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CT Findings of Ischemic Stroke in Eswatini

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Abstract: The incidence of stroke is increasing in sub-Saharan Africa, including in Southern Africa, as the region undergoes socioeconomic and epidemiological changes. We did a retrospective analysis of the findings of the non-contrast CT in the patients with the diagnosis of ischemic stroke to investigate the incidence and CT findings of ischemic stroke in Eswatini. A total of 218 patients were diagnosed with stroke by positive findings with non-contrast CT in the Department of Radiology, Mbabane Government Hospital in Eswatini. Excluding 18 patients with hemorrhagic stroke, a total of 200 patients with ischemic stroke were analyzed for the clinical and CT findings. The clinical information, which was available in 158 patients, was analyzed. CT findings were analyzed in 200 patients for the location of the ischemic lesions, extent, Oxford Community Stroke Project (OCSP) classification, vascular territory, extent, and associated findings. In patients associated with hemorrhagic transformation, we analyzed the clinical and CT findings including the vascular territory of the lesion and associated findings. As for the OCSP classification of the location of ischemic infarct, partial anterior circulation stroke is the most frequent type (59.5%), followed by lacunar syndrome (21%), posterior circulation (8.5%), and combined type (7.5%). Total anterior circulation stroke was 7 patients (3.5%). The assumed vascular territory of the ischemic lesion was the middle cerebral artery (MCA) territory as the most common vascular territory (41%), followed by deep areas, including basal ganglia, thalamus, midbrain, and pons (19.5%). Associated findings were brain atrophy in 83 patients (41.5%), followed by mass effect, in 21 patients (10.5%), including subfalcine herniation. The vascular territory of the lesion of ischemic infarct with hemorrhagic transformation in 12 patients (6%) was MCA in 9 patients (75%). In the 200 patients with ischemic infarct in the year 2022 in Eswatini, the frequent location of ischemic infarct was partial anterior circulation stroke (59.5%), followed by lacunar syndrome (21%). The most frequent vascular territory of the ischemic lesion was the middle cerebral artery (MCA) territory (41%), followed by deep areas, including basal ganglia, thalamus, midbrain, and pons (19.5%). Hemorrhagic transformation was found in 12 patients (6%), and most frequently in MCA territory. This study may be a helpful guide for further comprehensive study with a larger cohort of the clinical, imaging, and outcomes of stroke in southern Africa including Eswatini.

Keywords: ischemic stroke; infarct; vascular territory; hemorrhagic transformation; non-contrast CT; stroke in Africa; ischemic stroke in Eswatini

1. Introduction

Stroke or cerebrovascular accident (CVA) is an acute central nervous system (CNS) injury and one of the second leading causes of mortality globally and is the third leading cause of adult physical disability [1, 2]. Most deaths resulting from stroke occur in low- and middle-income countries. The incidence of stroke is increasing in sub-Saharan Africa, including in South Africa, as the region undergoes socioeconomic and epidemiological changes resulting in an increased burden of noncommunicable diseases and an aging population [3, 4].

Neuroimaging in stroke diagnosis, especially in acute ischemic stroke patients, plays an important role to differentiate other causes of stroke i.e., stroke-mimic situations such as tumors, seizures, and other metabolic

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or nervous disturbances. CT imaging may show early detection of hemorrhagic stroke, distinguish ischemic infarction, and decide on treatment planning using intravenous thrombolysis or not [1].

We did a retrospective analysis of the findings of the non-contrast CT in the patients with the diagnosis of ischemic stroke in the Department of Radiology, Eswatini in the year 2022 to investigate the incidence and CT findings of ischemic stroke in Eswatini.

2. Materials and Methods

2.1. subjects

In the year 2022, from January 1 to December 31, a total of 2255 patients have undertaken CT examinations. A total of 218 patients were diagnosed with stroke by positive findings with non-contrast CT. Those patients were divided into two groups, ischemic stroke, and hemorrhagic stroke. Excluding 18 patients with hemorrhagic stroke, a total of 200 patients with ischemic stroke were analyzed for the clinical and CT findings.

2.2. methods

Clinical findings were analyzed for age and sex distribution. The clinical information, which was written in the CT request form, and available in 158 patients, was analyzed. CT findings were analyzed for the location of the ischemic lesions, extent, Oxford Community Stroke Project (OCSP) classification, vascular territory, extent, and associated findings. In patients of ischemic infarct with hemorrhagic transformation, we analyzed the age and sex distribution and the CT findings including the vascular territory of the lesion and associated findings.

3. Results

A total of 218 patients were diagnosed with stroke using non-contrast CT among the 2255 patients of CT examinations in the Department of Radiology, Mbabane Government Hospital, Eswatini in the year 2022. Among the 218 patients with stroke, 200 patients (92%) were ischemic stroke, and 18 patients (8%) were hemorrhagic stroke in the diagnosis with non-contrast CT.

Of the 200 patients with ischemic stroke, the male was 87 patients (43.5%), and the female was 113 patients (56.5%). The age distribution was as follows [Table 1]. Below 10 years in 3 patients, twenties (20-29 years old) in 4 patients, thirties in 17 patients, forties in 21 patients, fifties in 27 patients, sixties in 52 patients, seventies in 51 patients, eighties in 20 patients, and over ninety in 5 patients. Of the 200 patients, findings of hemorrhagic transformation are noted in 11 patients (5.5%).

Table 1. Age distribution of the ischemic stroke and hemorrhagic transformation (n=200)

Age	Ischemic	HTF
<10	3	
20-29	4	
30-39	17	4 (23.5%)
40-49	21	2 (9.5%)
50-59	27	
60-69	52	2 (3.8%)
70-79	51	3 (5.9%)
80-89	20	1 (5.0%)
90<	5	
	200	12 (6%)

HTF: Hemorrhagic transformation

The clinical information was available in 158 patients of the 200 patients with ischemic stroke and summarized in Table 2. The hemiparesis of the limbs is the most common finding (51%), followed by hemiplegia (23%), hypertension (39%), retroviral disease (13%), aphasia (15%), and slurred speech (13%).

Table 2. Clinical information of 158 patients (of the 200 patients) of ischemic stroke.

Information	N=158
Hemiparesis (weakness)	80 (51%)
Hemiplegia	36 (23%)
Hypertension	61 (39%)
Retroviral disease	20 (13%)
Aphasia	23 (15%)
Slurred speech	21 (13%)
Suspicion of CVA (no details)	38
Diabetes	28
Seizure	14
Facial palsy	13
Heart Disease (RHD, VHD2, IHD, CHF)	5
Post-partum	2
End-stage renal disease	4

RHD/VHD/IHD: Rheumatic/Valvular/Ischemic heart disease, CHF: Congestive heart failure

The CT findings for the basis of diagnosis of ischemic stroke were a localized or extensive low attenuation of the brain parenchyma, different from the normal texture of the gray and white matter of the brain in all cases. Other findings were blurring of the distinction of the gray, and white matter, effacement of the normal pattern of the gyri and sulci, hyperdense middle cerebral artery sign, and insular ribbon sign.

The location of the lesions with ischemic infarct was multiple in 62 patients and single in 138 patients. The locations were on both sides of the brain in 52 patients, the left side in 67 patients, the right side in 79 patients, and in the middle in 2 patients. The details of the locations of the ischemic lesions are summarised in Table 3.

Table 3. The locations of the lesions of ischemic infarct in 200 patients.

m-B 53	Symmetric (12): B-BG (4), B-TBG(2), B-Cbl(1), B-F(3), B-TPOBG (2), Asymmetric (41): m-Both (41)
L (s) 62	L-BG(11), L-BGI (3), L-F (6), L-P (5), L-Cbl (2), L-FP(ACA) (2), L-FBG, FPBG(4), L-FT, FTP, BG,I, FTPO(MCA) (10), L-H(int carotid a) (4), L-I (2), L-PI. PO (3), L-T,TI,TP,TPI, TBG (9), Lt Mid-B (1)
	Vascular territories: L-BGI(16), L-MCA(37), L-ACA(2), L-PCA(3), L-ICA(4)
L (m) 4	m-L (4)
R (s) 75	R-BG(13), R-BGI(2), R-F(9), R-I(2), R-O(3), R-Cbl (4), R-FBG(3), R-FP(6), R-FPIBG(3), R-FP(ACA)(2), R-FPO, FTP, FTP, FTPBGI(MCA) (16), R-MidB(1), R-P, PO(3), R-Pons, MidB(2), R-T, TFP, TP(4), R-TPBI(2)
	Vascular territories: R-BGI(17), R-MCA(45), R-ACA(2), R-PCA(10), R-ICA(1)
R(m) 4	m-R (4)
Mid (s) 2	Mid-B(2)

m: multiple, B: both sides, BG: basal ganglia, F/T/P/O/I: frontal/temporal/parietal/occipital/insular, R/L: right/left, ACA/MCA/PCA/ICA: anterior, middle, posterior, internal cerebral artery territories.

The distribution of the lesions according to the OCSP classification is summarised in Table 4. Partial anterior circulation stroke is the most frequent type (59.5%), followed by lacunar syndrome (21%), posterior circulation (8.5%), and combined type (7.5%). Total anterior circulation stroke was 7 patients (3.5%).

Table 4. OCSP classification and assumed vascular territories of the ischemic stroke (n=200)

OCSP classification	No.	Vascular territory
Partial anterior circulation	86 (43%)	MCA 82 (41%), ACA 4 (2%)
Total anterior circulation	5 (2.5%)	ICA 5
Lacunar syndrome/small vessel	39 (19.5%)	BG, thalamus, midbrain, pons
Posterior circulation	14 (7%)	PCA 14
Combined anterior and posterior	8 (4%)	Cardiac or aortic

Multiple, both sides/cardioembolic	48 (24%)	Cardioembolic

Vascular territories were assumed for the 200 patients with ischemic stroke by the CT findings. The inclusion criteria of patients for analysis of vascular territories are the lesion of the unilateral vascular territory of a single area or multiple areas of the same vascular territory. The assumed vascular territory of the ischemic lesion is described in Table 4. The middle cerebral artery (MCA) territory was the most common vascular territory (41%) [Fig. 1], followed by deep areas, including basal ganglia [Fig. 2], thalamus, midbrain [Fig. 3], and pons (19.5%), posterior cerebral artery territory (PCA) [Fig. 4] (7%), anterior cerebral artery territory (ACA) [Fig. 5] (4%), and internal carotid artery territory (ICA) (2.5%).

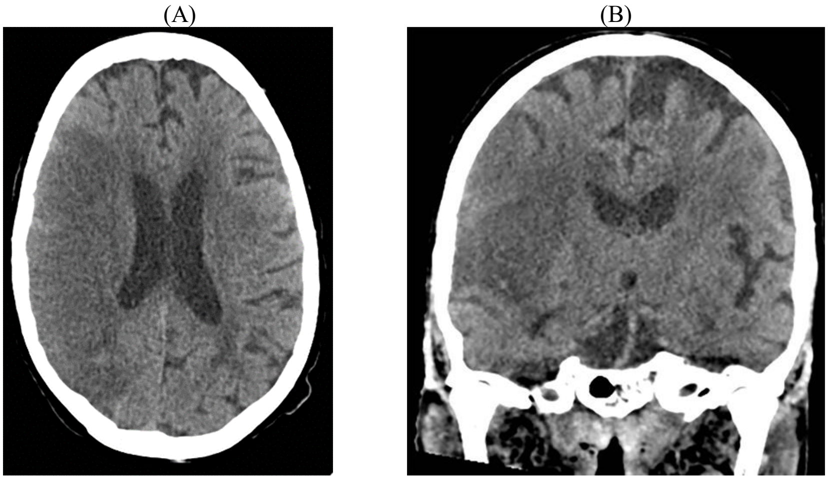

Fig. 1. F/75. (A) Transaxial view, (B) Coronary view. The chief complaints of this hypertensive patient were headache and dizziness. Non-contrast brain CT reveals an acute ischemic infarct of the right frontal, temporal, and parietal lobes, equivalent to the right middle cerebral artery territory. The right lateral ventricle is compressed. A dense MCA sign is noted due to the artery thrombotic occlusion at the M2 portion.

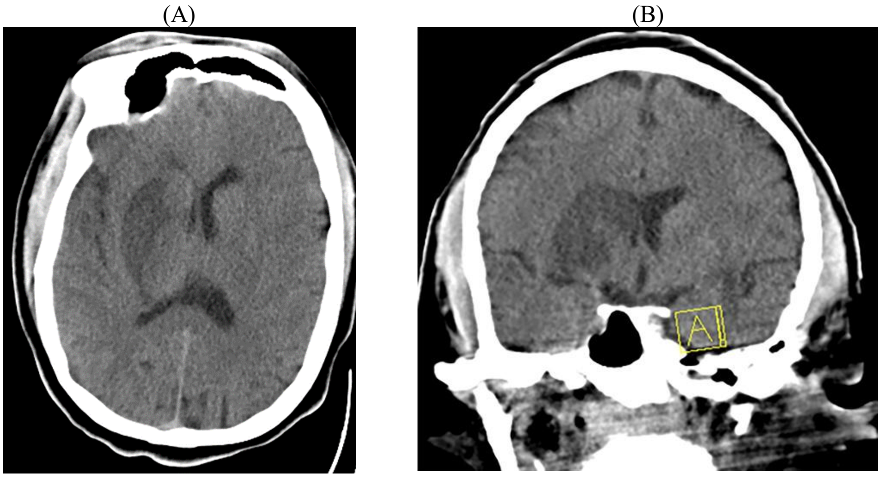

Fig. 2. F/50. (A) Transaxial view, (B) Coronal view. This patient complained of a sudden onset of the Left hemiparesis. Non-contrast brain CT shows an ischemic brain infarct of the lentiform nucleus of the right basal ganglia.

(A) (B)

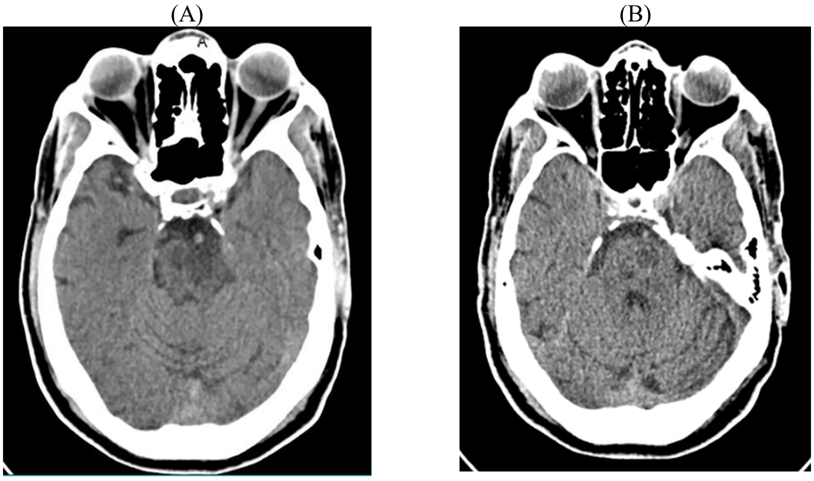

Fig. 3. M/74. (A) Midbrain level, (B) Pons level. This patient complained of a sudden attack of the right sided weakness. Non-contrast CT of the transaxial view reveals ischemic brain infarction of the left midbrain and pons junction level.

(A) (B)

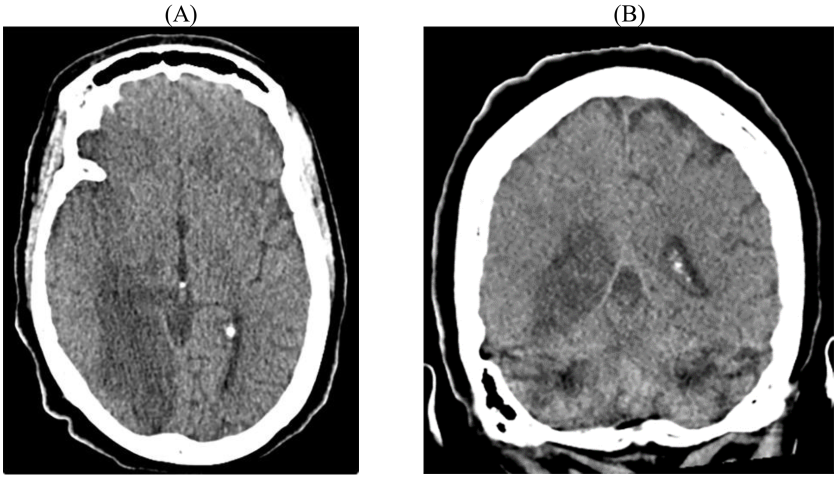

Fig. 4. F/ 63. (A) Transaxial view, (B) Coronary view. This patient complained of a sudden onset of the left hemiplegia. Non contrast brain CT reveals an acute ischemic infarct at the right occipital and medial temporal lobe. Thromboembolism of the territory of the right PCA is the most probable cause.

(A) (B)

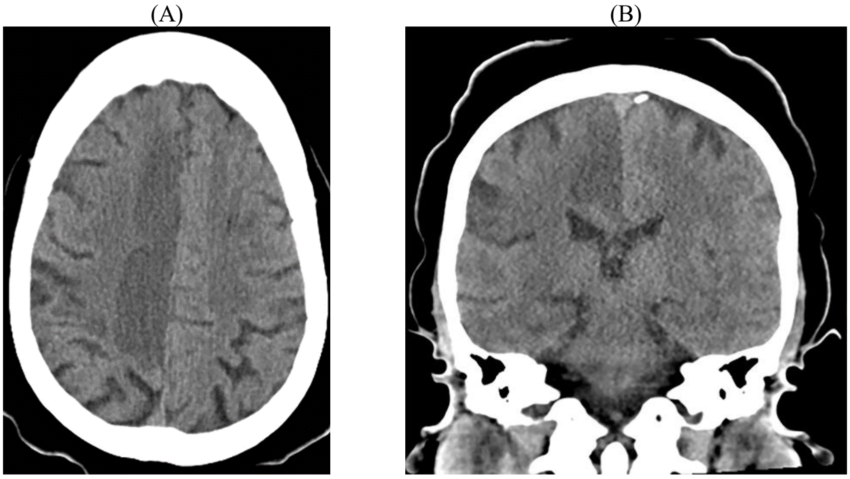

Fig. 5. M/35. (A) Transaxial view, (B) Coronal view. This patient was diagnosed with rheumatic heart disease with atrial fibrillation. There was a syncope attack with left hemiplegia. Non-contrast CT reveals an ischemic infarct of the medial side of the right frontoparietal lobe, as the anterior cerebral artery (ACA) territory ischemia.

The extent of the ischemic infarct area was measured visually into four grades, 1) focal or wedge in 57 patients (28.5%), 2) lobar in 52 patients (26%), 3) two lobar in 55 patients (27.5%), and 4) three lobar or more in 36 patients (18%).

Associated findings in the order of the frequency were brain atrophy in 83 patients (41.5%), followed by mass effect, in 21 patients (10.5%), including subfalcine herniation, in 13 patients, and compressed lateral ventricle, in 8 patients, sinusitis in 15 patients (7.5%), hemorrhagic transformation in 12 patients (6%), old neurocysticercosis in 5 patients (2.5%), and HIV encephalopathy in 3 patients (1.5%). Other associated conditions were fibrous dysplasia, left common carotid artery aneurysm, chronic otitis media with mastoiditis, parotid tumor, and bone metastasis of prostate cancer in one case respectively.

CT findings of ischemic infarct with hemorrhagic transformation were found in 12 patients (6%). The age distribution of the patients with hemorrhagic transformation, in each age group of ischemic infarcts, was 4 patients (23.5%) among 17 patients in their thirties, 2 patients (9.5%) among 21 patients in their forties, 2 patients (3.8%) among 52 patients of their sixties, 3 patients (5.9%) among 51 patients of their seventies, and one patient (5%) among 20 patients of their eighties. The vascular territory of the lesion of ischemic infarct with hemorrhagic transformation was MCA in 9 patients (75%), with 3 patients with left MCA, and 6 patients with right MCA. Lesions of the other 3 patients were multiple lesions on both sides of the brain. Intraventricular hemorrhage was associated in 2 patients (17%). The associated findings were mass effect in 6 patients (50%), including subfalcine herniation in 4 patients and compressed lateral ventricle in 2 patients, followed by brain atrophy in 2 patients.

4. Discussion

Stroke is an acute central nervous system injury and one of the leading causes of death in the world. Atherosclerotic disease of carotid origin and embolus associated with untreated atrial fibrillation are two of the most common causes of stroke in adults [1-4]. Thrombotic ischemic strokes are generally associated with atherosclerotic accidents and are classified based on the size of the involved vessels. Depending on the speed of the plaque rupture, it can be presented with sudden devastating damages or subtle pathologic changes that manifest slowly. In the latter situation, collateral circulation would partly compensate for the circulation loss. The small-vessel ischemic stroke could occur because of systemic and chronic diseases, such as diabetes [5, 6, 7]. Spontaneous arterial dissection is more common in patients younger than 40 years or after acute vascular trauma [6].

Generally, the time window for IV thrombolysis following the onset of stroke symptoms is considered as the 3- to 4.5-hour time window, which is markedly variable according to individual patients. Arterial occlusion in ischemic stroke causes decreased cerebral blood flow and cerebral perfusion pressure. The subsequent 3 stages are generally accepted. Stage 1 is compensatory cerebral autoregulation that maintains constant cerebral blood flow via maximal dilation of small arteries and arterioles and recruitment of collaterals, producing a compensatory increase in cerebral blood volume. In stage II, when maximal autoregulatory vasodilation is exhausted, oxygen extraction fraction is increased to maintain brain tissue oxygenation and metabolism necessary for cellular viability. In stage III, when the autoregulatory range of cerebral perfusion pressure reduction is overwhelmed at the ischemic core, it results in a decrease of cerebral blood volume and an ensuing decrease in cerebral blood flow until it crosses the threshold when collaterals fail, with venous collapse leading to tissue oxygenation decrease and, ultimately, bioenergetic cell death [8]. The ischemic penumbra refers to tissue at risk of infarction if reperfusion does not occur in a timely manner. This dysfunctional but salvageable tissue has been the target of all reperfusion and neuroprotection therapies.

There are different subtypes of stroke. The National Institute of Neurological Disorders and Stroke (NINDS) Stroke Data Bank recognized 5 major groups: 1) Brain hemorrhage, 2) brain infarctions, including atherothrombosis, 3) cardioembolic, 4) lacunar stroke, and 5) stroke from rare causes or undetermined etiology. The TOAST classification includes 1) large artery atherosclerosis, 2) cardioembolic stroke, 3) small vessel disease, 4) a stroke of other determined cause, and 5) a stroke of undetermined cause (cryptogenic). Oxfordshire Community Stroke Project classifications are 1) cerebral infarction, 2) lacunar infarct, 3) total anterior circulation infarct, 4) partial anterior circulation infarct, and 5) posterior circulation infarct [8, 9].

In our study, the etiological diagnosis was not finally proven. However, multiple ischemic lesions on both sides were found in 48 patients (24%), which were probably caused by cardiac embolism. Lacunar syndrome of the deep brain, including basal ganglia, thalamus, midbrain, and pons, was found in 39 patients (19.5%), which was caused by small vessel diseases. Total and partial anterior circulation syndrome were found in 91 patients (45.5%), which might be caused by large vessel atherosclerosis. The most common vascular territory was the middle cerebral artery (MCA) territory (41%), followed by posterior cerebral artery territory (PCA) (7%), internal carotid artery territory (ICA) (2.5%), and anterior cerebral artery territory (ACA) (2%).

For the different vascular territories of ischemic stroke, prospectively over a 9-year period, one study for 2213 individuals who sustained first-ever ischemic strokes and were admitted to an inpatient stroke rehabilitation program showed the results as follows. Strokes were divided into the anterior cerebral artery, middle cerebral artery (MCA), posterior cerebral artery, brain stem, cerebellar, small-vessel strokes, and strokes occurring in more than one vascular territory [5]. The most common stroke groups were MCA stroke (50.8%) and small-vessel stroke (12.8%). After adjustments for age, gender, risk factors, and admission year, the stroke groups can be arranged from most to least severe disability on admission: strokes in more than one vascular territory, MCA, anterior cerebral artery, posterior cerebral artery, brain stem, cerebellar, and small-vessel strokes [5].

The annual incidence rate of stroke in Africa is up to 316 per 100,000 individuals, which is within the highest incidence rates in the world, and the prevalence rate of 1,460 per 100,000 reported in one region of Nigeria, western Africa, is clearly among the highest in the world [10]. Africa has a slightly greater preponderance of small vessel disease-related stroke and intracerebral hemorrhagic lesions than elsewhere in the world [11]. In another report from Africa, the most common aetiological subtype of ischaemic stroke is small vessel disease (SVD) (42%), followed by large vessel atherosclerotic disease (17%), cardioembolism (11%), and undetermined (30%) [10].

Hypertension remains the most important modifiable risk factor for stroke in Africa, but others include diabetes mellitus, dyslipidemia, obesity, stress, smoking, alcohol use, physical inactivity, and an unhealthy diet [10]. Africans with HIV and stroke generally have a young age at stroke onset (<45 years), severe stroke presentation, the preponderance of ischaemic strokes, and advanced immunosuppression [11, 12]. In Sub-Saharan Africa, stroke represents an important part of the chronic disease burden. In a review article, the analysis showed that patients who had diabetes (OR = 2.39) and HIV (OR = 2.46) were at a significantly greater risk of suffering from all stroke types. Among case series, the pooled prevalence of hypertension was higher for hemorrhagic stroke than for ischemic stroke (73.5% versus 62.8%), while diabetes mellitus (DM) and atrial fibrillation (AF) were more prevalent among ischemic stroke compared to hemorrhagic stroke [13].

Another infectious risk factor unique in Africa is sickle cell disease (SCD). Cerebral ischemia in sickle cell disease is thought to result from the occlusion of distal internal carotid arteries, middle cerebral arteries, and anterior cerebral arteries [14]. Hemorrhagic strokes in SCD usually occur in the third decade of life and are associated with low steady-state hemoglobin levels and high steady-state leukocyte counts [10, 15].

There are multiple drivers of the rising burden of stroke in Africa. Early-life undernutrition might also partly explain the increased risk of cardiometabolic disorders among individuals who grow up in low-income rural areas. Late-life factors driving the increasing burden of stroke in Africa include a progressive increase in life expectancy, which is resulting in population aging. The limited availability of medication to control the more common risk factors is another factor. Drugs for the treatment of hypertension and diabetes are costly and might not be readily available in Africa [10, 15].

Stroke is the second leading cause of death after HIV/AIDS in South Africa. The most associated risk factors are hypertension, smoking, obesity, high cholesterol, diabetes, and HIV infection [11, 16, 17]. The timing of presenting to the emergency department was variable with some patients within 4.5 hours of the current window of thrombolytic therapy. But in most of the patients, the symptom onset was not recorded and presented beyond this window, and in a report, the average time to ED presentation was 33 hours, and the average time to CT scan was 6 hours [15, 16].

Pertaining to pre-hospital acute stroke care, a systematic review of studies from Africa found a median time from stroke onset to hospital admission of 31 hours, compared to the median onset-to-door time of 140 minutes in the European study (SITS EAST study) and 144 minutes in the American study (GWTG-Stroke study) [11-16]. In hospital-based acute stroke care, services for stroke often exist as part of general medical services, although stroke units have now been reported in some African countries. Neuroimaging services (CT or MRI) are essential for the accurate diagnosis of stroke, but the availability, accessibility, and affordability of these services are limited in many African settings [11, 13]. The availability of and access to acute reperfusion therapies in Africa are currently limited but growing.

The purpose of imaging diagnosis in the suspicion of stroke is to differentiate vascular from non-vascular lesions, such as tumors or infections, ischaemic from hemorrhagic stroke, arterial from venous infarction, and to distinguish anterior and posterior circulation strokes to determine the etiology of vascular occlusions, such as large vessel occlusion or cardiogenic embolism [8, 18]. CT is the primary imaging modality used for selecting appropriate treatment in patients with acute stroke. To make accurate and timely decisions regarding both (a) immediate treatment with intravenous tissue plasminogen activator up to 4.5 hours after a stroke at primary stroke centers and (b) transfer of patients with large-vessel occlusion (LVO) at CT angiography, CT is the central diagnostic modality at the comprehensive stroke centers for endovascular thrombectomy (EVT) up to 24 hours after a stroke [6].

Early ischemic changes on non-contrast CT appear as hypodensity (cytotoxic edema), loss of gray-white differentiation, cortical swelling, and loss of sulcation (effacement of brain sulcus from tissue swelling) [8]. CT findings of the early signs of proximal middle cerebral artery (MCA) large-vessel occlusive infarction seen at nonenhanced CT include loss of gray-white matter differentiation (GWD) at the insula, basal ganglia, and caudate head as well as sulcal effacement. These early findings may be found at the insula, caudate heads, and basal ganglia in the cases of proximal MCA thrombosis at nonenhanced CT [6, 8]. The hyperattenuating vessel sign can be seen in the intracranial ICA, M1, and M2 segments of the MCA, and A1 and A2 segments of the anterior cerebral artery (ACA) as well as the basilar and vertebral arteries [6, 8].

It is recommended that all patients should undergo CT angiography immediately after undergoing nonenhanced CT without being removed from the CT scanner. CT angiography has become the standard for rapidly accurately identifying intracranial large-vessel occlusion (LVO) [6]. Recent non-invasive neuroimaging for the diagnosis of ischemic stroke are multimodal CT and MRI, which enable prompt diagnosis, identify treatable underlying causes of stroke, and enhance the selection of candidates for reperfusion therapy. Multimodal CT includes CT angiography (CTA) and CT perfusion, whereas multimodal MRI includes parenchymal sequences such as diffusion-weighted imaging (DWI) with apparent diffusion coefficient (ADC) maps, gradient recalled echo (GRE), susceptibility-weighted imaging (SWI), fluid-attenuated inversion recovery (FLAIR), magnetic resonance angiography (MRA), and perfusion-weighted MRI [8].

Etiological diagnosis may be derived from the pattern of the lesions in CT imaging. An isolated lenticulostriate lesion points to a lacunar infarct. Multiple lesions in different vascular territories suggest a cardioembolic/aortoembolic source, whereas scattered lesions in one vascular territory are associated with large artery atherosclerosis [8, 18].

Hemorrhagic transformation refers to hemorrhagic infarction that occurs after venous thrombosis or arterial thrombosis and embolism [19]. Autopsy studies have reported an HT rate of 18–42% in acute ischemic stroke due to arterial occlusion [19, 20]. In a systematic review, the frequency of patients with any degree of hemorrhagic transformation of ischemic infarct (from petechial hemorrhage to frank hematoma formation) varied from 0% to 85%. This variability is due to the variable definition of hemorrhagic transformation and different cohorts and times of scan.

Our study found hemorrhagic transformation in 12 patients (6%). In 9 patients (75%), the location of the ischemic infarct with hemorrhagic transformation was the middle cerebral artery territory. And the younger aged patients in their thirties and forties showed a higher percentage, 23.5% and 9.5% of the patients with ischemic infarct of the same age group. However, age itself may not be the definite risk factor, over preceding other risk factors such as uncontrolled hypertension or other underlying pathology.

The blood–brain barrier (BBB) is a physiological barrier between the brain parenchyma and brain circulation that nourishes brain tissue, filters various substances from the brain to the blood, and protects the brain. The BBB is composed of endothelial cells, basement membrane, pericytes, and astrocytes, collectively referred to as the neurovascular unit and linked to circulating peripheral blood cells. Early disruption of the BBB plays a pivotal role in HT formation during acute ischemic stroke. Leukocyte types and various molecules are associated with HT after ischemic stroke. Neutrophils and brain tissue are major matrix-metalloproteinase-9 (MMP-9) sources within the first 18–24 h after stroke. Intravenous infusion of exogenous tissue plasminogen activator (tPA) can increase MMP-9 levels by activating neutrophils, and endogenous tPA can increase MMP-3 levels by acting on endothelial cell lipoprotein receptor protein (LRP) and can increase MMP-2 levels by activating platelet-derived growth factor-CC as a trigger via astrocyte platelet-derived growth factor receptor A [19].

Hemorrhagic transformation of ischemic infarct was radiologically defined in the National Institute of Neurological Disorders and Stroke (NINDS) recombinant tissue plasminogen activator study as acute infarction with punctate or variable hypodensity/hyperdensity, with an indistinct border within the vascular territory [21]. The radiographic definition of HT is generally classified by the European Cooperative Acute Stroke Study (ECASS). On CT scans, the severity of HT is divided into two stages: hemorrhagic infarction (HI) and parenchymal hemorrhage (PH) with or without mass effect. Each stage is divided into two subtypes [20]. According to European Cooperative Acute Stroke Study (ECASS), in the hemorrhagic infarct, the two subtypes are small petechial bleeding along the margins of the infarcted area and confluent petechial bleeding within the infarcted area. In the parenchymal hemorrhage, the two subtypes are hematoma in <30% of the infarcted area and hematoma in more than that [19, 21].

In a study assessing tinzaparin (low molecular weight heparin) versus aspirin, asymptomatic hemorrhagic transformation is increased in ischemic stroke with cortical syndromes including total anterior circulation syndrome and partial anterior circulation syndrome, and of the large vessel or cardioembolic etiology. Heparin does not increase asymptomatic hemorrhagic transformation. The asymptomatic hemorrhagic transformation of ischemic stroke is not associated with functional outcomes [21]. The risk of early neurological deterioration and of 3-month death was severely increased after parenchymal hemorrhage subtype-2, indicating that large hematoma is the only type of hemorrhagic transformation that may alter the clinical course of ischemic stroke [22].

In summary, we encountered 200 patients whom we diagnosed with ischemic stroke in the year 2022 in Eswatini and analyzed the clinical and CT findings of those patients. This study may be a helpful guide for further comprehensive study with a larger cohort of the clinical, imaging, and outcomes of stroke in southern Africa including Eswatini.

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