Chapter 1

# The Clinical Management of the Patient with Encephalitis

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Additional information is available at the end of the chapter

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## 1. Introduction

### 1.1. Initial approach

It is important to follow a structured systematic approach to ensure good clinical care of the patient and to aid diagnosis. In an acute situation, the patient's airway, breathing and circulation must be assessed. Therefore, a Primary survey is undertaken and for this an ABCD approach is employed.

Determining the patency of the airway is crucial for the survival of the patient. In the general assessment of airway patency a clinician must observe the face and the neck. Abnormalities in the jaw mouth and neck must be noted as these could lead to airway compromise and future complications. Speaking to the patient for example by asking their name and observing their response, such as able to communicate in full sentences is a good indicator of unobstructed airways. Changes in vocalisation can be due to Asthma, COPD, emboli, oedema or even pneumonia. If any of these conditions are suspected a definitive diagnosis must be obtained as any of these could lead to further deterioration of the patient.

The second stage of the primary survey is the assessment of breathing. We begin with observation of the patient. Looking for signs of respiratory distress is important and failure to recognise this can lead to fatal consequences. Signs of respiratory distress can be the use of accessory muscles or changes in chest movement and in some cases even both.

Observation of the chest for any deformity is important but systemic observation is crucially important as well because it can show signs of cyanosis. At this point the respiratory rate needs to me measured. Then proceed to auscultate the chest and then end in percussion. Oximetry is also undertaken to determine the patient's oxygen saturations.

The third step in the process is an assessment of the circulatory system. This is a multidimensional assessment and many factors must be taken into account. As part of the circulatory assessment, examination of the extremities is undertaken to determine if they are warm or cool as a way of assessing perfusion. Next press the nail bed for 5 seconds and if the refill is less than 2 seconds the capillary refill time is normal[1]. Now position the patient at a 45 degree angle and observe the filling of the jugular vein. This is an indicator of the Jugular Venous Pulse (JVP) and in a healthy person the filling should be less than 3cm[2].

The clinician should then proceed to measure the blood pressure and auscultate the heart for murmurs; if an abnormality is suspected an ECG should be performed. After these vital steps are done the clinician should move to the lower extremities. The clinician should start with palpating the peripheral pulses; femoral, popliteal and posterior tibial artery as well as the arteries of the upper limbs. Examination of the calf muscles should also be undertaken for DVT[3]. IV access should be obtained as soon as possible if there are signs of haemodynamic compromise.

Assessment of disability is the last step of primary assessment. The AVPU score can be calculated or a calculation of the Glasgow coma scale. Pupillary light reflex and posturing can indicate if there is neurological damage and the severity of encephalitis. A measurement of capillary glucose can also be performed in this stage.

## 2. Clinical interview

The clinical interview can be divided into presenting complaint, history of presenting complaint, past medical history, family history, medication history and social history. Each part can give insight into the likelihood of encephalitis and the important signs to look for while performing the clinical examination.

### 2.1. Personal history

We commence by obtaining the basic demographic details of the patient, confirming you have the correct patient by verifying the name, age and sex of the patient. These basic details can also give some insight into aetiological agents of encephalitis as different aetiological agents have their own archetypes in transmission regarding age and sex.

### 2.2. Chief complaint

The onset of symptoms can give an indication of the aetiology of encephalitis; however the incubations period of the pathogens vary and overlap so it can be difficult to determine the aetiology from the onset of the symptoms (table 1).

Fever is a common complaint in encephalitis. Fever characteristics can be significantly different in various causes of encephalitis (table 2). However caution must be taken as fever is pathognomonic for various illnesses ranging from infections to autoimmune or even malignancy.

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| Pathogen | Incubation |
|---|---|
| HSV | 2-12 days[1] |
| West Nile virus | 1-6 days[2] |
| JE | 5-15 days[3] |
| EBV | 30-50 days[4] |
| Mycoplasma | 7-21 days[5] |
| Bartonella henselae | 7-14 days[6] |
| syphilis | 9-60 days[1] |

1. INCUBATION PERIOD OF DISEASE Epidemiology Review (1983) 5(1): 1-15. Oxford Journals
2. Vector Competence of California Mosquitoes for West Nile virus : Laura B. Goddard,* Amy E. Roth,* William K. Reisen,* and Thomas W. Scott* :Emerg Infect Dis. 2002 December; 8(12): 1385–1391.
3. The Epidemiology of Japanese Encephalitis: Prospects for Prevention : David Vaughn, Charles Hoke : Oxford Journals Medicine Epidemiologic Reviews : Volume 14, Issue 1 : Pp. 197-221.
4. Epstein-Barr Virus-specific Serology in Immunologically Compromised Individuals[1] : Werner Henle, and Gertrude Henle : Accepted March 6, 1981. : Cancer Res November 1981 41; 4222
5. Epidemiology of Mycoplasma pneumoniae Infection in Families : Hjordis M. Foy, MD; J. Thomas Grayston, MD; George E. Kenny, PhD; E. Russell Alexander, MD; Ruth McMahan, MN : JAMA. 1966; volume 197( number11): pages 859-866
6. The expanding spectrum of Bartonella infections: I. Bartonellosis and trench fever: BASS, JAMES W. MD; VINCENT, JUDY M. MD; PERSON, DONALD A. MD : Paediatric Infectious Disease Journal: January 1997 - Volume 16 - Issue 1 - pp 2-10

**Table 1.** There is an overlap in the incubation period of the various aetiological agents of encephalitis.

| Aetiology | Fever characteristic |
|---|---|
| HSV | Mild pyrexia[2] |
| West Nile virus | Mild pyrexia[3] |
| JE | Mild pyrexia[4] |
| EBV | 38 - 40°C in the first week [1:pages 871-872] |
| Mycoplasma | Morning temperature spikes [1: page 871] |
| Tick borne encephalitis | Relapsing- biphasic[5] |
| Toxoplasmosis Gondi | Relapsing [1:page 873] |
| Bartonella henesle | Mild pyrexia [1: page 872] |

1. Fever of Unknown Origin: Clinical Overview of Classic and Current Concepts: Burke A. Cunha, MD, MACP : Infectious Disease Clinic of North America 21 (2007) Pages 867–915.
2. Herpes simplex virus infection: Dr Richard WhitleyMD, Bernard Roizman ScD: the Lancet : volume 357: Issue 9267, May 12 2001, pages 1513-1518
3. West Nile virus fever :Lásiková S, Moravcová L, Pícha D, Horová B. : Epidemiol Mikrobiol Imunol. 2006 Apr;55(2): 59-62
4. Transplacental Infection with Japanese Encephalitis Virus : Dr. U. C. Chaturvedi, A. Mathur, A. Chandra, S. K. Das, H. O. Tandon and U. K. Singh : Oxford Journals, Journal of Infectious Diseases : Volume 141, Issue 6 : Pp. 712-715
5. Tick-Borne Encephalitis : Uga Dumpis, Derrick Crook, and Jarmo Oksi : Oxford Journals , Clinical Infectious Diseases : Volume 28, Issue 4 : Pp. 882-890

**Table 2.** Fever characteristics of the various aetiological agents of encephalitis.

Cephalgia is another common symptom of encephalitis. The cerebellum, Dura Mater and bones of the skull are insensible to pain[4]. Cephalgia is usually due to vasculature or sinus pain so a differential diagnosis must be sought to eliminate other causes of cephalgia. A cognitive change within a patient requires further assessment. We need to consider if there are focal signs or if it a general deterioration of consciousness. There are no specific patterns of cognitive dysfunctions identified within any specific aetiological group nor are cognitive changes exclusive to encephalitis. Consequently a definitive diagnosis is required in such circumstances where the patient is obviously unstable and the clinician must determine a course of action to reach a diagnosis. This can be achieved by examination of the patient and thorough diagnostic tests. General cognitive changes[5] which can be encountered in an encephalitic patient would range from personality changes, mood disorders, amnestic disorders, hallucinations, and seizures.

Seizures and status epilepticus are a major concern in a patient with encephalitis. Depending on the aetiological agent seizures can be very common. In HSE virus encephalitis eliptogenic centres are located in the temporal and frontal cortices[6]. A seizure in a patient with HSV encephalitis is an indication of a poorer prognosis. In JE encephalitis periods of seizures alternating to periods of altered consciousness are common, they are however not as common in WN encephalitis and Murray Valley encephalitis[7].

### 2.3. Past medical history

Past medical history can demonstrate key risk factors of the patient suffering from encephalitis. For example any conditions which would leave the patient with an immunodeficiency like HIV, cancer or even a primary immunodeficiency in patient exhibiting symptoms of encephalitis would merit immediate diagnostic procedures. It is important to consider if the patient is up to date with their vaccinations. In an unvaccinated patient the most common cause of encephalitis would be due to a varicella virus[8]. The most common cause in a vaccinated patient is Herpes Simplex Virus[9] Others facts which need to be considered are a previous episode of fever as some causes of encephalitis have a pattern of remitting fever. It is also important to ask the patient is if they have any recollection of being bitten by mosquitoes or tics as this can indicate possible aetiological agents.

### 2.4. Family history

Primary immunodeficiency can predispose a patient to the risk of encephalitis as well as other infections. So if a history of immunodeficiency is obtained the patient should immediately commence treatment with immunglobulins[10].

An instance wherein family history is vital is if the aetiology of encephalitis is contagious and other members of the family experiences symptoms of an infection or has shown symptoms of encephalitis. This may be useful in reaching the diagnosis of encephalitis or even determining the aetiology of encephalitis. However the expression of symptoms in any illness is highly variable amongst individuals and that is something which should be kept in mind.

### 2.5. Medical history

Medications can cause cognitive changes and fever so a medication history should be obtained to ensure that the symptoms are not due to a chemical disturbance. Prescription drugs, non-prescription drugs and even recreational drug use should be noted.

### 2.6. Lifestyle history

Different continents have different common aetiological agents of encephalitis so a history of travel should be documented. If a person is inclined to an outdoor lifestyle this should also be taken into account as they are at a higher risk of being bitten by tics or mosquitos depending on their demographics. Seasons affect behaviour pattern for example mating pattern in mosquitos, so the season should also be noted.

## 3. Physical examination

After performing the primary survey and the clinical interview, the secondary survey can be undertaken. Some manifestations of encephalitis, which may be encountered, are discussed below.

### 3.1. General observation

During general observation we can start by assessing the skin. Some etiological agents which can cause encephalitis also cause dermatological lesions. A prime example is the most common viral cause of encephalitis which is HSV, HSV also causes herpetic skin lesions[11], which should been noted and is a good means to reach a fast diagnosis. Other aetiological agents which may also have dermatological signs are EBV[12] in which jaundice and oral petechiae can be observed. A patient infected with WNV occasionally will display a rash[13]. Basciliar angiomatosis[14] a vascular lesion of the skin which can extend to other organs is described as a Chancre and it is a diagnostic sign of primary syphilis. Untreated syphilitic patients can progress to encephalitis and observing skin changes can aid in diagnosis [15]

### 3.2. Examination of the eye

Many Etiological agents of Encephalitis can cause ocular symptoms. During a HSV infection the patient can develop keratoconjunctivits[16]. For a patient with an EBV infection a periorbital oedema may be noted[17]. Chorioretinitis[18] is a rare sequelae of West Nile Virus, Being rather uncommon it should still be excluded. Ocular manifestations of Mycoplasma pneumoniae infection other than conjunctivitis are uncommon[19]. The most frequent ocular manifestation of bartonella is neuroretinitis which is usually unilateral[20]. Interstitial keratitis is frequently reported in patients with syphilis[21].

### 3.3. Examination of the oral cavity

Oral involvement is common in many viral disorders. For example in HSV, ulcers on the buccal mucosa and the tongue are observed[22]. During a bout of EBV infection pharyngitis occurs in 80 - 90% of patients and is usually mild in nature and clears in 7 - 14 days[23]. WNV patients suffer from lymphadenopathy so the tonsils should be examined for any indications of tonsilitis[24]. Oral manifestations of primary syphilis are usually a solitary ulcer on the lip or tongue. Mucous patches and maculopapular lesions are the 2 principal features of secondary syphilis. Gumma formation and syphilitic leukoplakia are the manifestations of tertiary syphilis[25].

Periauricular lymph nodes are enlarged during West Nile Virus infection[26] Peripheral lymphadenopathy is a manifestation of tuberculosis[27]. Bartonella Henselae, the main causative agent of cat-scratch disease (CSD), appears to be the most common organism responsible for lymphadenopathy in adults and children[28].

### 3.4. Examination of the abdomen

After observation now palpations of the abdomen may be undertaken. Mycoplasma is a very rare cause of ascites more commonly cutaneous lesions upon the abdomen should be noted[29]. Other causes of ascites may be Bartonella Hensele and Syphilis. After general palpation the clinician may want to assess if individual organs can be palpated. Ascites can be evaluated by palpation and percussion. A first clue that there is a possibility of ascites is a rounded symmetrical abdomen with bulging flanks. Undulation test[30] is the gold standard in demonstrating ascites and within this test the clinician should feel for a fluid wave, which would account for a positive test. The clinician may also test to see if there is shifting dullness which is indicative of more than 500ml of ascetic fluid[31].

The clinician should then start with the liver. We position the patient in the recumbent position with the right-handed examiner on the right side of the patient[32]. EBV can cause significant hepatomegaly but it is not as common as splenomegaly[33]. HSV can cause hepatomegaly however it is rare. TB[34] causes hepatomegaly as it infiltrates most organs. Next we can perform the examination of the spleen. We position the patient in the supine position with the knee flexed. We begin below the left costal margin using the right hand firmly pushing down and then releasing[35]. Viral aetiologies in with splenomegaly can occur quite often is an EBV[36] infection. Another viral aetiology of encephalitis is WNV[37] and it is not as common as EBV in causing encephalitis coupled with splenomegaly. A very rare bacterial cause of encephalitis is TB[38], this bacteria infiltrates most organs so splenomegaly should not be ruled out. In order to complete a full physical examination you can perform a renal examination. However common aetiological agents for encephalitis do not usually cause renal disorders.

### 3.5. Neurological exam

Next we can perform a thorough neurological exam. This will not only indicate a possibility of encephalitis but also the extent of destruction within the cerebrum. To help differentiate meningitis from encephalitis, we can assess for nuchal rigidity by asking the patient to place

their chin on their sternum. Inability to do so is a sign of meningitis. Kerning's sign can also be performed.

### 3.6. Examination of cranial nervs

We move on to a comprehensive assessment of the cranial nerves (table 3).

| Cranial nerve | Test |
|---|---|
| Olfactory nerve | Smell [1 :page 111] |
| Optic nerve | Visual acuity, visual fields, ocular fundi[1 : page 116] |
| Optic nerve and oculomotor nerve | Pupillary reactions [1 : pages 116-149] |
| Oculomotor nerve, Trochlear nerve, Abducens nerve | Extraocular movement, including opening of the eye [1 :pages 149-208] |
| Trigeminal nerve | Facial sensation, movement of jaw, corneal reflexes [1 : pages 208-226] |
| Facial nerve | Facial movement, gustation [1 :pages 251-262] |
| Vestibulocochlear nerve | Hearing and balance [1 :Pages 263-269] |
| Glossopharyngeal nerve, Vagus nerve | Swallowing, elevation of palate, gag reflex, gustation [1 :pages 251] |
| Trigeminal nerve , Facial nerve, Vagus nerve , Hypoglossal nerve | Voice and speech [1 :pages 208 - 276] |
| Accessory nerve | Shrugging shoulders and turning of head [1 :pages 263-269] |
| Hypoglossal nerve | Tongue protrusion [1 :pages 270 - 276] |
| 1. Adapted from: The neurologic examination: Dejong, Russell N. pages 111 to 270 | |

**Table 3.** Methods to test cranial nerve function.

### 3.7. Motor system

Now we can assess the motor system to discover if any damage has been done to the motor system. We first start off by testing strength. Strength is tested by having the patient resist your force as you attempt to move their body part against the direction of pull of the muscle that you are evaluating. This is graded on a scale of 0-5, with "0" representing absolutely no visible contraction and "5" being normal[39]. Strength testing is used to decide whether there is a neurogenic weakness and to determine which muscles/movements are affected. In correlation with the remainder of the motor exam, it should be possible to determine the particular part of the nervous system that is responsible for producing the weakness.

Testing reflexes is an important part of differentiating whether weakness is of an upper or lower motor neuron type. A reflex can be abolished without damaging motor axons[40]. In the setting of the patient with known weakness, reflex testing is a powerful tool to investigate the cause.[41] Symmetry of the reflexes needs to be considered in determining pathology. Pathological "spread of reflexes" is another objective sign of hyperactivity e.g. sustained clonus. Babinski reflex is a pathological reflex seen in upper motor neuron damage. However the

validity of this reflex clinically argued as changes in foot tapping have been shown to more efficiently show upper motor neuron (UMN) lesions[42].

Muscle bulk can be primarily assessed by inspection. Symmetry is important, with consideration given to the dominance of the hand and overall body habitus. Generalized wasting or cachexia should be noted and may reflect systemic disease, including neoplasia. Severe atrophy strongly suggests denervation of a muscle, such as with lower motor neuron (LMN) lesions. The most common method is assessing muscle tone is passively moving the patients limb. Tone can either be decreased or increased. The two common patterns of pathologically increased tone, spasticity and rigidity[43]. We should consider the difference between spasticity and rigidity. Spasticity [44] is manifested as an increased resistance to ignition of movement proceeded with a rapid passive movement. Rigidity is an increase in tone which is seen throughout a variety of movements[45].

Coordination is tested as a part of a sequence of movements. Typically the patient is asked to hold his/her hands in front with the palms up, first with the eyes open and then closed (as when examining pronator drift, above). Now we should consider posture, gait and any abnormal movements. The patient should be able to stand erect with eyes open and closed to see if doing so incites abnormality in movements. Then you should ask the patient to walk and assess if there are any abnormalities in gait[46].

Now we can compare the differences between upper motor neurons and lower motor neuron lesion signs (table 4)

| Comparison | UMN | LMN |
|---|---|---|
| Location of symptoms | Contralateral[2: pages 254] | Ipsilateral [2: pages 254] |
| Reflexes | Absent [1: page 46] | Present [1: page 50] |
| Fasciculation | Absent [3: chapter 9] | Present[3: chapter 9] |
| Spasticity | Present[1: page 46] | Absent[1: page 50] |
| Flaccidity | Absent [2: pages 250] | Present[1: page 50] |

1. Adapted from: Reinhard Rohkamm, M.D., Color Atlas of Neurology, 2004 Thieme Pages 46 to 50.

2. Neuroanatomy text and atlas : john H. Martin third edition : 2003 McGraw-Hill

3. Merritt's Neurology 10th Edition (June 2000): by H. Houston Textbook of Neurology Merritt (Editor), Lewis P. Rowland (Editor), Randy Rowland By Lippincott Williams & Wilkins Publishers

**Table 4.** A comparison between Upper Motor Neurons and Lower Motor Neuron lesion signs

### 3.8. Sensory system

Somatic sensation can be tested using the dermatomes. However this is completely subjective to the patient's perception. It is up to the examiner to determine if indeed there is a loss of

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sensation and if the patient has the capacity to convey the results accurately. Changes in sensation and the symmetry of the changes should be noted. A comprehensive examination of the sensory system must be carried out.

## 4. Diagnostics

The principal goal in diagnostics is to identify if the patient is indeed suffering from encephalitis and then the aetiological agent of encephalitis. The most common causes of viral encephalitis are HSV and VZ encephalitis and they are the only curable causes also.

### 4.1. EEG

EEG changes in encephalopathies are similar to any encephalitis aetiological agent. There is a progressive increase in slow wave activities[47], the degree of which parallels the severity of brain dysfunction. A diffuse slow-wave background followed by the rapid development of periodic complexes in may be diagnostic of herpes-simplex encephalitis[48]. None of these patterns is specific to a particular pathophysiological process or diagnosis, but periodic epileptiform discharges are most likely to occur in an acute course of the disease[49].

### 4.2. Radiography

MRI is the most sensitive non-invasive test in early diagnosis of HSE due to its high sensitivity to inflammatory increased brain water content. The classical findings in herpes encephalitis are periodic lateral epileptiform discharge and hyper intense T2-weighted signal in the temporal lobe on MRI however these findings are nonspecific[50]. Japanese encephalitis MRI clues would be bilateral thalamic involvement; hemorrhagic involvement can be occasionally seen. Locations in which lesions can be seen are cerebrum, the midbrain and cerebellum, the pons and the basal ganglia. The locations in which hemorrhagic lesions can be seen are cortex, the midbrain, cerebellum, and pontine lesions[51]. Eastern equine encephalitis produces focal radiographic signs what distinguishes it from HSV encephalitis involvement of the basal ganglia and thalami[52]. An MRI preformed on a patient with Epstein-Barr virus encephalitis could show focal lesions in the basal ganglia[53]. The tick-borne encephalitis MRI revealed pronounced signal abnormalities in the basal ganglia and thalamus, without contrast enhancement[54]. Meningovascular syphilis can manifest T2-weighted hyper intense signal abnormalities, which are thought to represent cerebral infarctions[55].

### 4.3. Lumbar puncture

Lumbar puncture is indicated in a patient with suspected CNS infections (table 5).

Contraindications of lumbar puncture should be kept in mind. If the patient is showing signs of papilledema or an intracranial mass is suspected an urgent CT should be performed[56]. Local skin infections are an absolute contraindication and so are spinal deformities. Uncontrolled bleeding diathesis is also a contraindication.

| Steps | Procedure |
|---|---|
| 1 | Obtain consent |
| 2 | Position the patient in the lateral decubitus position[1] |
| 3 | Locate landmarks: between spinous processes at L4-5[1] |
| 4 | Prep and drape the area after identifying landmarks. Use lidocaine 1% with or without epinephrine[2] |
| 5 | Assemble needle either an A-traumatic or Quincke and manometer. A-traumatic can reduce a post lumbar puncture headache. Attach the 3-way stopcock to manometer[3] |
| 6 | Insert needle through the skin and advance through the deeper tissues. A slight pop or give is felt when the Dura is punctured.[4] |
| 7 | When CSF flows, attach the 3-way stopcock and manometer. Measure the intracranial pressure which should be 20 cm or less. [5] |
| 8 | If CSF does not flow, or you hit bone, withdraw needle partially, recheck landmarks, and re-advance[1] |
| 9 | Once the ICP has been recorded, remove the 3-way stopcock, and begin filling collection tubes 1-4 with 1-2 ml of CSF each[5] |
| 10 | Remove needle, and place a bandage over the puncture site. Instruct patient to remain lying down for 1-2 hours before getting up[1] |

1. Lumbar puncture: Anatomical review of a clinical skill : J.M. Boon[1,*], P.H. Abrahams[2], J.H. Meiring[1], T. Welch[3] Article first published online: 16 SEP 2004 : Clinical Anatomy : Volume 17, Issue 7, pages 544–553, 2004

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3. Choosing the best needle for diagnostic lumbar puncture : Damien Carson, MB BCh, FRCA and Michael Serpell, MB BCh, FRCA : Neurology July 1, 1996 vol. 47 no. 1 33-37

4. Lumbar Puncture : Miles S. Ellenby, M.D., Ken Tegtmeyer, M.D., Susanna Lai, M.P.H., and Dana A.V. Braner, M.D. New England Journal Med 2006; 355:e12September 28, 2006

5. Lumbar Puncture Technique: Thomas A. McLennan Canadian Medical Association Journal (1962) Volume: 86, Issue: 17, Pages: 789

**Table 5.** A step by step method of performing a lumbar puncture.

Once CSF is obtained test tube one sample is usually used for detecting protein and glucose levels. Test tube 2 is used to establish a possible etiological agent so can be used for serology and bacterial cultures. Test-tube 3 is used to establish cell count and finally test-tube 4 is reserved for any specifics tests.

### 4.4. Serology

Once you have a CSF sample it can be used to preform serology tests in order to identify any possible viral causes of encephalitis.

As the most common causes of encephalitis are viral, serology is a useful tool for diagnosis the aetiological agents of encephalitis. Routine PCR diagnosis of HSE type 1 and 2 is a highly sensitive and specific method for diagnosing encephalitis[57]. The identification of West Nile virus immunoglobulin M in cerebrospinal fluid is the recommended test to document central nervous system infection, but this test may not be positive in spinal fluid collected less than 8 days after the onset of symptoms[58].For the diagnosis of JE virus (JEV) infection an immunoglobulin M capture dot enzyme immunoassay can distinguish JEV from dengue infec-

tion[59]. The TaqMan assay was specific for WN virus and demonstrated a greater sensitivity than the PCR method [60].

## 5. Treatment

The treatment is mainly focused on medical treatment as surgery is rarely required. Medical treatments rely on the assessment of the patients' needs. Prioritising clinical care is crucial as encephalitis can be life threatening so focusing treatment on jus the aetiological agent is a flaw in the clinician's judgement. Ensuring the patient's vital signs stay within a physiological range and if an aetiological agent is discovered then treatment specified for that agent should be deployed.

### 5.1. Medical treatment

Encephalitis is a medical emergency. Initially as we discussed the ABCD guidelines should be followed. Then after the diagnostic steps are undertaken the patient should be isolated until the aetiology is determined as most viral causes are airborne (table 6).

| Virus | Treatment |
|---|---|
| HSV-1 and HSV-2 | Acyclovir[1] |
| Varicella-Zoster Virus | Acyclovir is recommended. Gancyclovir or adjunctive corticosteroids [2] |
| Cytomegalovirus | Gancyclovir [3] |
| EBV | Acyclovir initially or cidofovir once EBV identified[4]. |
| Herpes B virus | No drug has been shown to be effective, although valacyclovir is the preferred agent[1] |
| Human herpes 6 | Gancyclovir or foscarnet should be used in immunocompromised patients[1] |
| Measles | Steroid therapy [5] |
| St. Louis Encephalitis | Interferon alfa-2a [6] |

1. Herpes simplex virus infections of the central nervous system: encephalitis and meningitis, including Mollaret's.
2. Antiviral Therapy of Herpes simplex and Varicella-zoster Virus Infections Peter Wutzler Institute for Antiviral Chemotherapy, Clinicum of the University of Jena, Erfurt, Germany
3. Cytomegalovirus infection of the central nervous system. Griffiths P. Source Department of Virology, Royal Free and University College Medical School, London, UK
4. Diagnosis and treatment of viral encephalitis : A Chaudhuri, P G E Kennedy
5. Treatment of measles encephalitis with adrenal steroids : John E. Allen
6. Interferon-α protects mice against lethal infection with StLouisencephalitis virus delivered by the aerosol and subcutaneous routes : T.J.G Brooks, R.J Phillpotts

**Table 6.** The treatment modalities for viral aetiological agents of encephalitis.

The next most common aetiological cause of encephalitis is bacterial. Neurosyphillis treatment is based on administering Penicillin at the same levels of treponemicidal levels found in

CSF[61]. Mycoplasma pneumonia encephalitis therapy most frequently deployed is erythromycin or minocycline. A high cerebrospinal fluid cell count, cerebrospinal fluid protein elevation, and higher age were associated with an unfavourable outcome[62].

We have to consider systemic complications as well as CNS complications. Monitoring vital signs continuously is essential in ensuring no sequelae develop and if they do they are swiftly treated. In patients with elevated intracranial pressure (ICP), management with corticosteroids and mannitol should be considered[63]. Corticosteroids are thought to decrease cerebral oedema. Now we should consider treatment targeted to specific symptoms. For example seizures are treated by anticonvulsive therapy. Analgesics may be needed to relieve headaches. Antipyretics may be needed for temperature control. Sedatives may be prescribes for irritability or restlessness.

Rare forms of encephalitis include acute disseminated encephalitis and paraneoplastic encephalitis. Acute disseminated encephalomyelitis is treated with high-dose corticosteroids. Plasma exchange can be considered when corticosteroids have not shown any benefit. We can also use treated with high-dose intravenous immunoglobulin (IVIG) [64]. Paraneoplastic encephalitis responds to immunotherapy with IVIG or plasma exchange.

### 5.2. Surgical treatment

In patients who have failed to respond to therapy to control elevated intracranial pressure or are inevitable at risk of uncal herniation a decompressive craniectomy is indicated. Surgical decompression may reduce changes of serious morbidity and mortality[65].

### 5.3. Prognosis

Cerebral inflammation is an indicator of mortality initial leucocytosis and development of severe hyponatremia is an indicator or increased morbidity and risk of mortality. In Japanese encephalitis a virus-specific immunoglobulin response is a marker for low risk of mortality[66]. Even though acyclovir reduces risk of mortality a high rate of patients still have morbidities[67].

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