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Cases Of Viral Meningoencephalitis Presented In A Month Of January-February 2022 In Tertiary Care Centre In Southwestern Maharashtra

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Abstract

Background

Meningitis and meningoencephalitis are the serious and life threatening infection of the Brain. The outcome mainly depends on the host immunity and the pathogen . Prompt recognition of signs and symptoms of underlying disease and Correct diagnosis can lead to favorable outcome and can prevent the extent of serious long term or permanent Brain injury. Despite new availability of technologies for diagnosis correct diagnosis and exact pathogen causing disease is still a concern. This study shows the sudden outbreak of cases in southwestern Maharashtra with similar clinical presentation and with prompt treatment favorable outcome was noted.

Result

Total 10 cases of viral meningoencephalitis were admitted in a month of January and February 2022 in our tertiary care centre. Most of the cases i.e. 50% cases were seen having age more than 60 years of age, mean age 55.90±16.07 was observed. Out of 10 cases 50% were male cases and 50% were female cases. Altered Mentation (90%) was the most common clinical symptom seen in viral menigoencephalitis cases followed by Headache (70%), fever (50%), vomiting (50%), Brudginski (30%), Kernig (30%) and Seizure (30%). Of all the cases CSF analysis was done and protein, Sugar, LDH, ADA, RBCs(30%) . HSV AND Blood for Hb, TLC, Platelet count, Blood Urea, Sr. Creatinine (RFT), SGOT, SGPT, Direct bilirubin, Indirect bilirubin, Total bilirubin, Albumin, Globulin and Total protein were observed. 30% of cases shows derangement in RFT and LFT suggestive of MODF. Six out of Ten patients were able to receive microbiological confirmation (positive HSV PCR in CSF)

Conclusion

Viral meningoencephalitis is a devastating disease, which needs early recognition and prompt treatment in order to improve mortality and morbidity. Newer diagnostic and treatment modalities present a promising picture, although the disease burden still remains high.

Keywords: NIL

Introduction

The central nervous system (CNS) involvement is a unique feature of human viral infection. The severity of brain involvement and the disease's outcome are determined by the pathogen, the host's immunologic status, and environmental factors. Meningitis,

encephalitis, and meningoencephalitis are all symptoms of central nervous system infection, which can cause major public health problems, especially during outbreaks. [1] In many respects, infections of the central nervous system differ from infections of other body systems. The central nervous system is housed within a hard skull that is encased in layers of meninges and lacks lymphatics causing brain oedema. If infections of the central nervous system are not detected and treated promptly and aggressively, they can quickly spread and cause significant damage or even death. [2] Infectious diseases affecting the central nervous system, despite breakthroughs in antibiotic therapy and resuscitation, continue to be life-threatening, particularly in developing nations. [3] Bacteria, viruses, fungi, and parasites are among the pathogens that cause acute meningoencephalitis. [4] Meningoencephalitis is a condition in which the inflammatory process affects not only the meninges but also the brain parenchyma. Streptococcus pneumoniae, Neisseria meningitides, Group B Streptococcus Listeria monocytogene, and Haemophilus influenzae are the most common organisms that cause community-acquired bacterial meningitis. [5] Viruses are thought to be the most common cause of acute meningoencephalitis. Most cases of viral encephalitis in immunocompetent people are caused by herpes simplex virus type 1 (HSV-1), varicella-zoster virus (VZV), Epstein–Barr virus (EBV), mumps, measles, and enteroviruses [6]. The most prevalent etiological agents known to cause aseptic meningitis are enteroviruses. The herpes simplex virus is the most common cause of sporadic encephalitis worldwide. In immunocompromised patients, notably those on longterm steroid medication, fungal infections are more likely. [7] Although particular therapy is limited to a few viral agents, accurate diagnosis and supportive and symptomatic treatment (in the absence of specific therapy) are essential for the best prognosis (for reviews, see [1–7]).

Herpes simplex virus (HSV) is the most common infectious cause of sporadic encephalitis worldwide, with HSV-1 being the most common strain to cause HSV encephalitis (HSVE) . After primary mucocutaneous infection, HSV remains latent in sensory ganglia and 60–90% of the world's population is estimated to be seropositive for HSV . Possible pathogenic mechanisms for HSVE include reactivation of latent HSV in the trigeminal ganglia, with subsequent spread of infection to the temporal and frontal lobes, primary central nervous system (CNS) infection, and perhaps reactivation of latent virus within the brain parenchyma itself [8]. The introduction of acyclovir has decreased mortality rates from 70% to 15% , but early diagnosis and treatment initiation are major determinants of mortality [9].

Immunocompromised individuals seem to be at higher risk for severe HSV infections including meningoencephalitis , but the precise contribution of steroids remains unclear. Owing to their anti-inflammatory and immunomodulatory effects, glucocorticoids might in theory increase the rate of viral replication, but experimental animal models have yielded conflicting results regarding their effect on replication and clearance of HSV in the CNS [10]. Case reports have shown temporal associations between steroids and HSVE, but the relative contribution of steroids remains unclear because other factors, such as concomitant chemotherapy or direct insult to the CNS (radiation , surgery or close structures (eye , may have contributed to the HSVE in most of these cases. Here, we present a case of HSV-1 meningoencephalitis that developed following pulse-dose steroids for the treatment of interstitial lung disease in a patient with no history of chemotherapy or CNS insult. This case highlights the potential risk of pulse-dose steroids in patients with associated risk factors (age, critical illness immune paralysis, lymphopenia) and the need to rule out CNS infection in case of persistent, unexplained encephalopathy in critically ill patients.[10]

This, however, is depending on the continent as well as environmental conditions. Because the nervous system's anatomy determines the degree of inflammatory meningeal involvement in encephalitis, symptoms that resemble meningitis are invariably associated with encephalitis. Furthermore, the term 'viral meningoencephalitis' is frequently used in textbooks and review papers to refer to a viral infection of both the brain/spinal cord and the meninges. The general investigation of a patient with febrile sickness includes general investigations such as chest X-rays and blood cultures. Magnetic resonance imaging (MRI) is more sensitive and specific than computed tomography (CT) and should be used to assess viral encephalitis. [11-13]. Seasonal

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factors may also play a role; for example, Japanese encephalitis is more common during the rainy season, while arbovirus infections in the United States are more common in the summer and autumn. Infection with human enteroviruses is known to be more common in the summer and autumn. [14] A variety of ideas have been proposed based on clinical, laboratory, and ecological results, but none have been fully proven. One theory links the outbreaks to litchi fruits in Muzaffarpur, Bihar, because the outbreaks occur in India's litchi fruit farming region, and cases tend to peak during the litchi harvesting season. [15].

Our lacunae – we found sudden surge of cases of viral meningoencephalitis , mostly because of HSV-1 in our area during the period of Jan and Feb 2022 . All patients were elderly and mostly Farmer by occupation . The purpose of this case series was to create awareness about this condition and to look into clinical profile and outcome of this condition.

Material And Methods

Study Place: Tertiary care centre in Southwestern Maharashtra

Study Period: January – February 2022.

Inclusion Criteria:

All the cases above 18 years of age admitted with symptoms and signs of meningoencephalitis.

Diagnosed with viral meningoencephalitis confirmed with laboratory investigations and brain imaging

Clinical features like fever, headache, seizures, vomiting , altered sensorium

Exclusion Criteria:

Age less than 18 years

Assessment:

Data were extracted using a proforma that included detailed history, clinical examination details and requisite investigations. History and clinical findings attributable to the meningitis and meningoencephalitis were collected in detail, which included fever, headache, vomiting, neck stiffness, seizures, altered sensorium, and focal neurological deficits. In addition to basic investigations (which included complete blood count, blood sugar, renal and liver function tests, and chest X-ray), blood culture was also taken for all patients. All underwent lumbar puncture and CSF analysis was carried out that included sugar, protein, total count, differential count, Gram stain, bacterial culture, Ziehl Neelsen stain for acid-fast bacilli (AFB), India ink stain for Cryptococcus, cryptococcal antigen test and automated culture for AFB. Neuroimaging (CT or MRI brain) was carried out on all patients. CSF polymerase chain reaction (PCR) was carried out in selected patients. Herpes simplex virus (HSV) PCR was carried out in selected patients with suspected HSV encephalitis based on clinical, CSF and neuroimaging findings.

Results

Table1: Demographic characteristics

Demographic characteristics	**No of cases**	**Percentage (%)**
Age		
≤ 50	3	30
51 – 60	2	20
> 60	5	50
Mean±S.D	55.90±16.07	
Gender		
Male	5	50
Female	5	50

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Table 2: Clinical Symptoms

Clinical symptoms	No of cases	Percentage (%)
Fever	5	50
Headache	7	70
Seizure	3	30
Vomiting	5	50
Brudginski	3	30
Kernig	3	30
Altered Mentation	9	90

Table 3: CSF Analysis & Laboratory Parameters

CSF analysis	Mean±S.D	(CI 95%)
Protein	33.10±27.28	(13.58-52.62)
Sugar	73.50±17.80	(60.76-86.24)
LDH	208.6±273.4	(12.99-404.2)
ADA	11.18±4.30	(8.10-14.26)
Laboratory Parameters		
Hb	12.50±2.90	(10.42-14.58)
TLC	9280±2477	(7508-11052)
PLC	168100±159133	(51262-281938)
Blood Urea	62.40±39.13	(34.41-90.39)
Sr. Creat	1.77±0.77	(1.21-2.32)
SGOT	54.50±41.25	(24.99-84.01)
SGPT	43.20±34.83	(18.28-68.12)
Direct Bilirubin	2.06±2.40	(0.34-3.78)
Indirect Bilirubin	1.19±1.20	(0.33-2.04)
Total Bilirubin	1.63±1.11	(0.83-2.42)
Albumin	4.15±1.44	(3.11-5.18)
Globulin	2.75±0.66	(2.27-3.22)
Total Protein	4.36±1.76	(3.10-5.62)

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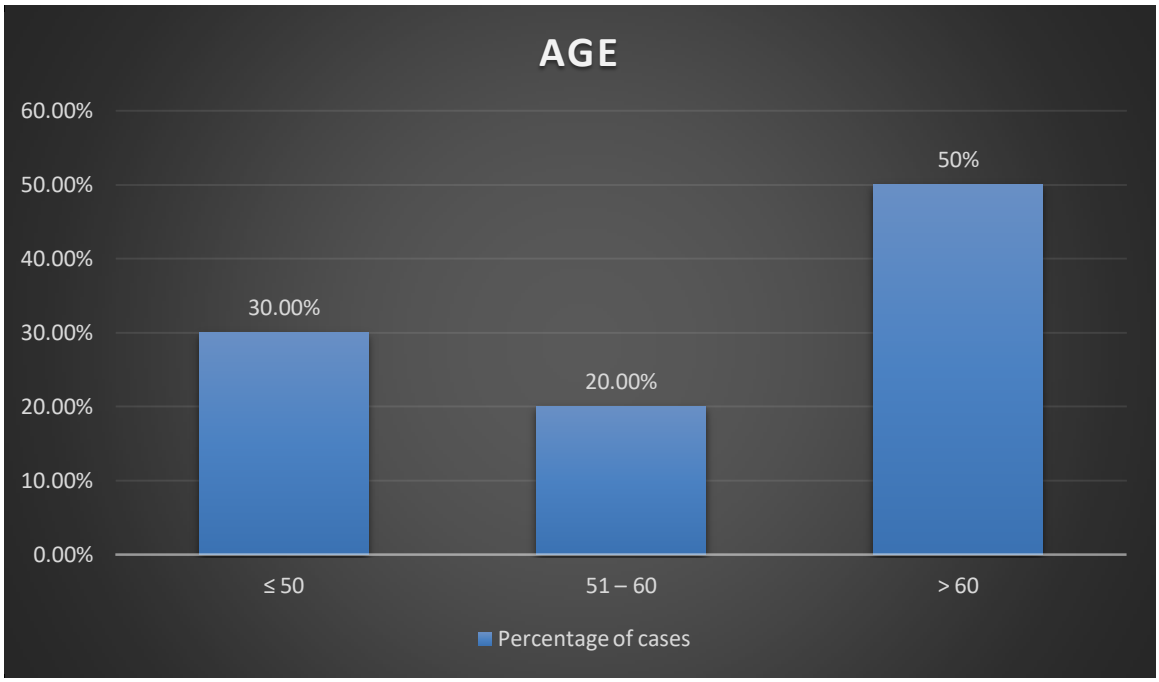
Table 4: Outcome

Outcome	No of cases	Percentage (%)
HSV PCR	6	60
Discharge	8	80
Dama	1	10
Death	1	10
Total	10	100

Graph 1: Age Distribution



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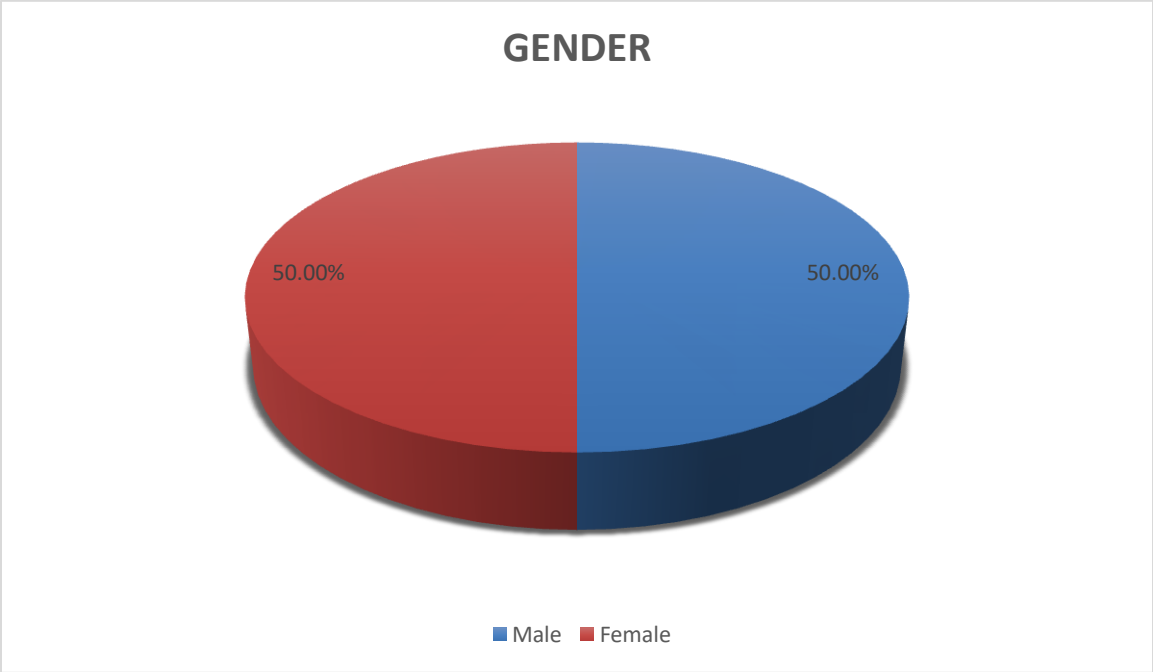
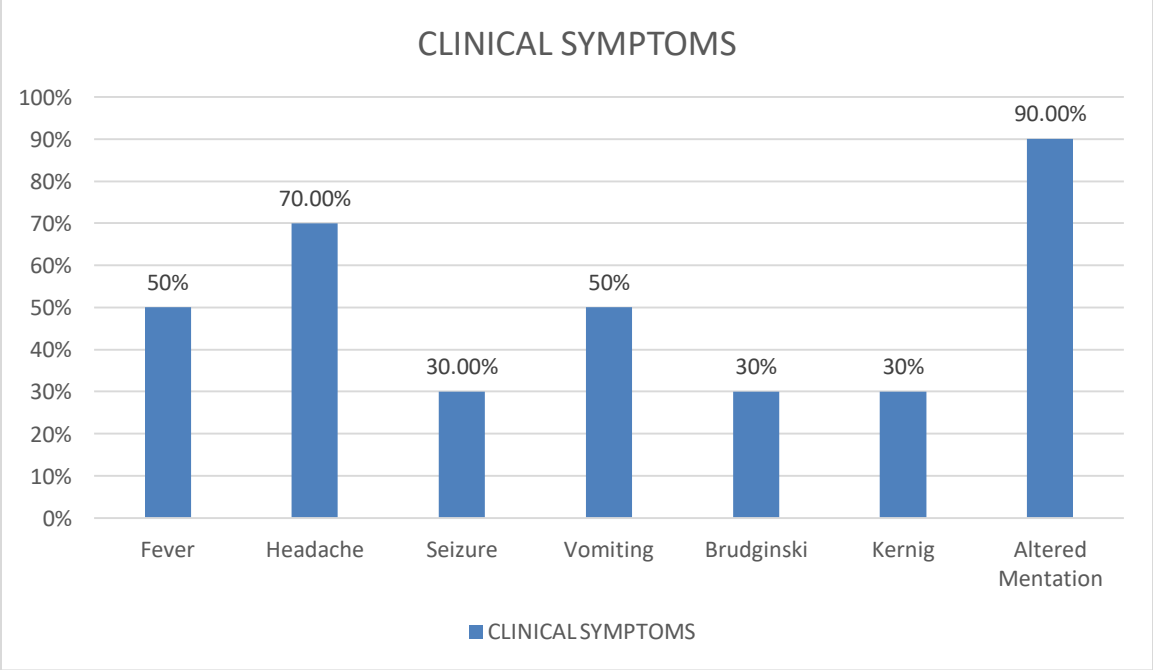
Graph 2: GENDER



Graph 3: CLINICAL SYMPTOMS



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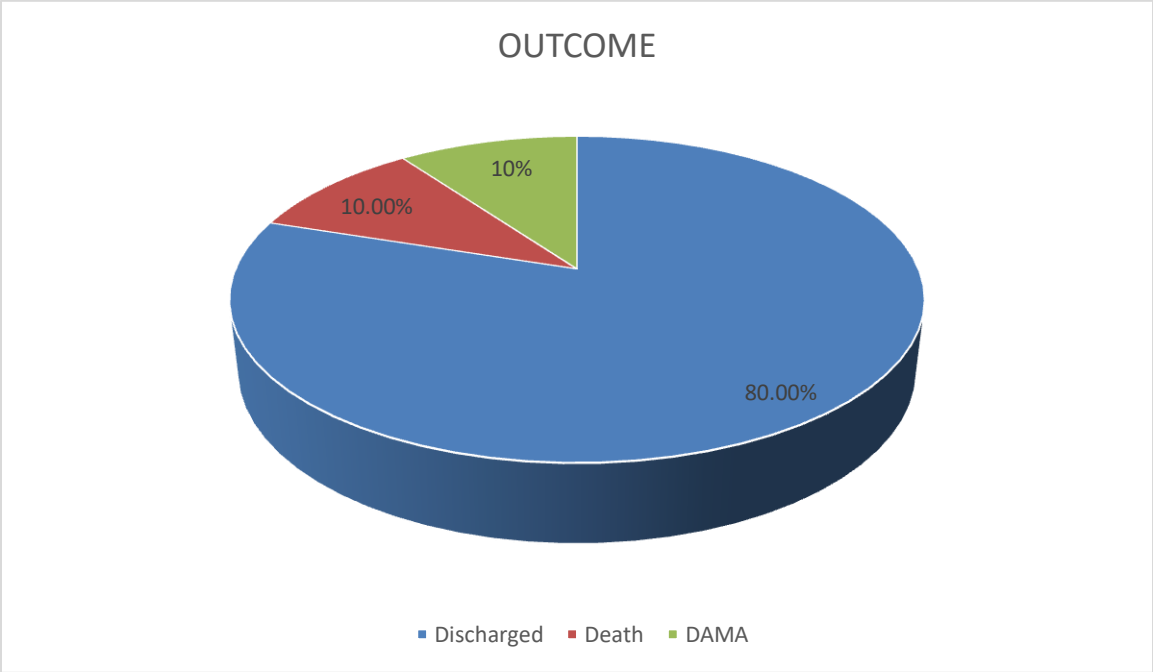
Graph 4: Outcome



Total 10 Cases Of Viral Meningoencephalitis Were Admitted In A Month Of January And February 2022 In Our Tertiary Care Centre. Most Of The Cases I.E. 50% Cases Were Seen Having Age More Than 60 Years Of Age, Mean Age 55.90±16.07 Was Observed. Out Of 10 Cases 50% Were Male Cases And 50% Were Female Cases (Table 1, Graph 1 And Graph 2). Altered Mentation (90%) Was The Most Common Clinical Symptom Seen In Viral Menigoencephalitis Cases Followed By Headache (70%), Fever (50%), Vomiting (50%), Brudginski (30%), Kernig (30%) And Seizure (30%) (Table 2, Graph3). Of All The Cases CSF Analysis Was Done And Protein, Sugar, LDH, ADA, Rbcs(30%) . HSV AND Blood For Hb, TLC, Platelet Count, Blood Urea, Sr. Creatinine (RFT), SGOT, SGPT, Direct Bilirubin, Indirect Bilirubin, Total Bilirubin, Albumin, Globulin And Total Protein Were Observed. 30% Of Cases Shows Derangement In RFT And LFT Suggestive Of MODF. (Table 3). On CT/MRI Examination One Case Was Observed Having Calcification In Posterior Falx, Persistent Cavum Septum Pellucidum & Vergae ; Partially Sclerotic Bilateral Mastoids, One Case Of OLD INTRAPARENCHYMAL HEMORRHAGE, PANSINUSITIS, One Case Having Sclerotic Bilateral Mastoid, One Case Having Generalised Cerebral & Cerebellar Atrophy, Ventriculomegaly, B/L Ganglionic Calcification, Minimal Periventricular Ischemic White Matter Disease, Ischaemic Foci In B/L Centrum Semiovale, Anterior Falcine Calcification, Mucus Retention Cyst With Thickening In Left Maxillary Sinus, Mucosal Thickening Noted In Multiple Sinuses, One Case Having Mild Generalised Cerebral & Cerebellar Atrophy, Ischemic Foci In B/L Centrum Semiovale, B/L Basal Ganglia Calcification, Changes Of Pansinusitis And One Case With Generalised Cerebral & Cerebellar Atrophy, Ventriculomegaly, Partially Empty Sella, Minimal Periventricular Ischemic White Matter Disease, Ischemic Foci Noted B/L Frontal Was Observed, One Case Of Generalised Cerebral & Cerebellar Atrophy, Ventriculomegaly, Partially Empty Sella, Minimal Periventricular Ischemic White Matter Disease, Ischemic Foci Noted B/L Frontal White Matter, B/L Hypoplastic Pcom Noted, One Case Of Mr Veno : No E/O Ic Sol/ Bleed / Mass Effect / Shift / No E/O Cvst, One Case Of Mucosal Thickening In Multiple Sinuses, No Enhancement On Contrast, Normal Mri Brain Plain And Contrast Study, One Case Of Acute Hemorrhagic Infarct, Generalised Cerebral And Cerebellar Atrophy And One Case Of Generalised Cerebral And Cerebellar Atrophy , Minimal Mucosal Thickening Of Lt Maxillary Sinus, B/L Partially Mastoid Sclerosis, Chronic Lacunar Infarct In B/L Basal Ganglia, Rt Thalamus, Lt Corona Radiata Was Observed. Out Of 10 Cases 8 Cases Were

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Discharged, 1 Case Had Taken Dama (Discharge Against Medical Advice) And 1 Mortality Was Observed.(Table 4, Graph 4)

Discussion

Between January and February 2022, a study was conducted in a tertiary care center in Southwestern Maharashtra. Ten cases of viral meningoencephalitis were diagnosed. The goal of this study was to look into the clinical profile of meningoencephalitis and its clinical outcome. Abnormal mentation were the most prevalent presenting symptoms. Other investigations with acute bacterial meningitis found comparable results. [15,16,17] Five patients had fever and vomiting symptoms, three cases each were seen having seizures, Brudginski and Kernig symptoms. Out of 10 cases 8 patients discharged, 1 patient took DAMA and 1 patient experienced death. A prospective cohort study of community-acquired bacterial meningitis in adults in the Netherlands found a similar result (41%).[17] A retrospective examination of 305 patients with acute bacterial meningitis found that 53 (17.4%) had received antibiotics prior to admission, with only one patient dying. The death rate among the remaining 252 individuals who had not received antibiotics prior to arrival was high (12%). This study emphasizes the need of initiating antibiotic medication as soon as possible. [18,19] Decreased Generalized cerebral and cerebellar atrophy, ventriculomegaly, b/l ganglionic calcification, limited periventricular ischemic white matter pathology, and ischaemic foci in the b/l centrum were all found on MRI brain scans in two patients Eight out of Ten patients with viral meningoencephalitis healed entirely with no lasting impairments; however, one patient died. These findings show that, despite early identification and appropriate treatment, some individuals with acute viral meningoencephalitis can develop a fulminant course, resulting in significant neurological impairment and a reduced quality of life. Patients with viral meningoencephalitis were diagnosed in all cases based on Clinical findings, CSF analysis and brain imaging findings. When CSF exhibited slightly higher protein, RBCs , low-to-normal blood sugar, and a moderately elevated lymphocyte count, a viral etiology was explored. In these individuals, bacterial cultures, Gram stain, AFB stain, and India ink stain were all negative. Six out of Ten patients were able to receive microbiological confirmation (positive HSV PCR in CSF); both displayed temporal cortex hyperintense signals on MRI brain. With acyclovir, eight out of ten patients healed completely and remained asymptomatic during follow-up Despite extensive diagnostic testing, the causative organism in many cases of suspected viral encephalitis (32–75%) could not be identified. In the California Encephalitis Project, 334 patients with encephalitis were investigated from 1998 to 2000; despite rigorous testing and review, no etiology could be determined in 208 (62%) of them.[20] Despite the fact that tuberculous and cryptococcal meningitis often have a subacute course, four patients with tuberculous meningitis and one with cryptococcal meningitis had abrupt onset sickness.

Conclusion

Viral meningoencephalitis is a devastating disease, which needs early recognition and prompt treatment in order to improve mortality and morbidity. Newer diagnostic and treatment modalities present a promising picture, although the disease burden still remains high.

Limitation

1. Unavailability to detect all viral markers
2. Long follow up not possible
3. Acute onset of illness
4. Commonest symptoms
5. Need for early diagnosis
6. Cause of mortality

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