EPIDEMIOLOGY, DIAGNOSIS, MANAGEMENT AND PREVENTION OF STROKE: A NARRATIVE REVIEW

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ABSTRACT

Introduction: **Stroke is a major public health problem globally, and surviving patients have very high risks of recurrence. Age, sex, ethnicity, transient ischaemic attack(TIA), and hereditary characteristics are non-modifiable risk factors while the major modifiable risk factors for stroke are hypertension, diabetes, lack of physical exercise, alcohol and drug abuse, cholesterol, diet management and cardiac diseases.**

Objective: **The aim of this narrative review was to discuss the current management and prevention of stroke.**

Methods: **In this review, the databases of Medline (PubMed), Scopus, Web of Science and Google Scholar were used for literature search. The MESH terms used included "Diagnosis of Stroke", "Epidemiology of Stroke", "Management of Stroke" and "Prevention of Stroke". The other terms were "Stroke Mimicks" and "Biomarkers of Stroke". Studies that evaluated the current management of stroke in adult patients were included. This article is guided by the Scale for the Assessment of Narrative Review Articles (SANRA).**

Results: **A total of 800 studies were found. After excluding duplicate papers and applying the inclusion and exclusion criteria, 72 studies were included for review. The included studies were published between 2010 and 2025. These studies included 9 Randomised Controlled Ttrials, a cohort study and other studies.**

Conclusions: **Computed tomography (CT) scan of the brain is the most common form of clinical imaging procedure used in the treatment of stroke and Non-contrast CT Scans are usually the first step in imaging process. Diffusion-weighted magnetic resonance imaging (MRI) is the most sensitive imaging technique, it shows ischaemic changes very early and is effective in the evaluation of cerebral changes in ischaemic stroke. Magnetic resonance imaging is more precise in excluding intracranial hemorrhage and MRI with DWI is more accurate in the revealing acute ischemic stroke. Stroke treatment mainly focuses on restoring blood flow to the brain and treating stroke-induced neurological injury.**

Keywords: Diagnosis, Epidemiology, Management, Prevention, Stroke mimicks, Biomarkers

INTRODUCTION

Stroke is a major public health problem globally, and a recurrence rate of 19.5% has been reported in a community study conducted in Rotterdam.[1] It is the third-largest cause of death in England.[2] The World Health Organization defines stroke as "rapidly developing clinical signs of focal (at times global) disturbance of cerebral function, lasting more than 24 hours or leading to death with no apparent cause other than that of vascular origin".[3] However, the American Stroke Association has developed a new definition of stroke which besides clinical definition includes "infarction of the brain, spinal cord, or retinal cell death due to ischemia, based on pathological, imaging, or other objective evidence of cerebral, spinal cord, or retinal focal ischemic injury in a definite vascular distribution".[4]

The majority of the Stroke cases are ischaemic (85%) while others (15%) are haemorrhagic.[5] In haemorrhagic stroke, following the break in the integrity of the wall

of an artery in the brain, there is consequent increase in the intracranial cavity volume due to the haemorrhage over time leading to an elevated intracranial pressure.[6] In ischaemic stroke, there is occlusion of a cerebral artery, followed by a reduction in blood flow to the distant tissue and subsequent ischaemic necrosis of the compromised cells in a few hours. The brain tissue is intricately sensitive to ischemia, and about 1.9 million neurons die every minute that blood is not supplying the brain tissue. However, optimal collateral circulatory competence was reported to be associated with slower infarct growth rates and prolongation of the therapeutic window.[7] Age, sex, ethnicity, transient ischaemic attack(TIA) and hereditary characteristics are non-modifiable risk factors while the major modifiable risk factors for stroke are hypertension, diabetes, lack of physical exercise, alcohol and drug abuse, cholesterol, diet management and cardiac diseases.[8]About 30% of patients die within a year of developing a stroke, with 50% of survivors suffering from a long-term disability. Alteplase treatment within six hours after ischaemic stroke was associated with a non-significant decrease in risk of death at three years. However, among patients who survived the acute phase, treatment was associated with increased long term survival which is reassuring for clinicians in doubt of benefits of thrombolysis.[9]

Epidemiology

The prevalence of stroke is highest in developing countries, with ischaemic stroke being the commonest.[10] The crude prevalence of stroke was 1.99 in males and 1.28 in females per 1000 population in a rural area study in Nigeria.[11] In another rural study in Nigeria, the crude prevalence was 8.5 per 1000 involving more than 1000 participants.[12] The prevalence was higher in men, increased with age and hypertension was a risk factor.[12] In a systematic review of stroke in Sub-Saharan Africa, the highest prevalence was reported to be 48.17 per 100 in Madagascar, while the lowest was recorded in Zimbabwe with 0.61 per 100.[13] In a study conducted in Ghana, the incidence rate of stroke was higher in males than females and the incidence increased with age.[14] In the United States, more than 795,000 people experience a stroke yearly.[15] Stroke is ranked as the second leading cause of death globally and around 5.5 million die annually of stroke.[16]

Though the prevalence of stroke is higher in older people, the prevalence has increased in the younger people over the years.[17] The predisposing factors to higher risk of stroke in younger women compared with younger men are preeclampsia, contraceptive use and hormonal therapy, as well as migraine with aura.[18] Brain infarction and intra-cerebral hemorrhage are commoner in older men but cardio-embolic stroke is commoner in older females.[19] Tobacco smoking, excessive alcohol consumption, myocardial infarction and arterial disorders are risk factors of stroke in men.

Pathophysiology of ischaemic stroke

Ischaemic stroke results from a sudden interruption of cerebral blood flow, typically due to arterial occlusion by a thrombus or embolus. This leads to a rapid decline in oxygen and glucose delivery to affected brain regions, initiating a cascade of metabolic and cellular disturbances. Energy failure from ATP depletion impairs ion pump function, causing neuronal depolarisation, intracellular calcium overload, and excessive release of excitatory neurotransmitters, particularly glutamate thereby triggering excitotoxicity. These events contribute to oxidative stress, mitochondrial dysfunction, and activation of apoptotic and necrotic pathways, culminating in cell death within the infarct core. Surrounding this core lies the ischaemic penumbra, a zone of functionally impaired but potentially viable tissue, which remains salvageable with timely reperfusion. Secondary injury mechanisms, including inflammation, leukocyte infiltration, and disruption of the blood-brain barrier, further propagate neural damage and cerebral oedema.[20,21,22]

Pathophysiology of haemorrhagic stroke

Haemorrhagic stroke is characterised by the extravasation of blood into the brain parenchyma (intracerebral haemorrhage) or the subarachnoid space (subarachnoid haemorrhage), most commonly resulting from chronic hypertension, cerebral aneurysm rupture, arteriovenous malformations, or trauma. The accumulation of blood within the cranial vault exerts a mass effect, raising intracranial pressure and compromising cerebral perfusion. In addition to the primary mechanical injury, the presence of blood and its breakdown products initiates a robust inflammatory response, oxidative damage, and activation of microglia. These processes disrupt the blood-brain barrier, promote vasogenic oedema, and exacerbate neuronal injury. In severe cases, increased intracranial pressure can lead to cerebral herniation, which is often fatal without prompt neurosurgical intervention.[23] The extent of secondary injury is a critical determinant of clinical outcomes in haemorrhagic stroke. After a head injury or ruptured aneurysm, blood accumulates in the subarachnoid space in subarachnoid haemorrhage.[10] The degree of a stroke's impact is directly associated with the period of the blood supply interruption.[15]

The role of biomarkers in the pathophysiology of stroke

Biomarkers are increasingly recognised as essential tools for elucidating the molecular and cellular mechanisms underlying both ischaemic and haemorrhagic stroke.

They provide valuable insights into key pathophysiological processes such as neuronal injury, inflammation, oxidative stress, blood-brain barrier (BBB) disruption, and coagulation abnormalities. Beyond their mechanistic relevance, biomarkers hold promise for improving early diagnosis, guiding therapeutic interventions, and predicting clinical outcomes.

In ischaemic stroke, the abrupt cessation of cerebral blood flow initiates a cascade of neurotoxic events, many of which are reflected in the release or alteration of specific biomarkers. Glial fibrillary acidic protein (GFAP) and S100 calcium-binding protein B (S100B) are released from astrocytes and serve as indicators of glial damage and BBB disruption. Neuron-specific enolase (NSE) and ubiquitin carboxy-terminal hydrolase L1 (UCH-L1) reflect neuronal injury and are associated with infarct size and neurological deficits. Inflammatory mediators such as interleukin-6 (IL-6), tumour necrosis factor-alpha (TNF-α), and C-reactive protein (CRP) are elevated early after stroke onset and contribute to secondary injury via leukocyte activation and endothelial dysfunction. Matrix metalloproteinase-9 (MMP-9) plays a pivotal role in degrading the extracellular matrix and tight junction proteins, exacerbating BBB permeability and increasing the risk of haemorrhagic transformation.[24,25]

In haemorrhagic stroke, the extravasation of blood into brain tissue induces mechanical damage and a potent inflammatory response. Biomarkers such as GFAP, NSE, and hemoglobin degradation products can help differentiate haemorrhagic from ischaemic stroke in the hyperacute phase. Elevated levels of D-dimer, fibrinogen degradation products, and thrombin-antithrombin complexes reflect coagulopathy and ongoing vascular injury. Inflammatory and oxidative stress markers, including heme oxygenase-1 (HO-1) and myeloperoxidase (MPO), contribute to secondary injury by promoting microvascular dysfunction and neuronal apoptosis.

Emerging classes of biomarkers, such as circulating microRNAs, extracellular vesicles, and metabolomic signatures, offer novel insights into stroke pathobiology and show potential for enhancing diagnostic specificity and prognostic accuracy. Importantly, the integration of biomarker profiles into clinical practice could enable personalised risk stratification and targeted therapeutic strategies, particularly in the context of precision medicine.[26,27]

METHODS

In this review, the data bases of Medline (PubMed), Scopus, Web of Science and Google Scholar were used for literature search. The MESH terms used included "Diagnosis", "Epidemiology", "Management" and "Prevention of stroke". The other terms were "Stroke mimicks" and "biomarkers". Studies that evaluated the current management of stroke in adult patients and studies published in English were included. This article is guided by the Scale for the Assessment of Narrative Review Articles. Excluded were studies not published in English and those that involved children.

RESULTS

A total of 800 studies were found. After excluding duplicate papers and applying the inclusion and exclusion criteria, 72 studies were included for review. The included studies were published between 2010 and 2025. These studies included 9 Randomised Control Ttrials, a cohort study and other studies.

DIAGNOSIS

Clinical presentation

Accurate diagnosis and classification will assist physicians in management and prediction of outcome in stroke cases. The National Institute for Health and Clinical Excellence (NICE) in United Kingdom recommends the use of a validated recognition tool called 'Recognition of Stroke In the Emergency Room' (ROSIER) for a rapid diagnosis of stroke. Its' sensitivity is 93%. The components include blood sugar, visual field, documentation of a history of seizures, level of consciousness, and also focal neurological weakness and speech problems.[28] NICE also recommended face, arm, speech test (FAST) by paramedics as a screening tool for stroke. The test assesses facial weakness, arm weakness, and speech disturbance in patients with str0oke, and it has a positive predictive value of ~80%. However, it does not take pre-existing disability into account, has a low sensitivity towards posterior circulation events and some of the differentials of stroke may be FAST positive, for instance Todd's paresis.[13]

The neurological symptoms depend on the site and the size of the lesion. Larger infarcts are associated with worse prognosis. The symptoms include hemiparesis, sensory dysfunction, dysarthria, facial asymmetry and impaired vision. The Modified Rankin scale is a clinician-reported measure of global disability and the most common outcome measure in stroke management.[15] The level of function is usually graded between 0 and 6 on the modified Rankin scale (mRS) score, with 0 signifying normal functional status and 6 showing lethal outcome. Patients that maintain self-care for activities of daily living corresponds to an

mRS score of 0–2. A score of 0–1 is usually classified as a perfect functional outcome.[15]

Clinical features of ischaemic stroke

Sudden weakness or numbness

One of the hallmark signs of an ischaemic stroke is sudden weakness or numbness, often localized to one side of the body. Affected individuals may experience facial drooping, difficulty lifting one arm, or weakness in a leg, leading to impaired mobility.

Speech difficulties

Patients may exhibit slurred speech or have difficulty articulating words, known as dysarthria or aphasia. These speech impairments can range from mild to severe and are often accompanied by challenges in understanding spoken language.

Visual disturbances

Sudden onset of visual problems can occur, including loss of vision in one or both eyes, double vision, or blurred vision. These symptoms may significantly impact the individual's ability to navigate their environment safely.

Loss of coordination and balance

Ischaemic stroke can disrupt the brain's coordination centers, resulting in loss of balance, difficulty walking, and a propensity for falls. These symptoms require immediate assessment to prevent further injury.

Severe headache

Although not as common in ischaemic strokes compared to haemorrhagic strokes, some patients may report an abrupt and intense headache, indicating potential underlying complications or secondary ischemia.

Altered consciousness or confusion

Depending on the extent of cerebral involvement, patients may exhibit confusion, disorientation, or decreased levels of consciousness. This variability highlights the complex nature of ischaemic stroke presentations.

Recognition and response

The clinical features of ischaemic stroke are diverse and can manifest suddenly, underscoring the need for prompt recognition and emergency response. Awareness of the F.A.S.T. method can empower both healthcare professionals and the public to act swiftly, potentially saving lives and reducing long-term disabilities. Efforts to enhance education on stroke recognition will play a critical role in improving outcomes for individuals experiencing ischaemic stroke

Clinical features of haemorrhagic stroke

Haemorrhagic stroke accounts for approximately 15-20% of all strokes and can be divided into two main types: intracerebral haemorrhage (ICH) and subarachnoid haemorrhage (SAH). The sudden onset of symptoms can lead to misdiagnosis and delays in treatment, making awareness of clinical features essential for healthcare providers and the general public.

Sudden severe headache

A hallmark of haemorrhagic stroke, particularly in cases of subarachnoid haemorrhage, is a sudden and intense headache, often described as a "thunderclap" headache or the "worst headache of one's life." This symptom warrants immediate medical evaluation.

Nausea and vomiting

Patients may experience nausea and vomiting, often resulting from increased intracranial pressure or irritation of the meninges due to the presence of blood.

Altered consciousness

The level of consciousness can vary significantly, ranging from mild confusion to a complete loss of consciousness (coma), depending on the location and volume of the bleed.

Neurological deficits

Sudden onset of focal neurological deficits is common, typically manifesting as weakness or numbness on one side of the body (hemiparesis or hemiplegia), which may affect motor skills and coordination.

Speech difficulties

Patients may present with slurred speech (dysarthria) or have difficulty communicating and understanding language (aphasia), reflective of the specific brain areas impacted by the bleed.

Seizures

Seizures may occur in response to irritation of brain tissue or increased intracranial pressure. Their presence should prompt urgent evaluation for potential underlying causes.

Vision problems

Patients may report sudden changes in vision, including blurred or double vision, relating to increased intracranial pressure or disruption of optic pathways.

Stiff neck and photophobia

In cases of subarachnoid haemorrhage, meningeal irritation might lead to stiffness of the neck and sensitivity to light (photophobia), necessitating further assessment.

Changes in heart rate and blood pressure

Patients may show variations in vital signs, including elevated blood pressure and alterations in heart rate, commonly resulting from compensatory mechanisms due to increased intracranial pressure.

Recognition and response

Timely recognition of the symptoms associated with haemorrhagic stroke is vital for effective management. Healthcare professionals should advocate for immediate medical evaluation when these symptoms are observed. Diagnostic imaging, such as CT scanning or MRI, is critical for confirming the diagnosis and determining the appropriate treatment pathway.

Imaging

Imaging plays a very significant role in the diagnosis of stroke and aids in appropriate therapy choice.[23] Its also useful in predicting prognosis and monitoring treatment.[24] Computed tomography(CT) scan of the brain is the most common form of clinical imaging procedure used in the treatment of stroke and Non-contrast CT Scans are usually the first step in imaging process.[25] The "gold standard" for stroke imaging is debated but generally points to Magnetic Resonance Imaging (MRI) with Diffusion-Weighted Imaging (DWI), especially for detecting acute ischemic strokes, due to its high sensitivity and ability to visualise early ischemic changes within minutes of onset. However, Computed Tomography (CT) is still often the initial and preferred imaging modality in hyperacute stroke due to its availability and faster acquisition time, particularly for distinguishing ischemic from hemorrhagic stroke.[26] They can swiftly identify or exclude haemorrhagic stroke. Diffusion-weighted magnetic resonance imaging (MRI) is the most sensitive imaging technique, it shows ischaemic changes very early, effective in evaluation of cerebral changes in ischaemic stroke and ruling out other conditions that might mimic stroke conditions. American Stroke Association recommends additional magnetic resonance imaging (MRI) after initial head imaging in cases in which initial imaging did not demonstrate infarction. According to a review by Akbarzadeh *et al.*, MRI is more precise in excluding intracranial hemorrhage and MRI with DWI is more accurate in the revealing acute ischemic stroke. However, due to the availability, cost, and lower acquisition time, CT is preferred in most healthcare settings.[27] Other uses of MRI include categorisation of etiologic subtype, evaluation of tissue injury, separating mimics from ischemia, deciding timing of anticoagulation, provision of imaging biomarkers.[28,29,30]

Biomarkers

Biomarkers are objective indicators which include proteins, ribonucleic acids, lipids or metabolites used to assess normal or pathological processes, assess responses to treatment and predict prognosis. They could improve the diagnosis and treatment of stroke but currently there is no marker with sufficient sensitivity, specificity, rapidity, precision, and cost-effectiveness to be used in the routine treatment of stroke. [31] Three biomarkers evaluated in humans (IL-6, Glu and TNF-α) had different chronological profiles and were non-specific to cerebral injury. However, they established good relationships with severity and outcomes of diseases. Similarly, IL-6, Glu and TNF-α were non-specific to cerebral ischemia as biomarkers in rats studies.[32] Improvement in patient outcomes in acute stroke requires a rapid and accurate diagnosis of stroke and its subtypes. A biomarker that could differentiate between hemorrhagic and ischemic stroke or risk of subsequent bleeding would, in theory, permit widespread initiation of thrombolysis in the ambulance and save valuable time and brain tissue.

Markers of brain tissue damage, particularly the highly abundant glial structural proteins like GFAP and S100β and the matrix protein MMP-9 offer this promise but have not yet been systematically evaluated at the earliest time points which matter most. To date, other highly abundant structural proteins such as those characteristic of axons, dendrites, and synapses or oligodendrocyte processes have rarely been considered for this role with the exception of the NR2 degradation product of the NMDA receptor and PARK7 which has a specific anti-oxidant role. However, it has to be concluded that none of the candidate markers described in this review have entered routine clinical use despite their obvious promise. More work is required before lives can depend on such measurements.[33]

Stroke mimics and chamelons

Mimics comprise about half of the cases admitted for stroke syndromes. The diagnosis of acute ischaemic stroke is not always simple because similar symptoms may resemble stroke symptoms in a number of conditions called stroke mimics. The frequency of stroke mimics depends on the evaluating Doctor and the facilities available at the hospital. There are two types of stroke mimics, Functional mimics and medical mimics. The latter comprising about 50% -80% of the stroke mimics.[34] Functional mimics or conversion disorders have clinical features that could be difficult to differentiate from ischaemic stroke and they are less common than medical mimics.[35] Stroke mimics have less clearly defined clinical features typical of well-defined stroke syndromes.[36] McClelland et al reported

in their study that stroke mimics were more common with younger ages and females.[37] Common clinical features include vertigo, dizziness, altered level of consciousness, tingling sensations, numbness, monoplegia, speech dysfunction, limb ataxia, seizures, migraine, depression, headache and visual disturbances.[38] Stroke mimics are non-vascular conditions that present with clinical features similar to that acute ischemic stroke. Examples include conversion disorders; seizures and postictal paralysis; toxic-metabolic disturbances; brain tumours; infections, and migraine. Some of them may present with imaging features of cytotoxic oedema and perfusion deficits that are also found in ischaemic stroke.[39,40]

Stroke mimics Stroke mimics encompass a range of medical conditions that manifest with clinical presentations similar to those of cerebrovascular accidents (CVAs), yet are not attributed to cerebrovascular pathology. Differentiating between true strokes and their mimics is essential for timely and effective management. Many conditions can present with neurological symptoms similar to stroke, complicating diagnosis and management. Recognition of these stroke mimics is vital to prevent unnecessary interventions and ensure that patients receive appropriate care.

Common stroke mimics

Seizures: The postictal state following a seizure may present with transient weakness, confusion, or altered awareness, mimicking stroke symptoms. Clinicians should consider seizure activity in patients presenting with sudden-onset neurological deficits.

Migraine aura: Migraine-related aura can result in neurological deficits such as visual disturbances, sensory changes, or weakness. These symptoms can be challenging to distinguish from those of an ischemic stroke.

Transient ischemic attack (TIA): A TIA presents with transient neurological symptoms that resolve within 24 hours. Although related to stroke pathology, the management and prognosis differ significantly from that of full-blown strokes.

Hypoglycemia: Severe hypoglycemia can induce confusion, dysphasia, and motor deficits. Rapid assessment of blood glucose levels is essential in suspected stroke cases.

Intoxication: Alcohol or substance use can lead to altered mental status and motor deficits that may be misinterpreted as a stroke. A thorough history and toxicology screening can aid in the differential diagnosis.

Bell's palsy: This condition, characterised by acute onset of unilateral facial weakness, may mimic the facial droop seen in stroke. Notably, Bell's Palsy typically spares the forehead due to the differential involvement of central versus peripheral pathways.

Multiple sclerosis (MS): Acute exacerbations of MS can produce neurological symptoms, including weakness and sensory changes that may resemble acute stroke syndromes due to the presence of demyelination.

Brain tumors: Primary or metastatic brain tumors can lead to increased intracranial pressure and focal neurological deficits, complicating the clinical picture and mimicking stroke presentations.

Neurological infections: Infectious processes such as meningitis or encephalitis may present with neurological deficits due to inflammation of the central nervous system, akin to stroke-like symptoms.

Psychogenic weakness: Psychogenic movement disorders can lead to weakness or paralysis without identifiable organic pathology, creating diagnostic challenges.

Peripheral nerve injury: Acute peripheral nerve injuries can result in weakness or sensory loss, which may be confused with central nervous system lesions.

Metabolic disorders: Conditions such as renal failure or hepatic encephalopathy can lead to altered mental status and neurological deficits, which warrant consideration in the differential diagnosis of stroke. Accurate and timely differentiation of stroke from its mimics is essential for optimising patient outcomes. Clinicians should maintain a high index of suspicion for these conditions when confronted with patients exhibiting acute neurological symptoms. Employing comprehensive clinical assessments, including imaging and laboratory studies, will facilitate correct diagnoses and appropriate treatment pathways.

Stroke chameleon

This refers to instances where stroke symptoms deviate from the classic presentations, potentially resulting in misdiagnosis and delayed treatment. Understanding these atypical manifestations is essential for healthcare professionals to enhance diagnostic accuracy and improve patient outcome. Stroke is typically characterised by unilateral weakness, speech disturbances, and other focal neurological deficits. However, certain cases may present with unusual clinical symptoms that obscure the diagnosis, necessitating heightened awareness among clinicians.

The term "stroke chameleon" has been coined to describe these atypical presentations which can include a myriad of non-specific symptoms.

Altered consciousness:Some patients may present with confusion or altered levels of consciousness rather than the standard motor or language deficits. This phenomenon can lead to significant diagnostic challenges.

Severe headache: Although headaches may accompany strokes, the sudden onset of a severe headache often indicates conditions like subarachnoid haemorrhage, resulting in potential misdiagnosis when associated with atypical stroke presentations.

Seizures: In certain cases, strokes may manifest with seizures, particularly in the presence of cortical involvement or brain lesions. This atypical presentation can divert attention from stroke as the underlying cause.

Non-specific symptoms: Symptoms such as dizziness, nausea, and neck pain may be reported, which are often less characteristic of classic stroke presentations, further complicating diagnosis.

Unilateral vs. Bilateral symptoms: While classic strokes usually present unilaterally, some patients may exhibit bilateral symptoms, adding an additional layer of complexity to the diagnostic process.

Implications for clinical practice: Recognising "stroke chameleons" is critical in emergency and acute care settings, as prompt identification and treatment of stroke significantly improve outcomes. Clinicians should maintain a high index of suspicion for stroke in patients presenting with atypical symptoms and utilise advanced neuroimaging techniques (such as CT and MRI) to facilitate accurate diagnosis. Atypical presentations of stroke, described as "stroke chameleons," necessitate increased awareness among healthcare providers. Enhancing recognition of these unusual symptoms is crucial for improving diagnostic accuracy and ensuring timely therapeutic interventions, ultimately reducing the burden of stroke-related morbidity and mortality. A stroke chameleon is a condition in which a stroke is shown by clinical symptoms that are unusual in stroke. The percentage of stroke mimics reduces with use of MRI at baseline. Chameleons are more problematic because patients are not recognised, and are not properly managed. [41]

Management

To improve outcome in management of stroke, it is expedient to intervene in the acute phase of stroke to improve the size of the stroke lesion, which becomes irreversible over the first few hours after the onset of symptoms. Ischaemic changes can be reversed only by early restoration of blood flow. Modern treatment approaches require excellent organisation in terms of early identification of stroke symptoms, as well as highly specialised and efficient in-hospital care to achieve early restoration of blood flow. Stroke treatment mainly focuses on restoring blood flow to the brain and treating stroke-induced neurological injury.[42]

The role of the stroke team in acute Stroke management

The stroke team is a specialised collective of healthcare professionals dedicated to the diagnosis, treatment, and rehabilitation of patients experiencing acute cerebrovascular incidents. The management of stroke requires a multifaceted and prompt response. A stroke team, comprises of various healthcare professionals and plays a critical role in delivering this response. The Stroke Team composes of emergency medical services representatives who are typically the first healthcare professionals to evaluate individuals presenting with stroke symptoms. Neurologists are essential to the acute management of stroke patients. Neurosurgeons provide surgical intervention and they are integral to the stroke team. Nurses provide immediate patient care, monitoring vital signs and neurological status. Radiologists conduct and interprete imaging studies to aid in the accurate diagnosis and subsequent management of stroke patients.

Rehabilitation therapists include physical therapists, who facilitate mobility recovery, occupational therapists, who assist patients in regaining the ability to perform activities of daily living, and speech therapists, who address communication and swallowing difficulties resultant from the stroke. Pharmacists oversee medication management, ensuring the appropriateness of pharmacological interventions and monitoring for potential drug interactions and side effects. Social workers and case managers provide support services that assist patients and families in navigating the healthcare system and transitioning to appropriate post-discharge care settings.

Research and quality improvement teams focus on data collection and analysis to refine care processes and improve patient outcomes. Community Outreach and Education team raises awareness on stroke prevention, symptom recognition, and the imperative of seeking immediate medical attention. The stroke team's coordinated approach is paramount in the timely and comprehensive management of stroke patients. The integration of the diverse skill sets of interprofessional team members enhances both immediate and long-

term patient outcomes, underscoring the necessity for a collaborative framework in stroke care.

Blood pressure management

Randomised control trial results suggested that lowering of Systolic Blood Pressure to <140 mm Hg in patients with acute Intra-cerebral haemorrhage may be beneficial in some patients. Rapid reduction in blood pressure may be advantageous in those with an acute ischemic stroke who present with a BP of ≤220/120.[43] Additionally, high blood pressure may be treated in the presence of heart failure, aortic dissection, acute myocardial infarction and subarachnoid haemorrhage. For BP of ≤220/120 without comorbid conditions requiring acute BP-lowering, it is sensible to initially lower BP by 15% although the benefit of lowering or re-instituting BP therapy in the first 48-72 hours is uncertain. However, hypotension and hypovolaemia should be avoided to ease systemic perfusion. High Blood Pressure is associated with poor outcome in ischemic stroke and haemorrhagic stroke, and it is also associated with haematoma expansion in intracerebral haemorrhage.[44,45]

Thrombolysis

In the management of ischaemic stroke, intravenous thrombolysis was effective in reversal of neurological deficit, but the therapy is not currently used in many places. This is useful in patients presenting within 4.5 h of onset of acute ischaemic stroke. Intravenous recombinant tissue plasminogen activator (alteplase-t-PA) is administered. Although it has been established that rtPA given within the first 3h is beneficial, rtPA given up to six hour post symptom onset might also be beneficial.[46,47] Tenecteplase has been found to be as effective as alteplase but may also lyse large vessel clots more effectively. Besides, it has better safety profile in thrombolyis than alteplase. Endovascular treatment with mechanical thrombectomy could be combined with intravenous thrombolysis in case of large vessel occlusion within six hours of onset of symptoms.[48,49]

Use of mannitol

Cerebral oedema is a major cause of early fatality and long-term disability after stroke. Mannitol is an osmotic agent and a free radical scavenger which might reduce oedema and tissue injuries in stroke. In a study conducted by Papagiani and colleagues, it was found that treatment of patients with mannitol in ischemic stroke-related cerebral oedema did not appear to affect the practical outcome and might raise mortality, irrespective of severity of stroke.[50] Mannitol seemed to be safe but might not improve outcome in patients with acute intracerebral hemorrhage according to the work done by Wang *et al.*[51] According to a review by Bereczki *et al.*, there was not enough evidence that support the routine use of mannitol for acute management of stroke. Further studies have to be done to prove that mannitol is beneficial in acute management of stroke. [52] However, mannitol was reported to effectively reduce pathological intracranial pressure proportionally to the degree of intracranial hypertension in a review conducted by Poole and colleagues.[53] Another study on the use of mannitol and normal saline in ischaemic stroke patients with midline shift showed increased cerebral blood flow in the contralateral hemisphere with mannitol but not with normal saline. So, higher perfusion pressures osmotic agents may increase Cerebral Blood Flow in normal brain tissue.[54] In a systematic review, Cook *et al.* reported that available evidence suggested hyperosmolar therapy may be useful in reducing intracranial pressure(ICP) increase or cerebral edema in patients with Subarachnoid haemorrhage, Traumatic brain injury, acute ischaemic stroke, intracranial haemorrhage, and HE, although neurological outcomes do not appear to be affected. Corticosteroids appeared to be useful in reducing cerebral edema in patients with bacterial meningitis, but not intracranial haemorrhage.[55]

Use of furosemide

Mannitol and furosemide are widely used for prevention and treatment of cerebral edema, increased intracranial pressure, and cerebral hernia. However, they should be used cautiously in ischaemic stroke because of the risk of Acute kidney injury, hence serum creatinine and estimated glomerular filtration rate should be monitored closely.[56]

Haemorrhagic stroke

Hemicraniectomy

After medical treatment, many patients have perpetual neurologic deficits such as hemiplegia, aphasia, and visual loss. Surgery for restoration of functions are important to complement medical treatment. The source of the bleed is located and surgically "clipped", the skull is drained to decrease pressure in the brain, blocking off any sources of bleeding, or performing minimally invasive "burr hole" for the removal of the intracerebral bleed. Procedures such as Stem cell transplantation, neuro-modulation and cortical stimulation techniques, and brain-computer interface technologies are likely to improve neuro- restoration and are subjects of future research.[51] For patients who did not respond well to medical management, decompressive surgery has been recommended for those with space occupying hemispheric infarction. The justification for this therapy is to create compensatory space to house the enlarging brain, consequently controlling intracranial pressure, and averting tissue injury.[57,58]

Decompressive surgery for malignant cerebral infarcts, at least in patients under the age of 60 years, is now also well-documented. Endovascular thrombectomy is recommended for anterior circulation stroke due to large-vessel occlusion. In this case, a rapid assessment is very important and management should start in six hours. The blood pressure should be monitored carefully during the procedure and hypotension should be treated aggressively as the need arises.[49] Stroke patients below 60 who has malignant middle cerebral artery region infarction should have decompressive hemicraniectomy.[2]

In the DESTINY (Decompressive Surgery for the Treatment of Malignant Infarction of the Middle Cerebral Artery) study by Juttler et al, they chose a sequential design which could be adjusted as the study progresses. Intention to treat analysis was used to avoid bias. However, combining per-protocol analysis with intention to treat analysis is usually recommended for a non-inferiority trial while the DESTINY trial was closer to a superiority trial. This study showed that hemicraniectomy significantly reduces death and improve outcome in respondents.[56]

Recent advances in stroke diagnosis and management

Stroke remains one of the leading causes of global morbidity and mortality, necessitating ongoing advancements in diagnosis and management to improve patient outcomes. Recent innovations in neuroimaging techniques, such as advanced diffusion-weighted imaging (DWI) and perfusion-weighted imaging (PWI), allow for the early detection of ischemic changes and assessment of collateral circulation, which are critical for timely interventions like thrombolysis and thrombectomy. The introduction of rapid CT angiography (CTA) has enhanced the ability to identify large vessel occlusions, facilitating quicker treatment decisions. Additionally, emerging blood-based biomarkers are being researched for their potential to differentiate between ischemic and hemorrhagic strokes, offering a promising avenue for enhanced diagnosis.[59,60]

In terms of management strategies, recent advancements in thrombectomy techniques have significantly improved outcomes for acute ischemic stroke patients. The latest devices, including advanced stent retrievers and aspiration thrombectomy systems, have demonstrated higher recanalisation rates and reduced complication risks, making mechanical thrombectomy a standard of care for appropriate patients. Moreover, novel thrombolytic agents like Tenecteplase are being evaluated for their safety and efficacy compared to traditional alteplase, potentially reshaping protocols for acute stroke treatment. Tele-stroke programs have also emerged as essential components of modern stroke care, allowing for rapid specialist consultations and interventions, particularly in remote or underserved regions.[61,62]

Finally, the shift toward personalized medicine is gaining traction in stroke management. By integrating genetic profiling, neuroimaging biomarkers, and patient-specific characteristics, healthcare providers can tailor treatment interventions to improve individual outcomes. Additionally, advances in neurorehabilitation techniques, including virtual reality and robotic-assisted therapies, are enhancing recovery by increasing patient engagement and motivation. These innovations represent a paradigm shift in stroke care, aiming not only to save lives but also to improve the quality of life for stroke survivors.[63]

Primary prevention

Cigarette smoking, physical inactivity, overweight and obesity, dyslipidemia, hypertension, and diabetes should be managed to decrease the risk of stroke according to American Heart Association. Guidelines recommend that some patients with atrial fibrillation be treated with warfarin or direct-acting oral anticoagulants for stroke prevention, depending on the clinical situation. Other risk factors include carotid artery disease, migraine with aura, sickle cell disease, alcohol or drug use, hypercoagulable states, and previous stroke or transient ischemic attack.[64] In ambulatory patients with high Blood Pressure, RCTs have consistently shown that antihypertensive pharmacotherapy is effective for primary and secondary prevention of stroke.[41,65] Blood pressure reduction by all classes of antihypertensive drugs is accompanied by significant decrease in stroke and major cardiovascular events. Warfarin is used for treatment of atrial fibrillation in prevention of stroke. However, Novel oral anticoagulants (NOACs) have advantages over warfarin in patients with atrial fibrillation(AF), but there was no strong evidence that NOACs should replace warfarin or low molecular weight heparin(LMWH) in primary prevention, treatment or secondary prevention of venous thromboembolism (VTE). [66, 67]

Secondary prevention

Secondary prevention of stroke will require effective management of risk factors like diabetes mellitus, hypertension, smoking and hyperlipidaemia. After an episode of stroke, the risk of recurrence, death, and use of health services is high. To achieve a good secondary prevention of stroke, surviving patients need to be followed up adequately including proper management of risk factors of recurrence. It was

reported that a systolic BP treatment target of <130 mm Hg, if tolerated, is reasonable for secondary stroke prevention.[68,69] Treatment of BP to <130/80 with diuretics and ACE inhibitors in all types of stroke, and dyslipidaemia with statins are recommended in secondary prevention of patients with non-cardioembolic TIA or stroke.[70] For the control of hypertension and other cardiovascular risk factors, strategies should include lifestyle measures, such as stopping smoking, exercise and reducing overweight. The pharmacotherapy include combinations of ACE inhibitors, diuretics, beta-blockers, aspirin and statins.[71]

Very early mobilisation was defined by Langhorne *et al.* as the beginning of sitting, standing and walking out of bed after stroke within 24 hours of stroke event based on severity of stroke. However, despite being a very big study with high external validity, the earlier the patients started mobilisation the worse their conditions.[72] Therefore there is a need to know who to target for early mobilisation and the characteristics of early mobilisation. In other to evaluate the effect of early mobilisation of patients with stroke, Herisson *et al.* conducted a randomised controlled trial of patients after stroke events. The first group seated out of bed one calendar day after a stroke event while the second group was seated out of bed on the third calendar day. No significant difference was found between the two groups, however there is need to conduct a bigger study because the sample size used in this study was relatively small.[73]

CONCLUSION

Although a lot have been done in stroke research which has led to improvement in patients' management and outcome, there is room for more studies in both medical and surgical management of stroke because the mortality is still relatively high. Primary and Secondary prevention of stroke is very important to avoid complications associated with stroke.

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Competing interests
The Authors declare that there is no conflict of interest.

Authors' Contributions
Azeez IA contributed to the conceptualisation, methodology, manuscript preparation, review, editing and final approval. Ogunniyi IA contributed to the conceptualisation, methodology, manuscript preparation, review, editing and final approval. Osarenkhoe OJ contributed to the conceptualisation, methodology, manuscript preparation, review, editing and final approval Adeyemo AK contributed to the conceptualisation, methodology, manuscript preparation, review, editing and final approval Adetona A contributed to the conceptualisation, methodology, manuscript preparation, review, editing and final approval, Lasisi AJ contributed to the conceptualisation, methodology, manuscript preparation, review, editing and final approval Alade HK contributed to the conceptualisation, methodology, manuscript preparation, review, editing and final approval, Oyelayo contributed to the conceptualisation, methodology, manuscript preparation, review, editing and final approval. Abdulraheem AB contributed to the conceptualisation, methodology, manuscript preparation, review, editing and final approval. Agbana RD contributed to the conceptualisation, methodology, manuscript preparation, review, editing and final approval.

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The data produced and analysed during the current study are not publicly available to keep the responses of the subjects confidential. However, they would be available from the corresponding author upon reasonable request

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