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Case Report

Internal Medicine Section

Hypoglycaemia-induced Parkinsonism in a Patient with Diabetes Mellitus and Tuberculous Encephalitis: A Complex Case Report

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ABSTRACT

Hypoglycaemia-induced Parkinsonism is a rare but significant complication of metabolic brain dysfunction, characterised by movement disorders following severe hypoglycaemia. We present a 65-year-old male with a 12-year history of type 2 diabetes mellitus, Autoimmune Haemolytic Anaemia (AIHA), and recent tuberculous encephalitis who was found unresponsive due to severe hypoglycaemia. Despite intravenous glucose administration, he developed new-onset Parkinsonism, manifesting as tremors, rigidity, and bradykinesia. Neurological imaging revealed hippocampal involvement on Magnetic Resonance Imaging (MRI), while a Dopamine Transporter (DaT) scan demonstrated reduced striatal uptake, confirming a dopaminergic deficit. The patient was managed with basal-bolus insulin therapy, corticosteroids for AIHA, and anti-tubercular treatment, while Parkinsonian symptoms improved with levodopa/carbidopa, trihexyphenidyl, and amantadine. Hypoglycaemia-induced neuronal injury results from metabolic failure, oxidative stress, excitotoxicity, and neuroinflammation, leading to selective neuronal necrosis, particularly affecting the basal ganglia, hippocampus, and substantia nigra. While some cases demonstrate reversible outcomes due to vasogenic oedema, others progress to irreversible neurodegeneration, emphasising the need for stringent glycaemic management and early recognition of neurological sequelae in diabetic patients.

Keywords: Loss of consciousness, Magnetic resonance imaging, Metabolic brain disorders, Movement disorders

CASE REPORT

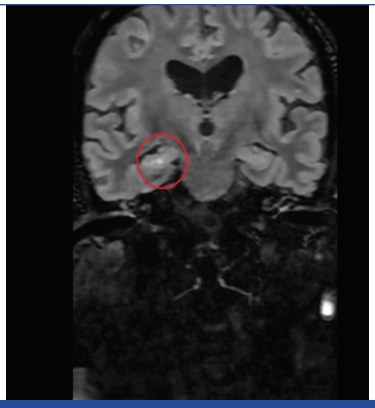
A 65-year-old male with a 12-year history of type 2 diabetes mellitus presented to the emergency department in a decreased responsive state. The patient was found unresponsive at home by his wife, who immediately called emergency services. The patient had a history of poorly controlled diabetes and his medication included metformin 500 mg twice daily and glimepiride 1 mg twice daily. The Haemoglobin A1c (HbA1c) level was recorded at 8.5%, reflecting poor long-term glycaemic control. He had experienced multiple hypoglycaemic episodes in the past year, characterised by confusion, sweating, and tremors, though none as severe as the current incident. Despite the duration of his diabetes, he exhibited no signs of microvascular complications such as retinopathy or nephropathy. Additionally, the patient had a history of AIHA, diagnosed 10 years ago and managed with intermittent steroid courses during haemolytic crises [Table/Fig-1].

Tuberculous encephalitis was suspected one month back due to presenting symptoms of progressive confusion, persistent fever, and neck stiffness, along with imaging findings on MRI of the brain with contrast revealed diffuse altered signal changes with increased signal intensity on T2-Fluid-Attenuated Inversion Recovery (T2-FLAIR) images in both hippocampi (right > left) and diffusion restriction in the same regions on Diffusion-Weighted Imaging (DWI) sequences [Table/Fig-2-4].

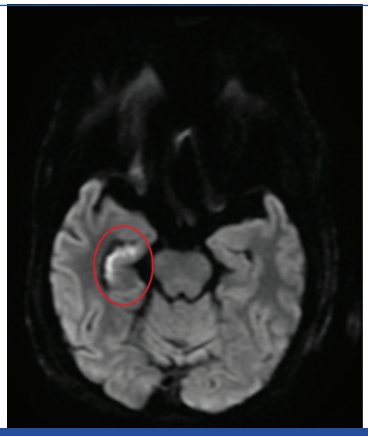
Cerebrospinal Fluid (CSF) analysis from a lumbar puncture also supported the tubercular pathology as it showed an elevated protein level (145 mg/dL), lymphocytic pleocytosis (98 cells/mm^3), and low glucose levels (32 mg/dL). These findings, combined with the endemic nature of tuberculosis in the patient's geographical area, prompted the empirical initiation of Anti-Tubercular Therapy (ATT) (isoniazid, rifampicin, pyrazinamide, and ethambutol) alongside corticosteroids (prednisolone) to address both the inflammatory response and the patient's AIHA. He recovered completely in

Crisis	Timeline	Trigger	Symptoms	Lab findings	Management	Outcome
First	10 years ago	None (initial diagnosis)	Fatigue, jaundice, dark urine, shortness of breath	Hb: 6.2 g/dL; Retics: 7%; Bilirubin: 4.5 mg/dL; Positive DAT	High-dose prednisolone; 2 units PRBC transfusion	Resolved in 2 weeks; Hb: 10 g/dL
Second	8 years ago	Viral upper respiratory infection	Severe fatigue, dizziness, worsening jaundice	Hb: 7.0 g/dL; Retics: 6.5%; Positive DAT	Prednisolone; No transfusion	Resolved in 3 weeks
Third	5 years ago	Stress (family bereavement)	Fatigue, palpitations, jaundice, mild chest discomfort	Hb: 6.8 g/dL; Retics: 8.1%; Positive DAT	Prednisolone; 1 unit PRBC transfusion; Psychosocial support	Resolved in 4 weeks; Hb: 9.5 g/dL
Fourth	3 years ago	Bacterial infection (fever, WBC ↑)	Jaundice, dark urine, pallor, fever, abdominal discomfort	Hb: 5.9 g/dL; Retics: 9.5%; Positive DAT	Prednisolone; Broad-spectrum antibiotics; 2 units PRBC transfusion	Resolved in 2 weeks; Hb: 10.1 g/dL
Fifth	1 year ago	Unknown	Fatigue, dizziness, dark urine	Hb: 7.3 g/dL; Retics: 8%; Positive DAT	Prednisolone; No transfusion	Resolved in 2 weeks

[Table/Fig-1]: Details of AIHA crises.
Hb: Haemoglobin; Retics: Reticulocytes; DAT: Direct antiglobulin test; PRBC: Packed red blood cells



[Table/Fig-2]: MRI Brain T2/FLAIR- Diffuse altered signal changes as evidenced by increased signal intensity on T2/FLAIR images in both the hippocampuses.



[Table/Fig-3]: MRI Brain DWI Diffusion restriction in both the hippocampuses (Right > Left).

Investigation	Findings	Remarks
MRI (T2/FLAIR)	Diffuse altered signal changes with increased signal intensity in both hippocampi (right > left)	Suggestive of inflammatory or infectious pathology
Diffusion-Weighted Imaging (DWI)	Diffusion restriction in both hippocampi (right > left)	Supports tuberculous encephalitis diagnosis
DaTscan	Reduced uptake in the striatum	Consistent with Parkinsonism
EEG	No epileptiform activity	Rules out seizures

[Table/Fig-4]: Radiological tests summary.
MRI: Magnetic resonance imaging; T2/FLAIR: MRI T2-fluid-attenuated inversion recovery; DaTscan: Dopamine transporter scan; EEG: Electroencephalography

two weeks and was discharged. Other comorbidities included hypertension, hyperlipidaemia, and coronary artery disease, managed with lisinopril, atorvastatin, and aspirin, respectively.

On arrival at the emergency department, the patient was drowsy but arousable to verbal stimuli. His vital signs included a blood pressure of 145/85 mmHg, a heart rate of 76 bpm, a respiratory rate of 14 breaths per minute, and oxygen saturation of 98% on room air. Initial laboratory findings revealed severe hypoglycaemia with a blood glucose level of 42 mg/dL [Table/Fig-5]. Intravenous glucose was administered, leading to partial improvement in consciousness.

During the hospital stay, approximately 48 hours after admission, the patient developed new motor symptoms, including tremors, muscle stiffness, and bradykinesia, which gradually became more pronounced. A neurological examination revealed a resting tremor in the right hand, cogwheel rigidity, bradykinesia, and postural instability, with difficulty maintaining balance. Cognitive assessment indicated mild cognitive impairment, as evidenced by a Mini-Mental State Examination (MMSE) score of 22/30. Non-motor symptoms included episodes of confusion, irritability, and depressive mood changes.

Further diagnostic workup included imaging and laboratory tests. A DaTscan demonstrated reduced uptake in the striatum, consistent

Investigation	Value	Reference range
Haemoglobin (g/dL)	10	11.5-15.0
Total leukocyte count (/µL)	9,400	4,000-10,000
Platelet count (/µL)	3,83,000	150,000-410,000
Mean corpuscular volume (fL)	79	78-98
Serum bilirubin (mg/dL)	0.21	0.2-1.20
Conjugated bilirubin (mg/dL)	0.11	Up to 0.5
Unconjugated bilirubin (mg/dL)	0.10	0.1-1.0
AST (U/L)	24	8-48
ALT (U/L)	29	7-55
Alkaline phosphatase (U/L)	38	40-130
Total protein (g/dL)	6.8	6.4-8.3
Serum albumin (g/dL)	4.9	3.5-5.2
Serum magnesium (mg/dL)	2.2	1.8-2.40
Serum phosphorus (mg/dL)	4.30	2.6-4.7
Serum sodium (mmol/L)	130.0	136-145
Serum potassium (mmol/L)	3.80	3.50-5.10
Serum chloride (mmol/L)	103	98-107
Urea (mg/dL)	29	17-49
Serum creatinine (mg/dL)	0.66	0.6-1.35
Serum calcium (mg/dL)	9.79	8.60-10.2
Prothrombin time (seconds)	13.20	10.24-12.71
INR	1.11	0.85-1.15
Vitamin D (ng/mL)	17.90	20-50
Total cholesterol (mg/dL)	199	<200
Triglycerides (mg/dL)	89	<150
HDL cholesterol (mg/dL)	50	>40
LDL cholesterol (mg/dL)	128	<100
Serum vitamin B12 (pg/mL)	290	180-890
HbA1c (%)	8.5	4-5.6%
CRP (mg/L)	9.0	>10.0
T3 (ng/mL)	1.49	0.64-1.52
T4 (µg/mL)	12.49	-11.7
TSH (microIU/mL)	1.71	0.4-4.68
Urine protein	Absent	Absent
Urine red blood cells	Absent	0-2 per hpf
Urine acetone	Absent	Absent
Urine glucose	Absent	Absent

[Table/Fig-5]: Laboratory tests.
AST: Aspartate transaminase; ALT: Alanine aminotransferase; INR: International normalised ratio; HDL: High-density lipoprotein; LDL: Low-density lipoprotein; HbA1c: Haemoglobin A1C; CRP: C-reactive protein; T3: Total thyroxine; T4: Total triiodothyronine; TSH: Thyroid-stimulating hormone

with Parkinsonism. An Electroencephalogram (EEG) ruled out seizures as a cause of altered mental status.

The management plan addressed multiple aspects of the patient's condition. Hypoglycaemia was acutely managed with intravenous glucose and close monitoring to prevent recurrence. The diabetes management regimen was adjusted by discontinuing metformin and glimepiride and initiating a basal-bolus insulin regimen. Steroid therapy was continued to manage AIHA, along with regular haemoglobin monitoring. Anti-tubercular treatment was maintained, with regular liver function tests to monitor for hepatotoxicity. For Parkinsonism, levodopa/carbidopa was initiated, along with trihexyphenidyl and amantadine to improve motor function and reduce rigidity. Physical therapy was incorporated to enhance mobility and balance.

Outcome and prognosis were closely monitored. Blood glucose levels stabilised with fewer hypoglycaemic episodes. Motor symptoms showed partial improvement with Parkinsonism treatment, though progression of the underlying neurodegenerative process was likely.

Cognitive function improved with better glycaemic control. The patient remained on ATT and required ongoing follow-up to manage his multifaceted health issues, including diabetes, AIHA, tuberculous encephalitis, and Parkinsonism.

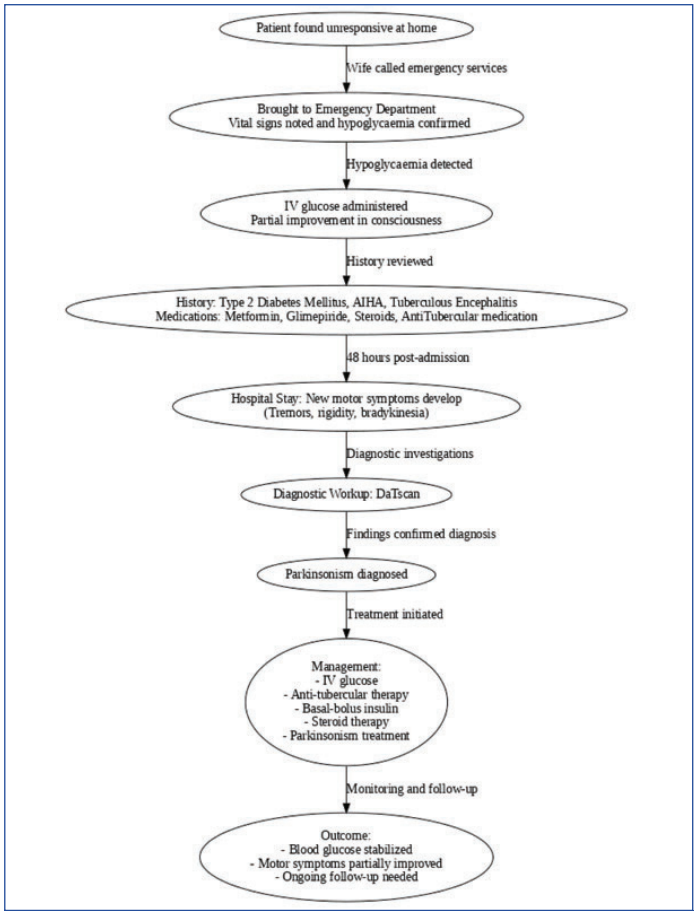
On follow-up, the patient demonstrated significant recovery and stabilisation over time. At two weeks post-discharge, his blood glucose levels were stable, and no further episodes of hypoglycaemia were reported. He tolerated the ATT well, with no signs of hepatotoxicity, and initial improvement in motor symptoms related to Parkinsonism was observed. At one month, continued stabilisation in glycaemic control was evident, and his haemoglobin levels confirmed that AIHA was well-controlled without the need for additional steroid courses. Neurologically, his motor symptoms showed further improvement, while his cognitive function remained stable, with an MMSE score of 28.

At three months, HbA1c levels indicated good glycaemic control, with no recurrence of haemolysis or worsening neurological symptoms. Liver function tests showed no adverse effects from ATT, and his cardiovascular parameters, including blood pressure and lipid levels, remained stable. By six months, the patient completed the intensive phase of ATT with sustained improvement in Parkinsonism symptoms, better balance, and mobility. His cognitive and cardiovascular health remained stable, and he reported satisfaction with his quality of life and daily activities.

At 12 months, the patient successfully completed the full course of ATT without recurrence of tuberculous encephalitis symptoms. His AIHA remained in remission, and his blood glucose levels consistently stayed within target ranges on basal bolus insulin regimen, with no hypoglycaemic episodes. Regular follow-ups every 3-6 months were planned to monitor for long-term complications and maintain his overall health. The patient's recovery highlights effective management of his complex medical conditions through a multidisciplinary approach [Table/Fig-6,7].

Drug	Dose	Frequency	Purpose/Comments
Isoniazid (H)	300 mg/day	Once daily	Anti-tubercular drug; supplemented with pyridoxine to prevent neuropathy.
Rifampicin (R)	600 mg/day	Once daily	Anti-tubercular drug; monitored for interactions and hepatotoxicity.
Pyrazinamide (Z)	1500 mg/day	Once daily	Anti-tubercular drug; monitor liver enzymes.
Ethambutol (E)	800 mg/day	Once daily	Anti-tubercular drug; monitor for optic neuritis.
Dexamethasone	12 mg/day (initial)	Taper gradually	Reduces central nervous system inflammation and intracranial pressure.
Metformin	500 mg	Twice daily	For Type 2 diabetes mellitus; discontinued due to recurrent hypoglycaemia.
Glimepiride	1 mg	Twice daily	For Type 2 diabetes mellitus; discontinued due to recurrent hypoglycaemia.
Levodopa/ Carbidopa	300 mg/day	Divided doses	For Parkinsonism; improves motor symptoms.
Trihexyphenidyl	2 mg/day	Twice daily	For Parkinsonism; reduces tremors and rigidity.
Amantadine	100 mg/day	Once daily	For Parkinsonism; helps manage dyskinesia.
Lisinopril	10 mg/day	Once daily	For hypertension; reduces blood pressure and cardiovascular risk.
Atorvastatin	20 mg/day	Once daily	For hyperlipidaemia; lowers cholesterol levels.
Aspirin	75 mg/day	Once daily	For coronary artery disease; antiplatelet therapy.
Prednisolone	40 mg/day	Once daily	For AIHA and inflammation; tapered gradually.

[Table/Fig-6]: List of medications.



[Table/Fig-7]: Clinical course and management.

DISCUSSION

Hypoglycaemia-induced neurological dysfunction results from a cascade of metabolic and neurochemical disturbances, ultimately leading to selective neuronal damage. In cases of severe hypoglycaemia, the glycolytic flux through the Embden-Meyerhof pathway is significantly reduced, thereby decreasing the Cerebral Metabolic Rate for glucose (CMRgl) [1]. This metabolic impairment disrupts oxidative phosphorylation, leading to an abrupt decline in Adenosine Triphosphate (ATP) levels and a subsequent rise in inorganic phosphate [2]. As the energy supply diminishes, neuronal survival becomes increasingly compromised, particularly in regions of the brain with high metabolic demands, such as the basal ganglia, hippocampus, and cerebral cortex.

A hallmark biochemical disturbance in hypoglycaemic brain injury is the excessive accumulation of excitatory amino acids, particularly aspartate, which contributes to excitotoxicity and neuronal damage. Due to the shortage of acetate required for condensation with oxaloacetate in the Krebs cycle, oxaloacetate accumulates, shifting the aspartate-glutamate transaminase reaction to the left. This shift results in a substantial increase in aspartate concentration, which spills over from intracellular stores into the extracellular space. The extracellular accumulation of aspartate, which can reach levels 1600% higher than normal, leads to neuronal hyperexcitation, excessive calcium influx, and excitotoxicity [3]. Unlike ischaemic brain injury, where glutamate accumulation drives excitotoxic damage, hypoglycaemia-induced neuronal injury is largely aspartate-mediated, explaining its distinct pathological features.

Studies have shown that hypoglycaemia-induced neuronal injury is linked to truncation of the Tricarboxylic Acid (TCA) cycle, shifting metabolism from a tricarboxylic to a dicarboxylic acid cycle [4]. This metabolic adaptation attempts to sustain neuronal survival by increasing reliance on aspartate transamination instead of glucose-derived pyruvate. As a result, the brain replenishes TCA cycle intermediates using glutamate and glutamine, leading to a relative increase in aspartate and a corresponding decrease in

glutamate/glutamine levels. This partial preservation of energy charge (25-30% of normal) delays neuronal death but does not prevent excitotoxic injury.

Histopathological findings in profound hypoglycaemia have shown selective neuronal necrosis in the cerebral cortex, hippocampus, and basal ganglia, with relative sparing of the brainstem [5]. Unlike ischaemic injury, which results in pannecrosis, hypoglycaemia preferentially affects neurons in highly metabolically active regions, leading to selective neuronal death while preserving surrounding glial and vascular structures. Hypoglycaemia has been shown to cause selective neuronal necrosis rather than ischaemic infarction, setting it apart from hypoxic-ischaemic injury, as demonstrated in neuropathological findings [5].

Neuroinflammation plays a crucial role in hypoglycaemia-induced neuronal damage. Hypoglycaemia triggers a strong proinflammatory response, with elevated levels of Tumour Necrosis Factor-alpha (TNF-α), Interleukin-1 (IL-1), Interleukin-6 (IL-6), and Interferon-γ (IFN-γ), leading to Blood-Brain Barrier (BBB) disruption and increased neuronal vulnerability [6]. The activation of microglia and astrocytes in hypoglycaemic states can exacerbate oxidative stress, contributing to long-term neurodegeneration, including Parkinsonism. Additionally, inflammatory cytokines disrupt endothelial function, increasing the risk of stroke, cerebral ischaemia, and chronic neuronal loss.

Cases of hypoglycaemia-induced Parkinsonism with reversible basal ganglia lesions have been documented [7]. MRI studies have shown that DWI abnormalities in hypoglycaemic encephalopathy often present as bilateral symmetrical lesions in the basal ganglia and cerebral cortex, with varying degrees of white matter involvement [8]. The extent of these abnormalities correlates with clinical outcomes, where widespread cortical and basal ganglia involvement is associated with persistent vegetative states, whereas selective white matter changes (such as splenial lesions) may indicate a more reversible injury pattern [8]. A 77-year-old diabetic patient who developed progressive gait disturbances, rigidity, and bradykinesia following hypoglycaemia showed bilateral basal ganglia hyperintensity on T2-weighted Imaging (T2WI) and DWI with an elevated Apparent Diffusion Coefficient (ADC) [7]. The patient's symptoms improved following the discontinuation of sulfonylureas, with MRI showing resolution of basal ganglia lesions within eight months. This suggests that in certain cases, vasogenic oedema rather than cytotoxic injury may play a role in reversible hypoglycaemia-induced Parkinsonism.

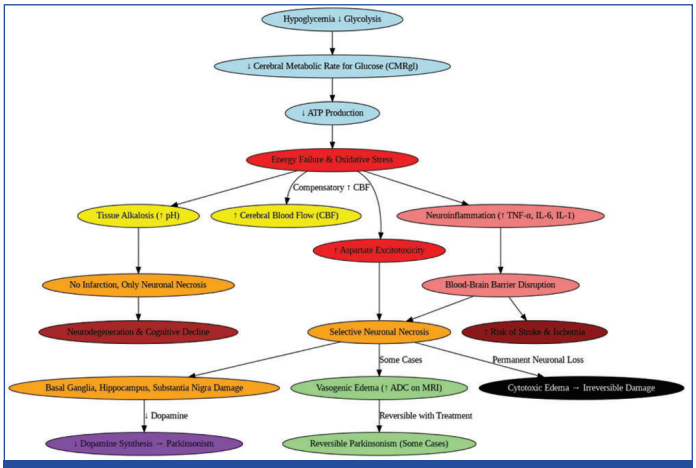
Another crucial aspect differentiating hypoglycaemic encephalopathy from ischaemic injury is the development of profound tissue alkalosis. In ischaemia, intracellular acidosis results from the accumulation of lactic acid due to anaerobic metabolism. However, in hypoglycaemia, lactate production is significantly reduced due to impaired glycolysis. Furthermore, the breakdown of proteins and deamination of amino acids generates excess ammonia, a potent base that further drives tissue pH towards alkalinity. This biochemical shift increases cellular pH to approximately 7.5, contrasting with the acidosis observed in ischaemic conditions [9]. The inability of hypoglycaemic brain tissue to generate sufficient lactate prevents infarction, leading instead to selective neuronal necrosis without the widespread pannecrosis typically seen in ischaemia.

Despite the drastic reduction in glucose availability, the brain attempts to sustain its metabolic functions through alternative energy substrates, including lactate, ketone bodies, and endogenous fatty acids [10]. However, these substrates are insufficient to fully compensate for the loss of glucose-derived ATP. The cellular redox state also shifts towards oxidation, as seen in the altered equilibrium of lactate/pyruvate, Nicotinamide adenine dinucleotide (NAD/NADH), and glutathione redox pairs [11]. Whether these oxidative imbalances contribute to long-term neurodegeneration remains an area of ongoing research.

Interestingly, Cerebral Blood Flow (CBF) increases in response to profound hypoglycaemia, possibly as a compensatory mechanism to enhance nutrient delivery to the brain [1]. Despite this increase, the Cerebral Metabolic Rate for Oxygen (CMRO2) remains relatively stable, suggesting the utilisation of alternative substrates for oxidative metabolism [12]. However, the inflammatory response to hypoglycaemia can further exacerbate BBB permeability through upregulated adhesion molecules {Intercellular Adhesion Molecule-1 (ICAM-1), Vascular Cell Adhesion Molecule-1 (VCAM-1)} and Matrix Metalloproteinase-9 (MMP-9), increasing the risk of delayed neuronal injury and impaired recovery [6].

Delayed recovery in hypoglycaemic brain injury extends beyond the acute phase. After glucose restoration, purine metabolism remains altered for up to 24 hours, and full metabolic recovery is delayed due to persistent abnormalities in fatty acid and branched-chain amino acid metabolism [4]. These long-term metabolic disturbances may contribute to neuronal membrane instability and delayed neurodegeneration, predisposing individuals to persistent movement disorders such as Parkinsonism.

[Table/Fig-8] illustrates the metabolic, inflammatory, and vascular pathways leading to hypoglycaemia-induced neuronal damage and Parkinsonism. This model incorporates mechanisms such as excitotoxicity (3), neuroinflammation (6), and reversible vs. irreversible basal ganglia injury [5,8].



[Table/Fig-8]: Pathophysiological mechanisms of hypoglycaemia-induced neuronal injury and parkinsonism.

CONCLUSION(S)

Hypoglycaemia-induced Parkinsonism represents a rare but significant neurological complication of metabolic brain dysfunction. The interplay of metabolic failure, excitotoxicity, neuroinflammation, and vascular compromise highlights the complexity of hypoglycaemic brain injury. While some cases demonstrate reversible outcomes due to vasogenic oedema, others progress to irreversible neurodegeneration, emphasising the critical need for early recognition and intervention. Glycaemic management remains the cornerstone of prevention, with a focus on avoiding recurrent hypoglycaemia, particularly in elderly diabetics and those on sulfonylureas or insulin therapy.

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