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Case Series

Radiology Section

Neuroimaging Perspectives on Metabolic Encephalopathies: A Case Series

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ABSTRACT

Encephalopathies arise from various causes, such as toxic exposures, autoimmune conditions and metabolic disturbances. Their symptoms are typically non specific and may include seizures, localised neurological issues, movement abnormalities, or more severe outcomes like coma, lasting complications, or death. Here, the authors enumerate a few commonly encountered metabolic encephalopathies, (2 females, 4 males) such as the involvement of the splenium in Cytotoxic Lesions of the Corpus Callosum (CLOCCS), the mamillary bodies in Marchiafava-Bignami Disease (MBD) and the lentiform nucleus in uraemic encephalopathy. Knowledge of these patterns aids in achieving a specific diagnosis. Each pattern is linked to the most likely differential diagnosis, aligning more closely with the real-world challenges faced by radiologists.

Keywords: Encephalopathy, Magnetic resonance imaging, Symmetrical

INTRODUCTION

Metabolic brain disorders arise from disturbances in the delicate balance of key physiological factors, including metabolic substrates, neurotransmitter levels, electrolytes, pH and cerebral blood flow, which are disrupted by internal dysfunctions [1]. The brain is highly vulnerable to damage from metabolic by-products and toxins. Magnetic Resonance Imaging (MRI) can reveal such damage at both early and later stages of the disease. However, the degree of brain damage observed in imaging may not always correspond to the severity of the patient's clinical symptoms [2]. Bilateral and symmetric lesions with restricted diffusion, mild or no mass effect and lack of enhancement are typical features [1]. The areas most commonly affected are the cortical grey matter, deep grey nuclei, thalami, periventricular white matter and the corpus callosum. Lesions with these characteristics may indicate underlying metabolic disturbances but are non specific and require proper clinical context for accurate interpretation [1]. These conditions are frequently encountered in emergency departments, where patients present with global cerebral dysfunctions such as acute confusion and delirium. Neuroimaging plays a crucial role in diagnosing these disorders and refining the differential diagnosis.

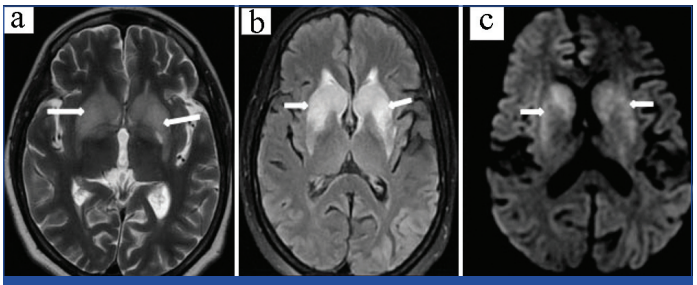
CASE SERIES

Case 1

A 46-year-old female patient presented with complaints of behavioural disturbances, swaying while walking, insomnia and difficulty in speech for the past five days. She has a known history of chronic kidney disease for four years and has been undergoing regular dialysis. She is not a known case of diabetes. She has a previous history of seizures three months ago (two episodes in a month lasting for three minutes each).

Blood investigations revealed elevated blood urea and creatinine levels. An MRI brain scan was performed, which showed bilateral T2/FLAIR symmetric hyperintensities involving the caudate nuclei, anterior limb of the internal capsule and putamen of both basal ganglia, which exhibit focal and variable diffusion restriction on Diffusion-Weighted Imaging (DWI), giving rise to the lentiform fork sign [Table/Fig-1a-c].

These imaging findings are consistent with uraemic encephalopathy, a condition commonly seen in patients with chronic kidney disease, particularly when dialysis is missed or insufficient. The involvement of multiple brain regions, especially with diffusion restriction in the globus pallidus, is typical of uraemic encephalopathy and correlates with the patient's elevated blood urea and creatinine levels.

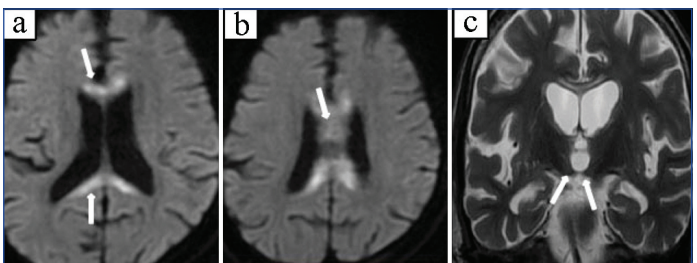


[Table/Fig-1]: a) T2 axial, b) Fluid-Attenuated Inversion Recovery (FLAIR) and c) DWI Imaging showing bilateral symmetrical basal ganglia hyperintensities with diffusion restriction. Note the typical fork like appearance of bilateral basal ganglia (lentiform fork sign) denoted by arrows suggestive of uraemic encephalopathy.

Case 2

A 56-year-old alcoholic male presented with complaints of an inability to walk, accompanied by shivering all over the body, anxiety with restlessness, irrelevant talk, slurred speech, fever and delirium for one week. During the clinical examination, the patient appeared malnourished and was in an altered state of consciousness (E3V3M3). There were no signs of meningeal irritation, such as neck stiffness or a positive Kernig's sign. The pupils were of normal size and reacted appropriately. The motor examination did not reveal any neurological deficits.

A complete blood count revealed leukocytosis (white blood count: 14.21×10^3 g/dL) and anaemia (haemoglobin: 10.9 g/dL). Urinalysis was negative. Computed Tomographic (CT) imaging was unremarkable. MRI brain imaging findings included subtle diffusion restriction mainly involving the body, splenium and genu of the corpus callosum, as well as atrophy of the bilateral mammillary bodies [Table/Fig-2a-c]. A diagnosis of Marchiafava-Bignami disease with metabolic encephalopathy was made based on the imaging findings.



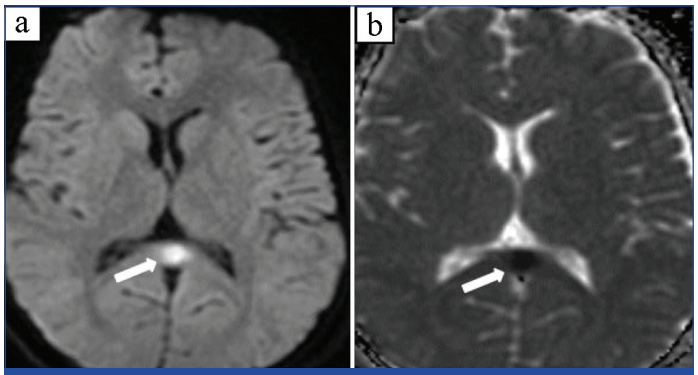
[Table/Fig-2a,b]: DWI images showing diffusion restriction in body, genu and splenium of corpus callosum (arrows). Also, note atrophy of bilateral mamillary bodies in image c (arrows).

A retrospective evaluation of his vitamin levels showed decreased levels of vitamin B, which further supports the diagnosis (68 pg/mL). The patient was started on vitamin B supplementation and his symptoms improved on follow-up. Alcohol cessation, rehabilitation and nutritional counselling were recommended.

Case 3

A 15-year-old male presented with symptoms of acute gastroenteritis, followed by altered sensorium for the past four days. On physical examination, the patient was conscious, alert and oriented, with no signs of muscle weakness or dizziness. He was referred to the Radiodiagnosis department for a CT brain scan, which did not reveal any significant abnormalities. Subsequently, an MRI brain scan was recommended. Laboratory investigations, including a haemogram, renal and liver function tests and serum electrolyte analysis, were all within normal limits.

The MRI findings demonstrated a small focal area of diffusion restriction in the splenium of the corpus callosum [Table/Fig-3a,b], with no other notable abnormalities. These imaging features were consistent with CLOCC.



[Table/Fig-3]: A DWI and B ADC depicting diffusion restriction in splenium of corpus callosum - s/o CLOCCS (ARROWS).

The patient was managed with antibiotics, including ceftriaxone and vancomycin, along with supportive care.

Case 4

A 38-year-old male with a history of chronic alcoholism, diabetes and hypertension presented with a two-week history of generalised fatigue and slowed activities. Initial evaluation at an external facility revealed severe hyponatraemia, with a sodium level of 107 mmol/L. Correction was performed using hypertonic saline, raising sodium levels to 138 mmol/L within two days.

Following sodium correction, the patient developed generalised limb stiffness, reduced speech output and tremors affecting all four limbs. He was subsequently referred for further assessment.

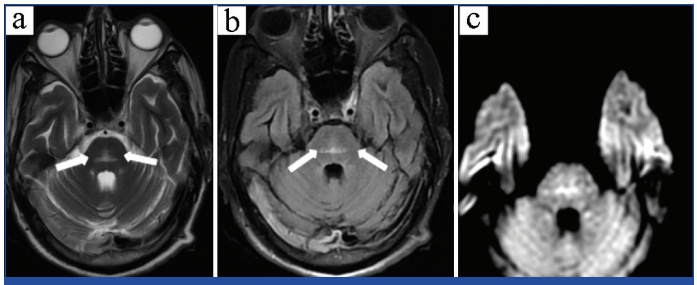
On clinical examination, the patient exhibited an abulic phenotype characterised by reduced responsiveness and significant extrapyramidal features, including limb rigidity, bradykinesia, tremors and gait instability. Laboratory tests at the study hospital showed normal sodium levels.

An MRI of the brain revealed a transverse band of central pontine hyperintensity and perpendicular signal (trident sign) with subtle diffusion restriction [Table/Fig-4a-c], suggestive of Osmotic Demyelination Syndrome (ODS).

The patient was managed with symptomatic treatment, including levodopa, pramipexole and physiotherapy. He showed substantial improvement at the time of discharge and continues to be under regular follow-up.

Case 5

A 22-year-old primigravida at 40 weeks and two days of gestation, in labour, was scheduled for an emergency caesarean section due to failure to progress. During her pregnancy, she had no history of



[Table/Fig-4]: A T2WI and B FLAIR images depicting transverse band of central pontine hyperintensity and perpendicular signal (trident sign) denoted by arrows with mild diffusion restriction on DWI image c consistent with central pontinemyelenolysis/ osmotic demeyelenation syndrome.

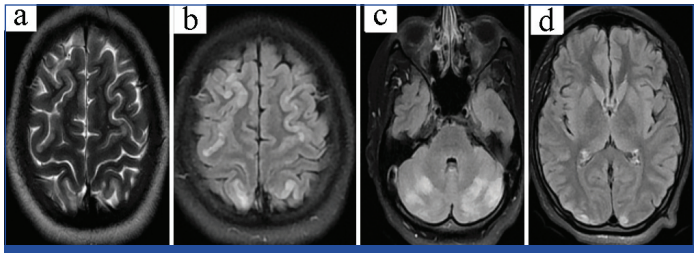
proteinuria, peripheral oedema, or neurological symptoms such as seizures. Her blood pressure remained consistently normal and she did not have chronic hypertension. Preoperative biochemical and haematological investigations were within normal limits.

On arrival in the operating room, her vital signs showed a blood pressure of 110/70 mmHg and a heart rate of 75 beats per minute. The surgery was uneventful and a healthy infant was delivered with Apgar scores of 9 and 10 at one and five minutes, respectively. The maternal blood pressure remained stable intraoperatively and no vasopressor medication was required.

On the second postoperative day, the patient developed a postural headache localised to the frontal and occipital regions. Conservative treatment for Post-Dural Puncture Headache (PDPH) was initiated. However, on postoperative day six, she experienced a generalised tonic-clonic seizure lasting a few minutes. Intubation was performed for airway management and she was treated with intravenous thiopental sodium (200 mg), diazepam (10 mg) and midazolam (3 mg). The patient regained alertness and orientation within a few minutes.

Her vital signs following the seizure showed a blood pressure of 180/100 mmHg, a heart rate of 74 beats per minute and a body temperature of 36.8°C. Haematological analysis revealed anaemia, with a red blood cell count of $3.68\times10^{6}/mm^{3}$, haemoglobin at 11.9 g/dL and haematocrit at 35.1%. Urine analysis indicated a trace amount of proteinuria. Neurological examinations, including assessments of higher cortical function, sensory and motor function, cerebellar function, gait and posture, were all normal.

Cerebrospinal Fluid (CSF) analysis showed normal levels of protein (28.2 mg/dL), glucose (47 mg/dL) and chloride (30.9 mEq/L). Brain Computed Tomography (CT) scans revealed no abnormalities. However, a MRI scan performed the same day identified nearly symmetrical cortical and subcortical patchy areas of FLAIR hyperintensities without diffusion restriction involving the bilateral frontoparietal lobes, parieto-occipital lobes and bilateral posterior cerebellar hemispheres [Table/Fig-5a-d]. These findings were consistent with Posterior Reversible Encephalopathy Syndrome (PRES). She was treated with intravenous thiopental sodium (200 mg), diazepam (10 mg) and midazolam (3 mg) for seizure management and antihypertensive treatment as required. The patient was doing well at the 15-day follow-up with normal blood pressure values.



[Table/Fig-5a-d]: MRI axial images showing nearly symmetrical cortical and subcortical patchy areas of FLAIR hyperintensities withoutdiffusion-FLAIR mismatch in the bilateral frontoparietal lobes, parieto-occipital lobes and bilateral posterior cerebellar hemispheres.

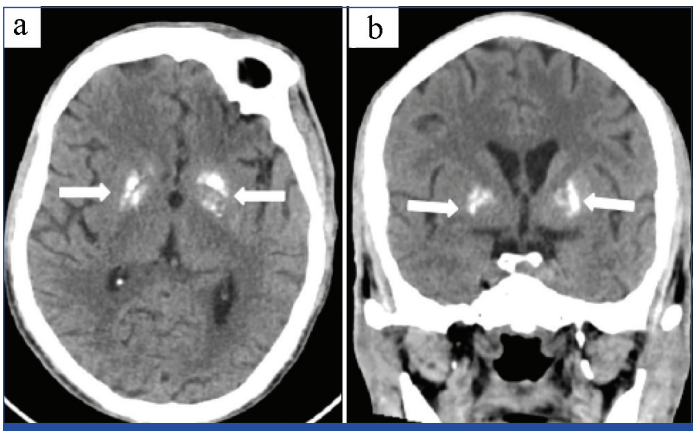
Case 6

A 46-year-old male presented to the emergency department after experiencing two episodes of seizures in one day, each lasting two minutes and occurring 30 minutes apart. He had no prior history of similar complaints. The patient had a history of diabetes and was on regular treatment. He was a non smoker, moderately built and well-nourished, with no evidence of skeletal abnormalities or neurocutaneous markers.

On examination, he was conscious, oriented to time and place and demonstrated normal memory and cognitive function. There were no signs of motor weakness, cranial nerve deficits, rigidity, tremors, or abnormal movements. His coordination was intact and systemic examinations revealed no abnormalities.

Laboratory investigations showed a normal haemogram, renal function and liver function tests. However, his Erythrocyte Sedimentation Rate (ESR) was elevated at 49. Serum calcium, magnesium, phosphorus and alkaline phosphatase levels were low. The Parathyroid Hormone (PTH) assay indicated reduced levels, while thyroid function tests were within normal limits. CSF analysis was normal. Additionally, Venereal Diseases Research Laboratory (VDRL) and Human Immunodeficiency Virus (HIV) tests returned negative results.

A CT scan of the brain was performed, which revealed bilateral symmetrical calcifications in the basal ganglia [Table/Fig-6a,b]. Subtle calcific densities were also seen in the bilateral corona radiata and centrum semiovale. These imaging findings are more likely due to hypercalcaemia and further investigation revealed low PTH levels (8.3 pg/L), suggestive of hypoparathyroidism. The patient was initiated on treatment with levodopa.



[Table/Fig-6]: a) Axial; and b) coronal plain CT images depicting bilateral symmetrical basal ganglia calcifications (denoted by arrows) consistent with hypoparathyroid related disorder.

DISCUSSION

Metabolic disorders are strongly associated with excitotoxic brain injury, primarily due to their role in triggering excessive glutamate release. While excitotoxic receptors are found throughout the central nervous system, certain brain regions are particularly vulnerable to this type of damage, such as the basal ganglia, thalami, cortical grey matter, periventricular white matter and the corpus callosum. Understanding this selective vulnerability is crucial, as it can help identify specific imaging patterns that suggest metabolic causes during the diagnostic process [1]. Some commonly encountered metabolic encephalopathies with classical imaging patterns are detailed below and simplified in tabular form for better comprehension [Table/Fig-7].

Metabolic brain disorders	Specific sites of involvement
Wernicke encephalopathy	Thalami, mammillary bodies and tectum
Chronic hepatic encephalopathy	Globipallidi (T1 high signal)
Parathyroid disorders	Thalami and globipallidi (T2 low signal)
Hyperammonemic encephalopathy	Insular and cingulate cortex
Cobalamin deficiency	Corticospinal tracts involvemnet
CLOCCs	Splenium
Marchiafava-Bignami Disease (MBD)	Corpus callosum
PRES	Parieto occipital vasogenicoedema
Osmotic Demyelination Syndrome (ODS)	Central pons
Wernicke encephalopathy	Thalami, mammillary bodies and tectum
Chronic hepatic encephalopathy	Globipallidi (T1 high signal)
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Osmotic Demyelination Syndrome (ODS)	Central pons

[Table/Fig-7]: Common metabolic encephalopathies and their specific sites of involvement.

URAEMIC ENCEPHALOPATHY

Uraemic encephalopathy, a metabolic disorder associated with both acute and chronic renal failure, results from the accumulation of endogenous uraemic toxins in patients with severe kidney dysfunction. The exact pathogenesis is complex and not fully understood, but the condition is believed to stem from neurotoxicity caused by the build-up of uraemic toxins, such as guanidine compounds [3]. Clinical manifestations are varied, including movement disorders (e.g., tremor, asterixis, myoclonus), seizures, cognitive impairment and altered mental status [4].

Imaging findings in uraemic encephalopathy typically follow three patterns: basal ganglia involvement (the most common), cortical or subcortical involvement and white matter changes (due to acute tubular necrosis or ATN). Bilateral, symmetric basal ganglia involvement is the most frequent imaging finding, typically accompanied by varying degrees of white matter and cortical involvement [5]. One notable sign is the “lentiform fork sign,” often seen in uraemic encephalopathy, which may suggest the presence of metabolic acidosis alongside uraemia. This sign, typically visible on T2-weighted and FLAIR images, is characterised by hyperintensity in the white matter surrounding the lentiform nuclei (internal and external capsules and medullary laminae). This pattern delineates the lateral and medial boundaries of the putamen, resulting in a distinctive appearance that is frequently observed in diabetic patients as well [6].

MARCHIAFAVA-BIGNAMI DISEASE (MBD)

The MBD is a rare condition primarily linked to chronic alcohol consumption and vitamin B complex deficiency. MBD presents in two clinical forms. Type A (acute) involves patients who exhibit seizures or coma, with widespread involvement of the corpus callosum, typically leading to death within a few days. Type B (chronic) is marked by mild encephalopathy and focal lesions, usually affecting the genu of the corpus callosum [7]. MBD often coexists with other alcohol-related pathological conditions and there have been cases where both Wernicke’s Encephalopathy (WE) and MBD occur simultaneously. Associated brain volume loss is also frequently observed [7].

Imaging plays a crucial role in diagnosing MBD, particularly through the identification of corpus callosum involvement. In the context of chronic alcohol use, selective damage to the middle layers of the corpus callosum is a key diagnostic feature, often referred to as the “sandwich sign.” In acute MBD, initial changes are best visualised on sagittal FLAIR images, showing central involvement of the corpus callosum while sparing the periphery. In Type B MBD, lesions typically begin in the genu and frontoparietal cortex, later progressing to involve the splenium. White matter changes may be observed, although they can vary [8]. Diffusion-Weighted Imaging (DWI) may initially show no abnormalities, with diffusion

restriction developing later. Acute lesions may demonstrate contrast enhancement. Chronic MBD is characterised by thinning of the corpus callosum and central linear hypointensities on T1-weighted images [8].

Cytotoxic Lesions of the Corpus Callosum (CLOCCS)

Reversible Splenial Lesions (RSLs), also referred to as CLOCCS, are secondary lesions associated with a variety of conditions. These lesions are generally reversible and tend to involve the splenium of the corpus callosum [9]. While the exact pathophysiology of RSLs remains unclear, it is believed to be related to excitotoxic intracellular and/or intramyelinic oedema, as the splenium has a high concentration of excitatory receptors, making it particularly susceptible to cytotoxic oedema [10].

The RSLs are most commonly associated with the use and subsequent withdrawal of antiepileptic drugs, typically occurring between 24 hours and three weeks after discontinuation. Other conditions linked to RSLs include viral infections (resulting in mild encephalopathy), metabolic disturbances such as hypoglycaemia, hypernatraemia and acute alcohol poisoning [11]. Clinically, patients with RSLs are often asymptomatic. Imaging findings typically show ovoid lesions in the central splenium, which appear hyperintense on T2-weighted and FLAIR images and hypointense on T1-weighted images. These lesions also demonstrate restricted diffusion and lack enhancement [11].

The RSL lesions are usually confined to the splenium, although in rare cases, they may involve the entire corpus callosum. While most lesions resolve completely, some patients may experience severe encephalopathy. In these cases, the lesions may not fully resolve, leading to the preferred terminology of “CLOCCS” [12].

Osmotic Demyelination Syndrome (ODS)

The ODS is an acute form of demyelination caused by rapid shifts in serum osmolality, most commonly due to the rapid correction of hyponatraemia. However, it is crucial to note that ODS can also occur in normonatraemic patients, as osmotic stress is the fundamental mechanism behind the disorder. A range of conditions and comorbidities can predispose patients to the development of ODS.

In the classic case, where rapid correction of hyponatraemia causes ODS, sodium levels are typically below 115 mmol/L and the correction rate exceeds 12 mmol/L per day. Oligodendrocytes, particularly in the pons, are especially vulnerable to osmotic changes, leading to demyelination that most commonly affects the brainstem region. This led to the original term, “central pontine myelinolysis.” However, ODS can also involve extrapontine cerebral regions, including the cortex and these cases are becoming more frequently reported.

Alcoholism is often cited as a risk factor for ODS following the rapid correction of hyponatraemia. Clinically, ODS presents with seizures and altered mental status, often following a biphasic pattern. Patients may initially experience altered mental status due to hyponatraemia, followed by improvement after sodium levels are corrected, only to deteriorate again within a week [13].

The typical imaging appearance of ODS involves well-demarcated, symmetric, rounded, or trident-shaped lesions in the central pons, which typically spare the peripheral pons and corticospinal tract areas. DWI can reveal restricted diffusion in the acute phase, as early as 24 hours after the onset of ODS, which is an important diagnostic marker for early detection [14].

Posterior Reversible Encephalopathy Syndrome (PRES)

Reversible Posterior Leukoencephalopathy Syndrome (PRES) is a clinicoradiological condition characterised by potentially reversible subcortical vasogenic brain oedema, often presenting with acute neurological symptoms such as seizures (60%-75%), altered mental status and headaches (20%-25%). It typically occurs in the context of various conditions, including hypertension, preeclampsia, renal failure, sepsis, thrombocytopenia and the use of cytotoxic or immunosuppressive medications, among others. In most cases, symptoms and imaging findings resolve after appropriate treatment [15].

Current theories suggest a shared pathophysiological mechanism involving endothelial injury and dysfunction, triggered by various causes (such as cytotoxic or immunogenic factors), leading to vasogenic oedema. This results in the accumulation of interstitial fluid in the subcortical white matter, with a particular affinity for the parietal and occipital lobes [16]. PRES can affect individuals of any age, although it is most commonly seen in young women.

Imaging findings are consistent with vasogenic oedema, often appearing as bilateral and asymmetric high signal intensity on T2-weighted and FLAIR images, typically involving the subcortical white matter. DWI and Apparent Diffusion Coefficient (ADC) maps are usually negative or show less extensive changes compared to FLAIR images, helping to distinguish PRES from conditions involving cytotoxic or intramyelinic oedema [16].

PARATHYROID DISORDERS

The primary imaging characteristics are consistent across all conditions, primarily involving calcium accumulation in the basal ganglia. CT scans reveal prominent, bilateral and symmetrical calcifications in the globus pallidus, putamen and caudate nuclei, with the thalamus, subcortical white matter and dentate nuclei potentially also being affected. On MRI, calcium deposits typically appear hyperintense on T1-weighted images and hypointense on T2-weighted images. Furthermore, T2-weighted or susceptibility-weighted imaging may reveal blooming artefacts, which are indicative of calcium deposits [17].

Other associated findings may include subcutaneous soft-tissue calcifications in the extremities.

HYPOGLYCAEMIC ENCEPHALOPATHY

Adult hypoglycaemic encephalopathy, also referred to as hypoglycaemic brain injury, results from an imbalance between glucose supply and utilisation within cerebral cells, ultimately causing brain damage. Clinical symptoms often include seizures, reduced consciousness and even coma, particularly in diabetic patients undergoing insulin therapy [18].

This condition tends to affect posterior and deep brain regions. Imaging studies frequently reveal symmetric hyperintensities on T2-weighted and FLAIR sequences, along with pronounced restricted diffusion in the parieto-occipital and temporal gyri on DWI. Basal ganglia involvement is also observed and may indicate a poorer prognosis. Another notable imaging feature is the preservation of the thalami, white matter and cerebellum. Early indicators can appear as sulcal effacement due to gyral swelling on T1-weighted MRI or hypoattenuation on CT scans [18].

Distinguishing hypoglycaemic brain injury from hypoxic-ischaemic brain injury is clinically important, as both conditions exhibit similar imaging characteristics. A key detail that can aid in differentiation is the presence of a history of cardiac arrest, which is commonly associated with hypoxic-ischaemic injury. Furthermore, hypoxic-ischaemic brain injury typically shows symmetrical involvement of the thalami and cerebellum, features that can help refine the diagnosis [19].

CONCLUSION(S)

Diagnosing metabolic brain disorders is a complex challenge, as it involves a diverse group of conditions. While many imaging features are non specific, some findings can be highly indicative of certain disorders. Imaging studies can also provide valuable prognostic

information, suggesting whether a condition may be reversible or indicative of significant damage to cerebral structures. Clinical history is vital, as it often points to toxic or metabolic causes over other conditions with similar imaging characteristics. Combining clinical data with imaging findings allows for a more accurate diagnosis. Recognising specific imaging features, such as symmetrical lesion patterns, characteristic locations and enhancement patterns, enables radiologists to narrow down the diagnosis, ultimately improving patient outcomes through a collaborative, multidisciplinary approach. MRI is crucial in the management of acutely encephalopathic patients, particularly in ruling out surgically treatable conditions that could be causing a decreased level of consciousness.

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