

## Ultrasound Evaluation of Extra Cranial Carotid Arteries and Its Correlation with Different Risk Factors of Atherosclerosis

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### Abstract

**Background:** Atherosclerosis, characterized by lipid accumulation and plaque formation within arterial walls, is a major cause of cardiovascular diseases (CVD) including coronary artery disease (CAD), cerebrovascular disease, and peripheral artery disease (PAD). The extra cranial carotid arteries are critical sites for atherosclerotic plaque development, potentially leading to ischemic strokes and other cerebrovascular events.

**Objective:** This review explores the role of ultrasound in evaluating extra cranial carotid arteries and examines the correlation between ultrasound findings and atherosclerosis risk factors, emphasizing its utility in risk stratification, guiding therapeutic decisions, and monitoring disease progression.

**Methods:** A comprehensive literature search was conducted using PubMed, MEDLINE, and Google Scholar. Studies relevant to ultrasound evaluation of extra cranial carotid arteries and the correlation with atherosclerosis risk factors were selected. Key search terms included "carotid artery ultrasound," "atherosclerosis," "risk factors," "intima-media thickness," and "carotid plaque."

**Conclusion:** Ultrasound, particularly Doppler ultrasound, is a non-invasive, safe, and reliable method for assessing carotid atherosclerosis. It provides real-time visualization of arterial structures and hemodynamics, enabling detailed evaluation of intima-media thickness, plaque characteristics, and luminal stenosis. Early detection and monitoring of atherosclerotic changes in the carotid arteries facilitate timely interventions, reducing the risk of cerebrovascular events. Correlating ultrasound findings with atherosclerosis risk factors is crucial for improving patient outcomes.

**Keywords:** Carotid Ultrasound, Atherosclerosis, Intima-Media Thickness, Carotid Plaque, Cardiovascular Risk Factors.

### 1. Introduction

Atherosclerosis is a chronic, progressive disease characterized by the accumulation of lipids, inflammatory cells, and fibrous elements within the arterial walls. This process leads to the formation of plaques, which can cause narrowing and hardening of the arteries, ultimately compromising blood flow. Atherosclerosis is a leading cause of cardiovascular diseases (CVD), including coronary artery disease (CAD), cerebrovascular disease, and peripheral artery disease (PAD). These conditions are major contributors to morbidity and mortality worldwide, posing significant public health challenges. The clinical manifestations of atherosclerosis range from asymptomatic disease to life-threatening events such as myocardial infarction and stroke<sup>[1]</sup>.

The extra cranial carotid arteries, which include the common carotid artery (CCA), internal carotid artery (ICA), and external carotid artery (ECA), are critical sites for the development of atherosclerotic plaques. These arteries supply blood to the brain, and atherosclerotic changes within them can lead to cerebrovascular events, notably ischemic strokes. Evaluating these arteries for atherosclerosis is crucial for identifying individuals at high risk for stroke and other cardiovascular events. Early detection and monitoring of

atherosclerotic changes in the carotid arteries can facilitate timely interventions and prevent adverse outcomes<sup>[2]</sup>.

Ultrasound imaging, particularly Doppler ultrasound, is a non-invasive, safe, and reliable method for assessing carotid atherosclerosis. It provides real-time visualization of arterial structures and blood flow dynamics, allowing for the detailed evaluation of the carotid arteries. Key ultrasound measurements in the assessment of carotid atherosclerosis include intima-media thickness (IMT), which is a marker of subclinical atherosclerosis, and the characterization of atherosclerotic plaques, including their size, composition, and surface features. Additionally, Doppler ultrasound can quantify the degree of luminal stenosis by measuring blood flow velocities, helping to assess the severity of arterial narrowing<sup>[3]</sup>.

Ultrasound's ability to detect and quantify atherosclerotic changes in the carotid arteries makes it an essential tool in both clinical practice and research. It aids in risk stratification, guiding therapeutic decisions, and monitoring disease progression or response to treatment<sup>[4]</sup>.

The aim of this narrative review is to explore the role of ultrasound in the evaluation of extra cranial carotid arteries and to examine the

correlation between ultrasound findings and various risk factors of atherosclerosis.

## 2. Methodology

A comprehensive literature search was conducted using databases such as PubMed, MEDLINE, and Google Scholar. Studies were selected based on their relevance to the ultrasound evaluation of extra cranial carotid arteries and the correlation with atherosclerosis risk factors. Key terms used in the search included "carotid artery ultrasound," "atherosclerosis," "risk factors," "intima-media thickness," and "carotid plaque."

### ❖ Anatomy

#### ✓ The Aorta

The aorta is the largest artery in the human body, originating from the left ventricle of the heart to deliver oxygenated blood throughout the body via systemic circulation. Anatomically, the aorta is divided into the thoracic aorta, extending from the heart down to the diaphragm, and the abdominal aorta, which continues from the diaphragm to the aortic bifurcation<sup>[5]</sup>.

#### ✓ Divisions of the Aorta

Based on the course of the aorta and the direction of blood flow, it is further divided into the ascending aorta, aortic arch, and descending aorta. The descending aorta is subdivided into the thoracic descending aorta and abdominal aorta. The aorta ends by splitting into the two common iliac arteries and the median sacral artery<sup>[5]</sup>.

#### ✓ Ascending Aorta and Aortic Sinuses

The ascending aorta starts at the aortic valve of the left ventricle and ends at the pericardial reflection, transitioning into the aortic arch. It runs alongside the pulmonary trunk within the pericardial sheath and supplies the heart through two coronary arteries. The aortic sinuses, or sinuses of Valsalva, are three small pockets located at the root of the ascending aorta, giving rise to the left and right coronary arteries, while the posterior aortic sinus does not give rise to any coronary arteries<sup>[6,7]</sup>.

#### ✓ Aortic Arch and its Branches

The aortic arch extends from the pericardial reflection, running posteriorly and crossing over the pulmonary trunk bifurcation, left pulmonary artery, and left main bronchus. It gives rise to the brachiocephalic trunk, left common carotid artery (CCA), and left subclavian artery. Variations in the branching pattern include shared origins and uncommon arteries such as the thyroid ima artery<sup>[7]</sup>.

#### ✓ Descending Aorta

The descending aorta begins where the aortic arch terminates, at the level of the fourth and fifth thoracic vertebrae intervertebral disc. The thoracic descending aorta provides several branches, including intercostal, subcostal, and bronchial arteries. The abdominal aorta starts at the

diaphragmatic hiatus, branching into the lumbar, renal, and various visceral arteries before bifurcating into the common iliac arteries at the fourth lumbar vertebra. The left and right CCAs supply the head and neck, dividing into the external carotid artery (ECA) and internal carotid artery (ICA) at the upper border of the thyroid cartilage<sup>[8]</sup>.

### ❖ Pathology of Atherosclerosis

Atherosclerosis is the primary cause of coronary, cerebral, and peripheral artery diseases, all of which have high morbidity and mortality rates. It is a vascular intimal disease affecting the entire vascular system, characterized by the formation of intimal plaques. These plaques can obstruct blood flow, leading to serious cardiovascular events. The Greek term "atherosclerosis" refers to the thickening of the arterial intima layer and the accumulation of fatty material within the plaques<sup>[9]</sup>.

The formation of atherosclerotic plaques begins with the deposition of small cholesterol crystals in the arterial intima and underlying smooth muscle. These plaques consist of a central core of fatty material covered by a fibrous cap. The accumulation of fat, macrophages, and fibrous tissue contributes to the growth of the plaque, which protrudes into the arterial lumen, reducing blood flow. As the disease progresses, connective tissue and calcium deposition cause arterial hardening (sclerosis). The uneven arterial surface can lead to clot formation and thrombosis, resulting in sudden blood flow obstruction<sup>[10]</sup>.

Cholesterol plays a significant role in the pathogenesis of atherosclerosis. While normal cholesterol levels can reduce the risk of clogged arteries, excessive low-density lipoprotein (LDL) cholesterol contributes to plaque formation. LDL is considered "bad" cholesterol because it promotes fatty buildups in arteries. High-density lipoprotein (HDL) cholesterol, on the other hand, helps remove LDL from the bloodstream, transporting it to the liver for breakdown. Plaques can be stable or unstable; unstable plaques are prone to rupture, potentially causing heart attacks or strokes. Carotid artery plaques are categorized based on their composition and can be homogenous or heterogeneous<sup>[11]</sup>.

## 3. Ultrasound

### ❖ Physical Principles of Ultrasound

Ultrasound imaging relies on the transmission and reflection of sound waves to create images of internal body structures. Sound waves, including ultrasound, require a medium to travel through and can be measured by their wavelength, frequency, and velocity. The interaction of ultrasound waves with tissues results in reflection, scattering, transmission, or attenuation, which are essential for image formation. **Table 1**

**Table (1)** Summary of Physical Principles and Wave Interactions in Ultrasound Imaging

Concept	Definition
<b>Sound</b>	A mechanical wave that moves in a straight line and requires a medium to travel through (Bushberg and Boone, 2011).
<b>Ultrasound</b>	A wave with a frequency greater than 20,000 hertz (Hz), exceeding the upper limit of human hearing (Bushberg and Boone, 2011).
<b>Waves</b>	<b>Longitudinal waves</b> measured by: - <b>Wavelength (<math>\lambda</math>):</b> The distance over which the shape of the wave repeats. - <b>Frequency (f):</b> The number of waves that travel through a spot in one unit of time. - <b>Velocity (v):</b> The speed of sound in a medium, dependent on wavelength ( $\lambda$ ) and frequency (f). $v=f\lambda$ . It is related to the medium's compressibility, being slow in gases, faster in fluids, and fastest in solids. (Bushberg and Boone, 2011).
<b>Wave Interaction with Tissue</b>	
<b>Reflection</b>	Occurs at the interface of two adjacent tissues with different acoustic impedances (z). The difference in impedance determines the amount of reflection. Reflected waves are used to create ultrasound images (Bushberg and Boone, 2011).
<b>Scattering</b>	Caused by interaction with a very rough surface or a small reflector, redirecting the sound wave in multiple directions, with only a small portion returning to the scan head (Bushberg and Boone, 2011).
<b>Transmission</b>	Some of the sound wave continues deeper into the body and is reflected from deeper structures. The amplitude of the sound wave decreases with depth (Bushberg and Boone, 2011).
<b>Ultrasound Image Formation</b>	The scan head of the ultrasound machine emits pulses of sound waves that interact with body tissues. Reflected waves return to the scan head and are processed into an image by the ultrasound

machine (Bushberg and Boone, 2011).

❖ **Ultrasound Transducer**

An ultrasound transducer operates on the piezoelectric effect, which allows it to convert electrical energy into mechanical sound waves and vice versa. Piezoelectric crystals within the transducer change shape when an electrical voltage is applied, causing the crystals to expand and contract, thus generating ultrasound pulses. The thickness of these crystals determines the frequency of the scan head: thinner crystals produce higher frequencies, while thicker crystals produce lower frequencies. The frequency of the ultrasound waves affects the image quality, with higher frequencies providing better resolution but less penetration, and lower frequencies offering deeper penetration but lower resolution<sup>[12]</sup>.

Medical ultrasound utilizes high-frequency sound waves, ranging from 2 to 15 MHz, to assist in the diagnosis and treatment of patients. The transducer sends ultrasound waves into the body, where they propagate through tissues and are reflected back as echoes. These echoes are converted back into electrical impulses by the transducer crystals and processed to form images on the screen. Reflection occurs at interfaces between tissues of different densities, with the amount of reflection proportional to the difference in impedance. Homogeneous fluids, such as blood or bile, appear as echo-free structures, while structures like bones or calcified plaques cause acoustic shadowing due to complete reflection of the sound waves<sup>[12]</sup>.

❖ **Doppler Ultrasound and Hemodynamics**

The Doppler ultrasound principle is used to measure the velocity of blood flow within vessels. When ultrasound pulses emitted by the transducer encounter moving blood, the frequency of the reflected waves changes in proportion to the blood's velocity, a phenomenon known as the Doppler shift. This shift is influenced by the initial frequency of the ultrasound waves, the velocity of the blood, and the angle of the transducer relative to the direction of blood flow. Blood flow patterns can vary from laminar to disturbed and turbulent, especially in the presence of stenosis or changes in vessel caliber. Spectral Doppler detects these velocity changes, with higher velocities indicating greater degrees of stenosis<sup>[13]</sup>.

❖ **Normal Carotid Arteries by Ultrasound**

The carotid artery wall on ultrasound generates parallel echoes corresponding to the adventitial and intimal layers, with a hypoechoic region representing the media between these lines. The intimal reflection should be straight, thin, and parallel to the adventitial layer, visible in longitudinal planes to ensure the image plane crosses the vessel diameter correctly. Intima-media thickness (IMT) is measured in the longitudinal

plane (B mode) at the far wall of the common carotid artery (CCA), 1 cm before bifurcation, with an upper normal average IMT up to 0.8mm. The variation in color degree from the periphery to the center of the artery indicates normal laminar flow, with potential flow disruptions at the carotid bulb due to vascular tortuosity or branching. The color duplex image displays aspects of carotid pulsatility, showing continuous color flow in the CCA and internal carotid artery (ICA) throughout systole and diastole, while the external carotid artery (ECA) shows decreased diastolic color flow [14].

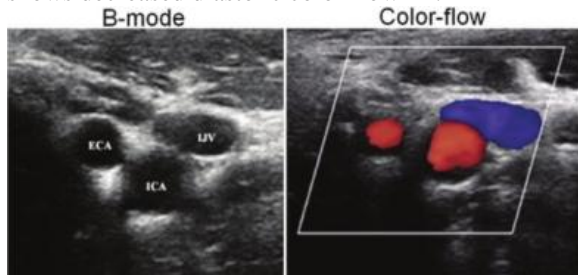


Fig.(1) ICA, ECA by color duplex [15].

❖ Carotid spectral Doppler characterization:

Pulsed Doppler is used to evaluate features and velocities of waveform of CCA, ICA and ECA. CCA has moderately broad systolic peaks and a moderate amount of flow throughout diastole. Normal ICA demonstrated low resistance flow, whereas a normal ECA showed high resistance flow [16].



Fig. (2) Typical wave form of CCA [16].

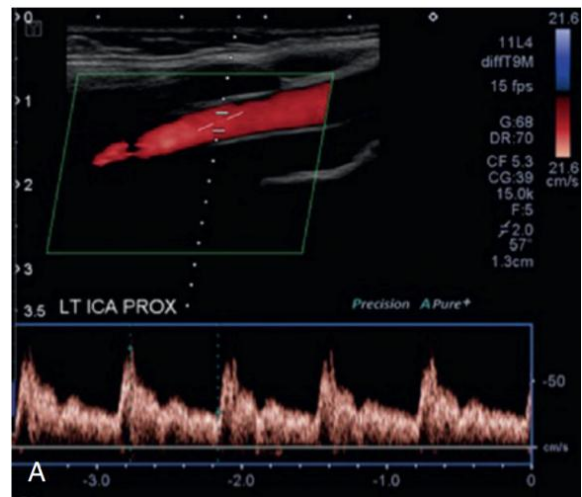


Fig. (3) Typical waveform of ICA [16].

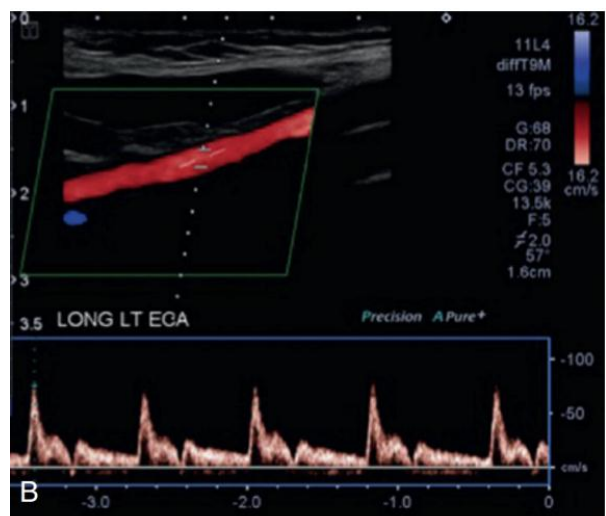


Fig. (4) Typical waveform of ECA [16].

❖ Differentiation between Carotid Artery and Jugular Vein

The common carotid artery (CCA) lies adjacent to the internal jugular vein (IJV), but the two can be easily distinguished. The CCA exhibits cephalic flow with a pulsatile pattern, while the IJV has caudal flow characterized by a continuous or undulating low-velocity waveform. The caliber of the CCA remains constant, whereas the IJV caliber varies significantly in response to respiration. Additionally, the CCA has a thick wall with a distinct intimal reflection, whereas the IJV wall is thin and lacks an intimal reflection [17].

❖ Differentiation between Internal and External Carotid Arteries

The internal carotid artery (ICA) and external carotid artery (ECA) can be differentiated based on several characteristics. The ECA is usually smaller than the ICA and has branches in the neck, whereas the ICA has no branches in the neck. The ECA is typically oriented anteromedially, while the ICA is oriented posterolaterally. On spectral waveform analysis, the ECA shows a high-resistance waveform,

whereas the ICA displays a low-resistance waveform<sup>[18]</sup>.

❖ **Atherosclerotic Changes of Extracranial Carotid Arteries**

The earliest atherosclerotic change is the diffuse thickening of the intima-media interface, with the upper normal average of intima-media thickness (IMT) estimated to be up to 0.8mm. Focal intimal thickening can develop into a plaque, defined as a focal IMT greater than 1.2mm. Plaque evaluation should be conducted in both transverse and longitudinal planes to avoid underestimation. Assessment is primarily done by B-mode ultrasound, with color and power Doppler imaging aiding in evaluating radiolucent plaques and detecting surface ulcers. Plaque characterization includes its site, echogenicity, surface, and the diameter stenosis it causes. Significant stenotic lesions often occur in the proximal internal carotid artery (ICA), while lesions at the common carotid artery (CCA) bifurcation can cause major hemispheric events. Echogenicity varies: anechoic plaques are similar to blood, hyper echoic plaques to adventitia, isoechoic plaques to the sternocleidomastoid muscle, and calcified plaques show intense posterior acoustic shadowing, preventing assessment of underlying material<sup>[16, 19]</sup>.

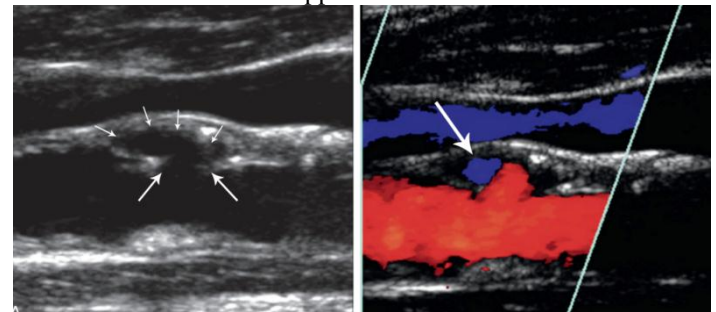
**Table (2)** Characteristics of Atherosclerotic Plaques in Carotid Arteries and Associated Imaging Features

Plaque Type	Description
<b>Anechoic Plaque (Radiolucent Plaque)</b>	Echo-lucent areas due to lipid core or hemorrhage. Homogeneous echo-lucent plaques are rare. Color Doppler and power Doppler imaging are useful for visualization. Difficult to distinguish hemorrhage from lipid core. Associated with increased LDL, plaque ulceration, and higher risk of cerebral ischemic symptoms (Castelblanco et al., 2019; Gupta et al., 2015).
<b>Isoechoic Plaque</b>	Increased echogenicity due to fibrous tissue content, related to collagen and smooth muscle cells. Not typically associated with cerebral ischemic symptoms (Nicolaidis et al., 2005).
<b>Hyperchoic Plaque</b>	Higher echogenicity than the sternocleidomastoid muscle, similar to the adventitia. Does not produce acoustic shadowing, indicating lack of calcification (Garg et al., 2016).
<b>Calcified Plaque</b>	Dystrophic calcification produces strong reflections and distal acoustic shadows. Calcification can be focal or diffuse. Large

calcifications generate shadows that obscure the arterial lumen, interfering with ultrasound diagnosis. Associated with late-stage plaque formation and neurological events (Lin et al., 2017).

❖ **Surface and Diameter Stenosis of Atherosclerotic Plaques**

Ischemic strokes are primarily caused by emboli from the heart, great vessels, or carotid system occluding distal intracranial vessels. High-grade stenosis (over 70% diameter stenosis) rarely causes intracranial hypoperfusion, but plaques, regardless of stenosis level, can be emboli sources if they have surface endothelial defects (ulcers). Ultrasound evaluation of plaque surfaces is crucial for detecting plaques prone to shedding emboli, though small ulcer craters on large, complex plaques can be difficult to detect. An ulcer is defined as a surface defect measuring 2mm by 2mm, and while ultrasound has low sensitivity for detecting small ulcers, it is specific for well-defined endothelial defects. To report an ulcer, it must not extend to the lumen intima interface, have sharp margins (differentiating from pseudo-ulcers), and show flow on color Doppler<sup>[20, 21]</sup>.



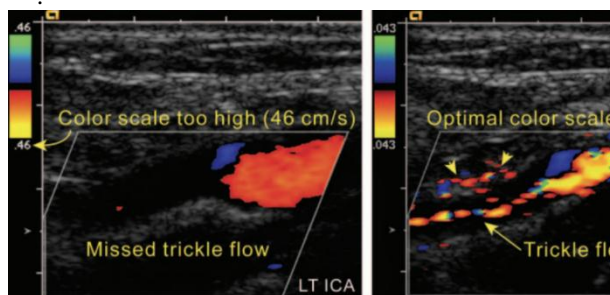
**Fig. (12)** Plaque ulcer by B-mode and color duplex<sup>[16]</sup>.

Diameter stenosis of a plaque is calculated by comparing the residual patent lumen with the total vessel lumen, as per the European Carotid Surgery Trials (ECST) method. However, for plaques at the proximal internal carotid artery (ICA), the North American Symptomatic Carotid Endarterectomy Trials (NASCET) method is preferred, comparing the smallest residual lumen diameter with the normal ICA lumen distal to the stenosis. Spectral Doppler plays a critical role in evaluating plaque stenosis severity, using parameters such as peak systolic velocity (PSV) of ICA, the ICA/CCA PSV ratio, and end diastolic velocity (EDV) of ICA. Elevated PSV is commonly used but can be misleading due to cardiac output changes, vessel tortuosity, or severe stenosis affecting contralateral ICA flow. In such cases, the ICA/CCA PSV ratio provides a more accurate assessment<sup>[22]</sup>.

❖ **Total vs Near Total Occlusion of Carotid Arteries**

Differentiating between total and near total occlusion of carotid arteries is crucial as total occlusion is not operable, whereas near total occlusion, a severe form of plaque stenosis, requires surgical intervention. The incidence of stroke in untreated near total occlusion is approximately 11% per year. Total occlusion typically occurs at or near the internal carotid artery (ICA) origin, showing anechoic or hypoechoic areas occupying the entire lumen with no detectable flow on color, power, or spectral Doppler. In some cases, the occluded vessel may not be identifiable, and diagnosis is based on visualizing a single artery from the bifurcation. A patent stump proximal to the occlusion may show bidirectional flow, representing forward systolic blood flow and its reflection caused by the occluding thrombus/plaque. The common carotid artery (CCA) may exhibit a high-resistance waveform due to blood flow diversion to the external carotid artery (ECA), which reflects the end-organ waveform unless ECA collateral to the ICA is present<sup>[16, 23]</sup>

Near total occlusion is identified by a very narrow residual channel (trickle flow) within the vessel, which shows low blood flow signals. For diagnosis, color sensitivity should be maximized, and power Doppler should be used. Distal to the near total occlusion site, there will be decreased blood flow and a tardus parvus waveform (delayed systolic upstroke and low PSV). In some severe cases, the PSV may appear in the range of mild stenosis or even normal, so the diagnosis should rely on the visual appearance in color or power Doppler rather than spectral Doppler parameters<sup>[24]</sup>



**Fig. (5)** Near total occlusion with presence of trickle flow after adjusting color scale<sup>[25]</sup>.

#### 4. Conclusion

Ultrasound, particularly Doppler ultrasound, is a non-invasive, safe, and reliable method for assessing carotid atherosclerosis. It provides real-time visualization of arterial structures and hemodynamics, enabling detailed evaluation of intima-media thickness, plaque characteristics, and luminal stenosis. Early detection and monitoring of atherosclerotic changes in the carotid arteries facilitate timely interventions, reducing the risk of cerebrovascular events. Correlating ultrasound findings with

atherosclerosis risk factors is crucial for improving patient outcomes.

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