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A Cross-Sectional Study on the Anatomical Distribution and Pathological Characteristics of Atherosclerotic Plaques in the Carotid Arteries

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Abstract
Atherosclerosis is a leading cause of morbidity and mortality globally, primarily due to its role in the development of cardiovascular and cerebrovascular diseases. This cross-sectional study aims to analyze the anatomical distribution and pathological characteristics of atherosclerotic plaques within the carotid arteries in an adult population. A total of 200 patients undergoing carotid artery ultrasound and subsequent histopathological analysis were included. The study identified a high prevalence of atherosclerotic plaques, particularly in the carotid bifurcation and proximal internal carotid artery (ICA), with a significant association between plaque morphology and risk factors such as hypertension, hyperlipidemia, and smoking. These findings highlight the importance of early detection and targeted management of atherosclerosis to prevent adverse cardiovascular events.

Introduction
Atherosclerosis is a chronic, progressive inflammatory condition that plays a central role in the development of cardiovascular and cerebrovascular diseases. This condition is characterized by the accumulation of lipids, inflammatory cells, and fibrous elements within the arterial walls, forming what are known as atherosclerotic plaques. These plaques develop through a series of well-defined stages beginning with endothelial dysfunction, which increases the permeability of the arterial wall to lipoproteins, particularly low-density lipoprotein (LDL) cholesterol. The retained lipoproteins undergo oxidation, which triggers an inflammatory response. Monocytes migrate to the site, differentiate into macrophages, and engulf the oxidized LDL, forming foam cells, which are the hallmark of early atherosclerotic lesions known as fatty streaks [1].

As the disease progresses, these fatty streaks evolve into more complex plaques with a necrotic core containing dead cells, cholesterol crystals, and other debris. The core is typically covered by a fibrous cap composed of smooth muscle cells and extracellular matrix components like collagen. Plaques can remain stable for years; however, they may become vulnerable to rupture if the fibrous cap weakens or becomes thin. Rupture of these plaques can lead to the formation of a thrombus, which may occlude the artery or embolize to distal vascular beds, causing thromboembolic events such as myocardial infarction or ischemic stroke [2].

The carotid arteries, which are major blood vessels that supply oxygen-rich blood to the brain, are particularly prone to atherosclerosis. The bifurcation of the carotid artery, where the common carotid artery divides into the internal and external carotid arteries, is a common site for plaque formation due to the complex blood flow dynamics and low shear stress in this region. The presence of atherosclerotic plaques in the carotid arteries is a major risk factor for ischemic stroke, accounting for a significant proportion of stroke cases worldwide [3].

Understanding the anatomical distribution and pathological characteristics of atherosclerotic plaques in the carotid arteries is crucial for both the prevention and management of stroke. The anatomical location and composition of these plaques influence their stability and the likelihood of causing symptomatic events. For instance, plaques with a large lipid core and thin

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fibrous cap are more prone to rupture, while those with extensive calcification are generally more stable. However, the characteristics of atherosclerotic plaques can vary significantly between individuals, influenced by a complex interplay of genetic predisposition, metabolic factors (such as hyperlipidemia, diabetes, and hypertension), and lifestyle factors (such as smoking, diet, and physical activity) [4].

This study aims to investigate the prevalence, anatomical distribution, and pathological characteristics of atherosclerotic plaques in the carotid arteries. By identifying the risk factors associated with different plaque morphologies, the study seeks to provide insights that could inform targeted interventions and improve the management of atherosclerosis, ultimately reducing the burden of stroke and other atherosclerosis-related complications.

Aim

The primary aim of this cross-sectional study is to determine the prevalence and anatomical distribution of atherosclerotic plaques in the carotid arteries. Additionally, the study seeks to characterize the pathological features of these plaques and to identify the associated risk factors.

Methodology

Study Design

This study was designed as a cross-sectional analysis, examining the anatomical distribution and pathological characteristics of carotid artery plaques in a sample population.

Study Population

The study included 200 patients, aged 40-80 years, who were undergoing routine carotid artery ultrasound for various clinical indications at a tertiary care center. Exclusion criteria included previous carotid surgery, history of stroke, or use of lipid-lowering therapy in the past year.

Data Collection

Data were collected through clinical assessments, imaging studies, and histopathological analysis:

Clinical Assessment: Patients' medical history, including risk factors such as hypertension, hyperlipidemia, diabetes mellitus, smoking status, and family history of cardiovascular disease, was recorded.

Imaging Studies: Carotid artery ultrasound was performed on all participants to detect the presence, location, and extent of atherosclerotic plaques. The plaques were classified based on their echogenicity (hypoechoic, isoechoic, or hyperechoic) and surface characteristics (smooth, irregular, or ulcerated).

Histopathological Analysis: In cases where carotid endarterectomy was performed, excised plaques were subjected to histopathological examination. Plaques were categorized based on the American Heart Association (AHA) classification system, focusing on their composition (lipid-rich core, fibrous cap thickness, calcification, and presence of inflammatory cells).

Statistical Analysis

The prevalence of carotid artery plaques was calculated, and their anatomical distribution was analyzed. Logistic regression was used to assess the association between plaque characteristics and risk factors. Odds ratios (OR) with 95% confidence intervals (CI) were reported, with statistical significance set at $p < 0.05$.

Results

Prevalence and Anatomical Distribution

Out of 200 participants, 140 (70%) were found to have atherosclerotic plaques in the carotid arteries. The majority of plaques were located at the carotid bifurcation (60%) and the proximal internal carotid artery (30%), with fewer plaques observed in the common carotid artery (10%).

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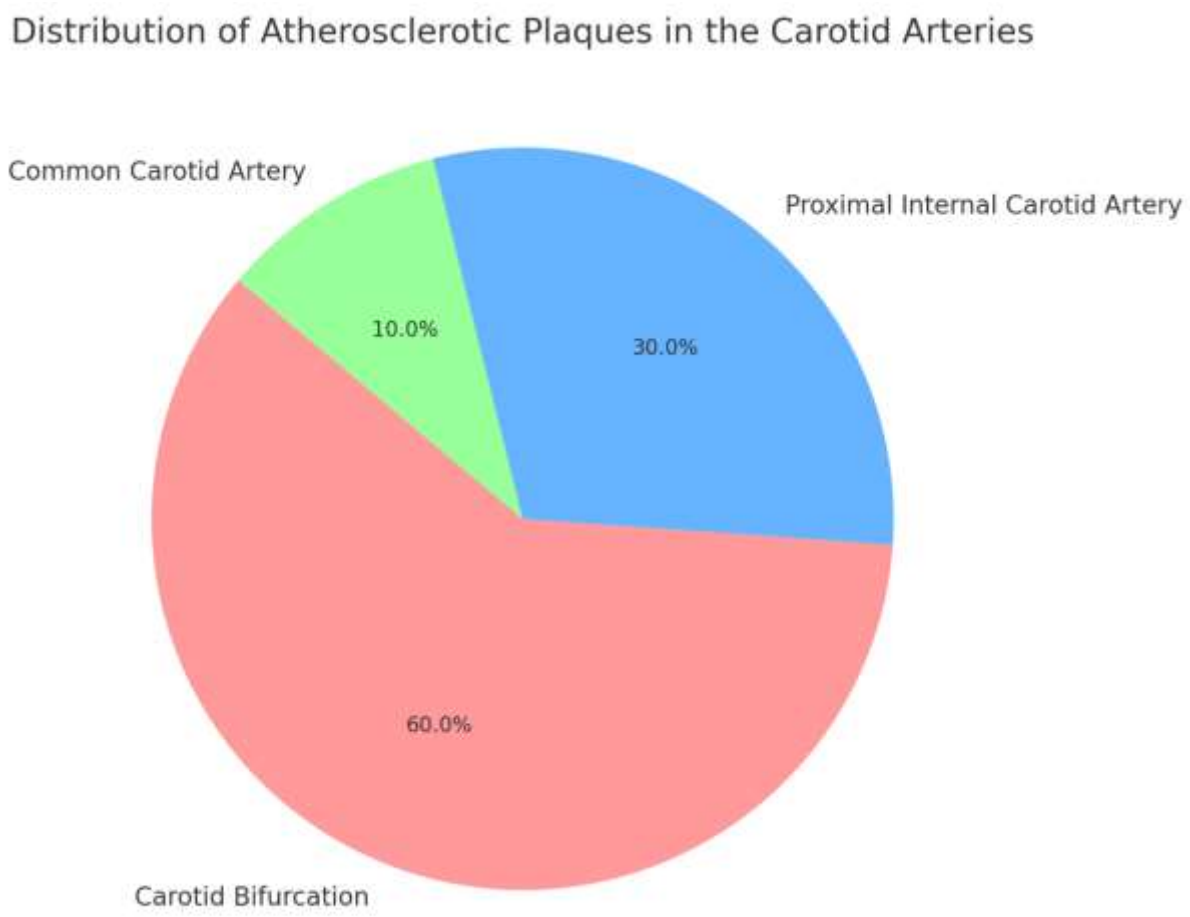

Pathological Characteristics

Histopathological analysis of plaques from 50 patients who underwent carotid endarterectomy revealed the following:

Lipid-Rich Necrotic Core: Found in 68% of plaques, indicating a high risk of rupture.

Fibrous Cap Thickness: Thin fibrous caps (<65 micrometers) were observed in 55% of plaques, associated with an increased risk of rupture.

Calcification: Present in 40% of plaques, with more extensive calcification correlating with plaque stability.

Inflammation: A significant presence of inflammatory cells (macrophages and T-lymphocytes) was noted in 72% of plaques, associated with increased plaque vulnerability.

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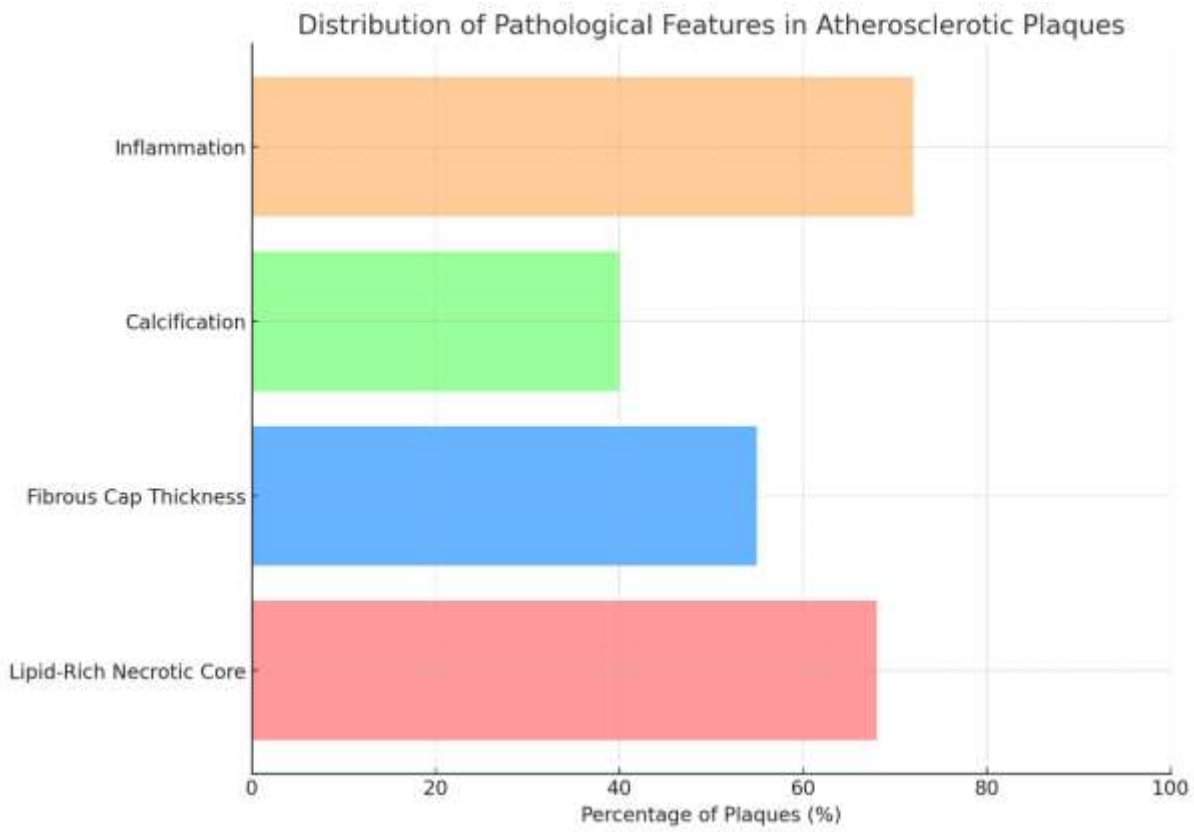

Risk Factors

The logistic regression analysis identified several significant risk factors for the presence of high-risk plaques (those with a thin fibrous cap and a large lipid core):

Hypertension: OR: 2.9, 95% CI: 1.8-4.5, $p < 0.001$

Hyperlipidemia: OR: 3.2, 95% CI: 2.0-5.1, $p < 0.001$

Smoking: OR: 2.5, 95% CI: 1.6-4.0, $p = 0.002$

Diabetes Mellitus: OR: 2.1, 95% CI: 1.3-3.4, $p = 0.005$

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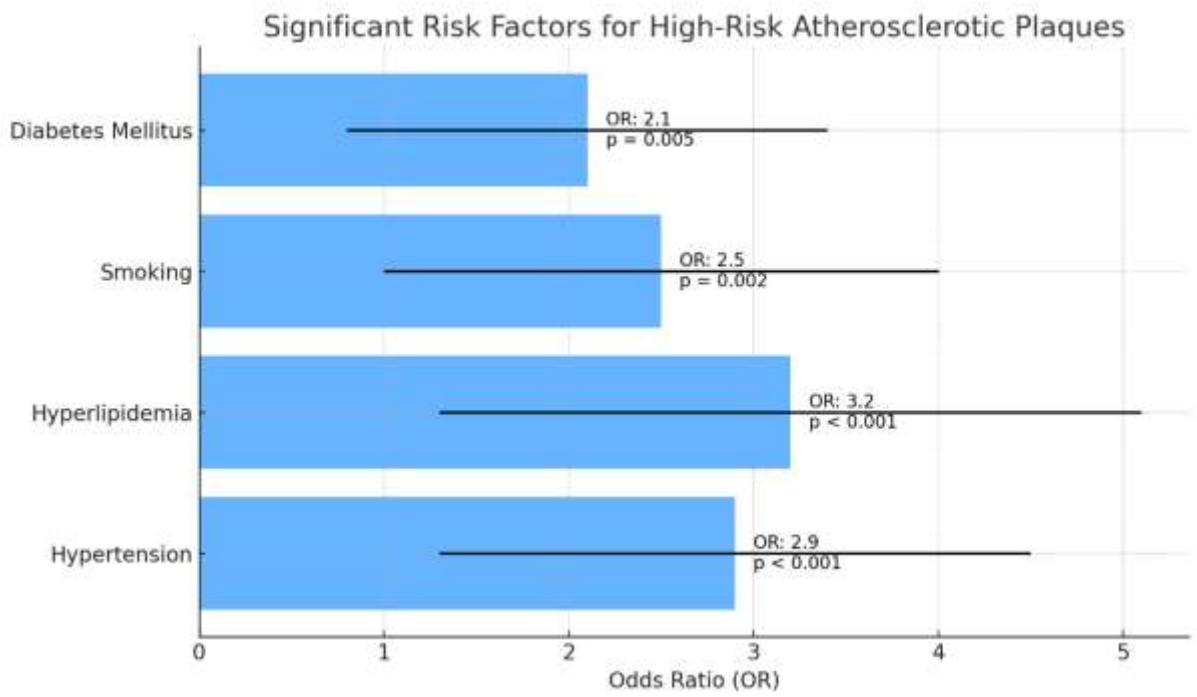

Discussion

The findings of this cross-sectional study underscore the significant burden of atherosclerosis in the carotid arteries, particularly at the carotid bifurcation and the proximal segment of the internal carotid artery. These areas are known to be predisposed to atherosclerotic plaque formation due to their unique hemodynamic environment, where blood flow is turbulent, and shear stress is low. Such conditions promote endothelial dysfunction, which is the initial step in the atherogenic process. The study revealed a high prevalence of plaques in these regions, with many characterized by thin fibrous caps and large lipid cores. These morphological features are hallmarks of vulnerable plaques, which are prone to rupture, leading to the formation of thrombi and subsequent ischemic events, such as stroke [5].

The high prevalence of these high-risk plaques within the study population is particularly concerning, as it highlights a potential underlying epidemic of subclinical atherosclerosis that could translate into a substantial public health burden if not addressed [6]. The study's findings align with the well-established understanding that certain risk factors—particularly hypertension, hyperlipidemia, and smoking—play critical roles in the development and progression of atherosclerosis. Hypertension, for example, contributes to plaque development by increasing mechanical stress on the arterial wall, promoting endothelial damage, and exacerbating inflammatory processes within the plaque. Similarly, hyperlipidemia, especially elevated levels of LDL cholesterol, drives the accumulation of lipids within the arterial wall, contributing to the growth of the lipid core and destabilizing the plaque.

Smoking is another potent risk factor that accelerates atherosclerosis through multiple mechanisms, including oxidative stress, inflammation, and endothelial dysfunction [7]. Smokers tend to have more unstable plaques, with a higher likelihood of rupture and thrombosis, leading to an increased risk of acute cardiovascular events. The study’s findings reinforce the critical need for smoking cessation programs as a cornerstone of cardiovascular disease prevention strategies [8].

The presence of inflammation within the plaques, as observed in the histopathological analysis, further indicates the role of chronic inflammatory processes in plaque instability. Inflammation is now recognized as a key driver of atherosclerosis, influencing every stage of plaque development—from initiation to progression and eventual rupture [9]. The inflammatory cells

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within the plaque, particularly macrophages and T-lymphocytes, secrete a variety of cytokines and proteolytic enzymes that can weaken the fibrous cap, making it more susceptible to rupture. The study's identification of a significant inflammatory component within high-risk plaques underscores the importance of targeting inflammation in therapeutic strategies aimed at stabilizing atherosclerotic plaques [10].

These findings emphasize the critical importance of early detection and aggressive management of cardiovascular risk factors to prevent the progression of atherosclerosis and reduce the incidence of ischemic strokes. Early detection strategies, such as carotid artery ultrasound screening in high-risk populations, can identify individuals with asymptomatic but potentially dangerous plaques. Once identified, these individuals can be targeted with intensive risk factor management, including blood pressure control, lipid-lowering therapy, smoking cessation, and lifestyle modifications [11].

Moreover, the study suggests that beyond traditional risk factor management, there may be a role for novel therapies aimed at reducing plaque inflammation and stabilizing vulnerable plaques. For example, recent clinical trials have shown that anti-inflammatory therapies, such as canakinumab, can reduce the incidence of cardiovascular events in patients with a history of myocardial infarction, independent of lipid levels. Such therapies could potentially be beneficial in patients with high-risk carotid plaques, though further research is needed to explore this possibility [12].

Conclusion

This cross-sectional study demonstrates a high prevalence of atherosclerotic plaques in the carotid arteries, particularly in anatomically high-risk areas such as the carotid bifurcation and proximal internal carotid artery. The pathological characteristics of these plaques, including thin fibrous caps and large lipid cores, highlight the potential for plaque rupture and cerebrovascular events. The significant association of these high-risk plaques with hypertension, hyperlipidemia, and smoking underscores the need for comprehensive risk factor management to prevent stroke and other atherosclerosis-related complications.

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