CD Alert

National Centre for Disease Control,
Directorate General of Health Services, Government of India

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CHANDIPURA VIRUS (CHPV)

INTRODUCTION

Acute Encephalitis Syndrome (AES) is a group of clinically similar neurologic manifestation caused by several different viruses, bacteria, fungus, parasites, spirochetes, chemical/ toxins, etc. Cases of AES have been on the rise in the recent years. The known viral causes of AES include JE, Dengue, HSV, Chandipura virus (CHPV), West Nile, etc. Of these, the Chandipura virus (CHPV), is an emerging tropical pathogen which was first discovered in 1965 from Chandipura village in Maharashtra. It belongs to genus vesiculovirus of Rhabdoviridae family.

Sporadic cases and outbreaks of CHPV in western, central, eastern and southern parts of the country, especially during the monsoon season, have been reported. CHPV gained global attention as an encephalitis causing virus after the outbreak in Andhra Pradesh in 2003 causing high fatality and was regarded as a human pathogen of public health importance.

The virus has been isolated from *Phlebotomine* and *Sergentomyia* species of sand-flies which is incriminated as principal vector of CHPV. It affects mostly children and is characterized neurologic dysfunctions; however, there is no evidence of human-to-human transmission.
Till date, presence of this virus is recorded from the Indian subcontinent (India, Bhutan and Nepal, Sri Lanka), and Africa. CHPV has caused explosive outbreaks in Maharashtra, Gujarat and Andhra Pradesh and has emerged as an important encephalitis-causing pathogen in India.

HISTORICAL BACKGROUND

First isolated in 1965, from blood of two adults with febrile illness while investigating persons with fever for dengue and chikungunya viruses in Chandipura of northern Maharashtra, this virus was named after the place of isolation as Chandipura virus (CHPV).
CHPV was initially considered as an orphan or concomitant virus due to low pathogenicity to cause infections in man and domestic animals.
In 1980, after a gap of two decades, potential of CHPV to cause mortality in humans was detected in Raipur district, when virus was held responsible for the death of an 11 years old child due to CHPV induced encephalopathy. Later, in 2003, as the Chandipura viral infection was associated with neurological complications and CHPV caused explosive outbreaks in Maharashtra, and Andhra Pradesh killing over 300 children with a CFR exceeding 50 per cent, it became recognized cause of Arboviral encephalitis.

GLOBAL & INDIAN SCENARIO

GLOBAL SCENARIO

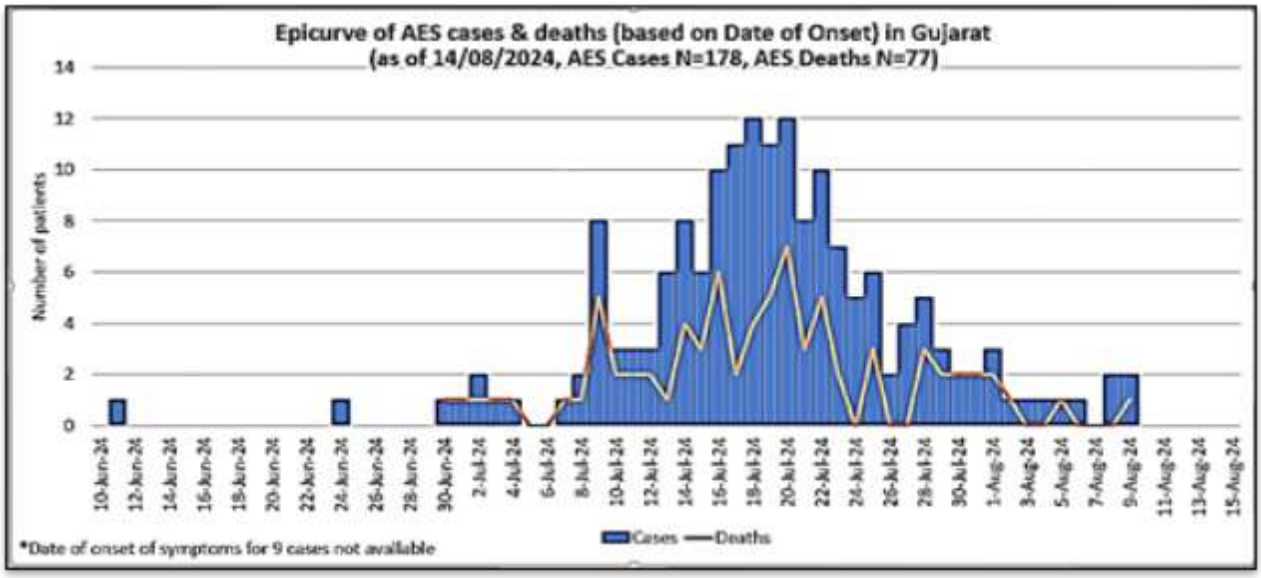
CHPV activity was prevalent in West Africa since 1975 as the virus has been isolated from a hedgehog and wild caught phlebotomine sand-flies from Nigeria and Senegal, respectively. CHPV activity was also reported from Sri Lanka as CHPV antibodies were detected in monkeys.

INDIAN SCENARIO

The epicenter of CHPV activity has been restricted to Central India comprising parts of Gujarat, Madhya Pradesh, Andhra Pradesh and Maharashtra since 1965.

Year	Activity
1965	First detected and isolated from Maharashtra, India
1967-1969	Isolated from Phlebotomine sandflies in Aurangabad, Maharashtra, India
1975	Isolated from Hedgehog in Nigeria, West Africa
1980	Isolated from a 11-year-old case of acute [fatal] encephalopathy syndrome in Raipur district, Madhya Pradesh (now Chhattisgarh), India
1993	Isolated from Phlebotomine sandflies in Senegal, Africa
1994-1997	Isolated from *Sergentomyia spp*. of sandflies in Senegal, Africa
2003	Isolated from human serum in an encephalitis outbreak, Andhra Pradesh, India Neutralizing (N) antibodies to CHPV detected in sera of pigs, buffalos, cattle, goats and sheep in the outbreak
2004	Isolated from human serum in an encephalitis outbreak in Vadodara and Panchmahal districts of Gujarat, India
2005-2006	Anti-CHPV N antibodies detected in both contacts and non-contacts of cases in similar extent, Hospital-based surveillance, Andhra Pradesh
2007	Isolated from human serum in an encephalitis outbreak in Nagpur division, Maharashtra, India
2010-2011	Anti-CHPV IgM antibodies detected in sera from fever cases, Panchamahal district Gujarat, India
2012	Anti-CPHV IgM antibodies detected in serum samples during an outbreak of acute encephalitis syndrome (AES), Vidarbha region, Maharashtra, India CHPV isolated from *Sergentomyia spp.* of sandflies Vidarbha region, Maharashtra, India
2024	Since early June 2024, cases of Chandipura- Acute Encephalitis Syndrome (AES) were reported in children under 15 years of age in Gujarat. As of August 31st, a total of 250 (RJ-72, GJ-178) AES cases have been detected out of which 66 were confirmed for CHPV. Among these, a total of 82 (RJ-5, GJ-77) cases resulted in death out of which 29 were confirmed for CHPV. Although the outbreak was primarily in Gujarat, some of the cases also resided in bordering areas of Rajasthan, Madhya Pradesh and Maharashtra.

Table 1: Chandipura virus (CHPV) global and Indian activity



EPIDEMIOLOGY

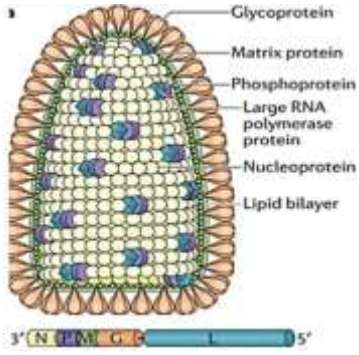
Agent

Chandipura virus is a member of the Rhabdoviridae family classified under genus vesiculovirus , originally an arthropod born virus (Arbovirus) and indigenous to India only. The term Rhabdo, meaning 'rod shaped' in Greek has been assigned due to the bullet shaped morphology of the viruses belonging to the family. Family Rhabdoviridae comprises of negative sense, single stranded viruses 150-165 nm long, 50-60 nm wide, showing distinct surface projections 9-11 nm in size & a stain-filled canal at the base of the virus particles. Amongst the 10 genera, genus Lyssavirus and genus vesiculovirus are of public health importance. Rabies virus, the prototype virus of genus

Lyssavirus, is the most important pathogen of Rhabdoviridae. Genus vesiculovirus, comprises viruses of human and veterinary importance; the prototype specimen being vesicular stomatitis Indiana virus, which infects cattle, horses, pigs, etc. causing mild flu-like symptoms.

CHPV is presently, considered to be the most significant pathogen of public health importance among the vesiculo-viruses due to its high case fatality rate.

Comparative sequence analysis concludes that CHPV is evolutionarily equidistant from new world vesiculo-viruses VSV Indiana (VSVind) and VSV New Jersey (VSVnj), and closely related to its Asian kin Isfahan. CHPV, like VSV, has an outer lipoprotein envelope that encloses a helical ribonucleoparticle (RNP), which further enwraps a non-segmented single-stranded RNA genome. The virus has five structural proteins, namely glycoprotein (G), matrix protein (M), large protein (L), nucleocapsid protein (N) and phosphoprotein (P) (Fig. 1).



Source: National Action Plan for Dog Mediated Rabies Elimination from India by 2030

Fig 1: Structure of Chandipura virus- Rhabdoviridae family

Host

CHPV has been isolated from human sera during outbreak and non-outbreak periods. Also, during the 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.

Neutralizing antibodies against the Chandipura virus have also been found in animals such as cattle, goats, pigs, buffalos, sheep, camels, wild macaques, rodents, frogs, lizards.

Experimentally, cells of insect origin and vertebrate animals were found to be susceptible to virus replication. The rate of replication in susceptible cell lines is recorded to be extremely high (within 24-48 hours).

The susceptibility of mice and humans to CHPV infection is age-dependent. Exposure of children below 15 years of age, as determined by the presence of anti-CHPV N antibodies, was found significantly less than adults indicating susceptibility of children.

Incubation period

The incubation period is typically short, ranging from a few hours to 5-6 days.

Disease distribution

The disease is predominantly rural, occurring during early monsoon and mostly affecting pediatric population under 15 years.

Vector

The principal vector for CHPV transmission is sandfly. Majority of the viruses in the genus vesiculovirus are transmitted by Phlebotomine sand-flies. Similarly, genus *Phlebotomus* and *Sergentomyia* spp. are indicated as vector of CHPV in India.

Detection and isolation of CHPV from *Sergentomyia* spp. demonstrates their anthropophagic nature. The vectorial capacity of mosquitoes in the transmission of CHPV has been proven experimentally. Under laboratory conditions, Aedes aegypti was found to be highly susceptible and could transmit the virus more efficiently than others through vertical and venereal routes. However, there had been no confirmed isolation of the virus from mosquito vectors in the wild. Ticks have also been implicated.

Sand-flies- Phlebotomine sandflies are small, fragile, blood-sucking insects. Only female sand-flies bite and suck blood from mammals, reptiles, and birds for egg production. Key vectors for Chandipura Virus (CHPV), Leishmaniasis and some viral diseases, over 30 species in the genus Lutzomyia and 40 species in Phlebotomus are known to transmit human pathogens like bacteria, protozoa, and viruses. Notably, they transmit viruses from the families Bunyaviridae, Reoviridae, and Rhabdoviridae. In India, P. papatasi, P. argentipes and Sergentomyia sp. are vectors for the Chandipura Virus.

Fig 2: Sandfly Source: Entomology division, NCDC

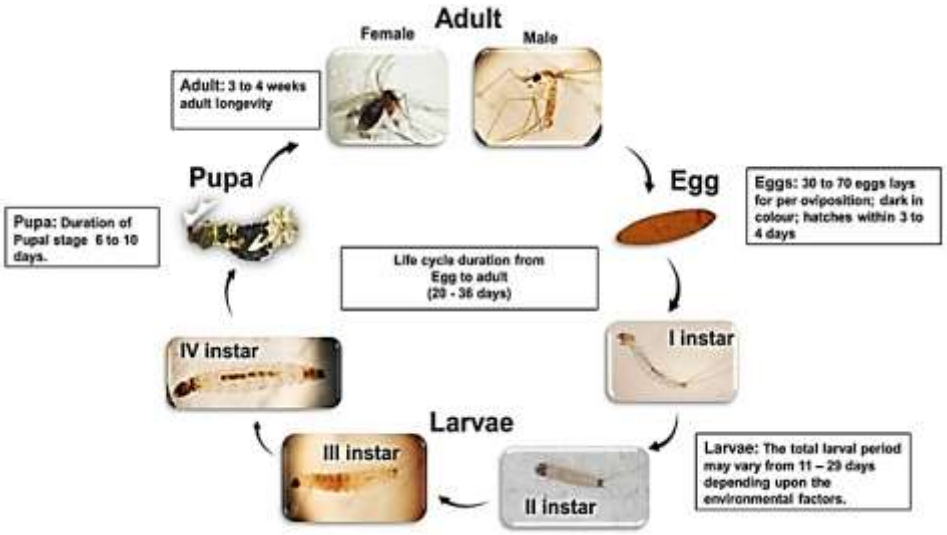


Figure 3: Life cycle of Phlebotomus argentipes

Life Cycle of Sandfly:

Egg: Females lay 30 to 70 eggs in moist, organic-rich environments. Eggs hatch in 3 to 4 days at 26± 2°C. **Larvae**: Creamy-white larvae with distinct head, thorax, and abdomen. It undergoes four instar stages, feeding on organic matter. The larval stage lasts 11 to 29 days.

Pupae: Pupae are comma-shaped and non-feeding, lasting 6 to 10 days. They turn from milky white to brown and exhibit distinctive caudal bristles. **Adult**: They are small, hairy insects 1.5 to 3 mm long. They exhibit unique wing venation and antenna structures useful for species identification. In males, two claspers are seen in the 10th abdominal segment, and in females has 2 small cerci in the 10th abdominal segment. The favorable conditions for sand fly breeding and feeding include alluvial soil with high sub-soil water (temperature 27 ± 2°C and a relative humidity >70%).

Reservoirs: The natural cycle of CHPV is to be understood fully; however, detection of anti-CHPV antibodies in rodents, cattle, goats, sheep, pigs, frogs, hedgehogs, and lizards indicates a wide range of hosts exposed to the virus in nature. Anti CHPV N antibodies have also been detected among healthy individuals with higher frequency among adults in past outbreaks (Andhra Pradesh 2003, Gujrat 2005), suggestive of possible endemicity and natural immunity.

Extrinsic incubation period of CHPV in P. argentipes is approximately 5–6 days. The short extrinsic incubation period favors rapid transmission of the virus.

Transmission:

Transovarial transmission (Laboratory confirmed) of the Chandipura virus (CHPV) has been reported in sandflies. CHPV passes through the midgut barrier within 4-5 days; subsequently, the virus crosses the salivary gland barrier within the next 24 hours, further allowing transmission to other vertebrate hosts.

Experimental studies with Phlebotomus papatasi demonstrating their potential not only to replicate the CHP virus but also to transmit the virus through vertical and venereal routes points towards maintenance of the virus in nature during the non-epidemic periods.

This mechanism could have helped the virus to remain dormant for prolonged periods and initiate outbreaks when sand-fly population increases under favourable conditions.

Additionally, experiments have shown that CHPV can multiply in Phlebotomus argentipes and can be transmitted by bite. The virus is disseminated to salivary gland by overcoming the midgut barrier within 4–5 days and is then transmitted to other vertebrate hosts by crossing the salivary gland barrier in the next 24 hours.

Vector biology

Breeding Habitat: Sandflies breed in damp soil in cattle sheds, human dwellings, rodent burrows, moist soils, often found in organic debris, etc.

Host Preference and Feeding Behavior: Sandflies are attracted to hosts by odor of CO2. Sandflies exhibit crepuscular (dawn and dusk) biting habits. Both male and female sandflies feed on plant juices and sugary secretions, and females also feed on animals or humans to produce eggs. Phlebotomus female sandflies take multiple blood meals to produce eggs.

Longevity: Adult sandflies can live 3 to 4 weeks. Generally, females having a longer lifespan than males. However, environmental conditions

influence the duration of longevity of sandflies.
Resting Sites: They seek dark, damp shelters with very high humidity and become active at dawn and dusk. Entomological assessments show presence of sand flies above 6ft in recent outbreaks.; but they cannot rest there for long and return to moisture. Phlebotomus sandflies prefer to rest indoors but also rest outdoors in considerable numbers.
Indoor resting sites include cracks and crevices in walls, roofs, floors, and rodent burrows.
Outdoor resting sites include cattle shelters, soil cracks, thick vegetations, tree holes, termite mounts, caves, bird tunnels, around the roots of large trees, bamboos, banana stumps, and rodent burrows.
Flight Range: Sandflies exhibit short hopping, typically covering less than 500 meters.

Seasonal prevalence: Sandflies proliferate in high humidity, warm temperatures, and abundant vegetation, causing seasonal prevalence to vary from region to region. In India, they are found year-round except in winter. Their density peaks during the onset of warm weather and monsoon/post-monsoon.

CASE DEFINITION

Suspected Case Definition of CHPV-Acute Encephalitis Syndrome (AES)

- Any case with age </= 15 years having

Abrupt onset of fever that may be associated with Vomiting and/or Diarrhea

With

- Change in mental status (irritability, somnolence or abnormal behavior greater than that seen with usual febrile illness)

& /OR

- New onset of seizures (that is not febrile seizures)

Probable case: A suspect case geographically/temporally linked to lab confirmed case.

Confirmed case: A suspect case with one or more of following laboratory results:

1. Presence of viral RNA by RT-PCR
2. IgM antibodies against CHP virus in blood/CSF by a validated serological test (ELISA) (In view of background positivity/ endemicity of Arboviral infections, single positive sample in serology must be cautiously interpreted)
3. Seroconversion in acute and convalescent blood/CSF samples by validated serological test
4. Virus isolation

CHPV can be either laboratory confirmed or probable when a suspected case with these features of AES is found in close geographic and temporal relationship to a laboratory confirmed case of CHPV in the context of an outbreak.

CLINICAL FEATURES

AES is defined by the World Health Organization (WHO) as “a syndrome in a person of any age, at any time of year, with the acute onset of fever and a change in mental status (including symptoms such as confusion, disorientation, coma, or inability to talk) AND/OR new onset of seizures (excluding simple febrile seizures).” CHPV is a priority pathogen causing Acute Encephalitis Syndrome.
The clinical presentation of CHPV infection can vary greatly, from sub-clinical to symptomatic cases which could be moderate and acutely life threatening. Cases can present as following

- Early Febrile symptoms:
- Sudden onset of high fever (often exceeding 39°C or 102.2°F)
- Headache
- Myalgia, Fatigue and malaise
- Nausea and vomiting
- Acute Neurological symptoms:
- Altered mental state (confusion, disorientation, drowsiness)
- Generalized convulsions
- Weakness or paralysis (can affect limbs or facial muscles)
- Signs of meningeal irritation (stiff neck, headache) (May rarely present as Aseptic meningitis)
- Decerebrate posture, leading to Grade IV coma within several hours
- Acute encephalitis / encephalopathy

Red Flag Signs for Referral for hospitalization: Refusal to feed, lethargy, altered sensorium, convulsion, bleeding from any site, decreased urine output, weak & fast pulses etc. in a suspected case of AES

In children: The disease affects mostly children under 15 years of age. The clinical picture consists of two phases.

1. Prodromal phase: Lasts 1–3 days prior, with fever, myalgia, malaise, and headache
2. Encephalitic phase: Persistent fever, decreasing level of consciousness, seizures, abnormal movements, or focal neurological deficits

Other clinical manifestations observed in past outbreaks were vesicular eruptions, bilateral crepitations, palpable liver and elevated levels of alanine aminotransferase and aspartate aminotransferase. Some cases may also have loose stools, respiratory problems, and decreased platelet counts.

Disease progression

Depending on the severity and age of the child and immune responses, some may succumb and some recover through post-encephalitic sequelae-cognitive decline, paresis, involuntary movements, and seizures. Some can have complete recovery.

Death may occur within 48-72 hours of symptom onset in absence of medical management. Rapid deterioration and mortality remain poorly understood, though hypotheses include encephalitis, acute brain events, spasms, or transient vasculitis-induced obstructions.

Case Fatality rate

Based on the past reported outbreaks of CHP virus, the case fatality rate ranges from 44% to as high as 78%.

DIAGNOSIS

The overall profile of investigations will depend upon clinician's assessment and possible differential diagnosis in a case presenting with AES. (Refer to the Investigations table III at Pg 7)

TREATMENT

The AES is a medical and neurological emergency, requiring immediate treatment. Hence ensure early hospitalization of the cases of AES.

Symptomatic treatment for syndromic approach of management of AES is as follows:

- **Rapid evaluation and stabilization before referral-** Earliest intervention starts at primary health care facility.
- **Maintain Airway.** Oxygen when needed to maintain SpO2 in normal range.
- Maintain Euglycemia. Monitor blood sugar and administer 10% glucose 5ml/kg stat when hypoglycemia is found.
  Intubate the patient having impaired airway reflexes, abnormal respiratory pattern, signs of raised ICP, oxygen saturation <92% despite high flow oxygen and GCS score <8 and begin
  assisted ventilation (i.e. intermittent positive pressure ventilation)
- Measures to maintain temperature.

Use correct size endotracheal tube (ET) as per table II below:

Age	Endotracheal tube size (internal diameter mm)
0-4 months	3-3.5
4-12 months	3.5-4
10-12 months	4.0
2-3 years	4.5
3-5 years	5.0
6-10 years	5.5
10-12 years	6.0

- ❖ If endotracheal intubation is not possible then patient may be shifted to higher facility with Laryngeal Mask Airway (LMA) or bag and mask ventilation.
- ❖ Rate of ventilation should be adjusted to 30-40 per minute in young infant for adequate oxygenation.

Shock management: Give 10-20 ml/Kg of Normal Saline IV bolus. Further fluid management should be based on response to bolus, causes and signs of shock and fluid requirement. Avoid overload of fluids.

Table III: **Investigations to establish the diagnosis and etiology of acute encephalitis:** (Transportation of samples to appropriate laboratory should be as per the guidelines.)	● CSF examination including cells, protein, sugar, microscopy, culture, and panel for neurotropic viruses. ● For virus studies: 3 ml of serum and 1 ml of CSF is to be sent to designated state laboratory/NIV Pune. ● Throat swab for viral PCR to be sent to NIV, Pune. ● Neuroimaging – Contrast enhanced MRI/CT scan of brain. ● CBC with peripheral smear. ● Rapid malaria Antigen Test
Investigations to look for organ dysfunction	● Random blood sugar ● Liver function test- S. Bilirubin, SGPT, SGOT ● Coagulation profile – PT, INR, aPTT, ● Renal function test – Blood Urea, Serum creatinine, S. Electrolytes ● If there is any evidence of cardiac dysfunction appropriate investigation may be asked for.
Investigations for systemic immune dysregulation	● Evidence of cytokine storm may be demonstrated by abnormal values of CRP, Ferritin and LDH.
Other Tests	● Any other Investigations if felt necessary by clinician.

- **Treatment of convulsion-**

<u>Loading dose</u>

❖ Injection Levetiracetam- Loading dose 40-60 mg/kg infused intravenously with normal saline over 5-15 minutes; maximum single dose is 4.5 grams.

❖ If Levetiracetam is not available, use Inj. Phenytoin Sodium 20 mg/kg/dose infused intravenously with normal saline over 20 minutes; maximum 1000 mg/dose;

Maintenance dose

❖ Inj. Levetiracetam 10mg/kg/dose intravenously twice daily initially and increase doses every 2 weeks by 10mg/kg/dose twice daily based on response up to the recommended dose of 30mg/kg/dose twice daily.

❖ Inj. Phenytoin Sodium 5 mg/kg/day (range 4-8 mg/kg/day); maximum 300 mg/day.

- **Treatment of active convulsion**

❖ Injection Lorazepam 0.1mg/kg (max. dose 4 mg) or Midazolam (0.1mg/kg) by intravenous route can be used while monitoring breathing. Benzodiazepines may aggravate hepatic injury if any so be cautious for its use.

❖ If intravenous or intramuscular routes are not accessible, intranasal midazolam at 0.2mg/kg (strengths available - 0.5mg/puff or 1.25 mg/puff) can be also given; total dose should be divided between two nostrils (for e.g; child with 10 kg weight – 2 puffs in each nostril). second spray may be used 10 minutes later if required

❖ OR Diazepam can be given per rectal 0.5 mg/kg in 2-6 years and 0.3mg/kg in 6-12 years

❖ Recovery position (as indicated in Fig 4) can prevent aspiration.

- **Hypoglycemia-** I.V. bolus of undiluted intravenous dextrose 2ml/kg 10% dextrose in neonate and 5ml/kg 10% dextrose in older children. (Monitor Blood glucose level). This is followed by IV drip- Glucose infusion at the rate of 6-8 mg/kg/min.

  Treat fever with Paracetamol in dose of 15mg/Kg/dose every 6 hourly.
- Vital monitoring is important during transport.
- **Management of raised intracranial pressure:** If there are features of raised intracranial pressure (headache, vomiting, blurring of vision, asymmetric pupils, tonic posturing, papilledema).

  Elevate head to 15-30 degrees if patient is not in shock. Keep the neck midline to facilitate venous drainage from the head.

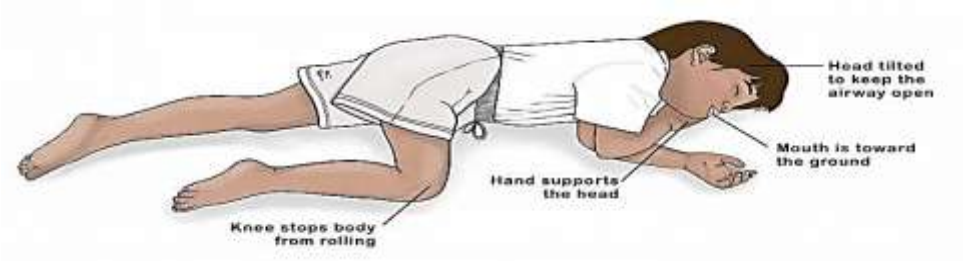


Fig 4: Recovery Position

- Glycerol 1.5 gm/kg every 6 hourly orally or thorough nasogastric tube is preferable. Duration should be decided based on response.
- Hypertonic (3%) saline is preferable to mannitol in the presence of hypotension, hypovolemia, and renal failure. Initial bolus dose of 3-5ml/kg to be given followed by maintenance at 0.5-1ml/kg/hour with serum Na+ monitoring 6 hourly (Target Na+ 145-160 meq/L). Too rapid treatment with hypertonic saline must be AVOIDED.
- Mannitol to be given IV in initial dose of 0.25-1g/kg/dose followed by 0.25-0.5g/kg/dose repeated 6 hourly for 48 hours

- Acyclovir to be started in suspected cases of herpes viral encephalitis in a dose of 10mg/kg I.V. 8 hourly. Duration of Acyclovir therapy is decided based on clinical course and investigations reports.
- A broad-spectrum antibiotic such as Injection Ceftriaxone in dose of 100mg/kg/day IV divided in 12 hourly doses to be given, as empirical therapy. Further treatment should be based on investigations reports.
- Anti-malarial (Artemisinin -based combination therapy) to be started based on peripheral smear for malaria parasite or RDT report.
- In suspected cases of Rickettsial disease causing AES, Doxycycline to be used in the dose of 2.2 mg/kg twice daily (maximum 100 mg per dose) for 7 days in children more than 40 kg weight or more than 8 years of age; in children less than 8 years of age give Inj Azithromycin 10mg/kg/day (maximum dose 500 mg per day) for 5-7 days.

  Duration of treatment depends upon response.

- **Acute liver injury** is cautiously treated and further worsening should be prevented. Blood products like Fresh Frozen Plasma (FFP) 10-15ml/kg & Injection Vitamin K 5- 10 mg I.V. as per PT INR for management and prevention of bleeding
- **Acute kidney injury** (AKI) as a complication of disease pathology should be managed with proper fluid resuscitation, pharmacotherapy, and dialysis. Maintain acid base balance and avoid nephrotoxic drugs.
- **Myocarditis** leading to heart failure, rhythm disturbances or cardiogenic shock should be managed cautiously with pharmacotherapy. Inj. **Dobutamine** to be used in dose of 2.5-20mcg/kg/min as continuous infusion (or Milrinone 0.25-0.75 mcg/kg/minute) while monitoring heart rate, blood pressure and capillary refilling time when the patient is normotensive; when hypotension is there, then use low dose Nor-epinephrine (0.02-0.2 mcg/kg/minute) or Epinephrine (0.1-0.2 mcg/kg/minute) infusion.
- Corticosteroids can be used if cytokine storm (macrophage activation syndrome) is suspected based on clinical condition and investigations.

❖ Inj. Methyl Prednisolone 1-2 mg/kg/dose in mild cases and 10-30 mg/kg/dose in severe cases, (maximum dose is 1000mg/dose) once daily for 5 days.

❖ If patient is in refractory shock, then Injection Hydrocortisone 1-2 mg/kg/dose (100mg/M^2/day in 3-4 doses) IV 6 hourly may be given.

For more details, refer to SOP FOR ACUTE ENCEPHALITIS SYNDROME (AES) MANAGEMENT – 2024

PREVENTIVE MEASURES

Vector control, hygiene, and public awareness are the only measures available against the disease.

Vector Control Methods

Indoor Residual Spray (IRS)

IRS involves the application of long-lasting insecticides on the inner walls of houses, killing sandflies that come into contact with treated surfaces.

Application and Requirement of Synthetic Pyrethroids:

There are 5 synthetic pyrethroids used in the program as per the Central Insecticide Board (CIB). These are Deltamethrin 2.5% WP, Cyfluthrin 10% WP, Lambdacyhalothrin 10% WP, Alphacypermethrin 5% WP, and Bifethrin 10% WP, as per the recommended dosage for malaria control programme by the Government of India and also used in sandfly control. Two rounds of synthetic pyrethroid indoor residual spraying (IRS) on all surfaces in domestic and peri-domestic areas that are sandfly-genic.

Target Areas: Focus on human and animal dwellings, especially in cracks, crevices, dark, moist places, rodent burrows and other places with soils rich in organic content.

Effectiveness:

Kills sand-flies that rest on treated surfaces, thereby reducing indoor vector populations. Reduces transmission risk by targeting sand-flies during their resting phase.

The sprayed surfaces should not be washed or mud plastered for a period of 3 months after the spray. This will prolong maximum effect of the insecticide.

Environmental Management:

Environmental management aims to reduce sandfly breeding sites and alter habitats to make them less conducive for sandfly proliferation.

Strategies:

- **Sanitation and cleanliness**: Regularly clean and remove organic debris in and around houses and animal shelters.
- **Vegetation Management**: Trim dense vegetation near dwellings, as thick foliage provides suitable shelter for resting and breeding sites.
- **Animal Shelter Management**: Maintain cleanliness and proper waste disposal in cattle sheds and animal shelters to reduce breeding sites.
- **Structural Modifications**: Seal the cracks and crevices in walls and floors with cement and use fine mesh screens on windows & doors to prevent sandfly entry.

Personal Protections:

- When possible, wear clothing to minimize the skin you have exposed ie., long sleeves skirt, pant, sock, tuck your shirt into your pants
- (wear full sleeves clothes) to reduce man vector contact.
- Use insect repellent like **DEET** (Diethyl-toluamide) etc.
- Sleeping on cots or beds will reduce the human vector contact.

CONTROL MEASURES DURING OUTBREAKS

- AES surveillance in under 15-year population.
- Phlebotomous papatasi is reported to be the vector of Chandipura Virus disease from Gujarat. It is not a vector of Visceral leishmaniasis (VL), that is transmitted by Phlebotomous argentipes. NCVBDC vector control strategy is Indoor Residual Spray (IRS) with synthetic pyrethroids like Alphacypermethrin 5% WP, 250gm of the same to be mixed in 10 litres of water. The residual efficacy is 10-12 weeks. IRS to be carried out in potential sandfly breeding places such as dark damp room, wall with cracks and cervices, storage area of fire wood, dung cakes. Ph. papatasi breeds and rests in cattle shades as well, hence in addition to human dwellings cattle shades are also to be sprayed.
- If the areas reporting Chandipura cases are not targeted for regular IRS rounds for malaria, even then the affected villages should be sprayed with Alphacypermethrin 5% as a focal round covering 50 houses (including cattle shed) or 500 meters of radius.
- Normally for VL, up to 6 feet of height is recommended for spray. In the current situation, as the affected areas are endemic for malaria as well, all surfaces in domestic and peri-domestic areas that are sandfly-genic should be sprayed.
- Use of Malathion 5% dust and Lindane is NOT recommended.
- Use repellents like DEET at least during close contact with cattle and before going to sleep.
- Information Education and Communication (IEC): Raising awareness about sandfly vector, Chandipura virus symptoms, promoting early medical consultation,

prevention and control measures and emphasising hygiene practices to reduce transmission risk.

VACCINATION

Currently, there is no vaccine available for CHPV

KEY MESSAGES

- Chandipura virus (CHPV), an emerging tropical pathogen, belongs to genus vesiculovirus of Rhabdoviridae family.
- It has caused explosive outbreaks in Maharashtra, Gujarat and Andhra Pradesh and has emerged as an important encephalitis-causing pathogen in India.
- The virus has been isolated from *Phlebotomine* and *Sergentomyia* species of sand-flies which is incriminated as principal vector of CHPV.
- It affects mostly paediatric population under 15 years and is characterized neurologic dysfunctions with high fatality rates.
- There is no evidence of human to human transmission.
- The AES is a medical and neurological emergency, requiring immediate treatment. Hence early hospitalization of the cases of AES is to be ensured.
- Symptomatic treatment for syndromic approach of management of AES is to be followed as per the SOP FOR ACUTE ENCEPHALITIS SYNDROME (AES) MANAGEMENT – 2024
- Understanding the virus's transmission dynamics, recognising its clinical manifestations, and implementing effective prevention and control strategies are essential for mitigating its impact on vulnerable populations.
- Continued research efforts, collaborative initiatives among health authorities, and community engagement are pivotal in developing targeted interventions, including vaccines and therapeutics, to combat Chandipura virus infections effectively.
- Proactive surveillance, vector control measures, community education, and early medical intervention play critical roles in reducing Chandipura virus transmission and mitigating its public health impact.

….about CD Alert

CD Alert is a technical bulletin of the National Centre for Disease Control (NCDC), Directorate General of Health Services, Ministry of Health & Family Welfare, Government of India, to disseminate specific and updated information on communicable diseases to medical fraternity and health administrators. The bulletin may be reproduced, in part or whole, for educational purposes, with due acknowledgement.

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