Chapter 10

Emergency Management of Acute Ischaemic Stroke

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Additional information is available at the end of the chapter

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Abstract

Stroke is a medical emergency with ischaemic stroke being the commonest type worldwide. Hypertension has been identified as the leading modifiable risk factor globally. The management of acute ischemic stroke is fast changing due to the advancement in technology and the introduction of intravenous recombinant tissue plasminogen activator. There is a limited time window for early intervention to salvage the ailing neurons. The first 24 hours of presentation is therefore crucial in management. Early recognition of stroke symptoms with rapid intervention can lead to a favourable outcome. Specialized care during the acute phase in the intensive care or stroke unit can improve the overall prognosis.

Keywords: ischaemic stroke, haemorrhagic stroke, emergency, management

1. Introduction

Stroke has been defined by the World Health Organization as "rapidly developing clinical signs of focal (at times global) disturbance of cerebral function, lasting more than 24hours or leading to death with no apparent cause other than of vascular origin" [1].

Stroke is a neurologic emergency with poorer prognosis in blacks [2, 3]. It represents one of the major causes of morbidity and mortality globally and ranks third as the most common cause of mortality in developed countries resulting in long term disability and accounting for 4.4 million deaths in the world [4, 5]. The severity of stroke varies widely, ranging from full recovery on one hand to both fatal and non-fatal events with neurological deficits and functional disabilities on the other hand [5–7].

Risk factors for stroke have been classified as modifiable and non-modifiable. The non-modifiable factors include sex, age, race, family history, genetic and low birth weight while the modifiable risk factors include hypertension, diabetes mellitus, hyperlipidaemia, atrial fibrillation, smoking, obesity, carotid artery disease, hyperhomocysteinemia, hypercoagulable states and select biomarkers [8].

Stroke increases sharply with age and the incidence of a first time stroke is about 200 per 100,000 annually with a prevalence of 5–12 per 1000 population. Stroke mortality rate is different among countries ranging from 20 to 250 per 100,000 populations annually [9]. In the UK about 90,000 females and 60,000 males die from stroke yearly with the approximate cost of stroke to NHS and social services being £2.3bn annually [10]. The risk of a recurrent stroke is very high among survivors. About 14% of patients who survive a stroke or TIA will have a recurrence in the first year; 22% of males and 25% of females will have mortality in the first year of an initial stroke and more than half of all stroke patients experience mortality within 8 years [5].

2. Classification

Stroke is classified into two major types: Ischaemic and haemorrhagic. Ischaemic stroke is by far the commonest, accounting for 85% of all strokes while haemorrhagic stroke accounts for 15% of strokes - intracerebral 10%, subarachnoid 5% [5].

Ischaemic stroke is the leading worldwide cause of morbidity and mortality in the developed world. About 8–12% of patients die within 30 days of their first stroke and those that survive the first attack are at increased risk of a recurrence [11]. Ischaemic stroke is caused by atherosclerotic vascular disease leading to occlusion and stenosis of major intracranial or extracranial arteries and constriction of small penetrating arteries of the brain. Cardioembolic stroke due to myocardial infarction is usually due to atherosclerosis of the coronary arteries. The resulting ischaemia leads to direct brain insult because of inadequacy of flow, hypoxia and metabolic substrate and institutes a cascade of neurochemical processes causing continuous damage within hours. Treatment of ischaemic stroke has been with the use of drugs such as fibrinolytic agents, anticoagulants and antiplatelets to improve blood supply to the brain. Prevention of stroke both at the primary and secondary levels is now possible because of availability of various safe and successful interventions directed at high risk individuals [5, 12].

3. Clinical presentation

Patients present with abrupt onset of focal neurological deficit such as facial paresis, arm drift, leg weakness and abnormal speech [13]. Although patients with acute ischaemic stroke do present with headache, vomiting, seizures, depressed level of consciousness; these symptoms are commoner in patients with haemorrhagic stroke. It is difficult, on the basis of clinical presentation,

to distinguish intracerebral haemorrhage from ischaemic stroke as they may look alike [14]. The duration of stroke onset should be noted as this is crucial in defining treatment options. Past medical and drug history (e.g heroin, amphetamines, and cocaine) should be obtained. History of vascular risk factors such as obesity, hypertension, hyperlipidaemia, diabetes mellitus, and smoking should also be obtained. The initial neurological assessment of the patient should be brief but detailed. Different stroke scales such as National Institutes of Health Stroke Scale (NIHSS) can be employed as this helps in determining the severity of the stroke [15]. Assessment of airway, breathing and circulation may precede a thorough evaluation of the stroke patient. A comprehensive physical examination is carried out by the attending physician or the stroke team. This may reveal an irregular pulse, bradycardia, cardiomegaly or heart murmurs. The blood pressure should also be checked [16].

4. Investigations

Besides doing basic investigations such as carotid Doppler, pregnancy test, full blood count, fasting lipid profile, blood sugar, serum homocysteine, serum electrolyte, urea and creatinine, coagulation studies (PT/INR/PTT), liver function tests, Haemoglobin AIc, electrocardiography (ECG), electroencephalopathy (EEG), toxicology screen, cardiac enzymes (CK,CK-MB, TROPONIN I and T) a brain Computed Tomography/Magnetic Resonance Imaging (CT/MRI) Scan is also required as this is the single most important investigation to help exclude a cerebral haemorrhage and stroke mimics. It confirms the diagnosis of ischaemic stroke allowing for prompt treatment of the condition. Increase in both cardiac Troponin T and Troponin I have been found to be associated with stroke severity and poor clinical outcomes [17, 18].

5. General supportive care

Issues to be focused on include: Airway management, hydration, increased intracranial pressure (ICP), Blood pressure control, Blood sugar control, Temperature.

5.1. Airway management

Coma is uncommon with ischaemic stroke patients. Patients who have neurological decline with reduced level of consciousness have challenges in maintaining their airway due to loss of protective reflexes [19]. This can result in aspiration, hypoxaemia or hypercapnia that may increase intracranial pressure by causing cerebral vasodilatation. The role of oxygen therapy in ischaemic stroke has been controversial due to failure of three clinical trials of hyperbaric oxygen to demonstrate efficacy. Supplemental oxygen can be administered at a dose of 10-15 L/min if there is evidence of hypoxia by pulse oximetry. This was shown to slow down the process of ischaemia and extend the therapeutic time window for thrombolysis [20, 21]. Patients with depressed level of consciousness should be intubated to avoid the risk of aspiration [22].

5.2. Hydration

Patients with ischaemic stroke should be routinely hydrated with isotonic saline. This helps ensure adequate perfusion to the ischaemic penumbra and may prevent infarct extension. Hypotonic solutions should be avoided as this may lead to increased cerebral oedema.

5.3. Increased intracranial pressure (ICP)

The head of the bed should be elevated at 30 degrees. Other measures to reducing ICP include administration of 0.5-1 g/kg of 20% mannitol as a bolus. Infusion of hypertonic saline solution (23.4%) can also be administered at a dose of 0.5–2.0 ml/kg as an alternative to mannitol especially in the setting of hypotension. Hyperventilation to a pCO2 of 28-35 mmHg has also been employed as a measure in reducing ICP.

5.4. Blood pressure management

High blood pressure is a frequent occurrence in acute ischaemic stroke. Although blood pressure declines spontaneously within 90 mins after stroke onset [23], about one third of the patients continue to have hypertension with an increased risk of poor outcome [24, 25]. The mechanisms implicated for the increase in blood pressure are multifactorial and include a previous history of hypertension, release of endogenous catecholamines, raised intracranial pressure (Cushing's reflex), infection, "White coat hypertension effect", prior alcohol intake, pain from urinary retention, impaired baroreceptor sensitivity and stress relating to hospitalization [25–29].

Hypotension, though uncommon in acute stroke, correlates with a poor clinical outcome. Causes implicated include infection, cardiac failure, arrhythmias, hypovolaemia and aortic dissection [30].

Rapid blood pressure reduction in acute ischaemic stroke reduces the cerebral blood flow thereby increasing the area of cerebral infarction and worsening neurological outcome [31]. For over three decades, there has been a controversy regarding the treatment of high blood pressure in the setting of acute ischaemic stroke [32, 33]. Some studies have observed a U-shaped relationship between the admission blood pressure and good clinical outcomes, with an optimal systolic blood pressure ranging from 121 to 200 mm Hg and diastolic blood pressure ranging from 81 to 110 mm Hg [34]. High blood pressure should not be treated in the first 24 hours after ischaemic stroke unless the systolic blood pressure is greater than 220, the diastolic blood pressure is greater than 120, the mean arterial blood pressure is greater than 130 mmHg or there are associated complications such as presence of myocardial infarction, aortic dissection or heart failure. At such times, the goal would be to reduce the blood pressure by 15%.

Recommendations for blood pressure control have been established regarding patients undergoing fibrinolytic therapy. The recommendations include a gradual approach to reducing the pressure below 185/110 mm Hg to qualify for fibrinolytic therapy with intravenous recombinant tissue plasminogen activator (rt-PA). Once intravenous (rt-PA) is given, the blood pressure must be maintained below 180/105 mm Hg to reduce the risk of intracerebral haemorrhage [16].

Antihypertensives given when considering re-perfusion therapy include IV Labetalol 10–20 mg over 1–2 minutes, may be repeated once, IV Nicardipine 5 mg/h, titrating up by 2.5 mg/h every 5–15 minutes, maximum 15 mg/h; when desired BP is reached, adjust to maintain proper BP levels. Other drugs such as hydralazine, enalaprilat, etc. may be considered where necessary. Do not administer rt-PA if the blood pressure is not maintained at or below 185/110 mm Hg. Blood pressure should be monitored every 15 minutes for 2 hours from the start of rt-PA therapy, then every 30 minutes for 6 hours, and then every hour for 16 hours. If the blood pressure still remains uncontrolled or diastolic BP >140 mm Hg, consider IV sodium nitroprusside [16].

5.5. Management of blood sugar

Hyperglycaemia occurs in about 20–40% of acute stroke patients with no previous diagnosis of diabetes mellitus [35]. There is overwhelming clinical evidence that correlates hyperglycaemia at the onset of acute ischaemic stroke with a negative outcome [36]. Hyperglycaemia influences neuronal damage by encouraging anaerobic metabolism and lactic acidosis within the ischaemic tissue, thus worsening outcome and heightening the risk of haemorrhagic transformation after thrombolysis [37]. Hyperglycaemia should be treated with insulin to achieve a blood sugar control between 7.7 and 10.0 mmol/l with close monitoring to avoid hypoglycaemia [16]. Insulin is indicated in the treatment of hyperglycaemia in acute ischaemic stroke because of its ability to reduce neuronal necrosis regardless of its effect on glucose levels [38].

5.6. Temperature control

About one third of patients presenting with stroke develop fever in the first few hours after stroke onset [39]. Increased body temperature of 37.5°C is associated with poor neurological outcome secondary to increased free radical production, increased metabolic demands and increased release of neurotransmitters [39, 40].

The source of the fever should be determined. Some of the possible causes of the fever include aspiration pneumonia and other respiratory infections, urinary tract infections or line infections, infective endocarditis, deep vein thrombosis/pulmonary embolism and cocaine intoxication. The guideline for the early management of acute ischaemic stroke recommends the lowering of temperature during the acute stroke period. Fever is managed strictly with antipyretics and appropriate antibiotics given if infection is suspected. The most frequently used antipyretic is acetaminophen. Aspirin, ibuprofen and indomethacin have also been considered in patients with reduced risk of bleeding [41].

5.7. Antiplatelet therapy

The commonly used antiplatelets include aspirin, clopidogrel and dipyridamole. The use of aspirin in acute ischaemic stroke was examined in CAST (the Chinese Acute Stroke Trial) and IST (the International Stroke Trial). In IST study, aspirin at a dose of 300 mg/day was found to reduce stroke recurrence within the first 14 days with no effect on early mortality. In the CAST study, aspirin 160 mg/day reduced the risk of recurrence and mortality in the first

28 days. Clopidogrel at a dose of 75 mg was found to have a risk reduction of 8.7% in the prevention of cerebrovascular and cardiovascular events [42]. Various studies have shown that the combination of dipyridamole and aspirin is superior to aspirin alone as an antithrombotic therapy after cerebral ischemia of arterial origin [43, 44].

5.8. Anticoagulant therapy

Heparin is not indicated for routine use in the treatment of acute ischaemic stroke. Some of the indications for its use include cerebral venous thrombosis, acute infarct with high grade carotid stenosis, cardiogenic emboli with high risk of recurrence, hypercoagulable states such as protein C deficiency, protein S deficiency, antithrombin III deficiency and antiphospholipid antibody syndrome. Other anticoagulants include warfarin which is useful in the prevention of stroke recurrence in atrial fibrillation patients [45]. Dabigatran has also been found to reduce the occurrence of stroke among non-valvular atrial fibrillation patients [46].

5.9. Statins

Statins have been observed to be efficacious in both primary and secondary prevention of stroke independent of cholesterol levels. This might be due to other beneficial effects of statins such as stabilization of atherosclerotic plaques, improvement of endothelial function, antioxidant properties, increased nitric oxide bioavailability, inhibition of inflammatory responses and immunomodulatory actions [47, 48]. The use of Statin early in stroke patients has been found to be strongly associated with improved post stroke survival, and discontinuation of statin, even for a brief period, has been associated with worsened survival [49].

6. Thrombolysis

The goal of reperfusion therapy for acute ischaemic stroke is prompt restoration of blood flow to regions of brain that are ischemic but not yet infarcted. This reduces the volume of brain damage, reduces oedema and improves outcome. The use of intravenous recombinant tissue plasminogen activator was approved by the US FDA in 1996 for use in acute ischaemic stroke patients presenting within 3 hours of stroke onset [50]. Its use is associated with favourable outcomes although increased risk of intracranial haemorrhage has been observed [16].

Intravenous recombinant tissue plasminogen activator (IV rt-PA) is administered when an acute stroke patient meets all of the inclusion criteria and none of the absolute exclusion criteria. The dose of the IV rt-PA is 0.9 mg/kg (maximum dose 90 mg). Infuse 0.9 mg/kg over 60 minutes, with 10% of the dose given as a bolus over one minute. Patient should be admitted into the intensive care unit or stroke unit for close monitoring. The infusion should be discontinued if the patient develops severe headache, acute hypertension, nausea, or vomiting or has a worsening neurological examination. An emergent CT scan should be requested for. Blood pressure monitoring and neurological assessment are carried out every 15mins during and after the administration of the IV rt-PA infusion for 2 hours, then every 30 minutes for 6 hours, then hourly until 24 hours after IV rt-PA treatment [16].

Inclusion criteria
Diagnosis of ischemic stroke causing measurable neurological deficit
Onset of symptoms <3 hours before beginning treatment
Aged ≥18 years
Exclusion criteria
Significant head trauma or prior stroke in previous 3 months
Symptoms suggest subarachnoid haemorrhage
Arterial puncture at non-compressible site in previous 7 days
History of previous intracranial haemorrhage
Intracranial neoplasm, arteriovenous malformation, or aneurysm
Recent intracranial or intraspinal surgery
Elevated blood pressure (systolic >185 mm Hg or diastolic >110 mm Hg)
Active internal bleeding
Acute bleeding diathesis, including but not limited to
Platelet count <100,000/mm^3
Heparin received within 48 hours, resulting in abnormally elevated aPTT greater than the upper limit of normal
Relative exclusion criteria
Recent experience suggests that under some circumstances—with careful consideration and weighting of risk to benefit—patients may receive fibrinolytic therapy despite 1 or more relative contraindications. Consider risk to benefit of IV rt-PA administration carefully if any of these relative contraindications are present:
Only minor or rapidly improving stroke symptoms (clearing spontaneously)
Pregnancy
Seizure at onset with postictal residual neurological impairments
Major surgery or serious trauma within previous 14 days
Recent gastrointestinal or urinary tract haemorrhage (within previous 21 days)
Recent acute myocardial infarction (within previous 3 months)

Adapted from Guidelines for the early management of patients with acute ischaemic stroke. Stroke 2013.

The checklist includes some FDA-approved indications and contraindications for administration of IV rt-PA for acute ischemic stroke. Recent guideline revisions have modified the original FDA-approved indications. A physician with expertise in acute stroke care may modify this list.

Onset time is defined as either the witnessed onset of symptoms or the time last known normal if symptom onset was not witnessed.

In patients without recent use of oral anticoagulants or heparin, treatment with IV rt-PA can be initiated before availability of coagulation test results but should be discontinued if INR is >1.7 or PT is abnormally elevated by local laboratory standards.

In patients without history of thrombocytopenia, treatment with IV rt-PA can be initiated before availability of platelet count but should be discontinued if platelet count is <100,000/mm^3.

aPTT indicates activated partial thromboplastin time; CT, computed tomography; ECT, ecarin clotting time; FDA, Food and Drug Administration; INR, international normalized ratio; IV, intravenous; PT, partial thromboplastin time; rt-PA, recombinant tissue plasminogen activator; and TT, thrombin time.

Table 1. Inclusion and exclusion characteristics of patients with ischemic stroke who could be treated with IV rtPA within 3 hours from symptom onset.

Inclusion criteria
Diagnosis of ischemic stroke causing measurable neurological deficit
Onset of symptoms within 3 to 4.5 hours before beginning treatment
Relative exclusion criteria
Aged >80 years
Severe stroke (NIHSS>25)
Taking an oral anticoagulant regardless of INR
History of both diabetes and prior ischemic stroke

INR indicates international normalized ratio; IV, intravenous; NIHSS, National Institutes of Health Stroke Scale; and rt-PA, recombinant tissue plasminogen activator.

Table 2. Additional inclusion and exclusion characteristics of patients with acute ischemic stroke who could be treated with IV rt-PA within 3 to 4.5 hours from symptom onset.

Placement of nasogastric tubes, indwelling bladder catheters, or intra-arterial pressure catheters should be delayed if the patient can be safely managed without them. A follow-up CT or MRI scan should be obtained at 24 hours after IV rt-PA before commencing anticoagulants or antiplatelets (**Tables 1** and **2**).

7. Endovascular treatments

This modality of treatment for acute ischaemic stroke is fast emerging. Mechanical clot retrieval with MERCI device (Mechanical Embolus Removal in Cerebral Ischaemia) has been employed. Other interventions have included mechanical clot aspiration with the Penumbra system. The Penumbra System (PS) is a new embolectomy device specifically designed to remove the thrombus in acute ischemic stroke secondary to large vessel thromboembolism. The device removes the thrombus through two mechanisms: aspiration and extraction [16]. Earlier trials, Trevo versus Merci Retrievers for Thrombectomy Revascularization of Large Vessel Occlusions in Acute Ischaemic Stroke (TREVO 2) and SWIFT, showed significantly higher recanalization rates associated with stent retriever devices compared to the first generation Merci Retriever [51, 52].

8. Complications

Patients who have sustained a stroke are prone to developing complications. About 30–60% of patients after acute ischaemic stroke develop these complications. The most frequent complications include respiratory and urinary tract infections, deep vein thrombosis (DVT) and pulmonary embolism (PE) [53]. Pulmonary embolism occurs in about 10% of patients post stroke. Deep vein thrombosis and pulmonary embolism tend to occur in the first three months post

stroke with an incidence of 2.5% and 1.2% respectively [54]. The risk of DVT/PE is increased in immobile and elderly patients with severe stroke [55]. Pressure sores are also common and have been attributed to poor nursing care. Stroke patients should therefore be turned frequently at 2-hourly interval to prevent this complication. Water bed can also be used.

9. Conclusion

Ischaemic stroke remains the commonest type of stroke worldwide. Its successful treatment is dependent on prompt restoration of blood flow to the penumbral tissue. Other supportive therapies have also been helpful in ensuring a favourable outcome. This was an overview of the management of the condition.

10. Intracerebral haemorrhage (ICH)

10.1. Introduction

This refers to bleeding into the brain parenchyma. Intracerebral haemorrhage is a devastating disease with increased morbidity and mortality constituting 15% of all stroke types [56, 57]. Factors associated with increased mortality include large clots, low Glasgow Coma Scale score, intraventricular haemorrhage and haematoma expansion. The causes of haematoma growth include a past history of stroke, liver disease, hyperglycaemia and hypertension [58]. The common sites for ICH include the basal ganglia, thalamus, brain stem and the cerebellum.

10.2. Causes

Causes of ICH have been classified into primary and secondary. Hypertension remains the most common modifiable risk factor for the development of ICH [59, 60] while cerebral amyloid angiopathy is the second most frequent risk factor in ICH leading to lobar haemorrhages . Other risk factors include increasing age, anticoagulation therapy, AV malformations, and aneurysms [61].

10.3. Clinical features

The symptoms are usually sudden in onset; most times occurring during exercise or emotional stress although it can also occur during routine activity [61, 62]. It is difficult, on the basis of clinical presentation, to distinguish ICH from ischaemic stroke as they may look similar.

Presentation of ICH differs depending on the size and location of the ICH. Symptoms that may suggest ICH include severe headache, vomiting, seizures, reduced level of consciousness. Headache is more frequent in patients with large haematomas and has been attributed to raised intracranial pressure and traction on the meningeal pain fibres. Small, deep haematomas rarely present with headache. [61]. About 15–23% of patients tend to have haematoma expansion and neurological deterioration in the first few hours of the event [63, 64].

10.4. Assessment

According to the guideline on the emergency diagnosis and assessment of an ICH patient, the following should be done:

A baseline severity score should be performed as part of the initial evaluation of patients with ICH (Class 1; level of evidence B); rapid neuroimaging with CT or MRI is recommended to distinguish ischaemic stroke from ICH (Class 1, level of evidence A); Computed Tomography Angiography (CTA) and contrast-enhanced CT may be considered to help identify patients at risk for hematoma expansion (Class 11b, level of evidence B) and CTA, CT venography, contrast-enhanced CT, contrast-enhanced MRI, MR angiography and magnetic resonance venography, and catheter angiography can be useful to evaluate for underlying structural lesions including vascular malformations and tumours when there is clinical or radiological suspicion (Class 11a, level of evidence B).

10.4.1. Diagnosis

Rapid diagnosis is essential in the management of the condition. Deterioration in the first few hours after onset has been reported due to haematoma expansion [64].

Initial assessment will include stabilization of patient by maintaining the airway. General physical examination and quick neurological examination should be performed on all patients. Vital signs should be measured. Baseline severity scale score like ICH score, Glasgow coma scale (GCS), NIHSS should be employed. The ICH score is a simple clinical grading scale, reliable and validated for rapid evaluation of ICH severity [63].

10.5. Investigations

Brain non- contrast CT Scan (NCCT) - this is the goal standard in diagnosing ICH. It is convenient and highly sensitive in the detection of ICH [65]. Other useful information that can be extracted from NCCT includes the location of ICH, intraventricular bleed, hydrocephalus, early signs of herniation, lesional oedema, and midline shift. ICH volume, a strong predictor of outcome can also be estimated. Brain MRI can help in identifying the exact neuroanatomic site as well as the aetiology [66]. Other investigations are same as for ischaemic stroke.

10.6. Management

Airway management is similar to that of acute ischaemic stroke.

10.6.1. Peri-haematomal oedema

This occurs in the first few days after intracerebral haemorrhage. It is significantly associated with hematoma expansion, increased intracranial pressure, mass effect, midline shift and brain herniation leading to poor functional outcome of ICH [67–69]. Agents that can reduce peri-haematomal oedema process provide protective effects for ICH. These include the use of osmotic diuretics such as Mannitol. Hypertonic saline can also be used. An earlier retrospective study

had reported rapid reversal of transtentorial herniation and decreased intracranial pressure with the use of 23.4% of hypertonic saline [70]. Another study had observed the superiority of hypertonic saline over mannitol in the treatment of increased intracranial pressure [71]. The routine use of mannitol in small ICH and asymptomatic peri-haematoma oedema should be discouraged.

10.6.2. Seizures

These are common in ICH occurring in up to 16% of the cases in the first week with most occurring at onset [72]. Lobar haematomas carry an increased risk of seizures than deep ICH [73]. A recent AHA/ASA guideline for management of spontaneous ICH recommend the use of antiepileptic drugs only in patients with clinical seizures and those with depressed mental status found to have electrographic seizures on EEG [74]. Drugs that have been used include intravenous Lorazepam (0.05–0.1 mg/kg), fosphenytoin (or Phenytoin 15-20 mg/kg), and valproic acid (15-45 mg/kg).

10.6.3. Hyperglycaemia

Hyperglycaemia at presentation portends a worse outcome. This is independent of diabetes mellitus [75]. Treatment involves the use of Insulin. Hypoglycaemia should be avoided.

10.6.4. Deep vein thrombosis

Symptomatic deep vein thrombosis occurs in 1–5% of patients with ICH with pulmonary embolism occurring in about 0.5–2% of such cases. It is therefore crucial to prevent both DVT and PE [76]. Prophylaxis for DVT includes the use of intermittent pneumatic compression devices (IPC) or compression stockings if IPC devices are not available. Subcutaneous low-dose unfractionated heparin can be used when the intracranial bleeding has been controlled within 48 hours of the admission [74].

10.6.5. Blood pressure

Lowering of blood pressure in the setting of ICH has been frequently practiced to reduce haematoma growth. However, the association between elevated BP and hematoma expansion remains controversial. An increasing blood pressure has been associated with haematoma expansion. The AHA/ASA guidelines recommend mean arterial Pressure of 130 mmHg. Titratable antihypertensive drugs such as Intravenous Labetalol (10–20 mg IV bolus, can be repeated up to max of 60 mg) and Nicardipine (5 mg/h up to 15 mg/h) are often used in acute ICH. Nitroprusside should be avoided because of its tendency to increase ICP.

10.6.6. Fever

This is a frequent occurrence in patients with ICH especially in those with intraventricular extension. Patients with persistent fever after ICH tend to have a worse prognosis [77].

10.6.7. Haemostatic therapy

The outcome of ICH is made worse by coagulopathy as this causes expansion of haematoma. Coagulopathy should therefore be reversed. Intravenous Vitamin K 10 mg and fresh frozen plasma 20 ml/kg can be given to patients with Warfarin related ICH. Alternatives to fresh frozen plasma include prothrombin complex concentrate and activated factor VII (Novoseven) [78]. Although recombinant factor VIIa was shown to be efficacious in reducing haematoma growth in phase II trial, it failed to demonstrate consistency in efficacy in subsequent trials. It is often used in patients with ICH associated haemophilia.

10.6.8. Intraventricular haemorrhage and hydrocephalus

About 45% of patients with intracerebral haemorrhage (ICH) develop intraventricular haemorrhage (IVH). ICH often predicts a poor outcome. There are two types of IVH; the primary –confined to the ventricles and secondary due to extension of an ICH. Secondary IVH is the commonest and is related to haemorrhages from hypertension involving the basal ganglia and the thalamus [79]. Treatment involves the use of intraventricular administration of rt-PA or urokinase. This was found to reduce mortality and morbidity by increasing blood clearance and clot lysis [80]. Unfortunately, the procedure was not without the risk of intracranial bleeding [81]. Other treatment options included an endoscopic surgical evacuation and ventriculostomy, ventriculoperitoneal shunting or lumbar drainage for hydrocephalus [82–84].

10.6.9. Surgical intervention

Controversies exist over the role of surgical haematoma evacuation. The International Surgical Trial in Intracerebral haemorrhage (ISTICH) and subsequent STICH 11 demonstrated no improvement for early haematoma evacuation in patients with supratentorial ICH [85, 86]. However in subgroup analysis, patients with superficial haematomas were more prone to a favourable outcome when managed surgically compared to deep ICH. In contrast to supratentorial haematomas, cerebellar ICH is a neurosurgical emergency requiring urgent evacuation as rapid deterioration can occur in the first 24 hours of onset. Indications for surgical intervention include haemorrhages greater than 3 cm and those with brainstem compression or hydrocephalus [87].

10.7. Stroke recovery and rehabilitation

Advancement in the treatment of acute stroke and the establishment of dedicated stroke units has led to an increase in the survival of stroke patients. Many of the survivors experience persistent difficulty in their activities of daily living. Moderate functional impairment has been observed in 40% of stroke patients with about 15–30% having severe disability [88]. Early initiation of effective rehabilitation post stroke has been found to enhance recovery process and minimize functional disability. Stroke rehabilitation is therefore crucial for recovery post stroke.

The services of rehabilitation involve a multidisciplinary approach comprising healthcare providers with training in neurology, rehabilitation nursing, physical therapy, occupational therapy and speech and language therapy. Other health professionals who play key roles in rehabilitation include social workers, psychologists, psychiatrists and counselors [89].

Stroke rehabilitation usually commences during the acute hospitalization when the patient has been stabilized medically and neurologically. The major concern in the acute phase are prevention of a recurrent stroke, prevention of complications, mobilizing the patient, promoting resumption of activities of daily living as well as providing emotional support to the patient and family. Thereafter the focus shifts to evaluation and recovery of any residual physical and cognitive deficits [90].

A patient with stroke is at risk of developing joint and muscle contractures. The reasons for this are multifactorial and include hemiparesis, impaired sensation, reduced level of consciousness, older age, incontinence and pressures sores. Early rehabilitation can reduce the contractures.

10.8. Conclusion

Recent advances in neuroimaging, organized stroke care, dedicated Neuro-ICUs, medical and surgical management have changed the management of ICH. Early airway protection, blood pressure control, rapid reversal of coagulopathy and surgical intervention may increase the chance of survival for patients with severe ICH.

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