Emergency Management of Stroke

Nedaa Mohammed A. Alromaili, Mahmoud Shehab Halawani, Intimani Abdula Rahman Malawi, Jumanah Sahal Malibari, Abdulrahman Mubarak Almutairi, Abdullah Abdulziz Alsaib, Bejad Nasha AL-Osaimi, Asmaa Idris Ali, Faisal Abdulrahman Althobaiti, Lamma Abdulmohsen A Alghiryafi

1 King Saud University, 2 Batterjee Medical College for Sciences and Technology, 3 Umm Alqura University, 4 Majmaah University, 5 King Abdulaziz University, 6 Taif University

ABSTRACT

A stroke takes place when the blood supply to the brain is interrupted or there is bleeding in the brain. Within a short time, brain cells start to die. It is critical to seek emergency care at the first sign of a stroke. Early treatment saves many lives and decreases the effects of stroke. If brain cells die or are damaged as a consequence of a stroke, symptoms take place in the parts of the body that these brain cells control. Examples of stroke symptoms comprised sudden weakness, paralysis or numbness of the face, arms, or legs (paralysis is an inability to move), trouble speaking or understanding speech and trouble seeing. A stroke is a serious medical condition that requires emergency care and may cause lasting brain damage, long-term disability or even death.

Keywords: stroke, thrombolytic therapy, emergency management, ischemic stroke, prevention.

INTRODUCTION

Acute ischemic stroke (AIS) is described by the sudden loss of blood flow to an area of the brain, normally in a vascular territory, causing in a corresponding loss of neurologic function. Additionally, earlier it was called cerebrovascular accident (CVA) or stroke syndrome, stroke is a nonspecific condition of brain damage with neuronal dysfunction that has numerous pathophysiologic reasons. Strokes can be distributed into 2 types: hemorrhagic or ischemic. Acute ischemic stroke is caused by thrombotic or embolic occlusion of a cerebral artery. Hemorrhagic stroke is less mutual than ischemic stroke (i.e., stroke initiated by thrombosis or embolism), epidemiologic investigations showed that only 8-18% of strokes were hemorrhagic. On the other hand, hemorrhagic stroke is allied with higher mortality rates than in case of ischemic stroke.

Patients with hemorrhagic stroke may show focal neurologic shortages like those of ischemic stroke, but have a tendency to be sicker than are patients with ischemic stroke. Nevertheless, patients with intracerebral bleeding probably had headache, nausea and vomiting, seizures, altered mental status and marked hypertension. About 800,000 individuals endure strokes every year in the United States. Nearly, 82-92% of these strokes were ischemic. Stroke is the fifth driving reason for grown-up death and incapacity, bringing about $72 billion in yearly cost. Between 2012 and 2030, add up to coordinate therapeutic stroke-related expenses are anticipated to triple, to $184.1 billion, with most of the anticipated increment in costs emerging from those 65 to 79 years old. Ischemic and hemorrhagic stroke can't be dependably separated on the premise of clinical examination discoveries alone. Advance assessment, particularly with cerebrum imaging tests (i.e., computed tomography [CT] scanning or magnetic resonance imaging [MRI]) is necessary.

MATERIALS AND METHODS

- Data Sources and Search terms

We conducted this review using a comprehensive search of MEDLINE, PubMed, EMBASE, Cochrane Database of Systematic Reviews and Cochrane Central Register of Controlled Trials from January 1, 1980, through August 31, 2017.

- Data Extraction

Two reviewers independently reviewed studies, abstracted data and resolved disagreements by consensus. Studies were evaluated for quality. A review protocol was followed throughout. The study was approved by the Ethics Board of King Saud University.

Signs and symptoms

Patients with intracerebral bleeds are more likely than those with ischemic stroke to have headache, altered mental status, seizures, nausea and vomiting, and/or marked hypertension. Even so, none of these findings reliably distinguished between hemorrhagic and ischemic stroke.
Ischemic Stroke | Hemorrhagic Stroke
---|---
Dysarthria | Right hemiparesis
Hemisensory deficits | Left gaze preference
 Aphasia | Right visual field cut
Ataxia | Right hemisensory loss
Facial droop | Aphasia
Monocular or binocular visual loss | Left hemiparesis
Visual field deficits | Left visual field cut
Diplopia | Left hemisensory loss
Vertigo (rarely in isolation) | Neglect (atypical)
Nystagmus | Right gaze preference
Sudden reduction in level of consciousness | 
Abrupt onset of hemiparesis, monoparesis, or (infrequently) quadriparesis | 

**Table 1:** signs and symptoms of stroke
Even though such symptoms can happen alone, they are more close to happen in combination. No historical feature differentiates ischemic from hemorrhagic stroke, even though nausea, vomiting, headache, and sudden change in level of consciousness are more common in hemorrhagic strokes. In younger patients, a history of recent trauma, coagulopathies, illicit drug use, migraines or use of oral contraceptives ought to be stimulated.

**Management of Stroke**
The significant objective of treatment in stroke is to preserve tissue in the ischemic penumbra, where perfusion is diminished however sufficient to stave off infarction. Tissue in this part of oligemia could be conserved by restoring blood flow to the conceded area and optimizing collateral flow. Recanalization approaches, containing the management of intravenous (IV) recombinant tissue-type plasminogen activator (rt-PA) and intra-arterial methods, try to establish revascularization so that cells in the penumbra may be rescued before irreversible damage happens. Restoring blood flow could diminish the impacts of ischemia only if performed rapidly. Notwithstanding constraining the period of ischemia, an alternative plan is to restrict the severity of ischemic injury (i.e., neuronal protection). Neuroprotective approaches are proposed to preserve the penumbral tissues and to broaden the time window for revascularization techniques. Right now, conversely, no neuroprotective agents have been appeared to affect clinical results in ischemic stroke.

**Table 2. General Management of Patients with Acute Stroke**

<table>
<thead>
<tr>
<th>Blood glucose</th>
<th>Treat hypoglycemia with D50 - Treat hyperglycemia with insulin if serum glucose &gt;200 mg/dL</th>
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<tbody>
<tr>
<td>Oral intake</td>
<td>NPO initially; aspiration risk is great, avoid oral intake until swallowing assessed</td>
</tr>
<tr>
<td>Oxygen</td>
<td>Supplement if indicated (Sa02 &lt; 94%)</td>
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<tr>
<td>Temperature</td>
<td>Avoid hyperthermia; use oral or rectal acetaminophen and cooling blankets as needed</td>
</tr>
<tr>
<td>Cardiac monitor</td>
<td>Continuous monitoring for ischemic changes or atrial fibrillation</td>
</tr>
<tr>
<td>Intravenous fluids</td>
<td>Avoid D5W and excessive fluid administration - IV isotonic sodium chloride solution at 50 mL/h unless otherwise indicated</td>
</tr>
</tbody>
</table>
• Emergency Reaction and Transport

Acknowledgment that a stroke may have happened, enactment of the emergency department and quick transport to the proper receiving facility are important to give stroke patients the most obvious opportunity for acute mediations. Patients with signs or symptoms of stroke, 29-65% used some features of the emergency medical services (EMS) system [9, 10].

The vast majority of the patients who call EMS were the individuals who present within 3 hours of indication beginning. Calls to the ambulance and the utilization of EMS were related with shorter time periods from symptom beginning to emergency department arrival [11, 12].

Stroke ought to be a priority dispatch with quick EMS reaction. EMS responders ought to play out a concise H&P, get time of symptom beginning or last known ordinary, implement a pre-hospital stroke appraisal, define blood glucose levels and give progress ahead of time to their ED goal as convenient a way as conceivable in order to permit planning and marshalling of work force and resources. The advancement of stroke centre designation such as centres would then turn into the favoured goal for patients with acute stroke side effects who use EMS. Information supporting the utilization of emergency air transport for individuals with acute stroke symptoms was restricted. Additional assessment of this transportation methodology is important to limit the conceivably high number of stroke mimics and to expand the suitable utilization of transport resources.

Telemedicine is additionally a technology that can give timely expert advice to rural and underserved clinics and hospitals [13].

• Blood Pressure Control

In spite of the fact that hypertension is normal in acute ischemic stroke and is related with poor result, investigations of antihypertensive treatment in this setting have created incompatible outcomes. A hypothetical disadvantage of blood pressure lessening is that elevated blood pressure may check dysfunctional cerebral autoregulation from stroke, however constrained proof recommends that antihypertensive treatment in acute stroke does not change cerebral perfusion [14].

For patients who are not applicants for fibrinolytic treatment, current guidelines suggested permitting moderate hypertension in many patients with acute ischemic stroke. Most patients encountered unconstrained diminishment in blood pressure over the initial 24 hours without treatment [15]. The exceptions would be patients who had dynamic comorbidities (e.g., aortic analysis, , decompensated heart failure and acute myocardial infarction [MI], hypertensive emergency) that necessitate emergent blood pressure management.

339 patients with ischemic stroke found that oral candesartan diminished combined vascular occasions yet had no impact on handicap [14]. However, the Scandinavian Candesartan Acute Stroke Trial (SCAST), a randomized, placebo-controlled., double-blind examination included 2029 patients, found no sign of advantage from candesartan yet found some proposal of damage [16]. In the single-blind, randomized China Antihypertensive Trial in Acute Ischemic Stroke (CATIS) study, which comprised 4,071 patients with acute ischemic stroke and raised blood pressure, instant blood pressure decreased with antihypertensive treatment within 48 hours of symptom onset did not decrease the risk for death or major incapacity.

CATIS accepted patients who taken fibrinolytic treatment. Mean systolic blood pressure was decreased from 166.7 to 144.7 mm Hg within 24 hours in the antihypertensive treatment group. Among the 2,038 patients who got antihypertensive treatment, 683 achieved the essential endpoint of death or significant handicap at 14 days or hospital release, matched with 681 of the 2,033 patients who got no antihypertensive treatment. At 3-month follow-up, 500 patients in the antihypertensive treatment group and 502 patients in the control group achieved the secondary endpoint of death or major incapacity [17].

A 2017 joint practice guideline from the American College of Physicians (ACP) and the American Academy of Family Physicians (AAFP) calls for physicians to start management for patients who have persistent systolic blood pressure at or above 150 mm Hg to achieve a target of less than 150 mm Hg to diminish danger for stroke, cardiac events, and death [18].

Consensus agreement is that these blood pressure guidelines should be maintained in the face of other interventions to restore perfusion, such as intra-arterial thrombolysis [13].
Table 3. Blood Pressure Management

<table>
<thead>
<tr>
<th>Blood Pressure</th>
<th>Treatment</th>
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<tbody>
<tr>
<td>Pretreatment:</td>
<td>Labetalol 10-20 mg IVP repeated every 10-20 minutes or Nicardipine 5 mg/h, titrate by 2.5 mg/h every 5-15 min, maximum 15 mg/h; when desired blood pressure reached, lower to 3 mg/h or Enalapril 1.25 mg IVP</td>
</tr>
<tr>
<td>Posttreatment:</td>
<td>Sodium nitroprusside (0.5 mcg/kg/min) Labetalol 10-20 mg IVP and consider labetalol infusion at 1-2 mg/min or nicardipine 5 mg/h IV infusion and titrate or Nicardipine 5 mg/h, titrate by 2.5 mg/h every 5-15 min, maximum 15 mg/h; when desired blood pressure reached, lower to 3 mg/h or Labetalol 10 mg IVP, may repeat and double every 10 min up to maximum dose of 300 mg</td>
</tr>
<tr>
<td>SBP &gt;185 or DBP &gt;110 mm Hg</td>
<td>SBP &gt;230 mm Hg or DBP 121-140 mm Hg SBP 180-230 mm Hg or DBP 105-120 mm Hg DBP &gt;140 mm Hg SBP &gt;220 or DBP 121-140 mm Hg or MAP &gt;130 mm Hg SBP &lt; 220 mm Hg or DBP 105-120 mm Hg or MAP &lt; 130 mm Hg</td>
</tr>
<tr>
<td>Posttreatment:</td>
<td>Sodium nitroprusside 0.5 mcg/kg/min; may reduce approximately 10-20% Labetalol 10-20 mg IVP over 1-2 min; may repeat and double every 10 min up to maximum dose of 150 mg or nicardipine 5 mg/h IV infusion and titrate or Nicardipine 5 mg/h, titrate by 2.5 mg/h every 5-15 min, maximum 15 mg/h; when desired blood pressure reached, lower to 3 mg/h Antihypertensive therapy indicated only if acute myocardial infarction, aortic dissection, severe CHF, or hypertensive encephalopathy present</td>
</tr>
</tbody>
</table>

- **Blood Glucose Control**
  Severe hyperglycemia seems to be freely allied with poor result and reduced reperfusion in thrombolysis, in addition to extension of the infarcted territory [20-22]. Furthermore, normoglycemic patients ought not to be given excessive glucose-containing IV fluids, as this can lead to hyperglycemia and can exacerbate
ischemic cerebral injury. Blood sugar control ought to be strongly preserved with insulin treatment with the aim of starting normoglycemia (90-140 mg/dL). Moreover, close monitoring of blood sugar level ought to continue during hospitalization to prevent hypoglycemia [13].

- **Intravenous Access and Cardiac Monitoring**
  Patients with acute stroke necessitate IV access and cardiac monitoring in the emergency department (ED). Patients with acute stroke are at danger for cardiac arrhythmias. Furthermore, atrial fibrillation might be related with acute stroke as either the cause (embolic disease) or as a complication [13].

- **Fibrinolytic Therapy**
  The main fibrinolytic specialist that has been appeared to profit chose patients with acute ischemic stroke are alteplase (rt-PA). While, streptokinase may profit patients with intense MI, in patients with intense ischemic stroke it has been appeared to build the danger of intracranial hemorrhage and death. Fibrinolytics (ie, rt-PA) reestablish cerebral blood stream in a few patients with intense ischemic stroke and may prompt change or determination of neurologic shortfalls. Sadly, fibrinolytics may likewise cause symptomatic intracranial discharge. Different intricacies incorporate conceivably extracranial discharge and angioedema or allergic responses [13]. An rt-PA stroke study group from the National Institute of Neurologic Disorders and Stroke (NINDS) first stated that the early management of rt-PA helped cautiously selected patients with acute ischemic stroke [19]. The FDA afterward accepted the utilization of rt-PA in patients who met NINDS criteria. Specifically, rt-PA had to be given within 3 hours of stroke onset and only after CT scanning had ruled out hemorrhagic stroke. Along these lines, fibrinolytic treatment regulated 3-4.5 hours after side effect beginning was found to enhance neurologic results in the European Cooperative Acute Stroke Study III (ECASS III), recommending a more extensive time window for fibrinolysis in deliberately chose patients [20]. On the premise of these and other information, in May 2009 the AHA/ASA reexamined the rules for the organization of rt-PA after intense stroke, growing the window of treatment from 3 hours to 4.5 hours to give more patients a chance to profit by this treatment [20-22].

- **Thrombolytic Therapy**
  Present treatments for acute ischemic stroke contain IV thrombolytic treatment with tissue-type plasminogen activator (t-PA) and endovascular treatments utilizing stent retriever devices. A 2015 update of the American Heart Association/American Stroke Association guidelines for the early treatment of patients with acute ischemic stroke suggested that patients suitable for intravenous t-PA ought to get intravenous t-PA though endovascular managements are being considered and that patients ought to get endovascular treatment with a stent retriever if they meet criteria [23]. Newer stroke trials have discovered the benefit of utilizing neuroimaging to select patients who are most likely to profit from thrombolytic treatment and the potential benefits of extending the window for thrombolytic therapy beyond the guideline of 3 hours with t-PA and newer agents. CT angiography may demonstrate the location of vascular occlusion. CT perfusion studies are capable of producing perfusion images and together with CT angiography are becoming more available and utilized in the acute evaluation of stroke patients [24]. The Diffusion and Perfusion Imaging Evaluation for Understanding Stroke Evolution (DEFUSE) trial recommended that there could be benefit of managing IV t-PA within 3-6 hours of stroke onset in patients with small ischemic cores on diffusion-weighted magnetic resonance imaging (MRI) and larger perfusion abnormalities (large ischemic penumbras) [25]. The Desmoteplase In Acute Ischemic Stroke (DIAS) trial required to display the benefit of managing desmoteplase in patients within 3-9 hours of onset of acute stroke with a significant mismatch (>20%) between perfusion abnormalities and ischemic core on diffusion-weighted MRI. Larger randomized trials of desmoteplase were negative [26]. A study by Jovin et al presented successful endovascular treatment beyond 8 hours from time last seen well in patients selected for treatment based on MRI or CT perfusion imaging. Revascularization was successful in about 73% of patients [27].

**Stroke Prevention**

Primary stroke prevention states to the treatment of individuals with no history of stroke. Secondary stroke prevention states to the treatment of individuals who have previously had a stroke or transient ischemic attack.

- **Primary Prevention of Stroke**
  Risk-reduction measures in primary stroke prevention may include the use of antihypertensive medications, anticoagulants, platelet antiaggregants, 3-hydroxy-3-methylglutaryl coenzyme A (HMG-CoA) reductase inhibitors
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