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Laboratory and clinical investigation of the 1974 influenza epidemic in Nigeria*

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Abstract

Altogether 13 strains of virus were isolated during the 1974 influenza epidemic in Nigeria. These A/Nigeria/1/74 strains were shown by haemagglutination-inhibition tests to be closely related to the A/Port Chalmers/1/73 virus. Antibody to the epidemic strains developed rapidly in the population and 80–95% of all age groups tested possessed high antibody levels; of 145 paired sera tested, 133 (92%) showed seroconversion to A/Nigeria/1/74 virus.

Outbreaks of influenza and other respiratory infections have been diagnosed clinically in Nigeria, the yearly periodicity of the major outbreaks coinciding with the onset of the Harmattan season (i.e., when cold winds and dust blow south from across the Sahara desert). These outbreaks start about November and fade out in February. Although there are so far no statistics on morbidity and mortality, it is suspected that these outbreaks claim many lives and many more people are usually unable to work. Even clinicians and medical scientists were alarmed by the extent and severity of the 1974 outbreak.

Despite the annual outbreaks, there has been no project to study influenza in Nigeria; indeed, the status of annually-recurring influenza in the tropical regions of Africa has not been clearly delineated. We report here our findings during the 1974 influenza outbreak in Nigeria.

Materials and methods

Collection of specimens. Specimens were collected from febrile patients reporting at the Outpatients Department; initially throat swabs were taken, but because most patients disliked this, nasal secretions were collected in the later parts of the study. The patients were made to pass their nasal phlegm into sterile containers. These were treated with an antibiotic solution containing 200 units/ml of penicillin and 200 μg/ml of streptomycin, and then stored at −20°C until used. Paired sera were also collected from some of the patients.

Virus isolation. Antibiotic-treated phlegm or throat swab extracts were inoculated by the amniotic route into 10-day-old embryonated eggs, each egg receiving 0.1 ml. The eggs were incubated at 37°C for 48 h, after which the infected amniotic fluid was harvested and tested for virus. Routine second and, in some cases, even third amniotic inoculations were carried out, before the positive materials were inoculated into the allantoic sac or before discontinuation of the isolation attempts.

Haemagglutination (HA) test. This test was carried out as reported previously (1), but with physiological saline as the diluent instead of Tris-saline.

Haemagglutination-inhibition (HI) test. The procedure was as described before (1). All test sera were first inactivated at 56°C for 30 min and then treated with 3 volumes of 0.111... mol/l of potassium periodate in saline for 15 min at 4°C; the reaction was stopped thereafter with one volume of glucose solution (100 g/l in physiological saline). Four HA units of virus were used in the test.

For the identification of the Nigerian isolates, reference antisera from the World Health Organization influenza kit, as well as antisera supplied by the WHO World Influenza Centre, London, were used.

Results

Identification of isolates. Altogether 13 haemagglutinating viruses were isolated, but 4 of these were lost in subsequent processing. The results of HI tests on the remaining 9 viruses, with reference antisera, are shown in Table 1. It can be seen that higher HI titres were obtained with the antisera to A/England/42/72 and A/Port Chalmers/1/73

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Table 1. Identification of the 1974 Nigerian influenzavirus isolates by HI tests with 5 different reference antisera

Lab. no. of isolate	A/HK/1/68	A/Eng./42/72	A/Pt. Chalm./1/73 [a]	A/Eng./42/72-eq./Prag./1/56	B/HK/5/72
7	40	320	640	320	< 10
44	20	160	320	160	< 10
53	40	320	320	160	< 10
76	160	320	640	80	< 10
77	80	320	640	160	< 10
85	40	640	1 280	160	< 10
86	80	640	640	160	< 10
89	40	320	320	160	< 10
91	40	160	640	320	< 10

[a] Tests at the WHO World Influenza Centre, London, demonstrated closer relation to A/Pt. Chalm./1/73 than to A/Eng./42/72 virus.

strains. Tests carried out at the WHO World Influenza Centre, London, showed a closer relationship of the Nigerian isolates (A/Nigeria/1/74) with A/Port Chalmers/1/73 virus than with A/England/42/72 virus (G. C. Schild, personal communication, 1974).

Seroconversion in human sera to A/Nigeria/1/74. Of 145 paired sera (early and late) collected during the outbreak, 133 (92.0%) showed significant seroconversion to the virus.

Age distribution of antibodies to A/Nigeria/1/74. There was no age-dependence in the distribution of antibodies, 80–95% of sera in all age groups having HI titres of $\geqslant 40$ (Table 2). One of the older patients studied, aged 48, died after a few days of illness; the only serum sample collected from him had an HI titre of 1/32 and a virus resembling denguevirus was isolated from this serum. This case will be reported in another paper.

Table 2. Antibody to A/Nigeria/1/74 virus in sera of different age groups

Age group (years)	Total no. of sera	No. of sera with HI titre of	
		< 40	$\geqslant 40$
0–9	159	17 (11.0) [a]	142 (89.0)
10–19	10	2 (20.0)	8 (80.0)
20–29	42	4 (10.0)	38 (90.0)
30–39	21	1 (5.0)	20 (95.0)
$\geqslant 40$	9	1 (11.0)	8 (89.0)
total	241	25 (10.0)	216 (90.0)

[a] Figures in parentheses are percentages.

Clinical picture. There was generally a sudden, but sometimes a slow, onset of malaise, headache, shivering, cough, and catarrh with pyrexia ranging from 37.2°C to 39.4°C. Headache, sore throat, and generalized body pains, especially in the back and joints, were a frequent complaint. Nausea, anorexia, and sleeplessness were sometimes complained of, as were pain behind the eyes, vomiting, and diarrhoea. Physical examination showed an ill-looking, shivering patient with a streaming nose, watery eyes, and pyrexia, but with no other localized signs. In two patients there was herpes labialis. The illness lasted a few days, and the majority of the patients were back at work after 7–10 days. Post-influenzal lethargy and debility were not pronounced features.

Discussion

In terms of comparative epidemiological patterns, it appears that " winter influenza " in the temperate regions of the world coincides with the " Harmattan influenza " of tropical West Africa, and " summer influenza " of the temperate regions coincides with the " wet season influenza " of West Africa. In our experience, more severe and extensive outbreaks of influenza and respiratory illnesses

occur during the Harmattan seasons than during the rainy seasons.

Although in our tests there was considerable cross-reaction between the A/Nigeria/1/74 virus and A/England/42/72 as well as A/Port Chalmers/1/73 viruses, the tests carried out in London (G. C. Schild, personal communication, 1974) showed the Nigerian isolates to be more closely related to A/Port Chalmers/1/73 virus, which represents the strains isolated in Australasia in September and October 1973 (3). This strain was also isolated in 1974 in Britain (G. C. Schild, personal communication). It is therefore of global epidemiologic interest that A/Nigeria/1/74 virus is closely related to A/Port Chalmers/1/73 virus. It should be mentioned that all our isolations were from Nigerian patients, who to the best of our knowledge had not travelled outside Nigeria during the year.

Antibody to A/Nigeria/1/74 virus was readily induced in the population during the epidemic, and between 80% and 95% of all age groups tested possessed a protective antibody level of at least 1/40 (Miller et al., as cited in (2)). Of the 145 paired sera that were collected, 133 (92%) demonstrated seroconversion to A/Nigeria/1/74 virus.

The rather unorthodox method of collecting nasal secretions was less objectionable to our patients than conventional throat swabbing, and it was found that the frequency of virus isolation was higher from nasal secretions than from throat swabs.

No influenzavirus was isolated during the milder, rainy season outbreak, although influenza-like infection of the upper respiratory tract was undoubtedly diagnosed clinically. It is conceivable that " wet season influenza " might be caused mostly by B strain viruses, which are more difficult to isolate by the methods we routinely employ. We are now conducting serologic tests for retrospective identification of the " wet season " specimens, using an influenza B seed from the WHO influenza kit.

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