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Research Article

Factors Affecting the Etiology of Isolated Thalamic Infarcts and Thalamic Infarcts with Extrathalamic Involvement: The Stroke Center Experience

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Objective: The thalamus is a control point that is a gateway for all sensory impulses except smell and contributes to regulating the sleep-wake process. Therefore, infarcts of the thalamus cause a diversity of symptoms along with a diversity of vascular anatomy. The fact that thalamic infarcts are mostly considered lacunar infarcts seems to create inadequacy in understanding the etiology. This study aimed to compare the etiologic, diagnostic, and therapeutic parameters of thalamic infarction.

Materials and Methods: This retrospective study analyzed 230 patients with thalamic infarction among 820 stroke patients admitted between January 2019 and April 2022. Patients with hemorrhagic stroke, malignancy, or infection were excluded. Demographic data, comorbidities, imaging results, and treatments administered were evaluated. All these results were analyzed in two subgroups: isolated thalamic and extrathalamic involvement.

Results: Bilateral thalamic involvement was 8.386 times more common in patients with extrathalamic participation. Patients using dual antiaggregants had 2.207 times higher isolated thalamic involvement, while those on oral anticoagulants had 2.378 times higher extrathalamic involvement. Pathologies in the basilar and vertebral arteries increased the risk of extrathalamic involvement by 2.660 times ($p<0.001$).

Conclusion: Thalamic infarcts cannot be considered exclusively lacunar. Extrathalamic involvement has been associated with worse functional scores and arterial pathologies. The protective effects of dual antiaggregants and oral anticoagulants vary depending on whether the thalamic infarct is extrathalamic or not, emphasizing the need for individualized therapies.

Keywords: Thalamus; Stroke, Extrathalamic involvement, Antiaggregant

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1. INTRODUCTION

The thalamus is a cerebral structure located in the dorsal part of the diencephalon, between the cerebral cortex and the midbrain, consisting of several interconnected gray matter nuclei separated by laminae of white matter and providing transmission between the cerebral cortex and peripheral structures, the spinal cord or brainstem.[1,2] The thalamus, which is considered a gateway for afferent signals from all systems except the sense of smell, provides filtration of incoming motor and sensory signals and transfers them to the cerebral cortex. As such, it governs our sensitivity to heat, light, and physical touch, and controls the flow of visual, auditory, and motor information. It plays a role in motivation and sleep-wake processes and controls proprioceptive sensation. The thalamus is involved in the modulation of these functions and is thus responsible for purposeful conscious behavior.[3]

Thalamic infarcts are known to occur most commonly with an etiology of small vessel disease and may show unilateral or bilateral involvement.[4-6] The thalamus is supplied by the polar artery (posterior communicating artery), paramedian thalamic-subthalamic arteries, inferolateral (thalamogeniculate) arteries, and posterior choroidal arteries (medial and lateral). These are all branches of the posterior cerebral artery (PCA). Considering that all arteries supplying the thalamus are terminal arteries, it can be assumed that thalamic infarctions are mostly lacunar and the etiology is small vessel disease. However, in vertebral and/or basilar artery-related infarcts in which the PCA is also affected, different clinical pictures may occur

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according to the characteristics of the arterial area affected by the infarct due to extrathalamic involvement as well as signs of thalamus involvement.[1,7,8] The incidence of PCA infarcts is 5-10% of all ischemic strokes and the most common causes are atherosclerosis and embolism. However, causes other than thrombotic processes such as dissection, hemorrhage, migraine, Moyamoya disease, fibromuscular dysplasia, mitochondrial diseases, reversible cerebral vasoconstriction syndrome, vertebrobasilar dolichoectasia and vasculitis, and central nervous system infections may also be involved in the etiology.[7,9-11]

Due to the differences in the localization of the affected vessel and the differences in the etiological causes, it is important to make an etiological evaluation of patients with isolated thalamic infarcts and patients with thalamic infarcts with extrathalamic involvement. In addition to this, it is an absolute necessity for the treatment of acute stroke to be directed towards this etiologic cause for the treatment to be applied correctly. Therefore, this study aimed to compare the diagnostic and therapeutic parameters of thalamic infarcted patients with isolated thalamic involvement and thalamic infarcted patients with extrathalamic involvement and to evaluate the relationship with etiologic causes.

2. MATERIAL AND METHODS

In this study, patients admitted to our hospital with a diagnosis of thalamic infarction were retrospectively analyzed. History and demographic data from all patients were recorded. Neurologic examination records including detailed tests of somatosensory functions and motor performance were also evaluated.

We retrospectively analyzed 820 patients with a diagnosis of stroke who were hospitalized and treated in our hospital between January 2019 and April 2022. Among these patients, patients without thalamic involvement, patients whose hospitalization and follow-up could not be performed by us after diagnosis, patients with malignancy, patients associated with infection, and patients diagnosed with hemorrhagic stroke were excluded. Ethics committee approval was obtained for the study (The Ethics Committee of the Antalya Education and Research Hospital approved this study (Number: 2024-152 KN: 7/11).

230 patients with thalamic stroke were included in the study and analyzed retrospectively. Etiological factors such as age, gender, lateralization, hypertension, diabetes mellitus, coronary and peripheral artery disease, history of previous cerebrovascular events, history of dementia, history of seizures, smoking, presence of atrial fibrillation, and family history were evaluated and recorded. In addition, functional assessments such as cognitive impairment, hemoglobin (Hb), Mean Corpuscular Volume (MCV), platelet, Mean Platelet Volume (MPV), hemoglobin A1C (HbA1c), and hyperlipidemia (HL) results, and medication history were recorded. In addition, the results of imaging scoring systems Alberta Stroke Program Early CT Score (ASPECT), Fazekas Scale and Computed tomographic angiography (CTA), tissue plasminogen activator (tPA) and Endovascular thrombectomy EVT procedures and Trial of Org 10172 in Acute Stroke Treatment (TOAST) criteria were recorded (Table-1,2).

Table 1.

Identification of risk factors for isolated thalamus involvement and extrathalamic involvement (Demographic data)

	Extrathalamic involvement			Univariate	
	No	Yes	Total	OR (%95 CI)	p
Age	69,1 ± 13,1	70,6 ± 13,7	69,8 ± 13,4	1,008 (0,989-1,028)	0,399
Gender					
Female	55 (55,6)	44 (44,4)	99 (43)	Reference	
Male	67 (51,1)	64 (48,9)	131 (57)	1,194 (0,707-2,016)	0,507
Lateralization					
Unilateral	121 (54,5)	101 (45,5)	222 (96,5)	Reference	
Bilateral	1 (12,5)	7 (87,5)	8 (3,5)	8,386 (1,015-69,301)	**0,048**
Hypertension					
(-)	52 (55,9)	41 (44,1)	93 (40,4)	Reference	
(+)	70 (51,1)	67 (48,9)	137 (59,6)	1,214 (0,715-2,060)	0,473
Diabetes Mellitus					
(-)	69 (51,9)	64 (48,1)	133 (57,8)	Reference	
(+)	53 (54,6)	44 (45,4)	97 (42,2)	0,895 (0,530-1,513)	0,679
Coronary Arterial Disease					
(-)	89 (55,3)	72 (44,7)	161 (70)	Reference	
(+)	33 (47,8)	36 (52,2)	69 (30)	1,348 (0,766-2,373)	0,300
Peripheral Arterial Disease					
(-)	119 (53,6)	103 (46,4)	222 (96,5)	Reference	
(+)	3 (37,5)	5 (62,5)	8 (3,5)	1,926 (0,449-8,254)	0,378
Cerebrovascular Disease					
(-)	97 (52,4)	89 (47,6)	186 (80,4)	Reference	
(+)	24 (54,5)	20 (45,5)	44 (19,1)	0,882 (0,458-1,698)	0,707
Dementia					
(-)	116 (52,5)	105 (47,5)	221 (95,2)	Reference	
(+)	6 (66,7)	3 (33,3)	9 (3,9)	0,984 (0,725-1,335)	0,917
Seizure					
(-)	121 (53,3)	106 (46,7)	227 (98,7)	Reference	
(+)	1 (33,3)	2 (66,7)	3 (1,3)	2,283 (0,204-25,536)	0,503

Table 1. (Continued)

Smoking					
(-)	102 (51,8)	95 (48,2)	197 (85,7)	Reference	
(+)	20 (60,6)	13 (39,4)	33 (14,3)	0,698 (0,329-1,481)	0,349
Atrial Fibrillation					
(-)	107 (54,9)	88 (45,1)	195 (84,8)	Reference	
(+)	15 (42,9)	20 (57,1)	35 (15,2)	1,621 (0,784-3,352)	0,192
Family History					
(-)	121 (52,8)	108 (47,2)	229 (99,6)		
(+)	1 (100)	0 (0)	1 (0,4)		

Table 2.

Identification of risk factors for isolated thalamus involvement and extrathalamic involvement (Test results, imaging results, and scoring systems)

	Extrathalamic involvement*			Univariate	
	No	Yes	Total	OR (%95 CI)	p
Cognitive Impairment					
(-)	106 (58,9)	74 (41,1)	180 (78,3)	Reference	
(+)	15 (31,3)	33 (68,8)	48 (20,9)	1,362 (0,874-2,123)	0,172
Hb	12,8 ± 1,8	12,7 ± 2,2	12,8 ± 2	0,960 (0,843-1,093)	0,538
MCV	83,9 ± 10,2	84,5 ± 7,6	84,1 ± 9,1	1,008 (0,979-1,037)	0,610
Plt	242,7 ± 84,1	249,8 ± 78	246 ± 81,2	1,001 (0,998-1,004)	0,510
MPV	10,8 ± 1	10,8 ± 1	10,8 ± 1	1,038 (0,797-1,353)	0,781
Hba1C	6,9 ± 1,9	7,2 ± 2,1	7,1 ± 2	1,083 (0,947-1,238)	0,245
HL					
(-)	110 (53,4)	96 (46,6)	206 (89,6)	Reference	
(+)	12 (50)	12 (50)	24 (10,4)	1,146 (0,492-2,669)	0,752
Medication Use					
No medicine	2 (40)	3 (60)	5 (2,2)	1,716 (0,281-10,471)	0,558
Single antiaggregant	41 (48,2)	43 (51,8)	84 (37,3)	1,363 (0,795-2,335)	0,260
Double antiaggregant	64 (64)	36 (36)	100 (43,5)	2,207 (1,292-3,768)	**0,004**
Oral anticoagulant	15 (35,7)	26 (64,3)	41 (18,3)	2,378 (1,188-4,760)	**0,014**
ASPECT Score					
<6	18 (43,9)	23 (56,1)	41 (17,8)	1,563 (0,792-3,086)	0,198
≥6	104 (55)	85 (45)	189 (82,2)	Reference	

Table 2. (Continued)

Fazekas Score					
0 - 1	84 (53,5)	73 (46,5)	157 (68,3)	0,9744 (0,541-1,645)	0,838
2 - 3	38 (52,1)	35 (47,9)	73 (31,7)	Reference	
CTA					
(-)	83 (63,4)	48 (36,6)	131 (57,0)		
(+)	39 (39,4)	60 (60,6)	99 (43,0)	2,660 (1,554-4,553)	**<0,001**
tPA					
(-)	117 (54,2)	99 (45,8)	216 (94,7)	Reference	
(+)	4 (33,3)	8 (66,7)	12 (5,3)	2,364 (0,691-8,084)	0,170
EVT					
(-)	120 (53,8)	103 (46,2)	223 (97,8)	Reference	
(+)	1 (20)	4 (80)	5 (2,2)	4,660 (0,513-42,356)	0,172
TOAST					
Large-artery atherosclerosis	9 (47,4)	10 (52,6)	19 (8,3)	1,41 (0,54 - 3,68)	0,482
Cardioembolism	16 (42,1)	22 (57,9)	38 (16,5)	1,75 (0,85 - 3,6)	0,131
Small-vessel occlusion	16 (59,3)	11 (40,7)	27 (11,7)	0,87 (0,38 - 2,01)	0,750
Stroke of other determined etiology	1 (33,3)	2 (66,7)	3 (1,3)	2,54 (0,23 - 28,65)	0,451
Stroke of undetermined etiology	80 (55,9)	63 (44,1)	143 (62,2)	Reference	

*n (%); mean ± s.deviation

Abbreviations: mRS: Modified Rankin Scale, Hb: Hemoglobin, MCV: Mean Corpuscular Volume, Plt: Platelet, MPV: Mean Platelet Volume, HbA1c: Hemoglobin A1c, HL: Hyperlipidemia, ASPECT: Alberta Stroke Program Early CT Score, CTA: Computed Tomographic Angiography, tPA: tissue plasminogen activator, EVT: Endovascular thrombectomy and TOAST Criteria: Trial of Org 10172 in Acute Stroke Treatment Criteria

Data were analyzed with IBM SPSS V23. Mann-Whitney U test was used to compare the values. Pearson chi-square and Continuity correction tests were used to analyze categorical data. A significance level of $p<0.05$ was taken.

3. RESULTS

There was no difference between the two groups according to age and gender. 8 of 230 patients had bilateral thalamic involvement. The rate of extrathalamic involvement in unilateral patients was 45.5%, while the rate of extrathalamic involvement in bilateral patients was 87.5%. Extrathalamic involvement in patients with bilateral thalamic involvement was 8.386 (95% CI= 1.015-69.301) times higher than those with isolated thalamic involvement (p=0.048).

When the data in Table 2 are evaluated; there was no significant difference between isolated thalamic and extrathalamic involvement in terms of cognitive impairment, hemoglobin, MCV, platelet, MPV, HbA1c, and hyperlipidemia. When evaluated according to the history of medication (antiaggregant or anticoagulant) that the patients were taking at the time of admission; no medication, single antiaggregant use, dual antiaggregant use, and oral anticoagulant use were compared. Accordingly, isolated thalamus involvement was 2.207 (95% CI=1.292-3.768) times higher than extrathalamic involvement in patients using dual antiaggregant (p=0.004). In contrast, extrathalamic involvement was 2.378 (95% CI=1.188-4.760) times higher than isolated thalamus involvement in patients on oral anticoagulants (p=0.004). There was no significant difference between the two groups in the comparison of patients with no other drug use and patients with single antiaggregant use. There

was no difference between the two groups in terms of ASPECT score and Fazekas score indicating lesion burden. Again, when compared according to TOAST criteria, there was no significant difference in both groups according to TOAST subgroups. In addition, isolated thalamic and extrathalamic involvement was compared according to the treatments applied to the patients and the results of both groups were similar in terms of tissue plasminogen activator therapy and endovascular thrombectomy. Finally, the risk of pathology in the basilar artery and vertebral arteries on CTA was 2.660 (95% CI=1.554-4.553) times higher in extrathalamic involvement than in isolated thalamic involvement ($p<0.001$).

4. DISCUSSION

The thalamus is a structure with important roles in the sleep-wake cycle, mediating cortical stimulus responses, emotions, sensory (gustatory, somatosensory, visual, and auditory) information processing and connection to the cortex, and many other cognitive functions.[12] Thalamic ischemia is a stroke localization that can be frequently seen both in isolation and in association with infarcts involving other structures.[13] The functional complexity of the nuclei of the thalamus and the potential for different variations in the arteries supplying the thalamus result in wide variations in the clinical presentation of thalamic infarcts.[4]

There are many studies in the literature on the relationship between the different arterial anatomy of the thalamus and stroke. Especially publications on Percheron artery infarcts have started to appear frequently in the literature. However, there are no studies on the etiology of isolated thalamic infarcts.[14-16]

In our study, involvement outside the thalamus was observed in a total of 108 patients. These patients were compared with patients with isolated thalamus involvement. Bilateral thalamus involvement was approximately 8 times more common in patients with extrathalamic involvement than in patients with isolated thalamus involvement. Although subgroup analysis could not be performed in this group due to the small number of patients, it was thought that the presence of bilateral thalamus and simultaneous extrathalamic involvement may be associated with percheron artery infarcts. There are articles supporting this in the literature.[15,16] The view that thalamic infarcts are predominantly lacunar infarcts does not fit the data of this study.[17,18]

The most important data of the study is the estimation of the risk related to the protective effect of the medical treatment (antiaggregant or anticoagulants) used by the patients before stroke. In the study, patients without medication, patients using single or dual antiaggregants, and patients using oral anticoagulants were compared. Accordingly, the results of single antiaggregant use were similar in terms of isolated thalamic stroke or stroke with extrathalamic involvement. However, isolated thalamic stroke was approximately 2.2 times higher than stroke with extrathalamic involvement in patients on dual antiaggregants. On the contrary, stroke with extrathalamic involvement was 2.3 times higher than isolated thalamic stroke in patients using oral anticoagulants. Accordingly, dual antiaggregant use was found to be a protective factor in terms of extrathalamic involvement. In addition, oral anticoagulant use was found to be a protective factor for isolated thalamic involvement. When these statistically significant differences obtained in terms of medical treatments are considered from an etiologic point of view, it was concluded that single antiaggregants used in the treatment of small vessel disease in the etiology of thalamic infarction did not make a significant difference in terms of isolated thalamic involvement and extrathalamic involvement. As a result, the choice of medications used for secondary prophylaxis due to previous cerebrovascular disease and/or coronary disease affected the infarct area in these patients. In patients with extrathalamic involvement, conditions such as stenosis, plaque, or dissection in large vessels increase extrathalamic involvement significantly.[19,20]

The study has some limitations. First, it is a retrospective study. Controlled prospective studies will be necessary to form a more definite opinion. Secondly, the areas of involvement of patients with extrathalamic participation are quite large and show differences. Finally, the

sample numbers in some subgroups are quantitatively low, although statistically significant.

In conclusion, it has been shown in this patient group that thalamic infarcts cannot be explained only by lacunar infarction etiology. Therefore, the etiology of thalamic infarcts should be investigated in more detail. In our study, results that were contrary to what is known in terms of medical treatment of thromboembolic processes were shown. Prospective studies with large samples are needed for more precise information.

Article Information Form

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Authors Contribution

Conceptualization, S.D.K., A.A.Y.
Methodology, S.D.K., E.Ö.G., E.A.
Investigation, S.D.K., M.V.
Resources, S.D.K., M.V., E.Ö.G.
Data curation, S.D.K., M.V., A.A.Y.
Writing—original draft preparation, S.D.K., A.A.Y., E.A.
Writing—review and editing, E.Ö.G., A.A.Y., M.V.
Supervision, E.Ö.G.
All authors have read and agreed to the published version of the manuscript.

The Declaration of Conflict of Interest/ Common Interest

No conflict of interest or common interest has been declared by authors.

The Declaration of Ethics Committee Approval

The Ethics Committee of the Antalya Education and Research Hospital approved this study (Number: 2024-152 KN: 7/11). Informed consent was obtained from all subjects and/or their legal guardians. All methods were carried out in accordance with relevant guidelines and regulations. All experiments were performed by relevant guidelines and regulations.

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REFERENCES

1. Bordes S, Werner C, Mathkour M, McCormack E, Iwanaga J, Loukas M, et al. Arterial supply of the thalamus: A comprehensive review. *World Neurosurg*. 2020 May;137:310-318. doi:10.1016/j.wneu.2020.01.237 Epub 2020 Feb 7. PMID: 32036065.
2. Habas C, Manto M, Cabaraux P. The cerebellar thalamus. *Cerebellum*. 2019 Jun;18(3):635-648. doi:10.1007/s12311-019-01019-3 PMID: 30827014.
3. Banerjee S, Shinde R, Sevick-Muraca EM. Probing static structure of colloid-polymer suspensions with multiply scattered light. *J Colloid Interface Sci.* 1999 Jan 01;209(1):142-153.
4. Atallah O, Almealawy YF, Alabide AS, Farooq M, Sanker V, Alrubaye SN, et al. Navigating the clinical landscape of artery of Percheron infarction: A systematic review. *eNeurologicalSci.* 2024 Aug 21;37:100521. doi:10.1016/j.ensci.2024.100521 PMID: 39257866; PMCID: PMC11382010.
5. Ortiz de Mendivil A, Alcalá-Galiano A, Ochoa M, Salvador E, Millán JM. Brainstem stroke: Anatomy, clinical and radiological findings. *Semin Ultrasound CT MR.* 2013 Apr;34(2):131-141. [PubMed]
6. Song YM. Topographic patterns of thalamic infarcts in association with stroke syndromes and aetiologies. *J Neurol Neurosurg Psychiatry.* 2011 Oct;82(10):1083-6. doi:10.1136/jnnp.2010.239624 Epub 2011 Mar 15. PMID: 21406535.
7. Knipe, H Jones, J et al. Thalamus http://radiopaedia.org/articles/thalamus Ve Gowda SN, Munakomi S, De Jesus O. Brainstem Stroke. In: StatPearls. Treasure Island (FL): StatPearls Publishing; February 25, 2024.
8. Wong KS, Caplan LR, Kim JS. Stroke mechanisms. *Front Neurol Neurosci*. 2016;40:58-71. doi:10.1159/000448302 Epub 2016 Dec 2. PMID: 27960181.
9. 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.

doi:10.1155/2014/154631 Epub 2014 Jul 2. PMID: 25100900; PMCID: PMC4101959.

10. Cereda C, Carrera E. Posterior cerebral artery territory infarctions. *Front Neurol Neurosci*. 2012;30:128-31. doi:10.1159/000333610 Epub 2012 Feb 14. PMID: 22377879.
11. Kuybu O, Tadi P, Dossani RH. Posterior cerebral artery stroke. 2023 Aug 8. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2025 Jan–. PMID: 30335329.
12. Chen XY, Wang Q, Wang X, Wong KS. Clinical features of thalamic stroke. *Curr Treat Options Neurol.* 2017 Feb;19(2):5. doi:10.1007/s11940-017-0441-x PMID: 28251587.
13. Song YM. Topographic patterns of thalamic infarcts in association with stroke syndromes and aetiologies. *J Neurol Neurosurg Psychiatry.* 2011 Oct;82(10):1083-6. doi:10.1136/jnnp.2010.239624 Epub 2011 Mar 15. PMID: 21406535.
14. Agarwal S, Chancellor B, Howard J. Clinical-radiographic correlates of Artery of Percheron infarcts in a case series of 6 patients. *J Clin Neurosci*. 2019 Mar;61:266-268. doi:10.1016/j.jocn.2018.11.030 Epub 2018 Nov 22. PMID: 30472345.
15. Li J, Ge J, Yang S, Yao G. Clinical review and analysis of artery of Percheron infarction. *IBRO Neurosci Rep.* 2023 May 27;15:17-23. doi:10.1016/j.ibneur.2023.04.006 PMID: 38204568; PMCID: PMC10776326.
16. Dogan SN, Cengel F, Bayrak AH, Yazgu R. Topographic evaluation of bithalamic infarcts: Are these due to occlusion of the artery of Percheron? *J Clin Neurosci.* 2021 Aug;90:99-104. doi:10.1016/j.jocn.2021.05.040 Epub 2021 Jun 7. PMID: 34275589.
17. Kato H, Izumiyama M, Izumiyama K, Takahashi A, Itoyama Y. Silent cerebral microbleeds on T2*-weighted MRI: correlation with stroke subtype, stroke recurrence, and leukoaraiosis. *Stroke*. 2002 Jun;33(6):1536-40. doi:10.1161/01.str.0000018012.65108.86 PMID: 12052987.
18. Chamorro A, Sacco RL, Mohr JP, Foulkes MA, Kase CS, Tatemichi TK, Wolf PA, Price TR, Hier DB. Clinical-computed tomographic correlations of lacunar infarction in the Stroke Data Bank. *Stroke*. 1991 Feb;22(2):175-81. doi:10.1161/01.str.22.2.175. PMID: 2003281
19. Prokopiv MM, Fartushna OY. Clinical syndromes of the thalamic stroke in the classical vascular territories: A prospective hospital-based cohort study. Wiad Lek. 2020;73(3):489-493. PMID: 32285819.
20. Pasterkamp G, Schoneveld AH, van Wolferen W, Hillen B, Clarijs RJ, Haudenschild CC, et al. The impact of atherosclerotic arterial remodeling on percentage of luminal stenosis varies widely within the arterial system. A postmortem study. Arterioscler Thromb Vasc Biol. 1997 Nov;17(11):3057-63. doi:10.1161/01.atv.17.11.3057 PMID: 9409293.