Neurological Changes: Case Based Problem Solving

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Patricia Benner’s Nursing Theory
Completing the rite of passage from novice to expert is not a forgone conclusion. Responding to patient needs goes beyond “knowing what” to do and develops into “knowing what” and “knowing why.”
OBJECTIVES

- Review distinct stroke mechanisms
- Explain waxing and waning syndromes and the implementation of appropriate nursing actions and interventions
- Discuss the anticipation, assessment and appropriate nursing actions in stroke patients with neurological decline
DISTINCT STROKE MECHANISMS

Trial of Org 10172 in Acute Ischemic Stroke - TOAST
Waxing and Waning

• To alternatingly increase (wax) and decrease (wane)
• Often referred to as “stuttering”
• What stroke mechanisms have the potential to demonstrate waxing and waning symptoms?
  – Small vessel disease
  – Large artery vasculopathy
• Mediating factors
  – Hemodynamics (Blood pressure, volume status)
  – Collateral circulation
  – Postural changes
• Importance
  – Clock restarts when symptoms resolve
  – May need repeat assessment for swallow and mobility
  – Patient education to prevent recurrence
  – Can we really stop the stuttering and prevent neurological deterioration?
47- year-old male with PMHx of HTN, HLD, CAD, and STEMI (s/p stent 2017) had acute onset of right sided weakness and slurred speech while at work (night shift). EMS was activated and he was transported to the hospital as a stroke alert. He had some improvement in his symptoms during transport.

- 0000: LKW
- 0042: ED Arrival
- 0050: NIHSS = 2
- CT head was negative for hemorrhage. CTA was negative for large vessel occlusion. IV alteplase was prepared, patient was medicated several times for elevated blood pressure (outside of parameters to start alteplase). During this time his symptoms resolved with a NIHSS of 0. Decision was made to hold off on alteplase.
- 0127: NIHSS = 0  all symptoms completely resolved
- 0132: His symptoms returned and worsened to a NIHSS of 10 for right sided weakness, facial palsy, and dysarthria.
Case 1 - Problem solving
What is the next best step?

A. Call the MD, explain to the family that patient waxing and waning is common with this type of stroke and the patient will likely get better soon.

B. Check BP, increase the HOB to 45 degree, call MD

C. Check BP, lay patient flat, prepare for possibility of alteplase administration

D. Repeat the CTA, transfer patient to the ICU
What actually happened...

- Alteplase was left mixed and ready for infusion
- Neuro checks were ordered every 15 minutes
- When symptoms recurred bedside nurse notified MD
  - BP was within parameters
  - IV alteplase was initiated
- Follow-up MRI revealed acute ischemia in the left internal capsule
- The etiology of his stroke is small vessel disease related to known vascular risk factors
- He was discharged on dual antiplatelets (for 21 days, then aspirin only) and statin for secondary stroke prevention. He was counseled extensively on risk factor modification.
Case Study 1: Summary

What stroke syndrome is this?
- Capsular warning syndrome

Teaching Points
- Frequent neurological monitoring
- Clear time stamp for resolution of symptoms
- Alteplase ready and BP within parameters when symptoms returned
Case Study 2

• 62 yo male with no known medical history, no PCP.
• Awoke in his normal state of health at 0500 and later developed acute onset of dysarthria and right arm and leg weakness while driving to work. He pulled over and activated EMS.
• 0600: LKW
• 0741: ED arrival
• 0759: NIHSS = 6
• CT head was negative for hemorrhage. CTA was negative for large vessel occlusion.
• Patient reported a similar episode ~1 month earlier, lasting ~10 minutes, he did not seek any medical care
• 0804: IV thrombolytics initiated
• 0945: NIHSS = 0
• 1130: NIHSS = 7
Case 2 - Problem solving
What is the next best step?

A. Check BP, treat with prn Labetalol for SBP> 160

B. Lay flat, make NPO, call MD, confirm IV access

C. No action the patient has already received maximal treatment with thrombolytics

D. Prepare to give oral Clopidogrel
Case Study 2

- MD notified of patient initial improvement post-alteplase and then worsening of dysarthria and right sided weakness
- Fluid bolus and flat bed rest
- 1330: NIHSS = 4
- Follow-up MRI revealed acute ischemia in the left pons.
- The etiology of his stroke is small vessel disease related to new diagnoses of hypertension and hyperlipidemia, and tobacco use.
- He was discharged on dual antiplatelets (for 21 days, then aspirin only) and statin for secondary stroke prevention. He was counseled extensively on risk factor modification

What actually happened...
Case Study 2: Summary

What stroke syndrome is this?
• Pontine warning syndrome

Teaching Points
• Ascertaining history of similar symptoms ~1 month earlier (this was documented on arrival to ED)
• Frequent neurological monitoring post-alteplase with prompt notification to MD of resolution and then worsening of symptoms
Summary: Stuttering Lacunar Stroke (SLS)

• Clinical characteristics
  • Rapidly improving and recurring symptoms
• Most Common SLS
  • Pontine Warning Syndrome
  • Capsular Warning Syndrome
• Pathophysiology underlying SLS presentation is unknown

• Data is limited on therapeutic approaches
  • Dual antiplatelets
  • Clopidogrel alone
  • Blood pressure augmentation
  • Postural changes
## Communication and Care Plan

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<th>Actions and Interventions</th>
<th>Patient/Family Education</th>
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<tr>
<td>Stroke risk factors</td>
<td>Current NIHSS</td>
<td>Frequent neurological assessment</td>
<td>Do not delay giving ASA/Plavix as ordered (no alteplase). Request PR ASA if needed</td>
<td>Stuttering course</td>
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<tr>
<td>LKW</td>
<td>Symptom resolution or stuttering</td>
<td>Hemodynamics</td>
<td>Lay flat with neurological decline and notify MD.</td>
<td>Lay down and notify nurse if worsening symptoms</td>
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<td>Chief complaint</td>
<td>Imaging results</td>
<td>Communicate and implement MD orders for BP parameters and neurological changes</td>
<td>• Individualized risk factors for lacunar stroke</td>
<td>• HTN, HLD, DM, and smoking</td>
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<td>Arrival NIHSS</td>
<td>Swallow assessment</td>
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<td>Symptom pattern</td>
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<td>Did they receive an intervention?</td>
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• 79 year old female with PMHx of HTN and dementia had acute onset of slurred speech and right sided facial droop, EMS was activated
• 1235: ED arrival
• 1254: NIHSS = 0
• On arrival to ED her symptoms had resolved. CT negative for hemorrhage. CTA near occlusive stenosis of left M1 segment of left MCA with robust collateral flow distally
• Due to high risk for stroke, she was admitted overnight for observation
• Recommendation keep patient flat as possible (allow to sit up for meals), continue permissive hypertension and load with aspirin 325 mg.
• 1859: MRI negative for stroke
• 1235 next day: Patient discharged with dual antiplatelets for 90 days (then ASA only) and statin for stroke prevention and her usual antihypertensives
• 1715: At home, acute onset right sided weakness and aphasia, EMS was activated
• 1817: ED arrival
• 1830: NIHSS = 6, BP 107/74
• CT negative for hemorrhage. CTA revealed occlusion of distal left M1 segment. Flow seen within distal left MCA branches suggesting a high-grade stenosis.
Case 3 - Problem solving
What is the next best step?

A. Keep HOB flat and prepare to administer IV fluids

B. Allow permissive hypertension to perfuse across the MCA stenosis

C. Get MRI scan, patient is not a candidate for any intervention

D. Keep HOB flat and prepare to administer IV thrombolytics and possible Cath lab
What actually happened...

- Patient was a candidate for IV thrombolytics
- 1858: IV alteplase was initiated
- 1920: NIHSS = 2
- IV fluids for BP augmentation
- Follow-up MRI revealed left frontal and temporal lobe acute ischemia
- The etiology of her stroke is intracranial atherosclerosis related to significant M1 stenosis and low blood pressure on admit creating a low flow over this significant stenosis
- She was discharged on dual antiplatelets (for 90 days, then ASA only) and statin for secondary stroke prevention. Antihypertensive dose was lowered.
Case Study 3

What stroke mechanism is this?
• Intracranial atherosclerosis

Teaching Points
• Family re-activating EMS for new onset of weakness and aphasia
• At first discharge consider holding antihypertensives to allow perfusion across the stenosis
• Avoid sudden drops in blood pressure
Summary: Large Artery Atherosclerosis

• Clinical characteristics
  • Crescendo TIA’s in same vascular territory
  • Waxing and waning symptoms related to postural changes and acute lowering of blood pressure (flow dependency)
  • Cerebral cortical impairment (aphasia, neglect, unilateral motor or sensory impairment)
  • Monocular vision loss
  • Acute speech deficit or change
  • Facial droop
  • Brain imaging (CTA) significant for (>50%) stenosis or occlusion of major intracranial artery (MCA, ACA, PCA,) or extracranial artery (ICA, VA) or branch cortical artery (presumably due to atherosclerosis)

• Therapeutic Approaches
  • Hyperacute – IV thrombolytics
  • Medical Management
    • Antiplatelets
      • Findings from CHANCE and POINT trials suggest DAPT may reduce the risk of further neurologic events in symptomatic patients with a minor stroke or TIA more than treatment with ASA alone (21 days)
    • Statin
      • Long term benefits of statin for patients at risk of atherosclerotic events is well established.
  • Surgical/Endovascular Treatment
    • The benefit of carotid intervention within two weeks of index event has been associated with a greater secondary preventative benefit, with a longer delay associated with less benefit to patient
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<td>Frequent neurological assessment</td>
<td>Do not delay giving ASA/Plavix as ordered (no alteplase). Request PR ASA if needed</td>
<td>Intracranial stenosis has a high risk of recurrence</td>
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<td>LKW</td>
<td>Symptom resolution or stuttering</td>
<td>Hemodynamics</td>
<td>Lay flat with neurological decline and notify MD</td>
<td>Long periods of sitting up in chair or ambulation can lead to worsening symptoms</td>
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<td>Chief Complaint</td>
<td>Imaging results</td>
<td>Communicate and implement MD orders for BP parameters and neurological changes</td>
<td></td>
<td>Avoid sudden drops in blood pressure and dehydration</td>
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<td>NIHSS on arrival</td>
<td>Swallow assessment</td>
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<td>Risk factors for atherosclerosis</td>
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Case Study 4

- 74 year-old male with history of CAD (s/p bypass surgery), HTN, HLD, history of stroke 2 years prior from a right ICA critical stenosis.
- He is in the hospital now, post op day #4 from a right carotid endarterectomy performed by vascular surgeon.
- He has been discharged awaiting a ride home.
- CNA becomes alarmed when she finds him in his chair “shaking all over”
- RN enters the room and finds him somnolent, confused, with new left sided weakness
- VS: BP 217/100, P 110, R 22, sats 85% (O2 placed)
- Stat CT/CTA head/neck unremarkable
Case 4 - Problem solving
What is the next best step?

A. Administer prn blood pressure medication and prepare to transfer to ICU

B. Gradually lower blood pressure in anticipation of giving alteplase

C. Implement seizure precautions for new onset seizure and anticipate EEG and MRI

D. Lay flat, allow permissive hypertension
Case Study 4

- Blood pressure rapidly lowered to his normal outpatient level
- Admission to the ICU
- Given 200 ml bolus 3% saline IV
- Improved over the next 3 days back to previous baseline

What actually happened...
Case 4 - Problem solving
What is the best explanation?

A. Hypertensive crisis
B. Acute stroke
C. Seizure with Todd’s paralysis due to electrolyte imbalance
D. Reperfusion syndrome
Reperfusion Syndrome

• “Paradoxical and complex phenomenon of exacerbation of cellular dysfunction and increase in cell death after the restoration of blood flow to previously ischemic tissues”

• Who’s at risk
  – Successful emergent endovascular reperfusion of occluded artery
  – Patients undergoing CAS or CEA with chronic severe stenosis

• Mediating Factors
  – Time of ischemia
  – Health of collaterals

• Complications
  – Penumbral damage
  – Ischemia Expansion
  – Hemorrhagic transformation
  – Seizures
  – Malignant cerebral edema
  – Herniation
Summary: Reperfusion Syndrome

• Clinical Characteristics
  • Decreasing LOC
  • Ipsilateral headache
  • Nausea/vomiting
  • Contralateral weakness
  • Seizure

• Pathophysiology
  • Rapidly restoring CPP through thrombolytic administration, mechanical thrombectomy or CAS/CEA
    • produces an inflammatory cascade leading to severe cerebral edema and deterioration of the salvageable penumbra
    • Impairs cerebral autoregulation
    • May lead to development of ICH

• Therapeutic Approaches
  • Lower blood pressure
  • Minimize edema
  • Treat seizure
  • Hold all antithrombotics

![Figure 1: Schematic representation of blood-brain barrier (BBB) changes in reperfusion injury.](image)
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<td>NIHSS post</td>
<td>Frequent neurological assessment</td>
<td>Treat blood pressures out of parameters and reassess response to treatment</td>
<td>Notify nurse of worsening or new symptoms including HA, nausea/vomiting new weakness</td>
</tr>
<tr>
<td>NIHSS and symptom pattern prior to intervention</td>
<td>intervention</td>
<td>Hemodynamics</td>
<td>Call RRT and notify MD of neurological change</td>
<td>Importance of blood pressure control</td>
</tr>
<tr>
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<td>Success of reperfusion</td>
<td>Communicate and implement MD orders for BP parameters and neurological changes</td>
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<tr>
<td></td>
<td>Imaging results</td>
<td>BP parameters will be different based on success of reperfusion</td>
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60 year-old male with PMH of HTN
0230: Presents to the ED with complaints of dizziness since yesterday, accompanied by n/v and occipital headache last two hours
Initial evaluation by ED MD suspects peripheral vertigo (CVA is in differential). CT head negative for hemorrhage. CTA negative for large vessel occlusion. Out of window for IV alteplase
0547: Admitted by hospitalist
1000: CT/CTA imaging reread by Neuroradiologist abrupt occlusion of the peripheral right PICA, concern for acute/subacute right PICA distribution infarct. MD notified and MRI ordered

1154: MRI shows acute infarction within right inferior cerebellum

1602: NIHSS of 2
Day 2
0816: NIHSS of 3
Hospitalist notes that patient symptoms now include slurred speech and right arm ataxia and orders a repeat CT
1435: Repeat CT shows evolving right cerebellar stroke with mild effacement of the fourth ventricle
2135: NIHSS of 5
Case Study 5

- Day 3
- 0852: More sleepy, slurred speech
- 1210: CT shows worsening edema resulting in early upward transforaminal herniation and obstruction of the lower fourth ventricle causing mild to moderate hydrocephalus
- 1500: NIHSS of 10
Case 5 - Problem solving
What is the next best step?

A. Call MD and discuss starting 3% saline
B. Increase HOB, make NPO and call SLP
C. Place the HOB flat to improve cerebral perfusion
D. Activate “rapid response team”
What actually happened...

- RRT activated for change in neurological status
- Patient was emergently taken to OR for emergent right-sided decompressive suboccipital craniectomy and EVD placement
- Patient remained in ICU for 7 days
- Bone flap was replaced and patient was discharged on day 20
Summary: Posterior Fossa Cerebellar Edema

• Clinical Characteristics
  • Large cerebellar stroke (PICA or SCA)
  • Delay prior to decline ~72 hours
  • Acute hydrocephalus, brain stem compression and death
  • Decreasing LOC
  • Cranial neuropathy
  • Bilateral posturing misinterpreted as a seizure
  • Outcomes excellent with early interventions

• Therapeutic Approaches
  • Frequent neurological assessment in an ICU setting
  • Minimize edema
  • Anticipate emergent craniectomy
# Communication and Care Plan

## Review Patient History
- Stroke risk factors
- LKW
- Chief complaint
- Arrival NIHSS
- Symptom pattern
- Did they receive an intervention?

## Current Situation
- Current NIHSS
- Imaging results
- Swallow assessment

## Ongoing Assessments
- Frequent neurological assessment
- Hemodynamics
- Communicate and implement MD orders for BP parameters and neurological changes

## Actions and Interventions
- Osmotherapy
- Anticipate STAT CT and emergent craniectomy with neurological decline

## Patient/Family Education
- High risk of neurological decline
- Notify nurse of worsening or new symptoms including HA, n/v, dizziness, vision changes
- Preparation for OR


Thank You