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Evaluation of Carotid Artery in Patients with Ischemic Stroke Using Carotid Doppler Ultrasonography in Dhamar Yemen

Mohammed Ali Al-Huthi*, Dhaifullah Jayed, Hadi Mujlli, Abdulsalam Almkdad

Department of cardiology, Faculty of Medicine, Thamar University, Yemen

***Corresponding author:**
Mohammed Ali Al-Huthi

dohuthi@yahoo.com

Tel: +967770006 006.

Department of cardiology, Faculty of Medicine, Thamar University, Yemen

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Abstract

Background: Stroke defined as an abrupt onset of neurologic deficit due to vascular cause. It is one of the leading causes of mortality and morbidity all over the world making early diagnosis and treatment inevitable. Lesions of extra cranial carotid arteries are implicated in majority of cases of acute ischemic stroke. Early detection of the athermanous changes in the carotid artery will reduce the stroke related morbidity and mortality. Carotid Doppler is a non-invasive imaging technique, with sensitivity approaching that of angiography. Aim: This study aimed to evaluate the role of carotid Doppler nosography in patients presenting with acute ischemic stroke and find association between carotid artery stenosis and the risk factors of cerebrovascular diseases in, Dhamar city, Yemen. Methods: A descriptive cross-sectional study was conducted over six months (October. 2021– April. 2022) among patients with confirmed ischemic stroke and who underwent carotid Doppler ultrasonography in Doppler units at AL-Ryadah International Hospital and Taiba Consultative Hospital, Dhamar city, Dhamar Governorate, Yemen. A pre-designated interview sheets was used for data collection by interviewing patients and their family members, and by extracting information from their medical files and carotid Doppler reports. The sheet was divided into four parts: the first part consisted of socio-demographic data, the second consisted of stroke side and it's relation to the side of carotid stenosis. The third part consisted of risk factors and the final part consisted of carotid doppler findings. Data analysis was done by using the Statistical Package for the Social Sciences (SPSS) program version 25. Results: Eighty-four patient were included in our study, among them, there were 54 patients (64.3%) were males and 30 (35.7%) were females. Their ages ranged from 22 to 100 years with mean age 64.9±1.70. Carotid stenosis was founded in 53.6% of participated patients, among those more than one-third (35.5%) had severe stenosis. The proximal part of internal carotid artery was most common location of plaque formation. Hypertension, smoking and diabetes were the most prevalent risk factors for cerebral ischemic stroke. Out of total 84 patients, 57 (67.9%) had hypertension, 28 (33.3%) had diabetes and 16 (19%) had previous stroke history. Conclusion: Carotid artery Doppler demonstrated atherosclerotic plaques and carotid stenosis in more than half of studied patients. Increasing age >50 years, male sex, smoking, hypertension, diabetes, and hyperlipidemia were associated with an increased rate of atherosclerosis, carotid stenosis in patients with ischemic stroke. Patients with risk factors of stroke should be scanned with Doppler sonography as early as possible.

Keywords: Stroke; Carotid artery; Doppler Sonography; Dhamar; Yemen

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Background

Stroke is the most common life-threatening neurological disorder and the most important single cause of disability. According to World Health Organization estimates for the year 2030, stroke will stay as the second leading cause of death along with Ischemic heart disease, both in developing as well as developed countries [1].

There are three basic categories of stroke. Ischemic stroke, hemorrhagic stroke and subarachnoid hemorrhage. Ischemic stroke is defined as thrombotic or ischemic occlusion of any vessel

resulting in loss of function with signs and symptoms depending on the territory supplied by the obstructed vessel [2-4]. The most common type of stroke is ischemic stroke with accounting for 75-85% among all types followed by hemorrhagic stroke 10-20% and subarachnoid hemorrhage is the least common with 5% of all cases [5, 6].

The most common cause of carotid artery stenosis is atherosclerosis. When carotid artery stenosis reaches greater than 50%, it is associated with significant risk of stroke [7]. Carotid artery atherosclerosis is one of the most important predictor of ischemic stroke [8]. There are many clinical modalities which can be used to diagnose carotid artery stenosis to prevent future occurrence of fatal events by early commencement of treatment and adopting preventive strategies. Among various clinical modalities, Doppler ultrasound stands out as an accurate, non-invasive, safe and cost-effective modality of evaluation of carotid arteries [8, 9]. It carries 96% sensitivity and 86% specificity. It has positive predictive value of 89% and negative predictive value of 94%. Its accuracy increases with increasing stenosis. It is 91% accurate with any degree of stenosis greater than 50% [10].

Ultrasound grading of carotid artery stenosis is operator dependent and may vary among laboratories [11, 12]. Color Doppler ultrasound is also helpful in some cases and may be followed by arteriography [13]. Doppler is considered more diagnostic than conventional gray scale duplex scanning as Doppler identified pseudo-occluded internal carotid artery stenosis in 94% patients whereas gray scale scanning identified stenosis in 27% patients in a study conducted by Berman et al [13].

Fatal and debiliting events associated with carotid artery stenosis demands for its screening in symptomatic as well as asymptomatic individuals. Mean annual stroke rate was 6% in symptomatic patients and 2% in symptomatic patients [14]. Doppler ultrasound is used for screening whereas angiography is used in high risk patients for diagnosis [15]. Regular screening for risk factors can reduce the incidence of new events due to early commencement of preventive strategies [16]. Ultrasonography is cost effective than angiography in initial screening of carotid artery atherosclerosis [15]. Current evidence doesn't support routine use of angiography in asymptomatic patients however Doppler ultrasonography can be easily performed with good results [15].

Study Justification

In Yemen, little is known about data the frequency of carotid artery disease among cerebrovascular ischemic stroke (CVS). There is no regional literature about this subject, and our study is the first study in Yemen at all.

Objectives

1. To study the colour Doppler findings of extra cranial carotid arteries among patients presenting with ischemic stroke in a tertiary care center, Dhamar city, Dhamar Governorate, Yemen.

2. To determine the prevalence and risk factors of extra cranial atherosclerosis among stroke patients using extra cranial duplex ultrasound and find the association between carotid artery stenosis and these risk factors.

Literature Review

Stroke

General considerations: Stroke is the second leading cause of death worldwide, with 6.2 million dying from stroke in 2015, an increase of 830,000 since the year 2000. The incidence rises steeply with age, with the highest incidence of stroke occurring in people older than 80 years. Men are at slightly higher risk of stroke compared to women except after age 80, and in younger ages Peripartum stroke is an important consideration [17]. In many lower- and middle-income countries it is rising in association with less healthy lifestyles [18]. While stroke has grown in incidence worldwide, it is declining among the affluent and rising among those with less access to medical care [19].

Definitions: The World Health Organization defines stroke as "rapidly developing clinical signs of focal (at times global) disturbance of cerebral function, lasting for more than 24 hours or leading to death, with no apparent cause other than of vascular origin [20].

i. Stroke is defined as a syndrome of rapid onset neurological deficit caused by focal cerebral, spinal or retinal infarction or haemorrhage [20].

ii. Transient ischaemic attack (TIA) means a brief episode of neurological dysfunction due to temporary focal cerebral or retinal ischaemic without infarction, e.g. a weak limb, loss of vision, usually lasting seconds or minutes with complete recovery. TIAs may herald a stroke. The arbitrary time of less than 24 hours is no longer used [20].

Pathophysiology: The underlying pathology responsible for stroke is either infarction or haemorrhage. Of the 180–300 patients per 100 000 population presenting annually with a stroke, 85% sustain a cerebral infarction due to inadequate blood flow to part of the brain, and most of the remainder have an intracerebral haemorrhage [18].

I. **Cerebral infarction**

Cerebral infarction is mostly caused by thromboembolic disease secondary to atherosclerosis in the major extra cranial arteries (carotid artery and aortic arch). About 20% of infarctions are due to embolism from the heart, and a further 20% are due to thrombosis in situ caused by intrinsic disease of small perforating vessels (lenticulostriate arteries), producing so-called lacunar infarctions. The risk factors for ischaemic stroke reflect the risk factors for the underlying vascular disease. About 5% are due to rare causes, including Vasculitis, endocarditis and cerebral venous disease.

Cerebral infarction takes some hours to complete, even though the patient's deficit may be maximal shortly after the vascular occlusion. After the occlusion of a cerebral artery, infarction may be forestalled by the opening of anastomotic channels from other arterial territories that restore perfusion to its territory. Similarly, reduction in perfusion pressure leads to compensatory homeostatic changes to maintain tissue oxygenation. These

compensatory changes can sometimes prevent occlusion of even a carotid artery from having any clinically apparent effect [18].

However, if and when these homeostatic mechanisms fail, the process of ischemia starts, and ultimately leads to infarction unless the vascular supply is restored. As the cerebral blood flow declines, different neuronal functions fail at various thresholds. Once blood flow falls below the threshold for the maintenance of electrical activity, neurological deficit develops. At this level of blood flow, neurons are still viable; if blood flow increases again, function returns and the patient will have had a transient ischaemic attack (TIA). However, if blood flow falls further, a level is reached at which irreversible cell death starts. Hypoxia leads to an inadequate supply of adenosine triphosphate (ATP), which leads to failure of membrane pumps, thereby allowing influx of sodium and water into cells (cytotoxic oedema) and release of the excitatory neurotransmitter glutamate into the extracellular fluid. Glutamate opens membrane channels, allowing influx of calcium and more sodium into the neurons. Calcium activates intracellular enzymes that complete the destructive process.

The release of inflammatory mediators by microglia and astrocytes causes death of all cell types in the area of maximum ischemia. The infarction process is worsened by anaerobic production of lactic acid and consequent fall in tissue pH. The final outcome of occlusion of a cerebral blood vessel thus depends on the competence of circulatory homeostatic mechanisms, the metabolic demand, and the severity and duration of the reduction in blood flow. Higher brain temperature, e.g. in fever, and higher blood glucose have both been associated with a greater volume of infarction for a given reduction in cerebral blood flow. Subsequent restoration of blood flow may cause haemorrhage into the infarcted area ('hemorrhagic transformation'). This is particularly likely in patients given thrombolytic drugs, and in patients with larger infarcts. Radio logically, a cerebral infarct can be seen as a lesion that comprises a mixture of dead brain tissue that is already undergoing autolysis, and tissue that is ischaemic and swollen but recoverable (the 'ischaemic penumbra'). The infarct swells with time and is at its maximal size a couple of days after stroke onset. At this stage, it may be big enough to exert mass effect clinically and radiologically; sometimes, decompressed craniotomy is required. After a few weeks, the oedema subsides and the infarcted area is replaced by a sharply defined fluid-filled cavity [18].

II. **Intracerebral haemorrhage**

Intracerebral haemorrhage causes about 10% of acute stroke events but is more common in low-income countries. It usually results from rupture of a blood vessel within the brain parenchyma but may also occur in a patient with SAH if the artery ruptures into the brain substance as well as the subarachnoid space. Haemorrhage frequently occurs into an area of brain infarction and, if the volume of haemorrhage is large, it may be difficult to distinguish from primary intracerebral haemorrhage both clinically and radiologically. Explosive entry of blood into the brain parenchyma causes immediate cessation of function in that area as neurons are disrupted and white-matter fibre tracts are split apart. The haemorrhage itself may expand over the first minutes or hours, or it may be associated with a rim of cerebral oedema, which, along with the haematoma, acts like a mass lesion to cause progression of the neurological deficit. If big enough, this can cause shift of the intracranial contents, producing transtentorial coning and sometimes rapid death. If the patient survives, the haematoma is gradually absorbed, leaving a haemosiderin-lined slit in the brain parenchyma [18].

Risk factors for stroke

i. Fixed risk factors [19]

a. Age

b. Gender (male >female except at extremes of age)

c. Race (African>Asian >European)

d. Previous vascular event:

- Myocardial infarction
- Stroke
- Peripheral vascular disease

e. Heredity

f. Sickle cell disease

g. High fibrinogen

ii. **Modifiable risk factors**

a. Hypertension

b. Cigarette smoking

c. Hyperlipidaemia

d. Diabetes mellitus

e. Oestrogen-containing drug

f. Polycythaemia

g. Heart disease:

- Atrial fibrillation
- Congestive cardiac failure
- Infective endocarditis

h. Excessive alcohol intake

Stroke etiology

i. Common causes [19]

a. Thrombosis

- Lacunar stroke (small vessel)
- Large-vessel thrombosis
- Dehydration

b. Embolic occlusion

- Artery-to-artery o Carotid bifurcation

o Aortic arch o Arterial dissection

- Cardioembolic

o Atrial fibrillation o Mural thrombus o Myocardial

infarction o Dilated cardiomyopathy o Valvular lesions o Mechanical valve o Bacterial endocarditis

- Paradoxical embolus o Atrial septal defect o Patent foramen ovale
- Stimulant drugs: cocaine, amphetamine

ii. Uncommon causes [19]

a. Hypercoagulable disorders

- Protein C & Protein S deficiency
- Antithrombin III deficiency
- Antiphospholipid syndrome
- Factor V Leiden mutation
- Prothrombin G20210 mutation
- Systemic malignancy
- Sickle cell anemia & B Thalassemia
- Polycythemia vera
- Systemic lupus erythematosus
- Homocysteinemia
- Thrombotic thrombocytopenic purpura
- Disseminated intravascular coagulation
- Nephrotic syndrome
- Inflammatory bowel disease
- Oral contraceptives

b. Venous sinus thrombosis

c. Vasculitis

- Systemic vasculitis granulomatosis with polyangiitis, Takayasu's,

Giant cell arteritis)

- Primary CNS vasculitis
- Meningitis (syphilis, tuberculosis, fungal, bacterial, zoster)

d. Noninflammatory vasculopathy

- Reversible vasoconstriction syndrome
- Fabry's disease
- Angiocentric lymphoma

e. Cardiogenic

- Mitral valve calcification
- Atrial myxoma
- Marantic endocarditis
- Libman-Sacks endocarditis

f. Subarachnoid hemorrhage

g. Eclampsia

Clinical manifestations

Onset is usually abrupt, and there may then be very little progression except that due to brain swelling. Clinical evaluation should always include examination of the heart for murmurs and rhythm irregularities. Auscultating over the carotid or subclavian vessels may reveal a bruit but is not sensitive enough to substitute for vascular imaging [20].

i. **Obstruction of carotid circulation**

a. Occlusion of the anterior cerebral artery distal to its junction with the anterior communicating artery: Weakness and cortical sensory loss in the contralateral leg and sometimes mild weakness of the arm, especially proximally. There may be a contralateral grasp reflex, paratonic rigidity, abulia (lack of initiative), or frank confusion. Urinary incontinence is not uncommon, particularly if behavioral disturbances are conspicuous. Bilateral anterior cerebral infarction is especially likely to cause marked behavioral changes and memory disturbances [17].

b. Middle cerebral artery occlusion: Contralateral hemiplegia, hemi sensory loss, and homonymous hemianopia (ie, bilaterally symmetric loss of vision in half of the visual fields), with the eyes deviated to the side of the lesion. If the dominant hemisphere is involved, global aphasia is also present. It may be impossible to distinguish this clinically from occlusion of the internal carotid artery. With occlusion of either of these arteries, there may also be considerable swelling of the hemisphere during the first 72 hours. For example, an infarct involving one cerebral hemisphere may lead to such swelling that the function of the other hemisphere or the rostral brainstem is disturbed and coma results. Occlusions of different branches of the middle cerebral artery cause more limited findings. For example, involvement of the superior division in the dominant hemisphere leads to a predominantly expressive (Broca) aphasia and to contralateral paralysis and loss of sensations in the arm, the face and, to a lesser extent, the leg. Inferior branch occlusion in the dominant hemisphere produces a receptive (Wernicke) aphasia and a homonymous visual field defect. With involvement of the nondominant hemisphere, speech and comprehension are preserved, but there may be a left hemispatial neglect syndrome or constructional and visuospatial deficits [17].

c. Occlusion of the ophthalmic or central retinal artery: Sudden painless visual loss with retinal pallor and a macular cherry red spot on fundoscopic examination. Sudden, transient vision loss in one eye (amaurosis fugax) is a TIA in this arterial territory. Patients with a cilioretinal artery (approximately 25%) may have macular sparing due to collateral blood supply [17].

ii. Obstruction of vertebrobasilar circulation

a. Occlusion of the posterior cerebral artery: May lead to a thalamic syndrome in which contralateral hemi sensory disturbance occurs, followed by the development of spontaneous pain and hyperpathia. There is often a macular-sparing homonymous hemianopia and sometimes a mild, usually temporary, hemiparesis. Depending on the site of the lesion and the collateral circulation, the severity of these deficits varies and

other deficits may also occur, including involuntary movements and alexia. Occlusion of the main artery beyond the origin of its penetrating branches may lead solely to a macular-sparing hemianopia [17].

b. Vertebral artery occlusion below the origin of the anterior spinal and posterior inferior cerebellar arteries: May be clinically silent because the circulation is maintained by the other vertebral artery. If the remaining vertebral artery is congenitally small or severely atherosclerotic, however, a deficit similar to that of basilar artery occlusion is seen unless there is good collateral circulation from the anterior circulation through the circle of Willis.

c. Lateral medullary syndrome: An obstruction of the posterior inferior cerebellar artery or an obstruction of the vertebral artery just before it branches to this vessel. This syndrome characterized by vertigo and nystagmus (vestibular nucleus), ipsilateral spinothalamic sensory loss involving the face (trigeminal nucleus and tract), dysphagia (nucleus ambiguus), limb ataxia (inferior cerebellar peduncle), and Horner syndrome (descending sympathetic fibers), combined with contralateral spin thalamic sensory loss involving the limbs [17].

d. Occlusion of both vertebral arteries and the basilar artery: Leads to coma with pinpoint pupils, flaccid quadriplegia and sensory loss, and variable cranial nerve abnormalities. With partial basilar artery occlusion, there may be diplopia, visual loss, vertigo, dysarthria, ataxia, weakness or sensory disturbances in some or all of the limbs, and discrete cranial nerve palsies. In patients with hemiplegia of pentane origin, the eyes are often deviated to the paralyzed side, whereas in patients with a hemispheric lesion, the eyes commonly deviate from the hemiplegic side. When the small paramedian arteries arising from the basilar artery are occluded, contralateral hemiplegia and sensory deficit occur in association with ipsilateral cranial nerve palsy at the level of the lesion [17].

e. Occlusion of any of the major cerebellar arteries: Leads to vertigo, nausea, vomiting, nystagmus, and ipsilateral limb ataxia. Contralateral spin thalamic sensory loss in the limbs may also be present. Occlusion of the anterior inferior cerebellar artery may cause deafness, ipsilateral facial spin thalamic sensory loss and weakness. Massive cerebellar infarction may lead to obstructive hydrocephalus, coma, tonsillar herniation, and death [17].

Investigations i. Neuroimaging

a. Brain Computed Tomography (CT): Will demonstrate haemorrhage immediately

but cerebral infarction is often not detected in the acute phase or only subtle changes are seen Repeat CT at 24–48hours may be helpful [20].

b. Brain Magnetic Resonance Imaging (MRI): Is more sensitive than CT for early changes of infarction (diffusion-weighted sequences, DWI) and for small infarcts. MRI also clearly demonstrates the extent and anatomy of an infarct and shows evidence of clinically silent simultaneous infarcts that indicate an embolic cause. MRI can also help identify the underlying cause, e.g. arterial dissection using specific sequences to show the false lumen (crescent sign) or venous cortical infarcts. Many stroke mimics, such as demyelination, are shown with MRI but not CT. For these reasons, MRI is increasingly used in routine assessment of stroke and is essential in younger patients or in those where the cause is uncertain [20].

c. Vascular imaging by carotid Doppler within 24 hours: Is essential to identify high grade symptomatic carotid stenosis requiring surgery. CT or MR angiography is now widely used to corroborate the results of Doppler, to identify arterial stenosis in the posterior circulation or intracranial vessels that are not visible on Doppler, and also to identify arterial dissection and venous sinus thrombosis. Catheter angiography is rarely needed following ischaemic stroke [20].

ii. Cardiac investigations

Identification of a cardio-embolic source of stroke, principally atrial fibrillation, is achieved with electrocardiography (ECG) or 24-hour ECG. Other causes, such as valve disease, patent foramen ovale or mural thrombus, require transthoracic

Echocardiography or trans-esophageal echo in selected patients. Recent research has shown that prolonged cardiac monitoring (e.g. with an implantable loop recorder) demonstrates paroxysmal atrial fibrillation in a significant minority of people with stroke of unknown cause [18].

iii. Other investigations

Routine blood tests blood count, erythrocyte sedimentation rate, glucose, clotting studies, lipids profiles. In young patients with stroke or in those individuals where there is no evidence of atherosclerosis or an embolic source, more specialist investigations may be required to look for an underlying vasculitic, inflammatory, infective, metabolic or genetic cause [18].

Management

Stroke is a medical emergency. Paramedics and members of the public are encouraged to make the diagnosis of stroke on a simple history and examination – FAST:

- Face–sudden weakness of the face
- Arm–sudden weakness of one or both arms
- Speech–difficulty speaking, slurred speech
- Time–the sooner treatment can be started, the better.

During initial assessment, immediate, continued and meticulous attention to the airway, blood pressure and swallowing is essential [18].

i. Acute management

a. Thrombolysis

Thrombolysis significantly increases the chances of having no or minimal disability after stroke, by reducing infarct size. Earlier treatment within the 4.5-hour time window significantly improves outcome, so every minute counts. Approximately

10% of patients are potential candidates for thrombolysis, most being excluded due

to late presentation outside the time window for treatment [19].

Two recent studies have shown benefit, with a low complication rate, from endovascular therapy (usually performed with retrievable stents) following alteplase therapy. In both studies, patients with proximal vessel occlusion and salvageable brain tissue were selected, with improved function and reduced mortality after treatment [19]. In a recent meta-analysis of five trials, endovascular thrombectomy has been shown to benefit most patients with an acute stroke caused by occlusion of the proximal anterior circulation. Currently thrombectomy is performed routinely for selected cases in a number of centres [19].

b. Antiplatelet therapy and anticoagulation

High-dose aspirin (300mg) is started 24hours after thrombolysis, or as soon as haemorrhage is excluded if thrombolysis is contraindicated, and continued for 2 weeks before switching to clopidogrel. The number needed to treat (NNT) to prevent one stroke is 100.

Anticoagulants are started for atrial fibrillation-associated cardio-embolic stroke usually after 2 weeks to reduce the risk of acute hemorrhagic transformation of infarcts (NNT=12). For arterial dissection, the risk of recurrent embolic stroke from the site of dissection is considered to be high enough to justify immediate anticoagulation or antiplatelet therapy, although controlled trial evidence is lacking. Venous sinus or cortical vein thrombosis causing stroke is also treated with anticoagulation. In addition to warfarin for oral anticoagulation, direct oral anticoagulants (DOACs) that inhibit factor Xa or thrombin, they have the advantage over warfarin of a wider therapeutic index with a lower rate of haemorrhage, no need for monitoring and few drug interactions [20].

DE compressive craniotomy

This should be performed within 48hours in MCA strokes causing infarction of more than 50% of the MCA territory to prevent coning and improve long-term outcome

[20].

Stroke units

Direct admission to a stroke unit has been demonstrated to be one of the most effective interventions in acute stroke, saving lives and reducing long-term disability. Specialized multidisciplinary teams and clear protocols for aspects of care, such as swallowing assessment, thromboembolism prevention, treatment of infections, management of hyperglycemia and other medical complications, improve quality and consistency of care, and thus outcomes. Early mobilization and access to physiotherapy, occupational therapy and speech therapy, as well as initiation of secondary prevention and patient education, are equally necessary. Early supported discharge and assessment of rehabilitation needs are also better coordinated on a stroke unit than a general ward [20].

ii. Secondary prevention interventions

a. Antihypertensive therapy

Recognition and good control of high blood pressure are the major factors in primary and secondary stroke prevention. Transient hypertension, often seen following stroke, usually does not require treatment, provided diastolic pressure does not rise above 100mmHg. Sustained severe hypertension needs treatment after 72 hours; blood pressure should be lowered slowly to avoid any sudden fall in perfusion [20].

b. Lipid-lowering therapy

Statins, typically atorvastatin 40 mg, should be offered to all patients unless there is a contraindication, aiming for a target total cholesterol below 4mmol/L (low-density lipoprotein <2mmol/L) [20].

c. Lifestyle modification and education

Education of patient and family is an essential aspect of secondary prevention. Smoking cessation and advice about diet, exercise, weight reduction and alcohol consumption should be started on the stroke unit and continued after discharge [20].

d. Surgery and stenting for carotid stenosis

High-grade symptomatic carotid stenosis is associated with a significant risk of recurrent stroke during the weeks after TIA or stroke. Carotid endarterectomy should be performed within 2 weeks in patients with 70–99% stenosis on the affected side, provided the initial stroke was not severely disabling. A second imaging modality, such as CT angiography, should be performed to confirm the results of Doppler studies. For patients with moderate symptomatic stenosis (50–69%), there is a modest benefit with intervention over the 3% stroke risk associated with the procedure itself. Carotid stenting is an alternative to surgery in some patients (major stroke risk is the same for surgery and stenting, but the chance of minor non-disabling stroke is higher for stenting) [20]. The case for intervention in asymptomatic stenosis is debatable. Patients with 70–99% stenosis may have a modest stroke risk reduction at 5 years, but moderate stenosis should be treated conservatively. Carotid occlusion is always treated conservatively (there is no risk of distal embolization) [20].

Carotid Doppler Ultrasonography

Introduction

Carotid Doppler ultrasonography is a popular tool for evaluating atherosclerosis of the carotid artery. Its two-dimensional gray scale can be used for measuring the intima-media thickness, which is very good biomarker for atherosclerosis and can aid in plaque characterization. The plaque morphology is related to the risk of stroke. The ulceration of plaque is also known as one of the strong predictors of future embolic event risk. Color Doppler ultrasonography and pulse Doppler ultrasonography have been used for detecting carotid artery stenosis. Doppler ultrasonography has unique physical properties. The operator should be familiar with the physics and other parameters of Doppler ultrasonography to perform optimal Doppler ultrasonography studies [21].

2.2.2Carotid artery anatomy and differentiation the ICA from

the ECA arteries the right carotid artery arises from the right brachiocephalic artery. Ultrasonography can show the most proximal segment of the common carotid artery. The left common carotid artery arises from the aortic arch. Ultrasonography cannot show the proximal segment of the left common carotid artery. The examiner should be able to differentiate the internal carotid artery (ICA) from the external carotid artery (ECA). The ICA is located posterior and lateral to the ECA. The ICA is slightly larger than the ECA. The ECA has branches such as the lingual artery, but the ICA does not. The Doppler spectrums from the ICA show a lower resistive pattern. The velocity difference between the systolic phase and diastolic phase of the ICA is smaller than that of the ECA. Another way to differentiate the ECA from the ICA is that during the acquisition of the ECA Doppler spectrum, placing the fingertips on the ipsilateral temporal artery generates a serration-like artifact on the Doppler spectrum from the ECA. This temporal artery tapping-induced artifact is not seen from the ICA. This so-called "temporal tapping" is a useful tool in differentiating the ICA from the ECA. Being certain of which is the ECA and the ICA is important in case one of them is occluded [21].

Patient position

For carotid ultrasonography, there are two options for the relative position between the patient and examiner. One is the overhead position, in which the examiner sits beyond the patient's head beside the end of the examination table and uses two hands for ultrasonography.

In this position, the examiner should use his right hand for the right carotid artery and use his left hand for the left carotid artery. The benefit of this position is that the examiner can use both hands and there are plenty of positions possible for the ultrasonography probe. The sonic window can be made wider and offers a clear view of the carotid artery especially from the poster lateral projection. The examiner should be familiar with using both hands, which requires some practice [21].

Another position is the usual lateral sitting position, which is used for most other ultrasonography examinations. The examiner uses his right hand for both carotid arteries. This position makes it easy to control the machines. However, the right posterior projection is a bit more difficult. Between these two choices, the overhead position for Doppler ultrasonography of the carotid artery is recommended [21].

A pillow is not necessary. In fact, it produces a poorer window for the carotid artery. The optimal patient head position is tilted about 45° away from the artery being examined. The neck of the patients should be relaxed. Contractions of the sternocleidomastoid muscle cause poor sonic penetration and make positioning of the probes difficult [21].

2.2.4 Intima-medial thickness

The intima-medial thickness (IMT) has been widely used as one of the parameters of atherosclerosis [22, 23]. The IMT is measured on a two-dimensional (2D) gray-scale image. The optimal gray-scale image of the longitudinal scan of the carotid artery, which passes by the center of the carotid artery, shows two bright interfaces along the artery wall. In the far wall, the upper bright line is the interface between the blood and intima, and the lower bright line is the interface between the media layer and adventitia layer. The interface between the intima and media does not produce any interface. The distance between the upper and lower bright line represents the thickness of the intima and media layer. It would be better that the carotid artery is parallel to the probe surface to minimize the overestimation of the IMT from the diagonal measurement. The IMT is generally measured on the distal common carotid artery at the far wall because the common carotid artery is easier to image and less variable than the ICA due to the angle of the beam or depth of the vessel. In one study, the success rate for far wall measurement was 89% (109/122) in the common carotid artery and 38%

(140/366) in the ICA [22]. The IMT should be measured on a segment without a focal lesion. Focal atherosclerotic lesions are much more common in the ICA than in the common carotid artery. Nowadays, many vendors provide an automated tool for measuring the IMT. Carotid artery atherosclerosis as measured by IMT is an independent risk factor for stroke and myocardial infarction [22, 24].

Plaque morphology and plaque volume

The plaque morphology, such as the echogenicity of the plaque, the surface, presence of ulceration, as well as the presence of plaque and stenosis, is important for predicting future cardiovascular events [25]. A description of the plaque morphology from a gray-scale image is highly recommended during carotid Doppler ultrasonography. The description should include the echogenicity of the plaque, the surface, and the presence of ulceration. The echogenicity of the plaque could be described as one of echogenic plaque, isochoric plaque, echo lucent plaque, or heterogeneous plaque. Iso-echoic plaque means that the echogenicity of the plaque is the same as that of the intima-media complex. The plaque surface can be described as smooth, irregular, or ulcerated. Plaque ulceration is associated with an increased risk of stroke [26, 27]. It is; however, very difficult to detect plaque ulceration by ultrasonography examination, and it is operator dependent. It is known that the sensitivity of detecting carotid plaque ulceration ranges from under 30% to over 80% when it is compared with pathological specimens [28, 29]. The effort to detect ulceration should be applied to increase accuracy in the assessment of risk of the patient with carotid plaque. Another problem is that such an ulcer is not clearly correlated with symptoms and is difficult to detect without careful gray-scale examination. The depression of the plaque surface by more than 2 mm is thought to indicate ulceration. The pattern of plaque ulceration can be cystic, bridge shaped, sponge-shaped, or a simple depression [30, 31]. To go into more detail on plaque echogenicity, it has been noted that symptomatic lesions are typically associated with purely or predominantly hypo echoic plaques. There has been an effort to measure plaque echogenicity quantitatively. Biasi et al, used longitudinal images of the plaque and vessel wall and measured the gray-scale median (GSM). The GSM of the blood pool was 0 to 5, and the GSM of the adventitia of the wall was 185 to 195 [32]. What they found was that the stroke risk during carotid stenting procedures is dependent on the GSM

of the plaque. Plaque with GSM values of 25 or less showed a 7.1% stroke risk while plaque with GSM values more than 25 showed only a 1.5% stroke risk. This means that echo lucent plaque is more vulnerable [31]. Recently, three-dimensional (3D) ultrasonography has been used for measuring plaque volume [33, 34]. On a 2D gray-scale image, plaque size can be measured based on length and height, but the total volume of the plaque cannot be measured. 3D ultrasonography showed good intra- and interobserver reproducibility for measuring total plaque volume [35]. The plaque volume can be used as a monitoring tool for atherosclerosis treatment. The plaque volume is known to increase without treatment and decrease with statin therapy [36]. 3D ultrasonography is thought to be useful for the monitoring of plaque and could also be useful for the evaluation of new treatments [37]. 3D ultrasonography volume measurements are more sensitive than IMT for the evaluation of carotid plaque progression post treatment. More specifically, while there was a significant change in the 3D plaque volume during the follow-up period, there was no change in the IMT.3D ultrasonography also can be used for plaque characterization. The limitation of 2D gray-scale evaluation of plaque is that single or even multiple images cannot represent the entire plaque volume.

Heliopoulos et al [38] tried to measure the echogenicity of the entire volume of plaque with 3D ultrasonography. In 110 symptomatic and 104 asymptomatic patients with carotid plaque disease, they assessed the mean gray value of the whole plaque and found a higher incidence of low echoic plaque in symptomatic patients than in asymptomatic patients, suggesting a higher risk of cerebral ischemia from the low echoic plaque [38].

2.2.6 Color Doppler ultrasonography and pulsed wave Doppler ultrasonography Color Doppler is color-encoded velocity information on a gray-scale image. Color Doppler is a good tool for visualizing the blood flow in the vessel and finding stenotic segments. To obtain a proper color Doppler image, an adequate acoustic angle is essential. With a linear probe, to generate a gray-scale image, the sonic beam needs to be perpendicular to the skin.

However, to obtain proper velocity information from color Doppler ultrasonography, the Doppler angle should be between 30° and 60°. The carotid artery is not a deeply located structure, and ensuring the proper angle of the Doppler probe surface relative to the common carotid artery is not easy. In contrast to measuring the IMT position, in which it is better for the vessel wall to be parallel to the probe surface, there should be an angle between the probe surface and vessel in color Doppler ultrasonography. One helpful technique for achieving this angle is the heel and toe technique. The heel and toe technique is a way of steering the probes. In the usual position for carotid artery scanning without any pressure, the probe surface will be parallel to the common carotid artery in most patients. Just pushing the head side edge or foot side edge will create a bit of an angle between the probe surface and vessel, and the optimal Doppler angle can be achieved. Adjusting the velocity range is one of the important ways of controlling Doppler ultrasonography parameters [39]. One of the purposes of color Doppler ultrasonography is finding any stenotic segment in the vessel. Because the flow volume through the vessel is constant, the velocity of the flow is fastest at the stenotic segment. If the upper limit of the color velocity scale is just below that of the flow velocity in the normal vessel, the increased flow velocity in the steno tic segment will be above the upper limit of the velocity scale and there will be an aliasing artifact. If there is a segment showing an aliasing artifact at the proper velocity scale setting, it means that the segment is stenotic. The usual normal velocity of the common carotid artery is 30-40 cm/sec [Kim S], but the velocity scale setting should be adjusted for each patient. However, to measure the exact flow velocity, we cannot rely on color Doppler imaging; we need pulsed wave Doppler. In pulsed wave Doppler, a small sample volume in the center of the vessel or in the stenotic segment will be used to check the velocity of the segment. The peak velocity is used for detecting significant stenosis. Angle correction is essential to measure the true flow velocity, and the angle correction should be along the flow direction, not along the vessel wall. The flow direction is usually not different from the vessel direction, but in cases of eccentric atherosclerotic plaque, the flow direction and vessel direction can be different [40].

Pitfalls in carotid Doppler ultrasonography

A calcified atheroma of the carotid artery can make it difficult to find a proper sonic window for color Doppler ultrasonography or pulsed wave Doppler. Trying different sonic windows such as poster lateral projection is helpful in some cases. If the obscured segment is less than 1 cm in size and there are normal waveforms at the proximal and distal parts of the plaque, it can be concluded that no significant stenosis exists. However, when the calcification is obscuring a large vascular segment, another imaging modality such as magnetic resonance angiography could be required [21]. In case of contralateral carotid artery stenosis, the PSV can be falsely elevated without significant stenosis. If there is critical stenosis of the ICA such as a residual luminal diameter of less than 0.7 mm, the peak systolic velocity of the affected segment can show pseudo-normalization. Color Doppler ultrasonography could depict this critical stenosis of the ICA [21].

Methods and Materials

Study Design

A descriptive cross-sectional study was conducted among patients with confirmed ischemic stroke who underwent carotid Doppler ultrasonography over six months (October. 2021 –

April. 2022) at Doppler units of two tertiary hospitals in Dhamar city.

Study Area & Setting

This study was carried out in Doppler units at AL-Ryadah International Hospital and Taiba Consultative Hospital, Dhamar city, Dhamar Governorate, Yemen.

Study Size

All patients with ischemic stroke were underwent carotid Doppler ultrasonography throughout the study's duration.

Exclusion criteria

i. Patients with ischemic stroke who weren't have carotid Doppler report.

ii. Patients with hemorrhagic stroke.

iii. Patients with history of head injury.

iv. Patients who refused to participate in this study.

Study Population

A total of 84 patients with ischaemic stroke and underwent carotid Doppler ultrasonography in Doppler units at AL-Ryadah International Hospital and Taiba Consultative Hospital, were successfully enrolled in our study.

Data Collection

Data were collected by a structured questionnaire including information about Sociodemographic characteristics (Age, Sex).

Detailed clinical history of risk factors such as hypertension, diabetes mellitus, hyperlipidemia, heart diseases and smoking were recorded from each patient.

The abnormal findings of carotid Doppler ultrasonography were documented in detail including which artery/s involved, side of lesions and the percentage of stenosis.

Statistical Analysis

Data processing, statistical analysis, and graph drawing were conducted using a Statistical Package for Social Sciences software SPSS (version 25.0).

Ethical Consideration

This study was introduced and approved by Thamar University Ethics Committee TUMEC (No: 22007). Consent was obtained from all participating patients before filling the questionnaire.

Results

A total of eighty-four patient were presented with acute ischemic stroke and underwent carotid Doppler during this study period. Among them, there were 54 patients (64.3%) were males and 30 (35.7%) were females. Their ages ranged from 22 to 100 years with mean age 64.9±15.6, the majority of patients 36 (42.9%) were aged between 51-70 years of age, followed by 31 (36.9%) were of age group above 70 years (**Table 1**).

Regarding to risk factors of cerebrovascular diseases among the studied patients, slightly less than half 40 (47.6%) were smokers, about one-third 28 (33.3%) were diabetic, the majority 57 (67.9%) had hypertension. Slightly more than one third 30 (35.7%) had heart disease, of those the most common type of heart diseases was ischemic heart disease, which represents about one half of all heart disease cases 14 (46.6%), other types were hypertensive heart disease, valvular heart disease and atrial fibrillation.

Other conventional risk factors were dyslipidemia in 10 (11.9%), chronic liver failure in 3 (3.6%) and chronic renal failure in 2 (2.4%). History of previous ischemic stroke was documented in 16 (19.0%) patients (**Table 2**) (**Figure 1**).

Blood pressure and heart beats were recorded for all study's participants and we found that, their systolic blood pressure was ranged from 90 to 230 mmHg with mean of 153.1 ± 31.8. The majority 57 (67.9%) of patients had high blood pressure, while hypotension was founded only in one patient (1.2%) (**Table 3**).

The majority 72 (85.7%) of studied patients had normal range of heart rate, while 11 patients had tachycardia (13.1%) (**Table 4**).

Carotid Doppler ultrasonography showed that, a normal findings were founded in about one third of the studied patients, while the majority 58 (69.0%) of patients had an abnormal findings with

Table 1. Socio-demographic characteristic of respondents (n = 84).

Characteristics		N	%
Gender	Male	54	64.30%
	Female	30	35.70%
Age Category	Less than 30 years	2	2.40%
	30 - 50 years	15	17.90%
	51 - 70 years	36	42.90%
	More than 70 years	31	36.90%

Table 2. Risk factors of cerebrovascular diseases among studied patients (n = 84).

Risk factor	Frequency	Percentage
Smoking	40	47.60%
Diabetes mellitus	28	33.30%
Hypertension	57	67.90%
Heart diseases	30	35.70%
Chronic renal failure	2	2.40%
Chronic liver disease	3	3.60%
Dyslipidemia	10	11.90%
History of previous stroke	16	19.00%

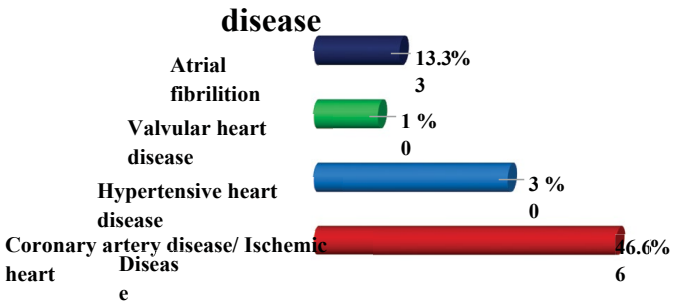

Figure 1 Type of heart disease among patients who had heart disease (n = 30).

Table 3. Blood pressure and heart rate values among studied patients (n = 84).

Variable		N	%
Blood pressure	Normal	26	31.00%
	High	57	67.90%
	Low	1	1.20%
Heart rate	Normal range	72	85.70%
	Tachycardia	11	13.10%
	Bradycardia	1	1.20%

Table 4. Carotid artery status, stroke side and plaques characteristics (n = 84).

Stroke side	Right	32	38.10%
	Left	52	61.90%
Carotid Doppler findings	Normal	26	31.00%
	Carotid artery stenosis	45	53.60%
	Diffuse atherosclerotic changes	13	15.50%
Side of carotid stenosis	Right	16	35.60%
	Left	10	22.20%
	Bilateral	19	42.20%
Location of atherosclerotic plaque	Internal carotid artery	19	42.20%
	Common carotid artery	9	20.00%
	Carotid bulb	15	33.30%
	Bifurcation of carotid artery	2	4.40%
Percentage of carotid artery stenosis narrowing	Mild (less than 50%)	20	44.40%
	Moderate (50 – 70%)	9	20.00%
	Severe (more than 70%)	16	35.60%
Percentage of significant stenosis	> 60%	23	51.10%
	< 60%	22	48.90%

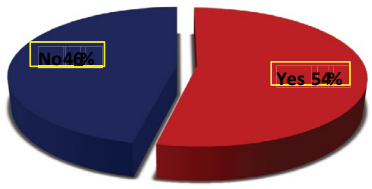

Figure 2 Prevalence of ceratoid artery stenosis among study participants (n=84).

some evidences of carotid atherosclerosis. Of the later, carotid artery stenosis (plaque) was founded in 45 (77.6%) patients and diffuses atherosclerotic changes founded in 13 (22.4%) patients.

Out of 84 patients included in this study, more than half 45 (53.6%) had carotid artery/arteries stenosis (**Figure 2**).

Plaques were found in proximal part of internal carotid artery in 19 patients (42.2%), carotid bulb in 15 patients (33.3%), and common carotid artery in 9 (20.0%) common carotid artery bifurcation in 2 patients (4.4%) (Table 4).

Regarding the side of carotid artery stenosis, the majority 19 (42.2%) of cases were have bilateral stenosis, followed by right side in 16 patients (35.6%), whereas the minority 10 (22.2%) of patients had left side stenosis.

When luminal narrowing of these affected carotid arteries with stenosis was calculated, it showed that 16 (35.6%) patients had severe i.e. more than 70% stenosis of the lumen, 9 (20.0%) had moderate i.e. 50-70% stenosis, 20 (44.4%) had mild i.e. less than 50% stenosis.

Among 45 patients who had carotid stenosis, percentage of significant stenosis > 60% was observed in more than half 23 (51.5%), while it was < 60% in the reminder of the patients 22.

By comparison the presence of carotid stenosis to patients' demographic characteristics and risks factors, we found that most of patients of elder age group (50 – 70 years and above 70 years of age) had carotid stenosis. 36 patients were aged between 50 – 70 years, of those the majority 22 (61.1%) had carotid artery stenosis, and 31 patients were above 70 years of age, also of the later the majority had carotid stenosis, while only the minority of patients less than 50 years of age had carotid stenosis, these findings was statistically significant with P value = 0.033.

From 54 male patients, the majority 32 (59.3%) had carotid artery stenosis, while only the minority of 30 female patients included in our study had carotid stenosis 43.3% (13 patients) but this wasn't statistically significant.

Carotid stenosis was founded in about one half (50.0%) of smoker patients and heart diseases patients (21 and 15 respectively).

The majority of diabetic, hypertensive and chronic liver disease patients had carotid stenosis (57.1%, 54.5% and 66.7% respectively), while there was no carotid stenosis in the two patients with chronic renal diseases included in our study, all of these findings were of no statistically significant.

The carotid stenosis was founded in the majority of patients with dyslipidemia and the majority of patients who had tachycardia (60.0% and 54.5% respectively).

Previous stroke history was reported in 16 patients, of those the majority 12 (75.0%) had carotid artery stenosis, which was statistically significant with P-value= 0.048 (**Table 5**).

Discussion

Large multicenter prospective studies such as North American Symptomatic Carotid Endartectomy Trial (NASCET) and European Symptomatic Trial (EST) have shown the etiological significance of carotid lesions in ischemic stroke patients [41]. The risk of ischaemic stroke increases with the degree of carotid stenosis [42]. Because patients with symptomatic occlusion of the internal carotid artery (ICA) and compromised cerebral blood flow are at risk for future ischaemic infarcts therefore early identification and accurate determination of the degree of carotid artery stenosis is useful in risk-factor management, with appropriate medical or surgical intervention [43].

The carotid stenosis was founded in the majority of the patients included in our study (45 patients (53.6%) out of 84 patients). Our result was in line with the finding of similar Pakistani and Chinese studies, which showed that the prevalence of carotid stenosis in their studies' patients was (56%) and (59.38%) respectively [44, 45].

The frequency of carotid stenosis in our study's participants was higher than frequency founded in similar Egyptian study, which published that carotid stenosis was founded in 41% of their study's patients [46]. Also it was higher than population in the Iran (49% of stroke patients [47].

Table 5. Carotid artery status vs. patients' characteristics and risk factors (n = 84).

		N	%	N	%	N	%		
Age	Less than 30 years	1	50%	1	50%	2	2.40%	8.373	0.033*
	30 – 50 years	3	20.00%	12	80.00%	15	17.90%		
	50 – 70 years	22	61.10%	14	38.90%	36	42.90%		
	More than 70 years	19	61.30%	12	38.70%	31	36.90%		
Gender	Male	32	59.30%	22	40.70%	54	64.30%	1.967	0.161
	Female	13	43.30%	17	56.70%	30	35.70%		
Smoking	Yes	21	50.00%	21	50.00%	42	50.00%	0.431	0.512
	No	24	57.10%	18	42.90%	42	50.00%		
Diabetes mellitus	Yes	16	57.10%	12	42.90%	28	33.30%	0.215	0.643
	No	29	51.80%	27	48.20%	56	66.70%		
Hypertension	Yes	30	54.50%	25	45.50%	55	65.50%	0.061	0.805
	No	15	51.70%	14	48.30%	29	34.50%		
Heart diseases	Yes	15	50.00%	15	50.00%	30	35.70%	0.239	0.625
	No	30	55.60%	24	44.40%	54	64.30%		
Type of heart diseases	IHD/ACS	7	50.00%	7	50.00%	14	46.70%	1.444	0.695
	HHD	4	44.40%	5	55.60%	9	30.00%		
	VHD	1	33.30%	2	66.70%	3	10.00%		
	AF	3	75.00%	1	25.00%	4	13.30%		
Chronic renal failure	Yes	0	0.00%	2	100%	2	2.40%	2.364	0.124
	No	45	54.90%	37	45.10%	82	97.60%		
Chronic liver failure	Yes	2	66.70%	1	33.30%	3	3.60%	0.214	0.643
	No	43	53.10%	38	46.90%	81	96.40%		
Dyslipidemia	Yes	6	60.00%	4	40.00%	10	11.90%	0.189	0.664
	No	39	52.70%	35	47.30%	74	88.10%		
Previous stroke	Yes	12	75.00%	4	25.00%	16	19.00%	3.816	0.048*
	No	33	48.50%	35	51.50%	68	81.00%		
Heart rate	Normal	38	52.80%	34	47.20%	72	85.70%	0.889	0.641
	Tachycardia	6	54.50%	5	45.50%	11	13.10%		
	Bradycardia	1	100.00%	0	0.00%	1	1.20%		

Among all patients with carotid stenosis, bilateral carotid athermanous plaques were seen in 19 (42.2%) patients in the current study. Bilateral plaques were observed in 31.7%, 35% and 46% according to Boliipo et al., Khan et al., and Chamarthi et al. respectively [48-50].

Sixteen patients (35.6%) had carotid stenosis more than 70%, while 20 patients (44.4%) showed stenosis between 50 – 70%. These percentages of carotid stenosis were consistent to that of a previous Egyptian study, which documented that a significant carotid stenosis (>70%) and stenosis (< 70%) were founded in 23.8% and 40.5% of their patients respectively.

In our study the sever carotid stenosis found to be higher than population of a previous Iranian study (18%) [47] and Saudi study (9%) This high percentage of significant carotid stenosis in our study's patients could be explained by the high percent of vascular risk factors associated with this group, which play an important role for developing atheroma.

In the present study, the most common site of carotid plaques carotid found at the proximal part of internal carotid artery (42.2%), followed by (33.3%) at the carotid bulb. Our findings were consistent with a similar study by Khan et al, who revealed that the most common site of carotid stenosis was at carotid bulb followed by proximal part of internal carotid artery while Bharathi et al, documented that carotid stenosis was found at the bifurcation of CCA, and the origin of ICA.

Older age is an important and well known risk factor for the development of Carotid artery atherosclerosis. In our study the majority (79.8%) of patients with stroke were older than 50 years and when we compared this age subgroup having carotid stenosis (41/45) to the patients having no stenosis (26/39) , the difference was statistically significant (P-value = 0.033).

These findings were consistent with certain international studies. An Indian study by Sethi et al found that mean age of patients with carotid lesion was 60.03 years as compared to 48.83 years in patients without any carotid lesion. Kerenyi also noted that mean age of the patient with CAS was 66.9 ±12.8 years.

The majority of our patients with carotid artery stenosis were males but this gender difference was found to be not significant (P-value = 0.161), which was consistent with certain previous studies. An Egyptian study by Khedr et al documented that the majority (87.4%) of patients with carotid stenosis were males Shaikh et al also noted similar finding, 66.6% of patients were males [45].

Conventional risk factors in the study participants were hypertension in 55 patients (65.5%), smoking in 42 (50.0%), heart diseases in 30 patients (35.7%), diabetes in 28 patients (33.3%),

previous stroke history in 19 patients (19.0%) and dyslipidemia in 10 patients (11.9%). These were the independent risk factors of ischemic stroke in previous published studies as well.

Hypertension was the most common risk factor present in 55 (65.5%) of cases either as a single risk factor or associated with other risk factors. Among all patients who had carotid stenosis, 30 (66.7%) was hypertensive. Elevated systolic blood pressure accelerates the progression of intima medial thickness (IMT) in the carotid artery. Our result was lines well with previous study by Patel et al., who reported that, hypertension was the most common and most significant risk factor.

Smoking is widely accepted as one of the important risk factor for ischaemic stroke in western countries, and is associated with the progression of carotid plaques Smoking is associated with raised fibrinogen levels, increased packed cell volume, and decreased macrophage activity changes in lipid biochemistry. Smoking increases arterial wall stiffness and alters the pattern of arterial blood flow. In our study smoking was present in 30 (35.7%) cases. However the association between smoking and carotid stenosis wasn't statistically significant. A similar finding was documented by Shaikh et al, who found that smoking was present in 43.59% cases [45].

Atherosclerosis is presumed to be accelerated in diabetes for a number of reasons. First, diabetes is associated with an increased risk of traditional coronary heart disease (CHD) risk factors, including hypertension, dyslipidemia, obesity, and hyperinsulinemia, other metabolic disturbances unique to diabetes, such as increased levels of circulating glucose, advanced glycation end products, and oxidation of lipoproteins might also increase the risk and rate of atherosclerosis. In our study, carotid stenosis was affected more than half 16 (57.1%) of diabetic patients, which was consistent with many previous studies.

Higher level of low density lipoprotein (LDL) cholesterol are associated with higher incidence of carotid atherosclerotic disease while high levels of high density lipoprotein (HDL) cholesterol have protective role In the present study, carotid stenosis was founded in most 6 (60%) of patients with hyperlipidemia. This was consistent with study conducted by Patel at al, which reported that atherosclerosis was present in most patients who had dyslipidemia.

In our study 15 (50.0%) of patients with heart diseases had carotid stenosis, of those 7 (46.7%) patients had Ischaemic heart disease, which was consistent with findings of previous study conducted by Khan et al, as their study showed that 25% of patients with coronary artery disease had carotid artery stenosis of more than 50% while overall about 94% of patients had some evidence of plaque. This discrepancy could be attributed to the small sample size in our study.

In our study a history of previous ischemic stroke was documented in 16 (19.0%) patients, of those 12 (75.0%) patients had carotid stenosis, which was statistically significant with P value = 0.048. Patel at all, reported that previous stroke history was present in 12 patients (26%).

Conclusion

Doppler sonography demonstrated that 53.6% patients with ischemic stroke had plaques involving carotid vessels. Significant carotid stenosis (> 70%) was observed in 35.6% patients. Most common site of plaque was proximal part of internal carotid artery. The plaques were most commonly noted on both sides. Increasing age >50 years, male sex, smoking, hypertension, diabetes and hyperlipidemia were associated with increased rate of atherosclerosis, carotid stenosis and ischemic stroke. Carotid Doppler is a very useful, noninvasive modality, accurate and less time consuming method in detecting the site, morphology of atherosclerotic plaque, quantifying the degree of stenosis. Early detection of stenosis by carotid Doppler in high risk patients would be of great importance in managing and preventing cerebral ischemia or infarct.

Recommendations

Doppler should be performed in all confirmed cases of stroke for detection the possibility of carotid stenosis in order to plan out medical / and surgical intervention for the primary as well as secondary prevention of cerebrovascular events.

Hypertension, diabetes mellitus, heart diseases, dyslipidemia, smoking and previous history of cerebrovascular events must be considered red flag signs for stroke. Their presence, especially if multiple, warrants thorough clinical auscultation, and if any suspicion, a carotid Doppler even in patients without stroke

In future routine screening of high risk patients at regular interval is necessary to prevent the occurrence of stroke.

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