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Neurological Manifestations and Outcomes of Chikungunya Virus Infection in Children

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ABSTRACT

Introduction: Chikungunya Virus (CHIKV) is a mosquito-borne arbovirus belonging to the family Togaviridae. Epidemics continue to occur with varied presentation in children. There have been few studies on the neurological impact of chikungunya in children.
Objective: To study the clinical profile of various neurological manifestations associated with CHIKV infections and their outcomes.
Methods: This is a retrospective study conducted in a tertiary care hospital. Study subjects includes all children between age of 1 month to 18 years with proven chikungunya infection. A positive diagnosis of CHIKV was made by testing for CHIK IgM in serum or anti-CHIKV IgM in the CSF, and by fulfilling the Encephalitis Consortium criteria for encephalitis. We assessed neurological sequelae during follow up.
Results: During the period from May 2013 to May 2021, 103 patients with CHIKV infection were diagnosed, and 32 patients met the inclusion criteria**.** On analysis of results, neurological manifestations occurred between 1-8 days after onset of fever. Majority of the cases belong to probable encephalitis (18). Severe form of infection is seen in Probable CHIKV encephalitis with prolonged hospital stay, requiring ventilator support and complications like seizures, cranial nerve palsy, visual disturbances. Less severe forms of infection are seen among possible encephalitis and Non encephalitis CHIKV associated CNS disease (NECACD).
Conclusion: CHIKV can cause neurological symptoms in children within 1-8 days, and should be considered as a potential cause of encephalitis. Early identification of CHIKV cases is crucial because of the economic burden they cause on developing tropical countries.

Keywords: chikungunya, encephalitis, acute disseminated encephalomyelitis, neurological infection**.**

INTRODUCTION

Chikungunya virus belongs to alphavirus genus and Togaviridae family.[1] This is a single chain linear RNA virus which is transmitted by Aedes group of mosquitos mainly Aedes Albopictus. Chikungunya term is derived from Swahili language which means "that which bends up" [2].

Chikungunya virus causes a self-limiting benign illness. In children the clinical manifestations resemble that of adults with some differences. In children commonly they present with abrupt onset of fever, rashes, arthralgia/arthritis, lymphadenopathy, swelling of eyelids, conjunctival injection and pharyngitis. Rarely they may have neurological manifestations like seizures, altered level of consciousness, blindness due to retrobulbar neuritis, and acute flaccid paralysis. In some cases, they may have febrile seizures, vomiting, abdominal pain, and constipation. [3,4]

The virus was isolated in 1952 and its disease was first reported in Tanzania in 1953[5,6]. Last massive outbreak in India was in 2006 during which 13.9 million suspected cases have been reported from 16 states and union territories[7]. In India the city of Delhi experienced an outbreak of chikungunya fever from August to October 2016. Epidemics related to chikungunya follow a cyclical and seasonal trend with an interepidemic period of 4-8 years. Chikungunya is still happening and we don't know much about how it affects children's nervous system.

METHODS

We conducted a retrospective study in tertiary care hospital after obtaining institutional review board approval. Study subjects included all children admitted to the hospital between the age of 1 month to 18 years with proven chikungunya infection and satisfying criteria of Encephalitis Consortium criteria for encephalitis[8]. The diagnosis of chikungunya was confirmed by presence of anti-CHIKV immunoglobulin M (IgM) in serum using ELISA and/or CSF for CHIKV RNA using Real time PCR.

Major Criteria:

Patients with altered level of consciousness or personality change or lethargy for ≥24h.

Minor Criteria:

1. Documented fever ≥38°C (100.4°F) within the 72h before or after presentation.
2. Generalised or partial seizures that is not fully because of a pre-existing seizure disorder.
3. New onset focal neurologic findings.
4. CSF analysis showing WBC count of ≥5/mm^3.
5. Brain imaging shows signs of either new or acute onset encephalitis.
6. Electroencephalogram abnormalities that are consistent with encephalitis and not because of another cause.

We eliminated other potential causes, such as trauma, tumors, alcohol abuse, and non-infectious factors.

For probable CHIKV-associated encephalitis, the criteria include the major criterion and 3 minor criteria. For possible CHIKV-associated encephalitis, the criteria include the major criterion and 2 minor criteria. When the major criterion is met alone or with 1 or 2 minor criteria other than fever, we diagnose Non-Encephalitic CHIKV-associated CNS disease (NECACD).

Medical records concerning patients were reviewed and data was abstracted using standard data collection form. Information recovered included clinical and laboratory data, status at discharge. We recorded complimentary investigations like CT brain, MRI brain and EEG.

Clinical features included in the data were fever, rashes, joint pains, seizures, visual disturbances, cranial nerve palsies, motor system abnormalities, altered sensorium, requirement of mechanical ventilation, and/or behavioral disturbances. Laboratory investigations included complete blood count, CRP, liver function tests, serum creatinine, serum electrolytes, investigations to rule out other infections, CSF cell counts,

sugars, protein, IGM chikungunya virus antibody. We recorded the following investigations data like CT brain, MRI brain, and EEG whenever required.

Statistical analysis:
Data was entered into Microsoft excel data sheet and was analyzed using SPSS 22 version software. Categorical data was represented in the form of Frequencies and proportions. **Chi-square test or Fischer's exact test** (for 2x2 tables only) was used as test of significance for qualitative data.
Continuous data was represented as mean and standard deviation. **ANOVA** was used as test of significance to identify the mean difference between more than two quantitative variables
Graphical representation of data: MS Excel and MS word was used to obtain various types of graphs
P value (Probability that the result is true) of <0.05 was considered as statistically significant after assuming all the rules of statistical tests.
Statistical software: MS Excel**,** SPSS version 22 **(**IBM SPSS Statistics, Somers NY, USA) was used to analyze data.

RESULTS

Total of 34 patients had positive CSF findings for CHIKV. We did not consider two cases because they also tested positive for dengue and Leptospira antigen respectively, which can cause encephalitis on their own. Out of 32 patients 18 were males and the rest were females. Majority of the subjects were aged between 1-10 years as shown in table 1. Based on clinical features, we divide the study population into three groups as probable encephalitis (18 cases), possible encephalitis (7 cases), Non-Encephalitic Chikungunya associated CNS Disease -NECACD (7 cases).
Clinical features:
Table-2 illustrates that probable encephalitis cases of CHIKV infection had high chances of seizures, altered sensorium. Ventilator requirement was more in probable cases. The mean duration of hospital stay was significantly more in probable encephalitis cases (18.2 days). One case had rashes and one case had joint pains.
Investigations:
Routine investigation results showed WBC counts ranging from 3070-21700 with higher mean values in NECACD cases (15252.9±6145.0), thrombocytopenia was seen in 4 cases of possible encephalitis, 4 in probable encephalitis and 3 in NECACD as shown in table 3. Hypoalbuminemia, hypokalemia, hyponatremia, hypocalcemia, thrombocytopenia was relatively more seen in probable encephalitis cases as seen in table 4. Lumbar puncture results showed higher protein and total counts in all cases, especially in probable encephalitis cases (CSF total counts–48.3±57.5, CSF protein–90.9±118). Neutrophil predominance in CSF analysis is seen in probable encephalitis group (17±23.2) whereas the other two groups had lymphocyte predominance as shown in table 5. Elevated liver enzymes were seen in all the cases.
IMAGING:
CT scan was done in 10 out of which 7 showed radiological changes like ill-defined hypodense areas in right temporoparietal occipital lobe, cerebral edema, basal ganglia hypodensities, ill-defined hypodense areas in superior vermis and cerebellum resulting in moderate hydrocephalus. 3 didn't show any radiological abnormalities. Out of the 7 cases only one case belongs to possible encephalitis rest all belong to probable encephalitis.
MRI brain findings showed Acute Disseminated
Encephalomyelitis/Encephalitis, posterior occipital, temporal, parietal hyperintensities and bilateral pulvinar and medial temporal hyperintensities in three cases having probable encephalitis.
Outcomes:
Two children developed cranial nerve palsies, one belonging to possible group had Lower Motor Neuron type of 7th nerve palsy and the other belonging to NECACD had 6th nerve palsy. 4 developed vision disturbances out of which 3 were probable encephalitis

and one was possible encephalitis. No mortality was observed in our study.

Out of the total 32, 6 did not follow up. A child with probable CHIKV encephalitis showed decreased academic performance and motor weakness, including gait disturbances and slurred speech. Cranial nerve palsies and vision disturbances were transient and completely recovered. We observed no sleep disturbances.

Table 1: Age and Gender distribution among study groups

	Probable		Possible		NECACD		P value
	N	%	N	%	N	%	
Age							
<1yrs	3	16.7%	0	0%	2	28.6%	0.854
1-5yrs	6	33.3%	2	28.6%	2	28.6%	
6-10yrs	5	27.8%	3	42.9%	2	28.6%	
>10yrs	4	22.2%	2	28.6%	1	14.3%	
Sex							
Female	7	38.9%	0	0%	3	42.9%	0.128
Male	11	61.1%	7	100%	4	57.1%	

Table 2: Clinical features among study groups

	Probable		Possible		NECACD		P value
	N	%	N	%	N	%	
Seizures	17	94.4%	4	57.1%	2	28.6%	0.003
Altered sensorium	14	77.8%	5	71.4%	3	42.9%	0.236
Joint pain	0	.0%	1	14.3%	0	.0%	0.158
Rashes	1	5.6%	0	.0%	0	.0%	0.669
Blurring of Vision	2	11.1%	0	.0%	0	.0%	0.436
Ventilation	8	44.4%	2	28.6%	1	14.3%	0.339
Shock	3	16.7%	1	14.3%	1	14.3%	0.983
DOHS	18.2	9.7	8.3	3.9	10.4	3.3	0.012

Table 3: Biological parameters among CHIKV infected cases

	GROUPS						P value
	Probable		Possible		NECACD		
	Mean	SD	Mean	SD	Mean	SD	
HB	10.6	1.4	10.6	1.4	10.4	1.2	0.957
TC	14667.1	5233.1	14667.1	5233.1	15252.9	6145.0	0.168
RBS	103.2	15.8	103.2	15.8	114.5	25.2	0.495
Creatinine	.6	.2	.6	.2	.5	.1	0.481
CA	7.9	.4	7.9	.4	8.2	.5	0.186
NA	137.0	4.9	137.0	4.9	133.3	2.9	0.478
K	4.5	.6	4.5	.6	4.1	.4	0.487
CL	101.4	6.9	101.4	6.9	103.1	4.5	0.744
SGOT	122.1	81.8	122.1	81.8	121.0	73.4	1.00
SGPT	83.8	65.6	83.8	65.6	84.2	64.1	0.801

Table 4: Comparison of hyponatremia, hypocalcaemia, hypoalbuminemia, thrombocytopenia among study groups.

		Probable		Possible		NECACD		P value
		N	%	N	%	N	%	
PLT	<150000	4	22.2%	4	57.1%	3	42.9%	0.222
	>150000	14	77.8%	3	42.9%	4	57.1%	
CA	<8	5	27.8%	3	42.9%	3	42.9%	0.672
	>8	13	72.2%	4	57.1%	4	57.1%	
NA	<135	8	44.4%	2	28.6%	5	71.4%	0.262
	>135	10	55.6%	5	71.4%	2	28.6%	
Albumin	<3.5	8	44.4%	4	57.1%	3	42.9%	0.825
	>3.5	10	55.6%	3	42.9%	4	57.1%	

Table 5: CSF analysis among study groups

	GROUPS						P value
	Probable		Possible		NECACD		
	Mean	SD	Mean	SD	Mean	SD	
C-G	56.3	12.3	56.3	12.3	53.4	15.9	0.519
C-P	52.8	28.6	52.8	28.6	38.5	24.0	0.381
C-TC	49.6	43.3	49.6	43.3	10.0	14.7	0.196
C-N	4.9	12.0	4.9	12.0	3.7	9.8	0.192
C-L	44.7	45.9	44.7	45.9	6.3	8.3	0.258

Table 6: Comparison of clinical features and outcomes of chikv encephalitis in various studies

Year of cases location	Samples	CSF	Neurological features	diagnosis	outcome
2006, India[22]	11	11 IgM CSF/ serum	Headaches, altered sensorium, ataxia, rigidity, opsoclonus; abnormal brain MRI	Encephalopathy	3 died
2006, India[21]	37	Virus Isolation/PCR/IgM CSF	Unknown	Encephalitis	7 died
2006, India[23]	11	Virus Isolation/PCR/IgM/Hemagglutination inhibition CSF/serum (6)	Unknown	Encephalitis	2 died
2006, Réunion[9]	16	PCR/IgM CSF/serum	Drowsiness, seizures, focal neurological signs; abnormal MRI brain (5) and EEG	encephalopathy (4) Encephalitis (12)	2 died, 5 disabled, 9 no neurological sequelae
2006, India[24]	27	PCR CSF (4) White cell count↑ (6/20), protein↑ (14/20)	59% abnormal behavior; 22% drowsiness, extrapyramidal; 11% seizures; abnormal MRI brain (1/4)	Encephalitis	21 improved, 4 no improvement, 2 died
2015, Honduras[25]	18	18 PCR/ serum (11) White Cell Count↑, protein↑	<12 month (11); seizures/lethargy/bulging fontanelle/irritability/hyperalgesia; abnormal MRI brain (5/5), abnormal EEG (7/14)	Meningoencephalitis	1 died
2016, India[26]	3	PCR serum	Children; seizures/altered sensorium	Meningoencephalitis	Recovered at 3-4 d (2), died after 6 h (1)
Present study	34	34 PCR CSF	convulsion, confusion, behavioral disorders, meningism; abnormal MRI (10/14),	Encephalopathy	No mortality

DISCUSSION

34 laboratory confirmed cases were included in our study with neurological manifestations. Very less emphasis has been placed on understanding neurological involvement of CHIKV in children. However, during the outbreak in La reunion island, 24% of children infected had neurological manifestations like simple and complex febrile seizures to meningeal

syndrome, acute encephalopathy, and encephalitis [9]. In India, during 2006 outbreak a study conducted by Lewthwaite P et al found 14% children had CNS infection[4]. CHIKV can cause various neurological symptoms in adults, such as encephalitis, myelopathy, myopathy, peripheral neuropathy, acute flaccid paralysis, and Gullian-barre syndrome[11]. Notably, there are very few case reports in children related to neuroinfection before 2006 outbreak.

Out of 34, 12 were from urban areas and 22 were from rural areas. 1-5 years age group was dominant in this work, unlike other studies where most of the patients were infants [9].

The exact way that viruses affect the central nervous system, either directly or indirectly through the immune system, is not understood**.** Some of the evidence suggests that virus can invade directly, as both RNA and virus has been isolated in CSF in severe disease[9]. Different cells like astrocytes, neurons, and oligodendrocytes can be infected by chikungunya in a lab setting. It was observed that astrocytes and neurons undergo apoptosis after infection[9]

Diagnosis of CHIKV infection in this work was done by IgM capture ELISA. Viral culture is the gold standard for diagnosis of chikungunya infection but is seldomly used. RT-PCR, real time loop mediated isothermal amplification (RT-LAMP) techniques can be useful for the rapid diagnosis because viral RNA can be detected as early as 8 days from the onset of symptoms. Usually in CHIKV infection, IgM antibodies are detected within 2 days and can persist for up to 3 months, but it has been reported to persist for 18 months after the onset of disease in 40% symptomatic patients. IgG antibodies persist for years1[12,13,14,15]. Some of the serological tests like ELISA, Indirect immunofluorescent method, hemagglutination inhibition or neutralization methods for detection of IgM and IgG antibodies against CHIKV in acute and convalescent sera are used.

In arboviral infections, encephalopathy is the most common neurological presentation. Encephalitis can be diagnosed if there is evidence of brain inflammation like CSF pleocytosis, brain imaging or focal changes on EEG[16,17]. In the present study the mean duration of onset of encephalitis was 1-8 days. In other studies, it was 1-13 days from onset of systemic infection[16]. Imaging abnormalities in chikungunya infection do not show a distinct pattern unlike herpes simplex, and cytomegalovirus infections. The abnormalities in our study were similar to other studies which include CT scan changes like edema, hypodensities and MRI showed T2 hyperintensities [16,17].

Patients with CNS disease were found to have coinfection with arboviral diseases[18,19], of which coinfection with dengue virus is the most common, some associations with zika virus have also been reported[18,20]. The severity of disease, pathogenesis, and presentation with coinfection compared to monoinfection is presently unknown[18]. In our study, one case had dengue coinfection and the other had coinfection with Leptospira.

No mortality has been reported in our study due to CNS infection. Three children under 18 years old died in the La Réunion epidemic. One child had acute disseminated encephalomyelitis, while the other died from hemorrhage, shock, and coma[9]. During the Delhi outbreak, a study reported a case that died from meningoencephalitis and liver involvement[21]. A detailed comparisons of the different studies in terms of neurological symptoms/outcomes arising during CHIKV is reported in Table-6 along with sample size and diagnosis.

Hyperpyrexia, hypotension, cerebral edema, hyponatremia, hypocalcemia can be implicated to cause severe CHIKV infection including encephalopathy and seizures.

Treatment of chikungunya infection is largely supportive; no specific antiviral is available till date. NSAIDS should not be used as this may precipitate bleeding

manifestations. Evidence suggests against use of corticosteroids, antibiotics, or antivirals in chikungunya infection management[15].

Preventive strategies involve personal protection and eliminating mosquito breeding sites. Insecticides and space spraying can also be used. Vaccine development is underway, phase 3 trials are over, 8% developed arthralgia following vaccination.

CONCLUSION

Neurological disease associated with chikungunya virus is being increasingly reported in adults and children. Neurotropic nature of CHIKV is a known entity leading to morbidity in affected children. It was clearly evident that CHIKV has neurological symptom onset over 1-8 day, specifically majority of the neurological symptoms occurred on 3 days followed by 2/5 days. Therefore, chikungunya encephalitis should also be considered as a differential in children presenting with encephalitis. Further the CHIKV cases had palsy related issues which has larger economic burden on developing tropical countries, hence it is very important to identify early in the course of illness.

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