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INVESTIGATION OF VOLUMETRIC ALTERATIONS IN THALAMIC SUBNUCLEI IN PROGRESSIVE AND STABLE MILD COGNITIVE IMPAIRMENT USING MAGNETIC RESONANCE IMAGING

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Abstract

Objective: This study aimed to evaluate the volumes of thalamic subnuclei known to function in large-scale cognitive networks between progressive mild cognitive impairment (pMCI) and stable MCI (sMCI) groups progressing to dementia.

Materials and Methods: Magnetic resonance imaging and clinical data of 31 pMCI (Age: 68.66±6.86; education: 15.71±2.46; gender: 15 female) and 31 sMCI (Age: 70.18±7.24; education: 16.23±2.68; gender: 13 female) patients with no statistically significant differences in age, gender, education, and follow-up interval (mean 21 months) from the Alzheimer's Disease Neuroimaging Initiative database were used. FreeSurfer software was used for individual thalamus segmentation and volume calculation. Thalamic nuclei were divided into anterior, medial, posterior, lateral, ventral, and intralaminar nucleus groups. The volumes of each nucleus were normalised using intracranial volume. Normalised volumes were compared between groups using independent samples t-test, and false discovery rate (FDR) correction was applied. Correlation analysis was performed using florbetapir (AV45) PET scores to evaluate the relationship between amyloid burden in the brain and volumetric decrease.

Results: Statistically significant volumetric decreases were detected in the bilateral anterior (Right: t=2.432 p_{FDR}=0.048; Left: t=2.327 p_{FDR}=0.048) and lateral (t=2.372 p_{FDR}=0.048) nucleus groups in pMCI compared to sMCI. A negative correlation was found between PET scores and bilateral anterior nucleus groups (Right: r=-0.353 p=0.026, left: r=-0.350 p=0.026).

Conclusion: In our study, the anterior thalamic nucleus group, closely associated with memory, showed reduced volume in MCI patients who progressed to dementia, and this reduction correlated with amyloid burden in the brain. Based on the findings of our study, the anterior thalamic nucleus group may provide supportive value to other MRI biomarkers in predicting conversion to dementia.

Keywords Thalamus · Mild cognitive impairment · Magnetic resonance imaging · Dementia · Volumetric analysis

INTRODUCTION

Mild cognitive impairment (MCI) is defined as a clinical condition characterised by cognitive impairment that exceeds what is expected for an individual's age and education level but without significant functional impairment in activities of daily living.

Furthermore, MCI, which corresponds to the prodromal phase of Alzheimer's disease (AD) dementia, is also defined as its potentially reversible phase (1). Clinically, MCI is divided into two types: progressive MCI (pMCI), which progresses to dementia over time, and stable MCI (sMCI), which maintains cognitive status for an extended period (2). Distinguishing these two groups is crucial for developing targeted interventions and follow-up strategies.

It has been accepted that the relationship between brain and behaviour lies at the core of large-scale neural networks and can be revealed by describing these networks in detail. In this context, network-based models are gaining importance to describe the internal organisation of cortical and subcortical components of large-scale neural networks (3). However, a centripetal approach is also needed to fully understand large-scale networks in which higher-level cognitive functions are performed. In this regard, the thalamus, a critical hub for regulating higher-level cognitive functions such as episodic memory, attention, and executive functions, has become a focal point (4, 5). In addition, structural and functional neuroimaging studies have also shown that thalamic atrophy and functional connectivity changes occur in the preclinical and prodromal stages of AD and are particularly associated with impairments in memory and executive functions (6-9). Accordingly, focusing on subnuclei of the thalamus in the process of dementia may allow for the evaluation of broader cortico-subcortical network disorders, moving beyond neurodegenerative models that have historically focused on the hippocampus and medial temporal lobe structures.

This study aimed to investigate the volumetric changes in six major nuclei groups (anterior, lateral, ventral, intralaminar, medial, and posterior) of the thalamus between the pMCI and sMCI groups using structural magnetic resonance imaging (MRI) data and to evaluate whether the thalamus has potential as an early biomarker to predict conversion to dementia.

MATERIALS AND METHODS

Participants

All data used in this study were obtained from the Alzheimer's Disease Neuroimaging Initiative (ADNI) database (https://adni.loni.usc.edu/). ADNI is a multicenter, longitudinal study to develop and validate biomarkers for the early detection and monitoring of AD. The ADNI study was approved by the Institutional Review Boards of all participating institutions, and written informed consent was obtained from all participants or their legal representatives. In this study, 3 Tesla structural MRI scans (baseline acquisition), 18F-florbetapir-positron emission tomography ([^{18}F] AV45-PET), and clinical data of a total of 62 individuals diagnosed with mild cognitive impairment (MCI) from the ADNI database were used. Participants who met the following criteria were included in the study: (i) diagnosis of MCI at baseline visit, (ii) documentation of whether participants converted from MCI to dementia at the follow-up visit, (iii) availability of baseline 3 Tesla T1-weighted structural MRI scans, and (iv) complete demographic and clinical data. In line with these criteria, participants were divided into two groups as pMCI and sMCI, depending on whether they showed a transformation from MCI to AD-type dementia at the end of the follow-up period.

Volumetric analysis of magnetic resonance imaging data

FreeSurfer (https://surfer.nmr.mgh.harvard.edu, version 7.2.0) software was used to segment anatomical MRI data. All volumetric analyses were conducted exclusively on baseline MRI images, which correspond to the initial clinical assessment. In the first stage, standard recon-all steps in FreeSurfer were applied to all individual MRI images (10, 11). After this stage, the skull peeling process and segmentation outputs of all individual MRI images were examined using Freeview. Following the quality control procedure, no manual correction was required, and no participants were excluded due to segmentation failures. In the advanced segmentation stage, 25 thalamus thalamic nuclei were divided into subnuclei based on the thalamus atlas created by Iglesias et al. using high-resolution MRI and histological data, and volume values were calculated for each participant (12). The 25 thalamic nuclei obtained were anatomically grouped and six subgroups were created. The six thalamic nucleus groups are presented in three dimensions in Figure 1. Additionally, to reduce the potential inter-individual influence of head size differences, all nuclei volumes were normalised using the estimated total intracranial volume.

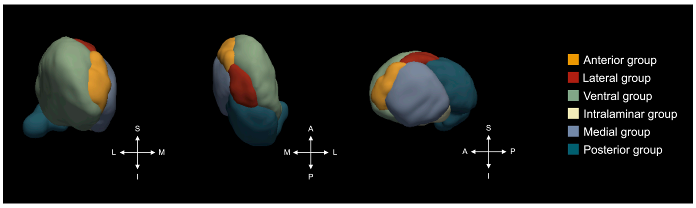

Figure 1. Three-dimensional representation of the six major thalamic nucleus groups. I: Inferior, S: Superior, A: Anterior, P: Posterior, M: Medial, L: Lateral

Statistical analysis

Statistical analyses were performed using SPSS (IBM SPSS Statistics version 30, https://www.ibm.com/tr-tr/products/spss-statistics) and R version 4.0.0 (https://www.r-project.org). First, the Shapiro-Wilk test was used to determine whether the data showed a normal distribution. Then, statistical analyses were performed using the independent sample t-test for continuous variables and the chi-square test for categorical variables. Multiple comparisons in the volumetric analyses of the thalamic subnuclei were controlled using the False Discovery Rate (FDR) correction. All p-values were adjusted using the P.adjust function in R (13). The FDR correction was applied across all 12 thalamic nuclei (6 left, 6 right). The significance threshold for volumetric statistical analyses was set at $p_{FDR}<0.05$ (FDR-corrected). Linear regression analysis was performed to estimate [^{18}F] AV45 PET reflecting the pathological burden of disease using the significant volumetric changes observed between groups as independent variables. Since three linear regression analyses were performed, Bonferroni correction was applied, and the significance threshold was set at p<0.017.

RESULTS

Demographic and clinical results

The statistical analysis results of demographic and clinical data are presented in Table 1. No statistically significant difference was found between the groups in terms of age, education status, and gender (t=0.761, p>0.05; t=0.971, p>0.05; χ^2=0.261, p>0.05, respectively). Additionally, no statistically significant difference was found between the groups in terms of the follow-up interval (t=0.844, p>0.05). In clinical evaluations, Mini Mental State Examination (MMSE) scores at follow-up were found to be lower in the pMCI group compared to the sMCI group (t=4.715, p<0.001), whereas there was no difference between the groups in MMSE-baseline scores (t=1.929, p>0.05).

Table 1. Demographic and clinical details of the groups

	Mean±standard deviation		Test	p values
	sMCI (n=31)	pMCI (n=31)		
Age	70.18±7.24	68.66±6.86	0.761†	N.S.
Education	16.23±2.68	15.71±2.46	0.971†	N.S.
Gender (Male/Female)	18 / 13	16 / 15	0.261‡	N.S.
Follow-up interval	21.48±10.71	21.68±10.92	0.844†	N.S.
MMSE (Baseline)	28.26±1.505	27.26±2.46	1.929†	N.S.
MMSE (Follow-up)	27.77±2.48	20.87±7.61	4.715†	<0.001

pMCI: Progressive mild cognitive impairment, sMCI: Stable mild cognitive impairment, MMSE: Mini Mental State Examination, †: Independent Samples t: Test, ‡: Chi-square, The significance threshold was set as p<0.05.

Volumetric analysis results

As a result of statistical analysis, significant volume decrease was detected in bilateral anterior group (Right: t=2.432, p =0.048; Left: t=2.327, p=0.048) and lateral group (t=2.372, p=0.048) thalamic nuclei in the pMCI group compared to the sMCI group. No statistically significant difference was detected in the other nucleus groups. Analysis results are presented in detail in Table 2 and Figure 2.

Table 2. Volumetric analysis of thalamic nuclei subgroups

	Volume-to-eTIV ratio (x10super [-6])		*t* values	P_{FDR}	Cohen's d	95% CI
	sMCI	pMCI				
Right						
Anterior group	100.607±19.545	89.839±15.021	2.432	**0.048***	0.618	[0.105 1.125]
Lateral group	97.817±21.055	85.978±18.143	2.372	**0.048***	0.602	[0.091 1.109]
Ventral group	1796.140±171.008	1730.902±172.925	1.494	0.120	0.379	[-0.125 0.880]
Intralaminar group	266.703±28.610	251.822 ± 25.525	2.161	0.051	0.549	[0.039 1.054]
Medial group	650.637±98.701	633.152±88.876	0.733	0.266	0.186	[-0.314 0.684]
Posterior group	1369.935±163.320	1330.365±138.482	1.029	0.205	0.261	[-0.240 0.760]
Left						
Anterior group	89.238±19.263	79.094±14.763	2.327	**0.048***	0.591	[0.080 1.098]
Lateral group	98.967±21.289	90.865±17.725	1.628	0.108	0.414	[-0.091 0.915]
Ventral group	1753.913±120.209	1690.246±169.640	1.705	0.108	0.433	[-0.073 0.935]
Intralaminar group	257.375±22.701	249.443±24.494	1.294	0.150	0.329	[-0.174 0.829]
Medial group	641.912±106.196	631.776±88.928	0.407	0.343	0.103	[-0.395 0.601]
Posterior group	1346.984±123.218	1323.680±139.437	0.697	0.266	0.177	[-0.322 0.675]

Values shown are in mean±standard deviation. Independent sample t-test analysis was performed and the significance threshold was determined as p<0.05 by applying false discovery rate correction to the results. pMCI: Progressive mild cognitive impairment, sMCI: Stable mild cognitive impairment, R: Right, L: Left; eTIV: Estimated total intracranial volume, CI: Confidence intervals

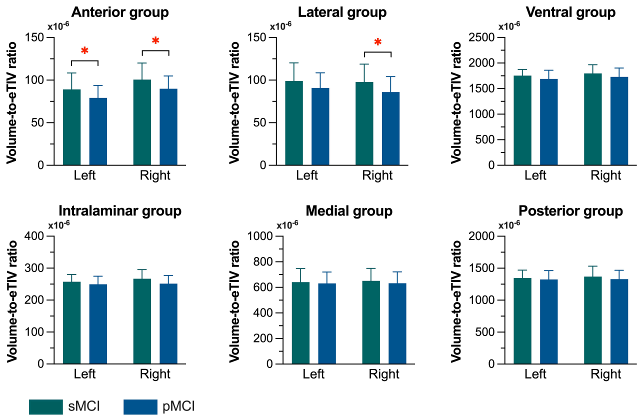

Figure 2. Plots of independent samples t-test comparisons for the thalamic nuclei are presented. Mean volumes to estimated total intracranial volume ratios [x10⁻⁶] have been reported (False Discovery Rate-corrected significance threshold *p<0.05). pMCI: Progressive mild cognitive impairment, sMCI: Stable mild cognitive impairment, eTIV: estimated Total Intracranial Volume

Linear regression analysis results

To investigate to what extent the bilateral anterior and right lateral group nuclei predicted PET scores, linear regression analyses were performed using each left and right anterior and right lateral group nuclei normalised volumes as the dependent variables and [^{18}F] AV45 PET scores as the independent variables. As a result of the multiple comparison correction, only the left anterior group nuclei surpassed the significance threshold. The left anterior group nuclei volume (R=0.391, R^2=0.153, F=6.847, p=0.013) explained 15% of the variance in PET scores (Figure 3). No statistically significant regression was found in the right lateral group nuclei (p>0.017) and right anterior group nuclei (p=0.017).

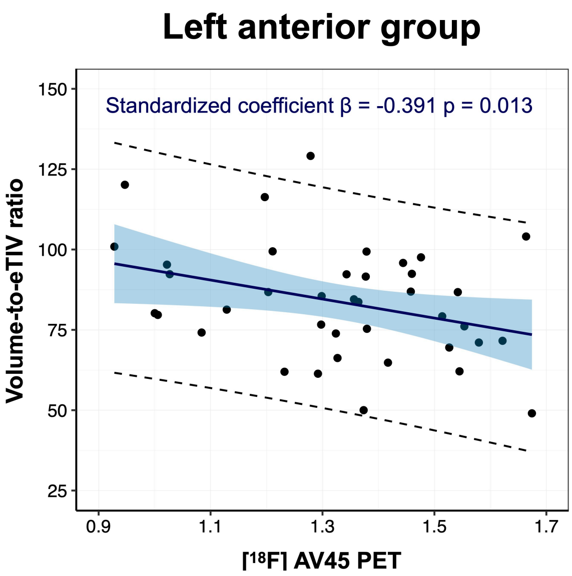

Figure 3. Linear regression analysis results between normalized volumes in left anterior group thalamic nuclei and [^{18}F] AV45 PET. The significance threshold in the analysis was set as p<0.017.

DISCUSSION

In this study, volumetric comparisons of six major thalamic nuclei groups were performed between the sMCI and pMCI groups using structural MRI data. As a result of the analysis, a decrease in the bilateral anterior and right lateral group nuclei was detected in pMCI compared to sMCI. In addition, to examine the relationship between the volume decrease in these anatomical regions and the amount of amyloid in the brain, regression analysis was performed between volume values and [^{18}F] AV45 PET scores, and a negative regression was found between the left anterior group nuclei and [^{18}F] AV45 PET.

Structural neuroimaging studies show that the total volume of the bilateral thalamus decreases in the clinical course of AD (6, 14, 15). A neuroimaging study focusing on the thalamus subgroup nuclei in AD reported that the volume of the medial group nuclei was reduced in the MCI and AD dementia groups compared to those with subjective cognitive impairment (8). Bernstein et al. reported that the anteroventral, mediodorsal, pulvinar, medial geniculate, and centromedian nuclei were significantly smaller in late MCI and AD cases than in healthy controls (9). Another neuroimaging study reported significant differences in the anterior, lateral, and posterior nucleus groups between the healthy control, MCI, and AD groups (7). Post hoc comparisons showed that the volume of the anterior and posterior nucleus groups was lower in the AD group compared to the healthy control and MCI groups (7). Cross-sectional studies in the literature consistently report a decrease in total thalamic volume in AD; however, findings regarding thalamic nuclei subgroups are inconsistent across the preclinical, prodromal, and dementia stages of the disease. Additionally, the effect sizes observed in our study (Cohen's d≈0.6) are moderate but consistent with a recent study showing subregion-specific volumetric reductions in thalamic nuclei in neurodegenerative conditions (16). However, these studies did not evaluate patients with MCI longitudinally, and it is unknown whether these patients are progressive or stable. Nevertheless, our findings of volume reduction in the anterior and lateral nuclei groups are consistent with studies reported in the literature.

When the anterior group nuclei of the thalamus are classified anatomically, they consist of anteroventral, anterodorsal, and anteromedial nuclei (12). When evaluated functionally, the laterodorsal nuclei are also considered within the anterior group nuclei due to their connections with the limbic system (17). In this context, when evaluating the study findings, the lateral group nuclei, which consist of the laterodorsal and lateroposterior nuclei, can be considered together with the anterior group nuclei. In the study, the difference detected between the groups only in the anterior and lateral group nuclei, especially in the right hemisphere, supports the fact that both nucleus groups work together. Anterior group nuclei are among the basic components of the Papez circuit, which play an important role in maintaining episodic memory functions (18). Furthermore, the anterior group of nuclei is located at the centre of the hippocampal–anterior thalamic axis defined by Aggleton and Brown (19). This critical location allows them to collaborate with the hippocampus, mammillary bodies, retrosplenial cortex, and prefrontal areas, thus ensuring the functional integrity of the Papez circuit (19-22). The study by Swartz and Black demonstrated that anterior–medial thalamic lesions are relatively frequent across different dementia syndromes and are strongly associated with accelerated cognitive decline (23). Furthermore, in Wernicke–Korsakoff syndrome, the anterior thalamic nuclei are particularly vulnerable, and neuronal loss in this region is considered a key contributor to the episodic memory impairments characteristic of the disorder (24). Moreover, the fact that amnestic syndromes have been reported in the literature as a result of cerebrovascular events affecting the anterior group nuclei also supports the importance of this nucleus group (25). When considered holistically, considering both their anatomical location and critical connections to the limbic system, the anterior group nuclei play a crucial role in memory functions. In this context, the volume reduction of the anterior group nuclei in pMCI detected in our study may underlie the progressive deterioration in memory functions.

Braak et al. reported that, as a result of postmortem examinations, the most prominent amyloid plaque accumulation in AD was seen in the anteroventral nucleus, which is located among the anterior group nuclei, and that this pathological plaque accumulation could prevent communication between limbic circuits (26). In our study, the negative correlation finding between the anterior group nuclei and [^{18}F] AV45 PET, which expresses the amount of amyloid plaque in the brain, supports this specific accumulation. In this context, considering the sensitivity of the anterior group nuclei to the accumulation of amyloid plaques and their critical role in memory functions, the significant volume loss and amyloid plaque accumulation in this nucleus group may be an important potential marker for the transformation to dementia.

Finally, although our study conducted specific and rigorous analyses, the relatively small sample size in each group can be considered a limitation. Therefore, we aimed to address this limitation by applying conservative statistical adjustments. In future studies, analyses focusing on thalamic subgroup nuclei in larger datasets will be necessary for elucidating the course of AD.

CONCLUSION

Our findings indicate that patients progressing to dementia show a more pronounced volume reduction in the anterior thalamic nuclei, suggesting that these nuclei may be particularly susceptible to early neurodegenerative processes and may contribute to disruptions in networks supporting episodic memory. Furthermore, although hippocampal atrophy remains a classical marker in AD, previous research points to thalamic degeneration as a factor that may influence or potentially accelerate cognitive decline. In conclusion, the anterior thalamic nuclei may support the classification performance of MRI-based biomarkers derived from larger cohorts and may show potential utility in identifying individuals at higher risk of progression to dementia in AD.

Ethics Committee Approval The Alzheimer's Disease Neuroimaging Initiative (ADNI) was approved by the institutional review boards of all participating ADNI sites reviewed and approved the data collection protocol provided by ADNI. This study was conducted using ADNI data. The ADNI study is ethically approved and operated in accordance with the Declaration of Helsinki, 1964.

Informed Consent Informed consent was not obtained because data from the Alzheimer's Disease Neuroimaging Initiative (ADNI) database was used.

Conflict of Interest The authors have no conflict of interest to declare.

Author Contributions Conception/Design of Study- E.H., C.S.; Data Acquisition- C.S.; Data Analysis/Interpretation-E.H., C.S.; Drafting Manuscript-E.H.; Critical Revision of Manuscript-E.H., C.S.; Final Approval and Accountability-E.H., C.S.; Technical or Material Support-E.H., C.S.; Supervision- E.H.

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References

1 Albert MS, DeKosky ST, Dickson D, Dubois B, Feldman HH, Fox NC, et al. The diagnosis of mild cognitive impairment due to Alzheimer's disease: recommendations from the National Institute on Aging-Alzheimer's Association workgroups on diagnostic guidelines for Alzheimer's disease. Alzheimers Dement 2011;7(3):270-9.

2 Han JW, Kim TH, Lee SB, Park JH, Lee JJ, Huh Y, et al. Predictive validity and diagnostic stability of mild cognitive impairment subtypes. Alzheimers Dement 2012;8(6):553-9.

3 Aggleton JP, Vann SD, O'Mara SM. Converging diencephalic and hippocampal supports for episodic memory. Neuropsychologia 2023;191:108728.

4 Mitchell AS, Sherman SM, Sommer MA, Mair RG, Vertes RP, Chudasama Y. Advances in understanding mechanisms of thalamic relays in cognition and behavior. J Neurosci 2014;34(46):15340-6.

5 Sherman SM. The thalamus is more than just a relay. Curr Opin Neurobiol 2007;17(4):417-22.

6 Zarei M, Patenaude B, Damoiseaux J, Morgese C, Smith S, Matthews PM, et al. Combining shape and connectivity analysis: an MRI study of thalamic degeneration in Alzheimer's disease. Neuroimage 2010;49(1):1-8.

7 Low A, Mak E, Malpetti M, Chouliaras L, Nicastro N, Su L, et al. Asymmetrical atrophy of thalamic subnuclei in Alzheimer's disease and amyloid-positive mild cognitive impairment is associated with key clinical features. Alzheimers Dement 2019;11:690-9.

8 Hari E, Ulasoglu-Yildiz C, Kurt E, Bayram A, Gurvit H, Demiralp T. Volumetric and functional connectivity changes of the thalamic nuclei in different stages of Alzheimer's disease. Clin Neurophysiol 2024;165:127-37.

9 Bernstein AS, Rapcsak SZ, Hornberger M, Saranathan M, Initiative ADN. Structural changes in thalamic nuclei across prodromal and clinical Alzheimer's disease. J Alzheimers Dis 2021;82(1):361-71.

10 Fischl B, Salat DH, Busa E, Albert M, Dieterich M, Haselgrove C, et al. Whole brain segmentation: automated labeling of neuroanatomical structures in the human brain. Neuron 2002;33(3):341-55.

11 Dale AM, Fischl B, Sereno MI. Cortical surface-based analysis. I. Segmentation and surface reconstruction. Neuroimage 1999;9(2):179-94.

12 Iglesias JE, Insausti R, Lerma-Usabiaga G, Bocchetta M, Van Leemput K, Greve DN, et al. A probabilistic atlas of the human thalamic nuclei combining ex vivo MRI and histology. Neuroimage 2018;183:314-26.

13 Benjamini Y, Hochberg Y. Controlling the false discovery rate: a practical and powerful approach to multiple testing. J R Stat Soc Series B Stat Methodol 1995;57(1):289-300.

14 Zidan M, Boban J, Bjelan M, Todorović A, Vujanić TS, Semnic M, et al. Thalamic volume loss as an early sign of amnestic mild cognitive impairment. J Clinical Neurosci 2019;68:168-73.

15 de Jong LW, van der Hiele K, Veer IM, Houwing JJ, Westendorp RGJ, Bollen E, et al. Strongly reduced volumes of putamen and thalamus in Alzheimer's disease: an MRI study. Brain 2008;131(12):3277-85.

16 Banerjee A, Yang F, Dutta J, Cacciola A, Hornberger M, Saranathan M. Atrophy patterns of deep gray matter nuclei in Alzheimer's disease and frontotemporal dementia. J Alzheimers Dis 2025;108(4):1760-77 .

17 Morel A, Magnin M, Jeanmonod D. Multiarchitectonic and stereotactic atlas of the human thalamus. J Comp Neurol 1997;387(4):588-630.

18 Papez JW. A proposed mechanism of emotion. Arch NeurPsych 1937;38(4):725-43.

19 Aggleton JP, Brown MW. Episodic memory, amnesia, and the hippocampal–anterior thalamic axis. Behav Brain Sci 1999;22(3):425-44.

20 Aggleton JP, Pralus A, Nelson AJD, Hornberger M. Thalamic pathology and memory loss in early Alzheimer's disease: moving the focus from the medial temporal lobe to Papez circuit. Brain 2016;139(7):1877-90.

21 Aggleton JP, O'Mara SM, Vann SD, Wright NF, Tsanov M, Erichsen JT. Hippocampal–anterior thalamic pathways for memory: uncovering a network of direct and indirect actions. Eur J Neurosci 2010;31(12):2292-307.

22 Jankowski MM, Ronnqvist KC, Tsanov M, Vann SD, Wright NF, Erichsen JT, et al. The anterior thalamus provides a subcortical circuit supporting memory and spatial navigation. Front Syst Neurosci 2013;7:45.

23 Swartz RH, Black SE. Anterior-medial thalamic lesions in dementia: frequent, and volume dependently associated with sudden cognitive decline. J Neurol Neurosurg Psychiatry 2006;77(12):1307-12.

24 Harding A, Halliday G, Caine D, Kril J. Degeneration of anterior thalamic nuclei differentiates alcoholics with amnesia. Brain 2000;123(1):141-54.

25 Cipolotti L, Husain M, Crinion J, Bird CM, Khan SS, Losseff N, et al. The role of the thalamus in amnesia: A tractography, high-resolution MRI and neuropsychological study. Neuropsychologia 2008;46(11):2745-58.

26 Braak H, Braak E. Alzheimer's disease affects limbic nuclei of the thalamus. Acta Neuropathol 1991;81(3):261-8.