Management of Stroke: Nursing Perspective

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Objectives

- Identify the main areas of nursing care for a patient who has a stroke
- Identify the knowledge and skills the nurses need in the care of a stroke patient
- Identify the behavioral characteristics of right brain–damaged stroke patients

Nurse’s Role

- Nurses play a significant role in stroke care
- Application of knowledge and nursing skills
  - Regular education program for nursing staff
  - Nursing practice is a reflection of their knowledge and skill sets
- Dedicated stroke unit
### Nursing Care
- Monitor patient 24/7 – ongoing basis
- Observe and report any physiological and psychological status of patient
- Need specialized training
  - Communication skills
  - Expertise in acute stroke practice

### Acute Stroke Care
- Careful monitoring
  - Could be hourly, every two or four hours
- Observe for any neurological deterioration
- Avoid complications

### Acute Stroke Care
Physiological parameters to be monitored
- Level of consciousness
- Oxygen saturation
- Blood pressure
- Blood glucose
- Hydration
- Pulse/heart rhythm
- Temperature
Knowledge and Skills

- Neurological assessment
- Blood pressure assessment
- Temperature assessment
- Respiratory assessment
- Swallow screen
- Blood glucose assessment
- Language and speech assessment
- Visual and spatial neglect
- Cognitive assessment
- Mobility assessment
- Urinary /bowel care
- Post-stroke depression

General Supportive Care

- Hypoxemia and hypotension should be avoided
- Prevent further cellular damage
- Administer Oxygen to hypoxemic patients to maintain O2 saturation > 94%
- Those at risk for airway obstruction, aspiration, or elevated ICP: head of bed elevated at 15° to 30°

General Supportive Care

Blood Pressure:
- Usually decreases spontaneously within 90 minutes after stroke
- Moderate hypertension:
  - could be beneficial in the acute stage by improving cerebral perfusion of ischemic tissues
  - Or
  - detrimental by exacerbating edema and hemorrhagic transformation
- Systolic blood Pressure > 220 mmHg or diastolic pressure > 105 mmHg increases the risk of hemorrhagic transformation
**General Supportive Care**

- Extreme arterial hypertension:
  - Leads to encephalopathy
  - Cardiac complications
  - Renal insufficiency
- Hypotension is detrimental by decreasing perfusion particularly the brain expanding ischemic injury

**Recommendation**

- Do not lower BP during the initial 24 hours of AIS unless BP is >220/120 mm Hg or with co-morbidity that would benefit lowering of BP (MI, HF, aortic dissection)

**Glucose Management**

- Hyperglycemia:
  - Associated with increased edema in patients with cerebral ischemia and increased risk of hemorrhagic transformation
- Recommendations:
  - Avoid hyperglycemia/hypoglycemia
  - Glucose levels: 140 – 180 mg/dl

Note: A study showed that aggressive control (glucose <126 mg/dl or <7 mmol/L increased infarct size)

**Hemodynamic Support**

- Fluid management
  - Isotonic saline
  - Avoid hypotonic hypo-osmolar fluids (D5W, D10W, LRSNS, D5 1/2NS, D10 1/2NS)
  - Fluids without dextrose are preferred
Neurological Assessment

- Level of consciousness
- Cognitive status: arousal, alertness, and orientation
- Glasgow Coma Scale (GCS)
- National Institute of Health Stroke Scale (NIHSS)

**Neurological Assessment**

- Level of consciousness
  - First change noted if patient is deteriorating
  - Could be subtle changes
  - If noted early by nurse, could notify the physician and change in LOC addressed
- Perform a head to toe neuro assessment at the beginning of the shift to obtain a baseline, monitor at frequent intervals allowing nurse to observe changes

**NIHSS**

- Helps quantify the degree of neurological deficits
- Facilitate communication
- Identify location of vessel occlusion
- Provide early prognosis
Neurological Complications

- 25% of patients deteriorate after initial stroke
- Causes of deterioration
  - Stroke progression (1/3)
  - Brain edema (1/3)
  - Hemorrhage (10%)
  - Recurrent ischemia (11%)

Hence the need for close observation and monitoring in a dedicated stroke unit

Mechanism of Brain Edema

Brain swelling occurs as a result of:

- Loss of transport of membrane transporters
- $Na^+$ and $H_2O$ influx into necrotic or ischemic cell
- Cytotoxic edema (peaks 3-4 days after injury)

Further swelling disrupts Blood brain barrier (BBB) → vasogenic edema

Cerebral Edema

- Significant cerebral edema is expected only in large-territory cerebral infarcts

Clinical patterns:
- Rapid and fulminant course – within 24-36 hours
- Gradual progressive course - over several days
- Initial worsening followed by a plateau or resolution - about a week
Cerebral Edema after Stroke

- Most specific sign is a decline in LOC
  - Due to brain edema shifting the thalamus and brainstem affecting the ascending arousal system
- Neurological deterioration usually occurs within 72-96 hours
- Others deteriorate at 4 – 10 days
  - When previously at-risk penumbra progresses to infarction
  - Followed by delayed swelling
  - Or hemorrhagic transformation

Note: If patients are intubated for mechanical ventilation, brain death is a possible outcome if no aggressive measures to relieve swelling are undertaken

Medical Management of Cerebral Edema

- Restriction of free water
- Avoid hypo-osmolar fluid
- Avoid excess glucose administration
- Prevent hypoxemia/hypercarbia
- Treat hyperthermia
- Avoid antihypertensive agents that induce cerebral vasodilatation
- Elevate head of bed at 20° to 30°

ICP Management

- Clinical deterioration is more often the result of displacement of midline structures rather than a mechanism of globally increased ICP
  - ICP is not increased in the early days after presentation with hemispheric infarct
  - In patients with cerebellar stroke early swelling and acute hydrocephalus may occur

Recommendations:
- Routine ICP monitoring is not indicated in hemispheric ischemic stroke
- Ventriculostomy is recommended in obstructive hydrocephalus after a cerebellar infarct but should be followed or accompanied by decompressive craniectomy (Class 1: Level of Evidence C)
ICP Management

- Mannitol 0.25 to 0.5 g/kg IV given over 20 minutes every 6 hours up. Maximum dose is 2g/kg
- Hyperventilation of intubated patients. Target is mild hypocapnia (Pco2 30-35 mm Hg). Benefit is short-lived.

Evolution of Ischemic Stroke

- 70M with altered LOC, Right facial Droop, right-sided weakness
- IV tPA given
- Dc nursing home
- Huge infarct involving Left MCA and Left ACA
- MRI obtained < 24 hours
Hemispheric Infarcts

CT Findings of Cerebral Infarction
- Progressive cerebral edema and mass effect
- Ipsilateral sulcal effacement
- Compression of ipsilateral ventricular system
- Shift of midline structures such as septum pellucidum
- Foramen of Monro or 3rd ventricle might be blocked
- Entrapment and dilatation of the contralateral lateral ventricle
- Obstructive hydrocephalus

CT Imaging
Findings that predict malignant edema and poor prognosis:
- Frank hypodensity within 6 hours
- Involvement of 1/3 or more of MCA territory
- Dense MCA sign or midline shift > 5mm within first 2 days
Hemorrhagic Transformation

- Common complication of severe stroke
- Manifestation of damage to the blood brain barrier (BBB)
- Loss of microvascular integrity
- Disruption of the neurovascular unit
- May be a consequence of recanalization and reperfusion of an infarcted area

Facts about Hemispheric Stroke

- Patients with significant swelling typically have occlusions of the ICA, MCA or both
- Infarctions from MCA branch occlusions typically do not result in swelling with significant mass effect

Other risk factors contributing to edema after ischemia
- Additional vascular territories
- Incomplete circle of Willis
- Marginal leptomeningeal collateral supply

Clinical Features of Hemispheric Stroke

- Hemiplegia
- Global or expressive aphasia
- Severe dysarthria
- Neglect
- Gaze preference
- Visual field defect
Other Significant Features

- Pupillary abnormalities
  - Reflection of significant brainstem shift
  - Typically not seen on initial presentation
  - Develop within 3 to 5 days
- Early Horner syndrome
  - May be due to an acute carotid artery occlusion or dissection
- Dominant hemispheric infarction
  - NIHSS is often > 20
- Non-Dominant hemispheric infarction
  - NIHSS is often > 15

“Obscure” Issues After Stroke

- Anxiety and depression
- Memory problems
- Neglect/inattention either of the person or environment
- Speech problems
- Poor coordination
- Sensory changes
- Sexual issues
- Visual loss
- Fatigue
- Incontinence

Comb and Razor Test

Purpose:
- Screens for unilateral spatial neglect (USN)
- Assess performance on functional activities such as using a comb, razor, or applying makeup
Comb and Razor Test: Procedure

- Ask patient to demonstrate the use of a comb or razor for 30 seconds
- Examiner sits opposite patient and holds the comb up saying: “I would like you to comb your hair, continue combing until I tell you to stop. Do you understand that? OK, now begin”
- Activate the stopwatch as soon as patient takes the comb
- Examiner observes and records the number of stroke on the left and right side of the head. Any strokes that are difficult to categorize are classified as ambiguous.
- At the end of 30 seconds, ask the patient to stop and take the comb from him or her.

Hemispatial Neglect

Painting and Drawing in USN

Kondo et al., (2012)
Cerebellar Stroke

- Can be difficult to diagnose when the chief complaints are dizziness, vertigo, and vomiting
- Attention to speech, gait, coordination, and eye movements is required
- Truncal ataxia is often missed during bedside examination
- Swelling after cerebellar stroke may result to:
  - Pontine compression
  - Acute hydrocephalus secondary to fourth ventricle compression

Cerebellar Stroke

Cerebellar Edema

- Most reliable clinical finding of swelling is decreased level of consciousness
- With pontine compression:
  - Ophthalmoparesis
  - Breathing irregularities
  - Cardiac dysrhythmias
- Peak swelling may occur days after the ischemia
Supportive Measures

Indications for endotracheal intubation

- Persistent or transient hypoxemia
- Upper airway obstruction with pooling of secretions
- Apneic episodes
- Development of hypoxic or hypercarbic respiratory failure evidenced by ABGs
- GTC seizures
- Recent aspiration

Recognition of Deterioration

- Signs of supratentorial deterioration
  - May present in two ways
    - Gradual rostrocaudal deterioration
      - Midposition pupils
      - Worsening motor responses
      - Progression to irregular breathing
    - Sudden deterioration
      - Unilateral dilated pupil
      - Progression to bilateral pupil dilation
      - Diminished motor response from localization to flexion rigidity

- Signs of brainstem dysfunction
  - Abnormal respiratory patterns
    - Central neurogenic hyperventilation
    - Apneustic breathing
    - Ataxic breathing
  - Posturing

Medical Options

Measures to reduce ICP

- Elevation of the head of bed to 30 degrees
- Osmotic therapy
  - Mannitol: single dose or as recurrent bolus or both
    - 15 g once
    - 0.5 to 1 g/kg
    - 1g/kg
    - 0.5 g/kg every 4 to 6 hours
  - Hypertonic saline – varying doses and concentration (3%, 7.5%, 23%)
- Hypothermia has been used but still with insufficient data
Neurosurgical Options

Recommendations:

- Decompressive craniectomy with dural expansion:
  - <60 years old, with unilateral MCA infarct who deteriorate neurologically within 48 hours despite medical therapy
  - Efficacy for > 60 years old is uncertain

- Suboccipital craniectomy with dural expansion:
  - Performed in patients with cerebellar infarctions who deteriorate neurologically despite maximal medical therapy

THANK YOU!

References


