# Chapter 8
# Strokes as Seen in Mulago Hospital, Uganda

**Stephen Muwonge Matovu and Robert Mukisa**

**Abstract** This chapter examines stroke as the leading cause of admission, disability and death in the Neurology unit at Mulago National Referral Hospital in Uganda. Patients are either referred from a primary health care center or present directly to the hospital. There is an increasing burden of stroke patients in the inpatient wards, outpatient clinic and the physiotherapy departments. This is because the population is undergoing a rapid epidemiological transition with increased exposure to, and development of, stroke risk factors, together with aging of the population. The clinical presentation of stroke, management and rehabilitation are discussed. Community awareness of the four preventative strategies of stroke, improved stroke care in the Neurology unit and rehabilitation are cornerstones in mitigating the stroke burden and its antecedent complications. This chapter, however, will mostly report on preliminary studies undertaken in Mulago hospital, Uganda to elucidate the burden of stroke at this tertiary care facility as there is a paucity of studies on the subject of stroke in Uganda.

**Keywords** Stroke burden • Risk factors • Stroke complications • Mulago hospital

## Introduction

Stroke is defined as rapidly developing clinical signs of focal (or global) disturbance of cerebral dysfunction with symptoms lasting 24 h or longer or leading to death with no apparent cause other than that of vascular origin [1]. Stroke is thus a term that is used to describe brain injury caused by an abnormality of the blood supply to a part of the brain.

Stroke is a relatively common presenting diagnosis to Mulago National Referral Hospital with the number of patients being admitted with stroke on the Neurology unit being on the increase. Between June and October 2011, 206 patients with stroke

S.M. Matovu (✉) • R. Mukisa
Department of Medicine, Mulago National Referral Hospital, Kampala, Uganda
e-mail: matmuwonge@yahoo.com



S. Musisi, S. Jacobson (eds.), *Brain Degeneration and Dementia in Sub-Saharan Africa*, DOI 10.1007/978-1-4939-2456-1_8

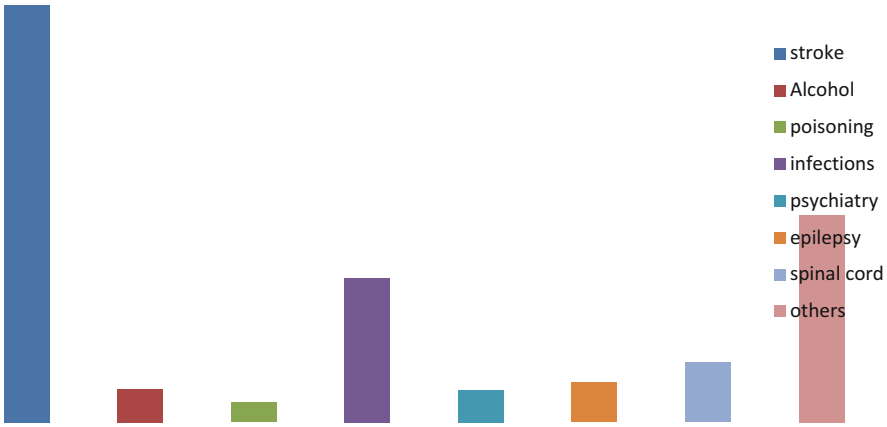


**Fig. 8.1** Disease burden on the neurology unit in Mulago Hospital, 2012 (Matovu et al. 2012 data unpublished)

were admitted compared to only 109 in 2005 and 60 in 1999 during the same months period [2]. Stroke is thus the leading cause of admission on the Neurology Unit at Mulago National Referral Hospital (Fig. 8.1).

## Risk Factors for Stroke

In a study carried out in Uganda's Mulago National Referral hospital, in Kampala, Nakibuuka et al. [3] investigated the main risk factors for ischemic and hemorrhagic stroke. The parameters noted were past medical history, physical examination findings, key laboratory investigations and Ultrasonography investigations. Table 8.1 below shows the main findings.

For both types of stroke (ischemic and hemorrhagic), the outstanding risk factors for stroke were Hypertension (BP > 140/90 mmHg), physical inactivity, alcoholism, dyslipidemias, Diabetes Mellitus, arthrosclerosis and a positive syphilis serology.

### *Hypertension*

More than 50 % of patients with either ischemic or hemorrhagic stroke reported a history of hypertension, in which more than half of the patients were found to have a blood pressure greater than 140/90 mmHg. Of these patients, however, 27 (32 %) patients reported sporadic use of anti-hypertensive medications, with only 7 patients (8 %) reporting regular medication use. Only 3 patients reported regular use of aspirin prophylaxis for stroke, and these all presented with ischemic stroke.

**Table 8.1** Risk factors of stroke among patients admitted to Mulago National Referral Hospital in 2012

| Risk factor | Percentage of patients | Percentage of patients |
|---|---|---|
| | **With ischemic stroke (N = 66)** | **With hemorrhagic stroke (N = 19)** |
| **I. Past medical history** | | |
| Hypertension | 57.6 | 68.4 |
| Physical inactivity | 40.9 | 36.8 |
| Diabetes mellitus | 12.1 | 5.2 |
| Current smoking | 7.6 | 5.2 |
| Alcohol | 18.2 | 21.1 |
| **II. Examination findings** | | |
| Irregularly irregular pulse | 22.7 | 5.3 |
| Systolic BP >140 mmHg | 47.0 | 73.7 |
| Diastolic BP >90 mmHg | 36.4 | 68.4 |
| **III. Laboratory findings** | | |
| Total cholesterol >200 mg/dl | 31.0 | 39.0 |
| LDL cholesterol >110 mg/dl | 43.1 | 33.3 |
| HDL cholesterol <40 mg/dl | 38.0 | 17.0 |
| Triglycerides >150 mg/dl | 23.1 | 33.3 |
| FBS >126 mg/dl | 43.1 | 55.6 |
| Reactive TPHA | 26.2 | 50.0 |
| Reactive HIV serology | 7.7 | 0 |
| **IV. Ultrasound findings** | | |
| Atherosclerosis on CUS | 46.0 | 46.7 |

## *Cardiac Causes: Embolic Source, Atrial Fibrillation and Stenosis*

In Nakibuuka's study, 18 patients had a suspected cardio-embolic stroke, with 18.8 % patients found to have an irregularly irregular pulse. However, none of these patients were aware of a previous diagnosis of atrial fibrillation or were taking any medications. Two of these patients were found to have severe valvular heart disease, suggesting that atrial fibrillation could be an important under-recognized cause of stroke in African populations [3–5]. Atherosclerosis in the carotid arteries and cardiac left ventricular hypertrophy were commonly found on physical examination.

## *Diabetes Mellitus, Dyslipideamias and Lifestyle*

Thirty eight patients were found to have a Fasting Blood Sugar (FBS) greater than 126 mg/dl, consistent with a diagnosis of Diabetes Mellitus, but only nine of these patients had a known previous diagnosis of the diabetes. Commonly recognized risk factors, such as hypercholesterolemia, physical inactivity, current smoking and alcohol were frequently found in this stroke victim population.

### *Syphilis and Human Immunodeficiency Virus*

Twenty six patients were found to have reactive serology for syphilis, and only five patients were found to have HIV diagnosed by Abbot test. All these patients were on treatment for these disorders.

### *Socio-demographic Associations of the Stroke Victims Seen at Mulago Hospital*

In terms of their sociodemographic characteristics, Nakibuuka et al. [3] found that the stroke victims were almost of equal gender representation with 51.8 % females but they had a bimodal age distribution with a peak in the 40–49 year age group (22.4 %) and in those aged 60–80 years (37.6 %). Generally, stroke was more common in older individuals and the mean age for all stroke patients was 62.2 years with the vast majority of them being above 40 years old (83.5 %). Most were married (61.2 %), had primary school education or below (54.1 %) and were employed (52.9 %) as shown in Table 8.2 below. These findings were similar to the findings of studies done in other African countries in Zimbabwe [6, 7].

**Table 8.2** Socio demographic characteristics of stroke patients admitted to Mulago National Referral Hospital

| Characteristics | Number (N = 85) | Percentage (%) |
|---|---|---|
| **Age (years)** | | |
| 20–29 | 8 | 9.4 |
| 30–39 | 6 | 7.1 |
| 40–49 | 19 | 22.4 |
| 50–59 | 11 | 12.9 |
| 60–69 | 16 | 18.8 |
| 70–79 | 16 | 18.8 |
| 80+ | 9 | 10.6 |
| **Gender** | | |
| Female | 44 | 51.8 |
| **Highest education level attained** | | |
| Never been to school | 14 | 16.5 |
| 0–7 years | 32 | 37.6 |
| 8–12 years | 23 | 27.1 |
| More than 12 years | 16 | 18.8 |
| **Marital status** | | |
| Never married | 6 | 7.1 |
| Married | 52 | 61.2 |
| Divorced | 17 | 20.0 |
| Widowed | 10 | 11.8 |

(continued)

**Table 8.2** (continued)

| Characteristics | Number (N = 85) | Percentage (%) |
|---|---|---|
| **Occupation** | | |
| Student | 3 | 3.5 |
| Unemployed | 37 | 43.5 |
| Employed | 45 | 52.9 |

**Table 8.3** Incidence of ischemic and hemorrhagic stroke according to gender and age group as seen at Mulago Hospital[a]

| | Ischemic stroke | | | | | | Hemorrhagic stroke | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | **Male N = 35** | | | **Female N = 31** | | | **Male (n = 9)** | | **Female N = 10** | |
| Age (years) | AS | CE | O | AS | CE | O | IPH | SAH | IPH | SAH |
| 21–30 | 0 | 3 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 31–40 | 1 | 0 | 0 | 0 | 0 | 3 | 1 | 0 | 0 | 0 |
| 41–50 | 2 | 1 | 0 | 1 | 2 | 0 | 1 | 1 | 1 | 1 |
| 51–60 | 7 | 1 | 1 | 1 | 2 | 2 | 1 | 0 | 1 | 1 |
| 61–70 | 3 | 2 | 2 | 5 | 3 | 2 | 2 | 0 | 3 | 0 |
| 71+ | 7 | 0 | 4 | 5 | 4 | 1 | 2 | 1 | 3 | 0 |
| Total | 20 | 7 | 8 | 12 | 11 | 8 | 7 | 2 | 8 | 2 |

[a]*AS* atherosclerotic, *CE* cardio-embolic, *O* other, *IPH* intraparenchymal hemorrhage, *SAH* subarachnoid hemorrhage

## Stroke Types

Strokes are divided into two very broad groups namely hemorrhagic strokes and ischemic strokes. In ischemia, there is not enough blood supply to allow continued normal functioning of the affected brain tissue. Brain ischemia is much more common in these than in those due to hemorrhage. About four strokes out of every five are ischemic. In hemorrhage, there are several different subtypes characterized by their locations inside of the skull. Hemorrhages within the brain substance (inside of the pia mater) are called intracerebral hemorrhages. Those between the pia mater and arachnoid are called subarachnoid hemorrhages. Regarding stroke types in Mulago National Referral hospital, 77.6 % were ischemic while 22.4 % were hemorrhagic stroke as confirmed by Computed Tomography scan of the brain. The incidence of both ischemic and hemorrhagic stroke increased with age as seen in Table 8.3 [3]. Mukisa et al. [8] in a separate study done in Mulago National referral hospital, had the same findings with the majority of patients admitted with stroke having ischemic stroke (82.4 %) and those with hemorrhagic stroke were 17.6 %. The observed ratio of ischemic to hemorrhagic stroke of 4:1 was similar to other studies among African populations [9–11]. Atherosclerotic stroke was the commonest ischemic stroke in etiology, being observed in 43.5 % patients with ischemic stroke. Intraparenchymal hemorrhage was the most common hemorrhagic stroke in etiology, in 78.9 % of patients with hemorrhagic stroke [3]. Table 8.3 summarizes these findings.

## Clinical Presentation of Stroke

### *Cerebral Infarction*

Here the symptoms reflect the vascular territory involved [12]. Dominant hemisphere stroke (left sided stroke) presents with aphasia, left gaze deviation (preference), right visual field deficit, right hemiparesis and right hemisensory loss. Non-dominant hemisphere stroke (right sided stroke) presents with left hemi-inattention, right gaze deviation (preference), left visual field deficit, left hemiparesis and left hemisensory loss. In Intracerebral haemorrhage, symptoms may progress over the first several hours as the haematoma expands. Vomiting may occur in most cases [13].

**Brainstem** involvement presents with diplopia/dysconjugate gaze, gaze deviation, paralysis/paresis of vertical eye movement, vertigo/tinnitus, nystagmus, Horner's syndrome, dysarthria, dysphagia, hemiparesis or quadriparesis, sensory loss in hemibody or all four limbs, crossed signs, nausea, vomiting, hiccups, decreased consciousness and abnormal respirations.

**Cerebellar** involvement presents with truncal/gait ataxia and ipsilateral limb ataxia. Cerebellar haematoma presents with sudden onset of headache, severe ataxia, dysarthria, nystagmus, vertigo and vomiting. Cerebrospinal fluid obstruction hydrocephalus with symptoms and signs of raised intracranial pressure.

**Supratentorial haematoma** presents with sudden onset of headache followed by either rapid loss of consciousness or a gradual deterioration in conscious level over 24–48 h due to mass effect. Hemiparesis, hemisensory loss and homonymous hemianopia are common. Third nerve palsy indicates transtentorial herniation.

**Pontine haematoma** presents with sudden loss of consciousness, quadriplegia, respiratory irregularities, slowed respiration, pinpoint pupils, pyrexia, dysconjugate eye movements and death often follows.

### *Subarachnoid Haemorrhage*

Here, the severity of the symptoms is related to the severity of the bleed and symptoms usually continue for many days. Sudden onset of a severe headache, transient or prolonged loss of consciousness or epileptic seizure may immediately follow. Nausea and vomiting are common. A stiff neck may be present after 3–12 h. Bleeding may injure adjacent tissue and produce symptoms such as limb weakness and inability to talk (Braunwald et al. Harrison's 16th edition page 2387).

Clinically, in a review study of admitted stroke patients at Mulago hospital neurology unit, Kwarisiima et al. [14] found that all (100 %) of admitted patients presented with focal neurological deficits with 76 % having motor deficits as the commonest neurological deficit followed by impaired level of consciousness which accounted for 61 % of the admissions. Table 8.4 summarizes these points.

**Table 8.4** Clinical presentation of stroke patients admitted in Mulago National Hospital

| Sign | Frequency N = 128 | Percentage |
|---|---|---|
| Sudden trouble in walking | 108 | 84 |
| Sudden weakness of the face | 109 | 85 |
| Sudden numbness of the faces | 80 | 63 |
| Sudden onset of vomiting | 37 | 29 |
| Loss of consciousness | 78 | 61 |
| Motor | 97 | 76 |
| Sensory | 2 | 02 |
| Motor and sensory | 29 | 22 |
| Glasgow coma scale | | |
| <9 | 38 | 30 |
| ≥9 | 90 | 70 |

## Investigations for Stroke at Mulago Hospital

### *Neuroimaging*

Mulago National Referral hospital is Uganda's national referral hospital and therefore a tertiary care hospital. As such the department of Radiology has a Computed Tomography (CT) scan. This enables confirmation of the diagnosis of stroke, establishes the underlying pathology, identifies the size and site of the lesion, and establishes the etiological mechanism. The brain Computed Tomography scan will establish the pathological diagnosis as either infarction or haemorrhage and exclude other conditions that may mimic stroke. In Mulago hospital 28 % of patients presenting with a suspected stroke were found to have a non-stroke diagnosis [3], a value much lower than 43 % found in a study done in Nigeria [15]. Nevertheless, it still emphasizes the relevance of CT scan as gold standard in stroke diagnosis [11, 15, 16] as Table 8.5 below illustrates.

### *Computed Tomography Appearance*

A haemorrhage is seen within a few minutes as an area of increased attenuation (Fig. 8.2). The appearances of an infarct change over the first few weeks, and not all infarcts show up on Computed Tomography scan (Fig. 8.3). Magnetic Resonance Imaging is more sensitive to small areas of ischemia and can detect the traces of old haemorrhage (haemosiderin deposits) indefinitely.

**Table 8.5** Non-contrast head CT- scan findings among patients at Mulago Hospital

| Non-contrast head CT scan finding | N (%) |
|---|---|
| Ischemic stroke | 66 (52.0) |
| Hemorrhagic stroke | 19 (15.0) |
| Neoplasm[a] | 8 (6.3) |
| Severe brain atrophy | 8 (6.3) |
| Subdural hematoma[a] | 6 (4.7) |
| Brain abscess[a] | 4 (3.1) |
| Toxoplasmosis | 4 (3.1) |
| Normal repeated CT scan on day 7 | 3 (2.4) |
| Intracranial hypertension | 3 (2.4) |
| Extensive calcifications | 2 (1.6) |
| Pan-sinusitis | 1 (0.8) |
| Meningoencephalitis | 1 (0.8) |
| Tuberculosis choroiditis | 1 (0.8) |
| Chronic epidural hematoma[a] | 1 (0.8) |

[a]Surgically correctable conditions


**Fig. 8.2** Computed tomography scan showing cerebral haemorrhage

## *Other Imaging Investigations*

### Doppler Ultrasound Scans of the Carotid Arteries

Doppler ultrasound scan of the carotid arteries is done at the Uganda Heart Institute, Mulago National Referral Hospital. It detects atherosclerotic plaques, discrete lesions, abnormal velocity flow patterns, irregular flow peaks, occlusion, dissection and evidence of internal carotid artery stenosis.

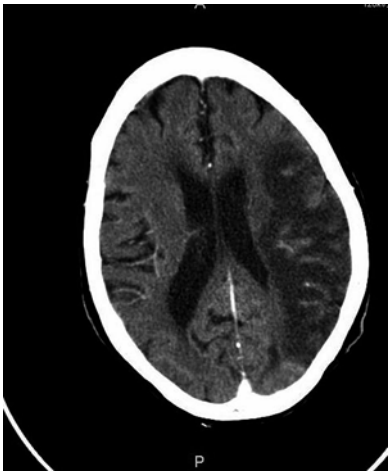


**Fig. 8.3** Computed tomography scan showing cerebral ischemia

**Cardiac Investigations**

These are done at the Uganda Heart Institute, Mulago National Referral Hospital. An Electrocardiogram is used detect atrial fibrillation. Transoesophageal echocardiography is used to identify sources of stroke such as akinetic wall segments, mural thrombi, severe valvular lesions, multiple valvular lesions, valvular vegetations, ventricular ejection fraction of <35 %, an atrial abnormality, ulcerated aortic atherosclerosis or dissection.

## *Laboratory Investigations in Acute Stroke*

The following essential investigations should be done routinely for patients admitted with stroke such as to the Neurology unit in Mulago.

***Full blood count***: Thos will indicate the presence of polycythaemia, raised white cell count due to infection, thrombocythaemia which increases the risk of infarction and thrombocytopenia which predisposes to haemorrhage

***Erythrocyte sedimentation rate***: If this is raised, it may indicate infection or Vasculitis

***Urea and electrolytes***: These are affected by dehydration which may result into hypo perfusion

***Fasting blood sugar***: Is often raised initially. If it persists then this confirms diabetes mellitus

***Syphilis serology***: This establishes a diagnosis of meningo-vascular neurosyphilis.

***Coagulation profile***: This is essential in hemorrhagic stroke and in young patients without identifiable risk factors

***Blood cultures***: Do this if you suspect bacterial endocarditis
***Fasting lipid profile***: This detects underlying dyslipideamias
***Lumbar puncture***: *It h*elps in confirming subarachnoid haemorrhage (xanthochromic), meningitis and encephalitis. Also do a VDRL on it to exclude syphilis

## Management

The overall aim of stroke care is to decrease morbidity and mortality, to optimize function recovery and to prevent recurrence of strokes. This is achieved by good nursing care, specific stroke treatment, maintenance of fluid and electrolytes, nutrition, avoiding systemic complications and early rehabilitation.

### *General Treatment in Caring for Acute Stroke Patients*

1. Start with hourly neurological observations and change to 4 hourly if stable. These include:
   - Level of consciousness using Glasgow Coma Scale
   - Vital signs
   - Oxygen saturation
2. Monitor blood glucose. If more than 11 mmol/L start insulin sliding scale
3. Intravenous fluids in dehydrated patients and those unable to swallow
4. Evaluate swallowing after 24 h
   - Observe the patient attempting to swallow using sips of water in upright position
   - Check for coughing or gagging reflexes
   - If swallowing is impaired keep nil per mouth.
   - Continue intravenous fluids for 48 h then start nasogastric tube feeding if still unable to swallow
5. Urinary catheterization if incontinent or in retention
6. Prevent constipation by adequate hydration and laxatives
7. Prevent pressure sores by advising a relative or a care taker to supervise 2 hourly turning
8. Decrease the risk of deep vein thrombosis by using compression stockings in addition to oral aspirin if intracerebral haemorrhage is excluded
9. Treat sepsis and pyrexia with antibiotics and antipyretics respectively
10. Early physiotherapy and mobilize out of bed
11. Occupational, speech and language therapy
12. The family is taught good nursing care/manual handling techniques before discharge home

## *Specific Treatments*

### Antiplatelet Drugs

All ischaemic strokes are started on aspirin immediately or as soon as the diagnosis is made followed by long term treatment. Aspirin, when given effectively, prevents deaths, major disability (dependence) and recurrent strokes when used as longer term therapy. The dose is 300 mg per os daily for the first 2 weeks followed by 75–150 mg per os thereafter. Patients that are intolerant of aspirin are treated with either clopidogrel or dipyridamole.

### Antihypertensive Drugs

Blood pressure lowering during the first 24–48 h is avoided as an acute drop in blood pressure can reduce perfusion to an already ischaemic brain. Treatment is considered only if the blood pressure is persistently elevated that is ≥180/105 mmHg in ischaemic stroke. Aim at a daily reduction of 10–20 mmHg. In intracerebral haemorrhage treatment is started if the blood pressure is more than 160/100 mmHg. In the acute phase, blood pressure reduction is achieved by treating with sublingual nifedipine and then orally twice daily. For a gradual reduction, use captopril, atenolol or hydrallazine as the alternatives used.

### Anticoagulation

Anticoagulants are used in patients with an ischaemic stroke and a cardiac embolic source or atrial fibrillation to prevent further strokes. Patients are first treated with aspirin as anticoagulation is started 2 weeks later after the stroke because of the risk of intracerebral haemorrhage. Warfarin is administered per os in a loading dose of 10 mg daily for 2 days followed by a maintenance dose depending on the prothrombin time or international normalized ratio (INR). The aim is to have and maintain an INR of two to three or a prothrombin time of twice the normal range.

### Thrombolysis

Thrombolytic therapy for ischaemic strokes with intravenous recombinant tissue plasminogen activators like alteplase is not possible in Mulago Hospital, because often, the minimum time of admission to Mulago hospital of patients with an acute stroke after its occurrence is 2 days [7]. Thrombolytic agents are only beneficial when administered within 3–6 h after stroke onset at maximum.

**Neurosurgery**

Evacuation of a cerebellar haematoma is life saving. Evacuation of supratentorial hematomas is done in only younger patients with deteriorating consciousness. Neurosurgery is beneficial for obstructive hydrocephalous following stroke.

## Stroke Complications

Stroke patients are at risk for complications which may lead to death. Cerebral edema, progression of the stroke and complications are responsible for the common neurological worsening in the first 48 h.

### *Acute Complications*

The main acute complications are aspiration pneumonia, pulmonary embolism, pressure sores and urinary tract infections. They occur in over 50 % of hospitalized stroke patients and are associated with a poor prognosis. Aspiration pneumonia is the main cause of death in stroke in hospitalized patients. It is frequent in patients with extensive strokes and coma. Management includes avoiding oral intake, chest physiotherapy and use of antibiotics.

Patients with stroke are at significant risk for deep vein thrombosis and pulmonary embolism. The use of low dose aspirin and compression stockings decreases this risk. Pressure sores, spasticity and contractures are common and are reduced by early patient positioning, 2 hourly turning, and passive exercises.

### *Chronic Complications*

These include disability, spasticity, contractures, pain, depression, dementia and late onset seizures. Post stroke depression is common occurring in over 50 % of the patients, especially in right sided stroke. It's treated using tricyclic antidepressants such as Imipramine or selective serotonin reuptake inhibitors such as Fluoxetine.

Dementia after stroke is common and is a major long term cause of dependency, particularly in the elderly [17, 18]. In Mulago hospital the prevalence of cognitive impairment among patients admitted with stroke was found to be 63 % [8]. The mild form was most common representing 27 % of the cases. The cognitively impaired with no dementia were more than those with dementia contributing 43 % and 20 % respectively of the stroke patient study population [8].

### *Rehabilitation*

In Mulago hospital, rehabilitation is one of the most important aspects in the care of stroke patients. The aim is to improve quality of life by reducing emotional, functional, cognitive, physical and communication disorders. Early mobilization and rehabilitation help and improve outcome. This is done on a daily basis on the Neurology unit. Physiotherapy maximizes functional recovery, occupational therapy is necessary for functional assessment and the provision of practical aids. Speech and language therapy helps with aphasia, dysarthria and dysphagia. Psychiatric consultation is routinely done for those found to be depressed.

## Prevention of Stroke

### *Lifestyle Measures*

These include the four-by-four action plan approach. (1) Diet entails eating fruits and vegetables, reducing salt, sugar and animal fat intake. (2) Increased physical activity is emphasized as this maintains the ideal body weight. (3) Moderation of alcohol intake and (4) stopping smoking are all important in both primary prevention of stroke at community level and secondary prevention when the stroke/transient ischaemic attack has occurred.

### *Secondary Prevention*

Stroke recurrence occurs in some patients admitted to Mulago hospital and attempts are routinely made to stop this using the following measures:

**Antiplatelete Drugs**
Low dose daily aspirin 75–150 mg decreases the risk of another ischaemic stroke. Clopidogrel 75 mg daily and dipyridamole are the alternative drugs in patients with aspirin intolerance. Antiplatelet therapy has to be continued indefinitely.

**Anticoagulants**
All ischaemic stroke patients presenting with atrial fibrillation or mitral valve disease are anticoagulated indefinitely with warfarin with a target International Normalized Ratio of 2.5, as prophylaxis or using aspirin if there is no contraindication to it. The annual risk of embolism with either valvular heart disease or atrial fibrillation is reduced by anticoagulating with warfarin.

**Antihypertensives**
Treatment of hypertension significantly reduces the risk of strokes. Blood pressure treatment is started in hypertensive stroke patients and continued indefinitely to be monitored on discharge from hospital.

**Statins**
Cholesterol lowering drugs, simvastatin 20–40 mg per os daily or another statin therapy for raised cholesterol levels prevents stroke in patients with a history of cerebrovascular disease and ischaemic heart disease.

**Carotid Stenosis**
Asymptomatic carotid stenosis of >70 % and symptomatic stenosis of <70 % are managed medically.

## Prognosis

The outcome for stroke patients is poor. The mortality within the first year is over 30 %, with a further one third disabled and about one third regaining independent living. The majority of deaths occur during the first week and month after the stroke and continues throughout the first year.

The 30-day mortality among adult patients with stroke admitted at Mulago hospital is 43.8 % [14]. This is similar to other early stroke case-fatality findings observed in African hospital-based studies [7, 19, 20]. It was significantly associated with older age, leucocytosis, impaired level of consciousness and high body temperature [14].

More patients with subarachnoid haemorrhage or intracerebral haemorrhage die within 30 days than do those with cerebral infarction. The risk of recurrence continues over time.

## References

1. WHO MONICA Project. Monitoring trends and determinants in cardiovascular disease: a major international collaboration. J Clin Epidemiol. 1988;41:105–14.
2. Matovu SM, Nakibuuka J, Mukisa R, Musubire A, Kaddumukasa M. Stroke burden on the Neurology Unit in Mulago Hospital, Uganda. 2012.
3. Nakibuuka J, Abwooli N, Namale A, Blondin N, Ddumba E. A descriptive epidemiological study on stroke in Kampala, Uganda: a hospital-based study. Afr J Neurol Sci. 2012;31(1):41–48.
4. Ntep-Gweth M, Zimmermann M, Meiltz A, Kingue S, Ndobo P, Urban P, Bloch A. Atrial fibrillation in Africa: clinical characteristics, prognosis, and adherence to guidelines in Cameroon. Europace. 2010;12(4):482–7.
5. Sliwa K, Wilkinson D, Hansen C, Ntyintyane L, Tibazarwa K, Becker A, Stewart S. Spectrum of heart disease and risk factors in a black urban population in South Africa (The Heart of Soweto Study): a cohort study. Lancet. 2008;371(9616):915–22.
6. Matenga J. Stroke incidence rates among black residents of Harare – a prospective community based study. S Afr Med J. 1997;87:606–9.
7. Rosman KD. Epidemiology of stroke, urban black population. Stroke. 1986;17:667–9.
8. Mukisa R, Ddumba E, Musisi S, Kiwuwa MS. Prevalence and types of cognitive impairment among patients with stroke attending a referral hospital in Uganda. Afr J Neurol Sci. 2011;30(2):56–63.

9. O'donnell MJ, Xavier D, Liu L, Zhang H, Chin SL, Rao-Melacini P, Rangarajan S, Islam S, Pais P, INTERSTROKE investigators, et al. Risk factors for ischaemic and intracerebral Haemorrhagic stroke in 22 countries (the INTERSTROKE study): a case-control study. Lancet. 2010;376:112–23.
10. Onwuchewa A, Bellgam H, Asekomeh G. Stroke at the university of port Harcourt teaching hospital, rivers state, Nigeria. Trop Doct. 2009;39(3):150–2.
11. Sagui E, Saliou PM, Dubecq C, Khadi BF, Niang A, Gning S, Jean-Pierre B, Sane M, Debonne JM. Ischemic and hemorrhagic strokes in Dakar, Senegal. A hospital based study. Stroke. 2005;36(9):1844–7.
12. Williams C, Shephard T. A brief descriptive analysis of stroke features in Virginia. Neuroepidemiology. 2003;22(1):31–6.
13. Ojemann R, Heros R. Spontaneous brain hemorrhage. Stroke. 1983;14:468.
14. Kwarisiima L, Mukisa R, Nakibuuka J, Matovu S, Katabira E. Thirty-day stroke mortality and associated clinical and laboratory factors among adult stroke patients admitted at Mulago Hospital, Uganda. AJNS.2014;33(1):79–86.
15. Ogun SA, Oluwole O, Ogunseyinde AO, Fatade B, Odusote KA. Misdiagnosis of stroke – a computerized tomography scans study. West Afr J Med. 2000;19(1):19–22.
16. Nyame PK, Jumah KB, Adjei S. Computerised tomography scan of the head in the evaluation of stroke in Ghanaians. East Afr Med J. 1998;75:17–9.
17. Adams GF, Hurwitz LJ. Cerebrovascular disability in the aging brain. Edinburgh/London: Churchill Livingstone; 1974.
18. Adams GF, Hurwitz LJ. Mental barriers to recovery from strokes. Lancet. 1963;2(7307): 533–7.
19. Connor MD, Walker R, Modi G, Warlow CP. Burden of stroke in black populations in sub-Saharan Africa. Lancet Neurol. 2007;6:269–78.
20. Garbusinski JM, van der Sande MA, Bartholome EJ, et al. Stroke presentation and outcome in developing countries: a prospective study in the Gambia. Stroke. 2005;36:1388–93.