



Science

## **STUDY ON CLINICO-ETIOLOGICAL CORELATION AND OUTCOME OF VIRAL MENINGOENCEPHALITIS IN PEDIATRIC AGE GROUP IN A TERTIARY CARE CENTRE**

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### **Abstract**

Meningoencephalitis remains the most significant cause of childhood deaths indeed a more measure cause of long term neurological handicap in children. Herpes simplex virus (HSV) encephalitis is recognized worldwide as the most frequent infectious encephalitis and the only one with a validated specific treatment. The incidence of viral encephalitis is 3.5-7.4 per 100,000 people per year. Aim & objectives: To study the demographic profile, etiological agent, clinical presentation, investigation findings and outcome of viral mengingoencephalitis in children. Material & Methods: The study was conducted in the pediatric department of SCB Medical College, Cuttack Odisha. Result: Highest incidence was observed in 1-5 years age group followed by 6-14 years. Males are more affected than Female. Highest number of cases occurred in summer season (45.2%). The common symptoms were fever (97.6%), irritability (80.9%), refusal to feed (80.9%), vomiting (80.9%), altered sensorium (76.1%), headache (61.9%), convulsion (52.3%). The common signs observed were temperature > 100.4 F (94.1%), meningeal sign (52.3%), GCS (Glasgow Coma Score) < 7 (52.3%), hypotension (50%), focal neurological deficit (52%). Viral marker were detected in 47.7% of cases. HSV (Herpes Simplex Virus) was common agent found among the positive viral marker detected. Conclusion: Viral mengingoencephalitis is a major health problem due to high morbidity and mortality. Early diagnosis is often difficult due to low incidence of disease specific signs. Detection of viral antibody is done by cerebrospinal fluid (CSF) Ig M & Ig G. PCR(Polymerase Chain Reaction) method is suggested as a better option which has more sensitivity and specificity and gives a rapid diagnosis.

**Keywords:** Meningoencephalitis; Herpes Simplex Virus; Polymerase Chain Reaction.

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## 1. Introduction

Meningoencephalitis remains the most significant cause of childhood deaths indeed a more measure cause of long term neurological handicap in children. HSV encephalitis is the most common form of viral encephalitis and has an incidence of 2-4 cases per 1 million population per year and accounts for 10% of all cases of encephalitis in the United States [1]. The annual incidence of viral encephalitis is most likely underestimated, especially in developing countries, because of problems with pathogen detection [2]. Japanese encephalitis (JE) affects at least 50,000 individuals per year. In a study from Finland by Rantalaiho T et al (1991) the incidence of viral encephalitis in adults was 1.4cases per 100,000 persons per years. HSV was the organism most frequently identified as the cause (16%), followed by VZV (5%), Mumps virus (4%) and Influenza A virus (4%) [3]. According to Long SS et al the clinical hallmark of acute encephalitis is the triad of fever, headache and altered mental status. Most of the patient with viral encephalitis presents with the symptoms of meningitis (fever, headache, neck stiffness, vomiting) followed by altered consciousness, convulsions and sometimes focal neurological signs, signs of raised intracranial pressure or psychiatric symptoms. There may be an association with history of infection elsewhere in the body [4]. Subacute sclerosing panencephalitis is a late complication of measles and presents four to ten years after the initial infection. Progression may be slow or rapid with personality change, dementia, seizures, ataxia and death. Progression of rubella panencephalitis is similar. Complications of viral meningoencephalitis are not infrequent, can develop early in the course of illness. Acute complication includes seizure, increase ICP, Coma, infarction, focal neurological deficit, ventriculitis. Long term sequelae are visual impairment, hearing defect, persistent seizure, behavioral disorder, mental retardation and developmental delay.

Thus, for reduction of high mortality and morbidity of viral meningoencephalitis in children there is a clear need for analyzing the clinical parameters of the disease. At the present time effective viral vaccines for Polio, Measles, Mumps, Rubella and Varicella are widely available but nevertheless prudent clinical judgment and timely intervention are highly warranted.

Keeping all these in mind, the present study was undertaken to determine the possible clinical and investigative clue of disease etiology as well as identifying those presenting clinical parameters which correlate with disease outcome.

## 2. Aim and Objectives

To study the demographic profile, etiological agent, clinical presentation, investigation findings and outcome of viral meningoencephalitis in relation to death, recovery and sequelae in children.

## 3. Methodology

After obtaining clearance from the institutional ethical committee the study was conducted in the pediatrics department of S C B Medical College, Cuttack. This is a prospective study done over a period of September 2015 to August 2017. All the babies admitted through OPD or emergency department with suggestive signs and symptoms of viral meningoencephalitis like fever, refusal to feeds, headache, vomiting, convulsions altered sensorium, lethargy were admitted and thorough systemic examination, biochemical and microbiological examinations of blood and CSF were done and those patients satisfying the inclusion criteria were included in the study.

Inclusion Criteria: i. age between 1month to 14 years. ii. C S F showing protein 50-200mg/dl, sugar normal or <40mg/dl, cells<1000/cmm.

Exclusion Criteria: i. Neonatal age group 0-1 months baby, ii. Those with clinical suspension and not evaluated and leave against medical advice. iii. Parents who did not gave consent for the study. iv. Alternative diagnosis was made during the course of hospitalization. All the cases included in the study were treated empirically with antiviral agents and as needed with antibiotics. Appropriate supporting care including attention to fluids, electrolytes, ventilations and parenteral nutrition was provided. 42 cases were included in our study after the criteria were satisfied.

All the studied cases were analyzed for various clinocobiochemical parameters in terms of outcome of the disease. CSF was cultured on specific media. Other investigations included complete blood counts, Chest X-rays, renal and liver function test, electrolytes, blood and urine culture. C-reactive protein (CRP) was determined in serum and categorized as positive or negative. HSV-1 & HSV-2 IgM detection was done by HSV-1& HSV-2 IgM fast EIA KIT. PCR, an invitro DNA amplification procedure in which millions of copies of a particular sequence could be produced within few hours. In this method detection of HSV-1, HSV-2 & VZA DNA were done in acute phase of disease.

Statistical analysis was done by chi-square test whenever applicable. P –value of <0.05 was considered to be significant.

#### 4. Observation

Table 1: Age and Sex distribution

Age	No of cases	Male	Female	Percentage in total
<1 year	3	2	1	7
1-5 year	20	13	7	48
6-14 year	19	9	10	45

The incidence of viral meningitis in our study 7% were infant,48% were from 1-5 years and 45% were included in 6-14 years of age group.

Table 2: Seasonal variation

Summer	19
Rainy	15
Winter	8

Our study showed highest seasonal occurrence of viral meningoencephalitis in summer 45.2% cases, followed by 36.8% in rainy and 19%in winter.

Table 3: Symptoms Observed

Symptoms	Survived (n=34)	Died (n=8)	Total (n=42)
Fever	34	7	41
Vomiting	27	7	34
Convulsion	22	6	22

Parotid swelling	12	0	12
Irritability	27	7	34
Headache	19	5	26
Refusal to feed	27	7	34
Altered sensorium	24	8	32
Lethargy	3	2	5

The fever was the commonest symptoms in all groups with total incidence of 97.6% followed by irritability (80.9%), refusal to feed (80.9%), altered sensorium (76.1%), headache (61.9%), convulsion (52.3%), parotid swelling (28.7%), lethargy (11.9%).

Table 4: Sign observed

Signs	Survived (n=34)	Died (n=8)	Total (n=42)
Temp >100.4F	32	6	40
RTI	8	4	12
GCS<7	15	7	22
Meningeal sign	16	6	22
Bulging fontanel	3	5	8
Weakness of limbs	5	2	7
Papilledema	5	6	11
Hypotension	14	7	21
Focal neurological deficit	17	5	22

The signs observed temperature >100.4 F was most common sign in 32(94.1%) children, followed by Meningeal sign in 52.3% children, GCS < 7(52.3%), hypotension (50%), focal neurological deficit (52%).

Table 5: Biochemical parameter

Parameter	Levels	Survived (n=34)	Died (n=8)	Total (n=42)
Cell count	<50/cmm	18	8	26
	50-1000/cmm	16	0	16
Sugar	<40mg/dl	14	2	16
	>40mg/dl	20	6	26
Protein	<45mg/dl	12	3	15
	>45mg/dl	62	5	27
Viral marker	+ve	12	8	20
	-ve	22	0	22
Hb	<10gm%	1	5	6
TLC	>1100/cmm	2	5	7
Serum CRP	>6mg/L	13	7	20
LFT	Abnormal	1	5	6
RFT	Abnormal	2	6	8
CT/MRI brain abnormality	Normal	21	-	21
	Swelling of parenchyma	3	-	3
	Infaction	4	-	4

On CSF study, cell count was found to be less than 50 /cumm in all patients who died and 56.3% of cases who survived. CSF sugar was found at <40mg/dl in 38% cases and 27% of cases were found protein value >45mg/dl. The different hematological parameters in meningoencephalitis in our study was serum CRP >6mg/L most frequent (47%), followed by TLC >11000/cumm in 16.7% of patient and Hb <10gm% in 14.2% of patients. High abnormal LFT (75%) and abnormal RFT (87.5%) were found in cases who are died. The CT and MRI brain abnormalities in meningoencephalitis shows 75% normal, swelling of parenchyma in 12% & Infarction in 13% of cases.

Table 6: Clinico-etiological correlation of viral meningoencephalitis

Features	Total (n=42)	HSV(n=12)	HZV(n=11)	Others (n=19)
Convulsion	22(52.4%)	7(58.3%)	7(63.6%)	8(42.1%)
Altered sensorium	34 (100%)	11(91.6%)	10(90.9%)	13(68.4%)
Meningeal sign	22(52.4%)	11(91.6%)	3(27.2%)	8(42.1%)
Papiledema	11(26.2%)	7(58.3%)	4(36.3%)	0(0%)
RTI	12(28.5%)	4(33.3%)	4(36.3%)	4(21.0%)
CSF cell count >50/cmm	16(38.0%)	4(33.3%)	0(0%)	12(63.1%)
CSF protein>45mg/dl	18(42.9%)	8(66.6%)	7(63.6%)	3(15.7%)
CSF sugar <40mg/dl	16(38.0%)	1(8.3%)	4(36.3%)	11(57.8%)

In Clinical presentation of our study showed altered sensorium in 13(68.4%) followed by convulsion & meningeal sign each 8(42.1%) of cases. in CSF study >50 cells count /cumm was found in >50% of the group. CSF protein was high in both HZV 6(66.6%) & VZV 7(63.7%) cases. Low CSF sugar was found in others 11(57.8%) cases.

Table 7: Comparison of CSF parameters between HSV &HZV Meningoencephalitis

Features	HSV	HZV	P-value
Mean CSF cell count	166.36± 206.68	4±5.82	1.00
Mean CSF protein	79.82±49.15	64.80±34.02	0.430
Mean CSF sugar	69.09±15.97	54.70±22.17	0.101

The CSF parameters between HSV & VZV meningoencephalitis showed no statistically significant among both groups.

Table 8: Comparison of outcome among HSV& HZV

Parameter	HSV	HZV	P-value
Mortality	7	1	0.0415
Sequelae	4	2	0.7253
Deafness	1	0	0.3276
Seizure	1	0	0.3276
Motor incordination	1	1	0.9486
Behavioral disorder	1	0	0.3276
Cranial nerve palsy	0	1	0.9645

The mortality in HSV was 7(58.5%) which was highest and statistically significant

Table 9: Correlation of parameters who survived and died

Parameters	Total (n=42)	Died(n=8)	Survived(n=34)	p-value
RTI	12	4	8	0.290
Altered sensorium	34	8	24	0.277
Convulsion	22	7	15	0.069
GCS<7	20	7	13	0.034
Intubation	15	6	9	0.030
Duration of illness>7 days	16	6	10	0.047

Among the different parameters studied, the incidence of GCS <7, intubation of with respiratory failure and duration of illness >5days were statistically significant among survivors and died.

Table 10: Comparison between parameters who survived and died

Parameters	Died (n=8)	Survived(n=34)	p-value
Mean CSF cell count	16±6.72	138.91±135.66	0.0374
Mean CSF protein	84.88±57.56	59.94±35.49	0.1249
Mean CSF sugar	64.50±33.22	61.81±28.30	0.8176

The mean CSF Cell count /cumm was statistically significant.

## 5. Discussion

The incidence of viral meningitis in our study 7% were infant, 48% were from 1-5 years and 45% were included in 6-14 years of age group. SA Fattah et al [5] reported 5-18 years age group comprises the highest number 44%. Our studies were similar to the finding of Aurelian L et al [6] and DeBiasi RL [7]. We observed 57.1% were male babies and 42.9% were female with a ratio of 1.3:1. Rantakallio P et al [8] reported 28.8% were female and 71.2% were male. Similar observations were made by Sejvar JJ [9], M Ward et al [10], Cochi et al [11].SA Fattah et al [5] reported Male-Female ratio was 33:17 which is similar to our study.

Our study showed highest seasonal occurrence of viral meningoencephalitis in summer 45.2% cases, followed by 36.8% in rainy and 19% in winter. Chalrs G, Prober and Laurl Dyer et al [12] show most cases in temperate climate during the summer and early rainfall. This is similar to our study. SA Fattah [5] reported patients were admitted round the year, higher during July – November. Rantakallio P et al [8] reported cases in month of May June and July. This discordant was may be due to regional variation.

In our study fever was the commonest symptoms in all groups with total incidence of 97.6% followed by irritability (80.9%), refusal to feed (80.9%), altered sensorium (76.1%), headache (61.9%), convulsion (52.3%), parotid swelling (28.7%), lethargy (11.9%). Predominance of common symptoms which overlap with other disease makes it difficult to pin point the diagnosis of meningoencephalitis. Similar features were reported by SA Fattah et al [5] neurological features (85%), 6% with pulmonary, 7% with combined neurological and pulmonary and 2% with other features. 13. Swadron SP [13] studied that febrile illness signs and symptoms of neuroinvasive disease such as headache, delirium, coma or new onset seizures were common features of meningoencephalitis. Schutte CM, Van Der Meyden CH et al [14] reported that most

common symptoms at presentation were fever in 77.5%, impaired consciousness in 56.5% and seizures 55.1% cases. 52.3% had GCS of 11-15 at presentation. According to Mustafa A et al [15] the most common clinical presentations were fever impaired consciousness, seizures, vomiting, behavioral changes, recent rash and recent history of seizures and respiratory tract infections. Steiner I et al [16] studied that generalized convulsions along with altered sensorium were significant findings in patient with viral encephalitis. Rantakallio P et al [8] shows fever in 78%, impaired consciousness in 57%, seizures in 55% and vomiting in 31% of cases. Higher incidence of neurological features in their study might be due to better identification of these features by the parents compared to our study where parents are not educated and fever was common complain. So incidence of fever was highest in our study.

Of all these signs observed temperature  $>100.4$  F was most common sign in 32(94.1%) children, followed by Meningeal sign in 52.3% children, GCS $<7$ (52.3%), hypotension (50%), focal neurological deficit (52%), features of RTI (28.5%) cases were found. Papilledema (26%), bulging fontanel (19%), weakness of the limbs (16.6%) were other presenting features. GCS  $<7$  (87.5%), hypotension (87.5%) were main signs who died. Suchett CM et al [14] reported 88% of patients with a GCS value of  $>12$  had good neurological outcome, while 88% of these of these with a GCS value of  $\leq 8$  had a poor outcome. Rantakallio P et al [8] reported that 52.3% had a GCS of 11-15 at presentation, RTI in 25%, neurological illness 19%, papilledema 28%, meningeal sign in 49% of cases.

On CSF study, cell count was found to be less than 50 /cumm in all patients who died and 56.3% of cases who survived. CSF sugar was found at  $<40$ mg/dl in 38% cases and 27% of cases were found protein value  $>45$ mg/dl. According to Rantakallio P et al [8] CSF cell count 0-50/cumm in 41%, CSF protein  $>45$ gm/dl in 22%, CSF sugar 50-75mg/dl in 60%, in 9% cases. Viral markers were detected in 47.7% of cases. The cases who were died, all (100%) had positive viral markers. Steiner I [16] observed the most common etiology of VE was enterovirus 71(42.1%), followed by measles(21.1%), varicella zoster virus (15.8%), herpes simplex virus (10.5%) and mumps(10.5%). According to Fidan Jmor et al [17] the most commonly identified agents based on virological and serological studies were VZV (24 cases), mumps virus (8 cases), HSV (7 cases) and measles virus (4 cases). The aetiology remain unclear in 37 children (39% cases). Rantalaiho et al [18] studied that in  $<14$  yrs age group 38.1% had positive viral marker. Rautonen J et al [19] reported both anti-HSV IgG and IgM antibodies were positive in 3(27%) of the 11 acute sera, where as a fourth sample was positive for anti-HSV IgG antibodies only. Simultaneously CSF analysis for HSV PCR was done within 5 days of illness in 6 of these 11 patients and it revealed positive in 2(33%).

The different hematological parameters in meningoencephalitis in our study was serum CRP  $>6$ mg/L most frequent (47%), followed by TLC  $>11000$ /cumm in 16.7% of patient and Hb  $<10$ gm% in 14.2% of patients. High abnormal LFT (75%) and abnormal RFT (87.5%) was found in cases who are died. Rantakallio P et al [8] shows that none of the hematological investigations revealed any significant trend. Serum Sodium was normal in 72.5% cases. TLC count  $>11000$  in 47% of cases.

The CT and MRI brain abnormalities in meningoencephalitis show 75% normal. Among abnormal finding swelling of the brain parenchyma was present in 10.7% and infarction in 14.2% cases.

Rautonen J et al [19] showed that C T scan revealed unilateral or bilateral hypodensity in temporal lobe with or without involvement of other areas(frontal, parietal or occipital) in 8(57%) of 14 patients. In 2(14%) patients the abnormalities were confined to either frontal or occipitoparietal lobe. in those 10 patients, the initial CT scan done on days1-8 from onset of symptoms was normal (50%). Hyper dense lesions of hemorrhage were observed in 6(60%) patients. Follow up CT scan of brain in the chronic stage of disease done for 3 patients' revealed encephalomalacia and brain atrophy. On MRI, done during the acute stage hyperintense signal (on T2-weighted images) and /or restricted diffusion abnormalities (on diffusion-weighted images) were seen in 10 (91%) of 11 patients. These correspond to edematous changes in the temporal lobes in 8(73%), while in 2(18%) patients localization was either to frontal lobe or parietooccipital region. Only one of 11(9%) MRI scan revealed no abnormality.

Out of 42 cases 8 (19%) patient died, 6 (14.2%) had some form of sequelae and 28(66.7%) recovered completely. Mortality was highest in age group of 1-5 years. Wong V, Yeung CY [20] showed that the mortality rate was 28%. Among the 41 survivors, 76 % were completely normal and 24% had neurological sequelae with focal neurological deficit in 29%, personality changes in 6%, moderate mental retardation in 2%; severe mental retardation in 4%, hyperactivity in 4% and epilepsy in 4%. The best predictors to unfavorable outcome were rapid rate of deterioration in conscious level after admission and the age of the patients. According to SA Fattah et al [5] outcome of patients showed 74% recovery, 19% death, and 7% referral to higher center. Rantakallio P [21] reported in his study out of 147 cases, 24(16.3%) died. For remainder at discharge, 48(32.7%) had major sequelae, 19(12.9%) had minor sequelae and 56(38.1%) recovered completely. Of the major sequelae seen, the commonest was motor deficits in 26(17.6%) followed by seizures in 14(9.5%) cases. Rautonen J et al [19] showed that the mortality rate of children with acute encephalitis is 3.8-28.04%. a study done by American paediatric population by Doachowske J B[22] showed that 40% of children who recovered from acute encephalitis had persistent non-progressive neurological abnormalities.

Out of 34 cases who survived 6(17.6%) cases showed sequelae at the time of discharge. Motor incoordination was present in 2(5.8%) cases. seizure disorder behavioral disorder, deafness; cranial nerve palsy each was present in 1(2.9%) cases. Rantakallio P [21] showed that at discharge, 48(32.7%) had major sequelae, 19(12.9%) had minor sequelae and 56(38.1%) recovered completely. Of the major sequelae seen, the commonest was motor deficits in 17.5%, followed by seizures in 9.5% cases. The more common minor sequelae seen at discharge were subtle neurological deficit in 7.5% and behavioral changes in 2% cases.

In Clinical presentation of our study showed altered sensorium in 13(68.4%) followed by convulsion & meningeal sign each 8(42.1%) of cases. in CSF study >50 cells count /cumm was found in >50% of the group. CSF protein was high in both HZV 6(66.6%) & VZV 7(63.7%) cases. Low CSF sugar was found in others 11(57.8%) cases. Mustafa A., Salih M et al [15] showed that the commonest initial presenting symptoms and signs were fever (100%), seizures (72%), irritability (50%) and weakness/ hemiparesis (39%). Wong V et al [20] reported that CSF pleocytosis is usually present in about 97%of cases, it may be absent in either immunocompetent or immunocompromised, elevated protein levels (65%) cases.



The comparison of clinical parameters, CSF parameters between HSV & VZV meningoencephalitis showed no statistically significant among both groups. According to Ugo K et al[23] nausea or vomiting 78% in HSV & 50% in VZV neck stiffness 100% in HSV & 38% in VZV. CSF Protein level is significantly high in patient with HSV type2 (median1205mg/dl) and in samples from those infected with ZVZ (median974mg/L). This is similar to our study.

Rantakallio P et al[8] shows that CSF cell count was significantly higher in patient with HSV-2 infection than in patients with enterovirus infection( $P<0.01$ ), and CSF protein levels were significantly higher in both patients with HSV-2 infection and patient with ZVZ infection. The CSF protein levels are significantly higher in CSF sample from patients infected with HSV type 2 and in sample from those infected with VZV.

The mortality in HSV was 58.5% which was highest, morbidity was 33.3%. The common sequelae were motor incoordination 4.7%, deafness 8.3%, seizures 8.3%, and behavioral disorder 8.3%. Our study is similar to the study done by Misra UK et al[24]. Rantakallio P et al [8] reported at the time of discharge 49% of patient either died or had major sequelae. This is in contrast with study by Rautonen et al [19] in which 9.5% of cases died or had severe damage. This discrepancy could be due to difference in race, etiological agent, the duration of illness at which the patient presented and inability of the family to afford tertiary care resulting in the patient being discharged once stabilized i.e. before optimum therapy. The latter limitation would make the patient appear worse off than those admitted for a longer period.

Among the different parameters studied, the incidence of GCS <7, intubation of with respiratory failure and duration of illness >5days were statistically significant among survivors and died. Our study was in accordance with the study done by Kennedy et al [25]. Rautonen et al [19] found that history of an onset of greater than 3days showed that an association with poor out comes.

## 6. Summary

Out of 42 cases 7% were infant, 48% were from 1-5years and 45% were from 6-14 years age group. As regards to sex distribution 57.1% were male babies, 42.9% were female babies. Highest number of cases occurred in summer season (45.2%). The common symptoms were fever (97.6%) irritability (80.9%), refusal to feed (80.9%), vomiting (80.9%), altered sensorium (76.1%), headache (61.9%), convulsion (52.3%), parotid swelling (28.7%), and lethargy (11.9%). The common signs observed were fever in 94.1%, meningeal sign in 52.3%, GCS<7 in 52.3%, hypotension 50%, focal neurological deficit 52% , features of RTI in 28.5% cases. Other presenting features were papilledema in 26%, bulging fontanales in 19% and weakness of limbs in 16.6% of cases. Cell count was found < 50/cmm in all patients who were died and 56.3% of cases who were survived. Viral markers were detected 47.7% of cases. The cases who died, all (100%) had positive viral markers. On radiological assessment about 75% had normal finding. Among abnormal finding swelling of the brain parenchyma was present in 10.7% and infarction in 14.2% cases. 19% patient had died, 14.2% had some form of sequelae and 66.7% had complete recovery. Mortality was highest in age group of 1-5 years. HSV was common agent found among the positive viral marker detected cases. GCS<7 and Intubation had significant higher association with mortality. CSF protein was high in both HZV (66.6%) & VZV (63.7%) cases. Gram stain and CSF culture was negative in all cases.

## 7. Conclusion

Viral meningoencephalitis is a major health problem due to high morbidity and mortality. Early diagnosis of disease is often difficult due to low incidence of disease specific signs. So early suspicion and a proper lumbar puncture can only help in early diagnosis. Over the years mortality of viral meningoencephalitis have been substantially reduced in spite of major advance in treatment modalities. Thus prevention is main stay to reduce the mortality and morbidity. Better management and prevention of sequelae can be possible by early detection of complication by wider and prudent use of MRI and CT scan. The incidence and sequelae of meningoencephalitis due to VZV and Mumps can be prevented by vaccination. Unavailability of specific diagnostic modalities to majority of children at remote unreachable places hinders formulating an effective treatment policy and adaptation of preventable strategies. Detection of viral antibody is done by CSF IgM & IgG. PCR method is suggested as a better option which has more sensitivity and specificity and gives a rapid diagnosis. Thus this can be useful in secondary care level also, from where majority of cases being referred and CSF viral culture facility is not available.

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