# THE PATTERN OF RADIOLOGICAL FINDINGS SEEN IN FOUR-VESSEL CEREBRAL ANGIOGRAPHY DONE AT KNH AND NAIROBI HOSPITAL

## DISSERTATION

TO BE SUBMITTED IN PART FULFILMENT FOR THE DEGREE OF MASTER OF MEDICINE IN DIAGNOSTIC RADIOLOGY, UNIVERSITY OF NAIROBI



BY

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To the patients with cerebrovascular pathologies; may the art of medicine continually unfold sense to you.

This book is dedicated to my parents who nurtured learning atmosphere against all the odds in my early life.

# Declaration

I, Dr. Chepsiror Kimeli David declare that the work contained herein is my original idea and has not been presented at any other place to the best of my knowledge.

Signature.....Date. 30/8/2006.....

## Approval by supervisors

This research dissertation has been submitted with our approval as university supervisors.

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## List of abbreviations

ACA .....Anterior Cerebral Artery

ACoA .....Anterior Communicating Artery

AICA.....Anterior inferior cerebellar artery

AJR .....American Journal of Radiology

ASVD.....Atherosclerotic vascular disease

AV.....Arteriovenous

AVM .....Arteriovenous - Malformation.

CNS.....Central nervous system

CT .....Computed Tomography

CTA. .....Computerized Tomographic Angiography

DDR.....Department of Diagnostic Radiology, University of Nairobi

DSA .....Digital Subtraction Angiography

DVA.....Developmental venous anomalies

FMD.....Fibromuscular dysplasia

HbSAg .....Hepatitis B surface antigen

HIV .....Human immunodeficiency virus

ICA.....Internal Communicating Artery

ICA-PCoA. .....Posterior Communicating Artery aneurysm

IU .....International Units

KNH.....Kenyatta National Hospital

MCA .....Middle Cerebral Artery

MRA. .....Magnetic Resonance Angiography

MRI.....Magnetic Resonance Imaging

PCoA.....Posterior Communicating Artery

PICA .....Posterior inferior cerebellar artery

PTI .....Prothrombin time index

SAH .....Subarachnoid haemorrhage

SHA. .....Superior Hypophyseal Artery

VA.....Vertebral Artery

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# ABSTRACT

## Introduction

Most of the intracranial cerebrovascular pathologies commonly present with subarachnoid hemorrhages and cerebrovascular accidents, which are associated with significant morbidity and mortality. Imaging plays a crucial role in diagnosis and subsequent management of these patients. The imaging modalities available include catheter angiography, computerized tomographic angiography and magnetic resonance angiography. Four-vessel cerebral Digital Subtraction Angiography (DSA) is used to make accurate diagnosis and pre-operative delineation of intracranial aneurysms, and arteriovenous malformation (AVM). It also provides information about the calibre and occlusion of cerebral vasculature. DSA thus plays an essential role in planning neurosurgical and interventional neuroradiological procedures

## Objective

The purpose of the study was to evaluate the pattern of cerebrovascular diseases as seen at a teaching and a tertiary angiographic centre in Nairobi.

## Methodology

This was a cross-sectional descriptive study of angiographic findings of patients who were done four-vessel studies at KNH/Nairobi Hospital CATH laboratories from January 2005 to January 2006.

## Results

A total of 88 patients were evaluated with four-vessel digital subtraction brain angiography during the study period. The male to female ratio was 1:1.1.The mean age was 40.3 years. Angiographic pathology was identified in 77.3% of the study subjects. Angiographic findings included aneurysm (40.9%), vascular stenosis/spasm (14.8%), AVM (8.0%) and angiographic normal (22.7%). Anterior (carotid) circulation

aneurysms arising within the circle of Willis accounted for 95.2% of all the aneurysms. Aneurysms were multiple in 16.7 % of all cases with male to female ratio of 1:5.

**Conclusion**

The pattern of cerebrovascular disease seen in KNH and Nairobi Hospital was comparable to those in other setups. Cerebrovascular pathologies were commonest in the age group 41-60 years. Aneurysms were the commonest cerebrovascular pathologies and they were significantly more ($p=0.007$) in females compared to males. Slightly more than a fifth of all cases were angiographically negative.

# 1.0: INTRODUCTION

The advent and near universal acceptance of non-invasive angiographic techniques such as computerised tomographic angiography (CTA) and magnetic resonance angiography (MRA) has significantly altered the criteria for catheter angiography. The non-invasive techniques coupled with three-dimensional capabilities provide unlimited range of projections with minimal vascular overlap. They are therefore ideal for screening populations with high risk of cerebrovascular diseases such as Fibromuscular dysplasia (FMD), Adult Polycystic kidney disease (APKD) and those with history of sudden onset of severe headache ('thunder clap headache')[1].

Conventional cerebral angiography though a gold standard for imaging cerebral vessels has largely been replaced by the non-invasive techniques. This is due to associated risks related to catheter placement, contrast agents and their injections. Modern cerebral angiography however entails use of intra-arterial digital subtraction techniques (DSA). These newer techniques involve use of reduced doses of contrast media, smaller calibre of catheters, and shortened duration of procedure. With above advances, modern cerebral DSA remains safe and reliable with an overall complication rate of less than one percent[2, 3, 4]. DSA as a dynamic near real time vascular study permits evaluation of entire carotid and vertebrobasilar systems with demonstration of vascular transit time. It allows accurate diagnosis and preoperative delineation of intracranial aneurysms, and arteriovenous malformation (AVM). It also provides information about the calibre and occlusion of cerebral vasculature. It thus plays an essential role in planning neurosurgical and interventional neuroradiological procedures[5].

DSA provides only two-dimensional projection of the cerebral vessels. The use of biplanar system improves visualization of pathologic entity, but numerous oblique views are often necessary to find intracranial aneurysms and precisely analyse their angioarchitecture.

Multiple oblique views may be automatically obtained using rotational angiographic acquisition. Three-dimensional DSA images can then be reconstructed from rotational sequences. Three-dimensional DSA supplements two-dimensional DSA in evaluation of complex intracranial aneurysms. This is because CTA and MRA have inferior spatial

resolution and lower sensitivities in detection of small (<3mm) intracranial aneurysms and do not enable imaging of entire cerebral vasculature[6].

There is little data on clinical epidemiological and radiological features of cerebrovascular diseases in East Africa. Ruberti (40) saw 6-8% of approximately 10,000 African patients in 30 years of practice. Moreover two-dimensional DSA, CTA and MRA are available only in few tertiary centres mainly located in Nairobi.

The purpose of this study was to evaluate the radiological findings in those patients who present for four-vessel angiography.

# 2.0: LITERATURE REVIEW

## 2.1: Cerebrovascular anatomy

Blood supply to intracranial structures is by internal carotid and vertebro-basilar artery systems, which anastomose at the ventral surface of the diencephalon forming an interconnecting arterial polygon (circle of Willis) [7].

## 2.2 Internal carotid artery circulation

Each internal carotid artery (ICA) arises from the bifurcation of common carotid artery at the mid cervical level. The extracranial segment of ICA consists of the carotid bulb and cervical segments. The cervical segment has neither narrowing nor dilatations, almost never branches and does not taper. The ICA enters the petrous portion of the temporal bone at the base of the skull through the carotid canal. Within the petrous bone, the carotid artery runs vertically and then turns horizontally at its genu to travel in an anteromedial direction, forming the carotid siphon. As the carotid artery passes above the foramen lacerum and under the Gasserian ganglion, it penetrates the lateral dural ring and turns medially, forming the lateral carotid loop, to enter the cavernous sinus. In the cavernous sinus, the carotid artery proceeds in a superomedial direction toward the posterior clinoid process. At the level of the posterior clinoid, the carotid artery makes a forward turn, forming the medial loop. The meningohypophyseal trunk originates at this level. The carotid then exits the cavernous sinus and enters the subarachnoid space.

The ophthalmic segment of the internal carotid artery extends from the distal dural ring to the origin of the posterior communicating artery. This is the longest subarachnoid segment of the internal carotid artery, and it possesses two major bends that create areas of haemodynamic stress that predispose to aneurysm formation. The first bend, best seen on lateral angiographic views, occurs as the carotid artery ascends and bends sharply posteriorly after penetrating the dura. The second bend, best appreciated on a dorsal (or anterior-posterior) angiographic view, is a gentler, medial-to-lateral curve as the artery runs medial to the anterior clinoid process and arcs laterally to ascend toward

the bifurcation. The ophthalmic segment has two major branches. The ophthalmic artery usually arises immediately beneath the optic nerve. The superior hypophyseal artery arises from the medial or ventromedial surface of the carotid, below the anterior clinoid process. Ophthalmic aneurysms typically arise along the first bend of the internal carotid artery, distal to the origin of the ophthalmic artery, and project either dorsally or dorsomedially toward the optic nerve. Superior hypophyseal artery aneurysms usually arise from the inferomedial surface of the internal carotid artery and project superomedially[7,8,9].

The posterior communicating artery originates from the posteromedial surface of the internal carotid artery and penetrates the membrane of Liliequist to join the posterior cerebral artery inside the interpeduncular cistern. Several perforators originate from the carotid or posterior communicating artery (anterior thalamoperforating arteries). Posterior communicating aneurysms project posteriorly and slightly inferiorly. The choroidal segment of the internal carotid artery begins at the origin of the anterior choroidal artery and ends at the carotid bifurcation. The anterior choroidal artery arises distal and lateral to the posterior communicating artery. The internal carotid artery then bifurcates into the anterior and middle cerebral arteries. The middle cerebral artery begins at the bifurcation of the internal carotid artery and runs along the sylvian fissure. It can be divided into the following 4 segments: An M1 segment located between the carotid bifurcation and the genu, an M2 segment that runs over the insular surface, an M3 segment that traverses the opercular surface of the sylvian fissure to reach the cortical surface, and a distal M4 segment consisting of its cortical branches.

The anterior cerebral artery (ACA) is the smaller of the two terminal ACA branches. It can be divided into the following three segments; A1, A2, and A3.The A1 segment extends medially from ACA origin to its junction with anterior communicating artery (ACoA). Medial lenticulostriate arteries arise from this segment. A2 segment includes the ACA from its junction with ACoA to its bifurcation into the pericallosal and callosomarginal arteries. Recurrent artery of Heubner is a lenticulostriate branch that typically arises from proximal A2.A3 segment consists of the cortical branches after the bifurcation near the callosal genu[7,8,9].

## 2.3 Vertebro-basilar circulation

Both vertebral arteries (VA) arise from the first part of each subclavian artery. Each course vertically through the foramina transversaria of upper six cervical vertebrae. The VA enters the subarachnoid space at the cranio-occipital junction. The first branch is the posterior spinal artery, which descends into the spinal cord. The vertebral artery then courses medially and superiorly around the medulla. The most important branch is the posterior inferior cerebellar artery (PICA), which travels in a posterior and lateral direction, just inferior to the olive. The basilar artery begins at the vertebrobasilar junction and courses superiorly toward the interpeduncular fossa. The first major branch of the basilar artery is the anterior inferior cerebellar artery (AICA), which courses laterally and posteriorly, to supply the inferior surface of the cerebellum. The superior cerebellar artery (SCA) originates just proximal to the basilar bifurcation and courses laterally to supply the superior cerebellar hemisphere.

The basilar artery terminates in the interpeduncular fossa where it bifurcates into the posterior cerebral arteries. The posterior cerebral artery consists of three segments as follows: The P1 segment, which extends from its origin at the basilar bifurcation to its junction with the posterior communicating artery and contains several posterior thalamoperforating arteries; the P2 segment, which courses through the crural and ambient cisterns, giving origin to the anterior temporal, hippocampal, medial posterior choroidal, and peduncular perforating arteries, middle and posterior temporal arteries, and lateral posterior choroidal arteries; and the P3 segment, which courses through the quadrigeminal cistern toward the calcarine fissure, where it divides into calcarine and parieto-occipital arteries[7,8,9].

## 2.4 Cerebral venous anatomy

The cerebral venous system is composed of dural sinuses and superficial and deep cerebral veins. Superficial sagittal sinus typically originates from crista galli anteriorly and extends posteriorly to its confluence (torcular Herophili) with straight and transverse sinus. Inferior sagittal sinus runs in the inferior margin of the falx cerebri. It joins with vein of Galen to form the straight sinus. Vein of Galen is a short venous

channel that receives blood mainly from internal cerebral veins and basal veins. Superficial cerebral veins drains into the superior sagittal sinus. Torcular Herophili divides into right and left transverse sinuses. Each transverse sinus continues as sigmoid sinus near the posterolateral wall of petrous temporal bone. Sigmoid sinus then continues inferiorly as internal jugular vein at the skull base [7,8,9].

## 2.5: Cerebrovascular pathology

Of all cerebrovascular accidents, 3% are due to subarachnoid hemorrhage (SAH), which is responsible for 5% deaths due to brain infarctions. The mortality rate (including deaths before admission may exceed 50% Intracranial aneurysms are the most common cause of non traumatic subarachnoid hemorrhage, occurring in 60% - 85% of cases [41]

## 2.6: Intracranial aneurysms

An aneurysm is an abnormal local dilatation in the wall of a blood vessel usually an artery, due to a defect, disease and injury. The three major types of intracranial aneurysms are saccular, fusiform and dissecting [8].

## 2.7: Classification of intracranial aneurysms

Intracranial aneurysms are classified as saccular, fusiform or dissecting. Saccular aneurysms include: developmental/degenerative, traumatic, mycotic, oncotic, flow related, vasculopathy related and drug related types.

## 2.8: Saccular aneurysms

These are rounded; berrylike outpouching that arises from arterial bifurcation points. There are true aneurysms i.e. they are dilatations of a vascular lumen caused by weakness of vessel wall layers [8]. Most intracranial aneurysms probably result from haemodynamically induced degenerative vascular injury [10]. The occurrence, growth, thrombosis, and even rupture of intracranial saccular aneurysms can be explained by

abnormal haemodynamic shear stresses on the walls of large cerebral arteries, particularly at bifurcation points.

The conditions associated with increased incidence of cerebral aneurysm are anomalous vessels, coarctation of the aorta, polycystic disease of the kidney, fibromuscular dysplasia, connective tissue disorder e.g. Marfan, Ehlers- Danlos, high flow states (e.g. AVM, fistula) and spontaneous dissection[8]

Gender influences the prevalence of aneurysms at certain anatomic locations. In females, the most common location of aneurysms is the supraclinoid segment of the internal carotid artery. In males, the most common site of ruptured aneurysms is the anterior communicating complex, while the most common reported site of unruptured aneurysms is the supraclinoid carotid. Females are more prone than males to developing aneurysms of the ophthalmic, cavernous, and posterior communicating segments of the internal carotid artery[11].

Intracranial aneurysms are multiple in 15 -20% of all cases. About 75% of patients with multiple intracranial aneurysm have two aneurysms, 15% have three and 10% more than three. A strong female to male ratio of 5:1 is observed. Aneurysms typically become symptomatic people aged 40 -60 years. In paediatric age group aneurysms are more often post traumatic or mycotic rather than degenerative and they have slight male predilection. Aneurysms in children are also larger than those found in adults averaging 17 mm in diameter[8,12].

85% of all intracranial aneurysms arise at anterior (carotid) circulation. Common locations are anterior communicating artery 30 -35% internal carotid artery (ICA) at the posterior communicating artery origin 30 -35% and the MCA bifurcation 20%[13].

Fifteen percent of all intracranial aneurysms arise on the posterior (vertebro-basilar) circulation 5% arise from basilar artery bifurcation and the remaining 1-.5% arise from posterior fossa sites include the superior cerebella artery and the vertebral artery at the origin of PICA. AICA aneurysms are rare[13].

Miscellaneous locations - saccular aneurysms are uncommon in locations other than the circle of Willis, the MCA bifurcation, or the pericallosal artery origin. When aneurysms occur at distal sites in the intracranial circulation, they often caused by trauma or infection.

Most aneurysms are asymptomatic until they rupture. On rupture they are associated with significant morbidity and mortality. The most common presentation of intracranial aneurysm is SAH. Other uncommon symptoms of intracranial aneurysm include cranial neuropathies, intracerebral haemorrhage, transient ischaemic attacks or cerebral infarction secondary to emboli. Vasospasm is the leading cause of disability and death from aneurysm rupture [14,15]

## 2.9: Fusiform aneurysms

These are also known as atherosclerotic aneurysms. The lesions are accelerated arterial ectasias that occur because of severe unusual form of arteriosclerosis .Arterial media damage results in stretching and elongation that may extend over a considerable length. The vessels may have more focal areas of fusiform or even saccular enlargement. Intraluminal clots are common and perforating branches often arise from entire length of the involved patent vessel. Fusiform aneurysms usually occur in older patients [8,10].
The vertebro-basilar system is commonly affected. Fusiform aneurysms may thrombose, producing brainstem infarction. They can also compress adjacent brain or cause cranial nerve palsies [8].

## 2.10: Dissecting aneurysms

In arterial dissection's blood accumulates within the vessel wall through a tear in the intima and internal elastic lamina. The consequence of this in terms of haemorrhage varies. If blood dissects subintimally, it causes luminal narrowing or even occlusion Dissecting aneurysms may arise spontaneously but more commonly, trauma or an underlying vasculopathy such as FMD is implicated. Most dissecting aneurysms that involve the craniocerebral vessels affect the extra cranial segment; intracranial

dissections are rare and usually associated with severe head trauma. ICA is commonly affected as well as VA [8,10,16,17].

## 2.11: Angiographic findings of intracranial aneurysms

A patent intracranial aneurysm is seen as contrast filled outpouching that commonly arises from an arterial wall or bifurcation. The circle of Willis and middle cerebral artery are the common sites. Thrombosed aneurysms usually have normal angiographic studies. Large thrombosed aneurysms can cause an avascular mass effect. The definitive diagnosis and preoperative delineation of intracranial aneurysms is achieved with catheter angiography. The role of diagnostic cerebral angiography in patients with non traumatic SAH is to identify the presence of any aneurysm, to delineate the relationship of aneurysm to its parent vessel and adjacent penetrating branches, to define the potential for collateral circulation to the brain and to arise for vasospasms[8].

Fusiform aneurysms have bizarre shape, with serpentine or giant configuration. Intraluminal flow is often slow and turbulent. Fusiform aneurysms typically do not have identifiable neck. Dissecting aneurysms are elongated; ovoid or saccular contrast collections that extend beyond vessel lumen [8,17,18].

## 2.12: Intracranial vascular malformation

Intracranial vascular malformations are traditionally divided into four basic types namely arterio-venous malformation, venous malformation, capillary telangiectasia and cavernous angioma [7].

### 2.12.1: Arterio-venous Malformations (AVM's)

AVM's are complex network of abnormal vascular channels that consist of arterial feeders, arterial collaterals, and the AVM nidus and enlarged venous outflow channels [19]. AVM's are categorized by their blood supply. Pial or parenchymal AVMs are supplied by carotid or vertebral circulation. Dural AVMs are supplied by external

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carotid circulation and mixed AVMs by both. A paediatric variant of AVM is the vein of Galen aneurysm in which AVM drains into and dilates great vein of Galen.

AVM's can be found throughout the CNS. Both sexes are equally affected. The common age of presentation is between 20 and 40 years. They may be microscopic or large enough to involve the entire hemisphere of the brain. 90% of AVMs are supratentorial and tend to occur at watershed areas and 10% are infratentorial. 70% of supratentorial AVM's are purely pial with no meningeal or dural blood supply. The remainder of lesions are partly dural or pial (mixed pial- dural AVM). Parenchymal AVMs are the common symptomatic vascular malformation [7,8,19].

Pial AVMs lie within brain parenchyma and derive blood from the cerebral arteries namely anterior cerebral artery (ACA) middle cerebral artery MCA, or posterior cerebral artery (PCA). The rapid shunting of blood typical of pial AVM's is visualized as an abnormal tangle of blood vessels with early and frequently rapid, venous drainage, uniquely demonstrated by 4-vessel angiography[8].

Dural AVM's are almost always infratentorial. They most frequently drain into the transverse and sigmoid sinuses in the posterior fossa, but may also involve the cavernous sinus, inferior petrosal sinus, and superior sagittal sinus. The occipital artery and meningeal branches of ECA are the vessels that mostly supply dural AVM components [7].

Symptoms of an AVM may include headache, weakness, numbness, visual problems or most often the abrupt onset of shock. Usually AVM's are clinically silent initially and then become symptomatic in second, third or fourth decade of life. Spontaneous rupture with haemorrhage is the presenting symptom in 30 -55% of pial AVM'S [20,21,22].

Angiography reveals certain features that are believed to correlate with increased risk of haemorrhage such as presence of intranidal, remote or peduncular aneurysms, central or deep venous drainage, stenosis of draining vein; and periventricular or intraventricular location.

CTA and MRA are usually the imaging studies of choice in-patients with suspected AVM. CT can identify intracranial haemorrhage, vascular calcifications associated with AVM. MRI typically demonstrates areas of parenchymal AVM involvement, showing both dilated feeding arteries and enlarged draining veins [23].

DSA still remains the criterion standard for characterization and delineation of brain and spinal AVM's. Patent parenchyma (pial) AVMs are seen as enlarged arteries and veins with AV shunting, "early" draining veins with minimal mass effect with "stagnant flow" and subtle AV shunting. Dural AVMs show enlarged dural arteries with AV shunting stenosis or occluded dural sinus is common. DSA can be used to measure the size of the AVM and judge the compactness of the nidus further, angiography can be used to evaluate venous drainage pattern (superficial, deep or mixed). In addition DSA frequently depicts associated risk factors for haemorrhage including aneurysms and venous stenosis [7,24].

**2.12.2: Venous angiomas**

•There are composed of radially arranged, dilated anomalous veins that converge into an enlarged, transcortical-draining vein [25].

Although the precise aetiology of venous angioma is unknown, these lesions are probably not true vascular malformation but instead represent extreme anatomic variants or developmental venous anomalies (DVA's)[26]. Arrested venous development after brain arterial system has been formed could result in retention of primitive embryologic medullary veins that drain into a single large draining veins and form a 'venous angioma

Venous angiomas are located in the deep cerebral or cerebellar white matter most often near the margin of adjacent ventricle. The most common site is adjacent to frontal horn of lateral ventricle [27] the next most frequent location is the cerebellum [28]. Most venous angiomas are asymptomatic and discovered incidentally at autopsy or on imaging studies. Headache, seizure and focal neurological deficit are less common.

Cerebral Angiograms always have normal arterial phase. Although a late capillary blush may be present, the pathognomonic Angiographic appearance of venous angioma is seen on venous phase images. A collection of dilated medullary veins ("medusa head") converges in an enlarged transcortical collector draining veins [28,29,30].

### 2.12.3: Vein of Galen malformation (VOGM)

VOGM is used to describe a heterogeneous group of anomalies with enlarged deep venous structures of Galenic system that are fed by abnormal midline Arterio-venous communications [31].

There are two basic types of VOGM.

**(1) Fistula group**

Single or multiple arteries drain directly into enlarged deep venous structures of the Galenic system. The most common abnormality is single or multiple direct artero-venous fistulae between-choroidal, or quadrigeminal arteries and a median venous sac. The sac probably represents persistence of a primitive venous channel that is embryonic precursor of the vein of Galen [32].

**(2) VOGM with parenchymal AVM**

The AVM is usually in the Thalamus or midbrain and its nidus has deep Galenic drainage. Venous outflow constrain is also common in this group [31]. Patients with fistulae present early in life (often at birth). High output congestive heart failure and macrocephaly with hydrocephalus are the common symptoms. Developmental delay and ocular symptoms are more common in VOGMs with true AVM nidus in the thalamus or brainstem.

Arterial supply to VOGMs is usually via enlarged choroidal and thalamoperforating branches. In fistula Group, posterior choroidal vessels are the dominant supply followed by anterior choroid, thalamoperforating, and anterior cerebral artery branches. In the nidus type, thalamoperforating vessels are common arterial feeders [8,31].

## 2.13: Atherosclerosis

Atherosclerosis is the most important cause of cerebral vascular stenosis in adults. Craniocerebral arteriosclerosis vascular disease (ASVD) occurs commonly and most severely at the internal carotid artery origin and the distal basilar artery [33,34].

ASVD is the principal cause of cerebral ischaemia and infarction and its sequale. Cerebral DSA permits an evaluation of the entire carotid and vertebro-basilar system, providing information about tandem atherosclerotic disease, plague morphology and collateral circulation [4,34]. ASVD is seen at angiography as vessel irregularity, elongation or tortuosity, and narrowing or frank occlusion.

In Cervicocranial Angiography in ASVD the three most important goals are as follows. Determine the degree of carotid stenosis, identify "tandem" lesions in the carotid siphon or intracranial circulation and evaluate existing and potential collateral circulation [4].

## 2.14: Non atheromatous causes arterial narrowing and occlusion

Other causes of vascular stenosis in children and older individuals include arterial dissection (traumatic, spontaneous/underlying vasculopathy), vasospasm (SAH, trauma infection) Fibrous muscular dysplasia, drug - abuse and tumour encasement (pituitary adenoma, nasopharyngeal squamous cell carcinoma). Idiopathic progressive Arteriopathy of childhood (Moyamoya) disease is an occlusive cerebrovascular disorder of unknown aetiology commonly seen in Japan but has been reported elsewhere as well [35].At angiography, contrast fills numerous enlarged lenticulostriate and thalamoperforator arteries, as well as dural, leptomeningeal and pial collateral vessels leading to "puff of smoke" appearance for which the disease is named. The Moyamoya pattern of collateral blood flow is non-specific and can be seen in any slowly progressive intracranial occlusive vascular disorder such as arteriosclerosis, radiation induced Angiopathy and sickle cell disease [36, 37].

# 3.0: JUSTIFICATION

There has been no similar cerebrovascular (DSA) study done in our setup before. A study by Ruberti [40] on the patterns of cerebral vascular disease in East Africans was based on intra-operative findings and at a time when DSA and CTA/ MRA were not available. Currently, CTA and MRA are available in a few tertiary health service provider centres in Nairobi and furthermore, they are still largely unaffordable to the majority of the patients. This situation has therefore left DSA studies as the most effective way of depicting cerebrovascular pathology in our setup.The findings of this study will broaden the understanding of the pattern of cerebrovascular pathologies in the local population.

Though interventional vascular radiology in Kenya is still rudimentary, the knowledge of pattern of four-vessel cerebrovascular disease diagnosed through DSA will lead to improved pick up of the cases from the population, better therapeutic decision making and planning of especially surgical intervention.

Possession by physicians of forehand knowledge of pattern of four-vessel cerebrovascular pathology for our setup will enhance their capability on alertness to be able to suspect clinically these pathologies and make focused and directed requests for appropriate radiological diagnosis. This in turn will ensure efficient and cost effective diagnosis and management of patients with these pathologies. The new knowledge generated vide this study will complement the one applicable now from western studies and allow our physicians to adapt it to our local settings and population.

# 4.0: OBJECTIVES

## 4.1: Broad objective

The main objective of this study was: to determine the pattern of cerebrovascular pathology as shown by 4 vessel cerebral angiography.

## 4.2: Specific objectives

1. To determine angiographic features of patients undergoing 4-vessel cerebral DSA study;
2. To determine the anatomical distribution of the lesions;
3. To correlate with age and sex distribution of these patients.

## 4.3: Research questions

1. What are the socio-demographic characteristics of patients who undergo 4-vessel cerebral DSA at KNH and Nairobi hospital?
2. How are age and sex related to radiological findings?
3. What is the anatomical distribution of the lesions that are found at 4-vessel cerebral DSA?

# 5.0: STUDY METHODOLOGY

## 5.1: Study area

The study was carried out at Kenyatta National Hospital (KNH) Cath-Lab and Nairobi Hospital radiology departments. KNH is the only national referral centre with cerebral DSA facilities. The same facility is used for cardio-angiographic procedures. Nairobi hospital is a non-profit making private facility. It has a well-equipped DSA facility and works in close collaboration with KNH Cath-lab. These are the only two institutions with neurointerventional capabilities in the country and may be the region as well.

## 5.2: Study population

The study population consisted of angiograms of patients who had been sent to KNH Cath-Lab and Nairobi Hospital radiology department for four-vessel cerebral angiograms from January 2005- January2006. All angiograms done and consented were included.

## 5.3: Study design

It was descriptive cross-sectional study. Patients who presented for four- vessel angiographies during the period of study were included consecutively. The patients clinical summary i.e. age, sex, serial no, x-ray no, radiological diagnosis were obtained from the request/report form.

## 5.4: Sample size determination

The sample size was all the patients that availed themselves for cerebral DSA within the period of one year extending from January 2005 to January 2006. On average one to two patients were examined at KNH / Nairobi hospital per week. The sample size was determined using the following formula by Fischer et al (1998).

$$n = \frac{z^2 p(1-p)}{d^2}$$

Where

n= desired sample size

z= standard normal distribution

p =known prevalence rate for the factor of interest under study

d=level of significance desired

When this formula was applied at d =0.05, z=1.96 and p =6%: Ruberti [42] found a prevalence of 6-8% cerebrovascular diseases in approximately 10, 000 African patients seen by him over 30 year period; the minimum sample was 87. However 88 angiograms were sampled.

## 5.5: Sampling procedure

All cerebral DSAs of consenting patients were studied consecutively for the period extending from January 2004 January 2006. All angiograms for patients referred for this procedure within this stipulated period were studied.

## 5.6: Neuroangiography team

The team included interventional radiologist, principal investigator, radiolographic technologist and assisting nurse. Access to intensive care facilities was readily available for the patient when required.

## 5.7: Equipment

The equipment used had the following specifications. Siemens Siregraph CF System, DSA devise used at 1200mA and 150kVP and angiographic suite with floating catheterisation tabletop.

Femoral catheterisation was used. The needle used was short bevelled, not larger than gauge 17 in adults, 18 in children and 19 in infants. Catheters utilised included 4-7

French diameters, including multipurpose, Simmons Headhunter, and MANI catheters Low osmolar non-ionic contrast medium (Iopamidol 300mg I/ml) was used in all studies because of minimal side effects.

## 5.8: Procedure/ technique

Patient preparation included: - Informed consent, prothrombin time index (PTI) - minimum accepted level was 75%, haemoglobin level of 10g\dl, H.I.V and HbSAg screening to prevent cross infection in a set up where sometimes catheter re-use was inevitable due to financial reasons. Other conditions met by patients included, good hydration to minimize contrasts media side effects, nil per oral for at least 4 hours prior to the procedure and Shaving of the inguinal region to remove hair that may contaminate the puncture site.

## 5.9: Angiographic technique

The patient was placed supine on an Angiographic table. Both femoral arteries are palpated and the easiest was selected for puncture (usually the right side was preferred). Appropriate guide wire and catheter were selected and their compatibility ascertained. Using aseptic technique, local anaesthetic was infiltrated on either side of artery down to the periosteum. A 5mm transverse incision was made over the artery to avoid binding of soft tissues on the catheter.

Correct puncture was made at the apex of arch of femoral artery with the needle directed 45° cephalad to skin surface .The needle was then in direct line with the lumen of the artery. The artery was immobilized by placing the index and middle fingers of the left hand on either side of the artery with the needle held in the right hand.

Commonly both anterior and posterior walls were punctured with a single thrust and pulsatile flow of blood obtained. The guide wire was then advanced up to the descending abdominal aorta, the soft tissue path dilated and catheter introduced over the guide wire.

The catheter was introduced in a rotating fashion holding the catheter as close to the skin as possible. The guide wire was removed as soon as possible followed by aspiration and flushing of catheter with heparinised saline.

Bilateral catheterization of both common carotid arteries (2-3cm below common carotid bifurcation) and catheterisation of one or both vertebral arteries was done. Both common carotid arteries were injected in lateral, oblique and anteroposterior (AP) views. Both lateral and frontal views of vertebrobasilar circulation were obtained. As deemed necessary additional discretionary views were obtained (e g reversed oblique view) during angiography, to elucidate overlapping vessels or further clarification, based on the region of interest. (8 -10mls of Iopamidol 300mgI/ml was used for each carotid and vertebral injection.

At the end of the procedure and withdrawal of the catheter, a short spurt of blood was allowed as manual pressure may trap any clots formed around the catheter tip. Pressure was applied at the puncture side just hard enough to prevent bleeding and preferably still allowing pulsation of the arteries peripherally to be felt (about 10 minutes). Protamine sulphate 1mg for each-100 IU of heparin was available in case of inadvertent bleeding.

## 5.10: Data collection tools

Required information elicited from the radiographs and reports was collected by the use of a structured data sheet. Patients' bio data was obtained from angiograms request forms. Available clinical summary and reports of other radiological investigations done was recorded on data sheet. Angiographic diagnosis was entered and indicated whether right, midline or left sided. The type, anatomical site and multiplicity of the lesion was also be recorded.

## 5.11: Validation of tools

Pre-test of data sheet was done with pre-existing four vessel angiograms at the DDR. The angiograms used for pre-testing were representative of spectrum of cerebrovascular

pathologies. At least two of each of categories were included; normal, aneurysm, AVM, and vascular stenosis

## 5.12: Control of biases

No angiograms done within the study period were excluded. Only patients who were done four-vessel examination within the study period were included. Angiograms were reported by principal investigator in consultation with practicing neurointerventional radiologist. Equivocal reports were further subjected to panel discussion at Clinico-Pathological radiological conference (CPRC)

## 5.13: Data management

Data was collected, cleaned and entered concurrently. Data analyses were done using Software Programme for Social Science research (SPSS version 11.5) and Excel for windows XP. Results were presented in form of frequency distributions and descriptive statistics. The Chi-square test of independence was used to test associations. Z tests for proportional and mean differences were also applied. The level of significance was set at $\alpha$=0.05.

## 6.0: ETHICAL CONSIDERATION

Before commencement of the study, a request was submitted together with a copy of this proposal to the ethical and research committee for both hospitals for approval. Patient's name was not recorded during the study in order to maintain confidentiality. Information acquired was used exclusively for research. For referral purposes, only the patient's hospital number was required. Informed signed consent was obtained from the patient before being included in the study (Annex C and D)

No other examination was done on a patient apart from the one requested by the primary physician – i.e. only patients sent for four-vessel cerebral angiography by attending physician would be considered.

The results from this study were delivered to KNH and Nairobi hospital ethical committees to assist in forming database for future studies and reference and to facilitate possible improvement in patient care.

# 7.0: RESULTS

During the study period, a total of 88 angiograms done at KNH and Nairobi Hospital were examined. Out of these 88 patients, 69 (78.4%) had a prior CT scan, 7 (8.0%) had a MRI and only 4 (4.5%) had both CT and MRI. Sixteen (18.1%) of cases underwent four-vessel studies solely on clinical suspicion.

## 7.1: Characteristics of the sample population

There were 41(46.6%) males and 47 (53.4%) females with a male to female ratio of 1:1.1. The sample population had mean age of 40.3 years with a minimum of 1 year, a maximum of 73 years, a median of 41 years and a standard deviation of 15.45. There was no statistically significant difference (p=0.58) in the mean age of the males (40.7 years) and females (40.0 years). The age and sex distribution of the sample population was as shown in Table 1. Overall, the 41-60 years age group comprised the commonest group (44.3%) referred for the angiographic examination. The 41-60 years age group was also the commonest group for females (53.2%) referred for angiographic examination but the 21-40 age group was the commonest for males (46.3%).

**Table 1: Age and sex distribution of the patients understudy (n=88)**

| | Male | | Female | | Overall | |
|---|---|---|---|---|---|---|
| **Age group** | **n** | **%** | **n** | **%** | **n** | **%** |
| 0-20 | 4 | 9.8 | 7 | 14.9 | 11 | 12.5 |
| 21-40 | 19 | 46.3 | 13 | 27.7 | 32 | 36.4 |
| 41-60 | 14 | 34.1 | 25 | 53.2 | 39 | 44.3 |
| 61-80 | 4 | 9.8 | 2 | 4.3 | 6 | 6.8 |
| **Total** | **41** | **100.0** | **47** | **100.0** | **88** | **100.0** |

## 7.2: Findings at angiographic examination

Angiographic pathology was identified in 77.3% of the study subjects. The commonest angiographic finding was aneurysm which comprised of 36 (40.9%) of the outcomes. The least common finding was AVM comprising of 7 (8.0%) of the findings. There were 20 (22.7%) cases of normal findings. The angiographic findings and sex distribution were as shown in Table2. The aneurysms were significantly more common (p=0.016) in females (51.1%) compared to males (29.3%). However AVMs were significantly more common (p=0.017) in males compared to the females. There were 12 (13.6%) other findings which included masses 9 (10.2%) and dural sinus thrombosis 3 (3.4%).

**Table 2: Sex distribution and the angiographic findings (n=88)**

| Angiographic Diagnosis | Male n (%) | Female n (%) | p-value |
|---|---|---|---|
| Aneurysm | 12 (29.3) | 24 (51.1) | 0.016 |
| AVM | 6 (14.6) | 1 (2.1) | 0.017 |
| Vasospasm | 8 (19.5) | 5 (10.6) | 0.122 |
| Others | 5 (12.2) | 7 (14.9) | 0.360 |
| Normal | 10 (24.4) | 10 (21.3) | 0.360 |
| **Grand Total** | **41 (100.0)** | **47 (100.0)** | |

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| Normal | 10 (24.4) | 10 (21.3) | 0.360 |
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Age distribution and angiographic findings were as shown in Table 3. Aneurysms (61.1%) and vascular stenosis (46.1%) were found to be more common in the age group 41-60 years. However, 57.1 of AVMs were found in the younger age group of 21-40 years. There were less pathological findings in the extremes of ages.

**Table 3: Age distribution and the angiographic findings (n=88)**

| Age group | Aneurysm n (%) | AVM n (%) | Vascular stenosis n (%) | Others n (%) | Normal n (%) |
|---|---|---|---|---|---|
| 0-20 | 3 (8.3) | 2 (28.6) | 1 (7.7) | 3 (25.0) | 2 (10.0) |
| 21-40 | 8 (22.2) | 4 (57.1) | 4 (30.7) | 5 (41.7) | 11 (55.0) |
| 41-60 | 22 (61.1) | 1 (14.3) | 6 (46.1) | 4 (33.3) | 6 (30.0) |
| 61-80 | 3 (8.3) | 0 (0.0) | 2 (15.4) | 0(0.0) | 1 (5.0) |
| **Grand Total** | **36 (100.0)** | **7 (100.0)** | **13 (100.0)** | **12 (100.0)** | **20 (100.0)** |

## 7.3: Other imaging studies versus angiographic findings

CT, comprising 69 (78.4%) of the cases, was the main preliminary imaging study done prior to referral for the four–vessel studies. Only 7 (8.0%) of the cases had MRI done. Sixty one (84.7%) of the cases with preliminary imaging studies and 7 (43.8%) of those referred solely on clinical suspicion were angiographically positive. Cases referred for angiographic examination with preliminary imaging studies were 7.1 times more likely to reveal angiographic pathology compared to those without (Chi-square = 12.5, p=0.0004).

Angiographic findings compared with the prior CT findings were as shown in Table 4. Most of aneurysms and vascular stenosis found at angiography (58.3% and 38.6% respectively) were reported as SAH at CT. Fifty seven percent of AVMs at angiography were reported as ICH at CT.

**Table 4: Angiographic findings compared with prior CT findings (n=88)**

| | Angiographic Findings | | | | |
|---|---|---|---|---|---|
| **Prior CT findings** | **Aneurysm n (%)** | **AVM n (%)** | **vascular stenosis n (%)** | **Others n (%)** | **Normal n (%)** |
| Aneurysm | 2 (5.6) | 0 (0.0) | 1 (7.7) | 1 (8.3) | 1 (5.0) |
| AVM | 1 (2.8) | 1 (14.3) | 0 (0.0) | 2 (16.7) | 1 (5.0) |
| ICH | 2 (5.6) | 4 (57.1) | 2 (15.4) | 2 (16.7) | 3 (15) |
| ICH+IVH | 1 (2.8) | 0 (0.0) | 1 (7.7) | 0 (0.0) | 1 (5.0) |
| SAH | 21 (58.3) | 0 (0.0) | 5 (38.6) | 1 (8.3) | 3 (15) |
| SAH+ICH | 0 (0.0) | 0 (0.0) | 0 (0.0) | 0 (0.0) | 1 (5.0) |
| SAH+IVH | 1 (2.8) | 0 (0.0) | 1 (7.7) | 0 (0.0) | 0 (0.0) |
| Others | 1 (2.8) | 0 (0.0) | 3 (23.1) | 5 (41.7) | 1 (5.0) |
| CT not done | 7 (19.4) | 2 (28.6) | 0 (0.0) | 1 (8.3) | 9 (45.0) |
| **Grand Total** | **36 (100.0)** | **7 (100.0)** | **13 (100.0)** | **12 (100.0)** | **20 (100.0)** |

Fifty (50) percent of the angiographically negative cases had a demonstrable pathology at CT of which 15% were reported as SAH at and another 15% as ICH; however, 45% had no preliminary CT done. The patients who presented for four-vessel studies with CT already done were 4.7 times more likely to reveal pathology at angiography than those without (Chi square 8.38, p= 0.004). Preliminary imaging modalities of the angiographic negative cases were as shown in Figure 1.

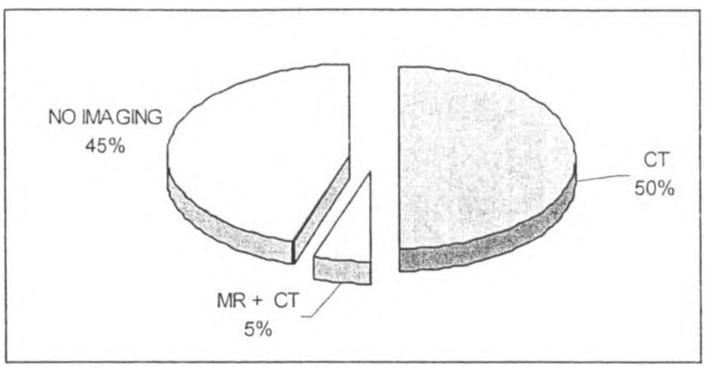


**Figure 1: Preliminary imaging studies of angiographic negative cases (n = 20)**

## 7.4: Anatomical distribution of aneurysms

The anatomical distribution of aneurysms was as shown in Figure 1. All the 36 aneurysm cases were intracranial. Out of these, 35 were saccular and only 1 was fusiform. Overall, the commonest anatomical site for aneurysm was at the junction of ICA and PCoA (30.6%). Of all the aneurysms, 16.7% were multiple; involving ICA-PCOA, MCA bifurcation and AICA. ICA-PCoA and multiple aneurysms were more common in females (33.3% and 20.8% respectively) compared to males (25.0% and 8.3% respectively). For multiple aneurysms the male to female ratio was 1:5.

Other uncommon sites of the aneurysms were ACA bifurcation, PCA-PCoA junction and ICA-SHA junction. Anterior (carotid) circulation aneurysms arising within the circle of Willis accounted for 95.2 % of all the aneurysms.

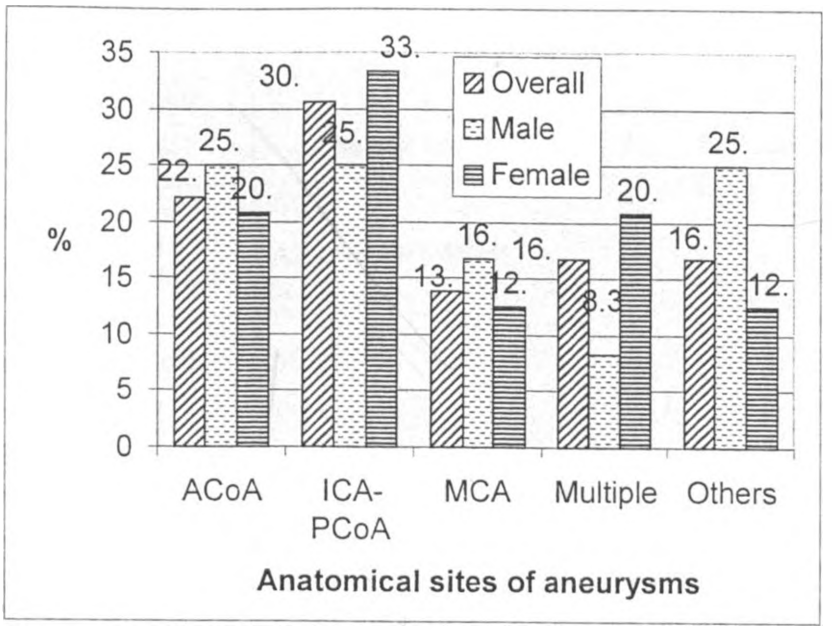


**Figure 1: Anatomical distribution of aneurysm (n=88)**

## 7.5: Anatomical distribution of vascular stenosis

The anatomical distribution of vascular stenosis was as shown in Table 5. The commonest anatomical site for stenosis was ICA (46.2%). Global involvement of cerebral arteries was found in 2 (15.4%) of the vascular stenoses cases. Of all vascular stenosis, 61.5% were non-atheromatous (vasospasms).

## 7.6: Anatomical distribution of AVMs

The AVMs were found only in the extracranial and supratentorial locations.

Fifty seven percent of the AVMs were found in the supratentorial location (Table 5). All the supratentorial AVMs were pial.

**Table 5: Anatomical sites of vascular stenosis and AVM**

| Angiographic Diagnosis | Site of lesion | N | Percent |
|---|---|---|---|
| Vascular stenosis | ACA | 2 | 15.4 |
| | All cerebral arteries | 2 | 15.4 |
| | ICA | 6 | 46.2 |
| | MCA | 1 | 7.7 |
| | PCA | 1 | 7.7 |
| | Vertebral | 1 | 7.7 |
| | **Total** | **113** | **100.0** |
| AVM | Extracranial | 3 | 42.9 |
| | Supratentorial | 4 | 57.1 |
| | **Total** | 7 | **100.0** |
| Others | | 12 | - |

# 8.0: DISCUSSION

The overall objective of this study was to determine the pattern of cerebrovascular pathology as shown by 4 vessel cerebral angiography. The study showed demonstrable angiographic pathology in 77.3% of the patients examined.

## 8.1: Socio-demographic Characteristics

The commonest age group examined was 41-60 years which comprised 44.3% of the sample population. The study also showed aneurysms and vascular stenosis being the common cerebrovascular pathologies in these age groups. This finding could be due to the age at which these pathologies become symptomatic. Osborn [8] stated that aneurysm typically become symptomatic in people aged 40-60 years. Ruberti [42] also showed that intracranial aneurysms typically become symptomatic at the age group 40-60 years. Graves et al [19] found that AVM's commonly present between 20 and 40 years. The study found 57.1% of AVM's in the age group 21-40 years.

### 8.2: Angiographic findings

Aneurysms were demonstrated in the majority (40.9%) and AVMs in the minority (8.0%) of the cases. However 22.7% of the cases were angiographically negative. This is likely due to the propensity of aneurysms to rupture causing SAH and hence becoming symptomatic. Angiographically negative cases were mainly due to spasm of the bleeding source, which could sometimes become angiographically occult and repeat angiography is advisable. These findings were generally comparable with what Sutton [38] documented, only that in his case, aneurysms were found in 55%, angiomas were in a significant minority (10%) and 15-20% were negative of the four-vessel studies.

There was male and female preponderance of AVMs and aneurysms respectively (Table 2). Ruberti [42] documented that aneurysms of internal carotid artery were more common in females while those arising from the ACA and ACoA were more common in males. Hartmann *et al* [43] demonstrated equal sex incidence of AVM's.

Un-ruptured aneurysms are usually asymptomatic. On rupture they are associated with significant morbidity and mortality with SAH being the commonest presentation. Vasospasm is the leading cause of disability and death from aneurysm rupture [14, 15]. The study showed that most of aneurysms and vascular stenosis found at angiography (58.3% and 38.6% respectively) were reported as SAH at CT.

Vascular stenosis/spasm was demonstrated in 14.7% of cases. Ruberti [42] in similar study showed vasospasm in 38.3%. The lower proportion in comparison to Ruberti's is likely due to increased number of patients currently undergoing other preliminary imaging examinations thus early detection of SAH that would have otherwise presented with vasospasms later.

## 8.3: Anatomical distribution

Osborn [13] documented that 85% of all intracranial aneurysms arise from anterior (carotid) circulation and 15% from the posterior (vertebro-basilar) circulation. This study however though comparable, showed a slightly higher figure of 95.2% for anterior (carotid) circulation aneurysms. However, Ruberti [42] and Kavous *et al* [41] showed similar findings (95.9% and 93% respectively).

Osborn [13] showed that the common aneurysm locations are anterior communicating artery (30 -35%), internal carotid artery (ICA) at the posterior communicating artery origin (30 -35%) and the MCA bifurcation (20%). In this study the common aneurysm locations were ICA- PCoA (30.6%) and ACoA (22.2%) aneurysms and least diagnosed was MCA (13.0%). These locations are in the circle of Willis and this where a high haemodynamic flow is found causing significant injury the vessels more so at the bifurcation causing aneurysmal dilatation. Ruberti [40] showed that ICA-PCOA, ACoA and MCA aneurysms constituted 43.1 %, 30.8% and 16.5% respectively in a study of 146 Kenyans. This compared well with this study except for the proportion of ACoA and MCA aneurysms. In a similar study Kavous *et al* [41] showed that anterior circulation aneurysms constituted 93% with ICA-PCOA (32.6%), ACoA (34.9%) and MCA (25.8%).These findings were comparable except for MCA aneurysms which had a lower proportion in this study.

Intracranial aneurysms are multiple in 15 -20% of all cases [39]. In another series, Sutton [38] established multiple aneurysms in between 5 to 15% of all cases. This study found comparable results with multiple aneurysms accounting to 16.7%. Kavous *et al* [41] also found that aneurysms were multiple in 10% of aneurysm cases. A strong female to male ratio of 5:1 is observed in multiple aneurysms [39]. This was comparable to the findings of this study.

Intracranial AVMs are supratentorial in 90% and infratentorial 10% of the cases [7, 8, 19]. All intracranial AVMs in this study were supratentorial and pial. No associated aneurysm was demonstrated with AVMs in this study. The ratio of aneurysms to AVMs in this study was 5:1. However, Ruberti [40] noted the ratio of intracranial aneurysm to AVM as 3:1 in Kenyan African population and 10:1 in the western series. He explained this with the fact that the AVMs manifest clinically early in life whilst the intracranial aneurysms are clinically more frequent at the age of between 40 and 60 years. He further noted that the Africans had a shorter span of life and therefore most did not reach the symptomatic age for aneurysms. Another explanation he gave was that the haemorrhages due to AVM were less catastrophic and give more often focal signs than subarachnoid haemorrhages which tended to be confused with meningitis, cerebral malaria and other diseases at general practitioner level. The explanations for the bigger ratio in this study than Ruberti's could be due to the more diagnosis of asymptomatic aneurysms by the newer imaging methods and the improvement of span of life for Kenyans compared to what it was at the time of his study 30 years ago.

# 9.0: CONCLUSIONS

Based on the findings of this study and on objectives that were set, the following conclusions were made:

The pattern of cerebrovascular disease seen in KNH and Nairobi Hospital was comparable to those in other setups. Cerebrovascular pathologies were commonest in the age group 41-60 years. Aneurysms and vascular stenosis /spasms were also the common cerebrovascular pathologies in this age group. AVMs were common in the younger age group of 21-40 years.

Aneurysms were the commonest cerebrovascular pathologies and they were significantly more ($p=0.007$) in females compared to males. Multiple aneurysms occurred in 17.9% of all aneurysm cases, with male to female ratio of 1:5. Majority of aneurysms (95.2%) occurred in the anterior (carotid) circulation; the common locations being ICA-PCoA (30.6 %), ACoA (22.2%) aneurysms and MCA bifurcation (13.0%).

The least common cerebrovascular pathology was AVM .All AVMs were supratentorial and pial. Slightly more than a fifth of all cases were angiographically negative and a half of them had been reported to have cerebrovascular disease at CT.

# 10.0: RECOMMENDATIONS

This study recommends the following:

1. With many Kenyans now reaching ages that cerebrovascular disease becomes symptomatic and in lifestyles that make cerebrovascular disease more common, there is need to increase use of non invasive and cheaper imaging techniques like CTA and MRA for screening the at risk populations.

2. There is need for follow-up of angiographically negative cases and possible repeat of angiography especially of those that had shown cerebrovascular disease at CT or MRI.

3. A larger series study need to be undertaken in order to establish the exact prevalence of cerebrovascular disease in the population and pick the rare cerebrovascular pathologies that were not encountered in this study.

4. There is need for a comparative study on cost effectiveness of four-vessel angiography with non-invasive angiographic techniques such as CTA and MRA.

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# 12.0: ANNEXES

**Annex A: Data collection form**

1. PATIENT NO:_____

2. AGE: _____ YEARS

3. SEX

M ☐

F ☐

4. CLINICAL SUMMARY

_____

_____

_____

5. CT DIAGNOSIS

_____

_____

(6) MRI DIAGNOSIS

_____

_____

| 7. ANGIOGRAPHIC DIAGNOSIS | RT | LT |
|---|---|---|
| AVM | ☐ | ☐ |
| ANEURYSM | ☐ | ☐ |
| ATHEROSCLEROSIS | ☐ | ☐ |
| NON-ATHEROMATOUS VASCULAR STENOSIS | ☐ | ☐ |

NORMAL ☐

OTHER ☐ ☐

(Specify)

8. SIZE OF THE LESION

_____ mm

9.TYPE OF ANEURYSM

SACCULAR ☐

FUSIFORM ☐

DISSECTING ☐

OTHERS ☐

10. LOCATION OF ANEURYSM

| CERVICAL | RT | LT |
|---|---|---|
| VERTEBRAL | ☐ | ☐ |
| ICA | ☐ | ☐ |
| OTHER (SPECIFY) | ☐ | ☐ |

| INTRACRANIAL | RT | LT |
|---|---|---|
| ICA | ☐ | ☐ |
| ACOA | ☐ | ☐ |

| | | |
|---|---|---|
| ACA | ☐ | ☐ |
| MCA | ☐ | ☐ |
| PCA | ☐ | ☐ |
| BASILAR ARTERY | ☐ | |
| VERTEBRAL ARTERY | ☐ | ☐ |
| OTHER (specify) | ☐ | ☐ |

11. TYPE/SITE OF INTRACRANIAL VASCULAR MALFORMATION

| | RT | LT |
|---|---|---|
| AVM | ☐ | ☐ |
| CAPILLARY TELAGIECTASIA | ☐ | ☐ |
| CARVENOUS ANGIOMAS | ☐ | ☐ |
| VENOUS MALFORMATION | ☐ | ☐ |

12. LOCATION OF AVM

| | RT | LT |
|---|---|---|
| SURATENTORIAL | ☐ | ☐ |
| INFRATENTORIAL | ☐ | ☐ |

13. TYPE OF AVM

| | RT | LT |
|---|---|---|
| PIAL | ☐ | ☐ |

| | | |
|---|---|---|
| DURAL | ☐ | ☐ |
| MIXED | ☐ | ☐ |

14. TYPE OF VASCULAR STENOSIS

| | |
|---|---|
| ATHEROMATOUS | ☐ |
| NON ATHEROMATOUS | ☐ |

| 15. LOCATION OF THE STENOSIS | RT | LT |
|---|---|---|
| CERVICAL ICA | ☐ | ☐ |
| CERVICAL VA | ☐ | ☐ |
| INTRACRANIAL ICA | ☐ | ☐ |
| INTRACRANIAL VA | ☐ | ☐ |
| BASILAR ARTERY | | ☐ |

**Annex B: Budget**

| Items | Units | Unit price (KShs.) | TOTAL (KShs.) |
|---|---|---|---|
| **Preparation of the proposal** | | | |
| ➢ Reference Materials | | | 12,000 |
| ➢ Stationary | | | 6,000 |
| ➢ Secretarial services | | | 5,000 |
| ➢ Binding | | | 3,000 |
| ➢ Ethical certification fees | | | 1,500 |
| **Transport** | | | |
| ➢ KNH – Nairobi Hospital - KNH | 120 | 300 | 36,000 |
| | | | - |
| | | | - |
| **Materials** | | | |
| ➢ Data sheets | 200 | 50 | 10,000 |
| ➢ Radiographs | 200 | 400 | 80,000 |
| ➢ | | | |
| **Data analysis** | | | 10,000 |
| **Report** | | | |
| ➢ Writing | | | 6,000 |
| ➢ Submission process | | | 5,000 |
| ➢ Corrections and binding | | | 10,000 |
| **Sub-Total** | | | **184,500** |
| **Contingencies** at 10 % of the Total | | | 18,450 |
| **GRAND TOTAL** | | | **202,950** |

**Annex C: Consent form (Swahili)**

**KIBALI CHA KUHUSIKA KATIKA UTAFITI**

Jina langu ni daktari David Chepsiror kutoka chuo cha udaktari,Chuo kikuu cha Nairobi.Ninafanya utafiti kuhusu magonjwa ya misisi ya ubongo kwa kutumia mbinu ya picha ya X-ray utakayofanyiwa(DSA).Hii inahusu utumisi wa sindano na dawa katika misisi ya mguu.DSA ina uweso wa kukudhuru kwa kusababisha uchungu, madhara kwa ubongo, misisi hata na figo. Lakini mitambo wa kisasa ina sababisha madhara chache sana chini ya asilimia moja.

Habari utakayotoa au ile itakayopatikana kukuhusu,itakuwa siri ya kutumika tukatika utafiti.Jina lako halitajumulishwa bali ile nambari ya matibabu tu ndilo litakalotumika Ripoti ya yale yatakayopatikana kwako na lolotelile litakalokusaidia litatumiwa kwa daktari wako.

Utafiti huu utasaidia katika kurekebisha na kuendelesha matibabu ya magonjwa ya sehemu ya misisi ya ubongo
Tafadhali kumbuka unashiriki kwa hiari yako.
Kama unakubali kushiriki;tafadhali weka sahihi yako hapa chini

Sahihi:

Tarehe:

Nambari:

Ninathibitisha kuwa muhusika ameelewa na kukubali kushiriki kwa utafiti huu.

Daktari Chepsiror David:

Sahihi:

Tarehe:

**Annex D: Consent form (English)**

## PARTICIPATION CONSENT FORM

My name is Dr Chepsiror .k. D, a master of Medicine student at Department of Diagnostic Radiology, University of Nairobi. I am doing a study on Cerebrovascular diseases seen at four-vessel cerebral angiography, and would wish to recruit you to participate. The information you will give or /and examination findings will be handled with utmost confidentiality.

This procedure involves intravenous placement of small calibre catheters, and reduced doses of low osmolar iodinated contrast medium. Potential risks include pain local swelling, aneurysm formation cerebrovascular accidents, arterial dissections, contrast medium reaction and renal insufficiency/failure. However modern cerebral DSA remains safe with overall complication rate of less than one percent.

Your name will not be included, except the serial number. The results of the study will be used to improve the management of cerebrovascular diseases. Please note that you are not obliged to participate and you have a right to decline or withdraw from the study

Thank you for your cooperation

If you accept to participate, please sign below

Signature:
Date:
Number:

I certify that the patient has understood and consented participation in the study.
Dr Chepsiror K D
Signature:
Date:

**Annex D: Consent form (English)**

## PARTICIPATION CONSENT FORM

My name is Dr Chepsiror .k. D, a master of Medicine student at Department of Diagnostic Radiology, University of Nairobi. I am doing a study on Cerebrovascular diseases seen at four-vessel cerebral angiography, and would wish to recruit you to participate. The information you will give or /and examination findings will be handled with utmost confidentiality.

This procedure involves intravenous placement of small calibre catheters, and reduced doses of low osmolar iodinated contrast medium. Potential risks include pain local swelling, aneurysm formation cerebrovascular accidents, arterial dissections, contrast medium reaction and renal insufficiency/failure. However modern cerebral DSA remains safe with overall complication rate of less than one percent.

Your name will not be included, except the serial number. The results of the study will be used to improve the management of cerebrovascular diseases. Please note that you are not obliged to participate and you have a right to decline or withdraw from the study

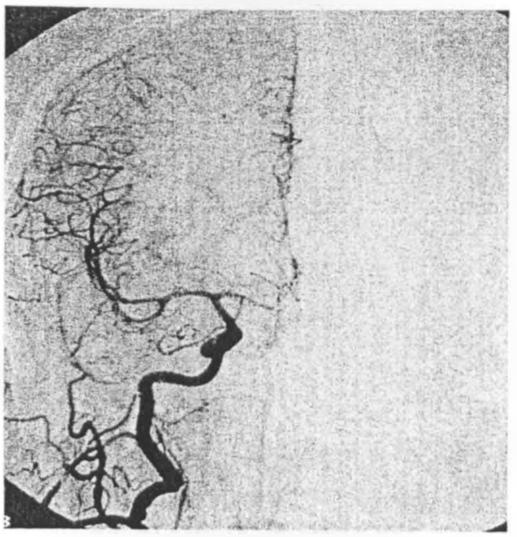
Thank you for your cooperation

If you accept to participate, please sign below

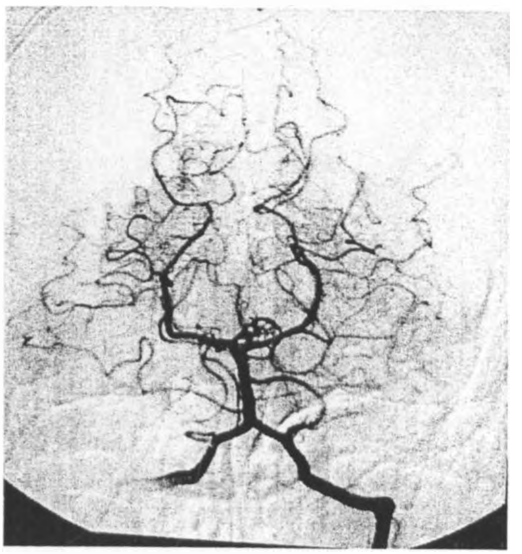
Signature:
Date:
Number:

I certify that the patient has understood and consented participation in the study.
Dr Chepsiror K D
Signature:
Date:

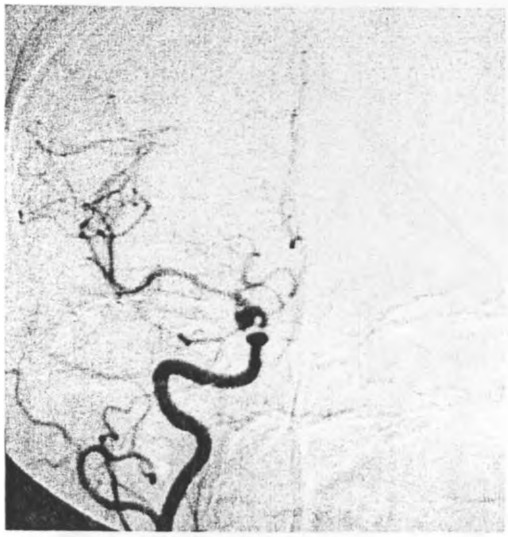
## Annex E. Angiographs of selected cases



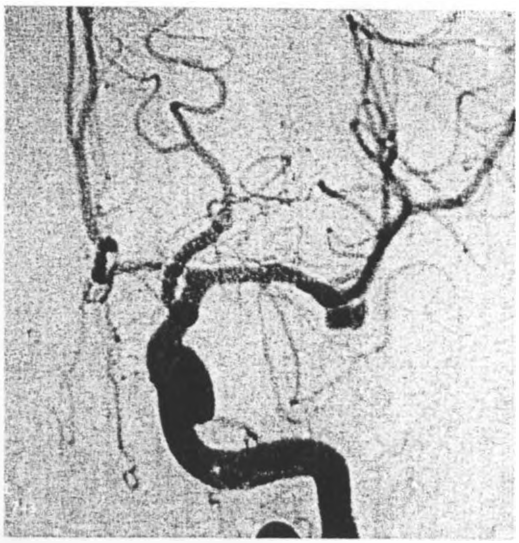
Picture 1: Normal Rt carotid angiogram –AP view



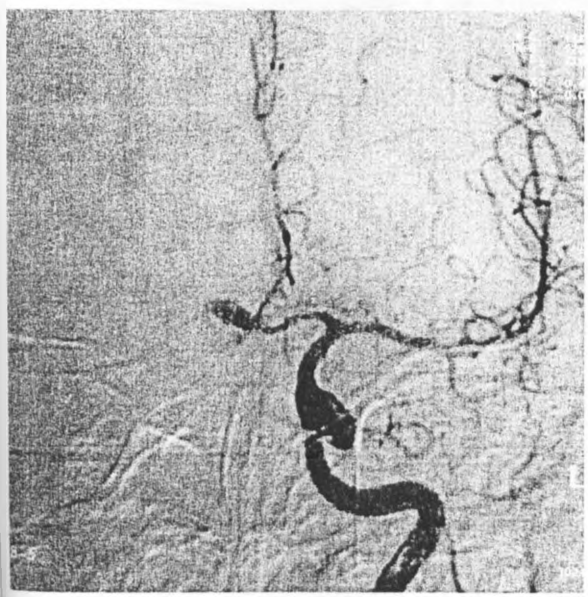
Picture 2: Normal vertebral angiogram –AP view



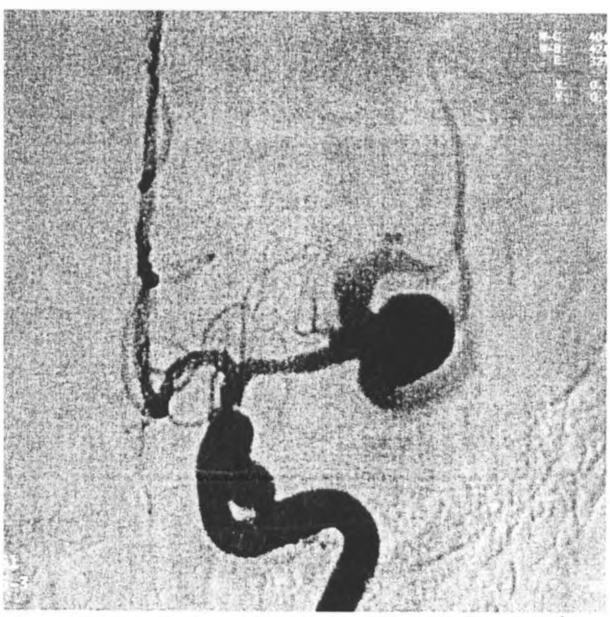
Picture 3: Oblique Rt carotid angiogram showing PCoA aneurysm in 36 years old female



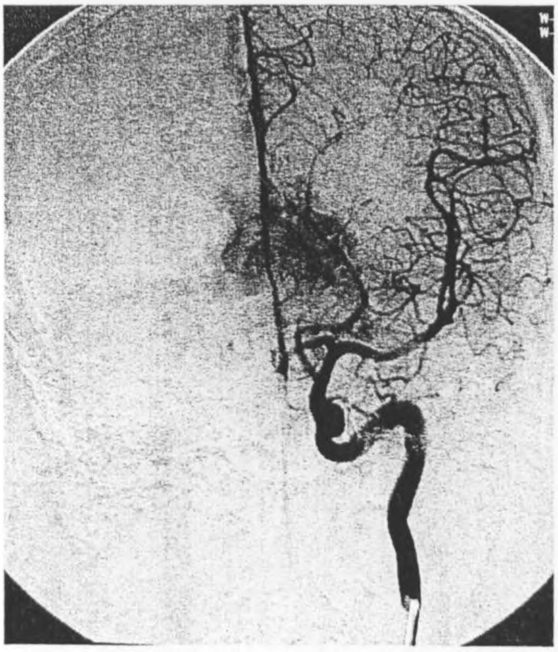
Picture 4: Left MCA bifurcation Aneurysm in 45 year old female



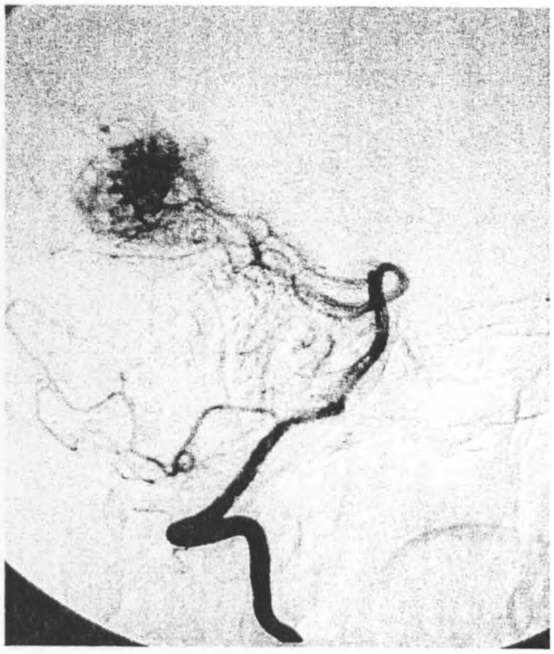
Picture 5: Left ACoA Aneurysm in 55 years old male



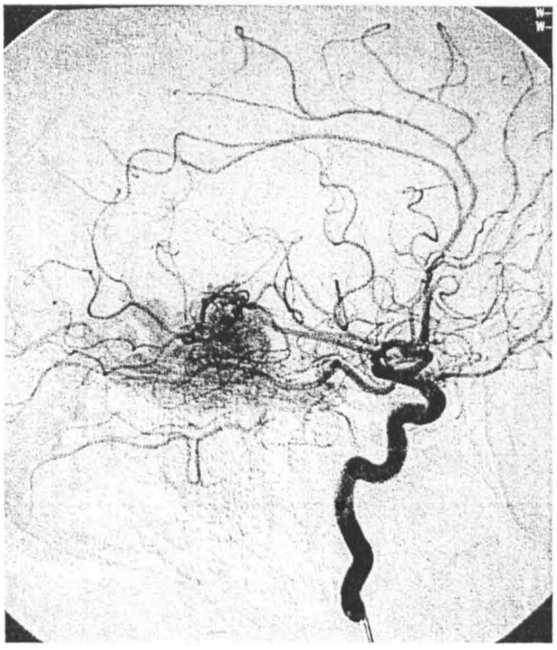
Picture 6: Left MCA bifurcation Aneurysm in 12 year old female



Picture 7



Picture 8



Picture 9

**Supratentorial Pial AVM supplied by MCA, PCA and ACA in 44 years old male (pictures 7-9)**