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Shafaq Saleem
Aga Khan University,Hospital,Karachi

Muhammad Saleem
Aga Khan University Hospital Karachi

Fatima Mubarak
Aga Khan University Hospital,Karachi

Salman Farooq
Khoula Hospital, Muscat, Oman

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CAUSES AND PRESENTATIONS OF BILATERAL THALAMIC LESIONS: A RETROSPECTIVE CROSS-SECTIONAL STUDY FROM A TERTIARY CARE HOSPITAL

Shafaq Saleem[1], Muhammad Saleem[1], Fatima Mubarak[2], Salman Farooq[3]
[1]. Section of Neurology, Department of Medicine, Aga Khan University Hospital
[2]. Radiology Department, Aga Khan University Hospital
[3]. Neurology Department, Khoula Hospital, Muscat, Oman

Corresponding author: Shafaq Saleem *Section of Neurology, Department of Medicine, Aga Khan University Hospital* ***Email:*** *drshafaqsaleem@gmail.com*

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ABSTRACT

Background and objective:

Bilateral thalamic lesions present a significant diagnostic and therapeutic challenge due to their critical role in sensory, motor, and cognitive functions. The objective of this study was to study the causes and presentations of bilateral thalamic lesions among patients presenting to a tertiary care hospital.

Methods:

This retrospective study reviewed 40 patients aged 18–80 years admitted to Aga Khan University Hospital between 2006 and 2023 with MRI-confirmed bilateral thalamic lesions. Data collected included demographics, clinical presentations, radiological findings, and final diagnoses. The data was analyzed through SPSS 22.0.

Results:

Of the 40 cases analyzed, the most common etiology was Artery of Percheron (AOP) infarction (27.5%), followed by Wernicke's encephalopathy (20%), Carbon monoxide poisoning (15%), lymphoma (12.5%), Fahr disease (12.5%), Cerebral Venous Sinus Thrombosis (7.5%), and Wilson's disease (5%). Clinical presentations included altered levels of consciousness, seizures, headache, and vertigo, with MRI revealing distinct patterns for each condition.

Conclusion:

Bilateral thalamic lesions encompass a wide range of etiologies, each influencing management and prognosis differently. This study underscores the need for precise radiological evaluation and clinical correlation to guide effective treatment strategies.

Keywords: Thalamus, Lymphoma, Vein of Galen, Wernicke's encephalopathy, Carbon monoxide

INTRODUCTION

The thalamus, a key gray matter structure within the dorsal diencephalon, plays a central role in relaying sensory, motor, and cognitive signals. Thalamic dysfunction has been linked to several forms of dementia, including vascular dementia, where strategic infarcts can lead to memory loss, apathy, and language deficits. In Alzheimer's disease degeneration of the anterior thalamic nuclei contributes to episodic memory impairment. In frontotemporal dementia, thalamic involvement may affect emotional regulation and behavior. Damage to thalamic regions can also result in emotional disturbances such as apathy and impaired recognition of negative emotions. Furthermore, lesions in the dominant thalamus may lead to thalamic aphasia, typically presenting as transcortical sensory or motor aphasia, with fluent or non-fluent speech and impaired naming or initiation.[1-3.]

Bilateral thalamic lesions, although infrequent, represent a particularly challenging subset of neurological disorders due to their impact on neurocognitive and sensorimotor pathways, which can result in severe clinical presentations, including altered consciousness, movement abnormalities, and deficits in memory and executive function.

The causes of such lesions are broad, spanning vascular, infectious, metabolic, toxic, and genetic etiologies.[1,4,5] Vascular lesions including ischemic lesions and bleeds, especially those stemming from an Artery of Percheron (AOP) infarction, are uniquely notable because this variant of the posterior cerebral circulation supplies both thalami from a single arterial source, creating a high risk for symmetrical thalamic involvement upon occlusion.[6]

Recent advances in neuroimaging, particularly high-resolution MRI techniques, have enhanced our ability to detect and characterize bilateral thalamic lesions early, enabling differentiation between potential causes. Diffusion-weighted imaging (DWI), Susceptibility-weighted imaging (SWI), and fluid-attenuated inversion recovery (FLAIR) sequences, for instance, facilitate distinguishing ischemic, infectious, and metabolic patterns, thereby guiding etiology-specific management strategies. This precision is particularly crucial as outcomes for patients with bilateral thalamic lesions vary significantly based on the underlying cause and timely intervention.

This study aims to identify the different etiologies associated with bilateral thalamic involvement and compare them with clinical and radiological features.

METHODS

This was a retrospective observational study conducted at Aga Khan University Hospital (AKUH), Karachi, enrolling patients admitted with bilateral thalamic lesions between January 2006 and December 2023. The study was exempted by the Aga Khan University Hospital (AKUH) Institutional Ethical Review Board under reference number 2024-9344-28276. All protocols adhered to ethical standards in data handling and patient confidentiality.

The study included patients with bilateral thalamic lesions identified on magnetic resonance imaging (MRI) between 18-80 years old. The study included 40 patients who were hospitalized for symptoms related to their thalamic findings. Data collected included age, clinical symptoms, neuroimaging, and diagnoses.

Patient records provided data on neurological symptoms, treatment, and MRI findings. MRI was the primary imaging modality, with T1, T2, FLAIR, and diffusion-weighted sequences for all patients and Magnetic Resonance Spectroscopy (MRS) in select cases. A senior and highly experienced neuro radiologist in their field assessed lesion characteristics, including location and signal intensity, and made the radiological diagnosis.

The data was analyzed for etiologies and clinical presentations using SPSS 22.0.

RESULTS

A total of 40 patients were included in this study with a male predominance of 60%. The distribution of diagnoses among patients with bilateral thalamic lesions revealed a variety of etiologies, including vascular, metabolic, toxic, and genetic causes.

The most frequent diagnosis was Artery of Percheron (AOP) occlusion, which accounted for 11 cases (27.5%). Patients with AOP occlusion primarily presented with altered level of consciousness (ALOC), headache, and vertigo. The second most common diagnosis was Wernicke's encephalopathy, identified in eight cases (20%). These patients presented with a combination of ALOC, headache, hand tremors, lethargy, and seizure-like activity. Carbon monoxide (CO) poisoning accounted for six cases (15%), with seizure and ALOC as the primary symptoms. Additional etiologies included bilateral thalamic lymphoma (n=5, 12.5%), Fahr disease (n=5, 12.5%), cerebral venous sinus thrombosis (CVST) (n=3, 7.5%), and Wilson disease (n=2, 5%). Notably, patients with bilateral thalamic lymphoma presented with ALOC alone, while those with Fahr disease exhibited a broader array of symptoms, including fever, decreased oral intake, sensory symptoms, and delayed menarche (Table 1). Dysarthria with deranged liver function tests was distinctive in patients diagnosed with Wilson disease.

TABLE I: SYMPTOMATOLOGY IN THALAMIC INVOLVEMENT							
S.no	Age range	Symptoms	Duration of symptoms	Diagnosis	Lesion Type	Clinical Follow-up	Radiology Follow-up Scan
1	18-80	ALOC Headache vertigo	4-24 hours	Artery of Percheron occlusion	Ischemic	Improved	Not available
2	18-80	ALOC Seizure	12 hours	Carbon monoxide poisoning	Hypoxic ischemic	Improved	Improved
3.	18-80	ALOC	2 days	Bilateral Lymphoma	Tumor	Static	Improved
4.	18-80	ALOC Decreased oral intake Sensory symptoms Delayed menarche	3-4 days	Fahr disease	Ischemia	Static	Static
5.	18-80	Headache ALOC	2 days	CVST	Ischemia	Improved	Not available
6.	18-80	Headache ALOC Hand tremors Lethargy Seizure	24-48 hours	Wernicke encephalopathy	Hypoxic ischemic	Improved	Improved
7.	18-80	Dysarthria /ALOC with deranged LFTs	1-2 months	Wilson disease	Ischemic	Static	Static

ALOC: Altered level of consciousness, LFTs: Liver function tests, CVST: Cerebral venous sinus thrombosis

Magnetic resonance imaging (MRI) radiologically confirmed bilateral thalamic involvement and delineated specific etiological patterns. Diffusion-weighted imaging (DWI) and fluid-attenuated inversion recovery (FLAIR) sequences were particularly valuable, aiding in differentiating ischemic from non-ischemic causes of bilateral thalamic lesions. Patients with AOP occlusion showed characteristic bilateral paramedian thalamic infarcts, while those with Wernicke's encephalopathy exhibited symmetric thalamic hyperintensities on FLAIR imaging. In cases of CO poisoning, MRI revealed hypoxic-ischemic changes, predominantly affecting the bilateral thalami (Table II).

Table II: MRI Features in Thalamic Involvement

S.No.	Diagnosis	T1 weighted Sequence	T2 weighted Sequence	FLAIR Sequence	T1/FLAIR Contrast Sequence (Y/N)	Diffusion weighted Sequence
1.	Artery of Percheron occlusion	Hypointense /Normal	Hyperintense	Hyperintense	No	Diffusion restriction in affeted thalamic area.
2.	Carbon monoxide poisoning	Hypointense /Normal	Hyperintense	Hyperintense	No	Diffusion restriction in basal ganglia and thalamus.
3.	Bilateral Lymphoma	Isointense /Hypointense	Hyperintense	Hyperintense	Yes	Restricted diffusion in the tumor area.
4.	Fahr disease	Hypointense	Hyperintense	Hyperintense	No	Negative Diffusion restriction
5.	CVST	Hypointense/Normal	Hyperintense	Hyperintense	Yes	Restricted diffusion in infarcted venous areas
6.	Wernicke encephalopathy	Hypointense	Hyperintense	Hyperintense	No	Restricte diffusion in thalamus and mammilary bodies.
7.	Wilson disease	Hypointense	Hyperintense	Hyperintense	No	Negative diffusion restriction.

MRI: Magnetic Resonance Imaging, DWI: Diffusion -weighted Image, FLAIR: Fluid-attenuated inversion recovery, AOP: Artery of Percheron, MRS: Magnetic Resonance Spectroscopy

DISCUSSION

The thalamus is frequently involved in various pathologies whether infective, vascular, inflammatory, or neoplastic. Being the vital structure involved in sensory, motor, and cognitive function, patients' presentation is similar with a different diagnosis. [6,7]

Our 17-year duration data showed ischemic insult to be a frequent cause of bilateral thalamus involvement due to artery of Percheron infarction. Overall, this uncommon condition accounts for 0.6% of all strokes.8 Supply to the thalamus is variable from anterior and posterior circulation via small vessels, commonly compromised due to small vessel disease followed by cardioembolic phemomena.[9,10] CVST caused by the vein of Galen occlusion is another cause involving the thalamus bilaterally besides hypertensive and amyloid angiopathy-related hemorrhage that presents with the bilateral thalamic lesion, highlighting the importance of correct radiological diagnosis besides clinical history and examination pivoting crucial role in management and recovery of the patient.

Wernicke's encephalopathy is another disease with a reversible and preventive cause of involvement of the thalamus shows reversible T2 hyperintensity specifically involving the medial thalamus.[11] It is also important to

note that Wernicke's encephalopathy can show normal brain imaging at times and even occur in non-alcoholics. This is crucial from clinical and radiological perspectives in making a diagnosis.

Besides the reversible causes, other etiologies can be commonly found in unilateral and bilateral thalamic involvement. Lymphomas are neoplasms that respond well to steroids and radiation therapy. However, it is important to differentiate lymphoma from other neoplasms with characteristic features.[10-12]. Besides lymphoma, glial tumors frequently involve the thalamus. Moreover, it should not be ignored that metastasis also infrequently can be found at this location.[10] Radiologists and physicians must be aware of the differences to guide a better approach.

The idiopathic calcification of the basal ganglia, Fahr Syndrome, does not restrict its deposition to basal ganglia the thalamus, and other deep brain structures are also involved, demonstrating symptoms according to the region affected.[13] This is important to consider as family screening due to disease inheritance is indicated.[14]

Carbon monoxide poisoning is a common entity in cold areas presenting neuropsychological sequelae. Radiologically it selectively damages globus pallidus, however, thalamus involvement can occur in isolation showing restricted diffusion and thalamic hemorrhage.[15]

This manuscript provides the differential diagnoses in patients with radiologically bilateral thalamic lesions, emphasizing both causes that respond well to prompt treatment and those associated with depositional or neoplastic processes. This highlights the critical role of radiological diagnosis in guiding effective management strategies. Our patient cohort demonstrates that while multiple etiologies can result in thalamic lesions with generally poor prognosis, many other conditions, such as infections, demyelination, congenital disorders, hypoxic injury, and various neoplasms beyond lymphoma, may also impact the thalamus bilaterally. Hence, patients should undergo a proper radiological workup to confirm the diagnosis.

CONCLUSION

This study highlights the etiologies, and clinical and radiological profiles of various diseases of thalamic etiology in our region emphasizing that its involvement can stem from diverse etiologies, each influencing management approaches differently.

REFERENCES

1. Herrero MT, Barcia C, Navarro JM. Functional anatomy of thalamus and basal ganglia. Childs Nerv Syst. 2002;18(8):386-404.
2. Schmahmann JD. Vascular syndromes of the thalamus. Stroke. 2003;34(9):2264-78.
3. Lazzaro NA, Wright B, Castillo M, Fischbein NJ, Glastonbury CM, Hildenbrand PG, et al. Artery of percheron infarction: imaging patterns and clinical spectrum. AJNR Am J Neuroradiol. 2010;31(7):1283-9.
4. Zuccoli G, Pipitone N. Neuroimaging findings in acute Wernicke's encephalopathy: review of the literature. AJR Am J Roentgenol. 2009;192(2):501-8.
5. Carrera E, Bogousslavsky J. The thalamus and behavior: effects of anatomically distinct strokes. Neurology. 2006;66(12):1817-23.
6. Özgür A, Esen K, Kaleağası H, Yılmaz A, Kara E, Yıldız A. Bilateral thalamic lesions: A pictorial review. J Med Imaging Radiat Oncol. 2017;61(3):353-60.
7. Hegde AN, Mohan S, Lath N, Lim CC. Differential diagnosis for bilateral abnormalities of the basal ganglia and thalamus. Radiographics. 2011;31(1):5-30.
8. Kumral E, Evyapan D, Balkir K, Kutluhan S. Bilateral thalamic infarction. Clinical, etiological, and MRI correlates. Acta neurologica Scandinavica. 2001;103(1):35-42.
9. Jiménez Caballero PE. Bilateral paramedian thalamic artery infarcts: report of 10 cases. J Stroke Cerebrovasc Dis. 2010;19(4):283-9.
10. Renard D, Castelnovo G, Campello C, Bouly S, Le Floch A, Thouvenot E, et al. Thalamic lesions: a radiological review. Behav Neurol.2014;2014:154631.
10. Manzo G, De Gennaro A, Cozzolino A, Serino A, Fenza G, Manto A. MR imaging findings in alcoholic and nonalcoholic acute Wernicke's encephalopathy: a review. Biomed Res Int. 2014;2014:503596.
11. Ota Y, Capizzano AA, Moritani T, Naganawa S, Kurokawa R, Srinivasan A. Comprehensive review of Wernicke encephalopathy: pathophysiology, clinical symptoms and imaging findings. Jpn J Radiol. 2020;38(9):809-20.
12. Menon G, Nair S, Sudhir J, Rao BR, Krishnakumar K. Bilateral thalamic lesions. Br J Neurosurg. 2010 Oct;24(5):566-71. doi: 10.3109/02688691003777915.
13. Saleem S, Aslam HM, Anwar M, Anwar S, Saleem M, Saleem A, et al. Fahr's syndrome: literature review of current evidence. Orphanet J Rare Dis. 2013;8:156.
14. Mufaddel AA, Al-Hassani GA. Familial idiopathic basal ganglia calcification (Fahr`s disease). Neurosciences (Riyadh). 2014;19(3):171-7.
15. Abdelhedi J, Bouchhima I, Turki E, Damak M, Boukhris A, Miladi MI, et al. Selective bilateral thalamic necrosis following carbon monoxide poisoning: Case report. J Neurol Sci. 2013;333:e315.

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Shafaq Saleem; design, data collection, data analysis, manuscript writing
Muhammad Saleem; concept, data analysis, manuscript writing
Fatima Mubarak; data collection, manuscript writing
Salman Farooq; concept, manuscript revision

All the authors have approved the final version to be published, and agree to be accountable for all aspects of the work.