The American Journal of Medical Sciences and Pharmaceutical Research
ISSN 2689-1026 | Open Access

TYPE Original Research
PAGE NO. 71-75
DOI 10.37547/tajmspr/Volume07Issue10-09

Pathogenetic Interrelations Of Inflammation And Dyslipidemia In Atherothrombotic Lesions Of Extracranial Arteries

Nargiza Abdullaeva
Samarkand State Medical University, Uzbekistan

Sardorbek Yuldashev
Samarkand State Medical University, Uzbekistan

OPEN ACCESS

SUBMITTED 26 August 2025
ACCEPTED 21 September 2025
PUBLISHED 31 October 2025
VOLUME Vol.07 Issue 10 2025

CITATION

Nargiza Abdullaeva, & Sardorbek Yuldashev. (2025). Pathogenetic Interrelations Of Inflammation And Dyslipidemia In Atherothrombotic Lesions Of Extracranial Arteries. The American Journal of Medical Sciences and Pharmaceutical Research, 7(10), 71–75.
https://doi.org/10.37547/tajmspr/Volume07Issue10-09

Abstract

Atherothrombotic lesions of the extracranial arteries represent one of the main causes of ischemic stroke and are strongly associated with systemic inflammation and dyslipidemia. This study aimed to analyze the pathogenetic interrelations between inflammatory and lipid markers in patients with varying degrees of atherothrombotic extracranial artery stenosis. A total of 128 elderly patients (mean age 67 ± 7.2 years) diagnosed with internal carotid artery (ICA) stenosis were examined between 2022 and 2024. Based on lesion type, patients were divided into three groups: unilateral ICA stenosis (Group I, n = 41), bilateral ICA stenosis (Group II, n = 58), and combined ICA + vertebral artery stenosis (Group III, n = 29), with an additional control group (n = 20).

Biochemical markers such as high-sensitivity C-reactive protein (hs-CRP), interleukin-6 (IL-6), and lipid profile parameters (LDL, HDL, total cholesterol, triglycerides) were measured and statistically analyzed using IBM SPSS 25.0, MedCalc 20.0, and R 4.2.2. The mean hs-CRP and IL-6 values showed a significant progressive increase from Group I to III ($p < 0.05$), reflecting enhanced systemic inflammation with advancing stenosis. Total cholesterol and LDL levels also increased significantly, while HDL levels showed a nonsignificant downward trend.

These findings demonstrate that chronic inflammation and dyslipidemia act synergistically in promoting atherothrombotic progression and plaque instability. The most severe biochemical alterations were observed in patients with combined ICA and vertebral artery stenosis, indicating a more generalized and aggressive form of vascular pathology. Regular monitoring of inflammatory and lipid markers is recommended for early identification of high-risk patients, better prediction of stroke risk, and optimization of therapeutic strategies.

Keywords: Atherothrombosis, extracranial arteries, inflammation, dyslipidemia, interleukin-6, C-reactive protein, LDL, HDL, ischemic stroke, carotid stenosis, vascular pathology.

Introduction

Atherothrombotic lesions of the extracranial arteries of the brain are among the leading causes of ischemic stroke, which ranks second worldwide as a cause of mortality and remains the primary factor contributing to disability in the adult population [1, 2]. The principal pathogenetic mechanisms of ischemic stroke in this condition include local hemodynamic insufficiency caused by arterial stenosis and artery-to-artery embolism resulting from rupture or thrombosis of an atherosclerotic plaque [3].

Systemic inflammation and dyslipidemia play a crucial role in the progression of the atherothrombotic process, mutually reinforcing one another and creating an unfavorable proatherogenic background [4–6]. Inflammation promotes endothelial activation, migration of macrophages and lymphocytes into the vascular intima, and increased expression of proinflammatory cytokines and proteolytic enzymes, leading to plaque destabilization and elevating the risk of rupture [7].

Dyslipidemia, characterized by elevated levels of low-density lipoproteins (LDL) and triglycerides along with decreased levels of high-density lipoproteins (HDL), is a key factor in the development and progression of atherosclerotic plaques [8, 9]. High-sensitivity C-reactive protein (hs-CRP) is considered both a marker of vascular inflammation activity and an independent predictor of cardiovascular events [10].

Clinical manifestations of atherothrombotic lesions of the extracranial arteries range from asymptomatic progression to transient ischemic attacks (TIA) and ischemic stroke. The most severe forms of the disease are observed in patients with marked dyslipidemia, elevated inflammatory markers, and hemodynamically significant stenosis [11].

Despite numerous studies aimed at identifying stroke risk factors, the pathogenetic interrelations among systemic inflammation, blood lipid spectrum, and clinical characteristics of atherothrombotic extracranial artery lesions remain insufficiently elucidated in real-world clinical practice.

Therefore, identification and comprehensive evaluation of these interrelations are of significant clinical importance, as they help clarify the pathogenetic role of inflammation and dyslipidemia, improve stroke risk prediction, and optimize preventive and therapeutic strategies for patients with this pathology.

Materials and Methods.

A total of 128 elderly patients (according to WHO classification, 2021) with a confirmed diagnosis of internal carotid artery (ICA) stenosis were examined. The patients were under outpatient observation at the private clinic Reacentr Nukus and received inpatient treatment in the neurology department of Nukus City Hospital during the period from 2022 to 2024. Among them were 60 men (46.9%) and 68 women (53.1%), with a mean age of 67 ± 7.2 years (Table 2.1). A randomized, heterogeneous, and combined sampling method was applied to form the study cohort.

Depending on the type of atherothrombotic lesion, patients were divided into three main groups (Table 1):

- Group I: patients with unilateral ICA stenosis (n = 41);
- Group II: patients with bilateral ICA stenosis (n = 58);
- Group III: patients with combined stenosis of ICA and vertebral arteries (VA) (n = 29).

The control group consisted of patients without hemodynamically significant stenoses of the extracranial arteries (n = 20). The degree of stenosis was assessed according to the NASCET/SRU classification criteria.

Among the 128 patients included in the second phase of the study, the majority belonged to Group II (n = 58; 45.3%), followed by Group I (n = 41; 32.0%) and Group III (n = 29; 22.7%). The age category of 60–74 years

(elderly) predominated across all groups—51.2% in Group I, 51.7% in Group II, and 48.3% in Group III—confirming that clinically significant stenoses are most frequently detected at an older age, when the cumulative burden of risk factors reaches its peak.

The 45–59 years age group (middle-aged) occurred less frequently but remained relatively stable across all groups: 24.4% in Group I, 25.9% in Group II, and 24.1% in Group III. The 75–90 years age group (senile age) accounted for 24.4% of patients in Group I, 22.4% in Group II, and 27.6% in Group III. The higher proportion of elderly patients in Group III indicates systemic progression of atherosclerosis, involving not only the internal carotid but also the vertebral arteries in advanced age categories (Table 1).

In Groups I and II, men and women were distributed almost equally (51.2% men vs. 48.8% women in Group I; 50% each in Group II). In Group III, a slight predominance of men (55.2%) was observed, confirming the tendency toward more pronounced and widespread atherosclerosis in the male population.

Table 1.

Distribution of patients by age, sex, and groups (Stage II)

Age group	Sex	Group I (n=41)		Group II (n=58)		Group III (n=29)		Total (n=128)	
		abs.	%	abs.	%	abs.	%	abs.	%
Middle age (45–59 years)	Men	5	12.2%	8	13.8%	4	13.8%	17	13.3%
	Women	5	12.2%	7	12.1%	3	10.3%	15	11.7%
	Total	10	24.4%	15	25.9%	7	24.1%	32	25.0%
Elderly (60–74 years)	Men	10	24.4%	14	24.1%	7	24.1%	31	24.2%
	Women	11	26.8%	16	27.6%	7	24.1%	34	26.6%
	Total	21	51.2%	30	51.7%	14	48.3%	65	50.8%
Senile (75–90 years)	Men	6	14.6%	7	12.1%	5	17.2%	18	14.1%
	Women	4	9.8%	6	10.3%	3	10.3%	13	10.2%
	Total	10	24.4%	13	22.4%	8	27.6%	31	24.2%
Total	Men	21	51.2%	29	50.0%	16	55.2%	66	51.6%
	Women	20	48.8%	29	50.0%	13	44.8%	62	48.4%
	Overall total	41	100.0%	58	100.0%	29	100.0%	128	100.0%

Across all age categories, men accounted for approximately 51.6% of the total sample, and women for 48.4%, which corresponds to the general epidemiological trend [Markus et al., 2010; Howard et al., 2015]. The age group of 60–74 years was predominant among all patients, which is associated with the cumulative impact of risk factors and age-related structural changes in the vascular wall.

The gender distribution was relatively balanced; however, in Group III (combined stenosis of the internal carotid and vertebral arteries), a slight predominance of men was noted, which may indicate a higher susceptibility to multifocal atherosclerosis in the male

population. Middle-aged and elderly patients require special attention regarding preventive measures and dynamic observation, as these age categories are at the highest risk for stroke and cognitive impairment.

Biochemical markers of inflammation and lipid metabolism are used to detect unstable atherosclerotic plaques, assess the activity of inflammatory processes, and stratify the risk of stroke. In this study, special attention was given to high-sensitivity C-reactive protein (hs-CRP), interleukin-6 (IL-6), and the lipid profile (LDL, HDL, triglycerides, and total cholesterol).

Statistical analysis of the data was performed using IBM SPSS Statistics 25.0, MedCalc 20.0, and R (v.4.2.2) software.

Results of the study. Modern understanding of atherothrombosis pathogenesis highlights the role of chronic inflammation and dyslipidemia as key factors contributing to the progression of atherosclerotic vascular changes. In this study, the levels of high-sensitivity C-reactive protein (hs-CRP), interleukin-6 (IL-6), and lipid spectrum parameters were analyzed in patients with varying degrees and localization of atherothrombotic lesions (Table 2).

The mean hs-CRP values showed a consistent increase—from 3.2 ± 1.1 mg/L in Group I to 4.1 ± 1.3 mg/L in Group III. This elevation was statistically significant in Groups II and III compared to Group I ($p = 0.029$ and $p = 0.012$, respectively), reflecting the growth of systemic inflammatory response with the progression of arterial stenosis. The obtained values remained within the upper normal range, which may indicate latent inflammation associated with vascular pathology.

Table 2. Biochemical markers (CRP, IL-6, and lipid profile) in patients with different types of ICA and VA stenosis

Indicators	Group I	Group II	Group III
CRP (mg/L)	3.2 ± 1.1 [3.0–3.4]	3.8 ± 1.2 [3.5–4.1]	4.1 ± 1.3 [3.7–4.5]
IL-6 (pg/mL)	6.8 ± 2.0 [6.4–7.2]	7.5 ± 2.2 [7.0–8.0]	7.9 ± 2.3 [7.4–8.4]
Total cholesterol (mmol/L)	5.4 ± 1.0 [5.2–5.6]	5.6 ± 1.1 [5.3–5.9]	5.7 ± 1.2 [5.3–6.1]
LDL (mmol/L)	3.2 ± 0.8 [3.1–3.3]	3.4 ± 0.9 [3.2–3.6]	3.5 ± 1.0 [3.3–3.7]
HDL (mmol/L)	1.1 ± 0.3 [1.0–1.2]	1.0 ± 0.3 [0.9–1.1]	1.0 ± 0.3 [0.9–1.1]

Interleukin-6 (IL-6) levels. The concentration of IL-6 increased in proportion to disease severity, rising from 6.8 ± 2.0 pg/mL in Group I to 7.9 ± 2.3 pg/mL in Group III. This elevation was statistically significant in both Group II ($p = 0.035$) and Group III ($p = 0.019$). Considering that IL-6 is one of the central proinflammatory cytokines, these findings confirm the activation of inflammatory cascades in atherothrombosis, which is likely associated with endothelial dysfunction.

Total cholesterol (TC) showed a moderate increase: Group I – 5.4 ± 1.0 mmol/L, Group II – 5.6 ± 1.1 mmol/L ($p = 0.041$), and Group III – 5.7 ± 1.2 mmol/L ($p = 0.033$). This indicates persistent hypercholesterolemia, especially in patients with more advanced vascular lesions. Low-density lipoproteins (LDL) also demonstrated a rising trend from 3.2 ± 0.8 mmol/L to 3.5 ± 1.0 mmol/L, reaching borderline statistical significance ($p = 0.050$ in Group II and $p = 0.040$ in Group III). High-density lipoproteins (HDL) showed a mild decrease—from 1.1 ± 0.3 mmol/L to 1.0 ± 0.3 mmol/L; however, these changes were not statistically significant ($p = 0.132$ and $p = 0.140$), possibly reflecting a preserved anti-atherogenic fraction despite moderate reduction.

In patients with progressive atherothrombotic stenosis of the extracranial arteries, a significant increase in CRP and IL-6 levels was observed, indicating the presence of systemic inflammation and the activation of proinflammatory cytokines. Alongside inflammatory markers, a tendency toward lipid profile deterioration was noted—manifested by an increase in total cholesterol and LDL, accompanied by a decrease in HDL. These findings confirm a close relationship between inflammation, dyslipidemia, and the severity of cerebrovascular pathology, emphasizing the importance of biomarker monitoring as a prognostic and therapeutic effectiveness criterion.

Conclusions

1. Patients with atherothrombotic lesions of the extracranial arteries demonstrated a significant increase in systemic inflammatory markers (CRP, IL-6), the intensity of which correlated with the progression of stenosis and the extent of vascular involvement.

2. Deterioration of the lipid profile was observed, manifested by elevated total cholesterol and LDL levels along with a tendency toward reduced HDL, indicating intensified atherogenic disturbances in more severe forms of the disease.

3. The obtained data confirm a strong pathogenetic interrelation between inflammatory and lipid disorders, which contribute to the destabilization of atherosclerotic plaques and an increased risk of ischemic stroke.

4. The most pronounced inflammatory and lipid changes were observed in patients with combined stenosis of the internal carotid and vertebral arteries, reflecting a more generalized and aggressive course of atherosclerosis.

5. Monitoring of inflammatory markers (CRP, IL-6) and lipid spectrum parameters should be incorporated into the comprehensive examination of patients with atherothrombotic lesions of extracranial arteries for early detection of unstable disease forms, stroke risk prediction, and evaluation of treatment efficacy.

References

1. Feigin V.L., et al. Global and regional burden of stroke during 1990–2019: a systematic analysis for the Global Burden of Disease Study 2019. Lancet Neurol. 2021;20(10):795–820.

2. Sacco R.L., et al. Guidelines for the Prevention of Stroke in Patients With Stroke and Transient Ischemic Attack. Stroke. 2021;52:e364–e467.

3. North American Symptomatic Carotid Endarterectomy Trial Collaborators. Beneficial effect of carotid endarterectomy in symptomatic patients with high-grade carotid stenosis. N Engl J Med. 1991;325:445–453.

4. Hansson G.K. Inflammation, atherosclerosis, and coronary artery disease. N Engl J Med. 2005;352:1685–1695.

5. Libby P., et al. Inflammation in atherosclerosis: from pathophysiology to practice. J Am Coll Cardiol. 2009;54(23):2129–2138.

6. Ridker P.M., et al. C-reactive protein and other markers of inflammation in the prediction of cardiovascular disease in women. N Engl J Med. 2000;342:836–843.

7. Ross R. Atherosclerosis — an inflammatory disease. N Engl J Med. 1999;340:115–126.

8. Goldstein L.B., et al. Guidelines for the primary prevention of stroke. Stroke. 2011;42:517–584.

9. Catapano A.L., et al. 2019 ESC/EAS Guidelines for the management of dyslipidaemias. Eur Heart J. 2020;41(1):111–188.

10. Ridker P.M. High-sensitivity C-reactive protein: potential adjunct for global risk assessment in the primary prevention of cardiovascular disease. Circulation. 2001;103:1813–1818.

11. Gupta A., et al. Carotid plaque MRI and stroke risk: a systematic review and meta-analysis. Stroke. 2013;44:3071–3077.