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ANALYSIS OF ACUTE ENCEPHALITIS: DIAGNOSIS AND MANAGEMENT

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Abstract:
Encephalitis results from inflammation of the brain parenchyma, and may be caused by infections or autoimmune conditions. The main objective of the study is to analyse the diagnosis and management of acute encephalitis. This descriptive study was conducted in SIMS, Lahore during January 2019 to July 2019. This study was done basically for the analysis of diagnosis and management of acute encephalitis. The clinical challenge is distinguishing causes of encephalopathy, including septic, metabolic, toxic and others, from patients who have encephalitis and therefore need specific treatments. Initial history should identify clues as to possible causes, including a full collateral history if available, in order to ascertain the true duration of the problem. It is concluded that neurologists must be familiar with the myriad causes of encephalitis in order to develop a practical approach to diagnostic testing and treatment.

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INTRODUCTION:

Encephalitis results from inflammation of the brain parenchyma, and may be caused by infections or autoimmune conditions. Diagnosis is typically made by a combination of clinical, laboratory, neuroimaging, and electrophysiologic findings. A number of case definitions have been developed, which generally require encephalopathy, as characterized by alteration in consciousness or personality change lasting for a sustained period of time (typically greater than 24 hours) [1].

To distinguish encephalitis from other causes of encephalopathy, key features include presence of fever, CSF pleocytosis, or MRI or EEG changes compatible with encephalitis. Although such definitions likely capture most patients with clinically significant encephalitis, some will be missed. For example, localized forms of brain inflammation (i.e., a unilateral brainstem process) may cause focal neurologic deficits without affecting consciousness or behaviour [2].

The typical patient presenting with encephalopathy is referred to the acute medicine team, but there are often significant delays in the suspicion of encephalitis being raised [3]. The urgent tasks facing the assessing doctor are to stabilise the patient and to rule out potentially life-threatening diagnoses, including HSV type 1 encephalitis which can kill rapidly and needs urgent antiviral treatment. Making this diagnosis hinges crucially on lumbar puncture (LP), which in practice is often delayed [4].

Once a treatable viral cause is excluded, autoimmune encephalitis may be considered, but tests for these conditions take longer to perform. Even with thorough investigation, between 37% and 62% of patients with encephalitis will have no identified cause, and the management of this group remains challenging [5].

The diagnosis of acute encephalitis is suspected in a febrile patient who presents with altered consciousness and signs of diffuse cerebral dysfunction. Worldwide, infection of the central nervous system is the commonest cause of acute encephalitis. Herpes simplex virus (HSV), varicella zoster virus (VZV), Epstein-Barr virus (EBV), mumps, measles, and entero viruses are responsible for most cases of acute viral encephalitis among immune competent individuals in the United Kingdom [6]. In a large Finnish study reported recently, VZV was found to be the commonest virus associated with encephalitis as well as meningitis and myelitis, comprising 29% of all confirmed or probable aetiological agents while HSV and entero viruses accounted for 11% each and influenza A virus 7% of the cases [1].

Objectives

The main objective of the study is to analyse the diagnosis and management of acute encephalitis.

MATERIAL AND METHODS:

This descriptive study was conducted in SIMS, Lahore during January 2019 to July 2019. This study was done basically for the analysis of diagnosis and management of acute encephalitis.

Clinical assessment

The clinical challenge is distinguishing causes of encephalopathy, including septic, metabolic, toxic and others, from patients who have encephalitis and therefore need specific treatments. Initial history should identify clues as to possible causes, including a full collateral history if available, in order to ascertain the true duration of the problem [7]. Evidence should be sought of a change in personality or behaviour, or periods of drowsiness or seizures (which may be subtle). A travel history should be obtained, including any contact with animals, fresh water, mosquito or tick bites, or exposure to illnesses in the community. Known immune compromise or risk factors for HIV infection should be established. Examination should establish conscious level, any focal neurological deficit, seizure activity or movement disorder [8].

Common causes of encephalopathy

- Anoxic/ischaemic.
- Metabolic.
- Nutritional deficiency.
- Toxic.
- Systemic infections
- Critical illness.
- Malignant hypertension.
- Mitochondrial cytopathy (Reye's and MELAS syndromes).

Diagnosis

The presence of fever in itself is not sufficient to make a diagnosis of infective encephalitis since encephalopathy may be precipitated by systemic infections or sepsis without cerebral inflammation (septic encephalopathy). Cerebral malaria is considered to be an example of infective encephalopathy rather than true encephalitis since the neurological symptoms of cerebral malaria result from brain hypoxemia and metabolic complications (hypoglycaemia and acidosis) due to the heavy parasitaemia of the red blood cells by *Plasmodium falciparum* leading to capillary occlusion [9].

Patients with neuroleptic malignant syndrome have fever, altered consciousness, and nuchal rigidity and may present even after the offending neuroleptic has been withdrawn.[3] Traumatic brain injury and ongoing epileptic seizures must be excluded before

making a diagnosis of acute encephalitis. Seizures are generalised in encephalopathy, although focal seizures and focal neurological deficit may rarely occur (for example, hypoglycaemic encephalopathy and hemiplegia).

Table 01: Initial evaluation of encephalitis in adults

Routine studies
CSF (unless contraindicated[b])
Opening pressure, leukocyte count with differential, erythrocyte count, protein, glucose
Gram stain and bacterial culture
HSV-1/2 PCR (if test available, consider HSV CSF IgG and IgM in addition)
VZV PCR (sensitivity may be low; if test available, consider VZV CSF IgG and IgM in addition)
Enterovirus PCR
Cryptococcal antigen or India ink staining
Oligoclonal bands and IgG index
Venereal Disease Research Laboratory
Serum
Routine blood cultures
HIV serology (consider RNA)
Treponemal testing (rapid plasma reagin, specific treponemal test)
Imaging
Neuroimaging (MRI preferred to CT, if available)
Chest imaging (chest x-ray or CT)
Neurophysiology
EEG
Other tissues/fluids
When clinical features of extra-CNS involvement are present, we recommend additional testing (e.g., biopsy of skin lesions; bronchoalveolar lavage or endobronchial biopsy in those with pneumonia/pulmonary lesions; throat swab PCR/culture in those with upper respiratory illness; stool culture in those with diarrhea); also see below
Conditional studies
Host factors
Immunocompromised—CMV PCR, HHV6/7 PCR, *Toxoplasma gondii*; MTB, fungal infections, WNV
Geographic factors
Africa—malaria, trypanosomiasias, dengue
Asia—Japanese encephalitis virus, dengue, malaria, Nipah virus
Australia—Murray Valley encephalitis, Kunjin virus, Australian bat lyssavirus
Europe—tick-borne encephalitis virus; if Southern Europe, consider WNV testing, Toscana virus testing
Central and South America—dengue, malaria, WNV, Venezuelan equine encephalitis

Management

The management of acute encephalitis can be guided by a practical approach involving 3 "Es": emergent issues, epilepsy, and etiology. Ideally, LP should be performed immediately in patients with suspected brain infection, and empirical treatment started immediately thereafter. However, if LP is delayed for more than 6 hours empirical aciclovir may be needed before LP.[6] Patients with HSV encephalitis are likely to remain PCR positive in CSF for at least the first few days after commencing treatment, so LP should still be performed as soon as possible in patients who have commenced acyclovir [10]. This will help to establish the diagnosis and therefore dictate the duration of treatment. The UK guidelines recommend that aciclovir should be continued for at least 2 weeks, at which point the LP should be repeated. If the HSV PCR is still positive, aciclovir should be continued with repeat LP every week until the PCR is negative. If the patient is completely well, some would suggest that repeat LP is not necessary [11,12].

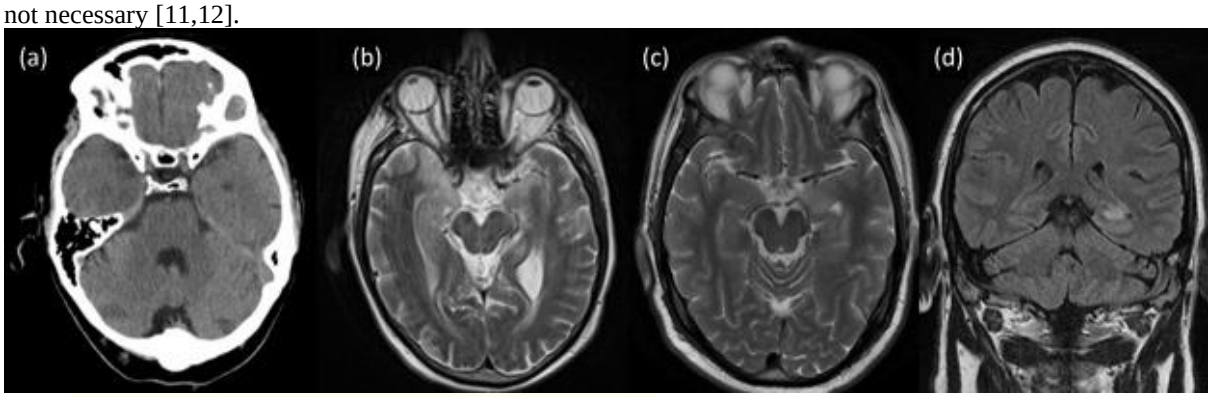

Figure 01: Brain imaging in encephalitis (**Reference:** Clin Med (Lond). 2018 Apr; 18(2): 155–159)

CONCLUSION:

It is concluded that neurologists must be familiar with the myriad causes of encephalitis in order to develop a practical approach to diagnostic testing and treatment. An understanding of recent advances in management, particularly with respect to autoimmune etiologies and critical care approaches, is equally important. Here, we summarize a general approach to the care of adult patients with encephalitis.

REFERENCES:

1. Dalmau J, Gleichman AJ, Hughes EG, et al. Anti-NMDA-receptor encephalitis: case series and analysis of the effects of antibodies. Lancet Neurol 2008;7:1091-1098.
2. Titulaer MJ, McCracken L, Gabilondo I, et al. Treatment and prognostic factors for long-term outcome in patients with anti-NMDA receptor encephalitis: an observational cohort study. Lancet Neurol 2013;12:157-165.
3. Prüss H, Finke C, Höltje M, et al. N-methyl-D-aspartate receptor antibodies in herpes simplex encephalitis. Ann Neurol 2012;72:902-911.
4. Hacohen Y, Deiva K, Pettingill P, et al. N-methyl-D-aspartate receptor antibodies in post-herpes simplex virus encephalitis neurological relapse. Mov Disord 2013.
5. Mohammad SS, Sinclair K, Pillai S, et al. Herpes simplex encephalitis relapse with chorea is associated with autoantibodies to N-Methyl-D-aspartate receptor or dopamine-2 receptor. Mov Disord 2013.
6. Graus F, Dalmau J. Paraneoplastic neurological syndromes. Curr Opin Neurol 2012;25:795-801.
7. Thakur KT, Motta M, Asemota AO, et al. Predictors of outcome in acute encephalitis. Neurology 2013;81:793-800.
8. Kalanuria AA, Geocadin RG, Püttgen HA. Brain code and coma recovery: aggressive management of cerebral herniation. Semin Neurol 2013;33:133-141.
9. Badjatia N. Fever control in the neuro-ICU: why, who, and when? Curr Opin Crit Care 2009;15:79-82.
10. Treiman DM, Meyers PD, Walton NY, et al. A comparison of four treatments for generalized convulsive status epilepticus. Veterans Affairs Status Epilepticus Cooperative Study Group. N Engl J Med 1998;339:792-798.
11. Silbergleit R, Durkalski V, Lowenstein D, et al. Intramuscular versus intravenous therapy for prehospital status epilepticus. N Engl J Med 2012;366:591-600.
12. Brophy GM, Bell R, Claassen J, et al. Guidelines for the evaluation and management of status epilepticus. Neurocrit Care 2012;17:3-23.