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Arterial Stiffness and Structure in Large Vessel Vasculitis: A Systematic Review with Meta-Analysis

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Abstract

Introduction: *Central artery stiffness and wall thickness are elevated in chronic states of inflammation and acutely-induced inflammatory responses. These conditions contribute to altered hemodynamics, increased vascular resistance, and impaired blood flow, potentially exacerbating the clinical outcomes. This study systematically reviewed central elastic artery stiffness as measured by pulse wave velocity (PWV) and wall thickening using carotid intima-media thickness (IMT) in patients with giant cell arteritis (GCA) and Takayasu arteritis (TAK).*

Methods: *Web of Science, PubMed (MEDLINE), and Cochrane library were systematically searched in October 2024. Twenty-two studies with 1,572 GCA or TAK patients met the criteria for meta-analysis.*

Results: *In GCA patients, carotid-femoral PWV (cfPWV) (SMD=0.23, 95%CI = [0.05, 0.41], P=0.001) and carotid artery IMT (MD=0.88, 95%CI = [0.03, 1.73], P=0.04) were higher than controls. Active GCA patients showed significantly higher carotid artery IMT (MD=1.55, 95%CI = [0.62, 2.49], P=0.001), while no significant difference was observed in inactive GCA patients (MD=0.88, 95%CI = [-0.5, 0.2], P=0.42). In TAK patients, cfPWV (SMD=0.84, 95%CI = [0.55, 1.13], P<0.00001), brachial-ankle PWV (SMD=1.03, 95%CI = [0.73, 1.33], P<0.00001), and carotid artery IMT (SMD=1.44, 95%CI = [1.20, 1.68], P<0.00001) were greater than in controls.*

Conclusion: *Patients with GCA or TAK demonstrated higher central artery stiffness and wall thickness than controls. Comprehensive cardiovascular risk management may be necessary for these patients even after controlling inflammation.*

Keywords: *Giant Cell Arteritis; Takayasu Arteritis; Arterial Stiffness; Atherosclerosis*

1. Introduction

Giant cell arteritis (GCA) and Takayasu arteritis (TAK) are rare large-vessel vasculitides that predominantly affect the aorta and its primary branches, leading to vascular inflammation, fibrosis, and stenosis [1]. The pathological processes in GCA and TAK involve immune-mediated inflammation that leads to intimal hyperplasia, disruption of the elastic lamina, collagen deposition, smooth muscle proliferation, and fibrosis [2]. Both TAK and GCA affect the aorta and its main branches. GCA primarily affects people over the age of 50 years and has a predilection for branches of the extracranial artery while TAK affects younger patients. These vascular changes could reduce the elasticity of arteries and elevate cardiovascular risks [3].

Similar to large vessel vasculitis, arterial stiffness is a clinical condition that predominantly affects the aorta and cardiothoracic arteries. Inflammation has been implicated in the pathophysiology of arterial stiffness [4]. We and others have demonstrated that central artery stiffness is elevated in chronic states of inflammation (e.g., Lupus) and acutely-induced inflammatory responses (e.g., eccentric exercise-induced muscle damage) [5-8]. Previous studies that have examined arterial stiffness in large vessel vasculitis are inadequate, potentially due to difficulty in imaging analysis [9], variability in study design, patient demographics, and disease stages [10, 11], making it difficult to conclude whether the vascular dysfunctions are greater in GCA and TAK patients. Additionally, it is unknown if arterial wall thickening in large vessel vasculitis is related to arterial stiffness in this patient population. Therefore, this systematic review and meta-analysis is aimed to evaluate the levels of central artery stiffness and carotid wall thickness in patients with GCA and TAK.

2. Methods

2.1. Protocol and Registration

A systematic review of studies focusing on macrovascular function and structure including arterial stiffness and carotid artery intima-media thickness in patients with large vessel vasculitis was undertaken. Endothelium-dependent vasodilation as assessed by flow-mediated dilation was initially included in the literature search. However, due to a lack of a sufficient number of quality studies, this measure was dropped from the analyses. This study was registered in the international prospective register of systematic reviews (PROSPERO) with a registration number of CRD42024603914. This review followed the preferred reporting items for systematic reviews and meta-analyses (PRISMA) guidelines [12].

2.2. Eligibility Criteria

The inclusion criteria for this study were as follows: 1) patients clinically diagnosed with GCA or TAK confirmed by clinical, imaging, or histological methods, 2) studies reporting PWV and/or IMT as a measure of vascular function or structure, 3) observational studies (cross-sectional, cohort, or case-control) and clinical trials providing data on PWV and/or IMT in GCA or TAK patients, 4) the vascular measures, PWV and/or IMT, in GCA or TAK patients were compared with healthy control group or disease control group who have other forms of vasculitis, vascular conditions, or in different phases of GCA or TAK.

The exclusion criteria included: 1) animal studies or in vitro research, 2) studies not specifically reporting data on PWV and/or IMT, 3) case reports or case series with fewer than 10 patients, 4) studies mainly involving other types of vasculitis, such as anti-neutrophilic cytoplasmic antibody-associated vasculitis, without specific subgroup data for GCA or TAK, and 5) studies published in languages other than English.

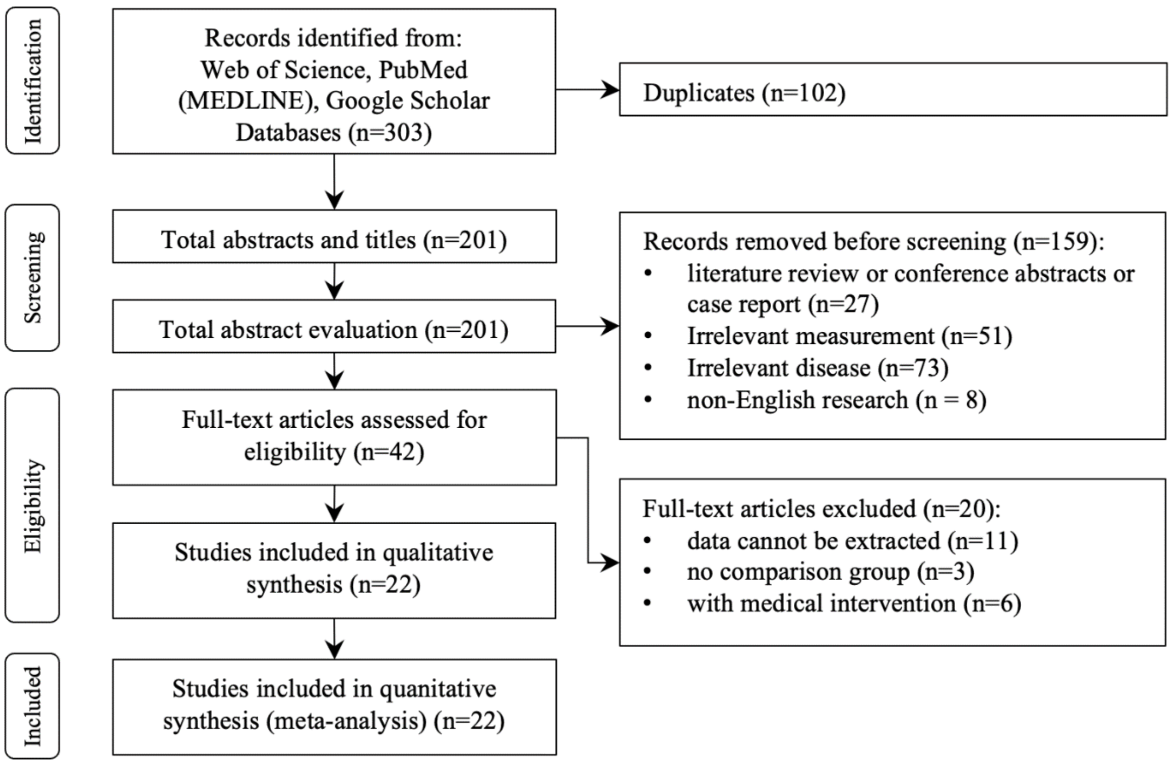

Fig 1. *Research screening process*

2.3. Search Strategy

A variety of databases including Web of Science core collection, PubMed (MEDLINE), and Cochrane library were systematically searched from the inception to October 2024. To ensure the inclusion of recent and relevant data, search filters were applied to prioritize studies published within the last ten years.

To enhance its specificity, medical subject headings derived from PubMed was carefully paired with additional keywords relevant to the topic.

Search strategy for Web of Science is demonstrated as below: TS=((((Giant Cell Arteritis) OR (Giant Cell Arteritides) OR (Horton Disease) OR (Horton's Disease) OR (Hortons Disease) OR (Horton's Giant Cell Arteritis) OR (Horton Giant Cell Arteritis) OR (Giant Cell Aortitides) OR (Giant Cell Aortic Arteritis) OR (Giant Cell Aortitis) OR (Juvenile Temporal Arteritis) OR (Juvenile Temporal Arteritides) OR (Cranial Arteritis) OR (Cranial Arteritides) OR (Temporal Arteritis) OR (Temporal Arteritides)) OR ((Takayasu Arteritis) OR (Pulseless Disease) OR (Takayasu Disease) OR (Takayasu Syndrome) OR (Takayasu's Arteritis) OR (Young Female Arteritis) OR (Young Female Arteritides) OR (Aortitis Syndrome))) AND (((Carotid Intima-Media Thickness) OR (Carotid Intima Media Thickness) OR (IMT) OR (Vascular Stiffness) OR (Arterial Stiffness) OR (Aortic Stiffness) OR (Carotid-Femoral Pulse Wave Velocity) OR (Carotid Femoral Pulse Wave Velocity) OR (Carotid-Femoral Pulse Wave Velocities) OR (Carotid Femoral Pulse Wave Velocities) OR (Brachial-Ankle Pulse Wave Velocity) OR (Brachial-Ankle Pulse Wave Velocities) OR (Brachial Ankle Pulse Wave Velocity) OR (Brachial Ankle Pulse Wave Velocities) OR (Pulse Wave Velocity) OR (Pulse Wave Velocities) OR (PWV)))).

2.4. Data Extraction

Two independent reviewers screened the titles and abstracts of the retrieved studies and extracted data. A third, senior reviewer was consulted to resolve discrepancies between reviewers. Duplications were excluded by employing the built-in duplicate detection feature in EndNote X9 software (Clarivate Analytics, Philadelphia, PA, USA) based on similarities in titles, authors, and publication years. Additional manual searches of the reference lists of recent review articles were conducted to identify any additional studies not captured by database search algorithms. The extracted information included publication year, disease type, participant information (gender/sex, age, disease duration, disease phase, and body mass index), comparison groups, and main outcomes.

2.5. Publication Bias and Research Quality Evaluation

The quality of the included studies was assessed using the Newcastle-Ottawa Scale [13]. No studies were classified as low quality (score <4), suggesting that the studies included in this analysis generally demonstrated adequate methodological rigor and a lower risk of bias.

2.6. Statistical Analyses

Statistical analyses were performed using RevMan 5.4 software (The Cochrane Collaboration, Copenhagen, Denmark). Standard mean difference (SMD) was used as the effect analysis statistic and the 95% confidential interval (CI) was provided when $p < 0.05$ was considered as a statistically significant difference. The heterogeneity was quantitatively determined by I^2. If there was no statistical heterogeneity among the results, the fixed effect model was used for meta-analysis. I^2 over 50 among the results was considered substantial heterogeneity. After excluding the influence of obvious clinical heterogeneity, the random effects model was used for meta-analysis. Subgroup analysis was applied if applicable.

3. RESULTS

3.1. Search Results and Characteristics of Included Research

Table 1. *Newcastle-Ottawa Scale for studies in meta-analysis*

Studies	Selection				Comparability	Exposure			Total quality score
	Is the case definition adequate?	Representativeness of the cases	Selection of controls	Definition of controls	Comparability of cases and controls	Ascertainment of exposure	Same method of ascertainment for cases and controls	Non-response rate	
Jud *et al.* [17]	1	1	0	1	1	1	0	0	5
Monjo-Henry *et al.* [20]	1	1	0	1	2	0	0	0	5
Milchert *et al.* [19]	1	1	0	1	0	1	0	0	4
Jud *et al.* [18]	1	1	0	1	1	0	0	0	4
Ješe *et al.*[16]	1	1	0	1	2	1	1	0	7

Hafner *et al* [15]	1	1	0	1	2	1	0	0	6
Gonzalez-Juanatey *et al* [14]	1	1	1	1	2	1	0	0	7
Ucar *et al.* [29]	1	1	1	1	2	0	0	0	6
Svensson *et al.* [28]	1	0	0	1	2	1	1	0	6
Chen *et al.* [22]	1	1	0	1	2	1	0	0	6
Zhang *et al.* [34]	1	1	0	1	2	1	1	0	7
Wang *et al.* [31]	1	1	0	1	2	0	0	0	5
Zhao *et al.* [35]	1	1	0	1	2	1	1	0	7
He *et al.* [24]	1	1	0	1	2	1	1	0	7
Yurdakul *et al.* [33]	1	0	0	1	2	0	0	0	4
Yang *et al.* [32]	1	1	0	1	2	1	0	0	6
Wang *et al.* [30]	1	1	0	1	2	0	0	0	5
Neto *et al.* [25]	1	1	0	1	2	0	0	0	5
Alibaz-Oner *et al.* [47]	1	0	0	1	2	1	0	0	5
Cho *et al.* [23]	1	0	0	1	2	0	0	0	4
Ng *et al.* [48]	1	1	0	1	1	0	0	0	4
Raninen *et al.* [27]	1	1	0	1	2	1	0	0	6

Each study is evaluated on the Newcastle-Ottawa Scale assessing three broad perspectives: Selection (4 pts), Comparability (2 pts), and Exposure (3 pts). Total quality scores range from 0 to 9, with higher scores indicating higher quality methodology

A total of 303 records were obtained after research screening. A total of 1,572 participants in 22 studies, including 736 GCA patients in 7 studies [14-20] and 836 TAK patients in 15 studies [21-35]. The research screening process is shown in **Fig. 1**. The characteristics of the included studies of PWV and IMT for meta-analysis are shown in **Table 2**.

Table 2. *Characteristics of included studies for the meta-analysis*

Studies	Year	Disease	Participants				Control group	Main outcomes
			Men/ Women (n)	Age (year) (mean ± SD)	Disease phase (active/inactive)	BMI (kg/m^2) (mean ± SD)		
Jud *et al.* [17]	2023	GCA	32/106	74.5 ± 7.7	0/138	26.5 ± 4.7	inactive cancer or suspected pulmonary embolism	cfPWV; carotid IMT
Monjo-Henry *et al.* [20]	2023	GCA	20/24	72.8 ± 7.6	44/0	27.1 ± 3.8	high cardiovascular risk	carotid IMT
Milchert *et al.* [19]	2022	GCA	28/53	73.0 ± 9.0	mixed	/	healthy	carotid IMT
Jud *et al.* [18]	2021	GCA	33/111	74.7	0/144	26.5 ± 4.6	inactive cancer or suspected pulmonary embolism	cfPWV; carotid IMT

Ješe *et al.* [16]	2021	GCA	87/161	75.2 ± 9.8	248/0	/	healthy	carotid IMT
Hafner *et al.* [15]	2014	GCA	13/28	78.4 ± 6.8	41/0	24.6 ± 4.2	healthy	carotid IMT
Gonzalez-Juanatey *et al.* [14]	2007	GCA	15/25	72.8 ± 6.7	0/40	/	healthy	carotid IMT
Ucar *et al.* [29]	2023	TAK	6/44	39.8 ± 8.2	20/30	26.1 ± 5.4	healthy	carotid IMT
Svensson *et al.* [28]	2022	TAK	3/17	41.7 ± 14.5	0/20	27.6 ± 4.5	healthy	carotid IMT
Chen *et al.* [22]	2022	TAK	28/154	35.3 ± 13.3	mixed	26.1 ± 33.6	healthy	baPWV
Zhang *et al.* [34]	2022	TAK	3/49	38.2 ± 12.7	23/29	/	inactive TAK	cfPWV
Wang *et al.* [31]	2020	TAK	0/67	39.7 ± 9.3	43/24	25.5 ± 3.1	healthy	baPWV
Zhao *et al.* [35]	2019	TAK	1/21	32	11/9	/	inactive	carotid IMT
He *et al.* [24]	2019	TAK	40/200	34 ± 14.8	64/176	22.7±3.3	inactive TAK	
Yurdakul *et al.* [33]	2018	TAK	2/31	39.1 ± 11.4	12/21	/	healthy	carotid IMT
Yang *et al.* [32]	2017	TAK	0/25	28.3 ± 6.2	25/0	20.7 ± 2.9	healthy	cfPWV
Wang *et al.* [30]	2015	TAK	12/36	45.0 ± 6.7	0/48	25.2 ± 5.6	coronary artery disease	baPWV
Neto *et al.* [25]	2014	TAK	0/27	32.4 ± 8.3	15/12	22.3 ± 2.6	healthy	cfPWV
Alibaz-Oner *et al.* [47]	2014	TAK	2/30	39.1 ± 11.4	11/21	/	healthy	carotid IMT
Cho *et al.* [23]	2010	TAK	1/11	38.0 ± 10.0	2/10	22.0 ± 2.7	healthy	carotid IMT
Ng *et al.* [48]	2006	TAK	0/10	40.8 ± 13.2	mixed	26.3 ± 3.1	healthy	cfPWV
Raninen *et al.* [27]	1996	TAK	2/14	45.7	4/12	/	healthy	carotid IMT

BMI = body mass index, GCA = Giant cell arteritis; TAK = Takayasu arteritis; cfPWV = carotid-femoral pulse wave velocity; baPWV = brachial-ankle pulse wave velocity; IMT = intima-media thickness

3.2. PWV in GCA

Carotid-femoral PWV (cfPWV) in GCA was shown in two studies [17, 18] (**Fig. 2a**) with non-significant heterogeneity (I^2 = 0). The fixed model was applied accordingly. In patients with GCA, cfPWV (SMD=0.23, 95%CI = [0.05, 0.41], P=0.001) was significantly higher than in controls.

3.3. Carotid Artery IMT in GCA

Carotid artery IMT in GCA were indicated in 5 studies [14, 17-20] as shown in **Fig. 2b**. A random model was applied based on the heterogeneity (I^2 = 93). Carotid artery IMT (MD=0.88, 95%CI = [0.03, 1.73], P=0.04) was significantly higher in patients with GCA than in controls. Subgroup analysis of difference disease stages (active/inactive) was performed in **Fig. 2c**.

Active GCA patients showed significantly higher carotid artery IMT (MD=1.55, 95%CI = [0.62, 2.49], P=0.001), while no significant difference was observed in inactive GCA patients (MD=0.88, 95%CI = [-0.5, 0.2], P=0.42).

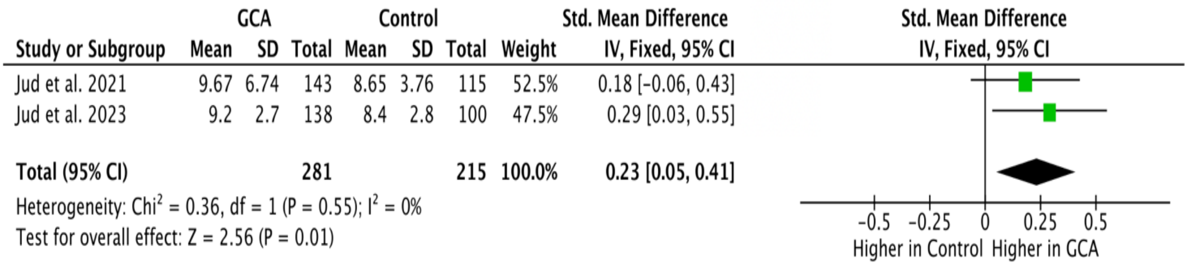

Fig 2a. *cfPWV in GCA*

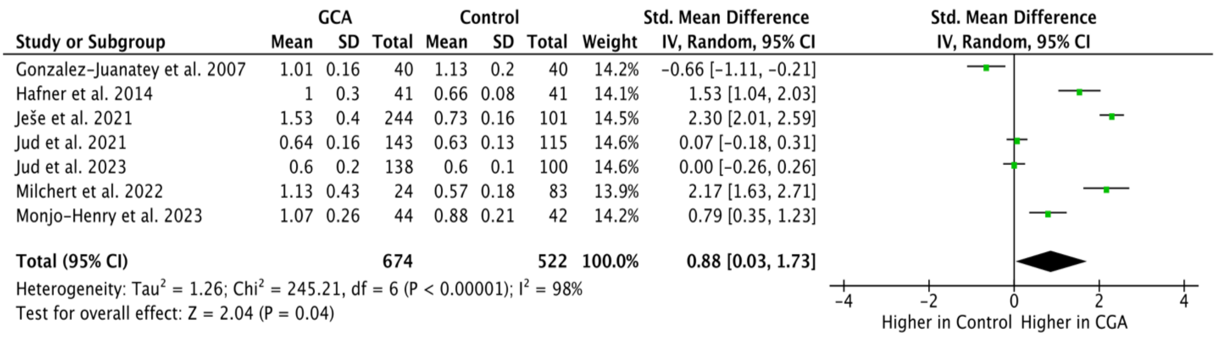

Fig 2b. *carotid artery IMT in GCA*

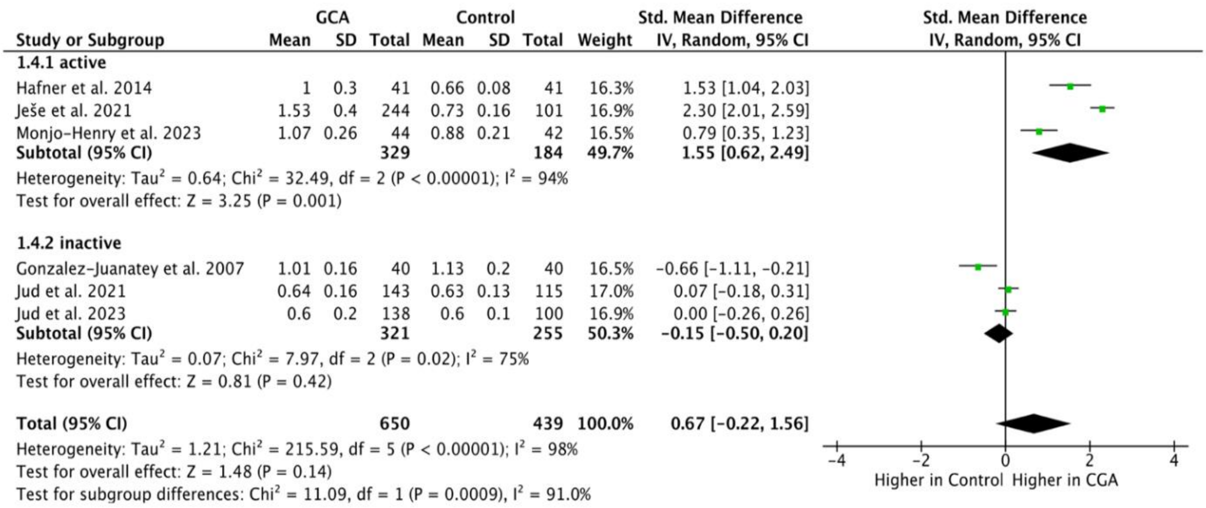

Fig 2c. *Carotid artery IMT in GCA (subgroup by disease phase)*

Fig 2. *Arterial stiffness as assessed by carotid-femoral pulse wave velocity (cfPWV) and carotid artery intima-media thickness (IMT) in giant cell arteritis (GCA)*

3.4. PWV in TAK

A total of 8 studies were included to show cfPWV [25, 26, 32, 34] and brachial-ankle PWV (baPWV) [22, 24, 30, 31] in TAK as displayed in **Fig. 3a** and **Fig. 3b**. While a fixed model was applied in cfPWV (I^2 = 0) due to low heterogeneity, a random model was used in baPWV (I^2 = 85).

Both cfPWV (SMD=0.84, 95%CI = [0.55, 1.13], P<0.00001) and baPWV (SMD=1.03, 95%CI = [0.73, 1.33], P<0.00001) were greater in patients with TAK than in controls.

3.5. Carotid Artery IMT in TAK

As shown in **Fig. 3c**, 7 studies were included to show carotid artery IMT [21, 23, 27-29, 33, 35] in patients with TAK. Low heterogeneity among the included studies was found in the analysis (I^2 = 35). Therefore, the fixed effect models were applied for analysis.

Carotid artery IMT was higher in patients with TAK than in controls (SMD=1.44, 95%CI = [1.20, 1.68], P<0.00001).

Study or Subgroup	TAK Mean	TAK SD	TAK Total	Control Mean	Control SD	Control Total	Weight	Std. Mean Difference IV, Fixed, 95% CI
Neto et al. 2014	9.77	3.49	27	7.83	1.06	27	27.7%	0.74 [0.19, 1.29]
Ng et al. 2006	12	4.3	10	8.3	1.1	11	9.6%	1.16 [0.22, 2.10]
Yang et al. 2017	8.37	2.23	25	6.46	1.15	25	23.9%	1.06 [0.46, 1.65]
Zhang et al. 2022	10.45	5.11	52	7.2	3.66	29	38.8%	0.69 [0.23, 1.16]
Total (95% CI)			**114**			**92**	**100.0%**	**0.84 [0.55, 1.13]**

Heterogeneity: $Chi^2 = 1.47$, df = 3 (P = 0.69); $I^2 = 0\%$
Test for overall effect: Z = 5.65 (P < 0.00001)

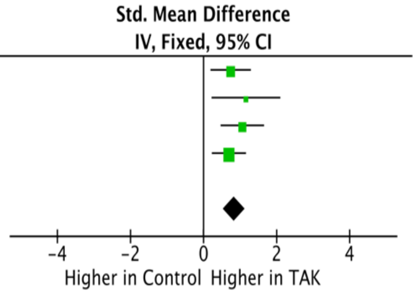

Fig 3a. *cfPWV in TAK*

Study or Subgroup	TAK Mean	TAK SD	TAK Total	Control Mean	Control SD	Control Total	Weight	Std. Mean Difference IV, Random, 95% CI
Chen et al. 2022	13.67	11.17	182	11.15	2.53	182	28.3%	0.31 [0.10, 0.52]
He et al. 2019	15.05	6.51	74	12.75	2.64	166	26.8%	0.54 [0.26, 0.82]
Wang et al. 2015	16.6	2.82	48	12.8	1.51	25	19.9%	1.53 [0.98, 2.08]
Wang et al. 2020	14.92	4.31	67	12.11	1.54	67	24.9%	0.86 [0.51, 1.22]
Total (95% CI)			**371**			**440**	**100.0%**	**0.75 [0.34, 1.17]**

Heterogeneity: $Tau^2 = 0.15$; $Chi^2 = 20.52$, df = 3 (P = 0.0001); $I^2 = 85\%$
Test for overall effect: Z = 3.56 (P = 0.0004)

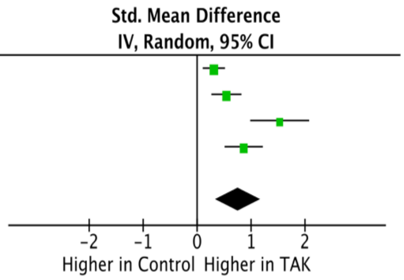

Fig 3b. *baPWV in TAK*

Study or Subgroup	TAK Mean	TAK SD	TAK Total	Control Mean	Control SD	Control Total	Weight	Std. Mean Difference IV, Fixed, 95% CI
Alibaz-Oner et al. 2014	1.1	0.3	32	0.68	0.8	18	16.4%	0.77 [0.18, 1.37]
Cho et al. 2010	1.16	0.48	23	0.46	0.07	24	11.5%	2.03 [1.31, 2.74]
Raninen et al. 1996	1.23	0.52	18	0.74	0.14	18	11.3%	1.26 [0.54, 1.98]
Svensson et al. 2022	1.03	0.41	17	0.52	0.11	17	9.3%	1.66 [0.87, 2.45]
Ucar et al. 2023	0.96	0.39	50	0.52	0.1	57	30.8%	1.58 [1.14, 2.02]
Yurdakul et al. 2018	1.1	0.3	33	0.7	0.09	20	14.3%	1.62 [0.97, 2.26]
Zhao et al. 2019	2.4	0.7	9	1.8	0.4	11	6.5%	1.04 [0.09, 1.99]
Total (95% CI)			**182**			**165**	**100.0%**	**1.44 [1.20, 1.68]**

Heterogeneity: $Chi^2 = 9.26$, df = 6 (P = 0.16); $I^2 = 35\%$
Test for overall effect: Z = 11.66 (P < 0.00001)

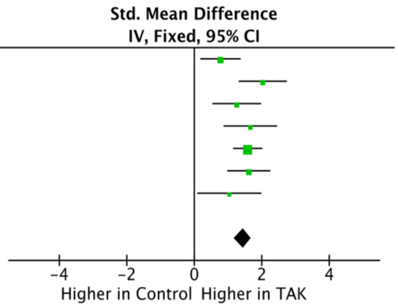

Fig 3c. *Carotid artery IMT in TAK*

Fig 3. *Arterial stiffness as assessed by carotid-femoral (cfPWV) and brachial-ankle PWV (baPWV) and carotid artery intima-media thickness (IMT) in Takayasu arteritis (TAK)*

4. Discussion

In the present systematic review and meta-analysis, we found that both GCA and TAK were associated with higher central artery stiffness and wall thickness as measured by PWV and carotid artery IMT, demonstrating elevated cardiovascular risks in patients with large vessel vasculitis. Even though treatments of the underlying vasculitis can reduce inflammation, the structural changes in the arterial wall may persist even after disease activity is controlled. The results of the present study indicate that comprehensive cardiovascular risk management may be necessary for these patients even after inflammation is controlled. Two vascular measurements included in the present study, PWV and IMT, are well-established markers of cardiovascular health. The observed increases in cfPWV in GCA and TAK suggest that systemic inflammation could lead to the stiffening of central arteries. Elevated cfPWV is associated with higher cardiovascular morbidity and mortality [36]. Additionally, the increase in baPWV reflects both central and peripheral vascular stiffness, highlighting a systemic issue in vascular health among TAK patients [24, 37]. Arterial stiffening in large vessel vasculitis was associated with the corresponding elevation in carotid wall thickness in the present study. The significant elevation in carotid artery IMT combined with increased values of PWV among patients with large vessel vasculitis may reflect the relation between systemic inflammation and vascular remodeling [38]. The measurement of carotid artery IMT serves as a surrogate marker for atherosclerosis [39], and its elevation suggests an accelerated process of atherosclerosis [40], which may predispose patients to cardiovascular events/diseases.

The immune-induced vascular inflammation in large vessel vasculitis can contribute to arterial stiffness because of the elevated cytokines led by granulomatous formation and proliferation of smooth muscle cells in arterial walls [2]. This is consistently reflected in the elevated PWV in both GCA and TAK in the present study. Stiffened arteries are associated with reduced nitric oxide bioavailability [41], a crucial vasodilator in inhibiting vascular smooth muscle contraction and maintaining vascular homeostasis [42], as well as increased production of reactive oxygen species [43, 44]. Therefore, emphasizing the critical role of managing inflammatory markers for arterial stiffness is important in improving cardiovascular outcomes in patients with large vessel vasculitis [38].

Combined with the extremely low disease rate [1] and diagnosis delay [45], research targeting cardiovascular health in patients with large vessel vasculitis is quite limited. As no systematic study has been found focusing on PWV and/or IMT in patients with GCA, to our knowledge, this is the first systematic review with meta-analysis to evaluate PWV and IMT in large vessel vasculitis. Compared with the existing studies [11, 46], the present study systematically included a larger number of studies of both GCA and TAK in the meta-analysis, providing an updated and more comprehensive analysis for this patient population. However, there are several limitations that must be emphasized. The limited number of studies in this field restrained the literature screening and inclusion strategy. Additionally, variabilities in study design and patient demographics could have introduced biases. Despite no significant heterogeneity was found for cfPWV and baPWV, high heterogeneity of carotid artery IMT of GCA and baPWV of TAK indicate some variabilities in results. Subgroup analysis was applied based on different disease stages in IMT of GCA. The significance for subgroup differences (P=0.0009) suggests that the disease stage (active vs. inactive) substantially influences the observed outcomes. This supports the need for separate analyses by disease stage. Despite this, we found that inactive GCA patients showed similar carotid artery IMT with normal control while that in active GCA patients were significantly higher, heterogeneity remains high in both subgroups. However, similar to baPWV of TAK, due to the insufficient studies, subgroups of control groups, age, or disease duration are not applicable.

Therefore, further high-quality original investigations are essential to better understand cardiovascular risk progression in GCA and TAK populations.

5. Conclusion

Patients with large vessel vasculitis showed elevated arterial stiffness and wall thickness. Future research should focus on elucidating the mechanisms underlying arterial stiffness and atherosclerosis in large vessel vasculitis, as well as exploring potential therapeutic interventions to monitor cardiovascular health in GCA and TAK management.

6. Funding

This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

7. Author Contributions

Conceptualization: Yanbing Zhou, Hirofumi Tanaka; Methodology: Yanbing Zhou, Lin-Sheng Chen, Writing - original draft preparation: Yanbing Zhou; Writing - review and editing: Yanbing Zhou, Lin-Sheng Chen, Curry L. Koening, Hirofumi Tanaka. All authors have read and agreed to the published version of the manuscript.

8. Data Availability Statement

The data presented in this study are from the included trials and are publicly available.

9. Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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