International Journal of Pharmacy and Pharmaceutical Research (IJPPR)
Volume 32, Issue 7, July 2026 **ijppr.humanjournals.com** ISSN: 2349-7203

Lipoproteins and Stroke: From Pathogenesis to Current and Emerging Therapies

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Received: 27 May 2026 Revised: 15 June 2026 Accepted: 28 June 2026

ABSTRACT

With ischemic stroke making up the majority of instances and hyperlipidemia being a key factor in its pathophysiology, stroke continues to be a significant worldwide health burden. The purpose of this study was to assess existing and new lipid-lowering techniques for stroke prevention as well as to provide an overview of the processes connecting dyslipidemia to cerebral infarction. The information was compiled from well-established research on oxidative stress, inflammation, lipoprotein biology, atherosclerosis, and pharmaceutical treatment. The review demonstrates that whereas inflammatory and oxidative mechanisms hasten plaque instability and thrombosis, high LDL-C encourages endothelial dysfunction, plaque development, and cerebral artery blockage. Beyond LDL-C, new research emphasizes the predictive and mechanistic roles of lipoprotein (a), ApoB/ApoA-I ratio, and HDL functioning in ischemic stroke risk. Statins, fibrates, ezetimibe, PCSK9 inhibitors, and inclisiran are among the modern treatments that successfully reduced cardiovascular risk and atherogenic lipids. Furthermore, patients with high vascular risk and persistent dyslipidemia may benefit from new treatments that target apoC-III and ANGPTL3. Overall, the results highlight the importance of improving broader lipid-related risk assessment and focusing on several pathways implicated in atherosclerosis and thrombosis in addition to reducing LDL-C in order to avoid stroke. Additional mechanistic research, multiethnic cohort studies, and drug repurposing techniques may assist improve results in stroke care and strengthen individualized preventative measures.

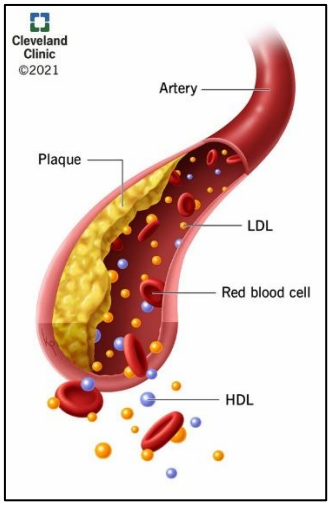
Keywords: Stroke, Hyperlipidemia (Dyslipidemia), Lipoproteins, Statin, Oxidative stress.

1. INTRODUCTION:

According to current Global Burden of Disease studies, stroke is the second greatest cause of death globally and the third highest cause of combined mortality and disability, making it one of the most important global health challenges. Nearly 87% of stroke cases are ischemic strokes, which happen when a blood clot or embolus blocks blood flow in the cerebral arteries. The number of deaths from ischemic stroke has climbed significantly over the previous few decades, from about 2 million in 1990 to 3.2 million in 2019. By 2030, this figure is predicted to approach 4.9 million. [3]

Hyperlipidemia contributes significantly to the development of atherosclerosis and is a significant risk factor for cerebral infarction. Increased oxidized low-density lipoprotein (ox-LDL) levels build up in the vascular endothelium, which attracts inflammatory cells and encourages macrophage infiltration and lipid absorption. These occurrences set off a sequence of pathological alterations that aid in the formation of atherosclerotic plaques. Large intracranial artery stenosis may result from progressive plaque accumulation. A thrombus may form as a result of plaque instability or rupture, and detached thrombotic debris may enter the bloodstream and obstruct brain arteries, resulting in an acute cerebral infarction. Neuro-inflammation and oxidative stress are both important processes in the development of this illness. [2]

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2. PATHOPHYSIOLOGY:

2.1. DYSLIPIDEMIA AND ATHEROSCLEROSIS:

The three layers of the artery wall—the adventitia, media, and intima—provide the anatomical foundation for the development of atherosclerosis. Lipid buildup and inflammatory cell recruitment start in the intima, which is made up of endothelial cells and sub-endothelial matrix. During atherosclerosis, the media, which is composed of smooth muscle cells and elastic fibers, experiences pathological remodeling and helps regulate vascular tone. Fibroblasts, immune cells, and vasa vasorum are found in the adventitia, the outermost layer, and they all contribute to the "outside-in" inflammatory response seen in advanced lesions. Endothelial dysfunction, lipoprotein retention, oxidative stress-induced low-density lipoprotein oxidation, and maladaptive immune activation are all intricately linked in the pathobiology of atherosclerosis. Oxidative stress, smoking, diabetes, hypertension, and hyperlipidemia are all part of the complicated pathophysiology, which allow low-density lipoproteins to enter the sub-endothelial region by damaging vascular endothelial cells. [21]

2.2. PLAQUE FORMATION:

Low-density lipoprotein (LDL) particles enter and remain in the artery wall's sub-endothelium, which initiates the development of atherosclerotic plaque. Oxidative alteration of these retained lipoproteins results in oxidized LDL (ox-LDL), which causes endothelial dysfunction and starts an inflammatory reaction. Circulating monocytes are drawn into the artery intima by the injured endothelium, where they undergo macrophage differentiation.

Through scavenger receptors, macrophages take up ox-LDL and change into lipid-filled foam cells, which are a hallmark of early atherosclerosis lesions. Vascular smooth muscle cell migration and proliferation are stimulated by the buildup of foam cells and the production of pro-inflammatory cytokines and growth factors. Atherosclerotic plaques develop and enlarge as a result of progressive lipid deposition, persistent inflammation, and cellular proliferation. This narrows the artery lumen and reduces blood flow.

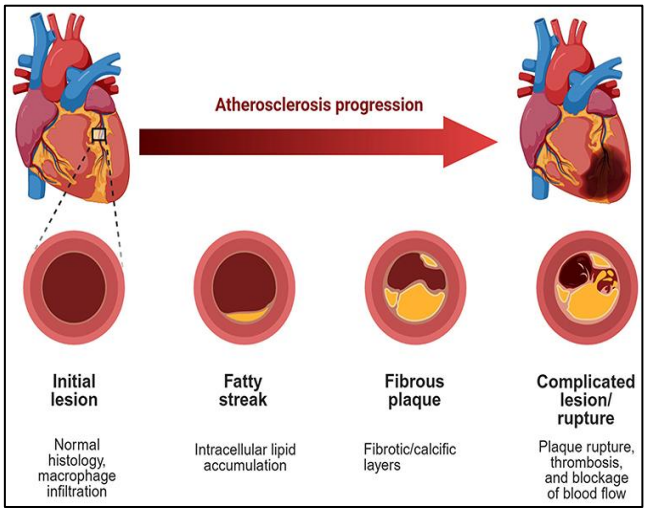
2.3. CEREBRAL ARTERY OCCLUSION:

Blockage of a cerebral blood vessel causes cerebral artery occlusion, which lowers cerebral perfusion. An embolic occlusion occurs when a clot or debris from the heart or proximal arteries travels to and obstructs a cerebral arterial. Thrombotic occlusions occur when a clot forms within the afflicted artery as a result of diseases like atherosclerosis or vascular damage. Emboli can less frequently enter the arterial circulation via a patent foramen ovale or other right-to-left cardiac shunt.

2.4. INFLAMMATION AND OXIDATIVE STRESS:

Cerebral infarction brought on by hyperlipidemia is significantly influenced by inflammation. Overproduction of inflammatory mediators accelerates the development and instability of atherosclerotic plaque by damaging vascular endothelial cells, stimulating smooth muscle cell proliferation, and increasing macrophage infiltration. Plaque rupture raises the risk of cerebral infarction by exacerbating inflammation and causing thrombus development.

An important factor in cerebral infarction linked to hyperlipidemia is oxidative stress. Reactive oxygen species (ROS) overproduction damages vascular endothelial cells, interferes with vascular function, and encourages the growth and migration of vascular smooth muscle cells (VSMCs). These alterations hasten the development of atherosclerotic plaque, which increases platelet activation and aggregation and raises the risk of cerebral infarction and thrombosis.



3. TYPES OF STROKE:

3.1. ISCHEMIC STROKE: [6]

The most common kind of stroke, ischemic stroke, happens when a cerebral or cervical blood artery is blocked, stopping blood supply to the brain. Thrombosis (local clot production), embolism (a clot moving from another area of the body, usually the heart), or severe artery stenosis can all cause this blockage. Sudden weakness or numbness of the face, arm, or leg, trouble speaking or interpreting speech, visual abnormalities, dizziness, loss of balance or coordination, and an abrupt, intense headache are common symptoms.

3.2. HAEMORRHAGIC STROKE: [7]

Intracerebral hemorrhage (ICH) and subarachnoid hemorrhage (SAH) are the two primary kinds of hemorrhagic stroke, which is the second most common type of stroke. In both situations, a blood artery bursts, resulting in bleeding into the subarachnoid space surrounding the brain in SAH or inside the brain tissue in ICH. These are serious incidents that have the potential to seriously harm the nervous system.

4. LIPOPROTEINS AND STROKE RISK:

4.1. LDL-C: [8]

Elevated low-density lipoprotein cholesterol (LDL-C), in particular, is a significant risk factor for cardiovascular illnesses and contributes to the development of ischemic stroke. With a population attributable fraction (PAF) of 25.78%, high LDL-C is the second most significant metabolic risk factor for ischemic stroke, behind higher systolic blood pressure. Dyslipidemia is a modifiable risk factor for ischemic stroke since it can be successfully treated with lipid-lowering medications. Both the first stroke and its recurrence can be avoided with appropriate cholesterol management. In people with carotid artery stenosis, rigorous cholesterol reduction with high-dose atorvastatin reduces the incidence of stroke by 33% and recurrent stroke by 16%, according to research.

4.2. HDL-C: [3]

Because HDL can enhance the elimination of cholesterol, reduce inflammation, prevent oxidative damage, and inhibit thrombosis, it is well known for its preventive properties against stroke. Additionally, by controlling blood clot formation, vascular health, and the progression of atherosclerosis, miRNAs contained by HDL help prevent stroke.

HDL may experience functional alterations that lessen its protective properties and turn it proinflammatory when oxidative stress or inflammation are present. Under such conditions, HDL may promote inflammation, apoptosis, and the formation of atherosclerosis rather than preventing illness.

The results of the many research that have looked into HDL's involvement in stroke are still conflicting. While some research indicates that HDL has a protective impact, other studies show no correlation at all or even an unanticipated rise in the risk of stroke with high HDL-C levels. These contradictory findings could be explained by a variety of factors, including genetic variances, sex and ethnic disparities, and the various functional characteristics of HDL.

4.3. ApoB & ApoA-I:

ApoA-I: [4]

The primary structural protein of high-density lipoprotein (HDL), apolipoprotein A-I (ApoA-I), is essential for preserving the stability, structure, and metabolism of HDL. By stimulating cholesterol efflux via ATP-binding cassette transporter A1 (ABCA1), activating lecithin-cholesterol acyltransferase (LCAT) for HDL maturation, and permitting cholesterol delivery to the liver via scavenger receptor class B type 1 (SR-B1), it promotes reverse cholesterol transport. ApoA-I oxidative modifications or genetic changes can reduce HDL function and raise the risk of cardiovascular disease. ApoA-I not only transports cholesterol but also has antioxidant, anti-inflammatory, anti-apoptotic, vasodilatory, and antithrombotic properties that restrict atherosclerosis and protect endothelial function. By preventing the development of hyperadhesive von Willebrand factor fibers, it also lessens platelet adhesion. The ApoB/ApoA-I ratio is a powerful predictor of ischemic vascular events, and stroke patients have been shown to have lower ApoA-I levels and reduced HDL-associated ApoA-I content.

ApoB: [4]

The primary structural protein of atherogenic lipoproteins, such as chylomicrons, VLDL, IDL, LDL, and lipoprotein (a), is apolipoprotein B (ApoB). It is generated in the liver as ApoB-100 and in the intestine as ApoB-48. Plasma ApoB levels properly represent the total amount of atherogenic particles since each atherogenic lipoprotein contains one ApoB molecule. By building up inside the artery wall and undergoing oxidative alteration, ApoB-containing lipoproteins cause endothelial dysfunction, inflammation, foam cell generation, and plaque development, all of which contribute to atherosclerosis. Additionally, oxidized ApoB promotes thrombotic processes, which increases the risk of ischemic stroke (IS). According to recent data, ApoB concentration and the ApoB/ApoA-I ratio are better indicators of the risk of IS and atherosclerotic cardiovascular disease than conventional lipid measurements like LDL-C. Atherosclerotic risk is further increased by genetic variations that affect ApoB metabolism. ApoB, which reflects the burden of atherogenic lipoproteins and their role in vascular damage and stroke, is hence a significant biomarker and therapeutic target.

4.4. Lipoprotein (a):[1]

Apolipoprotein B-100 is connected to apolipoprotein(a) [Apo(a)] to form lipoprotein(a) [Lp(a)], an LDL-like particle. Smaller Apo(a) isoforms are linked to higher cardiovascular risk, and elevated Lp(a) levels contribute to residual cardiovascular risk independent of LDL-C. Lp(a) has antifibrinolytic properties that favor thrombosis and carries oxidized phospholipids that increase oxidative stress and vascular inflammation, both of which contribute to atherosclerosis.

Elevated levels of Lp(a) have been associated with a higher risk of ischemic stroke, atherosclerotic cardiovascular disease, vascular event recurrence, and carotid plaque susceptibility. According to recent data, Lp(a) is a useful predictive biomarker for identifying those who are more likely to suffer an ischemic stroke.

5. CURRENT PHARMACOLOGICAL MANAGEMENT:

5.1. STATINS:[10]

The first-line treatment for hypercholesterolemia is statins, which work by blocking the enzyme that limits the rate at which cholesterol is synthesized, HMG-CoA reductase. This lowers the creation of cholesterol in the liver, raises the expression of the LDL receptor, and improves the removal of LDL from the bloodstream. Additionally, statins reduce the generation of very-low-density lipoprotein (VLDL), which lowers LDL-C levels by 30–50%. Lowering LDL-C with statins dramatically lowers all-cause mortality and atherosclerotic cardiovascular events, according to clinical research. Statins can elicit temporary muscle-related symptoms and, at higher doses, slightly raise the risk of diabetes in sensitive individuals, despite being typically well tolerated. Atorvastatin, rosuvastatin, simvastatin, pravastatin, lovastatin, fluvastatin, and pitavastatin are among the statins that are often used.

5.2. FIBRATES:[2]

Dyslipidemia is frequently treated with fibrates, including fenofibrate, bezafibrate, gemfibrozil, and fenofibric acid. By activating PPAR-α, which improves fatty acid oxidation, raises lipoprotein lipase activity, and decreases apolipoprotein C-III expression, they mainly lower triglyceride levels. By reducing cholesteryl ester transfer protein activity and encouraging the formation of ApoA-I and ApoA-II, fibrates also raise HDL-C levels.

In addition to decreasing cholesterol, fibrates have anti-inflammatory and antioxidant qualities that prevent the advancement of atherosclerosis. According to experimental research, PPAR-α activation may enhance neurological recovery following a stroke by lowering oxidative stress, neuroinflammation, and blood–brain barrier dysfunction via modifying pathways such as NF-κB, SIRT1, TLR4, AMPK, IL-1, and IL-6.

5.3. EZETIMIBE:[11]

By preventing the intestinal absorption of biliary and dietary cholesterol, ezetimibe reduces cholesterol levels. It can be used alone or in conjunction with statins to generate more potent lipid-lowering effects. It efficiently lowers LDL-C and non-HDL-C levels.

5.4. PCSK9 INHIBITORS:[10]

PCSK9 inhibitors, including evolocumab and alirocumab, are useful lipid-lowering medications used in patients with familial hypercholesterolemia or when statins alone are unable to meet LDL-C objectives. They improve LDL-C clearance and lower LDL-C levels by 60–70% by blocking PCSK9-mediated LDL receptor degradation. These medications are generally well tolerated and reduce the risk of cardiovascular events, such as myocardial infarction and stroke.

5.5. siRNA THERAPEUTIC:

INCLISIRAN:[12,13]

A long-acting small interfering RNA (siRNA) treatment called inclisiran reduces low-density lipoprotein (LDL) cholesterol by inhibiting the liver's production of PCSK9. It reduces PCSK9 synthesis and secretion by binding to PCSK9 mRNA and encouraging its destruction via the RNA-induced silencing complex (RISC). The RISC complex's enduring activity adds to its long-lasting impact. Inclisiran causes a long-lasting decrease in plasma LDL-C concentrations by reducing both intracellular and circulating PCSK9 levels.

6. EMERGING THERAPEUTIC TARGETS:

6.1. ApoC-III INHIBITORS: [15]

Apolipoprotein C-III (apoC-III) is known to inhibit lipoprotein lipase (LPL) and is an essential regulator of triglyceride metabolism. Moreover, apoC-III has recently been identified as a major risk factor for cardiovascular disease. Through transcriptional regulatory elements in the gene promoter region, glucose stimulates the production of ApoC3, whereas insulin inhibits it. Fibrates' hypotriglyceridaemic action can be partially explained by the activation of peroxisome proliferator-activated receptor-α, which lowers apoC3 expression.

6.1.1. Volanesorsen:

A 20-nucleotide second-generation 2'-O-methoxyethyl chimeric antisense inhibitor of apoC3 is called volanesorsen. mRNA was developed for the incredibly rare condition FCS, as well as possibly for other forms of hypertriglyceridemia and familial partial lipodystrophy.

6.1.2. Olezarsen:

Olezarsen, an ASO coupled with N-acetyl-galactosamine (GalNAc), specifically targets hepatic APOC3 mRNA and prevents apoC3 protein synthesis. Gal-NAc receptors are abundant in hepatocytes, which increases binding capacity and affinity. It may make it possible to lessen the risk of toxicity and lower the dosage of medications.

6.1.3. ARO-APOC3:

ARO-APOC3, an investigational RNAi treatment that targets APOC3, is being developed to treat patients with severe hypertriglyceridemia (SHTG), mixed dyslipidemia (MD), and FCS.

6.2. ANGPTL3 INHIBITORS: [15]

The 70 kDa glycoprotein ANGPTL3 is mostly expressed and secreted by hepatocytes. The conventional angiopoietin structure is present in the 460 amino acid polypeptide that comprises the protein structure of ANGPTL3. This structure includes a signal peptide with 16 amino acid residues, an N-terminal coiled-coil domain, a linker region, and a fibrinogen-like C-terminal domain. It has been shown to decrease LPL activity and increase TG-rich very low-density lipoprotein (VLDL). Furthermore, by inhibiting the activity of endothelial lipase (EL), which is encoded by LIPG, it can increase HDL-C levels. ANGPTL3 may also have an impact on the liver's synthesis of TG in VLDL and the hepatic removal of LDL particles via endothelial lipase-dependent VLDL and residual particle clearance or the LDLR.

6.2.1. Evinacumab:

Evinacumab is a fully human monoclonal antibody with a human IgG4 constant region. It has a high affinity for ANGPTL3 in humans, mice, rats, and monkeys. The primary mechanism of action is likely the potentiation of plasma lipase activity, which forms immune complexes by binding to ANGPTL3 in the circulation.

7. FUTURE PERSPECTIVES:

Beyond its circulating levels, HDL's anti-inflammatory, antioxidant, and antithrombotic properties play a major role in preventing stroke. To completely comprehend these protective advantages, however, more confirmation and mechanistic studies are required. HDL's concentration, particle size, and structural integrity can all be altered by diabetes, oxidative stress, inflammation, and interactions with extracellular vesicles. Large, multiethnic cohort studies evaluating HDL composition and function may help identify novel biomarkers associated with stroke risk.[3]

Drug Repurposing: One useful method for discovering novel pharmacological or therapeutic applications for existing drugs is drug repurposing. Finding novel pharmacological activities is significantly more advantageous than creating a completely new medication. First, because the medication being repurposed has already been successfully utilized in preclinical animals and/or humans, there is little chance of failure. Second, medication development time will be shortened and the parent medicine will be a well-known one with established safety standards. Finding a novel therapeutic use for an already-approved drug begins with docking studies.[17]

8. CONCLUSION:

The prevalence of ischemic stroke worldwide, which is mostly caused by atherosclerosis brought on by hyperlipidemia, continues to pose a serious threat to public health. Cerebral artery blockage is the result of a complex pathophysiology that includes dyslipidemia, oxidative stress, inflammation, and plaque development. This highlights the crucial role that lipid management plays in preventing stroke. Although LDL cholesterol is a significant modifiable risk factor, new knowledge about the complex functions of HDL, ApoB, ApoA-I, and lipoprotein (a) expands our knowledge of stroke risk and creates opportunities for more accurate biomarkers and focused treatments.

Lipid control and stroke risk reduction have greatly improved thanks to current pharmacological approaches, which are headed by statins and supplemented by fibrates, ezetimibe, PCSK9 inhibitors, and cutting-edge siRNA therapies like inclisiran. In the meanwhile, patients with complicated dyslipidemias have optimism thanks to new treatments that target apoC-III and ANGPTL3, which demonstrate the advancement in tackling residual risk factors. These advancements highlight how crucial it is to combine clinical care with molecular insights in order to maximize results.

In the future, multiethnic cohort studies, medication repurposing initiatives, and a deeper understanding of HDL's functional characteristics beyond its plasma levels should improve therapeutic alternatives and predictive models. As the intricate relationship between lipoproteins, inflammation, and vascular health is better understood, more individualized and efficient interventions may be developed in the future, which could revolutionize stroke prevention and treatment and lessen the devastating disease's worldwide effects.

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How to cite this article:
Vijayalakshmi M et al. Ijppr.Human, 2026; Vol. 32 (7):332-339.

Conflict of Interest Statement: All authors have nothing else to disclose.

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