patent. The liver weighed 2,350 g with a tight capsule and rounded edge. The parenchyma showed evidence of moderate congestion. The spleen weighed 325 g and was firm and heavily congested. The other organs were unremarkable.

Microscopic examination revealed the following pertinent information. Multiple sections of the trachea and both lungs showed widespread hemorrhage and pulmonary edema. There was a heavy mononuclear cell infiltrate throughout most of the sections of the lung, and the alveolar septae were congested and edematous with infiltration of mononuclear cells. In some areas there was evidence of hyperaeration in the form of rupture of alveolar septae. Some small bronchi were occluded by mononuclear cells in a serosanguinous fluid medium. The mucosa of the trachea and larger bronchi were infiltrated with mononuclear cells, and there were metaplastic changes of the tracheal mucosa. Some of the smaller arterioles were noticeably thickened. Throughout the lung there were very few polymorphonuclear leukocytes in the inflammatory infiltrates. The heart was essentially unremarkable.

Table 5. Selected clinical data for 12 surviving influenza A/New Jersey/76 patients.

Clinical sign	Patient no.											
	V1	V2	V3	V4	S1	S2	S3	S4	S5	S6	S7	S8
Admission temperature (F)	100.2	101.2	100.8	101.6	102.4	100.2	103.8	102.6	100.8	102.4	100.0	100.6
Leukocyte count ($\times 10^3$/mm^3)	6.4	15.4	6.0	10.0	7.0	7.3	7.5	5.5	16.4	9.6	15.0	13.4
Chest X ray*	NSA	RML	NSA	NSA	NSA	NSA	NSA	RLL	LLL	NSA	LLL†	NSA
β-hemolytic streptococci (group A) in throat culture	–	+	–	+	–	–	–	–	+	–	–	+
Days hospitalized	1	4	1	3	4	1	3	3	4	3	6	3
Symptoms recorded on admission												
Fever	+	+	+	+	+	+	+	+	+	+	+	+
Chills	+	+	+	+	+	+	+	+	+	+	–	+
Rhinitis	+	+	+	+	+	+	+	+	+	+	+	+
Sore throat	+	+	+	+	+	+	+	+	+	+	+	–
Myalgia	+	+	+	+	+	+	–	+	–	–	+	+
Cough	–	+	+	+	+	+	+	+	–	+	+	+
Productive cough	–	+	+	+	–	+	–	–	–	–	+	+
Chest pain	+	+	+	+	+	+	–	+	+	–	–	+
Headache	+	–	+	+	+	–	+	–	–	+	+	+
Vomiting	–	–	–	+	–	+	–	–	+	+	+	–
Diarrhea	+	–	–	–	–	–	–	–	–	–	–	–

NOTE. + = present; – = absent.

*NSA = no significant abnormalities, RML = right middle lobe pneumonia, RLL = right lower lobe pneumonia, and LLL = left lower lobe pneumonia.

†*Haemophilus influenzae* in sputum culture.

The liver was edematous with noticeable edema fluid between the hepatic cords and widening of the hepatic sinusoids. There were a few mononuclear cells in the portal areas. The spleen was heavily congested with red blood cells as were two small accessory spleens. Influenza A/New Jersey virus was cultured from the nasopharynx, trachea, and lung tissue by two independent laboratories. Cultures of lung, blood, and cerebrospinal fluid were negative for bacteria.

Discussion

A novel strain of influenza A virus, A/New Jersey/76 (Hsw1N1), was isolated from five soldiers admitted to the Fort Dix hospital from January 29 to February 4. Immediately after identification of this strain on February 13, a prospective search for new cases among patients with ARD was initiated, but none were found. Two additional case-finding methods also were considered: (*1*) surveillance of contacts of patients with influenza A/New Jersey viral isolates and study of those developing ARD; and (*2*) the retrospective identification of cases through the study of paired sera from the Adenovirus Surveillance Program [4]. The former approach was considered impractical because of the long period of time separating the illnesses of the identified patients and the start of this investigation, the large number of potential contacts for each BCT trainee (~200), and the diversity of respiratory tract pathogens prevalent on BCT posts in winter months. For example, in late 1975–early 1976 influenza A/Victoria virus, adenovirus types 4, 7, and 21, and coxsackievirus A21 were isolated from Fort Dix soldiers, in addition to the A/New Jersey influenza virus. The second method, the determination of antibody to A/Mayo Clinic antigen in Adenovirus Surveillance Program sera, was undertaken and identified eight additional A/New Jersey influenza patients.

Development of heterotypic A/Mayo Clinic antibody after exposure to influenza A (H3N2) strains must occur infrequently in order for serologic studies alone to be an acceptable method of case identification. There was no increase in A/Mayo Clinic antibody in any of 39 soldiers who received subunit influenza vaccine (table 3). This sample was small, but data reported by Hodder et al. [6] are consistent with a very low prevalance of heterotypic antibody in BCT trainees after influenza immunization. In that study titers of antibody to A/Mayo Clinic antigen were determined in 168 Fort Dix BCT trainees randomly selected from soldiers in their third or fourth week of training (those that began training on February 2 or 9, 1976). Since essentially all trainees received influenza vaccine before the first week of training, the prevalence of titers of antibody to A/Mayo Clinic antigen of $\geq$1:20 in this group should reflect heterotypic antibody development resulting from immunization with influenza A (H3N2) antigens. Only four (2%) had titers of $\geq$1:20. Soldiers studied by Hodder et al. [6] were 17–35 years of age, but most were younger than 20 years.

People immunized with H3N2 influenza strains who developed at least a fourfold increase in titer of antibody to influenza A (H3N2) antigen were studied at the CDC [7] and by Masurel [8]. In the CDC studies $\leq$6% of persons aged 18–34 years developed a fourfold or greater rise in titer of antibody to influenza A/swine antigen. In the latter study at least a fourfold rise in heterotypic antibody titer was observed in 10 (4%) of 277 persons 20–29 years old. Masurel did not observe heterotypic influenza A/swine antibody responses in any of 69 persons $\leq$19 years old, regardless of their response to the vaccine antigens [8].

Three (8%) of 40 patients with influenza A/Victoria isolates had a fourfold or greater rise in titer of antibody to A/Mayo Clinic antigen, but none of 27 soldiers with serologic evidence of influenza A virus infection developed fourfold increases in A/Mayo Clinic antibody (table 3). Similar studies of patients with influenza A (H3N2) infections, confirmed by isolation of the virus and/or serologic tests, were done by Noble et al. [7] and by Masurel [8]. In the former study 2% of 89 individuals 13–34 years of age had a fourfold or greater rise in antibody to influenza A/swine antigen. In Masurel's study three (4%) of 67 people developed fourfold or greater increases in heterotypic antibody. Therefore, rises in heterotypic influenza A/swine antibody occur infrequently in young adults immunized or infected with influenza A (H3N2) strains.

Although serology is more tenuous than viral isolation for case identification, the eight patients identified by serology alone all were hospitalized near the time patients with isolates of A/New

Jersey were admitted. Considering the clustering of these patients in time and the expected infrequent development of heterotypic antibody to A/Mayo Clinic antigen, it is probable that most, if not all, of these patients were infected with influenza A/New Jersey virus.

Serum specimens were obtained from three individuals with A/New Jersey isolates (table 2). HAI antibody titers to A/Mayo Clinic and A/New Jersey antigens for two of these three were of the same order of magnitude as the convalescent-phase titers observed for patients identified by serology alone. One patient lacked antibody to both the A/Mayo Clinic and A/New Jersey antigens. Although failure to detect an increase in HAI antibody titer following infection with influenza A/New Jersey virus has been reported [9], this patient's specimen could have been exposed to adverse environmental conditions.

Clinical study of the 12 surviving patients was restricted to the review of X rays and medical records since several of these patients could not be interviewed and the earliest a patient could be interviewed was almost three weeks after the onset of his illness. Although four had radiologic evidence of pneumonia, the clinical syndromes of these 12 patients were similar to those described for patients infected with other influenza A strains [10]. Caution should be exercised in attempts to draw further conclusions from these data. The sample was small; ARD wards were busy; records were abbreviated; histories were not obtained in a uniform manner; and antipyretic use prior to admission was not known. The autopsy findings of the single fatal case provide some insight into the pathogenicity of the influenza A/New Jersey virus since there was no complicating bacterial infection and the patient had no known preexisting disease. The severe edema, hemorrhage, and mononuclear infiltrates observed on postmortem examination of the lungs are consistent with viral pneumonia.

In summary, 13 cases of A/New Jersey/76 (Hsw1N1) influenza were identified at Fort Dix. Five patients had isolates of A/New Jersey virus. In this study, eight others were identified serologically. All 13 had been ill with ARD.

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After the isolation of A/New Jersey/76 (HswlN1) influenza virus from five soldiers at Ft Dix, New Jersey, case finding was initiated by obtaining specimens for viral isolation from 95 patients with acute respiratory disease and determining antibody to influenza A/Mayo Clinic/103/74 (HswlN1) antigen in paired sera from 74 soldiers who had been hospitalized with acute respiratory disease. Influenza A/New Jersey virus was not isolated, but serologic studies identified eight additional soldiers at A/New Jersey influenza patients. Development of heterotypic antibody to A/Mayo Clinic antigen following infection and/or immunization with

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influenza A(H3N2) strains was studied and was found to occur infrequently. One of the 13 identified patients had died and postmortem findings were consistent with viral pneumonia. For of the 12 surviving patients had radiologic evidence of pneumonia, but clinical syndromes in all 12 were similar to those described for other influenza A infections.