

PART II – NEUROLOGICAL DISORDERS

CHAPTER 5
STROKE

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NEUROLOGY IN AFRICA

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CHAPTER 5

STROKE

Introduction

Stroke is a major medical disorder caused by interruption of blood supply to the brain which results in a loss of neurological function. It usually occurs suddenly without warning and frequently results in death or disability. The two main mechanisms are blocking of the arteries causing ischaemia and rupture of the arteries causing haemorrhage. The aim of management of stroke is to try to limit the area of damage in the brain, assist recovery and prevent any recurrence. The management of stroke in high income countries over the last two decades has witnessed marked improvement with the creation of dedicated stroke care units in hospitals and a more active approach to the management and prevention of stroke. This chapter presents an overview of the main characteristics of stroke. The student should aim for an overall understanding of stroke, including its increasing burden, main causes and prevention, and in particular to be able to diagnose and manage a patient presenting with stroke.

Definition: Stroke is a sudden neurological deficit lasting more than 24 hours with no explanation other than a vascular cause. If the patient recovers fully within 24 hours without any neurological deficit, then this is classified as a transient ischaemic attack (TIA).

Epidemiology

Stroke is the third most common cause of death worldwide after heart disease and cancer. It is reported to be the leading neurological cause of death in Africa (Chapter 3). The annual incidence of stroke in high income countries is 2-3 per 1000 persons and the prevalence reaches 0.5 to >1% of the population in older age groups (>65 yrs). A similar high incidence rate has been reported recently in one study in Tanzania. The overall prevalence of stroke is reported to be lower in Africa with age adjusted rates being less than half that in high income countries. However with increasing urbanization and life style changes the burden of stroke is steadily increasing and it is now one of the leading causes of neurological admissions and death in urban hospitals throughout Africa.

AETIOLOGY

Stroke occurs as a result of ischaemia or haemorrhage

Ischaemia

Ischaemia accounts for >80% of strokes worldwide and for 60-80% in Africa. Ischaemia is caused by thrombosis or embolism resulting in loss of blood supply to part of the brain (Fig. 5.1). Thromboembolism is the main cause and arises from atheromatous plaques situated in the major blood vessels in the neck and brain (Fig. 5.5). This is termed athero-thromboembolism. It results in occlusion of the arteries supplying the brain, most commonly the middle cerebral artery (Figs. 5.2 & 3). A smaller number of ischaemic strokes arise from occlusion of the small end arteries arising from the larger blood vessels deep within the brain. These are called lacunar strokes. Ischaemia is also caused by cardioembolism. This arises mainly from mitral valve disease, atrial fibrillation, cardiomyopathy and much less frequently, recent myocardial infarction. Less common causes of ischaemic strokes are sickle cell disease, HIV infection, vasculitis and venous sinus thrombosis.

Haemorrhage

About 10-20% of strokes worldwide are caused by haemorrhage. This percentage is higher in Africa (20-40%) probably because of the high burden of untreated or inadequately treated hypertension. Haemorrhagic stroke occurs when there is sudden release of blood into the brain. The main types are intracerebral haemorrhage (ICH) and subarachnoid haemorrhage (SAH) (Figs. 5.4 & 6). Hypertension is the major cause of ICH and is also a risk factor for SAH. The sources of bleeding in chronic hypertension are ruptured Charcot-Bouchard micro aneurysms which form on small perforating end arteries deep in the brain. Less common sources of bleeding are arteriovenous malformations (AVMs), tumours, trauma and amyloid. The most common site affected is the internal capsule area which usually results in a complete hemiparesis. When the source of bleeding is in the brain stem, or cerebellum there is usually quadriplegia with cranial nerve palsies, ataxia and coma. Subarachnoid haemorrhage (SAH) is mainly caused by a ruptured underlying intracranial saccular (berry) aneurysm arising from the circle of Willis and less frequently AVM.

Key points

- stroke is a leading cause of death in adults in Africa
- the main causes are ischaemia & haemorrhage
- ischaemia is caused by atheroma & embolism
- haemorrhagic strokes arise from ICH or SAH
- main causes are hypertension & aneurysms

Pathogenesis

When the blood supply to the brain is lost acutely either as a result of ischaemia or haemorrhage, a core area of the brain will undergo infarction/necrosis. This core area of the brain is irreversibly damaged. However in ischaemia, because of collateral blood supply, a surrounding area called a penumbra remains potentially viable for a limited time. This time period is usually about 3-6 hours, during which it will recover if the blood supply is restored. This is the target for the early treatment directed at decreasing thrombosis and improving blood supply. The swelling in the brain is caused by cytotoxic and vasogenic oedema as a result of infarction or haemorrhage.

This is frequently responsible for the clinical deterioration in the days immediately following on the acute stroke.

Vascular Risk Factors

The main risk factors for stroke are hypertension, atrial fibrillation, diabetes mellitus, smoking and lack of exercise (Table 5.1). Together, these account for over two thirds of all strokes and represent modifiable risk factors. The risk of stroke increases exponentially with age, with a much greater risk in the elderly population. Hypertension is the most important modifiable risk factor for both ischaemic and haemorrhagic stroke. The risk almost doubles with every 7.5 mm Hg rise in diastolic pressure even within the normal range of blood pressure. Established cardiovascular disease is an important risk factor for stroke, particularly a previous stroke or TIA, atrial fibrillation, rheumatic mitral valve disease and heart failure. Diabetes, high cholesterol and low density lipoproteins, sickle cell disease, the oral contraceptive pill, migraine and infections are all known risk factors for ischaemic stroke. The main modifiable life style risk factors include diet, salt intake, obesity, lack of exercise, cigarette smoking and increased alcohol consumption.

Table 5.1 Main risk factors for stroke

Risk factors	Relative degree of risk
ageing	highest
hypertension atrial fibrillation previous stroke or TIA	very high
ischaemic heart disease diabetes	high
life style: diet increased salt intake lack of exercise smoking alcohol	moderate
obesity	low

Key points

- age is the strongest non modifiable risk factor for stroke
- hypertension and AF are among the main modifiable risk factors in secondary prevention
- life style is the major modifiable risk factor in primary prevention

Main causes of stroke

- atheroma
- hypertension
- cardioembolism

CLINICAL PRESENTATION

The key features of a stroke are a sudden onset of a focal neurological deficit in a person who was previously well. Strokes occur more frequently at night and in the early morning. The clinical findings will depend on the type of stroke, the vascular site affected and the underlying cause. The most common presentations are a sudden, unilateral loss of power or sensation in an arm or leg or both, a loss of speech, vision or balance (Table 5.2). These help to localize the site of origin of the stroke. There are no features that can reliably distinguish between ischaemia and haemorrhage, although headache, vomiting, complete hemiparesis, reduced level of consciousness and severe hypertension are more common in haemorrhage. In subarachnoid haemorrhage (SAH), the onset is characterized by a new, sudden and severe headache with neck stiffness, usually without any focal neurological deficit, but alteration or loss of consciousness may be present. If the patient is unable to give a history, then the details should be obtained from a relative. The general examination should be directed at looking for the main underlying risk factors for stroke, including hypertension, atrial fibrillation, cardiac murmurs, carotid bruits and signs of systemic illness. CT imaging is usually necessary to distinguish between the two main types of stroke.

Table 5.2 Main clinical features of stroke

<p>sudden onset either all at once or over minutes or hours</p> <p>focal neurological symptoms and signs</p> <p>loss of neurological function</p> <p>motor loss: weakness of one side or part of one side of the body</p> <p>sensory loss: decreased sensation on one side or part of one side of the body</p> <p>aphasia: loss or impairment of speech, understanding, reading or writing</p> <p>visual: loss of vision to one side, hemianopia (patient usually unaware)</p> <p>other symptoms: altered consciousness, dysphagia, dysarthria, ataxia, diplopia, quadriparesis</p>
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Localization

Ischaemic strokes can be divided into anterior and posterior circulation strokes. Anteriorly the internal carotid artery (ICA) divides to form the anterior and middle cerebral arteries (ACA & MCA). Posteriorly the vertebral arteries join at the lower pons to form the basilar artery which in turn divides into two posterior cerebral arteries (PCA). The anterior and posterior circulations are joined in front by the anterior communicating artery and at the back by the posterior communicating artery to form the circle of Willis (Fig. 5.1). This ensures collateral circulation in the brain. The MCA supplies the anterior lateral two thirds of the brain and the ACA supplies the remaining medial two thirds. The PCA supplies the posterior one third or the occipital lobe. The brain stem and cerebellum are supplied in turn by the vertebral and basilar arteries. The most common sites affected are the MCA followed by the ACA, followed by lacunar and PCA (Table 5.3).

Table 5.3 Localization and strokes

Artery	Main clinical findings
Internal carotid artery	hemiplegia, (arm = face = leg) hemisensory deficit hemianopia
Anterior cerebral artery	hemiplegia, (leg > arm)
Middle cerebral artery	hemiplegia & numbness (face = arm > leg) aphasia <i>*(if the dominant hemisphere involved)</i> hemianopia sensory inattention <i>(if the non dominant hemisphere involved)</i>
Posterior cerebral artery	hemianopia
Lacunar	hemiplegia, (face = arm = leg) hemisensory, (face = arm = leg)
Vertebro-basilar arteries (brain stem)	dysphagia, dysarthria, hemiplegia/quadriplegia cranial nerve palsies ataxia

* left hemisphere is dominant in most (>90%) right handed persons and in approx 70% of left handed persons

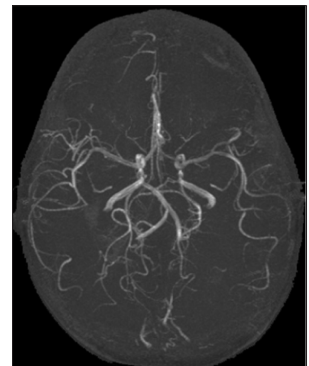
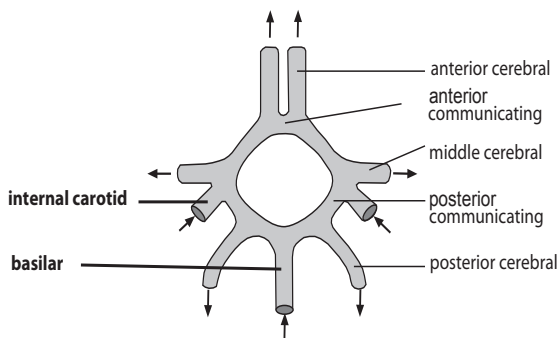


Figure 5.1 Circle of Willis. Angiogram of circle of Willis (COW). Normal (right).

Key points

- stroke is a sudden neurological deficit due to a vascular cause
- person is usually aware of a neurological deficit over minutes or less commonly hours
- hemiparesis is the most common finding
- neurological findings help to localize the site of the lesion
- most strokes occur in the anterior circulation

TRANSIENT ISCHAEMIC ATTACK (TIA)

A TIA is a sudden ischaemic focal neurological deficit that completely recovers in less than 24 hours. They typically last for minutes not hours. They are mostly caused by thromboemboli arising from the internal carotid arteries in the neck and their branches. Other sources of emboli are atrial fibrillation and heart disease. The vascular territory involved determines the

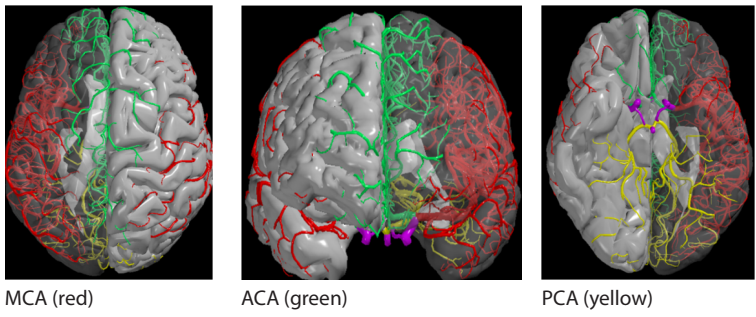


Figure 5.2 Territories supplied by main cerebral arteries

CT scans showing infarcts

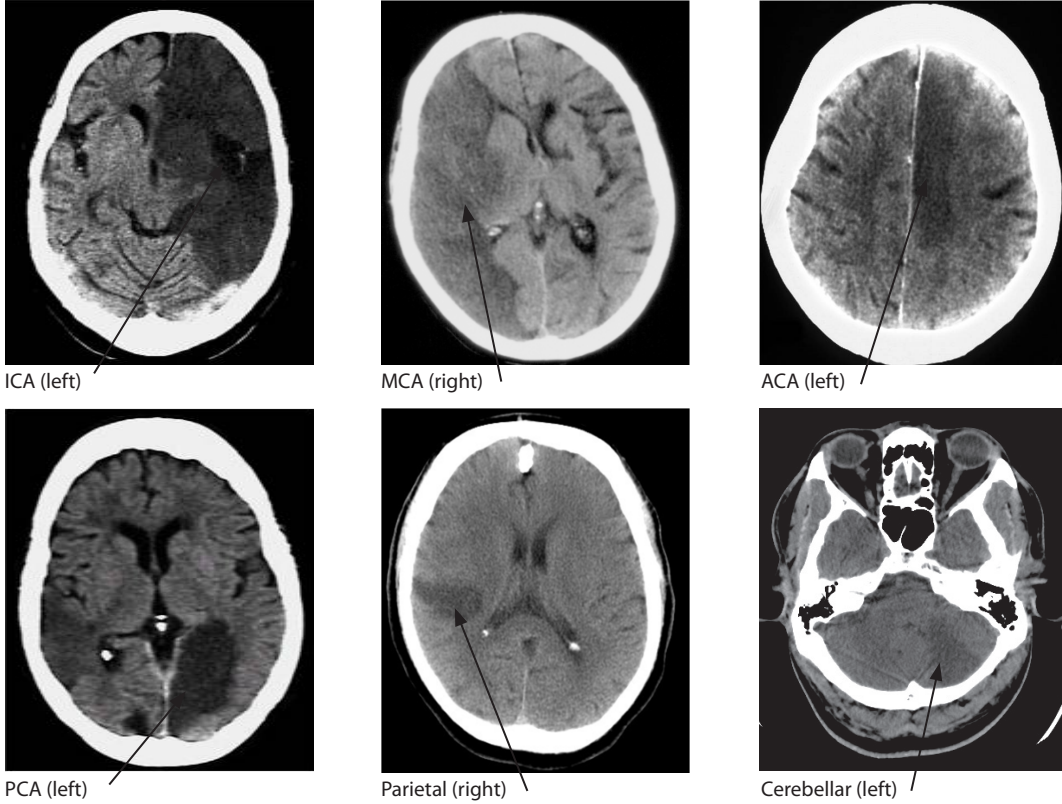


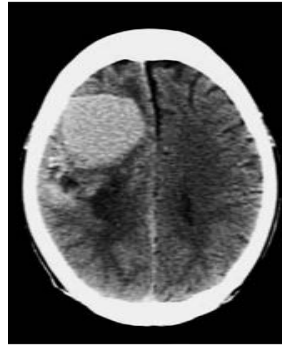
Figure 5.3 Infarction in territories of the main cerebral arteries

neurological findings and the presentations are similar to those already outlined for stroke but usually less severe. All TIAs should be investigated in a similar manner and with the same sense of urgency as stroke (Table 5.4). After a TIA the overall risk for stroke is about 10% per year, the greatest risk being in the days and weeks following the TIA. If the TIA lasts >90 mins in a person at risk, then the likelihood of a stroke is greatest (4-8%) within the next 48 hours and

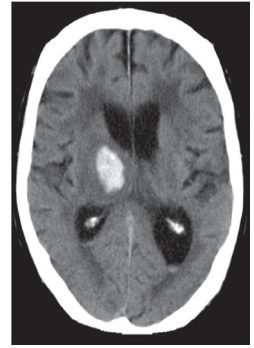
CT scans showing ICH



Frontal (right) & intraventricular



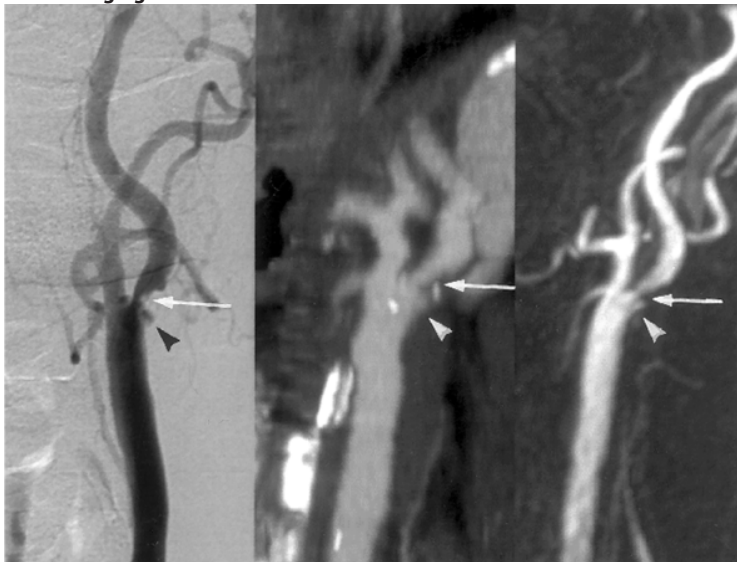
Frontal (right)



Internal capsule (right)

Figure 5.4 Intracerebral haemorrhage (ICH). CT scans showing ICH.

Carotid angiogram



Stenosis ICA

Figure 5.5 Internal carotid artery stenosis

the patient requires urgent hospital admission. The aim of investigations and management is to identify and modify preventable risk factors such as smoking, exercise, diet and alcohol and aggressively treat underlying diseases such as carotid artery stenosis (Fig. 5.5), hypertension, diabetes and atrial fibrillation (Table 5.1). Antiplatelet drugs and anticoagulants are used as in the prevention of stroke (Table 5.6).

Key points

- most TIAs last for minutes not hours
- if TIA lasts >1-2 hours, the risk of stroke is greatest over next 48 hours
- annual risk for stroke after a TIA is around 10%
- don't wait for stroke to happen
- manage & treat TIA as if it were a stroke

Differential diagnosis

A history of a sudden onset of focal neurological deficit is almost always diagnostic of stroke. The differential diagnosis includes other disorders presenting with similar acute or semi acute neurological presentations. These include opportunistic processes in HIV disease, subdural haematoma, mass lesions, Todd's paralysis after an unwitnessed seizure, and other causes of acute encephalopathy. However in these cases the correct diagnosis should be suggested by a different clinical history, sub-acute onset and progressive nature of the neurological deficit. Venous sinus thrombosis may present with a stroke but this is uncommon. The clinical context, usually a pre-menopausal female with typical fundoscopy changes of venous engorgement with haemorrhages should suggest the correct diagnosis. A history of recent head injury or fall suggests the possibility of subdural haematoma (SDH), although a history of trauma may be absent (Chapter 19). CT scan of the brain may be necessary to make the correct diagnosis. There are a number of other medical conditions that can mimic a stroke or TIA at onset; these include focal seizures, migraine, hypoglycaemia, syncope, and hysteria. These are usually self-limiting often with a history of similar previous episodes and have a normal neurological examination.

INVESTIGATIONS AND DIAGNOSIS

Stroke is a clinical diagnosis and investigations are directed at establishing the cause and preventing recurrences. The main investigations for stroke are outlined in Table 5.4. Computerised tomography (CT) of the head is the investigation of choice in stroke. Ideally this should be done within 24-48 hours of onset of the stroke. Its primary role is to rapidly exclude haemorrhage, thereby allowing the administration of an antiplatelet drug, usually aspirin. In addition, it can determine the nature, size and site of stroke and exclude other disorders. In haemorrhagic stroke, CT shows haemorrhage as a white or hyperdense area almost as soon as it occurs. In small bleeds, the white area persists for around 48 hours while larger bleeds may persist for 1-2 weeks. After two weeks a bleed becomes indistinguishable from an infarct on a CT. CT shows ischaemia as an ill defined dark or hypodense area but this can take 24-48 hours to appear on the scan. Not all infarcts show up on a CT because of decreased sensitivity, small size and also poor imaging of the posterior fossa. If the initial CT is normal and a stroke is still suspected then a scan repeated after 3-7 days may show an infarct. If the clinical diagnosis of a stroke is certain, then a repeat scan may be unnecessary. In SAH the CT is highly sensitive during the first few days, after which it becomes negative and the diagnosis is then confirmed by lumbar puncture showing altered blood or xanthochromia. Magnetic resonance imaging (MRI) is more sensitive than CT for detecting early and small vessel strokes.

Table 5.4 Investigations for stroke

Department	Investigation	Risk factor
Haematology	FBC, ESR, sickle cell test	anaemia, polycythaemia, infection, vasculitis, sickle cell disease
Biochemistry	blood glucose, creatinine, electrolytes, liver function tests, lipids	diabetes, renal disease, hyperlipidaemia
Serology	HIV, VDRL	infections
Microbiology	malaria parasites, blood culture (if febrile)	infections
Cardiology	ECG	atrial fibrillation, myocardial infarction
Imaging	chest X-ray CT/MRI of head	cardiomegaly, hypertension ischaemia or haemorrhage
Ultrasound	echo heart (if cardiac origin suspected) carotid doppler (if carotid origin suspected)	mitral valve disease, thrombus, endocarditis large vessel atheroma

Key points

- CT head may be necessary in suspected stroke to make the correct diagnosis
- CT scan can detect over 90% of all strokes
- CT scan done during the first 24-48 hours after onset of stroke may miss ischaemia
- CT scan done during the first 1-2 weeks after onset of stroke usually confirms haemorrhage

SUBARACHNOID HAEMORRHAGE

SAH is the term usually reserved for spontaneous or non traumatic bleeding into the subarachnoid space which occurs as a result of a ruptured saccular (berry) aneurysm or an arteriovenous malformation (AVM) (Fig. 5.6). The majority (75-80%) occur as a result of bleeding from a ruptured saccular aneurysm. These aneurysms arise mainly at the junctions of the arteries that form the circle of Willis in the subarachnoid space at the base of the brain. In 5-10% of cases the SAH arises from an AVM and in a small percentage of cases no cause is found. SAH occurs in 5-10/100,000 persons per year in the UK. The frequency of SAH in SSA is not known but it may be more common there. A patient presenting with SAH is usually younger than in other types of haemorrhagic stroke (ICH) and first degree relatives are at an increased risk of stroke. Other risk factors for SAH include hypertension and a history of smoking.

Clinical presentation

The main clinical feature of SAH is a sudden explosive severe headache described as “like being hit on the back of the head with a hammer.” There may be a prior history of sentinel or warning headaches for some weeks beforehand. The suddenness of onset helps to differentiate it from the pain of meningitis. The headache in SAH is usually accompanied by nausea, vomiting, fever, meningism and variable loss of consciousness. The loss of consciousness typically occurs at the moment of the bleed. The clinical findings vary from a fully alert patient with severe headache and meningism to a deeply comatose patient with decerebrate rigidity. Blood pressure is frequently elevated, mostly as a result of the SAH. Focal neurological signs and deficits may occur as a result of raised ICP, ICH or compression from the aneurysm. These include 3rd nerve

palsy, 6th nerve palsy, hemiparesis, bilateral extensor plantar responses and papilloedema with or without subhyaloid haemorrhages (10-20%). Focal neurological deficits are more common in ruptured AVMs.

Investigations

CT of the head is highly sensitive for SAH with >90% of patients showing evidence of fresh blood in the subarachnoid space or ventricles (Fig 5.6). This lasts for 24-48 hours after which it becomes negative. CT may not show evidence of an aneurysm unless it is large but will usually show evidence of an AVM especially if contrast is given. In a patient with suspected SAH if a CT is normal or unavailable then it is necessary to do lumbar puncture (LP) and check for fresh uniformly mixed blood which fails to clear in all 3 consecutive CSF samples. The opening CSF pressure may be elevated in SAH. Xanthochromia occurs when the CSF is uniformly straw or yellow in colour. This is due to the presence of degraded blood in the CSF which is older than 24 hours and it persists for up to 2 weeks. The CSF may be entirely normal if examined within the first few hours or later than 2 weeks after the bleed.

Key points

- most common cause of SAH is a ruptured saccular aneurysm
- diagnostic feature of SAH is a sudden severe explosive headache
- meningism is a key feature of SAH
- level of consciousness in SAH ranges from being fully alert to deep coma
- diagnosis confirmed by finding evidence of blood either on CT or in CSF

Management

SAH carries a very high mortality during the first few days and if left untreated, there is a significant risk of rebleeding (20-30%) over the next 6 weeks. Management of acute SAH is therefore directed towards immediate treatment and the prevention of further bleeding. Patients should be nursed in bed with the head elevated 10-20 degrees, resting in quiet surroundings with adequate analgesia to avoid pain and surges in blood pressure. Aspirin should be avoided and constipation prevented. Intravenous hydration should be with approximately 3 litres per day of normal saline to avoid hypovolaemia. Antihypertensive medications should be avoided to prevent hypotension. In order to reduce arterial vasospasm and cerebral infarction secondary to the irritative effect of blood on vascular smooth muscle, the calcium channel blocker nimodipine 60 mg 4 hourly is prescribed for 3 weeks. Seizures occur in approximately 10% of patients and usually respond to the phenytoin 300 mg daily after a loading dose of 900 mg.

Neurosurgical

Neurosurgical intervention is indicated for SAH patients who are fully conscious or mildly confused with minimal or no neurological deficits. Patients with altered level of consciousness, coma or focal neurological signs usually do not benefit from neurosurgical intervention. The overall aim of neurosurgical intervention aim is to occlude the ruptured aneurysm. This can be achieved by either a neurosurgeon placing a clip over the neck of the aneurysm or by the neuroradiologist endovascularly embolising the aneurysm by packing it with metal coils. The latter is now the preferred method for the occlusion of most aneurysms. The optimum time for neurosurgical management is within the first 3 days after the initial bleed although the aneurysm can be operated on or coiled later.

CT (without contrast)



Blood in the subarachnoid space

Magnetic resonance angiogram COW



Aneurysm in left posterior cerebral artery

Figure 5.6 Subarachnoid haemorrhage & aneurysm**Prognosis**

The case fatality rate for patients presenting with SAH due to aneurysms is high with >10% mortality within the first few days either as a result of the initial haemorrhage or its early complications. Of all those patients that do survive the initial bleed and do not have neurosurgical intervention, one third die within 3 months, one third go on to make a good recovery and one third are left with permanent neurological disability. Case fatality rates for patients presenting with SAH secondary to AVM are lower at around 10%.

Key points

- main aim of treatment is to prevent another bleed
- nimodopine helps to reduce vasospasm which may worsen the neurological deficit definitive surgical management is by either coiling or clipping the aneurysm
- overall mortality in SAH is high

MANAGEMENT

The overall aim of stroke care is to decrease morbidity and mortality, to optimise recovery of function and to prevent further strokes. This can be achieved by good nursing care, specific stroke treatment, maintenance of fluid and electrolytes, nutrition, avoiding systemic complications and early rehabilitation. The outcome improves when stroke care guidelines are followed and care takes place in a defined area in hospital by a dedicated team. In Africa hospital care starts usually with admission to a general medical ward. Management includes general (Table 5.5) and specific measures (Table 5.6).

Table 5.5 General measures in caring for acute stroke patient**1. Start neurological observations hourly and change to 4 hourly if stable**

- level of consciousness using GCS
- vital signs
- oxygen saturation

2. Monitor blood glucose (if >11 mmol/L start insulin sliding scale)

intravenous fluids in dehydrated patients, unable to swallow

<p>4. Evaluate swallowing after 24 hours</p> <ul style="list-style-type: none"> • observe the patient attempting to swallow sips of water in upright position • check for coughing or gagging • if swallowing impaired keep nil per oral (NPO) • continue iv fluids for 48 hours then start nasogastric tube feeding if still unable to swallow
<p>5. Urinary catheterization if incontinent or in retention</p>
<p>6. Prevent constipation by adequate hydration and laxatives</p>
<p>7. Prevent pressure sores by supervising 2 hourly turning*</p>
<p>8. Decrease the risk of deep vein thrombosis (DVT) by using compression stockings in addition to oral aspirin if ICH excluded</p>

** this is done best by a relative or carer permanently at the bedside*

Specific measures

Antiplatelet drugs

All ischaemic strokes should have aspirin immediately or as soon as possible after onset followed by long term treatment (Table 5.6). Ideally, haemorrhage should be first excluded on CT. If a CT scan is unavailable, then aspirin should be used cautiously in the first two weeks and then only in cases strongly suspected of having ischaemic stroke. Aspirin when given effectively prevents 15 deaths or major disability for about every 1000 patients treated during the first few weeks and prevents about a fifth of recurrent strokes when used longer term. There is a slight increased risk of gastro-intestinal haemorrhage. The dose is 300 mg po daily for the first 2 weeks followed by 75-150 mg po daily thereafter. Patients that are intolerant of aspirin should be treated with either clopidogrel or dipyridamole. Combination therapy with both aspirin and clopidogrel is increasingly used in acute stroke patients.

Blood pressure

Blood pressure (BP) rises after an acute stroke and tends to fall spontaneously after that. The modern management is to avoid lowering blood pressure during the first 24-48 hours as an acute drop in BP can reduce perfusion to an already ischaemic brain. Consider treatment only if BP is persistently elevated (Table 5.6). The upper limit of persistently elevated blood pressure in ischaemic stroke is systolic 180 mm Hg and diastolic 105 mm Hg. The aim is a daily reduction of 10-20 mm Hg. Lower levels should not be treated in the first 48 hours unless complicated by hypertensive encephalopathy, left ventricular failure or myocardial infarction. In ICH, the threshold for starting treatment is lower (>160/100, Table 5.6). If BP needs to be treated in the acute phase, consider using nifedipine sublingually for acute reduction and then orally twice daily. Other options include captopril for a more gradual reduction or atenolol and/or hydralazine.

Anticoagulation

Patients with a proven ischaemic stroke and a cardiac embolic source or atrial fibrillation should be anticoagulated to prevent further strokes (Table 5.6). Patients should be first treated with aspirin and anticoagulation be delayed for 2 weeks after the stroke because of the risk of intracerebral haemorrhage. Warfarin is then the drug of choice in a loading dose, usually 10 mg daily for 2 days followed by daily dose, depending on the prothrombin time or international normalized ratio (INR). The aim is to have and maintain an INR of 2-3 or a prothrombin

time of twice the normal range. In patients with mechanical valve prosthesis the target INR is 2.5-3.5.

Thrombolysis

This is a recent development in stroke management and dramatically improves the outcome in some ischaemic stroke patients. Thrombolytic therapy is with alteplase, an iv tissue recombinant plasminogen activator (rtPA), a thrombolytic agent. This is beneficial in some patients with ischaemic strokes who have no early CT evidence of completed infarction or bleed. It has to be given as soon as possible after the onset of the stroke usually within 3 hours or 6 hours at maximum. Any later treatment has increased risk of bleeding and a lack of efficacy. However implementation requires a trained medical team on call and emergency CT scanning facilities. Currently <5% of all stroke patients with access in high income countries are treated with thrombolysis.

Table 5.6 Drug treatments in stroke

Measure	Treatment	Dose	Side effects
Blood Pressure (Indication for Rx in 1st 48 hours) >180/105 in infarction >160/100 in haemorrhage	nifedipine	10 mg/sublingually stat 10/20 mg/po/bd	hypotension headache
	captopril	6.25/12.5 mg/po/bd	cough, allergy
	hydralazine	25/50 mg/im/po/tid	rash
	atenolol	50/100 mg/iv/po/od	asthma, depression, hypoglycaemia
Antiplatelet drugs Indication: ischaemia	aspirin	300 mg/po/stat 75-150 mg/po/od	indigestion, nausea, GI bleeding
	clopidogrel	75 mg/po/od	indigestion diarrhoea
	dipyridamole	200 mg/po/bd	headache, indigestion
Anticoagulation Indication: risk of cardiac embolism	warfarin	10 mg/po/od/for 2 days 1-5 mg/po/od (according to INR)	bleeding

Key points

- acute ischaemic strokes should have aspirin immediately
- avoid aggressively lowering blood pressure during the first 24-48 hours of acute stroke
- after first 48 hours, all persistently elevated blood pressures should be lowered
- embolic strokes should be anticoagulated, but not for two weeks after onset of stroke

COMPLICATIONS

Stroke patients are at risk for complications which may lead to death. Neurological worsening is common in the first 48 hours of stroke as a result of brain swelling, extension of the original stroke and complications. Patients with coma, extensive stroke and large haemorrhage have a poor prognosis.

Acute

The main acute complications are aspiration pneumonia, pulmonary embolism (PE), pressure sores and urinary tract infections. These occur in over half of hospitalized stroke patients and are associated with a poor prognosis. Pneumonia is the main cause of death in stroke in

hospitalized patients. This occurs as a result of aspiration and is more frequent in patients with extensive strokes and coma. Management includes avoiding oral intake, chest physiotherapy and early antibiotics. Patients with stroke are at significant risk for DVT and PE. The use of prophylactic low dose aspirin and compression stockings decreases this risk. Heparin is contraindicated in the first 2 weeks after stroke as it increases intracerebral bleeding. Pressure sores, spasticity and contractures are common after a stroke and are reduced by early patient positioning, 2 hourly turning, passive exercises and limb splinting.

Chronic

Long term complications include spasticity, contractures, pain, depression, dementia and late onset seizures. Post stroke depression is common occurring in over half the patients. It is important to recognize it and if necessary offer treatment with tricyclics or selective serotonin reuptake inhibitors. Dementia as a result of stroke is common and is a major long term cause of dependency, particularly in the elderly. Seizures occur in about 2% of acute stroke patients but usually resolve in a few weeks and don't require long term treatment with anticonvulsants. Late onset seizures (6–12 months after stroke) occur in around 5% of stroke patients and are persistent, but they respond well to phenytoin.

Rehabilitation

Rehabilitation is one of the most important aspects in the care of stroke patients. Early mobilization and rehabilitation have been shown to help and improve outcome. This should take place on a daily basis in the general medical ward or in a specialized stroke area. Physiotherapy maximises functional recovery, occupational therapy is necessary for functional assessment and the provision of practical aids and speech and language therapy helps with aphasia, dysarthria and dysphagia.

Palliative care

Many stroke patients have no hope of recovery, and the best management is to ensure their comfort and avoid any unnecessary investigations and further suffering. It can be very distressing for family to witness a dying patient with noisy and laboured breathing because of retained airway secretions. Care is best achieved by good nursing and adequate palliative analgesia. It is important to explain to family and carers what is happening and many will at this stage choose to care for the patient at home.

Key points

- main acute complications are aspiration pneumonia & pressure sores
- pneumonia is a leading cause of death in stroke
- early mobilization and rehabilitation are critical to recovery
- long-term complications are disability, pain, depression, dementia & seizures
- palliative care is important where recovery is unlikely

PREVENTION

Antiplatelet drugs

Low dose daily aspirin 75-150 mg decreases the risk of another stroke by about one fifth in ischaemic strokes. About 10% of patients don't tolerate aspirin because of gastrointestinal side effects, mainly indigestion, nausea and rarely bleeding. This can be decreased by the concomitant use of a proton pump inhibitor and by using alternative antiplatelet drugs. Clopidogrel 75 mg daily is the drug of first choice in patients with aspirin intolerance but is more expensive. The combination of clopidogrel and aspirin is considered more effective than aspirin alone but has an increased risk of bleeding. Dipyridamole may also be used. Antiplatelet therapy has to be continued indefinitely.

Anticoagulants

The annual risk of embolism with either valvular heart disease or atrial fibrillation is around 10% per year without anticoagulation. Anticoagulation with warfarin decreases this risk very significantly by >50% per year. All ischaemic stroke patients presenting with atrial fibrillation or mitral valve disease should be anticoagulated indefinitely unless there is a contraindication.

Blood pressure

Treatment of hypertension significantly reduces the risk of strokes. There is strong evidence that lowering blood pressure, irrespective of the previous baseline level down to 130/70 reduces the risk of stroke. A mean drop of 9 mm Hg systolic and 4 mm Hg diastolic reduces the relative risk of stroke by about a quarter. Blood pressure treatment should be started in hypertensive stroke patients 48 hours after onset of the stroke and continued and monitored on discharge from hospital.

Carotid Stenosis

Athero-thromboembolism arising from the carotid and vertebral arteries is the main cause of ischaemic stroke in high income countries. Symptomatic carotid stenosis of >70% is an indication for carotid surgery wherever this is available (Fig. 5.5). Asymptomatic carotid stenosis of >70% and symptomatic stenosis of <70% are managed medically. However atheroma arising specifically from the carotids appears to be an uncommon source of ischaemic stroke in Africa.

Other factors

Life style measures such as salt reduction, low animal fat diet, decreasing alcohol consumption, stopping smoking and increasing exercise are all very important in both primary prevention of stroke at community level and secondary prevention when the stroke/TIA has occurred. Education is vital to the successful implementation of these measures. The use of cholesterol lowering drugs, simvastatin 20-40 mg po daily or another statin has been shown to decrease coronary events and recurrent strokes and these should be prescribed if possible after an ischaemic event. The expected relative risk reduction is in the order of 20%. However, the statins are expensive and have side effects including myalgia, myositis and liver dysfunction, which can lead to them being discontinued in about 10% of patients. An elevation of creatine kinase (CK) occurs in many individuals on statins and values up to 1000 IU can be tolerated. Adequate long term control of diabetes is important.

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 continue throughout the first year. The risk of recurrence continues over time. Over half of all stroke survivors are dead within 5 years. The long term prognosis for stroke is probably worse in Africa because of the lack of secondary and tertiary care.

Prognosis of stroke after one year

- one third die
- one third are disabled and dependent
- one third are independent

The future

The majority of strokes occur in low and middle income countries where most of the world's population live. With increasing urbanization, this burden is set to continue and increase over time. In low income countries especially, the majority of people have limited or no access to facilities for the prevention and management of stroke. These limitations extend from a lack of awareness to lack of treatment, rehabilitation and prevention. These in turn are related to a lack of trained specialists, education, resources and research. Clearly there is a need for more research and intervention particularly in the area of primary and secondary prevention of strokes in low and middle income countries, not forgetting the very important link between patient care and primary prevention of stroke in the community.

Key points

- prevention of stroke is now a top priority in Africa
- primary prevention must include public education concerning necessary lifestyle changes
- includes decreasing total salt intake, stopping smoking, dietary changes & increasing exercise
- secondary prevention includes targeting known diseases & risk factors for stroke
- tertiary measures are important to cope with increasing burden of death & disability

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