# Initial general assessment

Sudden loss of focal brain function is the core feature of the onset of ischemic stroke. The goals in this initial phase include:

**A.** Medically stabilize the patient.

**B.** Reverse any conditions that are contributing to the patient’s problem.

**C.** Assess the pathophysiologic basis of the neurologic symptoms.

**D.** Screen for potential contraindications to thrombolysis in acute ischemic stroke patients.

**E.** Diagnosing an intracerebral haemorrhage (ICH) or subarachnoid haemorrhage (SAH) as soon as possible can be lifesaving. The presence of onset headache and vomiting favour the diagnosis of ICH or SAH compared with a thromboembolic stroke, while the abrupt onset of impaired cerebral function without focal symptoms favours the diagnosis of SAH.

**F. History and physical examination** should distinguish between seizures, syncope, migraine, and hypoglycaemia, which can mimic acute ischemia. In patients with focal signs and altered level of consciousness, it is important to determine whether the patient takes insulin or oral hypoglycaemic agents, has a history of a seizure disorder or drug overdose or abuse, medications on admission, or recent trauma.

### Acute stroke differential diagnosis

* Migraine
* Intracerebral hemorrhage
* Head trauma
* Brain tumor
* Todd’s palsy (paresis, aphasia, neglect, etc., after a seizure episode)
* Functional deficit (conversion reaction) Systemic infection
* Toxic-metabolic disturbances (hypoglycemia, acute renal failure, hepatic insufficiency, exogenous drug intoxication)

**G. Physical examination** should evaluate the neck and retro orbital regions for vascular bruits, and palpate of pulses in the neck, arms, and legs to assess for their absence, asymmetry, or irregular rate.

**1.** The heart should be auscultated for murmurs. Fluctuations in blood pressure occasionally precede fluctuations in clinical signs.

**2.** The skin should be examined for signs of endocarditis, cholesterol emboli, purpura, or ecchymoses. The funduscopic examination may reveal cholesterol emboli or papilledema. The head should be examined for signs of trauma. A tongue laceration may occur during a seizure.

**3.** The neck should be immobilized until evaluated radiographically for evidence of serious trauma if there is a suspicion of a fall. The chest x-ray is helpful if it shows cardiomegaly, metastases, or a widened mediastinum suggesting aortic dissection. Examination of the extremities is important to detect deep vein thrombosis.

**4. Breathing.** Patients with increased ICP due to haemorrhage, vertebrobasilar ischemia, or bihemispheric ischemia can present with a decreased respiratory drive or muscular airway obstruction. Intubation may be necessary to restore adequate ventilation. Patients with adequate ventilation should have the oxygen saturation monitored. Patients who are hypoxic should receive supplemental oxygen.

**H. Immediate laboratory studies**

**1.** All patients with acute neurologic deterioration or acute stroke should have an electrocardiogram. Chest radiography is indicated if lung or heart disease is suspected. Oxygen saturation or arterial blood gas tests are indicated if hypoxia is suspected.

**2. Blood studies include:**

**a.** Complete blood count including platelets, and erythrocyte sedimentation rate.

**b.** Electrolytes, urea nitrogen, creatinine.

**c.** Serum glucose. Finger stick for faster glucose measurement if diabetic, taking insulin or oral hypoglycaemic agents, or if there is clinical suspicion for hypoglycaemia.

**d.** Liver function tests.

**e.** Prothrombin time and partial thromboplastin time.

**f. Toxicology** screen and blood alcohol level in selected patients.

**g.** Blood for type and cross match in case fresh frozen is needed to reverse a coagulopathy if ICH is present.

**h.** Urine human chorionic gonadotropin in women of child- bearing potential.

**i.** Consider evaluation for hypercoagulable state in young patients without apparent stroke risk factors.

### Laboratory studies

Complete blood count and erythrocyte sedimentation rate

Electrolytes, urea nitrogen, creatinine, glucose

Liver function tests

Prothrombin time and partial thromboplastin time

Toxicology screen

Blood for type and cross match

Urine human chorionic gonadotropin in women of child-bearing potential Consider evaluation for hypercoagulable state in young patients without apparent stroke risk factors

**3.** Anticoagulant use is a common cause of intracerebral haemorrhage. Thus, the prothrombin and partial thromboplastin time and the platelet count should be checked. The effects of warfarin are corrected with intravenous vitamin K and fresh- frozen plasma (typically 4 units) in patients with intracerebral haemorrhage.

**4.** A drug overdose can mimic an acute stroke. In addition, cocaine, intravenous drug abuse, and amphetamines can cause an ischemic stroke or intracranial haemorrhage. Hyponatremia and thrombotic thrombocytopenic purpura (TTP) can present with focal neurologic deficits, suggesting the need for measurement of serum electrolytes and a complete blood count with platelet count.

**5. Hyperglycemia,** defined as a blood glucose level >108 mg/dL, is associated with poor functional outcome from acute stroke at presentation. Stress hyperglycemia is common in stroke patients, although newly diagnosed diabetes may be detected. Treatment with fluids and insulin to reduce serum glucose to less than 300 mg/dL is recommended.

**6. Hypoglycaemia** can cause focal neurologic deficits mimicking stroke. The blood sugar should be checked and rapidly corrected if low. Glucose should be administered immediately after drawing a blood sample in "stroke" patients known to take insulin or oral hypoglycaemic agents.

**7. Fever.** Primary central nervous system infection, such as meningitis, subdural empyema, brain abscess, and infective endocarditis, need to be excluded as the aetiology of fever. Common etiologies of fever include aspiration pneumonia and urinary tract infection. Fever may contribute to brain injury in patients with an acute stroke. Maintaining normothermia is recommended after an acute stroke. Prophylactic administration of acetaminophen (1 g four times daily) is more effective in preventing fever than placebo (5 versus 36 percent).

**8. Blood pressure management.** Acute management of blood pressure (BP) may vary according to the type of stroke.

**a. Ischemic stroke.** Blood pressure should not be treated acutely in the patient with ischemic stroke unless the hypertension is extreme (diastolic BP above 120 mm Hg and/or systolic BP above 220 mm Hg), or the patient has active ischemic coronary disease, heart failure, or aortic dissection. If pharmacologic therapy is given, intravenous labetalol is the drug of choice.

**b. Intracranial haemorrhage.** With ICH, intravenous labetalol, nitroprusside, or nicardipine, should be given if the systolic pressure is above 170 mm Hg. The goal is to maintain the systolic pressure between 140 and 160 mm Hg. Intravenous labetalol is the first drug of choice in the acute phase since it allows rapid titration.

**I. Neurologic evaluation.** The history should focus upon the time of symptom onset, the course of symptoms over time, possible embolic sources, items in the differential diagnosis, and concomitant diseases. The neurologic examination should attempt to confirm the findings from the history and provide a quantifiable examination for further assessment over time.

**J. Neuroimaging** studies are used to exclude haemorrhage as a cause of the deficit, to assess the degree of brain injury, and to identify the vascular lesion responsible for the ischemic deficit.

**1. Computed tomography.** In the hyper acute phase, a non- contrast CT (NCCT) scan is usually ordered to exclude or confirm haemorrhage. A NCCT scan should be obtained as soon as the patient is medically stable.

**a. Noncontrast CT.** Early signs of infarction include: Subtle parenchymal hypodensity, which can be detected in 45 to 85 percent of cases. Early focal brain swelling is present in up to 40 percent of patients with early infarction and also has been adversely related to outcome. A hyperdense middle cerebral artery (MCA) can be visualized in 30 to 40 percent of patients with an MCA distribution stroke, indicating the presence of thrombus inside the artery lumen (bright artery sign).

**2. Transcranial Doppler ultrasound (TCD)** visualizes intracranial vessels of the circle of Willis. It is a non-invasive means of assessing the patency of intracranial vessels.

**3. Carotid duplex ultrasound** is as a non-invasive examination to evaluate extra cranial atherosclerotic disease. It may help to establish the source of an embolic stroke, but is not used acutely.

### Initial management of acute stroke

Determine whether stroke is ischemic or hemorrhagic by computed tomography

Consider administration of t-PA if less than three hours from stroke onset

**General management:**

• Blood pressure (avoid hypotension)

• Assure adequate oxygenation

• Administer intravenous glucose

• Take dysphagia/aspiration precautions

• Consider prophylaxis for venous thrombosis if the patient is unable to walk

• Suppress fever, if present

• Assess stroke mechanism (e.g., atrial fibrillation, hypertension)

• Consider aspirin or clopidogrel (Plavix) therapy if ischemic stroke and no contraindications (begin 24 hours after t-PA).

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| **Antiplatelet Agents for Prevention of Ischemic Stoke** |
| • Enteric-coated aspirin (Ecotrin) 325 mg PO qd  • Clopidogrel (Plavix) 75 mg PO qd  • Extended-release aspirin 25 mg with dipyridamole 200 mg (Aggrenox)  one tab PO qd |
| **Eligibility criteria for the treatment of acute ischemic stroke with recombinant tissue plasminogen activator (rt-PA)** |
| **Inclusion criteria**  Clinical diagnosis of ischemic stroke, with the onset of symptoms within three hours of the initiation of treatment (if the exact time of stroke onset is not know, it is defined as the last time the patient was known to be normal), and with a measurable neurologic deficit. |
| **Exclusion criteria**  Historical  Stroke or head trauma within the prior 3 months Any prior history of intracranial haemorrhage Major surgery within 14 days  Gastrointestinal or genitourinary bleeding within the previous 21 days |
| **Clinical**  Rapid improving stroke symptoms  Only minor and isolated neurologic signs  Seizure at the onset of stroke with postictal residual neurologic impairments  Symptoms suggestive of subarachnoid haemorrhage, even if the CT is normal  Clinical presentation consistent with acute MI or post-MI pericarditis Persistent systolic BP >185 diastolic >110 mm Hg, or requiring aggressive therapy to control BP  Pregnancy or lactation  Active bleeding or acute trauma (fracture) |
| **Laboratory**  Platelets <100,000/mm3  Serum glucose <50 mg/dL or >400 mg/dL INR >1.5 if on warfarin  Elevated partial thromboplastin time if on heparin |
| **Head CT scan**  Evidence of haemorrhage  Evidence major early infarct signs, such as diffuse swelling of the affected hemisphere, parenchymal hypodensity, and /or effacement of  >35 percent of the middle cerebral artery territory |

**K. Thrombolytic therapy.** Patients presenting within three hours of symptom onset may be given IV alteplase (Activase) (0.9 mg/kg up to 90 mg; 10 percent as a bolus, then a 60 minute infusion).