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Original Research Article

A STUDY ON CLINICAL PROFILE AND AETIOLOGICAL CORRELATION OF BITHALAMIC LESIONS

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ABSTRACT

Background: Bithalamic lesions are uncommon yet clinically significant because of the thalamus's central role in cognition, consciousness, and sensorimotor integration. Bilateral involvement often produces varied and nonspecific neurological manifestations, complicating the early aetiological diagnosis. This study aimed to analyse the clinical profile, neuroimaging patterns, and aetiological spectrum of bithalamic lesions and associate clinical presentations with underlying causes and outcomes.

Materials and Methods: This retrospective descriptive study was conducted in the Department of Neurology at a tertiary care centre from December 2023 to November 2025. A total of 26 patients with neurological symptoms and bilateral thalamic involvement on brain CT or MRI were included. Demographic details, clinical presentations, neuroimaging patterns, aetiological diagnoses, and clinical outcomes were analysed. Data were summarised using descriptive statistics and expressed as frequencies and percentages.

Results: Most patients were aged <30 years (84.6%) and were female (53.8%). Wilson's disease was the most common aetiology (38.5%), followed by Wernicke's encephalopathy (15.4%), deep cerebral venous thrombosis, and Fahr's disease (11.5% each). Altered sensorium or encephalopathy was the most frequent clinical presentation (53.8%), followed by movement disorders (46.2% of patients). Bilateral thalamic T2/FLAIR hyperintensity was observed in all patients, whereas diffusion restriction was noted in 34.6% of patients. Treatable or potentially reversible conditions accounted for 73.1% of the cases. Mortality was observed in 15.4% of patients, predominantly in those with progressive neurodegenerative and mitochondrial disorders.

Conclusion: Bithalamic lesions demonstrate heterogeneous clinical and aetiological profiles. Neuroimaging plays a central role in the early diagnosis and aetiological differentiation of these conditions, facilitating timely intervention in reversible conditions and potentially improving clinical outcomes.

Keywords: Diagnostic Imaging, Magnetic Resonance Imaging, Nervous System Diseases, Thalamus, Computed Tomography

INTRODUCTION

The thalamus is a vital structure that plays a central role in sensory integration, motor control, cognition, behaviour, memory, and regulation of consciousness and the sleep–wake cycle.[1] Owing to its strategic anatomical location and extensive cortico-subcortical connectivity, thalamic involvement can result in a wide spectrum of neurological manifestations.[2] Lesions affecting both thalami simultaneously are relatively uncommon but are clinically significant because they often present with acute or subacute encephalopathy, movement disorders, visual disturbances, or cognitive impairment, frequently posing diagnostic challenges in routine neurological practice.[3]

Bithalamic lesions may arise from a diverse range of aetiologies, including vascular causes such as Artery of Percheron (AOP) infarction and deep cerebral venous thrombosis, metabolic and nutritional

disorders such as Wernicke's encephalopathy and Wilson's disease, infectious and post-infectious conditions, degenerative disorders such as Creutzfeldt–Jakob disease, and rare genetic or mitochondrial disorders such as Fahr and Leigh diseases.[3,4] Although the differential diagnosis of bilateral thalamic involvement is limited, overlap in clinical presentation often makes early aetiological distinction difficult. Delayed or incorrect diagnosis can result in significant morbidity or mortality, particularly in potentially reversible conditions.[5]

Computed tomography (CT) is useful in identifying calcifications and haemorrhage, while magnetic resonance imaging (MRI), including diffusion-weighted imaging and fluid-attenuated inversion recovery sequences, provides superior characterisation of lesion patterns.[6,7] Recognition of specific imaging signatures, when interpreted in conjunction with clinical features and laboratory findings, can help narrow the differential diagnosis and guide timely management.[8] Early identification is crucial in treatable conditions such as Wernicke's encephalopathy, deep venous thrombosis, and Wilson's disease, where prompt intervention can significantly improve outcomes.[9]

Despite the clinical importance of bithalamic lesions, systematic studies correlating clinical presentation with underlying aetiology remain limited, particularly in resource-constrained settings. Most of the available literature consists of isolated case reports or small case series focusing on individual conditions. The present study was undertaken to analyse the clinical profile, neuroimaging characteristics, and aetiological spectrum of patients presenting with bithalamic lesions and to correlate clinical presentation with underlying causes and outcomes. This study aims to provide a comprehensive descriptive overview that may assist clinicians in the early recognition, appropriate investigation, and optimal management of patients with bithalamic involvement.

MATERIALS AND METHODS

This retrospective descriptive study was conducted on patients with bithalamic lesions on neuroimaging at the Department of Neurology, Kalaignar Centenary Super Speciality Hospital (KCSSH), Guindy, Chennai, a tertiary care referral centre, from December 2023 to November 2025.

Inclusion criteria

Patients of any age and sex with neurological symptoms and bilateral thalamic involvement on CT or MRI, evaluated in the Neurology outpatient department, or admitted to the Neurology wards during the study period were included.

Exclusion criteria

Patients with unilateral thalamic lesions, poor-quality or incomplete neuroimaging, incomplete clinical records, or imaging in which bithalamic involvement could not be clearly established were excluded.

Methods: Clinical data were obtained from hospital records and included demographic details such as age and sex, presenting neurological symptoms, and the clinical course. Patients were categorised into age groups as young (<30 years) and elderly (≥60 years) for descriptive analysis.

Neuroimaging findings were reviewed for all the patients. The imaging modalities included brain CT and MRI. The assessed imaging characteristics included bilateral thalamic involvement in like diffusion restriction (vascular infarction), deep cerebral venous thrombosis, calcification, and associated brainstem or periaqueductal involvement. Aetiological classification was based on established clinical, laboratory, and neuroimaging criteria documented in the medical records.

Clinical outcomes at the last available follow-up were classified into four mutually exclusive categories: 1. Complete recovery (full resolution of neurological symptoms), 2. Partial recovery (clinical improvement without full resolution), 3. No significant improvement or stable course (persistent neurological deficits without meaningful change), and 4. Mortality (death attributable to the primary neurological disease).

Statistical analysis: Data were analyszed performed using **SPSS v29** standard statistical software Categorical variables are expressed as frequencies and percentages and are presented in tables. No inferential statistical analysis was performed as the study was descriptive. Missing or incomplete data were excluded from the analysis.

RESULTS

A total of 26 patients who met the inclusion criteria were identified and included in the analysis. Patients aged <30 years comprised 21 young adults and one paediatric patient, accounting for 22 patients (84.6%), while elderly patients (≥60 years) constituted 4 patients (15.4%). Females constituted 14 patients (53.8%) and males 12 patients (46.2%) [Table 1].

Table 1: Sex distribution of patients with bithalamic lesions

Demographic data	Category	n (%)
Sex	Female	14 (53.8%)
	Male	12 (46.2%)

Among the cohort, Wilsons disease was the most common aetiology, observed in 10 patients (38.5%) followed by Wernicke's encephalopathy in 4 patients (15.4%).

Deep cerebral venous thrombosis and Fahr's disease were identified in 3 patients each (11.5% each). Artery of Percheron infarct and Creutzfeldt–Jakob disease was noted in 2 patients each (7.7%). post-encephalitic sequelae (Japanese encephalitis) and Leigh disease were observed in one patient each (3.8%) [Table 2].

Table 2: Aetiological distribution of bithalamic lesions

Aetiology	n (%)
Wilson's disease	10 (38.5%)
Wernicke's encephalopathy	4 (15.4%)
Deep cerebral venous thrombosis	3 (11.5%)
Fahr's disease	3 (11.5%)
Artery of Percheron infarct	2 (7.7%)
Creutzfeldt–Jakob disease	2 (7.7%)
Post-encephalitic sequelae (JE)	1 (3.8%)
Leigh disease	1 (3.8%)

Altered sensorium or encephalopathy was observed in 14 (53.8%) patients, followed by movement disorders (dystonia, tremors or myoclonus) in 12 (46.2%) patients. Ataxia or gait disturbance was present in 9 (34.6%) patients. Seizures and cognitive or behavioural changes were observed in six (23.1%) patients [Table 3]. Visual symptoms or ophthalmoplegia were present in 8 (30.8%) patients, with retinal haemorrhages and optic disc hyperaemia observed in patients with Wernicke's encephalopathy (Figure 1).

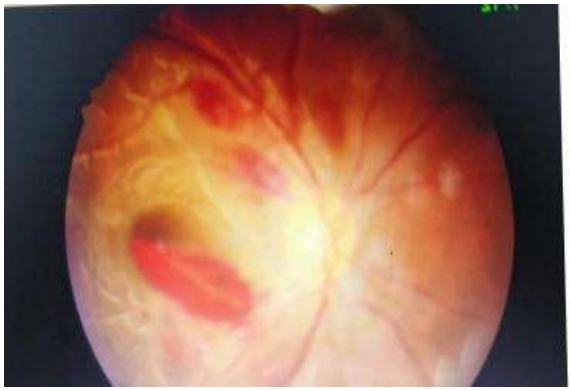

Figure 1: Fundus photograph showing multiple retinal haemorrhages with optic disc hyperaemia in a patient with Wernicke's encephalopathy

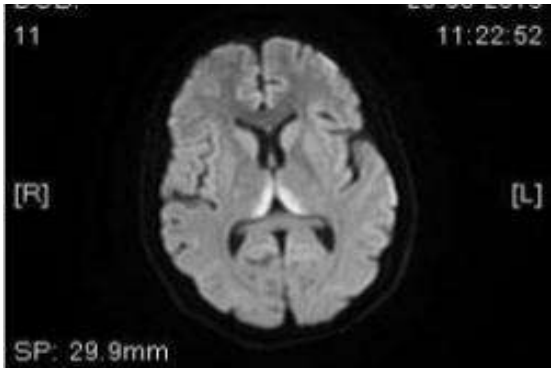

Figure 2: Bithalamic lesions in a patient with Wernicke's encephalopathy associated with retinal haemorrhages on fundoscopy

Figure 3: T2 hyperintensities in the periaqueductal grey matter and bithalamic lesions in a patient with Wernicke's encephalopathy

Table 3: Clinical presentation of patients with bithalamic lesions

Clinical Presentation	n (%)
Altered sensorium/encephalopathy	14 (53.8%)
Movement disorders (dystonia, tremor, myoclonus)	12 (46.2%)
Ataxia/gait disturbance	9 (34.6%)
Visual symptoms/ophthalmoplegia	8 (30.8%)
Seizures	6 (23.1%)
Cognitive/behavioural changes	6 (23.1%)

Bilateral thalamic T2/FLAIR hyperintensity was observed in all 26 patients (100%) (Figure 2). Diffusion restriction was observed in nine (34.6%) patients. Deep cerebral venous thrombosis and calcification was noted on CT in 3 patients each (11.5% each). Vascular infarction due to AOP involvement was noted in 2 patients (7.7%) [Table 4]. Brainstem or periaqueductal involvement was observed in 6 patients (23.1%), a characteristic finding in Wernicke's encephalopathy (Figure 3).

Table 4: Neuroimaging patterns in bithalamic lesions

Imaging Pattern	n (%)
Bilateral thalamic T2/FLAIR hyperintensity	26 (100%)
Diffusion restriction	9 (34.6%)
Deep venous thrombosis	3 (11.5%)
Vascular infarction (AOP)	2 (7.7%)
Calcification on CT	3 (11.5%)
Brainstem / periaqueductal involvement	6 (23.1%)

Treatable or potentially reversible conditions were observed in 19 (73.1%) patients, while progressive or fatal conditions were observed in 7 (26.9%) patients.

Based on the final clinical assessment, complete recovery was observed in 9 patients (34.6%), partial recovery in 10 patients (38.5%), no significant improvement/stable course in three patients (11.5%), and mortality in four patients (15.4%) [Table 5].

Table 5: Clinical outcome and treatability profile of patients with bithalamic lesions

Parameter	Category	n (%)
Treatability	Treatable / potentially reversible	19 (73.1%)
	Progressive/fatal	7 (26.9%)
Clinical outcome	Complete recovery	9 (34.6%)
	Partial recovery	10 (38.5%)
	No significant improvement / stable course	3 (11.5%)
	Mortality	4 (15.4%)

DISCUSSION

This study demonstrates that bithalamic lesions predominantly affect younger patients, are most commonly attributable to metabolic and vascular aetiologies, and frequently present with encephalopathy, with outcomes largely determined by the underlying cause. Variations in age and sex distribution across studies likely reflect differences in underlying aetiology, referral patterns, and study design rather than true epidemiological divergence. Bezerra et al. reported 2 cases of bilateral thalamic lesions due to deep cerebral venous thrombosis, both in female patients aged 18 and 49 years.[10] In a larger cohort, Gogineni et al. analysed 12 patients with deep cerebral venous thrombosis and reported a mean age of 32.25 ± 11.42 years, though with a contrasting male predominance (75%; 9 males and 3 females), again highlighting the predominance of younger age groups.[11] For metabolic aetiologies, Patel et al. described 4 adolescent patients with Wilson's disease, equally distributed between males and females, with ages ranging from 13 to 18 years.[12]

In our study, Wilson's disease was the most common aetiological factor, followed by Wernicke's encephalopathy, with vascular and metabolic causes, such as deep cerebral venous thrombosis and Fahr's disease, also contributing significantly. Less frequent aetiologies include AOP infarction, Creutzfeldt–Jakob disease, post-encephalitic sequelae of Japanese encephalitis, and Leigh disease. Supporting the vascular spectrum, Bezerra et al. identified deep cerebral venous thrombosis as the sole aetiology in 2 reported cases, with bilateral thalamic involvement confirmed on MRI and venous angiography.[10] Similarly, Gogineni et al. reported deep cerebral venous thrombosis as the exclusive cause in 100% (12/12) of their cohort, noting that deep venous system involvement constituted only 10.9% of all CVT cases at their centre, highlighting its rarity.[11] In contrast, isolated arterial causes have also been documented, as Bhattarai et al. reported AOP infarction as the sole aetiology in 100% (1/1) of their case,[13] while Wu et al. described bilateral anterior thalamic symmetrical infarction due to acute cerebral infarction, related to thalamic tubercular artery involvement rather than classic AOP occlusion.[14] From a metabolic perspective, Li et al. studied 33 patients with Wilson's disease and found abnormal thalamic signals on conventional MRI in 15 (45.5%) patients, whereas 18 (54.5%) had a normal-appearing thalamus.[15] These findings highlight the heterogeneous aetiological spectrum of bithalamic lesions, with metabolic and vascular causes predominating across studies.

In our study, altered sensorium or encephalopathy was the most common clinical presentation, with frequent occurrence of movement disorders, ataxia or gait disturbances, visual symptoms including ophthalmoplegia, seizures, and cognitive or behavioural changes. In line with these findings, Bezerra et al. reported that both patients presented with acute-onset headache and vomiting, followed by altered mental status characterised by abulia and prominent frontal release signs.[10]

Bhattarai et al. described a patient presenting with acute loss of consciousness, severe motor impairment, oculomotor abnormalities, and hemiplegia, consistent with encephalopathy and focal neurological deficits.[13] The clinical spectrum in a larger cohort, Kim et al. found that 17 out of 50 (34%) patients were neurologically symptomatic, with dysarthria being the most frequent manifestation, followed by tremor, dystonia, seizures, chorea, and psychiatric disturbances, and neurological symptoms present in 91.7% (11/12) of patients with T2-weighted signal abnormalities, indicating strong clinicoradiological correlation.[16] In vascular aetiologies, Gogineni et al. reported headache in 91.7% (11/12) of patients, seizures in 33.3% (4/12), and visual disturbances in 16.7% (2/12).[11] Studies demonstrate that altered consciousness and diverse neurological manifestations are hallmark clinical features of bithalamic involvement.

In our study, bilateral thalamic T2/FLAIR hyperintensity was the uniform imaging feature, with diffusion restriction and vascular or calcific changes observed in a subset of patients, occasional AOP infarction, and associated brainstem or periaqueductal involvement in some cases. In arterial infarction patterns, Bhattarai et al. demonstrated bilateral thalamic hypodensity on CT in 100% (1/1) of cases, involving the paramedian thalami with associated rostral midbrain involvement, consistent with AOP infarction.[13] Supporting venous aetiologies, Bezerra et al. demonstrated bilateral

thalamic hyperintensities on T2-weighted MRI in both patients, with deep cerebral venous thrombosis confirmed on venous MR angiography.[10] Kim et al. reported abnormal MR findings in 54% (27/50) of patients, with thalamic involvement seen in 50% (6/12) of those with T2-weighted abnormalities, along with frequent bilateral and symmetric involvement of the putamen (83%), caudate nucleus (67%), globus pallidus (58%), and midbrain (42%).[16]

Donohoe et al. demonstrated bilateral thalamic hypodensities on non-contrast CT and corresponding infarcts on MRI in 100% (1/1) of cases.[17] In venous thrombosis cohorts, Gogineni et al. reported thalamic involvement in 41.7% (5/12) of patients, frequently bilateral, with hemorrhagic infarctions in 75% (9/12) and MR venographic involvement of the internal cerebral vein and/or vein of Galen in 100% (12/12).[11] Complementing these findings, Wu et al. demonstrated bilateral symmetrical anterior thalamic hyperintensities on DWI, T2-weighted, and FLAIR MRI sequences in 100% (1/1) of cases, with corresponding CT hypodensities.[14] These findings indicate that bilateral and often symmetric thalamic imaging abnormalities are a consistent characteristic of diverse bithalamic pathologies.

In our study, the majority of cases were attributable to treatable or potentially reversible conditions, whereas a smaller proportion followed a progressive or fatal course. Clinical improvement was observed in some patients during follow-up, whereas others demonstrated a stable or variable trajectory, and mortality was confined to those with progressive neurodegenerative or mitochondrial disorders. In keeping with favourable outcomes in reversible conditions, Kim et al reported radiological improvement in 60% (9/15) after chelation therapy, with 67% (6/9) of patients with T2-weighted abnormalities showing improvement, a strong correlation between imaging and clinical response (Kendall tau-b = 0.755, $P < 0.001$), and radiological progression in only 11% (1/9).[16] Similarly, Patel et al. documented clinical improvement in 50% (2/4) of patients treated with zinc and penicillamine, while the remaining 50% (2/4) showed poor or no improvement.[12] In vascular aetiologies, outcomes were uniformly favourable, as Gogineni et al. reported improvement in 100% of patients, with modified Rankin Scale scores improving from 3–4 at baseline to 0–1 at three-month follow-up.[11] Prognosis correlates closely with etiology, favouring early recognition of reversible causes.

Strengths: The strengths of this study include the systematic clinical–radiological correlation, inclusion of a broad aetiological spectrum, and analysis of real-world data from a tertiary referral centre. The uniform use of neuroimaging for diagnosis has enabled consistent classification of bithalamic involvement across patients.

Limitations: This study was limited by its single-centre, retrospective design, which may restrict the generalisability of the findings. In addition, the descriptive nature and lack of long-term follow-up limited the assessment of disease progression and long-term outcomes across different aetiologies.

CONCLUSION

This study elucidates the heterogeneous clinical presentations and aetiological spectrum of bithalamic lesions, with metabolic and vascular causes predominating in younger patients and neurodegenerative disorders more prevalent in elderly. Neuroimaging is central to early aetiological identification, facilitating timely management of potentially reversible conditions. Future multicentre prospective studies with longer follow-up periods are required to refine diagnostic approaches and improve outcome prediction.

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