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Chandipura Virus: A Major Cause of Acute Encephalitis in Children in North Telangana, Andhra Pradesh, India

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A hospital-based surveillance was undertaken between May 2005 and April 2006 to elucidate the contribution of Chandipura virus (CHPV) to acute viral encephalitis cases in children, seroconversion in recovered cases and to compare the seroprevalences of anti-CHPV IgM and N antibodies in areas reporting cases with those without any case of acute viral encephalitis. During this period, 90 cases of acute encephalitis were hospitalized in the pediatric wards of Mahatma Gandhi Memorial (MGM) Hospital, Warangal. There were 49 deaths (Case Fatality Rate, i.e., CFR of 54.4%). Clinical samples and records were obtained from 52 suspected cases. The cases were below 15 years, majority in 0–4 years (35/52, 67.3%). Computerized tomography (CT) scans and cerebro-spinal fluid (CSF) picture favored viral etiology. No neurological sequelae were observed. CHPV etiology was detected in 25 cases (48.1%, n = 52; RNA in 20, IgM in 3 and N antibody seroconversion in 2). JEV etiology was detected in 5 cases (IgM in 4 cases and seroconversion in 1 case). Anti-CHPV IgM seroprevalence in contacts (26/167, 15.6%) was significantly higher ($P < 0.05$) than in non-contacts (11/430, 2.6%); which was also observed in children <15 years (19/90, 21.1% vs. 3/109, 2.7%). Anti-CHPV N antibody seroprevalence in <15 years contacts (66/90, 73.3%) and non-contacts (77/109, 70.6%) was significantly lower ($P < 0.05$) than in contacts (75/77, 97.4%) and non-contacts (302/321, 94.1%) more than 15 years respectively. CHPV appears to be the major cause of acute viral encephalitis in children in endemic areas during early monsoon months. ***J. Med. Virol. 80:118–124, 2008.*** © 2007 Wiley-Liss, Inc.

KEY WORDS: Chandipura virus; childhood acute viral encephalitis; seroconversion; surveillance; sero-epidemiological surveys

INTRODUCTION

Chandipura virus (CHPV), a *vesiculovirus* in the *Rhabdoviridae* family, was associated with outbreaks of acute encephalitis in children in various districts of Andhra Pradesh in 2003 [Rao et al., 2004] and Gujarat state in 2004 [Chadha et al., 2005]. These outbreaks were characterized by rapid onset of fever, central nervous system (CNS) involvement and high case fataity. Probably, due to rapid onset of the disease, only some cases had anti-CHPV IgM antibodies and hence the polymerase chain reaction (PCR) was used for diagnosis. The distribution of cases was patchy and the villages reporting cases were far away from each other making follow-up of cases a difficult task. Therefore, data on seroconversion in recovered cases were crucial.

During these outbreaks, anti-CHPV IgM antibodies were detected in some cases of fever, healthy family contacts and other individuals in the community indicating wider disease spectrum with the possibility of subclinical infections [Chadha et al., 2005]. The presence of anti-CHPV IgG antibodies in adults [Rao et al., 2004] and high frequency of neutralizing (N) antibodies, with increase in seroprevalences with age in the healthy human population [Chadha et al., 2005] suggested that the virus was endemic in the area.

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The study was planned to determine the contribution of CHPV to acute viral encephalitis cases in children, seroconversion in recovered cases and to compare the seroprevalences of anti-CHPV IgM and N antibodies in areas reporting cases with those without any cases of acute viral encephalitis.

METHODS

The study area, the north Telangana region of Andhra Pradesh state (Fig. 1), has the regional directorate of health services and the referral medical college hospital in Warangal city, serving the population of Warangal, Karimnagar, Khammam, and Adilabad districts. The general population density is 252 per square kilometers with 81% of the population residing in rural areas.

A hospital-based surveillance of cases with acute encephalitis in children, admitted to the Mahatma Gandhi Memorial Hospital of Kakatiya Medical College in Warangal city, was undertaken between May 2005 and April 2006. An acute viral encephalitis case was defined as a patient aged less than 15 years, presenting with acute onset of fever and central nervous system involvement in the form of one or more symptoms such as altered sensorium, unconsciousness, coma and convulsions; without signs of meningeal involvement; and negative for malaria, tuberculosis and other common bacterial causes. Clinico-epidemiological field investigations were undertaken through the field station established by the National Institute of Virology (NIV), Pune (India).

Fifty-two acute viral encephalitis cases, including three cases with acute onset of neurological involvement but without any records of fever were studied. Acute phase sera were collected within a week of the onset of illness. Convalescent phase sera were collected between 2 and 7 weeks (n = 9) and after 3 months (n = 2) of the onset of illness. A total of 167 sera from contacts residing in 10 villages reporting at least one case of acute viral encephalitis and 430 sera from subjects residing in 11 villages without any case (non-contacts) were collected for detection and comparison of viral activity.

Detection of CHPV RNA was done according to the method described earlier [Chadha et al., 2005]. PCR positivity was confirmed by sequencing all the PCR products. Alternate negative controls were included in all the PCR assays. Pre- and post-amplification procedures were conducted in well-segregated laboratories.

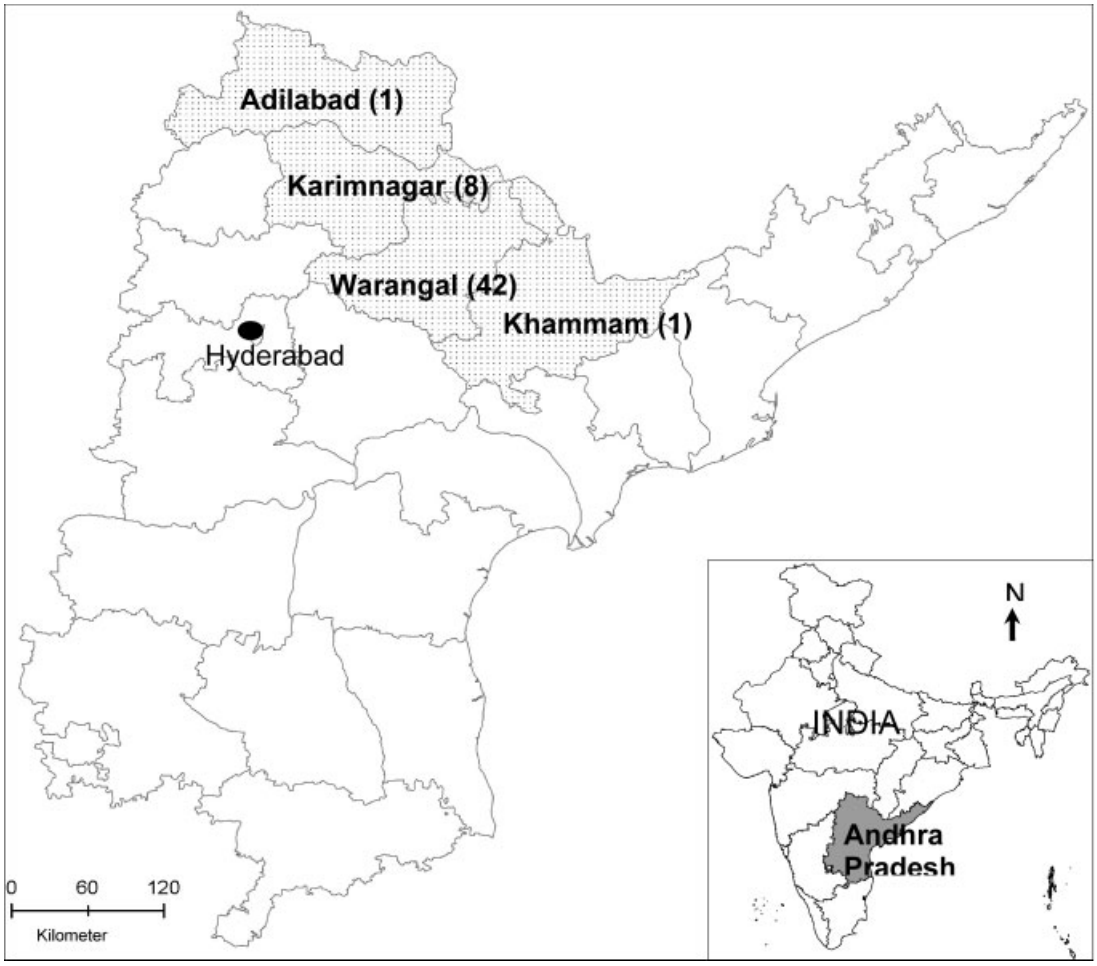

Fig. 1. Geographic distribution of acute viral encephalitis cases among children in North Telangana, Andhra Pradesh, India (May 9, 2005 to March 22, 2006, N = 52). The affected districts with the number of reported cases in brackets.

Patient sera were screened using in-house enzyme-linked immunosorbent assays (ELISAs) for the detection of IgM antibodies against Japanese Encephalitis virus (JEV), West Nile virus (WNV), and CHPV [Rao et al., 2004]. Negative controls included age-matched sera from apparently healthy children from an area not affected by the outbreak, and serum and CSF from children with flavivirus encephalitis. The cut-off value was determined as mean optical density for negative controls plus 3 SD.

Similarly, all sera were screened for neutralizing (N) antibodies against CHPV employing an in vitro neutralization test in Porcine Sarcoma (PS) cell cultures as per the protocol described previously [Rao et al., 2004]. The virus neutralizing antibody titer was expressed as the reciprocal of the highest antibody dilution capable of neutralizing 100 $TCID_{50}$ of CHPV. A titer of 1:10 was considered a positive result.

The proportions of contacts and non-contacts showing neutralizing (N) antibody were compared using Chi-square. The Institutional Ethical Committee approved the study protocol for clinical case investigations, laboratory diagnosis and seroepidemiological surveys in contacts and non-contacts.

RESULTS

According to the records of the hospital, between May 2005 and April 2006, 90 cases of encephalitis were admitted in the hospital, among which 49 were deaths (CFR 54.4%). Clinical data and the sera were obtained from 70 cases, out of which 52 patients were considered as suspects as per the case definition. As depicted in Figure 1, the majority of these cases were from Warangal district (42 cases), followed by other districts (Karimnagar-8 cases, Khammam-1 case, Adilabad-1 case). Clustering was not observed. The summary sheet of these 52 suspect patients is shown in Table I.

The ages of the patients ranged between 9 months and 13 years with maximum cases (35/52, 70%) in 0–4 years. Male to female ratio was 1:2. Case fatality was high (25/52, 48.1%) and majority of deaths (19/25, 76%) occurred within 48 hr of hospitalization. Hospital stay in survivors ranged between 2 and 9 days without any neurological sequelae at discharge from hospital and even at follow-up in the field in 11 cases. Acute onset of fever was reported in 49 of 52 cases, with duration less than 48 hr in majority (29/52, 54%). The symptoms included various degrees of altered consciousness (47/52, 90.4%) and convulsions (41/52, 78%). Other common symptoms included vomiting (23/52, 44%) and diarrhea (8/52, 16%).

Clinical neurological examination revealed decreased muscle tone and power with non-elicitable deep tendon reflexes and extensor plantar responses. Pupils were dilated with a sluggish reaction to light. Ocular fundi were normal in 2 patients. CT scans of 8 cases revealed diffuse swelling of the brain parenchyma and dilated ventricles. Routine laboratory evaluation was negative for malaria, tuberculosis and other common bacterial infections. CSF in 13 cases showed raised intra-cranial pressure, with normal glucose and slightly raised protein levels. There were no organisms and cells were less than 5 per cubic millimeter, predominantly lymphocytes.

The biweekly distribution of cases is shown in Figure 2. The bimodality in the epidemiological curve reflects exclusive CHPV cases (20/34, 58.8%) in early period and second rise of CHPV (5/18, 27.8%) with JEV (5/18, 27.8%) cases occurring after 15th September 2005. The differences in clinical presentations of CHPV and JEV confirmed cases are described in the following paragraph.

Twelve of 25 (48.0%) CHPV cases occurred in children under 5 years of age as compared to none of 5 JEV cases. Also, 8 out of 10 deaths among 25 CHPV cases occurred within 24 hr of the onset of illness, whereas there were no deaths among 5 JEV patients. Seventeen of 25 (68.0%) CHPV cases presented with history of fever less than 2 days and none in JEV cases. CSF cell counts were less than 2 per cubic millimeter in CHPV cases, while in JEV cases the cells ranged from 90 to 300. CSF protein levels in investigated CHPV cases were normal and in JEV cases these were raised. The CT scan images of two CHPV cases are shown in Figure 3. The ventricles were dilated in one case (Fig. 3a, case no. 9 in Table I and case no. 3 in Table II). Diffuse swelling of the white matter and effaced sulcal spaces were suggestive of infective etiology in the second case (Fig. 3b, case no. 12 in Table I and case no. 4 in Table II), which reported convulsions of acute onset without a history of fever.

CHPV RNA was detected in 20 (45.4%) of 44 cases and anti-CHPV IgM in 3 (6.8%) cases (Table I). Seroconversion was studied in 11 cases (Table II). Among five cases with evidence of CHPV RNA by PCR in acute phase sera, CHPV seroconversion was found in four cases—anti-CHPV IgM seroconversion (case no. 3), anti-CHPV N antibody seroconversion (case no. 1) and both anti-CHPV IgM and N antibody seroconversion in two cases (case no. 5 and 6). Two more cases, which were negative for both CHPV RNA and anti-CHPV IgM in the acute phase sera, seroconverted to anti-CHPV N antibodies (case no. 4 and 7) in the convalescent phase sera. Thus CHPV etiology was detected in 25 cases. Among these, 20 (8%) cases were reported between 9th May and 15th September 2005, the period of late summer and early monsoon seasons.

Anti-JEV IgM was detected in 4 of 37 (10.8%) investigated cases. One more case (Table II—case no. 10) seroconverted to anti-JEV IgM. All these 5 JEV cases were reported after 15th September 2005, which is the late monsoon season. In summary, among 52 cases, 25 (48.1%) cases were detected with CHPV etiology and 5 (9.6%) cases with JEV etiology. Thus, viral etiology could be established in 30 (57.7%) of 52 cases.

The comparisons of seropositivity to anti-CHPV IgM and N antibodies between contacts and non-contacts according to age groups are shown in Table III. Anti-CHPV IgM antibody seropositivity

TABLE I. Summary of Cases of Acute Viral Encephalitis Among Children With Epidemiological, Clinical, and Virological Features

Case no.	Date of admission (mm/dd/yyyy)	District	Age (years)	Sex	Clinical outcome	Fever (days)	Altered sensorium (days)	Unconsciousness (days)	Coma grade	Convulsions (days)	Vomiting (days)	Loose motions (days)	Post onset day of serum	CHPV RNA	Anti-CHPV IgM	Anti-JEV IgM	Anti-WNV IgM	Etiology
1	5/9/2005	Karimnagar	4	m	Death	0			3	0			0	Neg	Neg	Neg	Neg	Unknown
2	5/9/2005	Karimnagar	2	m	Death	0	0			0		0	1	Neg	Neg	Neg	Neg	Unknown
3	5/9/2005	Warangal	5	m	Death	1	0			0		0	0	Neg	Pos	Neg	Neg	CHPV
4	5/13/2005	Warangal	3	f	Death	1	1	0	3	0	0		1	Pos	Neg	Neg	Neg	CHPV
5	5/17/2005	Warangal	7	f	Death	0	0			2			3	Neg	Neg	Neg	Neg	Unknown
6	6/11/2005	Warangal	6	f	Discharged	3		0		1			3	Pos	Neg	—	—	CHPV
7	6/17/2005	Warangal	6	f	Death	1	0			0			2	Neg	Neg	—	—	Unknown
8	6/18/2005	Warangal	7	m	Discharged	4	0		4	0			6	Neg	Neg	Neg	Neg	Unknown
9	6/19/2005	Warangal	7	f	Discharged	1	1		4	0	0		6	Pos	Neg	Neg	Neg	CHPV
10	6/20/2005	Warangal	2	f	Death	1			3	1		0	1	Pos	Neg	—	—	CHPV
11	6/23/2005	Warangal	8	f	Death	0		0	3		0	0	0	Pos	Neg	—	—	CHPV
12	6/27/2005	Warangal	3	f	Discharged	no				0			3	Neg	Neg	—	—	CHPV Case 4 in Table II
13	6/29/2005	Karimnagar	11	m	Death	1		0	3	0	1	0	1	Pos	Neg	Neg	—	CHPV
14	6/30/2005	Warangal	7	f	Death	1		0		1	1		0	Neg	Neg	—	—	Unknown
15	7/4/2005	Warangal	8	f	Discharged		0			0	0	0	0	Pos	Neg	Neg	Neg	CHPV
16	7/4/2005	Warangal	6	m	Discharged	1	1				0		1	Pos	Neg	Neg	Neg	CHPV
17	7/5/2005	Warangal	10	f	Death	3	0			2			4	Neg	Neg	Neg	Neg	Unknown
18	7/6/2005	Warangal	4	f	Death	0	0		3	1			0	Pos	Neg	Neg	Neg	CHPV
19	7/8/2005	Warangal	3	f	Death	1	0	0		0	0		1	Pos	Neg	Neg	—	CHPV
20	7/9/2005	Warangal	1	f	Discharged	4	4			4			13	Neg	Pos	Neg	—	CHPV
21	7/10/2005	Warangal	6	f	Discharged	0	2	1	2				2	Neg	Neg	Neg	Neg	CHPV Case 7 in Table II
22	7/11/2005	Warangal	5	m	Discharged			0		0	0		0	Pos	Neg	Neg	Neg	CHPV
23	7/11/2005	Karimnagar	7	m	Discharged	1				1			2	Pos	Neg	Neg	Neg	CHPV
24	7/17/2005	Warangal	4	m	Death	1	0						1	Neg	Neg	Neg	Neg	Unknown
25	7/18/2005	Karimnagar	8	f	Discharged	1			3		0		1	Pos	Neg	Neg	Neg	CHPV
26	7/18/2005	Warangal	4	f	Death	1	0	0	3	0	0	0	1	Pos	Neg	Neg	Neg	CHPV
27	7/25/2005	Warangal	0	f	Discharged	4	0		3		0	10	9	Neg	Neg	Neg	—	Unknown
28	7/28/2005	Warangal	6	f	Death	3				0	0		3	Neg	Neg	Neg	—	Unknown
29	7/30/2005	Adilabad	1	f	Death	3				0			5	Pos	Pos	Neg	—	CHPV
30	8/3/2005	Warangal	4	f	Discharged	1		0	2	0			1	Pos	Neg	Neg	—	CHPV
31	8/23/2005	Warangal	4	m	Death	1	0			0			2	Neg	Neg	Neg	Neg	Unknown
32	8/29/2005	Warangal	2	m	Discharged	2	1		3	1	1		3	Neg	Pos	Neg	Neg	CHPV
33	9/3/2005	Warangal	2	f	Discharged	0		0		5			3	—	Neg	—	—	Unknown
34	9/7/2005	Khammam	9	f	Discharged	4	2						5	—	Neg	—	—	Unknown
35	9/18/2005	Warangal	0	m	Discharged	1	1			1			2	—	Neg	Neg	Neg	Unknown
36	9/20/2005	Warangal	13	f	Discharged	3	0						4	—	Neg	Pos	Neg	JEV
37	9/27/2005	Warangal	10	f	Discharged	3	1		1		0		6	Pos	Neg	Neg	Neg	CHPV
38	10/5/2005	Warangal	4	f	Discharged	5	5		3	5			6	Neg	Neg	Neg	Neg	Unknown
39	10/5/2005	Warangal	12	f	Discharged	0			0		0		3	Pos	Neg	Neg	Neg	CHPV
40	10/6/2005	Warangal	12	m	Discharged	3		0		1	1		5	Neg	Neg	Neg	Neg	JEV Case 10 in Table II
41	10/7/2005	Warangal	6	f	Death	2	0			0	2		3	Neg	Neg	Neg	Neg	Unknown
42	10/10/2005	Warangal	6	f	Discharged	4	0		2		2		5	Neg	Neg	Pos	Neg	JEV
43	10/18/2005	Karimnagar	7	m	Discharged	5			4	5	0		8	—	Neg	Pos	Pos	JEV
44	10/20/2005	Warangal	5	f	Discharged	0	0			0	0		1	—	Neg	Pos	Pos	JEV
45	10/21/2005	Warangal	4	f	Death	3	0		2	1	3		4	Neg	Neg	—	—	Unknown
46	10/23/2005	Karimnagar	3	f	Discharged	1			3	1			3	Pos	Neg	—	—	CHPV
47	10/24/2005	Warangal	6	m	Death	3	1		3	0			3	Neg	Neg	—	—	Unknown
48	10/25/2005	Warangal	7	m	Discharged	1		0		0			4	Neg	Neg	—	—	Unknown
49	10/28/2005	Warangal	13	m	Death	2				2			2	Neg	Neg	—	—	Unknown
50	11/3/2005	Warangal	3	F	Death	5	0			1			7	Pos	Neg	Neg	Neg	CHPV
51	12/20/2005	Karimnagar	10	f	Death	1	1			1	1		1	—	Neg	—	—	Unknown
52	03/22/2006	Warangal	6	m	Death	0	0			0			0	—	Neg	—	—	Unknown

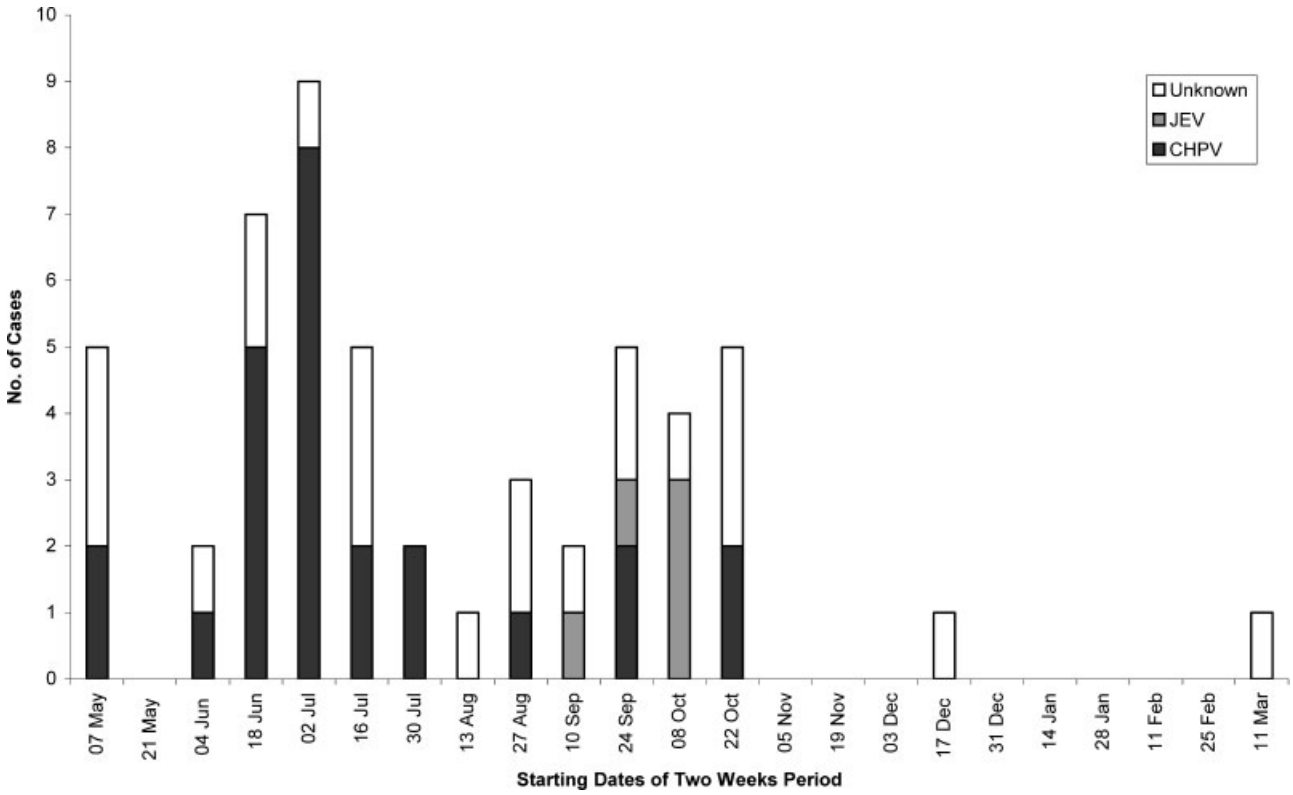

Fig. 2. Biweekly incidence of acute viral encephalitis cases among children in North Telangana, Andhra Pradesh, India (May 9, 2005 to March 22, 2006, N = 52). The numbers of cases are indicated on Y-axis, with the etiology indicated as JEV, CHPV and unknown, in decreasing shades of gray. X-axis shows the starting date of 2 weeks period.

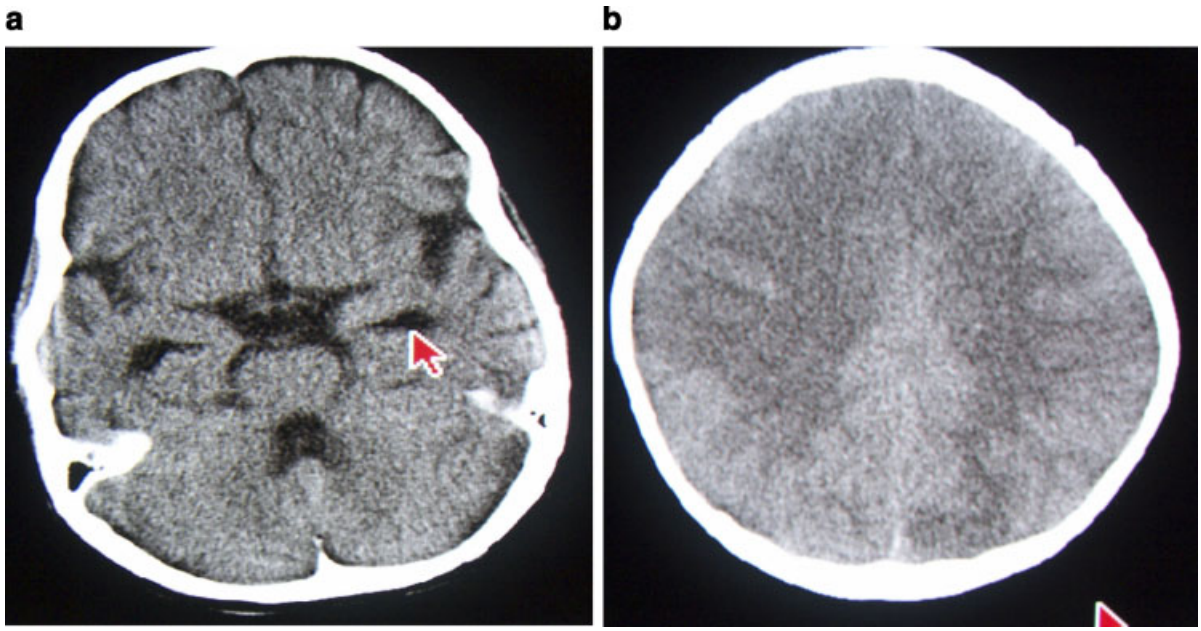

Fig. 3. Computerized tomographic (CT) scan images of two CHPV confirmed cases. **a**: Shows dilated temporal horns of both lateral ventricles (indicated by an arrow) in a section at suprasellar cistern. **b**: Shows diffuse white matter edema on both sides and effaced sulcal spaces in a section at the supraventricular region.

TABLE II. Seroconversion to Anti-CHPV IgM and N Antibodies and Anti-JEV IgM

Case no.	Post onset day of serum	CHPV RNA	Anti-CHPV IgM	Anti-CHPV N antibody result	Anti-CHPV N antibody titer	Anti-JEV IgM
1[a]	3	Pos	Neg	Neg	<1:10	—
	111	—	Neg	Pos	1:80	Neg
2	6	Neg	Neg	Neg	<1:10	Neg
	17	—	Neg	Neg	<1:10	Neg
3[a]	6	Pos	Neg	Pos	1:80	Neg
	17	—	Pos	Pos	1:80	Neg
4[a]	3	Neg	Neg	Neg	<1:10	—
	120	—	Neg	Pos	1:160	Neg
5[a]	0	Pos	Neg	Neg	<1:10	Neg
	16	—	Pos	Pos	1:640	Neg
6[a]	1	Pos	Neg	Neg	<1:10	Neg
	16	—	Pos	Pos	1:320	Neg
7[a]	2	Neg	Neg	Neg	<1:10	Neg
	12	—	Neg	Pos	1:160	Neg
8	4	—	Neg	—	—	Pos
	38	—	Neg	Neg	<1:10	Pos
9	6	Pos	Neg	Pos	1:40	Neg
	42	—	Neg	Pos	1:40	—
10[a]	5	Neg	Neg	Pos	1:40	Neg
	21	—	Neg	Pos	1:80	Pos
11	8	—	Neg	Neg	<1:10	Neg
	23	Neg	Neg	Neg	<1:10	—

[a]Indicates cases with seroconversion.

in contacts (26/167, 15.6%) was significantly higher ($P < 0.05$) than in non-contacts (11/430, 2.6%). Anti-CHPV IgM antibody seropositivity in contacts aged less than 15 years (19/90, 21.1%) was significantly higher ($P < 0.05$) than contacts aged 15 years or more (7/77, 9.1%). Fever was reported by 9 (5.4%) of 167 contacts and 21 (4.9%) of 430 non-contacts, among which anti-CHPV IgM was detected in 2 of 9 febrile contacts and 3 of 21 febrile non-contacts. Anti-CHPV IgM seropositivity in afebrile contacts (24/158, 15.2%) was significantly higher ($P < 0.05$) than afebrile non-contacts (8/409, 1.9%).

Anti-CHPV N antibody seropositivity in children less than 15 years contacts (66/90, 73.3%) and non-contacts (77/109, 70.6%) was significantly less ($P < 0.05$) than in more than 15 years contacts (75/77, 97.4%) and non-contacts (302/321, 94.1%) respectively.

DISCUSSION

Of the 52 suspected cases of viral encephalitis in children, evidence of recent viral infection was established conclusively in 30 (57.7%) cases. CHPV infection was detected in 25 (48.1%) cases with the majority (20 cases) in late summer and the early monsoon season. JEV infection was detected in 5 (9.6%) cases, all 5 cases detected after 15th September 2005, indicating different periods of JEV and CHPV activity. Warangal and Karimnagar districts have been identified as JEV endemic areas with occurrence of encephalitis cases in the late monsoon season. Over all, 25 cases could be attributed to CHPV, 5 cases to JEV and 22 cases remained undiagnosed.

It is important to note that due to rapid onset and high mortality, CHPV diagnosis is based mainly on detection of CHPV RNA. Though 19 of 22 undiagnosed cases were bled during the acute phase, CHPV RNA negativity in these samples could be due to the absence of viremia at sampling or the presence below the detection limit of the nested PCR. The possibility of circulation of another agent cannot be ruled out. Three undiagnosed cases were bled during the second week of illness. The late development of anti-CHPV IgM antibodies in few cases, less sensitivity of the assay used currently or other etiology may be possible in these cases. Follow up of 11 cases provided important information in relation to

TABLE III. Age-Specific Seroprevalences of Anti-CHPV IgM and N Antibodies in Contacts and Non-Contacts

Type of antibody	Anti-CHPV IgM antibody		Anti-CHPV N antibody	
Subject category	Contacts	Non-contacts	Contacts	Non-contacts
Age <15 years	19/90 (21.1)	3/109 (2.7)	66/90 (73.3)	77/109 (70.6)
Age ≥15years	7/77 (9.1)	8/321 (2.5)	75/77 (97.4)	302/321 (94.1)
Total	26/167 (15.6)	11/430 (2.6)	141/167 (84.4)	379/430 (88.1)

Figures indicate No. positive/No. Tested (percentage).

the possibility of misdiagnosis based on a single serum sample (Table II).

The clinical features were consistent with those described in 28 confirmed cases in Andhra Pradesh outbreak [Rao et al., 2004] and 19 cases in Gujarat [Chadha et al., 2005]. The CFR of 54% was also similar to that observed in the 2003 outbreak [Rao et al., 2004]. The results confirm conclusively the role of CHPV as the major cause of endemic acute viral encephalitis in children from the Telangana region of Andhra Pradesh.

Detection of anti-CHPV N antibody seropositivity to similar extent in villages reporting and not reporting encephalitis cases shows that the virus is highly prevalent in the region (Table III). The presence of anti-CHPV IgM antibodies in 2 of 9 febrile contacts and 3 of 21 febrile non-contacts indicate the varied clinical spectrum of the CHPV infection. The factors responsible for encephalitis in some children need to be examined. Exposure of children below 15 years of age, as determined by the presence of anti-CHPV N antibodies, was significantly less than adults indicating susceptibility of pediatric population. This is consistent with the fact that encephalitis cases occurred only among children as observed earlier [Rao et al., 2004; Chadha et al., 2005].

Sand flies have been implicated as vectors of CHPV by isolations in Aurangabad [Dhanda et al., 1970] and detection of viral RNA in Karimnagar [Geevarghese et al., 2005].

Thus, CHPV is an important cause of endemic acute viral encephalitis in children in Telangana region of Andhra Pradesh with seasonal increase in early monsoon. Proper surveillance and vector control strategies may help in case reduction.

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