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Virological and Serological Studies of Venezuelan Equine Encephalomyelitis in Humans

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During the 1971 epidemic of Venezuelan equine encephalomyelitis (VEE) in south Texas, 203 suspect VEE cases were evaluated by the Center for Disease Control. Sixty-seven were confirmed as cases of VEE. Laboratory confirmation was accomplished by isolation of VEE virus from a serum specimen taken during the acute illness in 50 (75%) of the confirmed cases. Serological confirmation was obtained in 17 cases (25%). Virus isolations were most often obtained from sera collected during the first 3 days of illness. Peak serum virus titers (algebraic mean, $10^{5.7}$ suckling mouse intracranial 50% lethal doses [$SMICLD_{50}$] per ml) occurred on day 2 of illness. One-half of the sera from which virus was isolated contained at least 10^5 $SMICLD_{50}$/ml, which has been shown to be sufficient to infect some vector mosquitoes. Blood from 13 virus-positive VEE cases was obtained 1 and 11 months after illness. Hemagglutination-inhibiting, complement-fixing, and neutralizing antibodies were formed by all 13 patients 1 month after illness. Hemagglutination-inhibiting antibody titers were essentially unchanged 11 months after illness. Complement-fixing antibody was undetectable 11 months after illness in 23% of cases and was detectable at dilutions of 1:8 or 1:16 in 77%. Neutralizing antibody (measured by log neutralization index) was not detectable 1 year after illness in one person (8%); titers had declined from 1.0 to 2.0 in 46%, were unchanged in 39%, and were not tested in one person (8%). No evidence of intrafamilial spread of VEE virus was obtained in either of two illness and antibody surveys. A randomized household illness and antibody survey of 681 Port Isabel residents revealed an inapparent infection ratio of 1:11 and an overall antibody prevalence of 3.2%.

South Texas experienced an epidemic of Venezuelan equine encephalomyelitis (VEE) during the summer of 1971 (23). Eighty-eight laboratory-confirmed human cases were reported to the Center for Disease Control (CDC) by the Texas State Health Department (6). An additional 22 persons are known to have acquired antibody (G. S. Bowen, T. Fashinell, P. Dean, and M. Gregg, *Clinical Aspects of Human Venezuelan Equine Encephalitis in Texas, 1971*, Pan American Health Organization Bulletin, in press) but were never officially reported by the Texas State Health Department. This is the only occasion on which VEE has occurred in epidemic form in the United States. It is the purpose of this paper to report the virological and serological results obtained by testing sera collected during the acute illnesses, 1 month later, and 11 months after the epidemic and to discuss some of the implications of the results.

MATERIALS AND METHODS

Detection of cases. All cases here reported became ill while residing or visiting in the lower Rio Grande Valley counties of Cameron and Hidalgo or in the counties near Corpus Christi (Nueces, Kleberg, San Patricio, Aransas). Cases were discovered as a result of an efficient hospital-based surveillance system (Bowen et al., in press). All suspect cases had clinical syndromes compatible with VEE virus infection, were reported to surveillance system personnel, and had blood collected for testing. Only persons from whom detailed clinical information was collected and reported by their physician, or who were examined and followed by one of us, are included in this report. The cases in this report all were confirmed in the laboratory by virus isolation from serum drawn during the acute stage of illness. The clinical signs and symptoms of these 40 cases fell into three syndromes: (i) encephalitis (25%); (ii) aseptic meningitis (10%); and (iii) febrile headache with myalgia (65%). No correlation between virus titer or serological response and clinical syndrome could be made.

Collection and processing of specimens. During the peak epidemic period in Cameron and Hidalgo counties, blood specimens were collected in physicians' offices, emergency rooms, and from inpatients by hospital laboratory personnel. These specimens were taken to hospital laboratories and held at 4°C

until collected daily and transferred to a single receiving laboratory. At the receiving laboratory, VEE-vaccinated personnel documented the specimens, centrifuged them, removed the clots, and transferred the sera to 1- or 2-dram screw-cap vials for shipment on dry ice to the CDC, Atlanta.

Sera were obtained from suspect and confirmed VEE patients 1 month after illness by their attending physicians, by hospital laboratory personnel, and during home visits by public health personnel. Follow-up serum specimens were obtained 11 months after illness by home visits to VEE cases who could still be located. Only 13 VEE cases who were originally virus positive and from whom a serum specimen had been obtained 1 month after illness could be found 11 months after illness. The large number of nonresidents and transients among the VEE cases prevented the finding of more cases.

Laboratory procedures. The procedures for virus isolation, identification, and characterization and for antibody testing have been described in detail by Calisher and Maness (3).

Kaolin-adsorbed sera were tested for hemagglutination-inhibiting (HI) antibody by a microtiter adaptation of the method of Clarke and Casals (7). Complement fixation (CF) was performed in the microtiter system by the Laboratory Branch Complement Fixation method of Casey (5). All human sera were tested in HI and CF tests for antibody to VEE, Eastern equine encephalomyelitis, Western equine encephalomyelitis, St. Louis encephalitis, and California (LaCrosse) encephalitis viruses. Sera were also tested in weaned mice for neutralizing (N) antibody against an epizootic strain of VEE virus from Guatemala (strain GJ9 1BJ). A constant serum-virus dilution technique was used, and results, in terms of neutralizing capacity, were recorded as log neutralization indexes.

Antibody and illness survey of family contacts and neighbors in Brownsville. Serum specimens from 32 family members and 67 neighbors of 13 VEE cases residing in Brownsville, Cameron County, Tex., were obtained 1 month after the outbreak. Illness histories were taken with each serum specimen. The sera were examined for presence of VEE antibody to determine whether or not intrafamilial spread of virus and/or asymptomatic cases had occurred.

Port Isabel antibody and illness survey. Six weeks after the epidemic, P. Dean, T. Fashinell, L. Schoenberger, R. Baskin, and M. Gregg of the Epidemic Intelligence Service, Bureau of Epidemiology, CDC, conducted an illness and antibody survey of residents of Port Isabel, Cameron County, Tex. This town was selected because many VEE cases were Port Isabel residents or were people vacationing in or near Port Isabel. The town was divided into five geographical areas, and a random sample of households was surveyed in each area. Serum specimens and illness histories were obtained from all family members above the age of 3 years when parental consent was obtained.

RESULTS

Laboratory confirmation of suspect cases. Serum specimens from 203 persons suspected of having VEE were sent to the CDC for evaluation. VEE virus was isolated from the serum of 50 of these patients, making rapid laboratory confirmation of most cases possible. An additional 17 cases were later confirmed to have VEE by fourfold rises in HI, CF, of N antibody titer. Of the 50 virus-positive cases, only 40 are included in the section on virological results, because information on duration of illness prior to the collection of the serum and clinical data were lacking. Only 13 cases were included in the section on serological results (Table 3), because these were the only cases that were virus positive during illness and from whom follow-up serum specimens were obtained both 1 month and 11 months after illness.

Virus isolations. Table 1 shows the number of virus isolations made from human sera by the day after onset of symptoms and the virus titer in these sera. Thirty-three of the 40 virus isolations (83%) were made from sera drawn the day of onset of symptoms (day 1) or during the next 2 days. Isolations were made, however, as late as 8 days after onset of symptoms.

Peak titers occurred the day after onset of symptoms (day 2). The mean titer of isolates from this day was $10^{5.7}$ suckling mouse intracranial 50% lethal doses ($SMICLD_{50}$)ml. Mean titers on days 1 and 3 were $10^{4.6}$ and $10^{3.8}$ $SMICLD_{50}$/ml, respectively. Isolations were made from sera of two patients on two different occasions (days 2 and 7 and days 4 and 6). It is certain, therefore, that VEE viremia in humans can last at least 6 days.

Viremia titers of all acute serum specimens irrespective of day after onset are shown in Table 2. Sixteen (40%) of the 40 sera tested contained only $10^{3.2}$ $SMICLD_{50}$ or less of VEE virus per ml; 2 contained $10^{3.3}$ to $10^{5.0}$ and 22 (55%) of the sera contained at least $10^{5.1}$ $SMICLD_{50}$/ml. The highest titer recorded was $10^{8.2}$ $SMICLD_{50}$/ml detected in two sera from different patients.

Serology. Serological results of 13 virus-posi-

TABLE 1. *Titers of VEE virus in blood of 40 patients (listed by day of symptoms)*

Day	No. of cases	Range of virus titers ($SMICLD_{50}$/ml)	Mean titer
1	11	2.7–7.0	4.6
2	12	2.7–8.2	5.7
3	10	<2.0–6.2	3.8
4	3	4.8–6.2	5.3
5	0		
6	2	<2.0–2.7	<2.3
7	1	2.5	
8	1	<2.0	
>8	0		

tive VEE patients whose sera were drawn during the acute illness, 1 month after illness, and again 11 months after illness are shown in Table 3. HI antibody was demonstrated 1 month after illness in all 13 patients tested. Titers ranged from 1:40 to 1:320. HI titers were essentially unchanged at 11 months.

In all cases, CF antibody was present 1 month after illness at serum dilutions of 1:16 to 1:64. One year after onset of illness, CF antibody was undetectable in three persons (23%); eight persons (62%) had titers of 1:8; and two others (15%) had titers of 1:16.

N antibody was detected in sera of all patients drawn 1 month after illness. Log neutralization indexes ranged from 2.1 to ≥4.8. Eleven months after illness N antibody was undetectable in one person (8%) and the log neutralization index had declined from 1.0 to 2.0 in seven persons (54%). N antibody was unchanged in four persons (31%) and was not tested at 1 month in one person because of a laboratory accident.

Antibody and illness surveys. Sera from 32 family members and 67 neighbors of 13 Brownsville VEE cases were serologically tested for antibody to VEE virus. Only 2 of these 99 persons (2%) had VEE N antibody. Neither was a family member of a case. Both reported an illness resembling that of VEE during the epidemic period.

During the antibody and illness survey conducted in Port Isabel, Tex., 681 residents out of an estimated 4,000 total population were sampled. Twenty-two of the 681 sera tested (3.2%) had VEE antibody. All seropositive individuals came from different families. Twenty of the 22 seropositive persons gave a history of VEE-compatible illness during the epidemic period. An inapparent infection ratio of 1:11 is calculated for these 22 infections. Thus, in both the Brownsville and the Port Isabel surveys, no evidence of intrafamilial spread of VEE virus was found. Likewise, very few asymptomatic infections were found during either survey.

Infections with other agents. All patients were evaluated for evidence of infection by Eastern equine encephalitis, Western equine encephalitis, St. Louis encephalitis, and California encephalitis viruses. No infections with these agents were identified. Fifteen serum pairs from children who had symptoms resembling those of VEE, but had no evidence of togavirus infection, were tested by a microneutralization technique for evidence of seroconversion to Coxsackie B1-6 viruses. Forty serum pairs from clinically ill persons who had no evidence of togavirus infection were tested by a microagglutination technique against a large battery of leptospiral antigens. Seroconversion

TABLE 2. *Titers of VEE virus in acute blood specimens of 40 naturally acquired human infections*

Virus titer ($SMICLD_{50}$/ml)	No. of patients	% of total
≤3.2	16	40.0
3.3-5.0	2	5.0
5.1-6.0	11	27.5
6.1-7.0	8	20.0
7.1-8.0	1	2.5
8.2	2	5.0

TABLE 3. *Results of virus isolation and serial serological tests on serum specimens from 13 patients, Texas 1971*

Patient	Age (yr)	Sex	Virus isolation		Serological results								
			Day of illness	Titer ($SMICLD_{50}$/ml)	0-7 Days			1 Month			11 Months		
					HI	CF	LNI[a]	HI	CF	LNI	HI	CF	LNI
J.Y.	6	F	2	5.2	<10	<8	0	160	16	4.0	160	16	3.6
M.T.	12	F	1	2.7	<10	<8	0	80	16	2.5	160	16	3.3
C.G.	14	F	1	5.2	<10	<8	0	80	64	3.5	320	8	2.3
E.G.	20	F	1	5.2	<10	<8	0	80	32	4.5	80	<8	2.8
J.R.	23	M	3	5.1	<10	<8	0	40	16	4.6	80	8	2.5
E.S.	29	M	2	6.2	<10	<8	0	160	32	3.3	80	8	2.1
V.H.	31	F	3	Pos NT[b]	<10	<8	0	80	16	NT[b]	160	<8	3.1
L.H.	42	F	4	4.8	<10	<8	0						
			6	2.3	<10	<8	0	80	32	2.1	80	8	2.7
C.B.	44	F	3	2.7	<10	<8	0	320	64	≥4.3	160	8	2.4
A.G.	48	F	2	6.5	<10	<8	0	80	32	≥4.8	40	8	1.8
A.G.	52	M	2	5.3	<10	<8	0	80	16	3.5	40	8	3.1
C.S.	53	F	2	5.2	<10	<8	0	80	64	>4.8	160	8	2.8
V.G.	3	M	3	2.3	20	<8	0	160	32	4.0	160	8	0

[a] LNI, Log neutralization index.
[b] Pos NT, Virus positive, titer not determined.

to Coxsackie B3 was demonstrated in 1 of the 15 (7%) serum pairs tested. Seroconversion to Coxsackie B4 was shown in a second pair (7%). Seroconversion to leptospiral antigens was shown in 2 of the 40 (5%) serum pairs tested. It is clear that the clinical syndrome of VEE is quite nonspecific and that infections with other agents can produce a very similar clinical picture.

DISCUSSION

Table 1 shows that virus isolations were made as late as 8 days after onset of symptoms, but that the vast majority (82.5%) were made from sera drawn the day of onset or in the 2 succeeding days. These results conform closely to those of Briceno-Rossi (1) and Sanmartin (18), who also found a greatly decreased incidence of viremia after day 3 of illness. These results suggest that isolation attempts could reasonably be limited to blood specimens obtained during the first 3 days of illness, especially during an epidemic, when rushed conditions prevail, and in laboratories where space or animals are limited.

Virus titers were determined for 40 of the virus-positive serum specimens by titration in suckling mice. More than one-half of the sera contained at least $10^{5.0}$ $SMICLD_{50}$/ml; however, 40% of the sera contained only $10^{3.2}$ $SMICLD_{50}$ or less per ml (Table 2). Virus titers determined here are intermediate compared to those detailed in other published reports. Briceno-Rossi (1) found VEE viremia titers of patients to be uniformly in excess of $10^{5.7}$ $SMICLD_{50}$/ml. However, he did not specify on which day of illness the blood specimens were collected. The titers of the isolates of Scherer et al. (20) ranged from 10^3 to 10^5 $SMICLD_{50}$/ml. Sanmartin (18) and Johnson et al. (15) reported titers below $10^{4.2}$ $SMICLD_{50}$/ml in the sera they tested. Differences in virus titers reported by several authors in widely spaced outbreaks can most likely be accounted for by (i) variability in the day of illness when sera were collected, (ii) time elapsed between collection of blood specimen and storage of sera in a low-temperature freezer, (iii) differences in virus strains, and (iv) differences in host response due to factors such as genetics, nutritional status, presence of other illnesses, etc.

The fact that over one-half of the human sera from which virus was isolated contained at least 10^5 $SMICLD_{50}$/ml is significant, because this is the level that has been shown to be sufficient to infect some vector mosquitoes (26) including *Psorophora confinnis*, which was one of the main VEE vectors during the 1971 Texas outbreak (25). Other important vectors during the 1971 outbreak included *Aedes sollicitans*, *Aedes thelcter*, and *Psorophora discolor* (25). *Aedes sollicitans* (4, 16), *Psorophora confinnis* (14, 16), and *P. discolor* (14, 16) are known to readily bite humans, and *A. thelcter* does so at least occasionally (16). Therefore, they could become infected with VEE virus if they fed upon a viremic person.

There are several inferences to be drawn from the fact that approximately one-half of the human sera contained enough virus to infect at least some vector mosquitoes. First, viremic humans could move virus from an epidemic area to a virgin area. Although horse movements might be restricted by quarantine measures, human movements would not be so limited. With the speed and ease of modern travel, epidemic VEE virus could be moved great distances very quickly. Second, where equines are immune, present in only small numbers, or absent, man-mosquito-man cycles could account for small outbreaks. If virus were introduced into an area by wild vertebrates, equines, or humans, a perpetuating cycle could be established with man as the source of infection for mosquitoes in the absence of susceptible equines. Third, amplification of previously established equine-associated epidemics could occur. New mosquitoes might be infected from human blood meals after the number of susceptible horses had been decreased by natural infection and vaccination, thus prolonging an outbreak.

Serosurveys in humans conducted 1 year or more after a VEE epidemic should use the HI or N test because a significant number of persons may have no detectable CF titer at that time. In our study, CF titers 1 year after illness were undetectable in 23% of patients. HI titers persisted unchanged from the 1-month to the 11-month bleedings. These results contrast with those of Sellers et al. (21), who found 4- to 32-fold declines in HI titers in eight of nine serum pairs obtained 2 and 14 months after virus isolation. No ready explanation is available for this difference in results, unless differences between virus strains or genetic differences between hosts were enough to produce a different host immunological response.

N antibody persistence after VEE infection appears to vary from individual to individual. One person had no detectable N antibody 11 months after infection. Sellers et al. (21) also found no detectable N antibody in 4 of 12 persons tested 14 months after illness. Thus, the present data indicate that the HI test can be used as well as or better than the neutralization test in serosurveys to detect past VEE activity in humans.

One interesting aspect of the Texas outbreak was the very low rate of asymptomatic infections. This is in marked contrast to published reports concerning the incidence of infection with other togaviruses that produced a high percentage of asymptomatic infections. Serosurveys in areas endemic for Western equine encephalitis virus have shown that from 10 (17) to 30% (9) of persons have antibody without evidence of previous illness. Likewise, inapparent infection ratios of 23:1 (10), 64:1 and 209:1 (14), 250:1 (12), and 500 to 1,000:1 (22) have been reported for other arboviruses (togaviruses).

Variation in case definition and time interval between epidemic and survey account for part of the above differences. Most of the surveys were conducted a long time after the outbreak took place. Overt encephalitis was the definition of a clinical case in references 15, 16, 25, and 26, not a history of recent milder illness as reported here.

The low inapparent infection ratio found during this outbreak is similar to that found by Gutierrez et al. (11), who found this ratio to be 1:10 during the 1969 Ecuador outbreak. Earlier reports by Hinman et al. (13), Sanmartin (18), and Scherer et al. (20) also indicated that a VEE virus infection is most often symptomatic. All these authors, however, found many persons with VEE antibody who had had no symptoms.

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