

# Risk factors for intracranial artery calcifications and their connections to cognitive impairment and cardiovascular illness

Nia Sharma

Sharma N. Risk factors for intracranial artery calcifications and their connections to cognitive impairment and cardiovascular illness. *J Neuropathol.* 2022; 2(5):48-9.

### ABSTRACT

Imaging frequently reveals arterial calcifications, although the precise implications are frequently ambiguous. The medial layer, which is made up of the tunica media and the internal elastic

lamina, is where arterial calcifications can be found. The risk factors for medial and intimal calcifications appear to vary. Intimal calcification risk variables included older age, smoking, hypertension, and a positive family history of vascular disorders, while medial calcification risk factors included diabetes mellitus and prior vascular disease in addition to older age and a positive family history. Intimal and medial calcifications have different effects in addition to having different risk factors.

**Key Words:** *Non-atherosclerotic*

### INTRODUCTION

The medial layer of the Intracranial Internal Carotid Artery (iICA) calcifications, in contrast, was histologically non-atherosclerotic, according to an autopsy research. Instead of stenosis, which causes persistent harm to the tissue the artery supplies, calcifications in this layer stiffen the artery and increase pulse wave velocity and pressure.

Studies did not distinguish between intimal and medial calcifications; instead, they simply looked at the clinical effects of arterial calcifications. Most research centered on calcifications in Coronary Arteries (CAC). It has been established that CAC is linked to both cerebrovascular disease and coronary artery disease. Some research has concentrated on calcifications in the intracranial arteries. These calcifications were linked to cerebrovascular illness, according to their research. Studies on cerebral artery calcifications in relation to vascular illness other than cerebrovascular disease are lacking. There is some evidence that intracranial artery calcifications are linked to cognitive outcomes including dementia and lower white matter and total brain volumes, which are all cognitively relevant.

In conclusion, the majority of earlier research on the clinical effects of arterial calcifications has concentrated on CAC. Furthermore, the distinctions between medial and intimal calcifications have rarely been considered in investigations on clinical outcomes. In spite of the fact that many individuals obtain imaging throughout their lives, it is unknown what would result from any discovered artery calcifications. For instance, brain imaging is typically performed on patients with

cognitive problems who are referred to a memory clinic using Magnetic Resonance Imaging (MRI) or Computed Tomography (CT). It is possible to measure artery calcifications, particularly using CT. So it is possible to look at the occurrence of intimal and medial intracranial artery calcifications, as well as risk factors and clinical outcomes of these calcifications, in the population of the memory clinic.

In this cross-sectional study, participants were drawn from the memory clinic at Tergooi Hospital in the Netherlands between April 2009 and April 2015. This study's sample size has already been discussed. Patients who did not have a CT scan were eliminated. The memory clinic team, which included a geriatrician, neurologist, neuropsychologist, and nurse, assessed each patient. Every patient completed a typical diagnostic process, which included physical and neurological examinations, evaluations of vital functions, and evaluations of educational attainment, cognitive testing, laboratory tests, electrocardiography, and brain imaging. The usual imaging procedure was a CT scan of the brain, and this was very seldom avoided.

This study demonstrated distinct risk factor profiles for intimal and medial dominant calcification patterns as well as the high prevalence of iICA calcifications in memory clinic patients. Stroke was linked to medial and intimal calcification patterns in iICA, as well as calcification severity. Intimal calcifications and the degree of calcifications in iICA were linked to myocardial infarction. Age and sex played a substantial role in the relationship between intracranial

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Editorial office, *Journal Of Neuropathology*, United Kingdom

Correspondence: Nia Sharma, Editorial office, *Journal Of Neuropathology*, United Kingdom, E-mail: [neuropathology@pulsusinc.com](mailto:neuropathology@pulsusinc.com)

Received: 4 September 2022, Manuscript No PULNP-22-5407; Editor assigned: 6 September 2022, PreQC No. PULNP-22-5407 (PQ); Reviewed: 19 September 2022, QC No. PULNP-22-5407 (Q); Revised: 22 September 2022, Manuscript No. PULNP-22-5407 (R); Published: 29 September 2022, DOI: 10.37532/pulnp.2022.2(5).48-9



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artery calcifications, the kind of cognitive disease, and cognitive performance. Our research contributes to the knowledge that there are several kinds of intracranial artery calcifications, which appear to be important for serious cardiovascular illnesses even though their function in dementia is still unclear.

The majority of the patients in our group had intracranial artery calcifications, which was a drawback of this study. However, even our smallest reference group included more than 100 participants. In our investigation, calcifications in the basilar artery were far less frequent. In the Netherlands, a community-based study examined the prevalence of vertebra basilar artery calcifications in the general population and discovered that basilar artery calcifications were likewise uncommon.

We were unable to measure the vertebral artery calcifications, which is a drawback. Another drawback was that we lacked data on the ECG, fasting glucose, HbA1c, and serum cholesterol levels. As a result, we might have overlooked some individuals who had high cholesterol, diabetes, or silent myocardial infarction. In this study, we discovered that medial and intimal iICA calcifications are mostly associated to vascular risk factors in various ways. Additionally, there were various correlations with cardiovascular illnesses. It is crucial to determine in which layer the calcifications occur since this may suggest that calcifications that are medially and initially positioned have different effects and may require different treatment methods. Future, long-term research is required to more fully explore the causes and effects of cerebral artery calcifications in people with memory disorders and in other contexts.