"NON-INFECTIOUS ETIOLOGY OF ENCEPHALOPATHY IN PATIENTS ADMITTED TO IMCU OF TIRUNELVELI MEDICAL COLLEGE"

DISSERTATION SUBMITTED

TO

THE TAMILNADU DR.M.G.R. MEDICAL UNIVERSITY, CHENNAI

In partial fulfilment of the requirements for the degree of

M.D. BRANCH – I (GENERAL MEDICINE)

Register No : 200120104005

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MAY-2023

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I, **Dr. BALU SUBRAMANYAM R K,** solemnly declare that the dissertation titled **"NON-INFECTIOUS ETIOLOGY OF ENCEPHALOPATHY IN PATIENTS ADMITTED TO IMCU OF TIRUNELVELI MEDICAL COLLEGE"** is a bonafide work done by me at Tirunelveli Government Medical College Hospital from 2021 to 2022 under the guidance and supervision of my unit chief, **Prof. Dr. S. ALAGESAN MD, DM.**, Professor and Head of General Medicine. This dissertation is submitted to The Tamil Nadu Dr. M.G.R. Medical University towards the partial fulfilment of the requirement of M.D. General Medicine degree examination.

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ACKNOWLEDGEMENT

I am grateful to **Dr. M. Ravichandran M.D**, Dean of Tirunelveli Government Medical College for allowing me the opportunity to successfully conduct this study in our institution.

I would like to thank wholeheartedly my **Guide, Prof. Dr. S.Alagesan M.D**, **D.M**, Professor and HOD of General Medicine department for his guidance and support that he provided throughout the study.

My sincere thanks to my **Co Guide, Dr. N.M.S. Ahamed M.D** for his patient guidance and valuable support provided during the study.

My sincere thanks to my Assistant Professors **Dr. T. Vinotha M.D, and Dr. K. R. Murugan M.D.,** for their help and encouragement.

I thank all my postgraduate colleagues for their constructive criticism, timely advice and support. I thank my family and my friends for their help and support.

Last but not the least I would like to thank my patients with gratitude for their cooperation during study and for teaching us the art of medicine and inciting us to learn more.

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PRINCIPAL INVESTIGATOR: DR R K BALU SUBRAMANYAM MBBS
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THE FOLLOWING DOCUMENTS WERE REVIEWED AND APPROVED

1.	TIREC Application Form	☐
2.	Study Protocol	☐
3.	Department Research Committee Approval	☐
4.	Patient Information Document and Consent Form in English and Vernacular Language	☐
5.	Investigator's Brochure	☐
6.	Proposed Methods for Patient Accrual Proposed	☐
7.	Curriculum Vitae of The Principal Investigator	☐
8.	Insurance /Compensation Policy	☐
9.	Investigator's Agreement with Sponsor	☐
10.	Investigator's Undertaking	☐
11.	DCGI/DGFT approval	☐
12.	Clinical Trial Agreement (CTA)	☐
13.	Memorandum of Understanding (MOU)/Material Transfer Agreement (MTA)	☐
14.	Clinical Trials Registry-India (CTRI) Registration	☐

THE PROTOCOL IS APPROVED IN ITS PRESENTED FORM ON THE FOLLOWING CONDITIONS

1. The approval is valid for a period of 2 year/s or duration of project whichever is later
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5. The TIREC will monitor the study
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Dr.K.Shantaraman, MD
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This is to certify that this dissertation work entitled **"NON-INFECTIOUS ETIOLOGY OF ENCEPHALOPATHY IN PATIENTS ADMITTED TO IMCU OF TIRUNELVELI MEDICAL COLLEGE"** of the candidate **Dr. BALU SUBRAMANYAM R K** with registration Number **200120104005** for the award of **M.D.** Degree in the branch of **GENERAL MEDICINE (I).** I personally verified the urkund.com website for the purpose of plagiarism check. I found that the uploaded thesis file contains from introduction to conclusion page and result shows **0 percentage** of plagiarism in the dissertation.

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Sources included in the report

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Non-infectious etiology of encephalopathy in patients admitted to IMCU of Tirunelveli Medical College
INTRODUCTION Encephalopathy is a broad clinicopathological condition, which involves diffuse brain dysfunction. This is characterised by delirium or an altered mental state. The treatment of encephalopathy is based on its etiology which can be due to metabolic, systemic infection, autoimmune, neurological, organ dysfunction, or toxin/drug-induced. [2] Encephalopathy is a multifactorial disease that involves reversible and irreversible causes. [2] The clinical hallmark of encephalopathy is an altered mental state, which is dependent on the severity of encephalopathy. The common neurological symptoms such are dementia, impaired cognitive functioning, personality changes, loss of focus, and lethargy are usually progressive and increase with the prolonged nature of the disease. Myoclonus (involuntary twitching of a muscle or group of muscles), nystagmus (rapid, involuntary eye movement), tremor, muscle atrophy and weakness, dementia, seizures, and loss of the ability to swallow or speak are some additional neurological symptoms that may manifest. [3] The underlying etiology and differential diagnosis of encephalopathy can be determined based on the patient's medical history including the onset of symptoms, progression, total duration, and physical examination. However, some patients may not able to provide a detailed history due to their condition. [4]
The onset of encephalopathy can be correlated with its etiology in the suspected high-risk group. Hyperacute-onset of encephalopathy can be due to various conditions, hence clinicians should consider the cerebrovascular disease, seizure, trauma, or migraine. The study aims to identify the non-infectious causes of encephalopathy to better understand the etiological differences and susceptible high risk groups. Early identification of the disease can help in preventing the progression of the disease and reduce the complication that arises with encephalopathy. The study also aims to identify the age group and specific disease group at risk of developing encephalopathy to prevent the occurrence by treating the causative factor promptly.
LITERATURE REVIEW Encephalopathy, which is derived from the Greek words en-cephalo, which means "

ABSTRACT

TITLE : **NON-INFECTIOUS ETIOLOGY OF ENCEPHALOPATHY IN PATIENTS ADMITTED TO IMCU OF TIRUNELVELI MEDICAL COLLEGE**

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BACKGROUND : Encephalopathy is a broad clinicopathological condition, which involves diffuse brain dysfunction. The clinical hallmark of encephalopathy is an altered mental state, which is dependent on the severity of encephalopathy. Encephalopathy is a multifactorial disease that involves reversible and irreversible causes including metabolic, systemic infection, autoimmune, neurological, organ dysfunction, toxin/drug-induced and many others. The underlying etiology and differential diagnosis of encephalopathy can be determined based on the patient's medical history including the onset of symptoms, progression, total duration, and physical examination with support of laboratory investigations. The study aims to identify the non-infectious causes of encephalopathy to better understand the etiological differences and susceptible high risk groups. Early identification of the disease can help in preventing the progression of the disease and reduce the complication that arises with encephalopathy.

AIM AND OBJECTIVES : The study aims to enumerate various non-infectious causes of encephalopathy in patients admitted to IMCU and to identify high-risk groups susceptible to various non-infectious causes of encephalopathy.

MATERIALS AND METHODS : This is a single center hospital based cross-sectional study done in the Department of general medicine of Tirunelveli medical college for a time period of 18 months (March 2021 - August 2022). The study included 235 participants who were recruited based on inclusion and exclusion criteria. Detailed history taking, assessment of sensorium using Glasgow Coma Score, routine blood investigations, relevant radiological investigations, body fluid analysis were done and collected data was analyzed using Statistical Package for Social Sciences.

RESULTS : The proportion of encephalopathy was seen higher in the age group between 40- 49 years of age (65 participants), followed by the age group of 50-59 years of age (64 participants). Male predominance was noted higher in the current study, with 140 male participants and 95 females. Regarding different etiologies, neurological cause was most common (68 participants), followed by encephalopathy due to systemic disease (62 participants), metabolic disorders (56 participants) and toxin/drug induced (49 participants). Type 2 diabetes was the most common comorbidity seen among the majority of the individuals (65 participants), followed by hypertension (58 participants) although 58 participants presented with encephalopathy with no comorbid condition. In addition, epilepsy, DCLD, CAD, CKD, and COPD were also seen among patients. Out of 235 participants, 134 (57%) of the participants were presented with a GCS score

of less than 10, and 101 (43%) participants showed a score above 10. The current study reports a significant difference in the GCS score due to various etiology of encephalopathy ($p < 0.0001$). The current study reports a significant difference in the etiology of encephalopathy in different age groups. The study found a significant difference between different age groups and the etiology of encephalopathy ($p < 0.0001$) with the age group of 40-49 and 50-59 years being more prone for encephalopathy with systemic diseases being the predominant cause. The current study did find a significant difference in the comparison of gender and etiology of encephalopathy ($p = 0.044$). However, the male gender was overall seen in higher numbers among various causes of encephalopathy.

CONCLUSION : The present study concludes that encephalopathy is a multifactorial disease with different etiological reasoning for each type of encephalopathy. The severity and morbidity remain high in every type of encephalopathy which increases with advanced age. The most cause of encephalopathy in 40-59 years age group was due to systemic disease, followed by neurological cause, and metabolic. The toxin/drug-induced encephalopathy was seen in the younger age group due to drug abuse or alcohol intoxication. The prognostic role of the GCS score can be beneficial in identifying the severity of the disease. The current study shows that the majority of patients got admitted with GCS <10 irrespective of the cause of encephalopathy. Hence it is essential to identify the root cause of encephalopathy to prevent morbid complications, reduce the risk of organ damage, and provide optimal therapeutic outcomes.

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INTRODUCTION

Encephalopathy is a broad clinicopathological condition, which involves diffuse brain dysfunction. This is characterised by delirium or an altered mental state. The treatment of encephalopathy is based on its etiology which can be due to metabolic, systemic infection, autoimmune, neurological, organ dysfunction, or toxin/drug-induced. [2]

Encephalopathy is a multifactorial disease that involves reversible and irreversible causes. [2] The clinical hallmark of encephalopathy is an altered mental state, which is dependent on the severity of encephalopathy. The common neurological symptoms such are dementia, impaired cognitive functioning, personality changes, loss of focus, and lethargy are usually progressive and increase with the prolonged nature of the disease. Myoclonus (involuntary twitching of a muscle or group of muscles), nystagmus (rapid, involuntary eye movement), tremor, muscle atrophy and weakness, dementia, seizures, and loss of the ability to swallow or speak are some additional neurological symptoms that may manifest. [3]

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The study aims to identify the non-infectious causes of encephalopathy to better understand the etiological differences and susceptible high risk groups. Early identification of the disease can help in preventing the progression of the disease and reduce the complication that arises with encephalopathy.

The study also aims to identify the age group and specific disease group at risk of developing encephalopathy to prevent the occurrence by treating the causative factor promptly.

LITERATURE REVIEW

Encephalopathy, which is derived from the Greek words en-cephalo, which means "in the brain," and pathos, which means "suffering," describes a change in mental status that affects a patient's cognition or level of arousal. There are many possible underlying causes of encephalopathy, which describes a clinical syndrome rather than a diagnosis. [1]

Although the term "encephalopathy" can refer to both focal and global brain insults, it is most frequently used to refer to the latter. Remember that focal cognitive deficit can mimic global encephalopathy syndromes, such as speaking difficulties like aphasia or memory loss like amnesia. A subtype of encephalopathy known as delirium causes the patient to experience a sharply altered mental state with waxing and waning attention.

The history (paying particular attention to the rate of onset, progression of symptoms, and overall duration), physical examination (mental status examination), laboratory/radiologic tests, and differential diagnosis of encephalopathy syndromes can be used to make these determinations. Patients with encephalopathies might not be able to give a history of their illness, so additional information from family (or others) is crucial. [2]

Encephalopathy that develops over any period may, in general, be caused by a systemic illness or a toxic-metabolic injury.

For each duration, broad categories of neurologic disease should be taken into account.

The below-given table shows the causes of encephalopathy over time;

Table -1 – Causes of encephalopathy depending on duration [1]

	Hyperacute	Acute	Subacute	Chronic
Primary Neurologic	● Vascular ● Seizure ● Migraine ● Trauma	● Vascular (SDH) ● Inflammatory (Acute demyelination) ● Infectious	● Vascular (SDH) ● Neoplasm-related ● Inflammatory ● Infectious	● Vascular (SDH) ● Degenerative ● NPH ● Infectious (HIV)
Systemic	● Hypertensive encephalopathy ● Metabolic encephalopathy	● Systemic infection	● Toxic/metabolic/drug-related ● Chronic systemic conditions; ● Heart Failure ● Endocrinopathy ● Malignancy ● Autoimmune ● OSA	

Consideration of cerebrovascular disease, seizures, trauma, or migraine should be made in the case of hyperacute-onset encephalopathy. Consideration of neurologic infection (bacterial or viral meningitis or encephalitis) or acute demyelination (such as acute disseminated encephalomyelitis or flare of multiple sclerosis) should be made in the case of acute-onset encephalopathy. Consideration of neoplasia (primary brain neoplasms or secondary metastasis) should be considered in cases of slowly progressing encephalopathy. Depending on the toxin or metabolite and the time course of its aberration, toxic and metabolic insults can cause encephalopathy over any time course. [3]

Common non-infectious causes of Encephalopathy [2] [4]

Neurological Causes

- Hemorrhagic stroke
- Ischemic Stroke
- Subarachnoid haemorrhage
- Seizures
- Cerebral venous thromboembolism
- Autoimmune encephalopathy
- Brain Metastasis

Encephalopathy due to systemic disease

- Uremic encephalopathy
- Hepatic encephalopathy
- Cardiogenic shock
- Carbon dioxide narcosis/ Hypercapnia

Metabolic Causes

- Hypoglycemia
- Hyperglycemia
- Hyponatremia
- Diabetic ketoacidosis
- Hyperosmolar hyperglycemic state

Toxin/Drug induced encephalopathy

- Benzodiazepine poisoning
- Alcohol intoxication
- Nitrobenzene poisoning
- Cypermethrin poisoning
- Wernicke encephalopathy
- Atropine induced delirium

HYPERACUTE CAUSES OF ENCEPHALOPATHY [1]

Primary Neurologic condition

Intracranial haemorrhage

It is categorized based on the compartment of the brain involved which includes; intracerebral, subarachnoid, subdural, or epidural space. The diagnosis is based on clinical parameters, however computed tomography (CT) is used for confirming diagnosis. [4]

A sudden, severe headache is a common symptom of subarachnoid haemorrhage, especially when it results from a burst cerebral aneurysm. Meningeal signs, such as meningismus, photophobia, nausea, seizures, and encephalopathy with a low level of arousal may also be present. Intraparenchymal haemorrhage frequently includes headache, nausea, seizures, hypertension, or altered level of consciousness in addition to focal neurologic deficits that match the hemorrhage's location.

Hypertension, cerebral amyloid angiopathy, head trauma, coagulopathy, brain metastases, and dural sinus thrombosis are among the common causes of intraparenchymal haemorrhage.

Head trauma is almost always linked to epidural and subdural hematoma. Although an epidural hematoma can cause a "lucid interval" before the patient's level of consciousness starts to decline, many patients experience

this from the moment it first appears. Damage to the middle meningeal artery frequently results in epidural hematoma, which allows arterial blood to gather in the epidural space and causes an abrupt rise in intracranial pressure and brain shift.

Ischemic stroke

Lack of blood flow to one or more areas of the brain results in infarction and is the cause of ischemic stroke. Most ischemic stroke patients have focal deficits because of the location of the ischemia or infarction, which determines the clinical syndrome (eg, weakness, aphasia). Infarctions of specific areas, such as the bilateral thalami (for example, "top of the basilar" syndrome), the right middle (inferior division) and anterior cerebral arteries, and diffuse and bilaterally located small emboli, can also result in global encephalopathy. Small-vessel strokes (such as lacunar strokes) may present with a stuttering pace over minutes to hours while embolic strokes typically present with a hyperacute presentation with symptoms that are maximal at the time of onset.

Seizures

Abnormal, excessive, rhythmic electrical discharges in the brain are responsible for seizures. Beyond this, their clinical presentation is highly variable and depends on where in the brain the abnormal electrical activity originates and whether it stays in one location (i.e. partial) or spreads to

more areas of the brain (ie, generalized). They typically manifest as discrete events with a rapid onset. Generalized seizures always cause a loss of consciousness and are followed by a postictal state, a variable-length period of global encephalopathy. When the cognitive networks are affected, partial seizures can result in encephalopathy and frequently cause focal symptoms that reflect the function of the area where the abnormal electrical activity occurs (e.g., motor, sensory, or autonomic). Patients who experience a sudden onset or fluctuating symptoms of encephalopathy should be evaluated for nonconvulsive seizures or seizures without obvious motor manifestations.

Migraine

A typical migraine is characterised by throbbing, unilateral headaches that last for several hours and are frequently accompanied by nausea, and fear of light and sound. These headaches frequently develop over several minutes and may be accompanied by a somatosensory or visual aura (such as a "scintillating scotoma" or spreading paresthesias). Confusional migraine, also known as acephalgic migraine, is a rare form of the condition where migraine symptoms include confusion. [5]

Traumatic brain injury

Hyperacute encephalopathy is frequently brought on by an acute traumatic brain injury. Depending on the extent of the injury, changes in both the

level of arousal (for example, lethargy, coma) and the contents of consciousness (for example, amnesia, disorientation) may take place. The mild traumatic brain injury with altered mental status known as a concussion is referred to as such. The onset of symptoms may be hours or days after impact, and concussed patients may experience a posttraumatic encephalopathy as part of a more general postconcussive syndrome (such as headaches, dizziness, mental fogginess, intolerance to loud noises, or bright lights). Fatigue, excessive sleeping or insomnia, personality changes (such as irritability, and labile affect), memory loss, poor concentration, and slow processing speed are just a few of the neuropsychiatric symptoms that may coexist with the syndrome. [6]

Encephalopathy due to systemic conditions

Sudden-onset encephalopathy can be brought on by metabolic disturbances (such as hyper- or hypoglycemia), medications, drugs, drug withdrawal, and other toxic exposures in addition to hypertensive emergencies (such as hypertensive encephalopathy/posterior reversible encephalopathy syndrome). Sudden-onset encephalopathy can also be a symptom of psychiatric conditions like panic attacks, nonepileptic seizures, fugue states, and psychosis.

ACUTE CAUSES OF ENCEPHALOPATHY [1]

Primary Neurologic Condition

Subdural hematoma

Hemorrhage into the subdural space is known as a subdural hematoma. The slow accumulation of blood can take days or even weeks to cause a clinically significant mass effect because venous bleeding is typically the cause. Headache, decreased arousal, focal deficits, cognitive decline, and seizures are typical symptoms. Head trauma (often mild), coagulopathies, anticoagulant drugs, and intracranial hypotension can all result in subdural hematomas (often due to lumbar puncture or shunt).

Demyelinating conditions (multiple sclerosis, neuromyelitis optica spectrum, acute disseminated encephalomyelitis)

Acute disseminated encephalomyelitis, neuromyelitis optica spectrum and multiple sclerosis are central nervous system demyelinating diseases. Even though multiple sclerosis is a chronic condition, it frequently experiences acute flare-ups that lead to specific neurologic deficits (such as transverse myelitis or optic neuritis). Diffuse or focused involvement of cognitive/behavioral networks can both result in encephalopathy. Acute multifocal demyelinating disorder of the central nervous system known as

acute disseminated encephalomyelitis typically develops after an infection, most frequently in children.

Acute Disseminated Encephalomyelitis (ADEM) - A progressive, rapid, and acute condition referred to as post-infectious encephalomyelitis, which is an autoimmune condition that occurs in the central nervous system. ADEM is clinically characterized by the demyelination of the brain and the spinal cord which results in inflammation following immunization or infection. [28]

Causes of ADEM - ADEM has been linked to a few contagious diseases and vaccinations. Cytomegalovirus, Epstein-Barr virus, herpes simplex, human herpes-virus-6, influenza virus, hepatitis A, human immunodeficiency virus, and mycoplasma pneumonia are the most frequently associated pathogens; however, the majority of the time, the pathogen responsible is not known. Borrelia burgdorferi, Leptospira, and beta-hemolytic streptococci are additional bacterial infections that are related. [33] [34]

Before there were immunization programs, the most common link was between ADEM and measles (in addition to an increased incidence in association with rubeola, rubella, mumps, varicella, and smallpox as well). Today, viral infections of the gastrointestinal or respiratory tracts are more frequently linked to ADEM.

The earliest vaccine to be linked to ADEM has been the rabies vaccine. It appears between 8 and 21 days after immunization in both adults and children. The vaccines for measles, pertussis, tetanus, influenza, hepatitis B, diphtheria, rubella, pneumococcus, varicella, smallpox, human papillomavirus, and poliomyelitis are among the less frequently associated vaccines. [34]

Pathophysiology of ADEM - Although the precise mechanism of ADEM is not fully understood, it is believed to result from inflammation brought on by an environmental stimulus in genetically susceptible people (such as vaccination or infectious disease). A further description of ADEM is that it is an autoimmune condition that results in demyelination of the central nervous system (CNS). [35]

It has been proposed that either a cell-mediated response or antibodies produced in response to an environmental trigger cross-react with myelin autoantigens (e.g., myelin basic protein, myelin oligodendrocyte protein, proteolipid protein) in the CNS, resulting in the demyelination characteristically seen in ADEM. The inflammation and circulating immune complexes that follow vaccination or infection may increase vascular permeability and cause congestion in the CNS, according to a different mechanism that has been put forth. It is believed that mononuclear infiltration of the CNS vasculature causes the edema that surrounds the vessels and, occasionally, hemorrhage that damages the neuronal cells

nearby (such as demyelination, necrosis, or gliosis) and, ultimately, determines the range of potential clinical presentations and prognoses seen in people with ADEM. The blood-brain barrier may break down as a result of the inflammation and increased vascular permeability, allowing antigens and inflammatory cells involved in the concurrent cell-mediated immune response to infiltrating the CNS. [36]

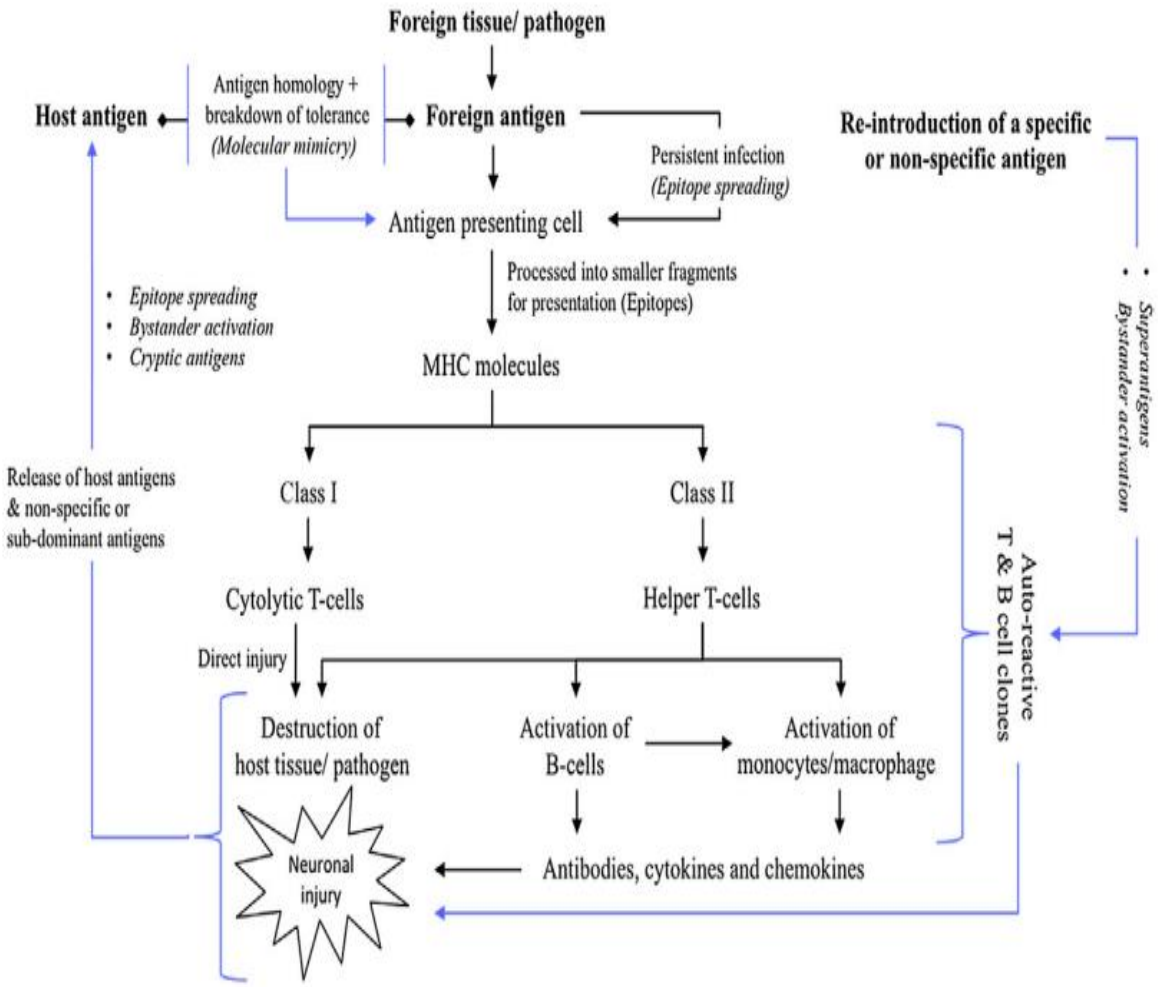

Figure 2 – Mechanism involved in ADEM

Diagnosis of ADEM - MRI is the preferred imaging technique for assessing ADEM. On T2-weighted, fluid-attenuated inversion recovery (FLAIR), proton-density, and echo-planar trace diffusion MRI sequences, it exhibits hyperintense lesions. T1-weighted sequences do not typically show lesions, though larger lesions may show up as hypointensities. Imaging of ADEM may show a single lesion (large or small, confluent or solitary) or multiple lesions throughout the white matter (periventricular and subcortical) and grey matter (basal ganglia, thalamus, cortex) of the brain. Most frequently seen are multiple, widespread, asymmetric lesions bilaterally throughout the brain. [37] The brainstem, cerebellum, and spinal cord may also have infratentorial lesions, but they rarely manifest alone without a corresponding lesion in the brain. On imaging, ADEM lesions frequently have indistinct margins. This may aid in separating these lesions from the multiple sclerosis lesions' having characteristic clear-cut margins. [35]

It is crucial to remember that ADEM can manifest without any outward signs of the disease and with a normal MRI (even after multiple scans). It is also possible in some circumstances for MRI lesions to develop weeks after the onset of symptoms. Repeat imaging is recommended, especially early in the course of the disease because, despite the patient's potential to continue asymptomatic, there may be fluctuations in lesions (e.g., new

lesions may appear while older lesions resolve). This is true even though the majority of MRI lesions resolve within 18 months. [35]

Although MRI is the preferred imaging technique, a CT scan might be recommended in an emergency to rule out any other neurological dysfunctions that might be life-threatening. A CT scan in the case of ADEM is typically unremarkable, particularly earlier in the course of the disease. Later stages of ADEM may manifest on a CT scan as focal or multifocal areas of white matter damage. In 50% to 80% of patients with ADEM, cerebral spinal fluid (CSF) analysis (e.g., following a lumbar puncture) may show abnormalities. These findings could be lymphocytic pleocytosis (less than 100 white blood cells/mL) and a marginally elevated CSF protein (less than 70 mg/dL). [32]

A diagnosis of ADEM cannot be made using a particular biomarker or diagnostic test. When a patient has multi-focal neurologic deficits without a history of prior neurologic dysfunction, it is taken into consideration. A diagnosis of ADEM will be further supported by one or more demyelinating lesions on a brain MRI, either supra- or infratentorial. These findings will help to confirm the diagnosis of ADEM when combined with a history of infection or immunization and abnormal CSF findings (but are not necessary to do so). [32]

Although there are no specific diagnostic standards for ADEM in adults, encephalopathy and multifocal CNS involvement are required for a diagnosis in children.

Primary central nervous system infections

(meningitis/encephalitis/abscess)

Encephalopathy due to Infectious causes continues to be much more prevalent in the majority of Asian nations. Meningitis and encephalitis, two infections of the central nervous system, can cause acute encephalopathy, which is frequently accompanied by headache and fever (as well as neck stiffness in the former). Bacteria, viruses, mycoplasma, rickettsiae, protozoa, metazoa, and fungi are examples of infectious agents responsible for encephalopathy. The distinct clinico-pathological disease caused by viral encephalitis is due to direct infection of neural cells accompanied by perivascular inflammation, neuronal destruction, neuronophagia, and tissue necrosis that is most commonly found in grey matter. The other condition is postinfectious encephalomyelitis which develops after a period of bacterial and viral infections. Although there is no proof that neural cells have been directly infected, there is widespread perivenular inflammation and demyelination that is restricted to the brain's white matter. [4]

ENCEPHALOPATHY DUE TO SYSTEMIC CONDITIONS

Uremic Encephalopathy [1]

Uremic encephalopathy can be characterized as the subacute or acute type of organic brain syndrome which is prevalent in acute or chronic renal failure with a glomerular filtration rate below 15% than normal. As with other organic brain syndromes, patients are presented with variable disorders of consciousness, psychomotor behavior alteration, impaired thinking, memory, speech, perception, sleep, and emotions. [7]

The term uremic encephalopathy refers to the non-specific neurologic symptoms of uremia. Sluggishness, easy fatigue, daytime drowsiness, and insomnia are the most common symptoms, including sleep-inversion, itching, inability to perform cognitive tasks, slurred speech, anorexia, nausea, and vomiting. [8] Other symptoms which are prevalent include; restlessness, myoclonus, asterixis, disorientation, confusion, and ataxia. In severe conditions convulsions and coma are reported. [8]

There are some particularly notable features of uremic encephalopathy. The degree to which renal insufficiency progresses determines the symptoms' severity and the overall rate of progression. Patients with acute renal failure typically experience more severe and rapid uremic symptoms than patients with chronic renal failure. Dialysis or renal transplantation can quickly reduce the symptoms, and maintenance dialysis regimens can

suppress them. Therefore, it is crucial to accurately identify the encephalopathy of renal failure because it can be quickly and effectively treated using widely accessible dialysis techniques. Uremic encephalopathy has undoubtedly varied and complex causes. [9] [10]

Pathophysiology of uremic encephalopathy [14] - Even though uremic encephalopathy is caused by a variety of factors, the majority of studies have found no connection between encephalopathy and any of the frequently observed signs of renal failure. There has been a lot of debate in recent years about PTH's potential function as a uremic toxin. There is a lot of evidence to support the idea that PTH may have negative effects on the central nervous system. [1] [13]

Even in the absence of compromised renal function, PTH has been shown to have effects on the human central nervous system. According to reports, one of the most typical signs of primary hyperparathyroidism is neuropsychiatric symptoms. EEG changes similar to those seen in patients with acute renal failure are also present in patients with primary hyperparathyroidism. [1]

It is still unclear how PTH might affect the central nervous system's ability to function. Patients with hyperparathyroidism have higher calcium levels in a variety of tissues, including the skin, cornea, blood vessels, brain, and heart. This finding raises the possibility that PTH may in some way make

it easier for Calcium ions to enter these tissues. The discovery of elevated calcium levels in the brains of both dogs and humans with secondary hyperparathyroidism and acute or chronic renal disease is consistent with the theory that some of the abnormalities in the EEG and dysfunction of the central nervous system observed in acute renal failure or chronic renal failure may be caused by a PTH-mediated increase in brain calcium. [1]

Diagnosis of Uremic Encephalopathy - Since patients with renal failure often have other concurrent illnesses that could also have other encephalopathic effects, the differential diagnosis is even more difficult. Dialysis therapy will help patients with renal failure return to a more typical body fluid composition. Despite the possibility that several causes of encephalopathy could manifest concurrently, uremic encephalopathy can typically be distinguished from other types of encephalopathies using standard laboratory techniques. [1]

It can be challenging to tell whether an encephalopathy is caused by hepatic or renal causes in patients who also have other medical issues, such as advanced liver disease with hepatic insufficiency. Because the primary pathway for urea elimination is unavailable in patients with renal failure, blood urea levels rise. The increased plasma urea contributes to the increased amount of urea that enters the colon. Then, colonic bacteria and mucosal enzymes react on urea similarly to how proteins and amino acids

do. As a result, uremic patients produce more ammonia, which could either raise plasma ammonia levels or cause this test to be read incorrectly.

This additional ammonia load may present a stress that the sick liver is unable to adequately handle in patients who also have cirrhosis or another type of liver failure. As a result, encephalopathy may develop, and blood and central nervous system ammonia levels may rise. Because cirrhosis and end-stage kidney disease both increase ammonia levels in the blood and central nervous system, patients with these conditions are particularly at risk for developing encephalopathy. It should be noted that in patients with severe liver disease, plasma urea and serum creatinine do not always accurately reflect renal function. Numerous patients with cirrhosis, ascites, and normal plasma urea and creatinine levels may have severe renal functional impairment, according to recent studies. Clinically, it may be challenging to distinguish hepatic from uremic encephalopathy in such patients. [11] [12] [13]

Hepatic encephalopathy

Hepatic encephalopathy (HE), also known as portal-systemic encephalopathy, is a complex syndrome that develops after liver cirrhosis or acute liver failure impairs hepatocellular function. Following hepatocellular impairment, more than 20 different compounds are present in the circulation in higher concentrations, but ammonia is the most

significant of these. HE is not a solitary clinical entity, though. It could be an indication of brain edema, brain atrophy, reversible metabolic encephalopathy, or any combination of these conditions. It is still unclear what mechanisms lead to brain dysfunction in liver failure. These elements have a direct connection to liver failure (e.g. decreased metabolism of ammonia). Unless the underlying liver disease is successfully treated, HE is associated with poor survival and a high risk of recurrence. [16] [17]

Pathogenesis of Hepatic Encephalopathy

Neurotoxins **-** The neurotoxin most closely related to HE is ammonia. The main source of ammonia is the gastrointestinal (GI) tract. Ammonia is produced by enterocytes from glutamine and by colonic bacterial catabolism of nitrogenous sources (such as blood after GI bleeding). Nearly all of the ammonia in the portal vein is eliminated by an intact liver, where it is converted to glutamine and kept out of systemic circulation. Because of impaired liver function and blood shunting around the liver, advanced liver disease is accompanied by an increase in blood ammonia. Since muscle is a key site for extrahepatic ammonia removal, muscle wasting, which is common in these patients, may also be a factor. [18]

Impairment of neurotransmission **-** In various experimental models of (mostly) acute liver failure, several neurotransmitter systems have been investigated using neurochemical, neurobehavioral, and

electrophysiological techniques. Most articles discuss modifications to the GABA-benzodiazepine-ergic, dopaminergic, serotoninergic, and glutamate-ergic neurotransmitter systems. [19] [20] [21]

In the brain, sera, and cerebrospinal fluid of people with type A and type C HE, substances that are involved in the activation of the GABAA-ergic neurotransmission have been isolated, characterised, and positively identified as benzodiazepines by gas chromatography-mass spectroscopy. [22] Strong, selective positive allosteric modulators of the GABAA receptor complex are neurosteroids. Patients with hepatic coma had higher levels of allopregnenolone and pregnenolone, two neurosteroid precursors, at pathophysiologically significant concentrations. The central nervous system symptoms of HE are caused by activation of the astrocytic 18-kDa translocator protein, formerly known as peripheral-type benzodiazepine receptors. [23] [24]

Systemic response to infections and neuroinflammation **-** Changes in cerebral blood flow, brain metabolites, and the release of inflammatory mediators are additional potential causes of brain dysfunction. Importantly, these processes take place without directly infecting brain tissue. Cirrhotic patients are known to have functional immunosuppression and to be more vulnerable to infections. It's unclear whether infections cause HE to worsen or whether the inflammatory response does. Proinflammatory cytokines

and mediators are released and circulated, which causes the systemic inflammatory response syndrome. From delirium to coma, sepsis-associated worsening encephalopathy is characterised by changes in mental status and motor activity. [25] [26]

Clinical presentation of Hepatic Encephalopathy

A wide range of generalised neurological and psychiatric manifestations are brought on by HE. Only psychometric tests focused on attention, working memory, psychomotor speed, and visuospatial ability, as well as electrophysiological and other functional brain measures, are altered by HE in its mildest form.

As HE worsens, the patient's relatives may notice personality changes like apathy, irritability, and disinhibition in addition to overt changes in consciousness and motor function. While complete sleep-wake cycle reversal is less frequently observed, disturbances of the sleep-wake cycle with excessive daytime sleepiness are more common. Patients may exhibit inappropriate behavior, an acute confusional state with agitation or somnolence, stupor, and ultimately coma [43]. Patients may also experience progressive disorientation to time and space. Disorientation or asterixis is used by the most recent ISHEN (International Society for Hepatic Encephalopathy and Nitrogen Metabolism) consensus as the first indication of overt HE. [23]

In reality, asterixis, also known as a "flapping tremor," is a negative myoclonus that results in loss of postural tone and is frequently present in the early to middle stages of HE that precede stupor or coma. Actions that call for postural tones, like hyperextending the wrists with separated fingers or rhythmically squeezing the examiner's fingers, are effective ways to elicit it. The tongue, eyelids, feet, legs, and arms are some other areas where asterixis can be observed. Asterixis can occur in other diseases, like uremia, so it is not pathognomonic of HE. Notably, the motor and mental (either cognitive or behavioural) signs of HE may not manifest or progress simultaneously in each person, resulting in diagnostic challenges. [23]

Classification of Hepatic Encephalopathy [27]

HE is generally classified based on four factors;

1. According to the disease :
    a. **Type A – acute liver failure**
    b. **Type B – portosystemic bypass or shunting**
    c. **Type C – Cirrhosis**
2. According to the severity : The divisions within the HE continuum are arbitrary. A grading scheme for such purposes is provided for clinical and research use. [Table 2]

Table 2 – West Haven Criteria (WHC) for hepatic encephalopathy and clinical description

WHC	ISHEN	Description	Operative criteria suggestions
Unimpaired		No encephalopathy history	Tested and normal
Minimal	Covert	Tests measuring psychomotor speed/executive functions or neurophysiological changes without clinical signs of mental change can be altered psychometrically or neuropsychologically .	Abnormal results of established psychometric or neuropsychological tests without clinical manifestations
Grade 1		Trivial lack of awareness	Despite being oriented in time and

		Euphoria or anxiety Shortened attention span Impairment of addition or subtraction Altered sleep rhythm	space (see below), the patient appears to have some cognitive/behavioral decay for his or her standards on clinical examination or as per the caregivers
Grade II	Overt	Lethargy or apathy Disorientation for time Obvious personality change Inappropriate behavior Dyspraxia Asterixis	Disoriented for time (at least three of the following are wrong: day of the month, day of the week, month, season, or year) ± the other mentioned symptoms
Grade III		Somnolence to semistupor	Disoriented also for space (at least three

		Responsive to stimuli Confused Gross disorientation Bizarre behavior	of the following wrongly reported: country, state [or region], city, or place) ± the other mentioned symptoms
Grade IV		Coma	Does not respond to any stimuli

3. According to the time course of the disease :
    a. Episodic HE
    b. Recurrent HE, occurring within a time interval of < 6 months
    c. Persistent HE, shows behavioral alterations with relapsed overt HE
4. According to the existence of precipitating factors :
    a. Non-precipitated
    b. Precipitated; bouts of episodic HE type C, factors include;
        - alkalosis;
        - dehydration;
        - fluid restriction;

- diuretics;
- diarrhea;
- vomiting;
- arterial hypotension/hypovolemia;
- gastrointestinal bleeding;
- peripheral vasodilatation;
- shock;
- hypoxia;
- anemia;
- high protein intake;
- constipation;
- hyponatremia;
- sedative drugs: morphine, benzodiazepines;
- hypokalaemia

Diagnosis of HE [23]

Laboratory testing - In HE patients with chronic liver disease, high blood ammonia levels by themselves do not add any diagnostic, staging, or prognostic value. The diagnosis of HE is called into question if an ammonia level is measured in a patient with overt HE and it is normal. Repeated measurements of ammonia may be useful to evaluate the effectiveness of "ammonia-lowering" medications. It may be logistically difficult to

measure blood ammonia accurately, which should be taken into account. Since ammonia can be found in plasma, arterial blood, or venous blood, the pertinent normal value should be used. There are numerous methods, but they should only be used when reliable analyses using laboratory standards are possible. [28]

The testing methods most widely used for HE are:

1. **Portosystemic encephalopathy (PSE) Syndrome Test**
2. **The Critical Flicker Frequency Test (CFF)**
3. **The Continuous Reaction Time Test (CRT)**
4. **The inhibitory control test (ICT)**
5. **The Stroop test**
6. **Electroencephalograph (EEG)**

Treatment of HE

Only overt HE is currently routinely treated. As its name suggests, minimal and covert HE is typically diagnosed using the techniques mentioned above because it is not readily apparent during a routine clinical examination. Even though it is subtle, minimal, and covert HE can have a significant impact on a patient's ability to go about their daily lives. Special circumstances, such as impairment in driving abilities, work performance, quality of life, or cognitive complaints, may exist where it may be necessary to treat such a patient. Patients with higher grades of HE who

need more intensive monitoring or who are unable to protect their airways should be treated in an intensive care setting. In patients with advanced cirrhosis, alternative causes of encephalopathy are not uncommon. According to medical terminology, an episode of encephalopathy may not be referred to as HE if other encephalopathy causes are present. Both hepatic and non-HE conditions are treated in the clinical setting. Controlling precipitating factors in the management of overt HE is of paramount importance, as nearly 90% of patients can be treated with just correction of the precipitating factor. [29]

Post-cardiac arrest encephalopathy

Post-cardiac arrest encephalopathy, also known as post-resuscitation encephalopathy, is becoming more common due to the growth of bystander cardiopulmonary resuscitation (CPR), portable defibrillators, and improvements in intensive care. [30]

Due to its higher blood supply and metabolic demand than the brainstem, the cerebral cortex is more susceptible to hypoxia and ischemia. The brainstem can be irreversibly damaged by prolonged ischemic or hypoxic insults, which can result in a diagnosis of brain death (defined as a coma with no brainstem reflexes) and termination of care unless the deceased person's organs are being harvested for donation. Due to diffuse laminar cortical necrosis, the initial coma in many patients progresses to a

vegetative state where there is no conscious awareness of the surroundings. The likelihood of neurological recovery for the cardiac arrest survivor is frequently assessed by the neurologist so that the family and primary caregivers can decide whether to withdraw life support in a well-informed manner. The Glasgow Outcome Scale (GOS) divides the prognosis for cardiac arrest into two categories: poor outcome, denoted by scores of 1 (death), 2, and 3, and good outcome, denoted by scores of 4 (moderate disability, able to participate in activities of daily life), and 5 (good recovery, able to return to work or school). [31]

The clinical findings and ancillary tests used to establish prognosis should have a nearly zero rate of false positives to determine the poor outcome to prevent discontinuing care in patients who have a chance for a good outcome. The neurological exam is still essential to determine the prognosis. On days 1-3 of post cardiac arrest, some ancillary tests, such as EEG (electrical silence and burst-suppression predict poor prognosis, whereas a reactive continuous pattern predicts good outcome), somatosensory evoked potentials (SSEPs, the nitrous oxide bilateral absence of response after stimulation of median nerve predicts poor prognosis), and brain MRI, can improve the prognostic accuracy regarding the long-term outcome. [31]

Long-term neurological complications resulting from a brain injury can occur after coma recovery in hypoxic-ischemic encephalopathy. Watershed infarctions, delayed post-anoxic myoclonus (Lance-Adams syndrome), additional movement disorders, cognitive impairment, and delayed demyelination are among them. [32]

Brain watershed regions are more prone to necrosis in the presence of prolonged hypotension and ischemia. Balint's syndrome (simultanagnosia, oculomotor apraxia, and optic ataxia) results from bilateral infarction of the watershed between middle and posterior cerebral arteries (visual association cortex, Brodmann areas 18/19). When evaluating the patient's response to repeated coma examinations, these syndromes need to be carefully taken into account. [33]

Although clonazepam, valproic acid, and levetiracetam are efficient in controlling myoclonus, it is typically necessary to combine two or more medications. Postanoxic myoclonus does not have a worse prognosis for encephalopathy than myoclonic status epilepticus and tends to improve over weeks to months. It is clinically challenging to differentiate between postanoxic myoclonus and myoclonic status epilepticus, which depends on EEG results. Periodic or continuous spike waves are linked to myoclonic status epilepticus. Hypoxic encephalopathy is more likely to cause post-

anoxic myoclonus, whereas cardiac arrest is more likely to cause myoclonic status epilepticus. [34]

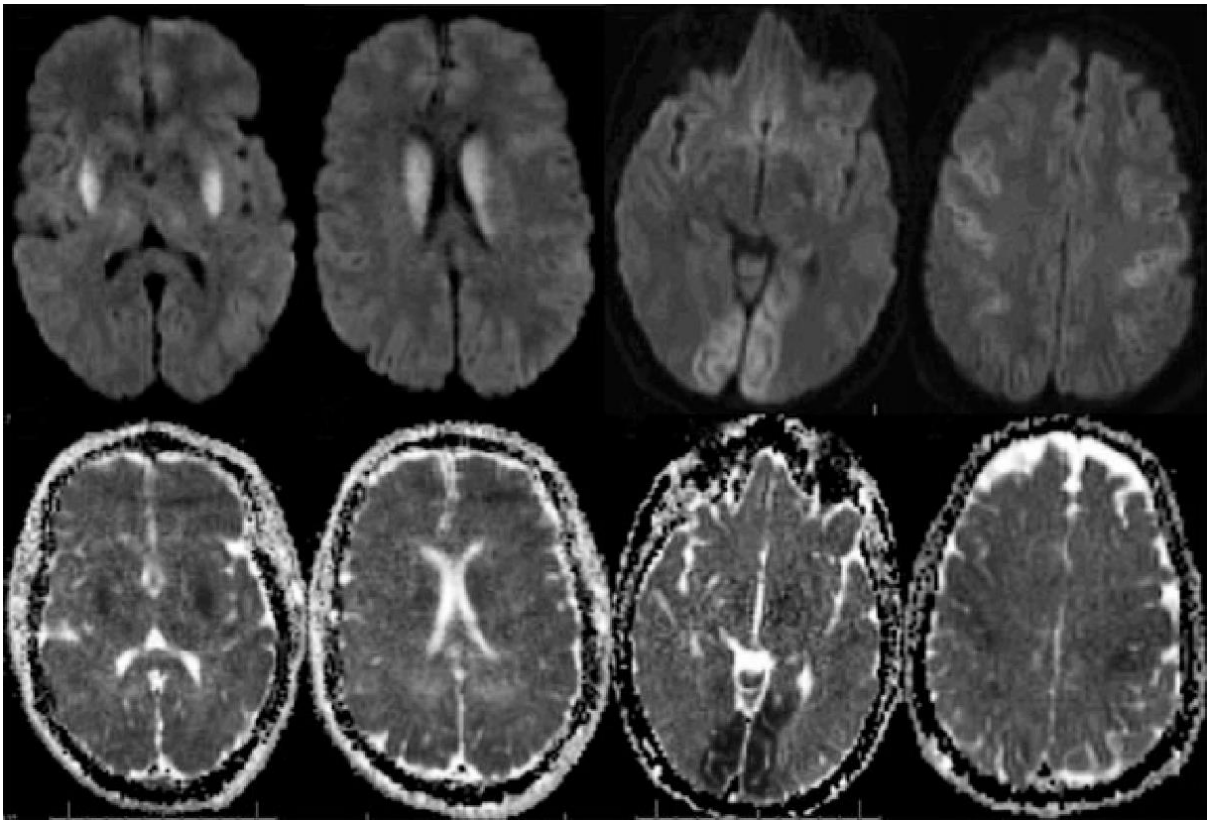

FIGURE 1 – Post-Cardiac arrest Encephalopathy - Brain MRI of a 24-year-old male who suffered an electrocution leading to cardiac arrest while working on a power line.

After a cardiac arrest, cognitive impairment can range from mild amnesia to severe dementia. The amnestic syndrome frequently stands out more than other cognitive deficits in the latter scenario. The vulnerability of the hippocampi to hypoxia and ischemia, particularly the pyramidal neurons of the CA1 subfield, accounts for dense amnesia. [33] [34]

Hypercapnic/ Carbon dioxide encephalopathy

Acute respiratory failure may be accompanied by hypercapnia or elevated carbon dioxide (CO2) levels, usually when chronic obstructive pulmonary disease (COPD) is being acutely exacerbated by an upper respiratory infection. (1) Decreased level of consciousness in those with hypercapnic encephalopathy; (2) headache (caused by cerebral vasodilation and increased intracranial pressure); (3) tremor and asterixis; and (4) altered mental status frequently accompanied by agitation and even combativeness if the patient is not comatose. An arterial blood gas is necessary for the diagnosis because oximetry may be normal after oxygen supplementation even though the PaCO2 level is elevated. The level of PaCO2 and the severity of respiratory acidosis are correlated with the severity of encephalopathy. As with a COPD exacerbation, antibiotics, a brief course of steroids, and bronchodilators are used to treat hypercapnic encephalopathy. [35]

The critical care management of acute intracranial hypertension from a variety of causes includes ventilator-induced hyperventilation to achieve hypocapnia in severe cases. As hypercapnia is treated, the encephalopathy typically gets better, but some patients may still struggle with executive dysfunction and attention deficits. [36]

METABOLIC ENCEPHALOPATHIES

Hypoglycemia

A reduced level of consciousness is the presenting complaint in patients suffering from hypoglycemia. It most frequently occurs as an unintended side effect of anti-diabetic medications, though it can also happen after an intentional (suicidal or fake) insulin overdose. Rarely, an occult pancreatic insulinoma can cause hypoglycemia, in which case the plasma level of C-peptide will be higher. Transient hemiparesis and seizures are additional potential clinical indicators. These symptoms may go away following a glucose infusion and/or glucagon injection. Prolonged and severe hypoglycemia can result in death or a persistent vegetative state, not to mention irreversible brain damage. MRI shows restricted diffusion in diffusion-weighted imaging (DWI) and apparent diffusion coefficient (ADC) sequences in basal ganglia, cerebral cortex, substantia nigra, and hippocampus, thought to be vulnerable to hypoglycemia due to their higher energetic demand. These MRI changes' prognostic significance is still up for debate. [38]

The level of glucose does not accurately predict clinical severity or prognosis. Although it is often difficult to determine, the most crucial prognostic factor is likely the duration of hypoglycemia.

Hyperglycemia

Patients with type 2 diabetes who have poor glycemic control run the risk of developing a nonketotic hyperosmolar coma, which has a higher mortality rate. The majority of patients are elderly and exhibit clear signs of dehydration during an examination. Coma in these patients is primarily brought on by cerebral dehydration secondary to osmotic imbalance and correlates with plasma glucose level and hyperosmolality. The neurological symptoms may include hemichorea-hemiballism, generalised or focal seizures, Epilepsia partialis continua, and stroke-like symptoms, which generally resolve after treating hyperglycemia. [39] [40]

Hyponatremia

Hyponatremia is characterised by a drop in serum sodium levels of 136 mmol/L. Hyponatremia greater than 125 mmol/L usually has no symptoms. Hyponatremia that is severe (125 mmol/L) and quick (within a few hours) can result in seizures, coma, brain herniation, death, headache, nausea, vomiting, cramps in the muscles, lethargy, restlessness, confusion, depressed deep tendon reflexes. Cerebral edema develops quickly in these severe cases as a result of the osmotic imbalance brought on by hyponatremia, which shifts water into the brain cells.

The focus of this chapter does not include the diagnostic assessment or therapeutic management of hyponatremia. Due to the rapid shrinkage of

the brain, aggressive correction of hyponatremia can cause osmotic demyelination of pontine (central pontine myelinolysis) and extrapontine neurons one to several days later. Quadriplegia, pseudobulbar palsy, seizures, and coma are all symptoms of the condition. Facial diplegia with automatic-voluntary dissociation, dysarthria, dysphagia, and emotional lability are also symptoms. Therefore, severe acute life-threatening hyponatremia should only be treated with hypertonic saline (3%). [41] [42]

Diabetic Ketoacidosis (DKA) induced encephalopathy [1] [23] [43]

Cerebral edema is prevalent in less than 1% of DKA patients. Narrowing of cerebral ventricles has been seen with the help of MRI and an initial pCO_2 level indicates the presence of cerebral edema.

The relationship between hypoperfusion and DKA encephalopathy is supported by subtle findings;

As a dynamic marker of neuronal dysfunction and integrity, N-acetyl aspartate (NAA) is a neuronal-axonal marker. During acute DKA, the basal ganglia's NAA to creatine ratio falls, which may indicate that the integrity of the neurons is weakened. Children who present with the most dehydration and hypocapnia, risk developing cerebral edema during DKA. Volume reduction may result in hypoperfusion and brain ischemia, particularly because DKA's accompanying hyperventilation can cause cerebral vasoconstriction particularly in more delicate regions like the

basal ganglia. Basal ganglia lactate peaks have been found using proton magnetic resonance spectroscopy, suggesting anaerobic cerebral metabolism. [1] [23]

On the other hand, cerebral edema in DKA always affects the entire brain. It makes sense that people are reluctant to give up on osmotic theories for the origin of cerebral edema associated with DKA. It is nearly impossible to control the rate of change of this gradient during the treatment phase, and both the hypertonicity in untreated DKA and the rapidly changing osmotic gradient in DKA resolution may contribute to fluid movement between neurons and interstitium. [23]

In a large DKA surveillance study conducted in the United Kingdom, it was discovered that, in the absence of cerebral edema, metabolic acidosis assessed via the serum pH directly relates to the level of consciousness, with confusion and agitation being associated with a pH of 6.96 and coma occurring at 6.88 [43]

AUTOIMMUNE ENCEPHALOPATHIES

Autoimmune encephalopathies are a diverse group of encephalopathies with immune-mediated pathogenesis that is either suspected or established. Recently, a consensus set of general diagnostic criteria for potential autoimmune encephalitis, as well as specific diagnostic criteria for each of these encephalitis and a diagnostic algorithm to group them, were published by an international group of top neuro immunologists. [43]

If the following three criteria are met, an autoimmune cause should be suspected (and an autoimmune encephalitis diagnosis may be made).

1. Sub-acute onset (rapid progression under three months) of encephalopathy
2. new focal CNS deficits, newly appearing seizures, CSF pleocytosis, abnormal MRI (T2/FLAIR hyper signal in one or both medial temporal lobes in limbic encephalitis or multifocal demyelinating or inflammatory lesions in grey matter, white matter, or both); and
3. a reasonable ruling out of other causes (such as infectious, toxic, metabolic, or stroke)

Table 3 – Differential Diagnosis of Autoimmune Encephalopathy [1]

[23]

	Primary CNS Vasculitis	*Hashimoto's Encephalopathy*	*Paraneoplastic Limbic Encephalitis*	*Anti-NMDAR Antibody Encephalitis*	*Anti-VGKC-Complex Antibody Encephalitis*	*Inflammatory CAA*
Median age (range)	50 (37-59)	56 (27-84)	63 (28-82) for anti-Hu	21 (1-85)	63 (19-83)	67 (43-82)
Sex (F:M)	1:1	3.5:1	1:3	4:1	1:2	1:1
Clinical picture	Headache, confusion, focal deficits, seizures	Headache, seizures, psychosis, ataxia, tremor, myoclonus, stroke-like deficits	Seizures, memory loss, psychosis	Psychosis, seizures, memory loss, movement disorders, dysautonomia, sleep disorders	Seizures (including faciobrachial dystonic) and memory loss (anti-Lgi1), neuromyotonia and Morvan's syndrome (anti-Caspr2)	Headache, confusion, seizures
Other clinical clues	Constitutional syndrome is rare	Hx of autoimmune hypothyroidism	Hx of smoking and constitutional syndrome, search for SCLC in all and testicular cancer in men	Search for teratoma in women	Underlying cancer is rare	Hx of lobar hemorrhage or AD-like cognitive decline, *APOE*ε4 genotype
Antibody	None	Anti-thyroid TPO/TG	Anti-Hu Anti-Ma2 Anti-CV2/CRMP5	Anti-NMDAR Ab (CSF more sensitive than serum)	Anti-Lgi1, anti-Caspr2 Others: anti-contactin-2, anti-DPPX	Anti-Aβ (CSF)
Other lab clues	ESR/CRP rarely increased	Normal or high TSH Normal free T3/T4	Tumor markers may be helpful	---	Hyponatremia (SIADH) in 60% (anti-Lgi1)	---
MRI findings	Multiple cortical and subcortical infarctions of different ages, perivascular and leptomeningeal Gd enhancement	Normal or non-specific WM T2/FLAIR	MTL T2/FLAIR	Normal or non-specific T2/FLAIR	MTL T2/FLAIR in 60% (anti-Lgi1)	Multiple cortical microbleeds, asymmetric WM T2/FLAIR
Other diagnostic tests	Angiogram, brain biopsy	EEG, brain biopsy	Whole body CT/PET scan, mammogram, testicular / transvaginal US	Whole body CT/PET scan, mammogram, testicular / transvaginal US	Whole body CT/PET scan, mammogram, testicular / transvaginal US	Amyloid PET/SPECT, brain biopsy
Pathology	Granulomatous angiitis, lymphocytic angiitis, fibrinoid necrosis, Aβ vascular deposits may be present	Perivascular lymphocytic infiltrate (non-vasculitic acute meningo-encephalitis)	1 Tumor immunoreactivity that disappears after pre-incubation with patient's serum 2 Neuronal loss, perivascular "cuffing" and microglial nodules in limbic structures	1 Immunoreactivity of patient's serum against neural tissue present in tumor 2 Neuronal loss, perivascular "cuffing" and microglial nodules	Neuronal loss, perivascular "cuffing" and microglial nodules in limbic structures	Aβ vascular deposits with: • Granulomatous angiitis (ABRA) or • Perivascular lymphocytic infiltrate (CAA-RI)

Anti-NMDA Encephalopathy [44-48]

In contrast to limbic encephalitis, anti-NMDA receptor antibody encephalitis affects more than just the medial temporal lobe. Anti-NMDA receptor antibody encephalitis was initially identified in young women as a paraneoplastic syndrome linked to ovarian teratoma, but it is now known to affect both sexes and a wider age range. In males of any age and, generally, in older (>45 years old) adult patients, the frequency of underlying tumours is very low; however, when they are discovered, they are typically carcinomas rather than teratomas. Finally, most patients exhibit comparable symptoms regardless of age. [44]

A primary psychiatric syndrome with psychosis, delusions, visual and auditory hallucinations, aggression, mania or depression, and occasionally catatonia is presented by some patients. [44] [45]

Children are more likely to experience abnormal movements, such as rhythmic unilateral dystonic postures, oral dyskinesias, and opisthotonos, as well as speech difficulties and atypical deficits like hemiparesis and ataxia. Adults are more likely to experience memory loss, dysautonomia (such as hypo or hypertension, hypo or hyperthermia, hypersalivation, diaphoresis, pupil dilation, and enuresis in children), and central hypoventilation. [46] Male adult patients present with seizures more

frequently than female patients do. Most patients experience similar symptoms regardless of age. [47]

There is no distinct MRI finding; it can be normal or show T2/FLAIR hyperintensities, typically outside the medial temporal lobe. In the majority of patients, CSF demonstrates lymphocytic pleocytosis with or without elevated protein and normal glucose. Positive oligoclonal bands are possible. Anti-NMDA receptor antibody detection in CSF is more sensitive than in serum (100% vs. 87%). Epileptiform activity may or may not be present, but EEG typically reveals focal or generalized slowing of background activity. In a subset of patients, a pattern known as "extreme delta brush" that consists of a delta rhythm with superimposed beta activity on the delta waves and is connected to a worse clinical outcome has been described. [47] [48]

Anti-VGKC-complex antibody encephalitis

Another potentially reversible cause of autoimmune encephalopathy is anti-voltage gated potassium channel (VGKC)-complex antibody encephalitis. It primarily impacts the medial temporal lobe, similar to paraneoplastic limbic encephalitis (i.e., anti-Hu or anti-Ma2) but different from anti-NMDA receptor antibody encephalitis. The variety of autoantibodies against the VGKC complex is growing which mainly

includes leucine-rich glioma inactivated 1 (LGI1) and contactin associated protein 2 (CASPR 2). [49] [50]

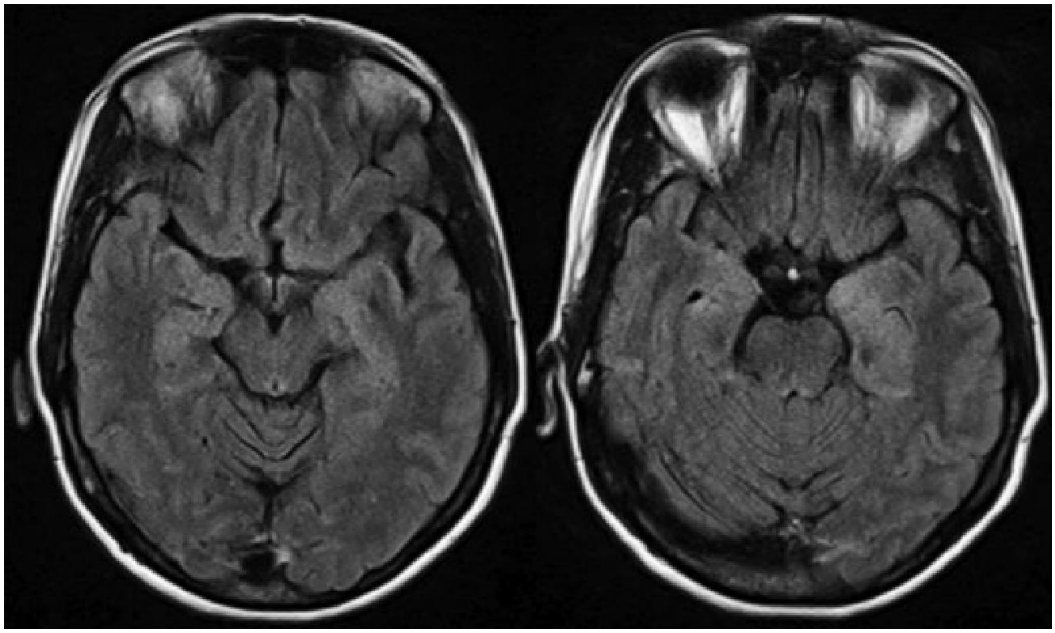

Figure 2- Anti-VGKC-complex antibody encephalitis

TOXIN INDUCED ENCEPHALOPATHIES

Toxin exposure leads to brain dysfunction, which is referred to as "toxic encephalopathy". There is a spectrum of symptoms associated with toxic encephalopathy, from subtle clinical disorders to overt subclinical deficits. The affected brain regions and cell types are related to the clinical signs and symptoms of toxic encephalopathy. The clinical characteristics, diagnostic modalities, and toxicological ramifications of toxic encephalopathy are schematically reviewed in this article. The review focuses on the most significant occupational causes of toxic encephalopathy, pharmaceutical causes and the neurotoxic effects of alcohol. [51]

Alcohol intoxication and withdrawal

When a patient with a history of alcohol abuse is admitted to the hospital for any other reason, alcohol withdrawal syndrome (AWS), a potentially serious complication, may occur. A constellation of autonomic and behavioural symptoms, such as nausea, vomiting, diaphoresis, tremor, hypertension, tachycardia, anxiety, insomnia, and agitation, typically appear 24 to 48 hours after the last drink. AWS can cause two potentially fatal complications, including delirium tremens (encephalopathy with delirium, tactile, auditory, and visual hallucinations, as well as a severe hyperadrenergic surge that can cause myocardial infarction and malignant

arrhythmias), alcohol withdrawal seizures, or even status epilepticus (typically generalised tonic-clonic) if it is not recognised or treated properly.

In addition to being effective at preventing and ending alcohol withdrawal seizures, benzodiazepines are still the first line of treatment for AWS. A symptom-triggered protocol is preferred over a scheduled "round-the-clock" protocol because it has been shown to lower the proportion of patients who require treatment, the total dose of benzodiazepine given to those who receive it, and the proportion of patients who require intubation and mechanical ventilation as a result of excessive sedation. Because of its simple metabolism to an inactive metabolite through hepatic glucuronidation and intermediate half-life (10–20 h), which is preserved even in cirrhotic patients, lorazepam is the preferred benzodiazepine. Due to their oxidation to active metabolites in the liver, diazepam and chlordiazepoxide have a longer half-life (>24 h) and can build up in the elderly and liver failure situations. Additionally, adipose tissue can accumulate diazepam, which has an unpredictable long half-life. [51]

Wernicke’s encephalopathy

The most common central nervous system (CNS) complication of alcohol abuse is Wernicke's encephalopathy (WE), which is caused by thiamine or vitamin B1 deficiency. Patients with alcoholism are more susceptible to

WE due to both their typically low dietary intake of thiamine and the direct effects of alcohol on thiamine metabolism. Alcohol interferes at various levels, reducing the availability of thiamine (intestinal absorption, activation, storage, and renal excretion). In addition, alcohol use increases the thiamine requirement because thiamine is necessary to metabolize alcohol. Besides alcohol-dependent patients, other patient groups at risk of WE are patients with hypomagnesemia (i.e., on chronic diuretics, on parenteral nutrition), patients on peritoneal dialysis or hemodialysis, patients with recurrent vomiting or chronic diarrhea and malabsorption. [52]

In 85% of cases, the presentation is mild, for instance with only gait imbalance and nystagmus, even though the full clinical picture is characterised by confusion, binocular diplopia, and gait ataxia.

The diagnosis of WE can be more difficult if there are signs of alcohol abuse present, such as intoxication or withdrawal symptoms, peripheral neuropathy (dry beriberi), or even chronic cerebellar degeneration. Although its sensitivity is only about 50%, T2/FLAIR/DWI hyperintensities of the periaqueductal grey matter, tectum, mammillary bodies, medial thalami, hypothalamus, and superior vermis of the cerebellum have shown >90% specificity. These MRI results are consistent with the autopsy findings of necrosis and petechial haemorrhages. Mammillary bodies and the medial thalamus are involved in confusion,

stupor, and coma; oculomotor and/or abducens nuclei and/or colliculi are involved in painless ophthalmoplegia, and the superior cerebellar vermis is involved in gait and trunk ataxia. Bilateral visual disturbances accompanied by papilledema and/or retinal haemorrhages, hypothermia or hyperthermia brought on by hypothalamic involvement, and epileptic seizures brought on by cortical involvement are rarer manifestations. There could be high-output heart failure (wet beriberi) with pulmonary and peripheral edema. [52]

Parenteral replacement of thiamine is preferred over oral therapy to ensure maximum bioavailability.

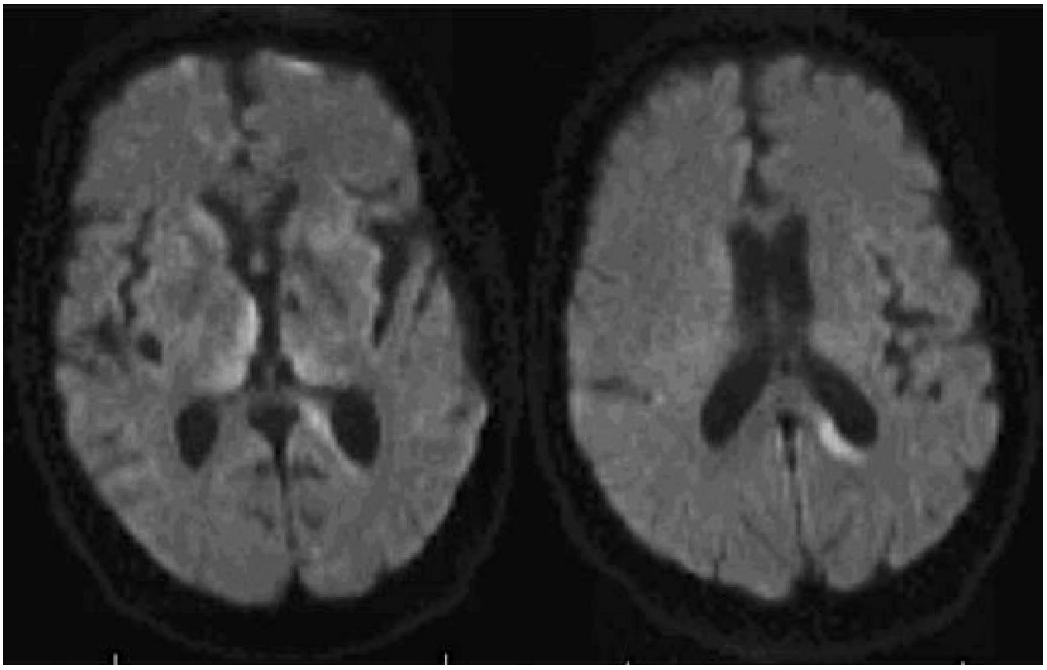

Figure 4 – Wernicke's encephalopathy

It is important to treat hypomagnesemia because it can make WE resistant to thiamine replacement. If hyponatremia is present, it should be treated carefully to prevent osmotic demyelination. Giving oral or parenteral carbohydrates to an alcoholic who is undernourished before thiamine can hasten WE and must be avoided. WE can be fatal or cause Korsakoff's psychosis, which is characterised by irreversible severe retrograde and anterograde amnesia, frequently accompanied by confabulations, as a result of deterioration and atrophy of the mammillary bodies and medial thalami. [52]

AIM AND OBJECTIVES

- To enumerate various non-infectious causes of encephalopathy in patients admitted to IMCU.
- To identify high-risk groups susceptible to various non-infectious causes of encephalopathy.

MATERIALS AND METHODS

Study design: Descriptive cross-sectional study.

Study duration: 18 months time period from getting thesis approval (March 2021 - August 2022).

Study population: Patients above 18 years of age admitted to IMCU with encephalopathy.

Sample size: 235

Patients above 18yrs admitted to IMCU with encephalopathy who meets the inclusion and exclusion criteria and consent to participate.

Inclusion criteria: All patients of age above 18 yrs admitted to IMCU who met any of the following criteria:

- GCS < 15
- not oriented in time, place, person
- unable to stay alert or awake

Exclusion criteria:

- Those patients who had a history of trauma
- Those patients who had a history of psychiatric illness
- Those patients with infectious etiology
- TC <4000 or >12,000

- Temp <36°C or >38°C
- Cultures positive
- Identified source of infection

Data collection techniques:

- Detailed history taking
- Assessment of sensorium using Glasgow Coma Score
- Routine blood investigations
- Relevant radiological investigations
- Body fluid analysis

Investigations:

- Vitals - BP, PR, RR, spo2
- Complete blood count
- Liver function test, Renal function test
- Serum electrolytes, Serum glucose
- Serum ammonia
- Arterial blood gas analysis
- Coagulation profile
- Chest X-ray, USG abdomen
- Echocardiography
- CT or MRI brain
- CT chest, abdomen as clinically indicated

- Blood culture/urine culture/sputum culture/pus culture as clinically indicated.
- Csf analysis whenever indicated.

Statistical analysis: Collected data were verified prior to computerized data entry. The Statistical Package for Social Sciences was used for statistical analysis of collected data.

RESULTS

Age distribution

The prevalence of encephalopathy was seen higher in the age group between 40-49 years of age (65 participants), followed by the age group of 50-59 years of age (64 participants).

Table 1 – Age distribution of patients

AGE GROUP	Frequency	Percent
20-29	27	11.5%
30-39	31	13.2%
40-49	65	27.7%
50-59	64	27.2%
60-69	43	18.3%
>70	5	2.1%
Total	235	100.0%

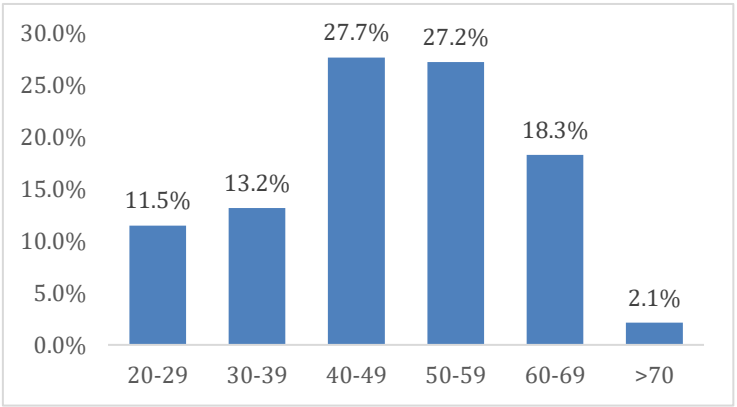

Figure 1 – Age distribution

Gender distribution

Male predominance was noted higher in the current study, with 140 male participants and 95 females.

Table 2- Gender distribution

SEX	Frequency	Percent
Female	95	40.4%
Male	140	59.6%
Total	235	100.0 %

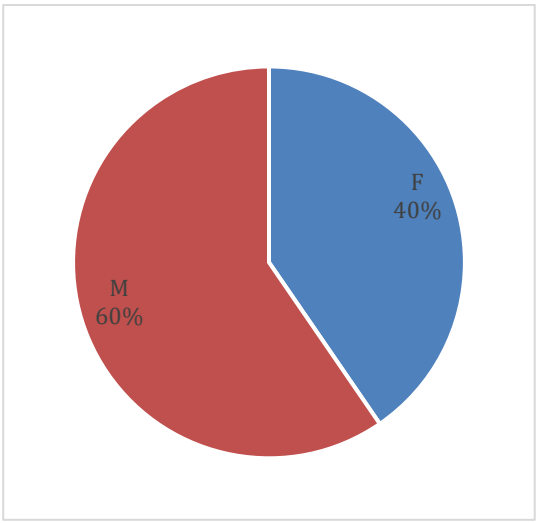

Figure 2 – Gender distribution of participants

Etiology of encephalopathy

The current study reports an encephalopathy concerning different etiology with the highest neurological cause (68 participants), followed by encephalopathy due to systemic disease (62 participants), and metabolic disorders (56 participants). In addition, toxin/drug induced encephalopathy was seen in 49 participants (20.9%).

Table 3 – Etiology of encephalopathy

ETIOLOGY OF ENCEPHALOPATHY	Frequency	Percent
Metabolic	56	23.8%
Neurological	68	28.9%
Systemic disease	62	26.4%
Toxins/drugs	49	20.9%
Total	235	100.0%

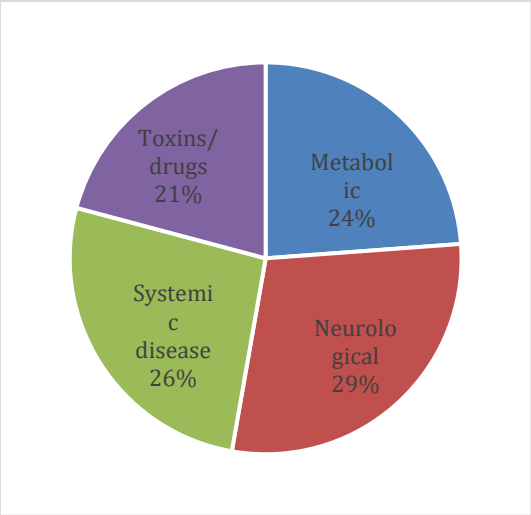

Figure 3 – Etiology for encephalopathy

Prevalence of the comorbid condition

Type 2 diabetes was seen among the majority of the individuals (65 participants), followed by hypertension (58 participants). Although, 58 participants presented with encephalopathy with no comorbid condition. In addition, epilepsy, DCLD, CAD, CKD, and COPD were seen among patients.

Table 4 – Comorbid conditions in encephalopathy

CO-MORBIDITIES	Frequency	Percent
Alcoholic liver disease	4	1.7%
Cancer	6	2.6%
COPD	10	4.3%
DCLD	20	8.5%
Epilepsy	14	6.0%
Hypertension	58	24.7%
Type 1 diabetes	6	2.6%
Type 2 diabetes	65	27.7%
CAD	11	4.7%
HfrEF	11	4.7%
CKD	17	7.2%
Nil	58	24.7%

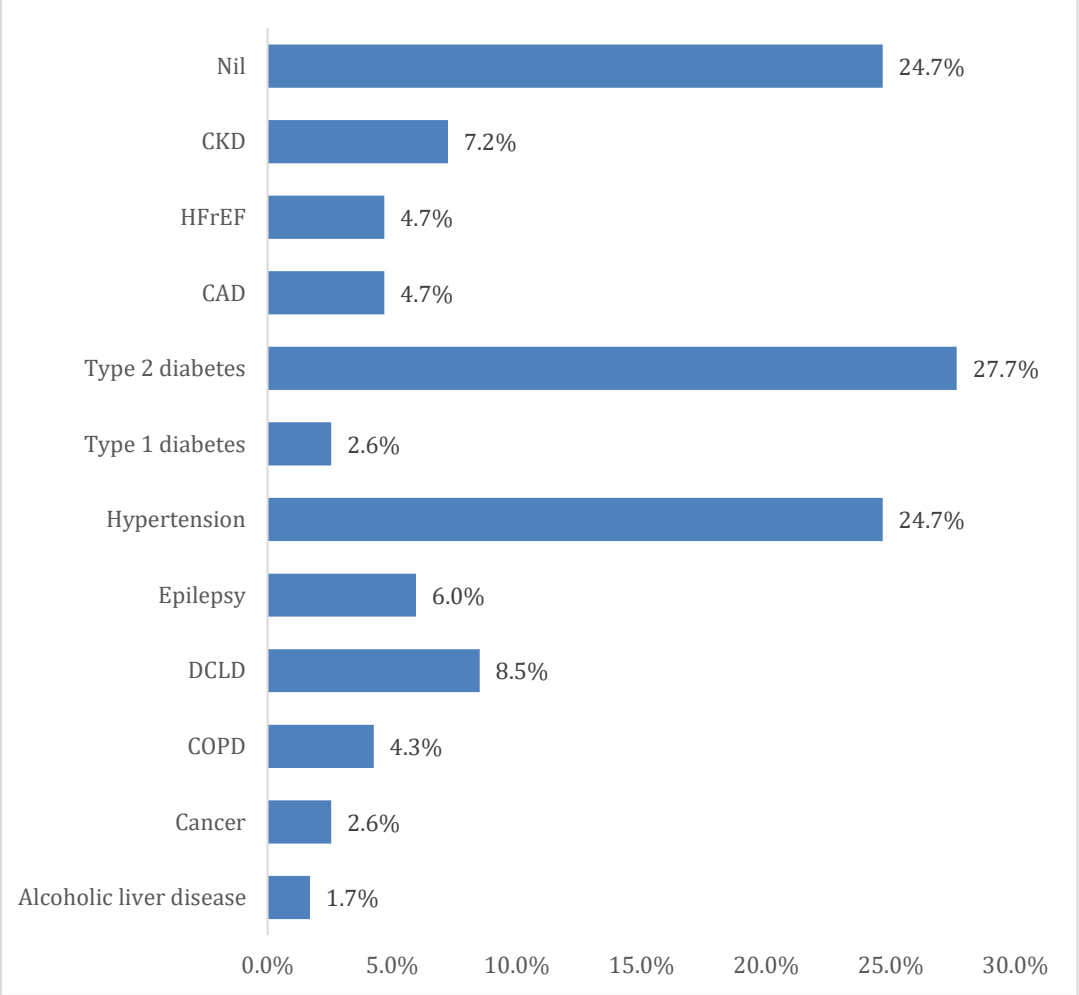

Figure 4 – Distribution of comorbid conditions

GCS Score

Out of 235 participants, 134 (57%) of the participants were presented with a GCS score of less than 10, and 101 (43%) participants shows a score above 10.

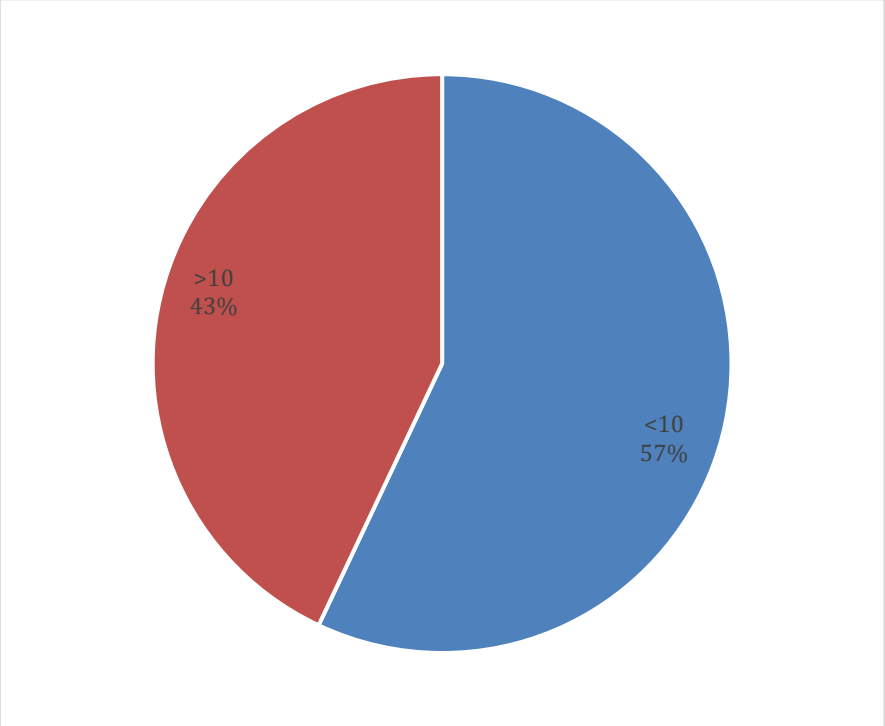

Figure 5 - GCS distribution

Cause for metabolic encephalopathy

The current study found diabetic ketoacidosis as the most common cause of metabolic encephalopathy (33.9%), followed by hypoglycemia (25%), and hyperosmolar hyperglycemic state (21.4%).

Table 5 – Cause of metabolic encephalopathy

Metabolic	Frequency	Percent
Diabetic Keto Acidosis	19	33.9%
Hyperosmolar Hyperglycemic State	12	21.4%
Hypoglycemia	14	25.0%
Hyponatremia	11	19.6%
Total	56	100.0%

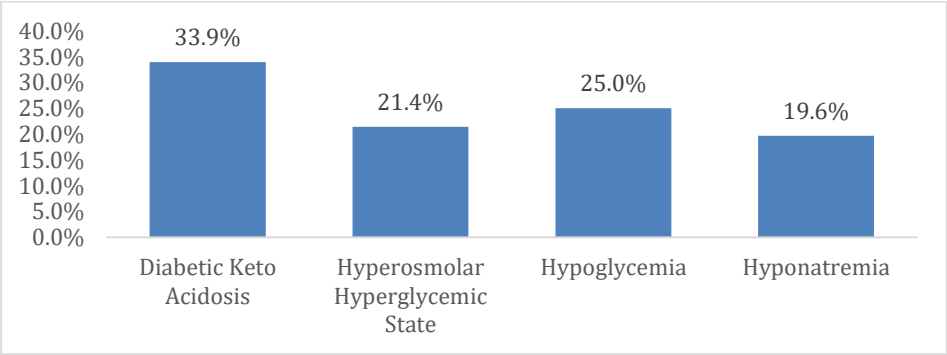

Figure 6 – Distribution of metabolic encephalopathy

Neurological Cause of Encephalopathy

Hemorrhagic stroke is one of the most common etiology reported in the current study including; 16 participants. The other cause comprises seizures (14 participants), ischemic stroke (13 participants), and cerebral venous thrombosis (9 participants). Less prevalent neurological causes include; brain metastasis and autoimmune encephalopathy.

Table 6 – Neuroglocial Causes of Encephalopathy

Neurological	Frequency	Percent
Autoimmune Encephalopathy	2	2.9%
Brain Metastasis	6	8.8%
Hemorrhagic Stroke	16	23.5%
Ischemic Stroke	13	19.1%
Seizure	14	20.6%
Seizure, Cerebral Venous Thrombosis	9	13.2%
Subarachnoid Hemorrhage	8	11.8%
Total	68	100.0%

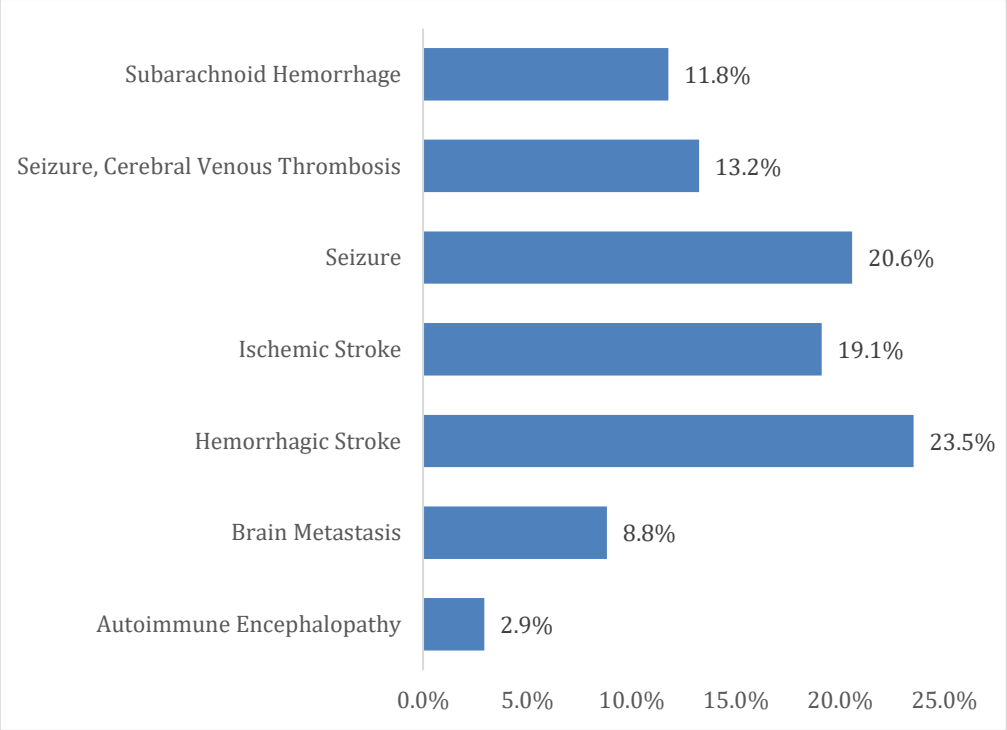

Figure 7 – Overall % distribution of neurological cause

Encephalopathy due to systemic disease

The study found that hepatic encephalopathy was the most prevalent cause of encephalopathy (33.9%), followed by uremic encephalopathy (29%), Cardiogenic shock (19.4%), and CO_2 narcosis (17.7%).

Table 7 – Systemic disease causing encephalopathy

Systemic disease	Frequency	Percent
Cardiogenic Shock	12	19.4%
Co2 Narcosis	11	17.7%
Hepatic Encephalopathy	21	33.9%
Uremic Encephalopathy	18	29.0%
Total	62	100.0%

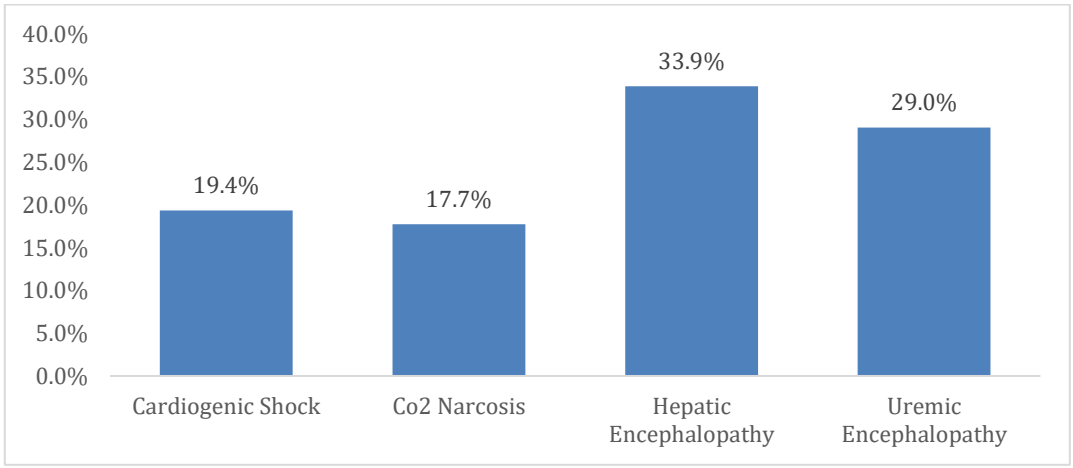

Figure 8 – % distribution of systemic disease

Toxin/drug-induced encephalopathy

Alcohol intoxication was predominant in the current study with 15 participants (30.6%), followed by atropine-induced encephalopathy in 13 participants (26.5%), and benzodiazepine poisoning in 9 participants (18.4%).

Table 8 – Toxin/drug induced encephalopathy

Toxins/drugs	Frequency	Percent
Alcohol Intoxication	15	30.6%
Atropine Induced	13	26.5%
Benzodiazepine Poisoning	9	18.4%
Cypermethrin Poisoning	5	10.2%
Nitrobenzene Poisoning	7	14.3%
Total	49	100.0%

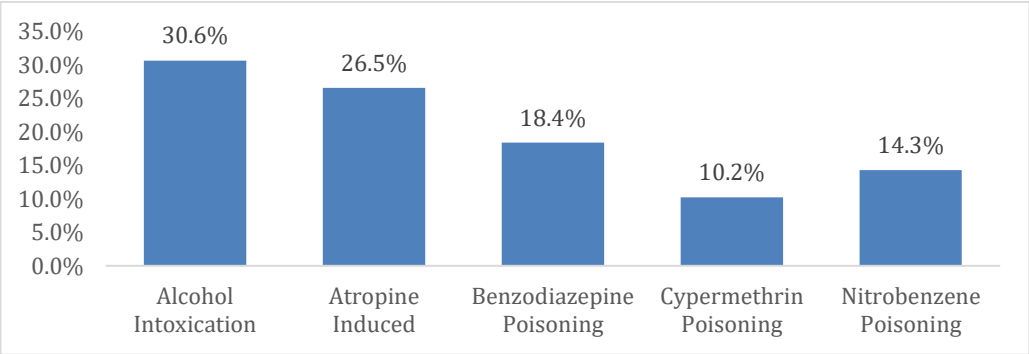

Figure 9 - % distribution of toxin-induced encephalopathy

Correlation of encephalopathy with age

The current study reports a significant difference in the etiology of encephalopathy in different age groups. The study found a significant difference between different age groups and the etiology of encephalopathy ($p < 0.0001$).

Table 9 – Correlation of age and causes of encephalopathy

			ETIOLOGY OF ENCEPHALOPATHY				Total	P value
			Metabolic	Neurological	Systemic disease	Toxins/drugs		
AGE GROUP	20 - 29	Count	7	9	0	11	27	<0.0001
		% within AGE GROUP	25.9%	33.3%	0.0%	40.7%	100.0%	
	30 - 39	Count	6	6	1	18	31	
		% within AGE GROUP	19.4%	19.4%	3.2%	58.1%	100.0%	
		Count	5	13	29	18	65	

	40 - 49	% within AGE GROUP	7.7%	20.0%	44.6%	27.7%	100.0%	
	50 - 59	Count	13	19	30	2	64	
		% within AGE GROUP	20.3%	29.7%	46.9%	3.1%	100.0%	
	60 - 69	Count	21	20	2	0	43	
		% within AGE GROUP	48.8%	46.5%	4.7%	0.0%	100.0%	
	>70	Count	4	1	0	0	5	
		% within AGE GROUP	80.0%	20.0%	0.0%	0.0%	100.0%	
Total		Count	56	68	62	49	235	
		% within AGE GROUP	23.8%	28.9%	26.4%	20.9%	100.0%	

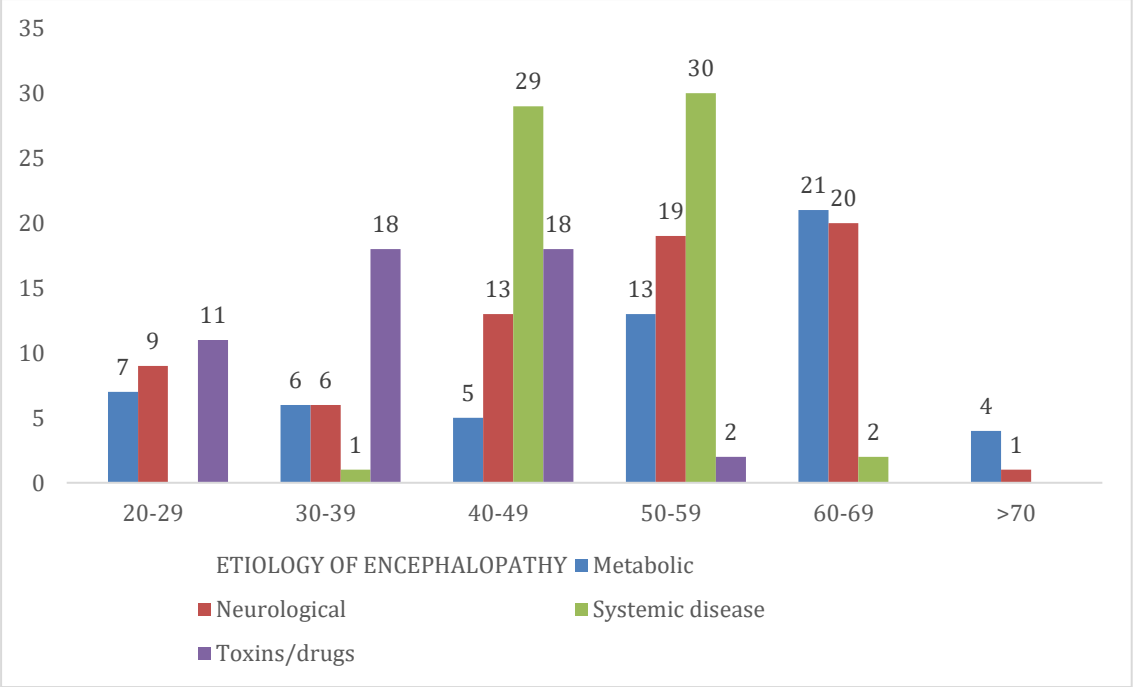

Figure 10 – Age distribution and etiology of encephalopathy

The given data has revealed that the age group of 40-49 and 50-59 years of age are more prone for encephalopathy; where the systemic disease is the predominant cause (29 participants in the age group of 40-49 years of age and 30 participants in the age group of 50-59 years). In addition, metabolic encephalopathy was seen higher in the age group of 60-69 years of age.

Gender distribution correlation with etiology

The current study did find a significant difference in the comparison of gender and etiology of encephalopathy (p = 0.044). However, the male gender was overall seen in higher numbers among various causes of encephalopathy.

Table 10 – Etiology of Encephalopathy

			ETIOLOGY OF ENCEPHALOPATHY				Total	P value
			Metabolic	Neurological	Systemic disease	Toxins/drugs		
SEX	F	Count	30	30	20	15	95	0.044
		% within SEX	31.6%	31.6%	21.1%	15.8%	100.0%	
	M	Count	26	38	42	34	140	
		% within SEX	18.6%	27.1%	30.0%	24.3%	100.0%	
Total		Count	56	68	62	49	235	
		% within SEX	23.8%	28.9%	26.4%	20.9%	100.0%	

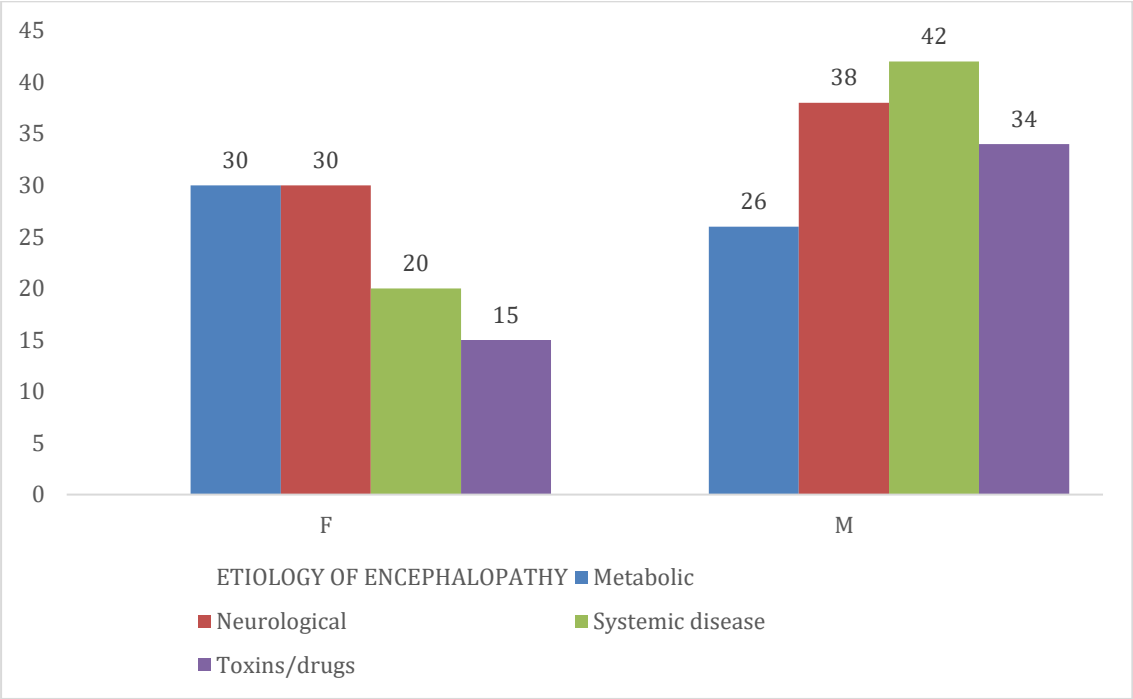

Figure 11 – Gender and etiology of encephalopathy

GCS Score and encephalopathy

The current study reports a significant difference in the GCS score due to various etiology of encephalopathy ($p < 0.0001$).

Table 11 – GCS score and etiology of encephalopathy

			ETIOLOGY OF ENCEPHALOPATHY				Total	P value
			Metabolic	Neurological	Systemic disease	Toxins/drugs		
GCS SCORE	<10	Count	42	30	42	20	134	<0.0001
		% within GCS SCORE	31.3%	22.4%	31.3%	14.9%	100.0%	
	>10	Count	14	38	20	29	101	
		% within GCS SCORE	13.9%	37.6%	19.8%	28.7%	100.0%	
Total		Count	56	68	62	49	235	
		% within GCS SCORE	23.8%	28.9%	26.4%	20.9%	100.0%	

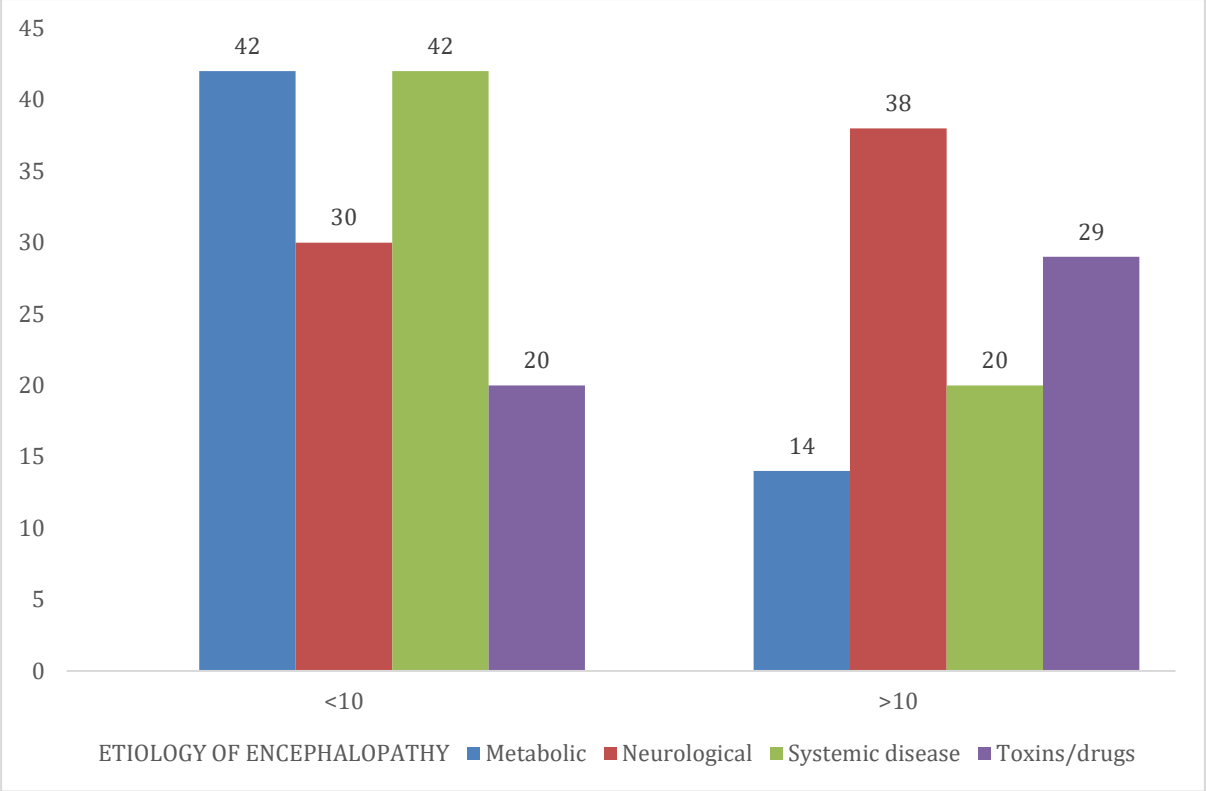

Figure 12 – GCS score and etiology

DISCUSSION

The current demographic and epidemiological data have suggested that encephalopathy has a significant correlation with age. Patients with age more than 60 years are at a high risk of developing encephalopathy. Whereas, in hospitalized patients, 10-40% of individuals are diagnosed with severe stages of encephalopathy.

The current study has reported the noteworthy cause of encephalopathy due to different pathogenesis which affects the overall clinical outcome in a patient. Our study has evaluated the correlation of encephalopathy concerning non-infectious causes which include; metabolic, neurological, systemic diseases and toxin/drug-induced encephalopathy in patients of different age groups.

The current study has reported that the prevalence of encephalopathy is more in the age group between 40-49 years of age and 50-59 years of age however, the study conducted by Belinda et.al. has reported that the age group most affected due to encephalopathy is the elderly group (60-75 years of age) with a mean age of 70.78 years. This difference can be due to the etiological nature of the disease. [50]

A male predominance was reported in the current study which was similar to the Belinda et.al findings which reported a higher prevalence of encephalopathy among 140 male participants out of 251. [50] In addition,

the study conducted by Nirhale et.al. also reported a high prevalence of males presented with encephalopathy. However, the current study did not find a significant correlation between gender and non-infectious cause of encephalopathy ($p = 0.044$). [51]

A comorbid condition such as type 2 diabetes was the most prevalent condition presented with encephalopathy (65 participants), followed by hypertension (58 participants), and DCLD (20 participants). However, 58 participants were found with no presence of comorbidity. The study by Belinda et.al also reported the found similar comorbid condition in patients presented with encephalopathy, out of which hypertension and diabetes were the most prevalent conditions. [50] [53]

The etiology behind encephalopathy was evaluated where we report that neurological cause is the most common reason for the development of encephalopathy (29.8%), followed by systemic disease (26.45%), metabolic disease (23.8%) and toxin/drug-induced (20.9%). Out of 235 patients, 68 patients were diagnosed with encephalopathy due to neurological disease. In neurological disease, hemorrhagic stroke (16 patients) was the most prevalent, followed by seizure-induced encephalopathy (14 patients). Similar findings were reported by Belinda et.al where the neurological cause was the most common cause for the development of encephalopathy (38.65% of patients), which comprised

cerebrovascular accident (58 patients) and ischemic stroke (42 patients). [50] In addition, the study conducted by Leong et.al. also reported causes of encephalopathy in which the neurological cause was the most common with 22.7%, followed by metabolic causes in 7.8% of the patients. [52]

Encephalopathy related to metabolic causes shows a high prevalence of diabetic ketoacidosis in the current study with clinical presentation in 19 patients, followed by the hyperosmolar hyperglycemic state in 12 patients, hypoglycemia in 14 patients, and hyponatremia in 11 patients. Similar findings were reported by the survey study conducted by Sathirapanya et.al, which reported hypoglycemia, hyperglycemia, and electrolyte imbalances as the most common cause of metabolic encephalopathy. [53] Frontera et.al. also reported a high prevalence of metabolic encephalopathy in 331 patients with hypoxic-ischemic encephalopathy and 156 patients with uremic encephalopathy. [54]

Among Toxin/drug induced encephalopathy, alcohol intoxication was seen to be the most prevalent cause of encephalopathy (30.6%), which was followed by atropine-induced encephalopathy (26.5%), and benzodiazepine poisoning (18.4%).

The current study reports patients with a GCS score < 10 in 134 patients which shows that encephalopathy is one of the morbid conditions. However, encephalopathy due to systemic disease and metabolic

encephalopathy were most common with low GCS scores (31.3%). The study reports a significant difference ($p < 0.001$) for low GCS scores and various etiology behind encephalopathy. This can be correlated to the findings of Belinda et.al, which reported that patients usually presented with low GCS scores due to a late diagnosis of encephalopathy with no adequate treatment plans. [50]

In the corresponding study, a GCS score between 3-8 was seen in 53 patients, and a score of 9-12 was seen in 41 patients. This signifies that encephalopathy if not detected and treated appropriately, can result in severe morbid conditions. [52]

The undiagnosed etiology of encephalopathy can result in an extended hospital stay with a high mortality rate especially among elderly patients. [56] The common causes of death due to encephalopathy are due to systemic, metabolic, neurologic, or infections. Study by Leong et.al reported neurological causes are the most significant cause of mortality among patients (68%). [52]. In addition, the study conducted by Sarin et.al. also reported high mortality rates among patients due to neurological (69.44%) and metabolic (35.29%) as the prevalent cause of encephalopathy. [55]

Hence the current study proposes that early detection of such encephalopathy based on its root cause can help in treating the disease more efficiently with better therapeutic outcomes.

SUMMARY

- The prevalence of encephalopathy was seen higher in the age group between 40-49 years of age (65 participants), followed by the age group of 50-59 years of age (64 participants).
- Male predominance was noted higher in the current study, with 140 participants and 95 females.
- Different etiologies of encephalopathy were identified with the highest numbers for neurological cause (68 participants), followed by encephalopathy due to systemic disease (62 participants), and metabolic disorders (56 participants).
- Type 2 diabetes was seen among the majority of the individuals (65 participants), followed by hypertension (58 participants).
- Diabetic ketoacidosis is the most common cause of metabolic encephalopathy (33.9%), followed by hypoglycemia (25%), and hyperosmolar hyperglycemic state (21.4%).
- Hepatic encephalopathy was the most prevalent cause of encephalopathy (33.9%), followed by uremic encephalopathy (29%), Cardiogenic shock (19.4%), and CO_2 necrosis (17.7%).
- Alcohol intoxication was predominant in the current study with 15 participants (30.6%), followed by atropine-induced encephalopathy

in 13 participants (26.5%), and benzodiazepine poisoning in 9 participants (18.4%).

- The study found a significant difference between different age groups and the etiology of encephalopathy ($p < 0.0001$).
- Metabolic encephalopathy was seen higher in the age range of 60-69 years of age.
- The current study did find a significant difference in the comparison of gender and etiology of encephalopathy ($p = 0.044$).
- The current study reports a significant difference in the GCS score due to various etiology of encephalopathy ($p < 0.0001$).

CONCLUSION

The present study concludes, that encephalopathy is a multifactorial disease with different etiological reasoning for each type of encephalopathy. The severity and morbidity remain high in every type of encephalopathy.

The study found that the age group at risk of developing encephalopathy are between the age of 40-59 years, male gender, and with the presence of comorbid conditions such as diabetes and hypertension.

The study reports a high prevalence of encephalopathy caused by systemic diseases among participants above 40 years of age which includes 29 patients presented with systemic cause of encephalopathy in the age group of 40-49 years of age and 30 patients were evident with systemic cause of encephalopathy in the age group of 50-59 years of age respectively. The most cause of encephalopathy in this 40-59 years age group was due to systemic disease, followed by neurological cause, and metabolic. The toxin/drug-induced encephalopathy was seen in the younger age group due to drug abuse or alcohol intoxication.

The prognostic role of the GCS score can be beneficial in identifying the severity of the disease. The current study shows that the majority of patients (134 participants) got admitted with GCS <10 irrespective of the cause of encephalopathy.

Hence it is essential to identify the root cause of encephalopathy to prevent morbid complications, reduce the risk of organ damage, and provide optimal therapeutic outcomes.

LIMITATIONS

- It is a single centred study conducted in a tertiary care centre.
- Comparatively small sample size of the study limits it's generalizability.
- Study excluded trauma patients which makes it susceptible to selection bias.

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PROFORMA

Name :
Age :
Sex :
Occupation :
Address :
IP no :
DOA :

Co-morbidities :

On admission

GCS : Eye opening -
Verbal response -
Motor response -

Vitals : BP -
PR -
RR -
SpO2 -
Temperature -

Investigations : Total count(cells/mm3) -
Hemoglobin(g/dl) -
PCV -
MCV(fl) -
Platelet count(lakh cells/mm3) -

RBS(mg/dl) -
Urea(mg/dl) -
Creatinine(mg/dl) -
Sodium(mEq/l) -
Potassium (mEq/l) -
Magnesium(mg/dl) -

Total bilirubin(mg/dl) -
Direct bilirubin(mg/dl) -
Indirect bilirubin(mg/dl) -
SGOT(iu/l) -
SGPT(iu/l) -
Total protein(g/dl) -
Serum albumin(g/dl) -

Serum globulin(g/dl) -
Serum ammonia(mcg/dl) -
PT(sec) -
APTT(sec) -
INR -

ABG -
Urine analysis -
Ascitic fluid analysis -
CSF analysis -
Blood culture -
Urine culture -

Chest X ray -
USG abdomen -
Echocardiography -
CT chest -
CT brain -
MRI brain -
EEG -

CONSENT FORM

Format for Informed Consent Form for Subjects/ Guardian of the Subjects

Informed Consent form to participate in a research study

Study Title:

Study Number:

Subject's Initials:

Subject's Name:

Date of Birth/ Age:

(i) I confirm that I have read and understood the information sheet dated …… for the above study and have had the opportunity to ask questions. [ ]
(ii) I understand that my father / mother / husband / wife / son / daughter's participation in the study is voluntary and that he/she is free to withdraw at any time, without giving any reason, without his/her medical care or legal rights being affected. [ ]
(iii) I understand that the Ethics Committee and the regulatory authorities will not need my permission to look at my father / mother / husband / wife / son / daughter's health records both in respect of the current study and any further research that may be conducted in relation to it, even if he/she withdraws from the trial. I agree to this access. However, I understand that my father / mother / husband / wife / son / daughter's identity will not be revealed in any information released to third parties or published. [ ]
(iv) I agree not to restrict the use of any data or results that arise from this study provided such a use is only for scientific purpose(s). [ ]
(v) I agree for the participation of my father / mother / husband / wife / son / daughter in the above study. [ ]

Signature (or Thumb impression) of the Subject/Legally Acceptable Guardian:
Date:
Signatory's Name:
Signature:
Relationship with subject:
Address:

Signature and name of investigator:
Signature or thumb impression of the Witness:
Date:
Name & Address of the Witness:

நோயாளிகளுக்கு அறிவிப்பு மற்றும் ஒப்புதல் படிவம்
(மருத்துவ ஆய்வில் பங்கேற்பதற்கு)

ஆய்வு செய்யப்படும் தலைப்பு:
பங்கு பெறுவரின் பெயர்:
பங்கு பெறுவரின் வயது:

		பங்கு பெறுவர் இதனை ✓ குறிக்கவும்
1.	நான் மேலே குறிப்பிட்டுள்ள மருத்துவ ஆய்வின் விவரங்களை படித்து புரிந்து கொண்டேன். என்னுடைய சந்தேகங்களை கேட்கவும், அதற்கான தகுந்த விளக்கங்களை பெறவும் வாய்ப்பளிக்கப்பட்டுள்ளது என அறிந்து கொண்டேன்.	☐
2.	நான் இவ்வாய்வில் தன்னிச்சையாக தான் பங்கேற்கிறேன். எந்த காரணத்தினாலோ எந்த கட்டத்திலும், எந்த சட்ட சிக்கலுக்கும் உட்படாமல் நான் இவ்வாய்வில் இருந்து விலகி கொள்ளலாம் என்றும் அறிந்து கொண்டேன்.	☐
3.	இந்த ஆய்வு சம்பந்தமாகவோ, இதை சார்ந்து மேலும் ஆய்வு மேற்காள்ளும் போதும் இந்த ஆய்வில் பங்குபெறும் மருத்துவர் என்னுடைய மருத்துவ அறிக்கைகளை பார்ப்பதற்கு என் அனுமதி தேவையில்லை என அறிந்து கொள்கிறேன். நான் ஆய்வில் இருந்து விலகிக் கொண்டாலும் இது பொருந்தும் என அறிகிறேன்.	☐
4.	இந்த ஆய்வின் மூலம் கிடைக்கும் தகவலையோ, முடிவையோ பயன்படுத்திக் கொள்ள மறுக்க மாட்டேன்.	☐
5.	இந்த ஆய்வில் பங்கு கொள்ள ஒப்புக் கொள்கிறேன் எனக்கு கொடுக்கப்பட்டஅறிவுரைகளின் படி நடந்து கொள்வதுடன், ஆய்வை மேற்கொள்ளும் மருத்துவ அணிக்கு உண்மையுடன் இருப்பேன் என்று உறுதியளிக்கிறேன். என் உடல் நலம் பாதிக்கப்பட்டாலோ, அல்லது எதிர்பாராத, வழக்கத்திற்கு மாறான நோய்குறி தென்பட்டாலோ உடனே இதை மருத்துவ அணியிடம் தெரிவிப்பேன் என உறுதி அளிக்றேன்.	☐

பங்கேற்பவரின் கையொப்பம் / ..... இடம் .....
கட்டைவிரல் ரேகை
பங்கேற்பவரின் பெயர் மற்றும் விலாசம் .....
ஆய்வாளரின் கையொப்பம் /..... இடம் .....
ஆய்வாளரின் பெயர் .....
மையம் .....
கல்வியறிவு இல்லாதவற்கு (கைரேகை வைத்தவர்களுக்கு) இது அவசியம் தேவை
சாட்சியின் கையொப்பம் /..... இடம் .....
பெயர் மற்றும் விலாசம் ....

Sno	AGE(years)	SEX	ETIOLOGY OF ENCEPHALOPATHY	DIAGNOSIS	O-MORBIDITIE	SBP	DBP	PR	RR	SpO2(% in roo	TEMPERATURE	GCS(on admiss	GCS SCORE	TC(cells/mm3)	Hb(g/dl)	PCV	MCV(fl)	PLATELETS(lal	RBS(mg/dl)	UREA(mg/dl)	CREATININE(r	SODIUM(mEq/l	POTASSIUM(m	MAGNESIUM(r	TOTAL BILIRU	DIRECT	INDIRECT	SGOT(IU/L)	SGPT(IU/L)	TOTAL PROTE	ALBUMIN	GLOBULIN	PT(sec)	APTT(sec)	INR	SERUM AMMONIA(mc	ABG	URINE ANALYSIS	BLOOD CULTURE	URINE CULTURE	CSF ANALYSIS	ASCITIC FLUID ANALYSIS	CHEST X RAY	CT CHEST	USG ABDOMEN	ECHO	CT BRAIN	MRI BRAIN	EEG
1	58	M	metabolic	hypoglycemia	hypertension	140	90	112	18	96	37	E2V2M4	8	10400	12	0.4	86	2.4	54	28	0.9	141	3.8	1.8	0.9	0.3	0.6	32	38	6.4	3.7	2.1					N	N					N				N		
2	61	F	metabolic	hyperosmolar hyperglycemic state	type 2 diabetes	130	70	92	16	98	37	E2V2M5	9	11000	11	0.4	84	2.1	586	31	0.8	138	3.9	1.9	0.9	0.3	0.6	34	37	6.2	3.5	2.1	12	36	0.9		N	glycosuria 2+		no growth			N		N		N		
3	40	F	metabolic	diabetic keto acidosis	type 2 diabetes	130	80	82	28	98	37	E3V4M4	11	9800	12	0.4	88	2.2	432	30	0.8	142	3.7	2	0.8	0.2	0.6	33	32	6.8	3.9	2.2	12	34	1.1		metabolic acidosis	ketone +					N		N		N		
4	67	M	metabolic	hyperosmolar hyperglycemic state	hypertension	130	90	72	16	99	37	E2V2M4	8	7300	13	0.4	91	2.9	612	32	0.8	137	4.1	1.9	0.8	0.2	0.6	32	38	6.7	3.8	2.2					N	glycosuria 2+		no growth			N		N		N		
5	56	F	metabolic	hypoglycemia	-	130	80	116	18	96	38	E2V2M4	8	10800	11	0.4	82	1.9	57	38	1.1	140	4.1	1.7	0.9	0.3	0.6	37	38	5.9	3.5	2.1	13	35	1.1		N	N		no growth			N				N		
6	28	M	metabolic	diabetic keto acidosis	type 2 diabetes	120	80	92	32	97	38	E3V4M4	11	7800	13	0.4	84	2.4	396	32	0.9	139	3.9	1.9	0.8	0.2	0.6	34	36	7.1	4.2	2.3					metabolic acidosis	ketone +		no growth			N				N		
7	46	M	neurological	autoimmune encephalopathy	-	130	80	78	14	99	37	E3V4M5	12	8200	13	0.4	87	2.8	134	31	0.8	142	3.7	1.9	0.8	0.2	0.6	33	31	6.9	3.9	2.3					N	N	no growth	no growth	anti VGKC Ab+		N	N	N		N	T2 hyperintensity in mesial	
8	51	F	neurological	subarachnoid hemorrhage	hypertension	160	90	76	15	96	37	E3V4M5	12	9200	12	0.4	88	2.6	132	34	0.9	140	3.9	1.8	0.9	0.3	0.6	36	31	6.6	3.7	2.1	11	36	1		N	N					N		N	EF 43%, LVH	perimesencephalic subarachnoid		
9	37	F	neurological	seizure, cerebral venous thrombosis	-	130	70	72	14	98	37	E3V4M4	11	8700	12	0.4	84	2.5	119	32	0.8	142	3.8	1.9	0.8	0.2	0.6	38	41	6.4	3.7	2.1	12	35	0.9		N	N					N		N		superior saggital sinus thrombosis	superior saggital sinus thrombosis	N
10	64	M	neurological	ischemic stroke	type 2 diabetes	180	100	72	16	96	38	E2V3M4	9	10700	12	0.4	85	2.2	225	38	0.9	141	3.9	1.8	0.9	0.3	0.6	35	38	6.2	3.5	2.1	13	36	1.1		N	glycosuria 1+		no growth			N		N		right mca infarct	right mca infarct	
11	61	M	neurological	hemorrhagic stroke	hypertension	160	100	70	14	97	38	E2V2M4	8	11200	12	0.4	87	2.4	121	32	1.1	143	3.8	1.7	0.9	0.3	0.6	39	42	6.6	3.7	2.2	14	35	1		N	N		no growth			N		N	EF 41%	pontine hemorrhage	pontine hemorrhage	
12	24	F	neurological	seizure	epilepsy	120	80	82	16	96	38	E3V4M4	11	8600	14	0.4	85	2.6	135	31	0.7	140	4.1	2.1	0.8	0.2	0.6	32	35	6.9	4.2	2.1					N	N					N				diffuse cerebral edema	N	epileptiform spikes in parietal lobe
13	67	M	neurological	brain metastasis	lung cancer, hypertension	140	90	88	14	97	37	E2V3M4	9	10700	11	0.4	84	2.1	138	36	1.1	139	4.1	1.8	0.9	0.3	0.6	35	39	6.1	3.6	2.2	14	35	1		N	N	no growth	no growth			right upper lobe	right upper lobe	N	EF 41%	left frontal lobe metastasis	left frontal lobe metastasis	
14	54	M	neurological	subarachnoid hemorrhage	hypertension	160	100	74	16	96	38	E3V4M4	11	8300	12	0.4	87	2.5	152	38	1.1	142	3.8	1.7	0.8	0.2	0.6	33	38	6.2	3.8	2.1	12	37	0.9		N	N					N			EF 45%, LVH	perimesencephalic subarachnoid		
15	26	F	metabolic	diabetic keto acidosis	type 1 diabetes	120	70	92	32	97	37	E2V2M4	8	11000	13	0.4	88	2.6	386	32	0.9	140	3.7	1.9	0.9	0.3	0.6	36	38	6.9	3.9	2.2					metabolic acidosis	ketone +	no growth	no growth			N		N		N		
16	71	M	metabolic	hyponatremia	hypertension	130	90	82	16	96	37	E2V2M4	8	9400	12	0.4	89	2.4	124	39	1.2	122	3.5	1.8	0.9	0.3	0.6	38	34	6.1	3.6	2.2					low sodium	N		no growth			N		N		age related cortical atrophy		
17	59	F	metabolic	hypoglycemia	-	140	80	106	18	99	37	E2V4M4	10	7600	13	0.4	87	2.5	51	36	0.9	139	3.9	1.7	0.8	0.2	0.6	35	36	6.5	3.8	2.1					N	N					N				N		
18	61	M	metabolic	diabetic keto acidosis	type 2 diabetes	120	80	88	16	97	37	E2V3M4	9	8500	12	0.4	83	2.6	590	41	1.2	137	3.8	1.8	0.9	0.3	0.6	39	35	6.4	3.7	2.1					metabolic acidosis	glycosuria 2+		no growth			N		N		N		
19	47	M	metabolic	hyponatremia	alcoholic liver disease	110	80	82	14	96	37	E2V3M4	9	7600	12	0.4	91	2.5	112	39	1.2	126	3.7	1.7	4.1	2	2.1	176	104	6.1	3.6	2.2	16	42	1.5	58	low sodium	N				high SAAG>1.1	N		parenchymal liver disease		N		
20	35	F	metabolic	diabetic keto acidosis	type 2 diabetes	120	80	88	28	98	38	E3V4M5	12	9600	13	0.4	88	2.8	406	38	1.2	139	3.8	1.9	1	0.4	0.6	36	39	6.6	3.7	2.3					metabolic acidosis	ketone +	no growth	no growth			N		N		N		
21	65	M	metabolic	hypoglycemia	-	110	70	92	16	97	37	E2V2M4	8	10200	13	0.4	84	2.4	54	34	0.8	142	4	2.1	0.8	0.2	0.6	34	35	6.4	3.6	2.2					N	N		no growth			N				N		
22	58	F	metabolic	diabetic keto acidosis	type 2 diabetes	130	80	86	22	98	37	E3V4M5	12	6900	13	0.4	87	2.6	611	42	1.3	136	3.8	1.8	0.9	0.3	0.6	37	32	6.5	3.8	2.1					metabolic acidosis	glycosuria 2+		no growth			N		N		N		
23	47	M	systemic disease	cardiogenic shock	CAD, HFrEF, type 2 diabetes	90	60	108	18	93	38	E2V2M4	8	9200	13	0.4	85	2.4	216	48	1.4	137	4.1	1.7	3.9	1.9	2	52	68	6.4	3.6	2.2	12	37	1.1		N	glycosuria 1+					cardiomegaly		congestive hepatopat	EF 28%, global hypokine			
24	57	M	systemic disease	CO2 narcosis	COPD	140	80	104	22	91	37	E2V3M4	9	11200	14	0.5	84	2.6	183	38	1.1	141	3.9	1.9	1.1	0.4	0.7	39	35	6.8	3.9	2.3					respiratory acidosis	N					emphysema	emphysema		EF 42%, moderate PHTN			
25	45	M	systemic disease	hepatic encephalopathy	DCLD	110	70	86	16	96	37	E2V2M4	8	9800	12	0.4	85	2.3	124	42	1.2	136	3.8	1.7	8.2	4	4.2	152	98	6	3.5	2.2	18	46	1.9	149	N	N	no growth	no growth		high SAAG>1.1	N		liver cirrhosis		N		
26	51	F	systemic disease	uremic encephalopathy	CKD, type 2 diabetes	160	90	82	14	97	37	E2V4M5	11	7400	11	0.3	81	2.1	175	236	7.9	138	5.5	2.2	0.9	0.2	0.7	38	36	6.1	3.4	2.2	14	37	1.2		N	protein 3+		no growth			N		contracted kidneys		N		
27	55	M	systemic disease	CO2 narcosis	COPD, hypertension	150	90	112	24	92	37	E2V4M4	10	8500	15	0.5	87	2.7	142	39	1.1	141	3.9	1.9	0.8	0.2	0.6	36	39	6.7	3.8	2.3					respiratory acidosis	N					emphysema	emphysema		EF 45%, moderate PHTN			
28	48	F	systemic disease	cardiogenic shock	CAD, HFrEF, type 2 diabetes	90	60	116	20	94	37	E3V4M5	12	6400	13	0.4	84	2.6	198	41	1.3	138	3.8	1.7	3.8	1.7	2.1	72	76	6.5	3.7	2.2	13	41	1.1		N	glycosuria 1+					cardiomegaly		congestive hepatopat	EF 29%, global hypokine			
29	46	M	systemic disease	hepatic encephalopathy	DCLD	100	70	88	16	97	37	E2V3M4	9	7200	11	0.4	86	2.5	135	38	1.2	137	3.9	1.8	9.6	4.4	5.2	162	112	6.1	3.5	2.3	19	48	2.1	156	N	N	no growth	no growth		high SAAG>1.1	N		liver cirrhosis		N		

Sno	AGE(years)	SEX	ETIOLOGY OF ENCEPHALOPATHY	DIAGNOSIS	O-MORBIDITIE	SBP	DBP	PR	RR	SpO2(% in roo	TEMPERATURE	GCS(on admiss	GCS SCORE	TC(cells/mm3)	Hb(g/dl)	PCV	MCV(fl)	PLATELETS(lal	RBS(mg/dl)	UREA(mg/dl)	CREATININE(r	SODIUM(mEq/l	POTASSIUM(m	MAGNESIUM(r	TOTAL BILIRU	DIRECT	INDIRECT	SGOT(IU/L)	SGPT(IU/L)	TOTAL PROTE	ALBUMIN	GLOBULIN	PT(sec)	APTT(sec)	INR	SERUM AMMONIA(mc	ABG	URINE ANALYSIS	BLOOD CULTURE	URINE CULTURE	CSF ANALYSIS	ASCITIC FLUID ANALYSIS	CHEST X RAY	CT CHEST	USG ABDOMEN	ECHO	CT BRAIN	MRI BRAIN	EEG
30	55	F	systemic disease	uremic encephalopathy	CKD, type 2 diabetes	150	90	84	18	96	38	E2V3M4	9	10400	10	0.3	84	2.4	210	284	7.1	138	5.6	2.1	1.1	0.3	0.8	41	46	6.2	3.6	2.3	15	38	1.3		N	protein 3+		no growth			N		contracted kidneys		N		
31	43	M	systemic disease	uremic encephalopathy	CKD, type 2 diabetes	160	90	86	16	94	38	E3V4M5	12	11200	10	0.4	82	2.6	268	235	6.9	141	5.1	1.9	0.9	0.3	0.6	68	52	6.8	3.9	2.3						N	protein 2+		no growth			N		contracted kidneys		N		
32	48	M	systemic disease	hepatic encephalopathy	DCLD	120	70	78	14	98	37	E2V4M4	10	6500	13	0.4	85	2.7	146	34	0.9	140	3.9	1.8	7.4	4	3.4	168	159	6.9	4.1	2.3	16	39	1.9	169	N	N	no growth	no growth		high SAAG>1.1	N		liver cirrhosis		N		
33	37	M	systemic disease	hepatic encephalopathy	DCLD	130	80	92	24	87	37	E3V4M5	12	8800	14	0.4	87	2.9	129	35	0.8	139	4.1	1.9	8.3	4.2	4.1	182	136	6.6	3.9	2.2	18	42	2.2	152	N	N	no growth	no growth		high SAAG>1.1	N		liver cirrhosis		N		
34	43	M	systemic disease	uremic encephalopathy	CKD, type 2 diabetes	160	90	102	16	98	38	E3V4M5	12	9200	13	0.4	86	2.6	188	237	7.4	140	3.9	1.8	1.1	0.3	0.8	35	36	7.1	4.2	2.3					N	protein 2+		no growth			N		contracted kidneys		N		
35	28	F	toxins/drugs	cypermethrin poisoning	-	110	80	98	14	99	37	E3V4M4	11	8900	13	0.4	84	2.3	158	32	0.8	138	3.7	2.1	0.8	0.2	0.6	37	34	6.8	3.9	2.3					N	N					N				N		
36	42	F	toxins/drugs	benzodiazepine poisoning	type 2 diabetes	120	80	82	14	98	37	E2V4M4	10	8100	13	0.4	87	2.4	206	33	0.9	139	3.8	1.9	0.9	0.2	0.7	35	38	6.5	3.8	2.2					N	N					N						
37	45	M	toxins/drugs	nitrobenzene poisoning	hypertension	140	90	88	22	89	37	E3V4M4	11	9600	14	0.4	85	2.8	176	36	0.8	142	3.9	2.1	0.8	0.2	0.6	32	39	6.9	4.1	2.2					metabolic acidosis	N					N						
38	31	M	toxins/drugs	alcohol intoxication	-	130	80	82	16	99	38	E2V3M4	9	8800	14	0.4	92	2.7	112	38	0.9	140	3.8	1.9	1.2	0.4	0.8	58	67	6.8	3.9	2.3	12	37	1.1		N	N					N		N		N		
39	39	F	toxins/drugs	cypermethrin poisoning	-	120	70	78	14	98	37	E3V4M5	12	10300	13	0.4	87	2.4	138	35	0.8	139	4.1	2.1	0.8	0.2	0.6	36	35	6.7	3.8	2.3					N	N					N				N		
40	48	M	toxins/drugs	atropine induced	hypertension	140	80	118	16	98	38	E3V3M4	10	9500	13	0.4	89	2.6	143	41	1.1	141	3.9	1.8	0.9	0.3	0.6	38	36	6.6	3.9	2.2					N	N					N						
41	58	M	neurological	hemorrhagic stroke	hypertension	180	110	74	18	96	37	E2V2M4	8	10400	12	0.4	86	2.4	168	28	0.9	141	3.8	1.8	0.9	0.3	0.6	32	38	6.4	3.7	2.1	13	37	1.1		N	N		no growth			N		N	EF 50%, LVH	right capsuloganglionic bleed		
42	61	F	neurological	ischemic stroke	hypertension	170	100	76	16	98	37	E2V2M5	9	11000	11	0.4	84	2.1	143	31	0.8	140	3.9	1.9	0.9	0.3	0.6	34	37	6.2	3.5	2.1	12	36	0.9		N	N		no growth			N		N	EF 48%	left mca infarct	left mca infarct	
43	40	F	neurological	seizure, cerebral venous thrombosis	-	130	80	82	16	98	37	E3V4M4	11	9800	12	0.4	88	2.2	132	30	0.8	142	3.7	2	0.8	0.2	0.6	33	32	6.8	3.9	2.2	12	34	1.1		N	N					N		N		right transverse sinus	right transverse sinus	N
44	41	M	neurological	autoimmune encephalopathy	-	130	70	72	16	99	37	E3V4M5	12	7300	13	0.4	91	2.9	145	32	0.8	141	4.1	1.9	0.8	0.2	0.6	32	38	6.7	3.8	2.2					N	N	no growth	no growth	anti NMDA Ab+		N	N	N		N	N	
45	56	F	neurological	brain metastasis	breast cancer	130	80	80	14	96	38	E2V2M4	8	10800	11	0.4	82	1.9	151	38	1.1	140	4.1	1.7	0.9	0.3	0.6	37	38	5.9	3.5	2.1	13	35	1.1		N	N	no growth	no growth			lung metastasis	lung metastasis	N	EF 42%	right parietal lobe metastasis	right parietal lobe metastasis	
46	28	M	neurological	seizure	epilepsy	120	80	88	16	97	38	E3V4M4	11	7800	13	0.4	84	2.4	163	32	0.9	139	3.9	1.9	0.8	0.2	0.6	34	36	7.1	4.2	2.3					N	N					N				diffuse cerebral edema	N	epileptiform spikes in frontal lobe
47	49	M	neurological	seizure	epilepsy	130	80	78	14	99	37	E3V4M5	12	8200	13	0.4	87	2.8	134	31	0.8	142	3.7	1.9	0.8	0.2	0.6	33	31	6.9	3.9	2.3					N	N	no growth	no growth			N		N		diffuse cerebral edema	N	epileptiform spikes in temporal lobe
48	51	F	neurological	subarachnoid hemorrhage	hypertension	160	90	76	15	96	37	E3V4M5	12	9200	12	0.4	88	2.6	132	34	0.9	140	3.9	1.8	0.9	0.3	0.6	36	31	6.6	3.7	2.1	11	36	1		N	N					N		N	EF 43%, LVH	perimesencephalic subarachnoid		
49	37	F	neurological	seizure, cerebral venous thrombosis	-	130	70	72	14	98	37	E3V4M4	11	8700	12	0.4	84	2.5	119	32	0.8	142	3.8	1.9	0.8	0.2	0.6	38	41	6.4	3.7	2.1	12	35	0.9		N	N					N		N		superior saggital sinus thrombosis	superior saggital sinus thrombosis	N
50	64	M	neurological	ischemic stroke	type 2 diabetes	180	100	72	16	96	38	E2V3M4	9	10700	12	0.4	85	2.2	225	38	0.9	141	3.9	1.8	0.9	0.3	0.6	35	38	6.2	3.5	2.1	13	36	1.1		N	glycosuria 1+		no growth			N		N		right mca infarct	right mca infarct	
51	61	M	neurological	hemorrhagic stroke	hypertension	160	100	70	14	97	38	E2V2M4	8	11200	12	0.4	87	2.4	121	32	1.1	143	3.8	1.7	0.9	0.3	0.6	39	42	6.6	3.7	2.2	14	35	1		N	N		no growth			N		N	EF 41%	pontine hemorrhage	pontine hemorrhage	
52	24	F	neurological	seizure	epilepsy	120	80	82	16	96	38	E3V4M4	11	8600	14	0.4	85	2.6	135	31	0.7	140	4.1	2.1	0.8	0.2	0.6	32	35	6.9	4.2	2.1					N	N					N				diffuse cerebral edema	N	epileptiform spikes in parietal lobe
53	67	M	neurological	brain metastasis	lung cancer, hypertension	140	90	88	14	97	37	E2V3M4	9	10700	11	0.4	84	2.1	138	36	1.1	139	4.1	1.8	0.9	0.3	0.6	35	39	6.1	3.6	2.2	14	35	1		N	N	no growth	no growth			right upper lobe	right upper lobe	N	EF 41%	left frontal lobe metastasis	left frontal lobe metastasis	
54	54	M	neurological	subarachnoid hemorrhage	hypertension	160	100	74	16	96	38	E3V4M4	11	8300	12	0.4	87	2.5	152	38	1.1	142	3.8	1.7	0.8	0.2	0.6	33	38	6.2	3.8	2.1	12	37	0.9		N	N					N			EF 45%, LVH	perimesencephalic subarachnoid		
55	26	F	metabolic	diabetic keto acidosis	type 1 diabetes	120	70	92	32	97	37	E2V2M4	8	11000	13	0.4	88	2.6	386	32	0.9	140	3.7	1.9	0.9	0.3	0.6	36	38	6.9	3.9	2.2					metabolic acidosis	ketone +	no growth	no growth			N		N		N		
56	71	M	metabolic	hyponatremia	hypertension	130	90	82	16	96	37	E2V2M4	8	9400	12	0.4	89	2.4	124	39	1.2	122	3.5	1.8	0.9	0.3	0.6	38	34	6.1	3.6	2.2					low sodium	N		no growth			N		N		age related cortical atrophy		
57	59	F	metabolic	hypoglycemia	-	140	80	96	15	99	37	E2V4M4	10	7600	13	0.4	87	2.5	51	36	0.9	139	3.9	1.7	0.8	0.2	0.6	35	36	6.5	3.8	2.1					N	N					N				N		
58	62	M	metabolic	diabetic keto acidosis	type 2 diabetes	120	80	88	16	97	37	E2V3M4	9	8500	12	0.4	83	2.6	590	41	1.2	137	3.8	1.8	0.9	0.3	0.6	39	35	6.4	3.7	2.1					metabolic acidosis	glycosuria 2+		no growth			N		N		N		

Sno	AGE(years)	SEX	ETIOLOGY OF ENCEPHALOPATHY	DIAGNOSIS	CO-MORBIDITIE	SBP	DBP	PR	RR	SpO2(% in roo	TEMPERATURE	GCS(on admiss	GCS SCORE	TC(cells/mm3)	Hb(g/dl)	PCV	MCV(fl)	PLATELETS(lal	RBS(mg/dl)	UREA(mg/dl)	CREATININE(r	SODIUM(mEq/l	POTASSIUM(m	MAGNESIUM(r	TOTAL BILIRU	DIRECT	INDIRECT	SGOT(IU/L)	SGPT(IU/L)	TOTAL PROTE	ALBUMIN	GLOBULIN	PT(sec)	APTT(sec)	INR	SERUM AMMONIA(mc	ABG	URINE ANALYSIS	BLOOD CULTURE	URINE CULTURE	CSF ANALYSIS	ASCITIC FLUID ANALYSIS	CHEST X RAY	CT CHEST	USG ABDOMEN	ECHO	CT BRAIN	MRI BRAIN	EEG
59	43	M	metabolic	hyponatremia	alcoholic liver disease	110	80	82	14	96	37	E2V3M4	9	7600	12	0.4	91	2.5	112	39	1.2	126	3.7	1.7	4.1	2	2.1	176	104	6.1	3.6	2.2	16	42	1.5	58	low sodium	N				high SAAG>1.1	N		parenchymal liver disease		N		
60	35	F	metabolic	diabetic keto acidosis	type 2 diabetes	120	80	88	28	98	38	E3V4M5	12	9600	13	0.4	88	2.8	406	38	1.2	139	3.8	1.9	1	0.4	0.6	36	39	6.6	3.7	2.3					metabolic acidosis	ketone +	no growth	no growth			N		N		N		
61	65	M	metabolic	hypoglycemia	-	110	70	92	16	97	37	E2V2M4	8	10200	13	0.4	84	2.4	54	34	0.8	142	4	2.1	0.8	0.2	0.6	34	35	6.4	3.6	2.2					N	N		no growth			N				N		
62	58	F	metabolic	diabetic keto acidosis	type 2 diabetes	130	80	86	14	98	37	E3V4M5	12	6900	13	0.4	87	2.6	611	42	1.3	136	3.8	1.8	0.9	0.3	0.6	37	32	6.5	3.8	2.1					metabolic acidosis	ketone +		no growth			N		N		N		
63	47	M	systemic disease	cardiogenic shock	CAD, HFrEF, type 2 diabetes	90	60	108	18	93	38	E2V2M4	8	9200	13	0.4	85	2.4	216	48	1.4	137	4.1	1.7	3.9	1.9	2	52	68	6.4	3.6	2.2	12	37	1.1		N	glycosuria 1+					cardiomegaly		congestive hepatopat	EF 28%, global hypokine			
64	57	M	systemic disease	CO2 narcosis	COPD	140	80	104	22	91	37	E2V3M4	9	11200	14	0.5	84	2.6	183	38	1.1	141	3.9	1.9	1.1	0.4	0.7	39	35	6.8	3.9	2.3					respiratory acidosis	N					emphysema	emphysema		EF 42%, moderate PHTN			
65	45	M	systemic disease	hepatic encephalopathy	DCLD	110	70	86	16	96	37	E2V2M4	8	9800	12	0.4	85	2.3	124	42	1.2	136	3.8	1.7	8.2	4	4.2	152	98	6	3.5	2.2	18	46	1.9	149	N	N	no growth	no growth		high SAAG>1.1	N		liver cirrhosis		N		
66	51	F	systemic disease	uremic encephalopathy	CKD, type 2 diabetes	160	90	82	14	97	37	E2V4M5	11	7400	11	0.3	81	2.1	175	186	6.8	138	4.3	2.2	0.9	0.2	0.7	38	36	6.1	3.4	2.2	14	37	1.2		N	protein 3+		no growth			N		contracted kidneys		N		
67	55	M	systemic disease	CO2 narcosis	COPD, hypertension	150	90	112	24	92	37	E2V4M4	10	8500	15	0.5	87	2.7	142	39	1.1	141	3.9	1.9	0.8	0.2	0.6	36	39	6.7	3.8	2.3					respiratory acidosis	N					emphysema	emphysema		EF 45%, moderate PHTN			
68	48	F	systemic disease	cardiogenic shock	CAD, HFrEF, type 2 diabetes	90	60	116	20	94	37	E3V4M5	12	6400	13	0.4	84	2.6	198	41	1.3	138	3.8	1.7	3.8	1.7	2.1	72	76	6.5	3.7	2.2	13	41	1.1		N	glycosuria 1+					cardiomegaly		congestive hepatopat	EF 29%, global hypokine			
69	49	M	systemic disease	hepatic encephalopathy	DCLD	110	70	88	16	97	37	E2V3M4	9	7200	11	0.4	86	2.5	135	38	1.2	137	3.9	1.8	9.6	4.4	5.2	162	112	6.1	3.5	2.3	19	48	2.1	156	N	N	no growth	no growth		high SAAG>1.1	N		liver cirrhosis		N		
70	55	F	systemic disease	uremic encephalopathy	CKD, type 2 diabetes	150	90	84	18	96	38	E2V3M4	9	10400	10	0.3	84	2.4	210	192	7.1	138	4.2	2.1	1.1	0.3	0.8	41	46	6.2	3.6	2.3	15	38	1.3		N	protein 3+		no growth			N		contracted kidneys		N		
71	27	M	toxins/drugs	alcohol intoxication	-	130	80	112	16	99	38	E3V4M5	12	11200	13	0.4	89	2.6	128	32	0.8	141	4.1	1.9	0.9	0.3	0.6	68	52	6.8	3.9	2.3	11	36	1.1		N	N					N		N		N		
72	33	F	toxins/drugs	benzodiazepine poisoning	-	120	70	78	14	98	37	E2V4M4	10	6500	13	0.4	85	2.7	146	34	0.9	140	3.9	1.8	0.8	0.2	0.6	36	32	6.9	4.1	2.3					N	N					N				N		
73	38	M	toxins/drugs	nitrobenzene poisoning	-	130	80	92	24	87	37	E3V4M5	12	8800	14	0.4	87	2.9	129	35	0.8	139	4.1	1.9	0.9	0.3	0.6	39	38	6.6	3.9	2.2					metabolic acidosis	N					N						
74	43	M	toxins/drugs	atropine induced	-	120	70	124	16	98	38	E3V4M5	12	9200	13	0.4	86	2.6	134	37	0.9	140	3.9	1.8	1.1	0.3	0.8	35	36	7.1	4.2	2.3					N	N					N				N		
75	28	F	toxins/drugs	cypermethrin poisoning	-	110	80	98	14	99	37	E3V4M4	11	8900	13	0.4	84	2.3	158	32	0.8	138	3.7	2.1	0.8	0.2	0.6	37	34	6.8	3.9	2.3					N	N					N				N		
76	42	F	toxins/drugs	benzodiazepine poisoning	type 2 diabetes	120	80	82	14	98	37	E2V4M4	10	8100	13	0.4	87	2.4	206	33	0.9	139	3.8	1.9	0.9	0.2	0.7	35	38	6.5	3.8	2.2					N	N					N						
77	50	M	toxins/drugs	nitrobenzene poisoning	hypertension	140	90	88	22	89	37	E3V4M4	11	9600	14	0.4	85	2.8	176	36	0.8	142	3.9	2.1	0.8	0.2	0.6	32	39	6.9	4.1	2.2					metabolic acidosis	N					N						
78	31	M	toxins/drugs	alcohol intoxication	-	130	80	82	16	99	38	E2V3M4	9	8800	14	0.4	92	2.7	112	38	0.9	140	3.8	1.9	1.2	0.4	0.8	58	67	6.8	3.9	2.3	12	37	1.1		N	N					N		N		N		
79	39	F	toxins/drugs	cypermethrin poisoning	-	120	70	78	14	98	37	E3V4M5	12	10300	13	0.4	87	2.4	138	35	0.8	139	4.1	2.1	0.8	0.2	0.6	36	35	6.7	3.8	2.3					N	N					N				N		
80	48	M	toxins/drugs	atropine induced	hypertension	140	80	118	16	98	38	E3V3M4	10	9500	13	0.4	89	2.6	143	41	1.1	141	3.9	1.8	0.9	0.3	0.6	38	36	6.6	3.9	2.2					N	N					N						
81	58	M	neurological	hemorrhagic stroke	hypertension	180	110	74	18	96	37	E2V2M4	8	10400	12	0.4	86	2.4	168	28	0.9	141	3.8	1.8	0.9	0.3	0.6	32	38	6.4	3.7	2.1	13	37	1.1		N	N		no growth			N		N	EF 50%, LVH	right capsuloganglionic bleed		
82	62	F	neurological	ischemic stroke	hypertension	170	100	76	16	98	37	E2V2M5	9	11000	11	0.4	84	2.1	143	31	0.8	140	3.9	1.9	0.9	0.3	0.6	34	37	6.2	3.5	2.1	12	36	0.9		N	N		no growth			N		N	EF 48%	left mca infarct	left mca infarct	
83	40	F	neurological	seizure, cerebral venous thrombosis	-	130	80	82	16	98	37	E3V4M4	11	9800	12	0.4	88	2.2	132	30	0.8	142	3.7	2	0.8	0.2	0.6	33	32	6.8	3.9	2.2	12	34	1.1		N	N					N		N		right transverse sinus	right transverse sinus	N
84	41	M	neurological	seizure	epilepsy	130	70	72	16	99	37	E3V4M5	12	7300	13	0.4	91	2.9	145	32	0.8	141	4.1	1.9	0.8	0.2	0.6	32	38	6.7	3.8	2.2					N	N	no growth	no growth			N	N	N		N	N	epileptiform spikes in parietal lobe
85	56	F	neurological	brain metastasis	breast cancer	130	80	80	14	96	38	E2V2M4	8	10800	11	0.4	82	1.9	151	38	1.1	140	4.1	1.7	0.9	0.3	0.6	37	38	5.9	3.5	2.1	13	35	1.1		N	N	no growth	no growth			lung metastasis	lung metastasis	N	EF 42%	right parietal lobe metastasis	right parietal lobe metastasis	
86	28	M	neurological	seizure	epilepsy	120	80	88	16	97	38	E3V4M4	11	7800	13	0.4	84	2.4	163	32	0.9	139	3.9	1.9	0.8	0.2	0.6	34	36	7.1	4.2	2.3					N	N					N				diffuse cerebral edema	N	epileptiform spikes in frontal lobe
87	49	M	neurological	seizure	epilepsy	130	80	78	14	99	37	E3V4M5	12	8200	13	0.4	87	2.8	134	31	0.8	142	3.7	1.9	0.8	0.2	0.6	33	31	6.9	3.9	2.3					N	N	no growth	no growth			N	N	N		N	N	N

Sno	AGE(years)	SEX	ETIOLOGY OF ENCEPHALOPATHY	DIAGNOSIS	CO-MORBIDITIE	SBP	DBP	PR	RR	SpO2(% in roo	TEMPERATURE	GCS(on admiss	GCS SCORE	TC(cells/mm3)	Hb(g/dl)	PCV	MCV(fl)	PLATELETS(lal	RBS(mg/dl)	UREA(mg/dl)	CREATININE(r	SODIUM(mEq/l	POTASSIUM(m	MAGNESIUM(r	TOTAL BILIRU	DIRECT	INDIRECT	SGOT(IU/L)	SGPT(IU/L)	TOTAL PROTE	ALBUMIN	GLOBULIN	PT(sec)	APTT(sec)	INR	SERUM AMMONIA(mc	ABG	URINE ANALYSIS	BLOOD CULTURE	URINE CULTURE	CSF ANALYSIS	ASCITIC FLUID ANALYSIS	CHEST X RAY	CT CHEST	USG ABDOMEN	ECHO	CT BRAIN	MRI BRAIN	EEG
88	52	F	neurological	subarachnoid hemorrhage	hypertension	160	90	76	15	96	37	E3V4M5	12	9200	12	0.4	88	2.6	132	34	0.9	140	3.9	1.8	0.9	0.3	0.6	36	31	6.6	3.7	2.1	11	36	1		N	N					N		N	EF 43%, LVH	perimesencephalic subarachnoid		
89	38	F	neurological	seizure, cerebral venous thrombosis	-	130	70	72	14	98	37	E3V4M4	11	8700	12	0.4	84	2.5	119	32	0.8	142	3.8	1.9	0.8	0.2	0.6	38	41	6.4	3.7	2.1	12	35	0.9		N	N					N		N		superior saggital sinus thrombosis	superior saggital sinus thrombosis	N
90	64	M	neurological	ischemic stroke	type 2 diabetes	180	100	72	16	96	38	E2V3M4	9	10700	12	0.4	85	2.2	225	38	0.9	141	3.9	1.8	0.9	0.3	0.6	35	38	6.2	3.5	2.1	13	36	1.1		N	glycosuria 1+		no growth			N		N		right mca infarct	right mca infarct	
91	62	M	neurological	hemorrhagic stroke	hypertension	160	100	70	14	97	38	E2V2M4	8	11200	12	0.4	87	2.4	121	32	1.1	143	3.8	1.7	0.9	0.3	0.6	39	42	6.6	3.7	2.2	14	35	1		N	N		no growth			N		N	EF 41%	pontine hemorrhage		
92	24	F	neurological	seizure	epilepsy	120	80	82	16	96	38	E3V4M4	11	8600	14	0.4	85	2.6	135	31	0.7	140	4.1	2.1	0.8	0.2	0.6	32	35	6.9	4.2	2.1					N	N					N				diffuse cerebral edema	N	epileptiform spikes in parietal lobe
93	68	M	neurological	brain metastasis	lung cancer, hypertension	140	90	88	14	97	37	E2V3M4	9	10700	11	0.4	84	2.1	138	36	1.1	139	4.1	1.8	0.9	0.3	0.6	35	39	6.1	3.6	2.2	14	35	1		N	N	no growth	no growth			right upper lobe	right upper lobe	N	EF 41%	left frontal lobe metastasis	left frontal lobe metastasis	
94	54	M	neurological	subarachnoid hemorrhage	hypertension	160	100	74	16	96	38	E3V4M4	11	8300	12	0.4	87	2.5	152	38	1.1	142	3.8	1.7	0.8	0.2	0.6	33	38	6.2	3.8	2.1	12	37	0.9		N	N					N			EF 45%, LVH	perimesencephalic subarachnoid		
95	26	F	metabolic	diabetic keto acidosis	type 1 diabetes	120	70	92	32	97	37	E2V2M4	8	11000	13	0.4	88	2.6	386	32	0.9	140	3.7	1.9	0.9	0.3	0.6	36	38	6.9	3.9	2.2					metabolic acidosis	ketone +	no growth	no growth			N		N		N		
96	69	M	metabolic	hyponatremia	hypertension	140	90	82	16	96	37	E2V2M4	8	9400	12	0.4	89	2.4	124	39	1.2	122	3.5	1.8	0.9	0.3	0.6	38	34	6.1	3.6	2.2					low sodium	N		no growth			N		N		age related cortical atrophy		
97	59	F	metabolic	hypoglycemia	-	130	80	96	15	99	37	E2V4M4	10	7600	13	0.4	87	2.5	56	36	0.9	139	3.9	1.7	0.8	0.2	0.6	35	36	6.5	3.8	2.1					N	N					N				N		
98	62	M	metabolic	diabetic keto acidosis	type 2 diabetes	120	80	88	16	97	37	E2V3M4	9	8500	12	0.4	83	2.6	590	41	1.2	137	3.8	1.8	0.9	0.3	0.6	39	35	6.4	3.7	2.1					metabolic acidosis	glycosuria 2+		no growth			N		N		N		
99	65	M	metabolic	hyponatremia	-	110	80	82	14	96	37	E2V3M4	9	7600	12	0.4	91	2.5	112	39	1.2	126	3.7	1.7	1.1	0.3	0.8	37	41	6.1	3.6	2.2	14	37	1.1		low sodium	N		no growth			N		N		N		
100	36	F	metabolic	diabetic keto acidosis	type 2 diabetes	120	80	88	28	98	38	E3V4M5	12	9600	13	0.4	88	2.8	406	38	1.2	139	3.8	1.9	1	0.4	0.6	36	39	6.6	3.7	2.3					metabolic acidosis	ketone +	no growth	no growth			N		N		N		
101	66	M	metabolic	hypoglycemia	-	110	70	92	16	97	37	E2V2M4	8	10200	13	0.4	84	2.4	54	34	0.8	142	4	2.1	0.8	0.2	0.6	34	35	6.4	3.6	2.2					N	N		no growth			N				N		
102	58	F	metabolic	hyperosmolar hyperglycemic state	type 2 diabetes	130	80	86	14	98	37	E3V4M5	12	6900	13	0.4	87	2.6	611	42	1.3	136	3.8	1.8	0.9	0.3	0.6	37	32	6.5	3.8	2.1					N	glycosuria 2+		no growth			N		N		N		
103	47	M	systemic disease	cardiogenic shock	CAD, HFrEF, type 2 diabetes	90	60	108	18	93	38	E2V2M4	8	9200	13	0.4	85	2.4	216	48	1.4	137	4.1	1.7	3.9	1.9	2	52	68	6.4	3.6	2.2	12	37	1.1		N	N					cardiomegaly		congestive hepatopat	EF 28%, global hypokine		
104	57	M	systemic disease	CO2 narcosis	COPD	140	80	104	22	91	37	E2V3M4	9	11200	14	0.5	84	2.6	183	38	1.1	141	3.9	1.9	1.1	0.4	0.7	39	35	6.8	3.9	2.3					respiratory acidosis	N					emphysema	emphysema		EF 42%, moderate PHTN		
105	44	M	systemic disease	hepatic encephalopathy	DCLD	110	70	86	16	96	37	E2V2M4	8	9800	12	0.4	85	2.3	124	42	1.2	136	3.8	1.7	8.2	4	4.2	152	98	6	3.5	2.2	18	46	1.9	149	N	N	no growth	no growth		high SAAG>1.1	N		liver cirrhosis		N		
106	52	F	systemic disease	uremic encephalopathy	CKD, type 2 diabetes	160	90	82	14	97	37	E2V4M5	11	7400	11	0.3	81	2.1	175	186	6.8	138	4.3	2.2	0.9	0.2	0.7	38	36	6.1	3.4	2.2	14	37	1.2		N	protein 3+		no growth			N		contracted kidneys		N		
107	55	M	systemic disease	CO2 narcosis	COPD, hypertension	150	90	112	24	92	37	E2V4M4	10	8500	15	0.5	87	2.7	142	39	1.1	141	3.9	1.9	0.8	0.2	0.6	36	39	6.7	3.8	2.3					respiratory acidosis	N					emphysema	emphysema		EF 45%, moderate PHTN		
108	48	F	systemic disease	cardiogenic shock	CAD, HFrEF, type 2 diabetes	90	60	116	20	94	37	E3V4M5	12	6400	13	0.4	84	2.6	198	41	1.3	138	3.8	1.7	3.8	1.7	2.1	72	76	6.5	3.7	2.2	13	41	1.1		N	glycosuria 1+					cardiomegaly		congestive hepatopat	EF 29%, global hypokine		
109	49	M	systemic disease	hepatic encephalopathy	DCLD	110	70	88	16	97	37	E2V3M4	9	7200	11	0.4	86	2.5	135	38	1.2	137	3.9	1.8	9.6	4.4	5.2	162	112	6.1	3.5	2.3	19	48	2.1	156	N	N	no growth	no growth		high SAAG>1.1	N		liver cirrhosis		N		
110	55	F	systemic disease	uremic encephalopathy	CKD, type 2 diabetes	150	90	84	18	96	38	E2V3M4	9	10400	10	0.3	84	2.4	210	192	7.1	138	4.2	2.1	1.1	0.3	0.8	41	46	6.2	3.6	2.3	15	38	1.3		N	protein 3+		no growth			N		contracted kidneys		N		
111	27	M	toxins/drugs	alcohol intoxication	-	130	80	86	16	99	38	E3V4M5	12	11200	13	0.4	89	2.6	128	32	0.8	141	4.1	1.9	0.9	0.3	0.6	68	52	6.8	3.9	2.3	11	36	1.1		N	N					N		N		N		
112	33	F	toxins/drugs	benzodiazepine poisoning	-	120	70	78	14	98	37	E2V4M4	10	6500	13	0.4	85	2.7	146	34	0.9	140	3.9	1.8	0.8	0.2	0.6	36	32	6.9	4.1	2.3					N	N					N						
113	38	M	toxins/drugs	nitrobenzene poisoning	-	130	80	92	24	87	37	E3V4M5	12	8800	14	0.4	87	2.9	129	35	0.8	139	4.1	1.9	0.9	0.3	0.6	39	38	6.6	3.9	2.2					metabolic acidosis	N					N						
114	43	M	toxins/drugs	atropine induced	-	120	70	124	16	98	38	E3V4M5	12	9200	13	0.4	86	2.6	134	37	0.9	140	3.9	1.8	1.1	0.3	0.8	35	36	7.1	4.2	2.3					N	N					N						
115	28	F	toxins/drugs	cypermethrin poisoning	-	110	80	98	14	99	37	E3V4M4	11	8900	13	0.4	84	2.3	158	32	0.8	138	3.7	2.1	0.8	0.2	0.6	37	34	6.8	3.9	2.3					N	N					N						
116	42	F	toxins/drugs	benzodiazepine poisoning	type 2 diabetes	120	80	82	14	98	37	E2V4M4	10	8100	13	0.4	87	2.4	206	33	0.9	139	3.8	1.9	0.9	0.2	0.7	35	38	6.5	3.8	2.2					N	N					N						

Sno	AGE(years)	SEX	ETIOLOGY OF ENCEPHALOPATHY	DIAGNOSIS	CO-MORBIDITIE	SBP	DBP	PR	RR	SpO2(% in roo	TEMPERATURE	GCS(on admiss	GCS SCORE	TC(cells/mm3)	Hb(g/dl)	PCV	MCV(fl)	PLATELETS(lal	RBS(mg/dl)	UREA(mg/dl)	CREATININE(r	SODIUM(mEq/l	POTASSIUM(m	MAGNESIUM(r	TOTAL BILIRU	DIRECT	INDIRECT	SGOT(IU/L)	SGPT(IU/L)	TOTAL PROTE	ALBUMIN	GLOBULIN	PT(sec)	APTT(sec)	INR	SERUM AMMONIA(mc	ABG	URINE ANALYSIS	BLOOD CULTURE	URINE CULTURE	CSF ANALYSIS	ASCITIC FLUID ANALYSIS	CHEST X RAY	CT CHEST	USG ABDOMEN	ECHO	CT BRAIN	MRI BRAIN	EEG
117	50	M	toxins/drugs	nitrobenzene poisoning	hypertension	140	90	88	22	89	37	E3V4M4	11	9600	14	0.4	85	2.8	176	36	0.8	142	3.9	2.1	0.8	0.2	0.6	32	39	6.9	4.1	2.2					metabolic acidosis	N						N							
118	31	M	toxins/drugs	alcohol intoxication	-	130	80	82	16	99	38	E2V3M4	9	8800	14	0.4	92	2.7	112	38	0.9	140	3.8	1.9	1.2	0.4	0.8	58	67	6.8	3.9	2.3	12	37	1.1		N	N						N		N		N			
119	39	M	toxins/drugs	alcohol intoxication	-	120	70	78	14	98	37	E3V4M5	12	10300	13	0.4	87	2.4	138	35	0.8	139	4.1	2.1	0.8	0.2	0.6	36	35	6.7	3.8	2.3					N	N						N							
120	48	M	toxins/drugs	atropine induced	hypertension	140	80	118	16	98	38	E3V3M4	10	9500	13	0.4	89	2.6	143	41	1.1	141	3.9	1.8	0.9	0.3	0.6	38	36	6.6	3.9	2.2					N	N						N							
121	58	M	neurological	hemorrhagic stroke	hypertension	180	110	74	18	96	37	E2V2M4	8	10400	12	0.4	86	2.4	168	28	0.9	141	3.8	1.8	0.9	0.3	0.6	32	38	6.4	3.7	2.1	13	37	1.1		N	N		no growth				N		N	EF 50%, LVH	right capsuloganglionic bleed		
122	62	F	neurological	ischemic stroke	hypertension	170	100	76	16	98	37	E2V2M5	9	11000	11	0.4	84	2.1	143	31	0.8	140	3.9	1.9	0.9	0.3	0.6	34	37	6.2	3.5	2.1	12	36	0.9		N	N		no growth				N		N	EF 48%	left mca infarct	left mca infarct	
123	40	F	neurological	seizure, cerebral venous thrombosis	-	130	80	82	16	98	37	E3V4M4	11	9800	12	0.4	88	2.2	132	30	0.8	142	3.7	2	0.8	0.2	0.6	33	32	6.8	3.9	2.2	12	34	1.1		N	N						N		N		right transverse sinus	right transverse sinus	N
124	41	M	neurological	seizure	epilepsy	130	70	72	16	99	37	E3V4M5	12	7300	13	0.4	91	2.9	145	32	0.8	141	4.1	1.9	0.8	0.2	0.6	32	38	6.7	3.8	2.2					N	N	no growth	no growth			N	N	N		N	N	N
125	61	F	neurological	brain metastasis	breast cancer	130	80	80	14	96	38	E2V2M4	8	10800	11	0.4	82	1.9	151	38	1.1	140	4.1	1.7	0.9	0.3	0.6	37	38	5.9	3.5	2.1	13	35	1.1		N	N	no growth	no growth			lung metastasis	lung metastasis	N	EF 42%	right parietal lobe metastasis	right parietal lobe metastasis	
126	28	M	neurological	seizure	epilepsy	120	80	88	16	97	38	E3V4M4	11	7800	13	0.4	84	2.4	163	32	0.9	139	3.9	1.9	0.8	0.2	0.6	34	36	7.1	4.2	2.3					N	N					N				diffuse cerebral edema	N	epileptiform spikes in frontal lobe
127	32	F	neurological	seizure	epilepsy	130	80	78	14	99	37	E3V4M5	12	8200	13	0.4	87	2.8	134	31	0.8	142	3.7	1.9	0.8	0.2	0.6	33	31	6.9	3.9	2.3					N	N	no growth	no growth			N	N	N		N	N	N
128	52	F	neurological	subarachnoid hemorrhage	hypertension	160	90	76	15	96	37	E3V4M5	12	9200	12	0.4	88	2.6	132	34	0.9	140	3.9	1.8	0.9	0.3	0.6	36	31	6.6	3.7	2.1	11	36	1		N	N						N		N	EF 43%, LVH	perimesencephalic subarachnoid		
129	38	F	neurological	seizure, cerebral venous thrombosis	-	130	70	72	14	98	37	E3V4M4	11	8700	12	0.4	84	2.5	119	32	0.8	142	3.8	1.9	0.8	0.2	0.6	38	41	6.4	3.7	2.1	12	35	0.9		N	N						N		N		superior saggital sinus thrombosis	superior saggital sinus thrombosis	N
130	64	M	neurological	ischemic stroke	type 2 diabetes	180	100	72	16	96	38	E2V3M4	9	10700	12	0.4	85	2.2	225	38	0.9	141	3.9	1.8	0.9	0.3	0.6	35	38	6.2	3.5	2.1	13	36	1.1		N	glycosuria 1+		no growth				N		N		right mca infarct	right mca infarct	
131	62	M	neurological	hemorrhagic stroke	hypertension	160	100	70	14	97	38	E2V2M4	8	11200	12	0.4	87	2.4	121	32	1.1	143	3.8	1.7	0.9	0.3	0.6	39	42	6.6	3.7	2.2	14	35	1		N	N		no growth				N		N	EF 41%	pontine hemorrhage	pontine hemorrhage	
132	24	F	neurological	seizure	epilepsy	120	80	82	16	96	38	E3V4M4	11	8600	14	0.4	85	2.6	135	31	0.7	140	4.1	2.1	0.8	0.2	0.6	32	35	6.9	4.2	2.1					N	N						N				diffuse cerebral edema	N	epileptiform spikes in parietal lobe
133	68	M	neurological	ischemic stroke	type 2 diabetes	140	90	88	14	97	37	E2V3M4	9	10700	11	0.4	84	2.1	138	36	1.1	139	4.1	1.8	0.9	0.3	0.6	35	39	6.1	3.6	2.2	14	35	1		N	N	no growth	no growth			right upper lobe	right upper lobe	N	EF 41%	left frontal lobe metastasis	left frontal lobe metastasis	
134	54	M	neurological	subarachnoid hemorrhage	hypertension	160	100	74	16	96	38	E3V4M4	11	8300	12	0.4	87	2.5	152	38	1.1	142	3.8	1.7	0.8	0.2	0.6	33	38	6.2	3.8	2.1	12	37	0.9		N	N						N			EF 45%, LVH	perimesencephalic subarachnoid		
135	26	F	metabolic	diabetic keto acidosis	type 1 diabetes	120	70	92	32	97	37	E2V2M4	8	11000	13	0.4	88	2.6	386	32	0.9	140	3.7	1.9	0.9	0.3	0.6	36	38	6.9	3.9	2.2					metabolic acidosis	ketone +	no growth	no growth				N		N		N		
136	69	M	metabolic	hyponatremia	hypertension	130	90	82	16	96	37	E2V2M4	8	9400	12	0.4	89	2.4	124	39	1.2	124	3.5	1.8	0.9	0.3	0.6	38	34	6.1	3.6	2.2					low sodium	N		no growth				N		N		age related cortical atrophy		
137	60	F	metabolic	hypoglycemia	-	140	80	96	15	99	37	E2V4M4	10	7600	13	0.4	87	2.5	51	36	0.9	139	3.9	1.7	0.8	0.2	0.6	35	36	6.5	3.8	2.1					N	N						N				N		
138	59	M	metabolic	hyperosmolar hyperglycemic state	type 2 diabetes	120	80	88	16	97	37	E2V3M4	9	8500	12	0.4	83	2.6	585	41	1.2	137	3.8	1.8	0.9	0.3	0.6	39	35	6.4	3.7	2.1					N	glycosuria 2+		no growth				N		N		N		
139	44	M	metabolic	hyponatremia	alcoholic liver disease	110	80	82	14	96	37	E2V3M4	9	7600	12	0.4	91	2.5	112	39	1.2	126	3.7	1.7	4.1	2	2.1	176	104	6.1	3.6	2.2	16	42	1.5	53	low sodium	N				high SAAG>1.1	N		parenchymal liver disease		N		
140	36	F	metabolic	diabetic keto acidosis	type 2 diabetes	120	80	88	28	98	38	E3V4M5	12	9600	13	0.4	88	2.8	406	38	1.2	139	3.8	1.9	1	0.4	0.6	36	39	6.6	3.7	2.3					metabolic acidosis	ketone +	no growth	no growth				N		N		N		
141	66	M	metabolic	hypoglycemia	-	110	70	92	16	97	37	E2V2M4	8	10200	13	0.4	84	2.4	54	34	0.8	142	4	2.1	0.8	0.2	0.6	34	35	6.4	3.6	2.2					N	N		no growth				N				N		
142	58	F	metabolic	hyperosmolar hyperglycemic state	type 2 diabetes	130	80	86	14	98	37	E3V4M5	12	6900	13	0.4	87	2.6	611	42	1.3	136	3.8	1.8	0.9	0.3	0.6	37	32	6.5	3.8	2.1					N	glycosuria 2+		no growth				N		N		N		
143	47	M	systemic disease	cardiogenic shock	CAD, HFrEF, type 2 diabetes	90	60	108	18	93	38	E2V2M4	8	9200	13	0.4	85	2.4	216	48	1.4	137	4.1	1.7	3.9	1.9	2	52	68	6.4	3.6	2.2	12	37	1.1		N	glycosuria 1+					cardiomegaly		congestive hepatopat	EF 28%, global hypokine			
144	57	M	systemic disease	CO2 narcosis	COPD	140	80	104	22	91	37	E2V3M4	9	11200	14	0.5	84	2.6	183	38	1.1	141	3.9	1.9	1.1	0.4	0.7	39	35	6.8	3.9	2.3					respiratory acidosis	N						emphysema	emphysema		EF 42%, moderate PHTN			
145	50	M	systemic disease	hepatic encephalopathy	DCLD	110	70	86	16	96	37	E2V2M4	8	9800	12	0.4	85	2.3	124	42	1.2	136	3.8	1.7	8.1	4	4.1	152	98	6	3.5	2.2	18	46	1.9	157	N	N	no growth	no growth		high SAAG>1.1	N		liver cirrhosis		N		

Sno	AGE(years)	SEX	ETIOLOGY OF ENCEPHALOPATHY	DIAGNOSIS	CO-MORBIDITIE	SBP	DBP	PR	RR	SpO2(% in roo	TEMPERATURE	GCS(on admiss	GCS SCORE	TC(cells/mm3)	Hb(g/dl)	PCV	MCV(fl)	PLATELETS(lal	RBS(mg/dl)	UREA(mg/dl)	CREATININE(r	SODIUM(mEq/l	POTASSIUM(m	MAGNESIUM(r	TOTAL BILIRU	DIRECT	INDIRECT	SGOT(IU/L)	SGPT(IU/L)	TOTAL PROTE	ALBUMIN	GLOBULIN	PT(sec)	APTT(sec)	INR	SERUM AMMONIA(mc	ABG	URINE ANALYSIS	BLOOD CULTURE	URINE CULTURE	CSF ANALYSIS	ASCITIC FLUID ANALYSIS	CHEST X RAY	CT CHEST	USG ABDOMEN	ECHO	CT BRAIN	MRI BRAIN	EEG
146	53	F	systemic disease	uremic encephalopathy	CKD, type 2 diabetes	160	90	82	14	97	37	E2V4M5	11	7400	11	0.3	81	2.1	175	186	6.8	138	4.3	2.2	0.9	0.2	0.7	38	36	6.1	3.4	2.2	14	37	1.2		N	protein 3+		no growth			N		contracted kidneys		N		
147	55	M	systemic disease	CO2 narcosis	COPD, hypertension	150	90	112	24	92	37	E2V4M4	10	8500	15	0.5	87	2.7	142	39	1.1	141	3.9	1.9	0.8	0.2	0.6	36	39	6.7	3.8	2.3					respiratory acidosis	N					emphysema	emphysema		EF 40%, moderate PHTN			
148	48	F	systemic disease	cardiogenic shock	CAD, HFrEF, type 2 diabetes	90	60	116	20	94	37	E3V4M5	12	6400	13	0.4	84	2.6	198	41	1.3	138	3.8	1.7	3.8	1.7	2.1	72	76	6.5	3.7	2.2	13	41	1.1		N	glycosuria 1+						cardiomegaly		congestive hepatopat	EF 29%, global hypokine			
149	46	M	systemic disease	hepatic encephalopathy	DCLD	110	70	88	16	97	37	E2V3M4	9	7200	11	0.4	86	2.5	135	38	1.2	137	3.9	1.8	9.6	4.4	5.2	162	112	6.1	3.5	2.3	19	48	2.1	162	N	N	no growth	no growth			high SAAG>1.1	N		liver cirrhosis		N		
150	55	F	systemic disease	uremic encephalopathy	CKD, type 2 diabetes	150	90	84	18	96	38	E2V3M4	9	10400	10	0.3	84	2.4	210	192	7.1	138	4.2	2.1	1.1	0.3	0.8	41	46	6.2	3.6	2.3	15	38	1.3		N	protein 3+		no growth				N		contracted kidneys		N		
151	27	M	toxins/drugs	alcohol intoxication	-	130	80	86	16	99	38	E3V4M5	12	11200	13	0.4	89	2.6	128	32	0.8	141	4.1	1.9	0.9	0.3	0.6	68	52	6.8	3.9	2.3	11	36	1.1		N	N						N		N		N		
152	34	F	toxins/drugs	benzodiazepine poisoning	-	120	70	78	14	98	37	E2V4M4	10	6500	13	0.4	85	2.7	146	34	0.9	140	3.9	1.8	0.8	0.2	0.6	36	32	6.9	4.1	2.3					N	N						N						
153	38	M	toxins/drugs	nitrobenzene poisoning	-	130	80	92	24	87	37	E3V4M5	12	8800	14	0.4	87	2.9	129	35	0.8	139	4.1	1.9	0.9	0.3	0.6	39	38	6.6	3.9	2.2					metabolic acidosis	N						N						
154	44	M	toxins/drugs	atropine induced	-	120	70	124	16	98	38	E3V4M5	12	9200	13	0.4	86	2.6	134	37	0.9	140	3.9	1.8	1.1	0.3	0.8	35	36	7.1	4.2	2.3					N	N						N						
155	28	M	toxins/drugs	alcohol intoxication	-	110	80	98	14	99	37	E3V4M4	11	8900	13	0.4	84	2.3	158	32	0.8	138	3.7	2.1	0.8	0.2	0.6	37	34	6.8	3.9	2.3					N	N						N						
156	42	F	toxins/drugs	benzodiazepine poisoning	type 2 diabetes	120	80	82	14	98	37	E2V4M4	10	8100	13	0.4	87	2.4	215	33	0.9	139	3.8	1.9	0.9	0.2	0.7	35	38	6.5	3.8	2.2					N	N						N				N		
157	45	M	toxins/drugs	nitrobenzene poisoning	hypertension	140	90	88	22	89	37	E3V4M4	11	9600	14	0.4	85	2.8	176	36	0.8	142	3.9	2.1	0.8	0.2	0.6	32	39	6.9	4.1	2.2					metabolic acidosis	N						N						
158	29	M	toxins/drugs	alcohol intoxication	-	130	80	82	16	99	38	E2V3M4	9	8800	14	0.4	92	2.7	112	38	0.9	140	3.8	1.9	1.2	0.4	0.8	58	67	6.8	3.9	2.3	12	37	1.1		N	N						N		N		N		
159	42	M	toxins/drugs	alcohol intoxication	-	120	70	78	14	98	37	E3V4M5	12	10300	13	0.4	87	2.4	138	35	0.8	139	4.1	2.1	0.8	0.2	0.6	36	35	6.7	3.8	2.3					N	N						N				N		
160	48	M	toxins/drugs	atropine induced	hypertension	140	80	118	16	98	38	E3V3M4	10	9500	13	0.4	89	2.6	143	41	1.1	141	3.9	1.8	0.9	0.3	0.6	38	36	6.6	3.9	2.2					N	N						N						
161	55	M	neurological	hemorrhagic stroke	hypertension	180	100	74	18	96	37	E2V2M4	8	10400	12	0.4	86	2.4	168	28	0.9	141	3.8	1.8	0.9	0.3	0.6	32	38	6.4	3.7	2.1	13	37	1.1		N	N		no growth				N		N	EF 50%, LVH	right capsuloganglionic bleed		
162	63	F	neurological	ischemic stroke	hypertension	190	100	76	16	98	37	E2V2M5	9	11000	11	0.4	84	2.1	143	31	0.8	140	3.9	1.9	0.9	0.3	0.6	34	37	6.2	3.5	2.1	12	36	0.9		N	N		no growth				N		N	EF 48%	left mca infarct	left mca infarct	
163	40	F	neurological	seizure, cerebral venous thrombosis	-	130	80	82	16	98	37	E3V4M4	11	9800	12	0.4	88	2.2	132	30	0.8	142	3.7	2	0.8	0.2	0.6	33	32	6.8	3.9	2.2	12	34	1.1		N	N						N		N		right transverse sinus	right transverse sinus	N
164	54	M	neurological	hemorrhagic stroke	hypertension	170	90	72	16	99	37	E3V4M5	12	7300	13	0.4	91	2.9	145	32	0.8	141	4.1	1.9	0.8	0.2	0.6	32	38	6.7	3.8	2.2	13	35	1.2		N	N	no growth	no growth				N	N	N	EF 44%, LVH	left capsuloganglionic bleed		
165	56	F	neurological	ischemic stroke	hypertension	180	100	80	14	96	38	E2V2M4	8	10800	11	0.4	82	1.9	151	38	1.1	140	4.1	1.7	0.9	0.3	0.6	37	38	5.9	3.5	2.1	13	35	1.1		N	N	no growth	no growth				N		N	EF 42%	right mca infarct	right mca infarct	
166	28	M	neurological	seizure	epilepsy	120	80	88	16	97	38	E3V4M4	11	7800	13	0.4	84	2.4	163	32	0.9	139	3.9	1.9	0.8	0.2	0.6	34	36	7.1	4.2	2.3					N	N						N				diffuse cerebral edema	N	epileptiform spikes in frontal lobe
167	46	M	neurological	hemorrhagic stroke	hypertension	170	90	78	14	99	37	E2V2M4	8	8200	13	0.4	87	2.8	134	31	0.8	142	3.7	1.9	0.8	0.2	0.6	33	31	6.9	3.9	2.3	12	36	1.1		N	N	no growth	no growth				N	N	N	EF 45%, LVH	pontine hemorrhage		
168	53	F	neurological	hemorrhagic stroke	hypertension	160	90	76	15	96	37	E3V4M5	12	9200	12	0.4	88	2.6	132	34	0.9	140	3.9	1.8	0.9	0.3	0.6	36	31	6.6	3.7	2.1	11	36	1		N	N						N		N	EF 43%, LVH	right capsuloganglionic bleed		
169	36	F	neurological	seizure, cerebral venous thrombosis	-	130	70	72	14	98	37	E3V4M4	11	8700	12	0.4	84	2.5	119	32	0.8	142	3.8	1.9	0.8	0.2	0.6	38	41	6.4	3.7	2.1	12	35	0.9		N	N						N		N		superior saggital sinus thrombosis	superior saggital sinus thrombosis	N
170	64	M	neurological	ischemic stroke	type 2 diabetes	180	100	72	16	96	38	E2V3M4	9	10700	12	0.4	85	2.2	225	38	0.9	141	3.9	1.8	0.9	0.3	0.6	35	38	6.2	3.5	2.1	13	36	1.1		N	glycosuria 1+		no growth				N		N		right mca infarct	right mca infarct	
171	63	M	neurological	hemorrhagic stroke	hypertension	160	100	70	14	97	38	E2V2M4	8	11200	12	0.4	87	2.4	121	32	1.1	143	3.8	1.7	0.9	0.3	0.6	39	42	6.6	3.7	2.2	14	35	1		N	N		no growth				N		N	EF 41%	pontine hemorrhage		
172	24	F	neurological	seizure	epilepsy	130	80	98	16	96	38	E3V4M4	11	8600	14	0.4	85	2.6	135	31	0.7	140	4.1	2.1	0.8	0.2	0.6	32	35	6.9	4.2	2.1					N	N						N				diffuse cerebral edema	N	epileptiform spikes in parietal lobe
173	69	M	neurological	ischemic stroke	type 2 diabetes	170	90	88	14	97	37	E2V3M4	9	10700	11	0.4	84	2.1	138	36	1.1	139	4.1	1.8	0.9	0.3	0.6	35	39	6.1	3.6	2.2	14	35	1		N	N	no growth	no growth				N		N	EF 41%	right mca infarct	right mca infarct	
174	54	M	neurological	hemorrhagic stroke	hypertension	160	100	74	16	96	38	E3V4M4	11	8300	12	0.4	87	2.5	152	38	1.1	142	3.8	1.7	0.8	0.2	0.6	33	38	6.2	3.8	2.1	12	37	0.9		N	N						N			EF 45%, LVH	left capsuloganglionic bleed		

Sno	AGE(years)	SEX	ETIOLOGY OF ENCEPHALOPATHY	DIAGNOSIS	CO-MORBIDITIE	SBP	DBP	PR	RR	SpO2(% in roo	TEMPERATURE	GCS(on admiss	GCS SCORE	TC(cells/mm3)	Hb(g/dl)	PCV	MCV(fl)	PLATELETS(lal	RBS(mg/dl)	UREA(mg/dl)	CREATININE(r	SODIUM(mEq/l	POTASSIUM(m	MAGNESIUM(r	TOTAL BILIRU	DIRECT	INDIRECT	SGOT(IU/L)	SGPT(IU/L)	TOTAL PROTE	ALBUMIN	GLOBULIN	PT(sec)	APTT(sec)	INR	SERUM AMMONIA(mc	ABG	URINE ANALYSIS	BLOOD CULTURE	URINE CULTURE	CSF ANALYSIS	ASCITIC FLUID ANALYSIS	CHEST X RAY	CT CHEST	USG ABDOMEN	ECHO	CT BRAIN	MRI BRAIN	EEG
175	26	F	metabolic	diabetic keto acidosis	type 1 diabetes	120	70	92	32	97	37	E2V2M4	8	11000	13	0.4	88	2.6	479	32	0.9	140	3.7	1.9	0.9	0.3	0.6	36	38	6.9	3.9	2.2					metabolic acidosis	ketone +	no growth	no growth			N		N		N		
176	71	M	metabolic	hyponatremia	-	130	90	82	16	96	37	E2V2M4	8	9400	12	0.4	89	2.4	124	39	1.2	127	3.5	1.8	0.9	0.3	0.6	38	34	6.1	3.6	2.2					low sodium	N		no growth			N		N		age related cortical atrophy		
177	60	F	metabolic	hypoglycemia	-	140	80	96	15	99	37	E2V4M4	10	7600	13	0.4	87	2.5	57	36	0.9	139	3.9	1.7	0.8	0.2	0.6	35	36	6.5	3.8	2.1					N	N					N				N		
178	63	M	metabolic	hyperosmolar hyperglycemic state	type 2 diabetes	120	80	88	16	97	37	E2V3M4	9	8500	12	0.4	83	2.6	585	41	1.2	137	3.8	1.8	0.9	0.3	0.6	39	35	6.4	3.7	2.1					N	glycosuria 3+		no growth			N		N		N		
179	44	M	metabolic	hyponatremia	alcoholic liver disease	110	80	82	14	96	37	E2V3M4	9	7600	12	0.4	91	2.5	112	39	1.2	126	3.7	1.7	4.8	2.2	2.6	176	104	6.1	3.6	2.2	16	42	1.5	58	low sodium	N				high SAAG>1.1	N		parenchymal liver disease		N		
180	36	F	metabolic	diabetic keto acidosis	type 2 diabetes	130	80	88	28	98	38	E3V4M5	12	9600	13	0.4	88	2.8	446	38	1.2	139	3.8	1.9	1	0.4	0.6	36	39	6.6	3.7	2.3					metabolic acidosis	ketone +	no growth	no growth			N		N		N		
181	66	M	metabolic	hypoglycemia	-	110	70	92	16	97	37	E2V2M4	8	10200	13	0.4	84	2.4	54	34	0.8	142	4	2.1	0.8	0.2	0.6	34	35	6.4	3.6	2.2					N	N		no growth			N				N		
182	58	F	metabolic	hyperosmolar hyperglycemic state	type 2 diabetes	130	80	86	14	98	37	E3V4M5	12	6900	13	0.4	87	2.6	611	42	1.3	136	3.8	1.8	0.9	0.3	0.6	37	32	6.5	3.8	2.1					N	glycosuria 2+		no growth			N		N		N		
183	47	M	systemic disease	cardiogenic shock	CAD, HFrEF, type 2 diabetes	90	60	108	18	93	38	E2V2M4	8	9200	13	0.4	85	2.4	216	48	1.4	137	4.1	1.7	3.9	1.9	2	52	68	6.4	3.6	2.2	12	37	1.1		N	glycosuria 1+		no growth			cardiomegaly		congestive hepatopat	EF 26%, global hypokine			
184	57	M	systemic disease	hepatic encephalopathy	DCLD	130	80	104	22	91	37	E3V3M4	9	11200	14	0.5	84	2.6	183	38	1.1	141	3.9	1.7	6.8	3.8	3	142	136	6.8	3.9	2.3	15	48	1.7	172	N	N		no growth		high SAAG>1.1	N		liver cirrhosis		N		
185	45	M	systemic disease	hepatic encephalopathy	DCLD	110	70	86	16	96	37	E2V2M4	8	9800	12	0.4	85	2.3	124	42	1.2	136	3.8	1.7	8.2	4	4.2	152	98	6	3.5	2.2	18	46	1.9	149	N	N	no growth	no growth		high SAAG>1.1	N		liver cirrhosis		N		
186	53	F	systemic disease	uremic encephalopathy	CKD, type 2 diabetes	160	90	82	14	97	37	E2V4M5	11	7400	11	0.3	81	2.1	175	186	6.8	138	4.3	2.2	0.9	0.2	0.7	38	36	6.1	3.4	2.2	14	37	1.2		N	protein 3+		no growth			N		contracted kidneys		N		
187	55	M	systemic disease	hepatic encephalopathy	DCLD	130	80	112	24	92	37	E2V4M4	10	8500	15	0.5	87	2.7	142	39	1.1	141	3.9	1.9	7.8	4	3.8	135	147	6.7	3.8	2.3	14	39	1.4	153	N	N		no growth		high SAAG>1.1	N		liver cirrhosis		N		
188	48	F	systemic disease	uremic encephalopathy	CKD, type 2 diabetes	160	90	116	20	94	37	E3V4M5	12	6400	13	0.4	84	2.6	198	179	7.2	138	4.6	1.7	1.2	0.4	0.8	72	76	6.5	3.7	2.2	13	41	1.1		N	glycosuria 1+		no growth			cardiomegaly		contracted kidneys	EF 29%,			
189	46	M	systemic disease	hepatic encephalopathy	DCLD	110	70	88	16	97	37	E2V3M4	9	7200	11	0.4	86	2.5	135	38	1.2	137	3.9	1.8	9.6	4.4	5.2	162	112	6.1	3.5	2.3	19	48	2.1	156	N	N	no growth	no growth		high SAAG>1.1	N		liver cirrhosis		N		
190	55	F	systemic disease	uremic encephalopathy	CKD, type 2 diabetes	150	90	84	18	96	38	E2V3M4	9	10400	10	0.3	84	2.4	210	192	7.1	138	4.2	2.1	1.1	0.3	0.8	41	46	6.2	3.6	2.3	15	38	1.3		N	protein 3+		no growth			N		contracted kidneys		N		
191	27	M	toxins/drugs	alcohol intoxication	-	130	80	86	16	99	38	E3V4M5	12	11200	13	0.4	89	2.6	128	32	0.8	141	4.1	1.9	0.9	0.3	0.6	68	52	6.8	3.9	2.3	11	36	1.1		N	N					N		N		N		
192	34	F	toxins/drugs	benzodiazepine poisoning	-	120	70	78	14	98	37	E2V4M4	10	6500	13	0.4	85	2.7	146	34	0.9	140	3.9	1.8	0.8	0.2	0.6	36	32	6.9	4.1	2.3					N	N					N						
193	38	M	toxins/drugs	atropine induced	-	130	80	118	24	87	37	E3V4M5	12	8800	14	0.4	87	2.9	129	35	0.8	139	4.1	1.9	0.9	0.3	0.6	39	38	6.6	3.9	2.2					N	N					N						
194	44	M	toxins/drugs	atropine induced	hypertension	140	90	124	16	98	38	E3V4M5	12	9200	13	0.4	86	2.6	134	37	0.9	140	3.9	1.8	1.1	0.3	0.8	35	36	7.1	4.2	2.3					N	N					N						
195	28	M	toxins/drugs	alcohol intoxication	-	110	80	98	14	99	37	E3V4M4	11	8900	13	0.4	84	2.3	158	32	0.8	138	3.7	2.1	0.8	0.2	0.6	37	34	6.8	3.9	2.3					N	N					N		N		N		
196	42	F	toxins/drugs	benzodiazepine poisoning	type 2 diabetes	120	80	82	14	98	37	E2V4M4	10	8100	13	0.4	87	2.4	206	33	0.9	139	3.8	1.9	0.9	0.2	0.7	35	38	6.5	3.8	2.2					N	N					N				N		
197	45	M	toxins/drugs	atropine induced	hypertension	140	90	132	22	89	37	E3V4M4	11	9600	14	0.4	85	2.8	176	36	0.8	142	3.9	2.1	0.8	0.2	0.6	32	39	6.9	4.1	2.2					N	N					N						
198	32	M	toxins/drugs	alcohol intoxication	-	130	80	82	16	99	38	E2V3M4	9	8800	14	0.4	92	2.7	112	38	0.9	140	3.8	1.9	1.2	0.4	0.8	58	67	6.8	3.9	2.3	12	37	1.1		N	N					N		N		N		
199	39	M	toxins/drugs	alcohol intoxication	type 2 diabetes	120	70	78	14	98	37	E3V4M5	12	10300	13	0.4	87	2.4	235	35	0.8	139	4.1	2.1	0.8	0.2	0.6	36	35	6.7	3.8	2.3					N	N					N		N		N		
200	48	M	toxins/drugs	atropine induced	hypertension	140	80	118	16	98	38	E3V3M4	10	9500	13	0.4	89	2.6	143	41	1.1	141	3.9	1.8	0.9	0.3	0.6	38	36	6.6	3.9	2.2					N	N					N						
201	58	M	neurological	hemorrhagic stroke	hypertension	180	110	74	18	96	37	E2V2M4	8	10400	12	0.4	86	2.4	168	28	0.9	141	3.8	1.8	0.9	0.3	0.6	32	38	6.4	3.7	2.1	13	37	1.1		N	N		no growth			N		N	EF 50%, LVH	right capsuloganglionic bleed		
202	70	F	neurological	ischemic stroke	hypertension	170	100	76	16	98	37	E2V2M5	9	11000	11	0.4	84	2.1	143	31	0.8	140	3.9	1.9	0.9	0.3	0.6	34	37	6.2	3.5	2.1	12	36	0.9		N	N		no growth			N		N	EF 48%	left mca infarct	left mca infarct	
203	40	F	neurological	hemorrhagic stroke	hypertension	180	110	82	16	98	37	E3V4M4	11	9800	12	0.4	88	2.2	132	30	0.8	142	3.7	2	0.8	0.2	0.6	33	32	6.8	3.9	2.2	12	34	1.1		N	N					N		N	EF 45%, LVH	pontine hemorrhage		

Sno	AGE(years)	SEX	ETIOLOGY OF ENCEPHALOPATHY	DIAGNOSIS	CO-MORBIDITIE	SBP	DBP	PR	RR	SpO2(% in roo	TEMPERATURE	GCS(on admiss	GCS SCORE	TC(cells/mm3)	Hb(g/dl)	PCV	MCV(fl)	PLATELETS(lal	RBS(mg/dl)	UREA(mg/dl)	CREATININE(r	SODIUM(mEq/l	POTASSIUM(m	MAGNESIUM(r	TOTAL BILIRU	DIRECT	INDIRECT	SGOT(IU/L)	SGPT(IU/L)	TOTAL PROTE	ALBUMIN	GLOBULIN	PT(sec)	APTT(sec)	INR	SERUM AMMONIA(mc	ABG	URINE ANALYSIS	BLOOD CULTURE	URINE CULTURE	CSF ANALYSIS	ASCITIC FLUID ANALYSIS	CHEST X RAY	CT CHEST	USG ABDOMEN	ECHO	CT BRAIN	MRI BRAIN	EEG
204	41	M	neurological	hemorrhagic stroke	hypertension	170	100	72	16	99	37	E3V4M5	12	7300	13	0.4	91	2.9	145	32	0.8	141	4.1	1.9	0.8	0.2	0.6	32	38	6.7	3.8	2.2					N	N	no growth	no growth			N		N	EF 51%, LVH	left capsuloganglionic bleed		
205	56	F	systemic disease	cardiogenic shock	CAD, HFrEF	90	60	102	22	94	38	E2V2M4	8	10800	11	0.4	82	1.9	151	59	1.4	138	4.1	1.7	0.9	0.3	0.6	37	38	5.9	3.5	2.1	13	35	1.1		N	N	no growth	no growth			cardiomegaly		congestive hepatopat	EF 24%, global hypokine			
206	52	M	systemic disease	CO2 narcosis	COPD	120	80	98	26	93	38	E3V4M4	11	7800	13	0.4	84	2.4	163	32	0.9	139	3.9	1.9	0.8	0.2	0.6	34	36	7.1	4.2	2.3					respiratory acidosis	N					emphysema	emphysema	N	EF 46%	N		
207	46	M	systemic disease	hepatic encephalopathy	DCLD	130	80	78	14	99	37	E3V4M5	12	8200	13	0.4	87	2.8	134	31	0.8	142	3.7	1.9	8.1	4.1	4	128	163	6.9	3.9	2.3	15	40	1.6	167	N	N	no growth	no growth		high SAAG>1.1	N		liver cirrhosis		N		
208	53	F	systemic disease	uremic encephalopathy	hypertension	160	90	76	15	96	37	E3V3M4	10	9200	12	0.4	88	2.6	132	192	6.9	140	4.9	1.8	0.9	0.3	0.6	36	31	6.6	3.7	2.1	11	36	1		N	N		no growth			N		contracted kidneys	EF 43%, LVH	N		
209	56	M	systemic disease	CO2 narcosis	-	130	70	94	22	93	37	E3V4M4	11	8700	12	0.4	84	2.5	119	32	0.8	142	3.8	1.9	0.8	0.2	0.6	38	41	6.4	3.7	2.1					respiratory acidosis	N					emphysema		N	EF 44%, moderate PHTN	N		
210	64	M	systemic disease	cardiogenic shock	type 2 diabetes	90	60	122	18	94	38	E2V3M4	9	10700	12	0.4	85	2.2	225	46	1.5	141	3.9	1.8	0.9	0.3	0.6	35	38	6.2	3.5	2.1	13	36	1.1		N	glycosuria 1+		no growth			cardiomegaly		N	EF 26%, global hypokine			
211	45	M	systemic disease	hepatic encephalopathy	DCLD	140	90	84	14	97	38	E2V2M4	8	11200	12	0.4	87	2.4	121	32	1.1	143	3.8	1.7	7.8	3.8	4	139	127	6.6	3.7	2.2	18	45	1.8		N	N		no growth			N		liver cirrhosis				
212	43	F	systemic disease	uremic encephalopathy	CKD, type 2 diabetes	160	90	82	16	96	38	E3V4M4	11	8600	14	0.4	85	2.6	135	172	7.4	140	4.9	2.1	0.8	0.2	0.6	32	35	6.9	4.2	2.1					N	protein 3+		no growth			N		contracted kidneys		N		
213	69	M	systemic disease	cardiogenic shock	CAD, HFrEF	90	70	112	22	95	37	E2V3M4	9	10700	11	0.4	84	2.1	138	36	1.1	139	4.1	1.8	0.9	0.3	0.6	35	39	6.1	3.6	2.2	14	35	1		N	N					cardiomegaly		N	EF 31%, global hypokine			
214	54	M	systemic disease	CO2 narcosis	COPD, hypertension	160	90	98	24	93	38	E3V4M4	11	8300	12	0.4	87	2.5	152	38	1.1	142	3.8	1.7	0.8	0.2	0.6	33	38	6.2	3.8	2.1	12	37	0.9		N	respiratory acidosis	N				emphysema	emphysema	N	EF 42%, moderate PHTN	N		
215	26	F	metabolic	diabetic keto acidosis	type 1 diabetes	120	70	92	32	97	37	E2V2M4	8	11000	13	0.4	88	2.6	459	32	0.9	140	3.7	1.9	0.9	0.3	0.6	36	38	6.9	3.9	2.2					metabolic acidosis	ketone +	no growth	no growth			N		N		N		
216	67	F	metabolic	hyponatremia	hypertension	130	90	82	16	96	37	E2V2M4	8	9400	12	0.4	89	2.4	124	39	1.2	127	3.5	1.8	0.9	0.3	0.6	38	34	6.1	3.6	2.2					low sodium	N		no growth			N		N		N		
217	60	F	metabolic	hypoglycemia	type 2 diabetes	140	80	114	16	99	37	E2V4M4	10	7600	13	0.4	87	2.5	58	36	0.9	139	3.9	1.7	0.8	0.2	0.6	35	36	6.5	3.8	2.1					N	N					N				N		
218	70	M	metabolic	hyperosmolar hyperglycemic state	type 2 diabetes	120	80	88	16	97	37	E2V3M4	9	8500	12	0.4	83	2.6	590	41	1.2	137	3.8	1.8	0.9	0.3	0.6	39	35	6.4	3.7	2.1					N	glycosuria 3+		no growth			N		N		N		
219	65	F	metabolic	hyperosmolar hyperglycemic state	type 2 diabetes	110	80	82	14	96	37	E2V3M4	9	7600	12	0.4	91	2.5	604	39	1.2	126	3.7	1.7	4.1	2	2.1	176	104	6.1	3.6	2.2	16	42	1.5		N	N		no growth			N		N		N		
220	37	F	metabolic	diabetic keto acidosis	type 2 diabetes	130	90	104	28	98	38	E3V4M5	12	9600	13	0.4	88	2.8	406	38	1.2	139	3.8	1.9	1	0.4	0.6	36	39	6.6	3.7	2.3					metabolic acidosis	ketone +	no growth	no growth			N		N		N		
221	67	M	metabolic	hypoglycemia	-	110	70	92	16	97	37	E2V2M4	8	10200	13	0.4	84	2.4	54	34	0.8	142	4	2.1	0.8	0.2	0.6	34	35	6.4	3.6	2.2					N	N		no growth			N				N		
222	58	F	metabolic	hyperosmolar hyperglycemic state	type 2 diabetes	130	80	92	18	98	37	E3V4M5	12	6900	13	0.4	87	2.6	621	42	1.3	136	3.8	1.8	0.9	0.3	0.6	37	32	6.5	3.8	2.1					N	glycosuria 2+		no growth			N		N		N		
223	47	M	systemic disease	uremic encephalopathy	CKD, type 2 diabetes	160	100	108	18	93	38	E2V2M4	8	9200	13	0.4	85	2.4	216	198	7.4	137	5.4	1.7	3.9	1.9	2	52	68	6.4	3.6	2.2	12	37	1.1		N	glycosuria 1+		no growth			N		contracted kidneys				
224	57	M	systemic disease	hepatic encephalopathy	DCLD	140	80	104	22	91	37	E2V3M4	9	11200	14	0.5	84	2.6	183	38	1.1	141	3.9	1.9	8.6	4.1	4.5	137	124	6.2	3.9	2.3				162	N	N		no growth		high SAAG>1.1	N		liver cirrhosis		N		
225	45	M	systemic disease	hepatic encephalopathy	DCLD	110	70	86	16	96	37	E2V2M4	8	9800	12	0.4	85	2.3	124	42	1.2	136	3.8	1.7	8.2	4	4.2	152	98	6	3.5	2.2	18	46	1.9	149	N	N	no growth	no growth		high SAAG>1.1	N		liver cirrhosis		N		
226	53	F	systemic disease	uremic encephalopathy	CKD, type 2 diabetes	160	90	82	14	97	37	E2V4M5	11	7400	11	0.3	81	2.1	175	186	6.8	138	4.3	2.2	0.9	0.2	0.7	38	36	6.1	3.4	2.2	14	37	1.2		N	protein 3+		no growth			N		contracted kidneys		N		
227	55	M	systemic disease	hepatic encephalopathy	DCLD	150	90	112	24	92	37	E2V4M4	10	8500	15	0.5	87	2.7	142	39	1.1	141	3.9	1.9	7.8	3.9	3.9	148	153	6.7	3.8	2.3	14	38	1.4	153	N	N		no growth		high SAAG>1.1	N		liver cirrhosis		N		
228	48	F	systemic disease	uremic encephalopathy	CKD, type 2 diabetes	170	100	116	20	94	37	E3V4M5	12	6400	13	0.4	84	2.6	198	175	6.9	138	4.9	1.7	3.8	1.7	2.1	72	76	6.5	3.7	2.2	13	41	1.1		N	glycosuria 2+		no growth				N		contracted kidneys		N		
229	46	M	systemic disease	hepatic encephalopathy	DCLD	110	70	88	16	97	37	E2V3M4	9	7200	11	0.4	86	2.5	135	38	1.2	137	3.9	1.8	9.6	4.4	5.2	162	112	6.1	3.5	2.3	19	48	2.1	156	N	N	no growth	no growth		high SAAG>1.1	N		liver cirrhosis		N		
230	55	M	systemic disease	hepatic encephalopathy	DCLD, hypertension	150	90	84	18	96	38	E2V3M4	9	10400	10	0.3	84	2.4	210	54	1.3	138	4.2	2.1	8.9	4.3	4.6	147	163	6.2	3.6	2.3	15	38	1.3		N	N		no growth		high SAAG>1.1	N		liver cirrhosis		N		
231	27	M	toxins/drugs	alcohol intoxication	-	130	80	86	16	99	38	E3V4M5	12	11200	13	0.4	89	2.6	128	32	0.8	141	4.1	1.9	0.9	0.3	0.6	68	52	6.8	3.9	2.3	11	36	1.1		N	N					N		N		N		
232	34	F	toxins/drugs	atropine induced	-	120	70	132	14	98	37	E2V4M4	10	6500	13	0.4	85	2.7	146	34	0.9	140	3.9	1.8	0.8	0.2	0.6	36	32	6.9	4.1	2.3					N	N					N						

Sno	AGE(years)	SEX	ETIOLOGY OF ENCEPHALOPATHY	DIAGNOSIS	CO-MORBIDITIE	SBP	DBP	PR	RR	SpO2(% in roo	TEMPERATURE	GCS(on admiss	GCS SCORE	TC(cells/mm3)	Hb(g/dl)	PCV	MCV(fl)	PLATELETS(lal	RBS(mg/dl)	UREA(mg/dl)	CREATININE(r	SODIUM(mEq/l	POTASSIUM(m	MAGNESIUM(r	TOTAL BILIRU	DIRECT	INDIRECT	SGOT(IU/L)	SGPT(IU/L)	TOTAL PROTE	ALBUMIN	GLOBULIN	PT(sec)	APTT(sec)	INR	SERUM AMMONIA(mc	ABG	URINE ANALYSIS	BLOOD CULTURE	URINE CULTURE	CSF ANALYSIS	ASCITIC FLUID ANALYSIS	CHEST X RAY	CT CHEST	USG ABDOMEN	ECHO	CT BRAIN	MRI BRAIN	EEG
233	38	M	toxins/drugs	atropine induced	-	130	80	126	18	96	37	E3V4M5	12	8800	14	0.4	87	2.9	129	35	0.8	139	4.1	1.9	0.9	0.3	0.6	39	38	6.6	3.9	2.2					N	N					N						
234	63	M	metabolic	hyperosmolar hyperglycemic state	type 2 diabetes	120	80	88	16	97	37	E2V3M4	9	8500	12	0.4	83	2.6	590	41	1.2	137	3.8	1.8	0.9	0.3	0.6	39	35	6.4	3.7	2.1					N	glycosuria 2+		no growth			N		N		N		
235	56	F	metabolic	hyperosmolar hyperglycemic state	type 2 diabetes	130	80	92	14	96	37	E2V3M4	9	7600	12	0.4	91	2.5	546	39	1.2	126	3.7	1.7	1.1	0.4	0.7	46	62	6.1	3.6	2.2					N	glycosuria 3+		no growth			N		N		N		