Comprehensive Clinical Case Study: Acute Ischemic Stroke

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History and Physical

Source of Information

Information was obtained from the patient’s chart and family. The patient is aphasic and is unable to provide accurate information.

Chief Compliant

Unable to obtain from patient due to aphasia. Per son: “I found her slumped over on her chair at home. She was confused, and could not move her right arm or leg.”

History of Present Illness

This is a 73-year old Caucasian female presenting to emergency department (ED) for evaluation of right hemiparesis and confusion. The patient was last seen normal approximately about one hour ago per son, who lives with the patient. She was found in the chair with her crocheting materials in her lap, confused and unable to move the right side of her body. The patient is on coumadin for history of atrial fibrillation. However, coumadin dosing was stopped for five days prior to an eye surgery (for retinal detachment) which she underwent four days ago. Coumadin was restarted 24 hours post surgery at a dose of 5 mg by mouth (PO) daily. The INR on admission is subtherapeutic at 1.3. The patient is currently in atrial fibrillation on the heart monitor. Subjective assessment limited due to expressive aphasia. The family denies history of headache, migraines, trauma, falls, previous cerebrovascular accidents (CVA), transient ischemic attacks, and seizures. No history of pain is identified, except occasional knee pain which Tylenol® is effective for pain relief.

Home Medications
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Metoprolol 25 mg PO BID, lisinopril 20 mg PO daily, coumadin 5 mg PO daily, simvastatin 40 mg PO daily, glyburide 10mg PO BID, multivitamin one tab PO daily, calcium carbonate 600 mg PO BID, acetaminophen 650 mg PO every six hours PRN pain. The patient does not have a history of use of any alternative and complementary therapies.

Medical History

**Childhood illnesses.** None

**Adult illnesses.** Hypertension and hyperlipidemia diagnosed at age 42; diabetes diagnosed at age 45; atrial fibrillation diagnosed at age 69

**Surgeries/Procedures.** Tonsillectomy and adenoidectomy at age of 12; hysterectomy at age 48; axilla abscess debridement at age 55; laproscopic cholecystectomy at age 69; eye surgery for retinal detachment on 10/20/13


**Immunizations.** Up-to-date on immunizations except flu vaccine, which the patient has not received this season. Last tetanus-diphtheria booster received 2010. Pneumonia vaccine received 10/2012.

Personal/Social History

The patient is a retired secretary, and currently enjoys volunteering at a local library. She is widowed and lives at home with her son and his family in a split-level home. Functional status assessment reveals she was independent in activities of daily living (ADLs) and was even able to manage financial bills before this hospitalization. The patient is a non-smoker without history of alcohol or drug use. No concern for any financial stress. Per family, she enjoys crocheting and donating hand-made hats to a local women’s shelter. Other activities include baking, gardening, and spending time with her three grandchildren. She was described by her family as a “proud and
happy grandma” who is the “center of [our] family tree.” No concern for elderly abuse or depression/suicidal history.

**Family History**

The patient’s mother passed away at the age of 84 from a myocardial infarction. She had a history of diabetes, osteoporosis, hypertension, and hyperlipidemia. The father died at the age of 82 from acute respiratory failure secondary to pneumonia. Father’s medical history consisted of hypertension, hyperlipidemia, sleep apnea, seizures, and two CVAs. The patient has two younger sisters who are alive. The 68-year old sister has a history of hypertension, diabetes, and a transient ischemic attack. The other sister, who is 70 years old, has a history of COPD, diabetes, and kidney disease. Cause of death of grandparents is unknown.

**Review of Symptoms**

**General.** Subjective assessment is limited due to expressive aphasia and drowsiness. However, the patient is able to nod yes/no to most questions. Family is at bedside and will also be able to assist with history. The patient is generally in good health. Blood pressure and diabetes are usually well-controlled, though the patient has not had a recent annual physical (last physical assessment in January 2012) and does not routinely check blood pressure or blood glucose levels at home. The patient is independent with medication management. No recent history of chills or fever. Oral and fluid intake adequate; good appetite. No recent weight loss or weight gain.

**Neurological.** Having numbness/tingling in right arm and leg and face. New onset confusion earlier today per family. No light-headedness, tremors, or falls.

**HEENT.** Head: Denies headache. Eyes/Ears: Denies visual or hearing deficits, and tinnitus. No photophobia. Nose: Denies discharge or congestion. Throat: Denies sore throat.

**Neck.** No pain or stiffness.
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**Respiratory.** Denies shortness of breath, coughing, or pain during inspiration/expiration.

**Cardiovascular.** No chest pain, diaphoresis, dizziness, or swelling. Occasional palpitations.

**Gastrointestinal/Abdominal.** Denies hiccups, nausea and vomiting, abdominal pain, constipation/diarrhea. Usual flatulence. Unable to obtain information about bowel movement. No rectal bleeding.

**Genitourinary.** No urinary urgency, frequency, or burning. No history of urinary tract infections. Denies incontinence and flank pain. Unable to obtain information about urine appearance.

**Genitalia.** Deferred. No history of sexually transmitted diseases.

**Musculoskeletal.** Occasional knee pain per family (unable to rate pain level due to aphasia). Denies history of trauma with or without falls. No history of fractures.

**Integumentary.** Denies itching, wounds, blisters, or thinning of nails or hair.

**Psychosocial.** Denies outbursts of anger, anxiety, and social withdrawal/depression.

**Hematologic.** No active bleeding issues. No history of anemia.

**Endocrine.** Denies diaphoresis, increased thirst or urination, irritability, weight loss/gain.

**Physical Examination**

**General.** The patient is alert, appropriately nods yes/no. Height is five feet five inches, weight is 138 pounds, Body Mass Index (BMI) is 23.0 kg/m2 (normal BMI 18.5-24.9 kg/m2).

**Vital signs.** Blood pressure is 175/90 mmHg, left arm sitting; apical pulse is 100 beats per minute and irregular; respiratory rate is 22 breaths per minute with normal depth and effort; temperature is 99.2°F oral; oxygen level of 98% on two liters of oxygen via nasal cannula.
Neurological. Glasgow Coma Scale of 14; National Institute of Health Stroke Scale (NIHSS) score of 17. Drowsy but following commands appropriately on left side. Confusion state is slightly better than this a.m. Expressive aphasia present; attempting to communicate but speech is dysarthric and negligible. Pupils are equal, round, and reactive to light, 3 mm with conjugate gaze, extra-ocular muscles intact with presence of right homonymous hemianopia. Corneal reflex is present. Mild right sided neglect. Face is asymmetrical with right facial droop. Tongue deviation to the right. No ataxia on left side; unable to test ataxia on right side. Attempted to test sensory component, but due to expressive aphasia the patient is unable to answer questions regarding testing. Normal Babinski reflex.

HEENT. Head is normocephalic, no tenderness on palpation. Frontal and maxillary sinuses are non-tender to palpation. Mild periorbital edema on right eye from recent eye surgery, clear conjunctiva. Ears in normal position, tympanic membrane is gray. Clear nares. Lips and mucosa pink and moist. Dentures removed. Delayed gag and cough reflex.

Neck. No pain or jugular venous distension. No thyromegaly or palpable adenopathy. Trachea is midline. Bilateral +2 carotid pulses without bruits.

Chest. No tenderness on palpation. No masses, lesions, crepitus.


Cardiovascular. Bilateral, equal, +2 radial and brachial pulses. Bilateral, equal, +1 posterior tibial and dorsalis pedis pulses. Capillary refill of less than three seconds. Irregular rate
and rhythm, atrial fibrillation on monitor. S3/S4 absent. No murmurs, clicks, snaps, or rubs.

Peripheral edema absent in bilateral upper and lower extremities.

**Abdomen.** Abdomen is flat, non-distended, non-tender, and non-guarding. Normal bowel sounds in all four quadrants. No bruits. Umbilicus midline. No protrusion, rigidity, or ascites. No palpable oragnomegaly or masses. Negative for tenderness at the costovertebral angle.

**Musculoskeletal.** Arms and legs are equal in length. No erythema, crepitation, or spinal tenderness. No kyphosis or scoliosis. Gross and fine sensory and motor strength intact in left upper and lower extremities, 5/5. Right hemiparesis, right upper and lower extremity moves only to deep pain, 1/5.

**Integumentary.** Skin warm and dry. Sutures over right eyelid and right eyebrow from recent eye surgery; site is clean, dry, and intact without erythema. No other lesions, abrasions, rash, or vecchymosis. Nail beds are pink without clubbing. No Lindsey’s nails.

**Laboratory and Radiography Findings**

For patients suspected of having an acute CVA, routine tests are conducted to assist in exclusion of differential diagnosis. Due to the narrow therapeutic window for treatment of acute ischemic CVA, an initial test of computed tomography (CT) without contrast is necessary to eliminate a diagnosis of intracranial hemorrhage (sensitivity and specificity for intracranial hemorrhage is ~100%) (Kranz & Provenzale, 2013). The results of the CT imaging also can eliminate the differential diagnosis of a space-occupying lesion (e.g. brain tumor or abscess). A magnetic resonance imaging (MRI) test may cause a treatment delay for a CVA with an ischemic etiology. The door-to-CT interpretation should be within 45 minutes to optimize patient outcome (American Heart Association/American Stroke Association [AHA/ASA], 2013). Although a cerebral angiography remains the gold standard for complete evaluation of intra-cranial and
extra-cranial vessels in CVA, it is not an appropriate test in an acute situation where fibrinolysis treatment is being considered. A more definitive diagnostic imaging for an ischemic CVA should be considered at a later stage when the patient is more stable (Johnson, 2012). To evaluate if electrolyte abnormalities or hypoglycemia are the potential etiologies of focal neurological deficits, a complete metabolic panel (CMP) with renal function studies are indicated. A complete blood count (CBC) with platelet count is needed to assess if the patient is a candidate for fibrinolysis. In addition, prothrombin time (PT), activated partial thromboplastin (aPTT), and international normalized ratio (INR) are critical to obtain since the patient was taking home medication coumadin before admission (Nazarko, 2013).

Since CVA and acute myocardial infarction (MI) can co-exist and MI may also be the source of embolization, all potential acute CVA patients are also evaluated for cardiovascular disease (AHA/ASA, 2013). Recent episode of MI, especially in transmural MI and a MI involving the anteroapical ventrical wall, may be a source of emboli (Longo et al., 2012). A chest x-ray exam was obtained to estimate degree of cardiac and/or pulmonary decompensation. In addition, a baseline electrocardiogram (EKG) was appropriate in this case to confirm the diagnosis of atrial fibrillation. Cardiac biomarkers assist in identification of concurrent myocardial ischemia and were also obtained. Although troponin level have increased sensitivity (85%) and specificity (90%) and is the preferred biomarker, creatine phosphokinase (CK-MB) was also drawn (Pagana & Pagana, 2010). An echocardiography will also be obtained in a non-acute phase to assess valvular function and the presence of thrombus in the left atrium (Karabay et al., 2013). A lipid panel will also be obtained when the patient has not eaten anything by mouth for at least 12 hours in order to have accurate results.

Table 1. CMP with Creatinine Clearance, Magnesium, and Phosphorus
**Table 2. CBC with Differential**

<table>
<thead>
<tr>
<th>Lab</th>
<th>Results</th>
<th>Normal Values</th>
<th>Lab</th>
<th>Results</th>
<th>Normal Values</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Sodium</strong></td>
<td>138 mEq/L</td>
<td>135-148 mEq/L</td>
<td><strong>Globulin</strong></td>
<td>2.4 g/dL</td>
<td>1.9-3.6 g/dL</td>
</tr>
<tr>
<td><strong>Potassium</strong></td>
<td>3.9 mEq/L</td>
<td>3.4-5.3 mEq/L</td>
<td><strong>A/G Ratio</strong></td>
<td>2.1</td>
<td>0.8-2.6</td>
</tr>
<tr>
<td><strong>Chloride</strong></td>
<td>101 mEq/L</td>
<td>96-110 mEq/L</td>
<td><strong>Total Bilirubin</strong></td>
<td>0.5 mg/dL</td>
<td>0.2-1.9 mg/dL</td>
</tr>
<tr>
<td><strong>Glucose</strong></td>
<td>162 mg/dL</td>
<td>70-100 mg/dL</td>
<td><strong>AST (SGOT)</strong></td>
<td>28 U/L</td>
<td>0-45 U/L</td>
</tr>
<tr>
<td><strong>Potassium</strong></td>
<td>3.9 mEq/L</td>
<td>3.4-5.3 mEq/L</td>
<td><strong>ALT (SGPT)</strong></td>
<td>17 U/L</td>
<td>0-40 U/L</td>
</tr>
<tr>
<td><strong>BUN</strong></td>
<td>12 mg/dL</td>
<td>7-20 mg/dL</td>
<td><strong>Alkaline Phosphatase (ALP)</strong></td>
<td>42 U/L</td>
<td>23-150 U/L</td>
</tr>
<tr>
<td><strong>Creatinine</strong></td>
<td>0.8 mg/dL</td>
<td>0.8-1.4 mg/dL</td>
<td><strong>Magnesium</strong></td>
<td>2.0 mEq/L</td>
<td>1.6-2.4 mEq/L</td>
</tr>
<tr>
<td><strong>BUN/Creat Ratio</strong></td>
<td>15:1</td>
<td>10:1-20:1</td>
<td><strong>Phosphorus</strong></td>
<td>3.0 mEq/L</td>
<td>2.5-5.2 mEq/L</td>
</tr>
<tr>
<td><strong>Calcium</strong></td>
<td>9.0 mg/dL</td>
<td>8.5-10.5 mg/dL</td>
<td><strong>Creatinine Clearance</strong></td>
<td>75 mL/min</td>
<td>88-128 mL/min (female)</td>
</tr>
<tr>
<td><strong>Total Protein</strong></td>
<td>7.0 g/dL</td>
<td>6.0-8.3 g/dL</td>
<td><strong>Albumin</strong></td>
<td>3.9 g/dL</td>
<td>3.5-5.2 g/dL</td>
</tr>
<tr>
<td><strong>Hemoglobin</strong></td>
<td>12.5 g/dL</td>
<td>12.1-15.1 g/dL (female)</td>
<td><strong>Band Neutrophils</strong></td>
<td>2.2%</td>
<td>2-5%</td>
</tr>
<tr>
<td><strong>RBC</strong></td>
<td>4.8 m/mm3</td>
<td>4.2-5.4 m/mm3</td>
<td><strong>Lymphocytes</strong></td>
<td>32.0%</td>
<td>14.0-51.0%</td>
</tr>
<tr>
<td><strong>Hemoglobin</strong></td>
<td>36.0 %</td>
<td>36.1-44.3% (female)</td>
<td><strong>Monocytes</strong></td>
<td>3.3%</td>
<td>2-8%</td>
</tr>
<tr>
<td><strong>Hematocrit</strong></td>
<td>88.4 fL</td>
<td>80.0-100.0 fL</td>
<td><strong>Eosinophils</strong></td>
<td>1.5%</td>
<td>1-3%</td>
</tr>
<tr>
<td><strong>MCV</strong></td>
<td>88.4 fL</td>
<td>80.0-100.0 fL</td>
<td><strong>Basophils</strong></td>
<td>0.5%</td>
<td>0.0-1%</td>
</tr>
<tr>
<td><strong>MCH</strong></td>
<td>28.2 pG</td>
<td>27.0-31.0 pG</td>
<td><strong>Absolute Segmented Neutrophil</strong></td>
<td>5.4 k/mm3</td>
<td>1.5-8.0 k/mm3</td>
</tr>
<tr>
<td><strong>MCHC</strong></td>
<td>34.3 g/dL</td>
<td>32.0-36.0 g/dL</td>
<td><strong>Absolute Lymphocyte</strong></td>
<td>2.6 k/mm3</td>
<td>0.9-4.1 k/mm3</td>
</tr>
<tr>
<td><strong>RDW</strong></td>
<td>11.7%</td>
<td>9.0-15.0%</td>
<td><strong>Absolute Monocyte</strong></td>
<td>0.7 k/mm3</td>
<td>0.2-1.1 k/mm3</td>
</tr>
<tr>
<td><strong>Platelets</strong></td>
<td>267,000 k/mm3</td>
<td>150,000-400,000 k/mm3</td>
<td><strong>Absolute Eosinophil</strong></td>
<td>0.27 k/mm3</td>
<td>0.0-0.6 k/mm3</td>
</tr>
<tr>
<td><strong>Segmented Neutrophils</strong></td>
<td>60.5%</td>
<td>40.0-76.0%</td>
<td><strong>Absolute Basophil</strong></td>
<td>0.18 k/mm3</td>
<td>0.0-0.3 k/mm3</td>
</tr>
</tbody>
</table>
Table 3. Anticoagulation Panel

<table>
<thead>
<tr>
<th>Lab</th>
<th>Results</th>
<th>Normal Values</th>
</tr>
</thead>
<tbody>
<tr>
<td>PT</td>
<td>13.5 sec</td>
<td>11-13.5 sec</td>
</tr>
<tr>
<td>aPTT</td>
<td>29 sec</td>
<td>25-35 sec</td>
</tr>
<tr>
<td>INR</td>
<td>1.3</td>
<td>2-3 for target treatment goal for atrial-fibrillation</td>
</tr>
</tbody>
</table>

(Normal values from Pagana & Pagana, 2010)

Table 4. Additional Laboratory Findings: Troponin, CK-MB, B-type natriuretic peptide (BNP), Homocysteine level

<table>
<thead>
<tr>
<th>Lab</th>
<th>Results</th>
<th>Normal Values</th>
</tr>
</thead>
<tbody>
<tr>
<td>Troponin</td>
<td>&lt;0.01 ng/mL</td>
<td>&lt;0.01 ng/mL</td>
</tr>
<tr>
<td>CK-MB</td>
<td>0.8 mcg/L</td>
<td>0-3 mcg/L</td>
</tr>
<tr>
<td>BNP</td>
<td>62 pg/mL</td>
<td>&lt;100 pg/mL</td>
</tr>
<tr>
<td>Homocysteine level</td>
<td>55 mg/L</td>
<td>&lt;13 µmol/L</td>
</tr>
</tbody>
</table>

(Normal values from Pagana & Pagana, 2010)

Table 4. EKG Findings

**EKG:** Abnormal EKG. Irregular R-R intervals, QRS <0.12, and no P-waves suggestive of atrial fibrillation. Ventricular rate 102 beats per minute.

Table 5. Radiography Findings

<table>
<thead>
<tr>
<th>X-ray of Chest</th>
<th>Normal chest x-ray without infiltrates or atelectasis. No cardiomegaly.</th>
</tr>
</thead>
<tbody>
<tr>
<td>CT Scan of Head without Contrast</td>
<td>No acute intracranial hemorrhage or mass effect noted. No acute disease process.</td>
</tr>
</tbody>
</table>
Diagnosis

The patient is presenting with signs and symptoms of acute ischemic CVA, likely due to cardioembolic etiology. Approximately 85% of CVAs in the United States (U.S.) are ischemic; 15% are hemorrhagic. Stroke is a medical emergency because the disease is the fourth leading cause of death and the leading cause of long-term disability. Early intervention is the basis for better patient outcomes (AHA/ASA, 2013; CDC, 2013). The vigilant efforts of the Healthy People 2010 goals have lead to the success of reduction of incidence of cardiovascular disease, including CVAs (AHA/ASA, 2013).

Computed tomography of head without contrast imaging findings are commonly normal in an acute phase of an ischemic CVA because the infarct may not be evident for two to three days after its occurrence (Johnson, 2012). Therefore, the clinical presentation of the patient and the elimination of other differential diagnoses allows for the establishment of the diagnosis. The clinical presentation of a CVA reflects the extent of damage in the specific intra-cranial or extra-cranial vessel involved (Connell & Hartigan, 2011). The mostly likely vessel involved in this scenario is the left anterior main division of the middle cerebral artery (MCA). Deficit in left MCA leads to contralateral hemiplegia with hemisensory loss and homonymous hemianopia. An insult to the anterior main division of the MCA commonly causes expressive aphasia due to damage in the Broca’s area (Trupe et al., 2013). Drowsiness is common in ischemic CVAs due to reduction in blood flow to the brain. The extent of drowsiness depends on function of collateral blood flow (Connell & Hartigan, 2011).

The pathogenesis of ischemic CVA in this case scenario stems from cardioembolism, which is responsible for 20% of all ischemic CVAs (Longo et al., 2012). The discontinuation of coumadin therapy for five days (prior to the recent eye surgery) without bridging with other
anticoagulants resulted in sub-therapeutic INR and increased risk of emboli formation. Cardiogenic emboli from atrial fibrillation often affect large intra-cranial vessels such as the MCA. Thrombi formed in the atrial wall detach and embolize into the arterial circulation. Ischemia to the brain tissue leads to apoptotic cellular death from the lack of glucose and ATP of mitochondria. The damaged cellular lipid membranes produce free radicals which further cause catalytic destruction (McCance, Huether, Brashers, & Rote, 2010).

The patient has multiple other risk factors for stroke other than atrial fibrillation, including hypertension, diabetes, hyperlipidemia, and advanced age. A high homocysteine level (> 13 µmol/L) is also an independent risk factor for cardiovascular disease. The development of atherosclerosis doubles in patients with high homocysteine levels from the toxic effect of methionine (an amino acid) degradation on intima of the blood vessels (Ashjazadeh, Fathi, & Shariat, 2013). These risk factors are associated with endothelial dysfunction causing arterial wall damage from inflammatory cytokines, cellular proliferation, and abnormal vasomotion modulation (Deanfield, Halcox, & Rabelink, 2008).

**Differential Diagnosis**

One critical differential diagnosis to evaluate in patients suspected of having a CVA is intracranial hemorrhage (ICH) or hemorrhagic CVA. Early elimination of the diagnosis can allow focus on treatment options (i.e. with thrombolytics) for an ischemic CVA. Intracranial hemorrhages are usually associated with spontaneous rupture of an artery from uncontrolled hypertension. Other causes of ICH consist of arteriovenous malformation, cerebral aneurysm, and coagulopathy (McGrath et al., 2012). The focal neurologic deficits of ICH are abrupt in onset with accompanied vomiting, worsen over 30-60 minutes, and are generally more severe in nature than ischemic CVA deficits (fatality rate of ICHs is 50%) (Longo et al., 2012). Deep
drowsiness and coma ensue as the hemorrhage compresses the brainstem. Other clinical manifestations of ICHs are similar to that of ischemic CVA and include contralateral hemiplegia, homonymous visual field defect, aphasia, and sensory loss (Parker, Rhoney, & Xi, 2010). The lack of evidence of acute hemorrhage on the CT of head without contrast findings eliminates the diagnosis of an ICH.

The next potential differential diagnosis to consider is a transient ischemic attack (TIA). The signs and symptoms, risk factors, and causes of TIAs are similar to ischemic CVAs, making differentiation difficult between the two conditions. With a TIA, however, neurological deficits caused by focal brain or retinal ischemia are brief and typically last less than one hour and completely resolve within 24 hours. In an episode of TIA, the occluded blood vessel is reopened (possibly from a free flowing thrombi) resulting in neurologic improvement. The NIHSS score in TIA is usually less than four, illustrating a minor neurologic dysfunction (Ferrari et al., 2010). A common symptom in TIA is amaurosis fugax, or transient ipsilateral monocular blindness caused by an embolus to the central retinal artery. Although mild neurologic deficits are characteristic of TIAs, urgent evaluation is required because the risk of a CVA after a TIA is 10-15% in the first three months, and 50% in the coming years (Longo et al., 2012). The diagnosis of TIA is critical to rule out, especially for patients who are potential candidates for fibrinolysis. Fibrinolysis is contraindicated when neurological improvement is noted on physical examination (AHA/ASA, 2013). The diagnosis of TIA is unlikely in this case because the patient has significant deficits without signs of neurological improvement. The NIHSS score during physical examination was 17 and remained so until treatment was administered.

The third possible diagnosis is a brain tumor. Clinical manifestations vary depending on the type, location, and growth of the mass. Focal neurologic deficits such as hemiparesis, sensory
and visual disturbances, aphasia, agraphia, and ataxia result from compression of neurons and white matter by the expanding tumor and edema (Mogensen, 2008). The signs and symptoms develop gradually and the full presentation of the disease is only evident when the tumor is well advanced. Occasionally, a brain tumor associated with hemorrhage presents with an abrupt onset of symptoms similar to a CVA (Cahill, Lo-Biondo-Wood, Bergstrom, & Armstrong, 2012). Generalized or focal seizure activity from disruption of cortical circuits is accompanied in 30% of brain tumor cases (Longo et al., 2012). Nonfocal neurological deficit such as a headache occurs as a result of brain irritation or from increased intracranial pressure (ICP). Papilledema is also due to elevated ICP and is present in 10% of patients with brain tumors (Huttner, 2013). Nausea and vomiting, malaise, anorexia, weight loss, and fever are also common systemic symptoms suggestive of metastatic (rather than primary) brain tumor (Mogensen, 2008). The diagnosis of a brain tumor in eliminated for this patient because the CT of head without contrast does not illustrate a space-occupying lesion or mass effect. In cases where a high clinical suspicion of brain tumor exists, a MRI is recommended because of the test’s high sensitivity (92%) and specificity (96%) to detect small tumors and tissue edema (Onwuchekwa & Onwuchekwa, 2010). However, in this case, a tumor is unlikely due to the lack of evidence of a lesion on the chest x-ray and a negative patient and family history of cancer. Most metastatic brain tumors (84%) originate from the primary cancer site in the lung (Cahill et al., 2012).

Finally, the differential diagnosis of Todd’s paralysis is considered. In Todd’s paralysis, hemiparesis is followed by an episode of seizure (usually recurrent partial motor seizures or generalized tonic-clonic seizures). The symptoms can last up to 36 hours before resolving completely (National Institute of Neurological Disorders and Stroke [NINDS], 2013). Other symptoms of Todd’s paralysis are associated with the specific area of the brain the seizure
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occurred. For example, a seizure in the occipital lobe may result in visual disturbances or blindness, ataxia, or vertigo in the post-ictal state. Left hemispheric (frontal, parietal, or temporal lobes) seizure can cause aphasia or difficulty with memory (Trupe et al., 2013). The etiology of the condition can be attributed to neuronal exhaustion from hypoxia or from the release of inhibitory neuronal discharge. Also, recurrent seizures can exacerbate damage to a localized region of the brain (Longo et al., 2012). Todd’s paralysis can be ruled out in this case because the patient does not have a history of seizures. Additionally, the CT of head without contrast does not show any source of possible brain irritability, such as a previous CVA, edema, or other lesions. An electroencephalogram (EEG) and/or a MRI will be obtained if patient’s presentation demonstrates signs of clinical or subclinical seizures.

Plan

Pharmacological/Procedural Treatment of Acute Ischemic CVA

Treatment goals in acute complications. Stroke is characterized by primary failure of focal tissue oxygenation. Therefore, immediate efforts are taken to prevent or reverse brain injury by optimizing cerebral perfusion. The main goal is to oxygenate the zone of penumbra which remains viable for hours after initial injury. The collateral blood supply around the ischemic area allows for some degree of blood flow to the penumbra (Agarwal et al., 2013). Critical care treatment is required to prevent further hypoxemia and hypotension. Medical support is necessary to prevent further complications (e.g. acute thrombus, pneumonia, seizures). Stroke team consisting of neurologists, neurosurgeons, ED nurses and physicians, will be notified prior to patient arrival in order to provide comprehensive care and to prevent delay in treatment (Class I, Level of Evidence B) (AHA/ASA, 2013).
**Priority one: Airway, breathing, circulation.** Hypoxemia and hypotension can exacerbate ischemic brain injury (Agarwal et al., 2013). The AHA/ASA (2013) recommends continuous cardiac monitoring for assessment of heart rate/rhythm, blood pressure (BP), respiratory rate, and oxygen saturation (*Class I; Level of Evidence B*). The vital signs are currently stable and the patient is able to maintain the airway on two liters of oxygen via nasal cannula. Thus, no concern for acute respiratory failure is identified. Common causes of hypoxia in CVA include aspiration, pneumonia, hypoventilation, and decreased consciousness due to brain stem dysfunction (Agarwal et al., 2013). An oxygen saturation of >94% is recommended for an acute ischemic CVA and an order for supplemental oxygen use will be prescribed (*Class I; Level of Evidence C*).

A continued state of normothermic is also a treatment goal. Hyperthermia (temperature > 38.6°C) in the setting of acute ischemic CVA is related to poor neurological outcomes because elevated temperatures increase metabolic demands and enhance the production and release of neurotransmitters and free radicals (McCance et al., 2010). An as needed order of acetaminophen 650mg per nasogastric tube every 4-6 hours and cooling blankets/apparatus will be ordered for a temperature >38.6°C (*Class I; Level of Evidence C*). If the body temperature exceeds 39°C, further evaluation with blood, urine, and/or sputum cultures will be necessary to determine the source of hyperthermia (Chesnutt & Zamora, 2008). An Advanced Practice Nurse (APN) with a Certificate to Prescribe (CTP) or with a Certificate toPrescribe Externship (CTP-E) is able to prescribe acetaminophen, within the APN standards and scope of practice (Ohio Board of Nursing [OBN], 2013).

Maintaining an adequate BP is essential to prevent complications. Although an elevated BP is common and essential for cerebral perfusion to the ischemic tissue during an acute
ischemic CVA, extreme hypertension (>220/120 mmHg) is unfavorable because it leads to cardiac and renal complications, encephalopathy, and exacerbation of cerebral edema (AHA/ASA, 2013). Consistent elevation of BP can also result in a hemorrhagic transformation of an acute ischemic CVA (McGrath et al., 2012). Conversely, hypotension is detrimental because it decreases perfusion to the ischemic brain and further causes ischemic injury to the penumbra. According to the AHA/ASA (2013) guidelines, BP lowering during the initial 24 hours of acute ischemic CVA is not recommended. However, BP reduction is appropriate in cases where BP is >220/120 mmHg or if a specific concomitant medical condition warrants BP lowering. If BP reduction is required, BP should be reduced no more than 15% during the first 24 hours after onset of a CVA (Class I; Level of Evidence C). Consistent with the AHA/ASA (2013) guidelines, the current antihypertensive home regimen therapy will be paused and restarted after 24 hours if the patient is medically and neurologically stable (Class IIa; Level of Evidence B). Target BP goal for this patient will be discussed in the next section.

**Priority two: Fibrinolysis for recanalization.** The use of fibrinolytic therapy with recombinant tissue-type plasminogen activator (rtPA) alteplase within 4.5 hours of symptom onset has demonstrated beneficial effects of reducing total ischemic time and restoring blood flow to the penumbra (Class I; Level of Evidence B). However, benefit of therapy is time dependent and earlier administration of rtPA within 3 hours is preferred for maximum benefits and unfavorable outcomes (Class I; Level of Evidence A). The established goal of door-to-needle time (administration of rtPA) is less than 60 minutes (Class I; Level of Evidence A) (AHA/ASA, 2013).

A meta-analysis of four clinical trials with 1847 patients illustrated the most neurologic improvement (i.e. improvement in the NIHSS score) at three months occurred with treatment of
IV rtPA within 1.5 hours of CVA symptom onset when compared with placebo (RR 2.81 [95% CI 1.75-4.50]). Treatment with IV rtPA initiated within 1.5 hours to three hours of symptom onset was also favorable at three month follow-up in comparison with the control group (RR 1.55 [95% CI 1.12-2.15]). Although neurologic improvement of CVA patients was noted at three months in the treatment group when rtPA was administered three to 4.5 hours after symptom onset, the favorable outcomes were less than the treatment groups which administered IV rtPA within three hours (RR 1.40 [95% CI 1.05-1.85]) (Lees et al., 2010). Desirable outcomes are achieved with earlier fibrinolysis because the longer the duration of transient neurological deficits, the chance of developing neuroanatomically focal abnormalities increases significantly. These abnormalities are inadvertently associated with an increased risk of hemorrhage (Kerr et al., 2012).

The patient is appropriate to receive fibrinolytic therapy as the patient meets all the inclusion criteria of 1) diagnosis of ischemic CVA with measurable neurological deficit that is not improving, 2) onset of symptoms less than 4.5 hours, and 3) older than 18 years of age. There are no presenting contraindications for treatment with rtPA for this patient. Exclusion criteria are associated with conditions that may increase the risk of bleeding and include: head trauma or previous CVA within three months, history of previous intracranial hemorrhage, active internal bleeding, arterial puncture at non-compressible site in past seven days, elevated BP (SBP > 185 mmHg/DBP > 110 mmHg), platelet count < 100,000/mm³, INR > 1.7, PT > 15 seconds, or aPTT > 35 seconds, blood glucose < 50 mg/dL, and multilobar infarction on CT of head without contrast. Relative contraindications include minor or improving CVA symptoms, pregnancy, seizure at onset with post-ictal state, major surgery or trauma within previous 14 days, recent gastrointestinal bleeding within past 21 days, and an acute MI within previous three months. If
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contraindications for IV fibrinolysis exist, intra-arterial fibrinolysis or mechanical thrombectomy is reasonable (Class IIa; Level of Evidence C) (AHA/ASA, 2013).

The administration dose of rtPA is 0.9 mg/kg (maximum dose 90 mg) over 60 minutes, with 10% of the dose given over one minute (as a bolus) (AHA/ASA, 2013). The patient will be admitted to an intensive care unit (ICU) for frequent monitoring. Target BP goal during and after rtPA administration is SBP <180 mmHg and DBP < 105 mmHg to minimize the risk of intracranial hemorrhage. Anti-hypertensives with a non-vasodilating effect, such as labetalol, nicardipine, are preferred (AHA/ASA, 2013; Longo et al., 2013) (Class IIa; Level of Evidence C). Injury to the brain impairs autoregulatory mechanisms such as a constrictive response to pathological vasodilation (Kerr et al., 2012). Labetalol can be given as an IV bolus (10-20mg over one to two minutes) or as a continuous infusion (2-8 mg/min) to maintain target BP levels. Nicardipine is usually administered as a continuous infusion, with initial dosing of 5 mg/hour and titrated up by 2.5 mg/hour every five to 15 minutes (maximum 15 mg/hour) to achieve the desired effect. If BP remains elevated and target BP goal is not achieved, nitroprusside continuous infusion with titration therapy at dose range of 3-10 mcg/kg/minute may be considered (AHA/ASA, 2013). An APN with a CTP or with a CTP-E is able to prescribe anti-hypertensives, within the APN standards and scope of practice (OBN, 2013). However, an APN with a CTP or with a CTP-E is unable to prescribe thrombolytic agents such as rtPA (OBN, 2013).

Meticulous management of BP and neurologic symptoms during and after TPA is crucial for reducing the risk of hemorrhage and detecting early complications. A comprehensive assessment is conducted every 15 minutes for two hours from the start of rtPA therapy, then every 30 minutes for six hours, and then every one hour for 16 hours (AHA/ASA, 2013).
Immediate cessation of rtPA therapy is merited if the patient develops a severe headache, nausea/vomiting, acute hypertension, or worsening neurological examination. These symptoms may be due to the presence of intracranial hemorrhage and an emergent CT of head without contrast is warranted to assess the cranium vault (Kerr et al., 2012).

**Priority three: Nothing-by-mouth (NPO) and IV hydration.** Stoke patients are at an increased risk of airway compromise because of impairment of the oropharyngeal mobility, decreased mental status, and loss of protective reflexes such as cough and gag (Burns, Green, Metivier, & Defusco, 2012). Patients with an infarct in the brain stem or major hemisphere, or with multiple infarctions, are at highest risk of pneumonia (Ji et al., 2013). Initial bedside screening evaluation by a nurse or speech pathologist is necessary to prevent aspiration pneumonia and address swallowing impairment (AHA/ASA, 2013). Adequate nutrition is required for recovery and prevention of medical complications. In addition, inadequate volume leads to increased viscosity in the acute period of ischemic CVA. An amplified hematocrit is related to decreased perfusion and expansion of the size of the infarct. Proper hydration allows reduction of viscosity of blood, enhance blood flow through microvascular and collateral circulation, and boost oxygen-carrying capacity (Crary et al., 2013).

Since the patient is drowsy and has a significant facial droop, the patient will be placed on a strict nothing-by-mouth diet until swallowing evaluation is completed. Intravenous hydration with isotonic 0.9% saline will be prescribed at a rate of 100 mL/hour. Hypotonic or dextrose-containing fluids are avoided in ischemic CVA because of the potential for exacerbation of brain edema due to the intercellular fluid distribution of the fluid (Burns et al., 2012). An artificial nasogastric feeding tube will be inserted for medication and nutrition administration if the patient fails the bedside swallowing assessment (Class I, Level of Evidence
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B) (AHA/ASA, 2013). An APN with a CTP or with a CTP-E is able to prescribe isotonic IV fluids, within the APN standards and scope of practice (OBN, 2013).

Priority four: Blood glucose management. The presence of high blood glucose increases the risk of stroke by threefold (Longo et al., 2012). Hyperglycemia has been associated with intracranial hemorrhage and worse clinical outcomes among CVA patients treated with IV rtPA. Blood glucose elevations during acute CVA are due to impaired glucose metabolism due to stress reaction (Calleja et al., 2011). Insulin resistance results in increased levels of plasminogen and fibrinogen, thus enhancing coagulation process and impairing fibrinolysis (promoting the development of thrombosis). Additionally, systemic complications are produced by the negative effects of diabetes on endothelial function and platelet aggregation (Bruno et al., 2008).

Although no proven effect of tight blood glucose control for CVA patients has been established by clinical trials, the AHA/ASA (2013) guidelines support the target goal of American Diabetic Association (ADA) for blood glucose maintenance between 140 to 180 mg/dL in acute CVA (Class IIa; Level of Evidence C). Blood glucose levels will be checked every six hours while the patient is NPO. Hyperglycemia treatment in the acute setting will include: Halting of oral home medications and adding correctional sliding scale insulin protocol with scheduled once daily basal insulin (for 62 kg patient, 15 units of lantus subcutaneous at bedtime) (Bruno et al., 2008). An APN with a CTP or with a CTP-E is able to prescribe insulin and hypoglycemic agents, within the APN standards and scope of practice (OBN, 2013).

Priority five: Thrombosis prophylaxis. Deep vein thrombosis (DVT) and pulmonary embolism (PE) are more likely to occur in CVA patients who have limited immobility due to lower extremity or pelvis paralysis. Occurrence of a DVT or PE slows recovery and rehabilitation after a CVA (Kuwashiro et al., 2012). The patient will be monitored for signs and
symptoms of DVT such as pain, redness, and swelling in the upper or lower extremities. Anticoagulants (i.e. low-molecular-weight-heparin or unfractionated heparin) are not appropriate at this time since the patient will be receiving IV rtPA. An anticoagulant will be prescribed after a CT of head without contrast has been obtained 24 hours after administration of rtPA to rule out intracranial hemorrhage. Initiation of anticoagulation therapy within 24 hours of treatment with IV rtPA is not recommended (Class III, Level of Evidence B. In the meantime, the use of external compression device will be utilized as a DVT/PE prophylaxis (Class IIa, Level of Evidence B) (AHA/ASA, 2013).

**Priority six: Additional testing.** After stabilization of the patient’s condition, additional testing is required to assess other possible risk factors of the etiology of the acute ischemic CVA and to establish secondary prevention measures. From reviewing the patient’s past medical history of hyperlipidemia, a major risk factor for stroke, a lipid panel will be ordered to assess if an adjustment of the lipid-lowering drug therapy is necessary (AHA/ASA, 2011). The patient will remain NPO for at least eight hours prior to the lipid panel testing. A transthoracic echocardiography (sensitivity 82-87%, specificity 90-100%) will be obtained to evaluate for other cardioembolic causes and will assess cardiac and valvular function, ejection fraction, and the presence of thrombi in the left atrium (Karabay et al., 2013). Carotid stenosis may also be the cause of an ischemic injury to the brain due to decreased blood flow. A carotid ultrasonography is the non-invasive test of choice to assess bilateral internal carotid arteries (sensitivity 90%, specificity 94%) (Johri et al., 2013). To assess the adequacy of glycemic control, a hemoglobin A1C level will also be obtained.

**Non-Pharmacological Therapy**
**Priority seven: Acute rehabilitation therapy.** An appropriate component of comprehensive stroke care includes physical, occupational, and speech therapy. The goal of therapy is to educate the patient and the family about the neurological deficits and to prevent further complications associated with immobility and/or swallowing or cognition such as pneumonia, DVT and PE, pressure ulcers of skin, falls, and muscle contractures (Burns et al., 2012). Rehabilitation is considered a tertiary approach in CVA prevention. The program’s focus is to optimize recovery by providing an individualized, progressive regimen of exercises. Physical therapy following a CVA has demonstrated recruiting of unused neural pathways resulting in an improvement of hemiparesis (Hakkennes et al., 2012). Stroke rehabilitation can play a crucial role in regaining a considerable portion of the functions that have been impaired as a consequence of the CVA. Adequate rehabilitation is associated with increased autonomy, improved quality of life, and a lower depression rate (AHA/ASA, 2013).

**Follow-Up and Monitoring Parameters**

Monitoring a patient for complications after an initial acute brain insult is of paramount importance. Reoccurrence of a CVA is possible and the patient is closely monitored for neurological deterioration by performing detailed neurological assessment (with NIHSS score) every one to two hours after completion of rtPA infusion. Hemorrhagic conversion of an ischemic CVA is likely especially for those patients with a larger CVA, are of older age, or with a cardioembolic pathogenesis (Burns et al., 2012). Incidence of symptomatic hemorrhage is also higher in patients who received fibrinolysis therapy (i.e. rtPA) that who did not, 5.9% versus 1.7% ($p < 0.05$) (Lees et al., 2010). Most fatal hemorrhages occur within the first 24 hours after IV rtPA administration (Burns et al., 2012). The presence of worsening neurological symptoms such as decreased mental status, headache, increased BP and heart rate, and vomiting warrant an
immediate CT of head without contrast imaging to assess for hemorrhage (Kranz & Provenzale, 2013). According to the fibrinolysis administration protocol, a 24-hour follow-up CT of head without contrast is also ordered for patients who received IV rtPA (AHA/ASA, 2013). If hemorrhage is discovered on the CT imaging, a neurosurgical consultation is essential to evaluate if a surgical intervention to evacuate the hematoma is necessary. In addition, if the follow-up CT imaging reveals a CVA greater than two-thirds of the MCA area, a neurosurgical consultation will also be appropriate because there is a delayed risk of brain herniation from brain stem compression (Rahme et al., 2012). A decompressive surgery may be required for intracranial pressure release and for herniation prevention (Class I, Level of Evidence B). A hemicraniectomy for MCA infarction can decrease mortality from 80% to 20% (McKenna et al., 2013).

The patient will be monitoring for signs and symptoms of active systemic bleeding after administration of the fibrinolysis therapy. Although intracranial hemorrhage is the most common after rtPA administration, hemorrhage can occur at any site in the body. The patient will be maintained on a strict bedrest during and at least four hours post-rtPA administration and bleeding precautions will be instituted (Whiteley et al., 2012). Vital signs and neurological assessment will be monitored every one hour (or sooner if patient is unstable) during the acute phase of the CVA (AHA/ASA, 2013). Coagulation panel consisting of aPTT, PT/INR, and a CBC will be monitored for bleeding complications (Lexi-Comp, 2013). Signs of possible hemorrhage to monitor include tachycardia, hypotension, decreased level of consciousness, and a reduction of hemoglobin/hematocrit level. Gastrointestinal signs and symptoms of bleeding include nausea, vomiting, and a positive hemoccult result (Longo et al., 2012). Angioedema with
rtPA administration may occur in 1-5% of patients. Empiric monitoring of lips, tongue, and oropharynx for edema is recommended (Whiteley et al., 2012).

Acute ischemic CVA also results in development of cytotoxic edema from the infarcted or necrotic tissue. Cerebral edema can increase intracranial pressure and lead to neurological deficits (Burns et al., 2012). Medical management of cerebral edema includes restriction of free water, avoidance of hypo-osmolar solutions, and minimization of hypoxemia and hypercarbia. Head of the bed is elevated at least 30° to assist with venous drainage and decreased edema. Severe elevations in intracranial pressure are managed by hypertonic saline or mannitol, hyperventilation, intraventricular drainage of cerebrospinal fluid, and decompressive surgery. Hyperventilation (PCO₂ 30-35 mmHg) induces cerebral vasoconstriction, and thereby decreasing cerebral blood flow and intracranial pressure (Rahme et al., 2012). Hypertonic saline and mannitol reduce intracellular fluid via osmotic diuresis. Mannitol 0.25 to 0.5 g/kg is administered intravenously over 20 minutes and can be given every six hours; Three percent hypertonic saline is administered intravenously at a rate of 30 ml/hour until the target sodium level is achieved (serum sodium goal of 150-155) (Katzung, Masters, & Trevor, 2012). High intracranial pressures can also cause disruption of neuronal circuit abnormalities which lead to the occurrence of seizures (Longo et al., 2012). For immediate cessation of seizures, lorazepam 1-2 mg IV every 10 minutes (for a maximum of 8 mg) may be given (Lexi-Comp, 2013). Although AHA/ASA (2013) guidelines do not recommend a specific antiepileptic drug for use in acute ischemic CVA, levetiracetam is most commonly used as an initial drug of choice for patients with a diagnosis of a primary neurological disorder (Burns et al., 2012; Katzung, Masters, & Trevor, 2012). Levetiracetam is administered intravenously in acute settings with a dose ranging from 500-1500 mg every 12 hours (Lexi-Comp, 2013). An APN with a CTP or with a CTP-E is able to prescribe
mannitol, hypertonic fluids, lorazepam, and levetiracetam within the APN standards and scope of practice (OBN, 2013).

The administration of antiplatelet and anticoagulation adjunctive therapy within 24 hours of IV fibrinolysis is contraindicated (Class III, Level of Evidence C). Anticoagulation with enoxaparin 40 mg subcutaneous (SQ) daily for DVT and PE prophylaxis and antithrombotic regimen with aspirin 81 mg orally (PO) daily (initial dose of 325 mg) for secondary prevention of recurrent CVA will be initiated if the 24-hour follow-up CT imaging validates the lack of a hemorrhage or a CVA expansion (Class I; Level of Evidence A). Aspirin is preferred than clopidogrel because maximum platelet aggregation effect is delayed (for five days) if treated with clopidogrel (Katzung, Masters, & Trevor, 2012). Low-molecular-weight-heparin (i.e. enoxaparin 40 mg SQ daily) was supported to reduce the risk of venous thromboembolism more than unfractionated heparin in ischemic CVA patients (18% versus 10%, RR 0.57 [95% CI 0.44-0.76], \( p = 0.001 \)) (Sherman et al., 2007). An APN with a CTP or with a CTP-E is able to prescribe enoxaparin and aspirin, within the APN standards and scope of practice (OBN, 2013).

Acute ischemic patients are at risk for MI, congestive heart failure, and significant arrhythmias. In addition, the risk of development of significant cerebral edema increases 48-72 hours after onset of CVA symptoms. Cerebral edema causes neurological deficits and increases the risk of acute respiratory (Burns et al., 2012). Therefore, continuous cardiovascular monitoring is crucial (Class I: Level of Evidence B). The following parameters will be ordered (as discussed previously) after reviewing the patient’s current medical condition and past medical history. These parameters will take effect if the current prescribed treatment plan has failed to achieve the target levels (AHA/ASA, 2013):
Call Clinician If:

<table>
<thead>
<tr>
<th>Condition</th>
<th>Threshold</th>
</tr>
</thead>
<tbody>
<tr>
<td>Systolic Blood Pressure</td>
<td>&gt; 180 mmHg or &lt; 120 mmHg</td>
</tr>
<tr>
<td>Diastolic Blood Pressure</td>
<td>&gt; 105 mmHg or &lt; 80 mmHg</td>
</tr>
<tr>
<td>Heart Rate</td>
<td>&gt; 110 beats per minute or &lt; 50 beats per minute</td>
</tr>
<tr>
<td>Temperature</td>
<td>&gt; 101.5°F or &lt; 95°F</td>
</tr>
<tr>
<td>Respiratory Rate</td>
<td>&gt; 30 breaths per minute or &lt; 8 breaths per minute</td>
</tr>
<tr>
<td>Oxygen Saturation</td>
<td>&lt; 90%</td>
</tr>
<tr>
<td>Urine Output</td>
<td>&gt; 300 mL/hr or &lt; 30 mL/hr for two hours</td>
</tr>
<tr>
<td>Blood Glucose</td>
<td>&gt; 150 mg/dL or &lt; 70 mg/dL</td>
</tr>
</tbody>
</table>

Although the current atrial fibrillation ventricular heart rate is fairly stable at 100-102 beats per minute as illustrated on the EKG and cardiac monitor, a plan for heart rate control is warranted to prevent further complications such as tachycardia-induced cardiomyopathy. Heart rate control is preferred than rhythm control in atrial fibrillation because fewer toxic medications and medical procedures are indicated for the same end-point results (Heist, Mansour, & Ruskin, 2011). The Atrial Fibrillation Follow-Up Investigation of Rhythm Management (AFFIRM) (2002) demonstrated incidence of increased mortality in the rhythm control group compared with the rate control group (24% versus 21%, \( p=0.08, \) RR 1.15 [95% CI 0.99-1.34]). In addition, the rate of CVA occurrence was not reduced in the rhythm control group in comparison with rate control (Wyse et al., 2002).

According to the American College of Cardiology Foundation/American Heart Association guidelines (2011), the target heart rate control for atrial fibrillation patients is less than 110 beats per minute (Wann et al., 2011). In the acute care setting, intravenous (IV) medications are preferred for rate control with conversion to oral agents at discharge for long-term therapy. Before discharge, the patient will be bridged from heparin to warfarin therapy, and heparin will be discontinued when the target INR of 2-3 for atrial fibrillation has been achieved (Mazighi, Meseguer, Labreuche, & Amarenco, 2012). Medications such as beta blockers and
channel blockers are effective to prolong the refractoriness of the AV node in order to decrease the ventricular rate in atrial fibrillation (Heist, Mansour, & Ruskin, 2011). In a post-analysis of the AFFIRM study, beta blockers were more efficacious than calcium channel blockers in achieving the target rate (70% versus 54%, \( p < 0.05 \)) (Olshansky et al., 2004). Since lowering BP too drastically is detrimental in an acute ischemic CVA and because the current heart rate is stable, no treatment for rate control is indicated at this time. However, in the follow-up care of the patient, heart rate and rhythm and BP will be monitored and appropriate treatment with either metoprolol 5-10 mg IV or diltiazem bolus of 0.25 mg/kg IV followed with continuous IV diltiazem infusion rate at 5-15 mg/hour (titrated to heart rate <110 beats per minute) will be prescribed (Heist, Mansour, & Ruskin, 2011). An APN with a CTP or with a CTP-E is able to prescribe anti-hypertensives and warfarin, within the APN standards and scope of practice (OBN, 2013).

Follow-up monitoring of the ordered tests will be evaluated and treatment adjusted as needed. Home lipid-lowering drug (simvastatin) dosage will be amended if lipid panel yields abnormal results (i.e. LDL > 70) (AHA/ASA, 2013). If carotid ultrasound reveals carotid stenosis, carotid endarterectomy (CEA) or stenting may be performed. In the acute setting, the intervention for recanalization may be emergent if the primary cause of the CVA is total or near-total occlusion caused by severe atherosclerosis (AHA/ASA, 2013). Usually, the surgical and endovascular management is reserved for secondary CVA prevention rather than acute CVA treatment. These procedures are generally avoided in an acute setting because there is an increased risk for further ischemic insult to the brain (Burns et al., 2012). In a non-acute period, CEA or stenting may be considered if the ipsilateral artery is 70-99% occluded (Class I, Level of Evidence A). Carotid stenting is an alternative to CEA when medical conditions are present that
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increase the risk of surgery (Class IIb, Level of Evidence B). If the artery is 50-70% occluded, the patient will be medically managed with anticoagulation; the patient will be monitored annually if the artery is <50% occluded (Class I, Level of Evidence B) (AHA/ASA, 2011). The patient is also at high risk for the development of thrombi in the left atrium attributable to the history of atrial fibrillation. If thrombi are detected on the transthoracic echocardiogram, a transoesophageal echocardiogram (TEE) (sensitivity 95%, specificity 100%) will be conducted for cardioversion/stratification for anticoagulation (Karabay et al., 2013).

Depression is highly prevalent in post-CVA acute state, affecting between 25-75% of CVA survivors. However, depressive symptoms may be unrecognized in patients due to the presence of CVA-related deficits. Depression is related to a high level of limitations in cognitive and functional capabilities (Janneke et al., 2013). Other risk factors include low social support and lesion location. Frontal/anterior CVAs are related to a higher occurrence of post-CVA depression. Untreated depression leads to poor rehabilitation efforts, poor prognosis, and increased mortality (Taylor-Pillae, Hepworth, & Coull, 2013). The patient will be monitored for post-CVA depression symptoms of: social withdrawal, lack of energy, irritability/fraustration, feelings of hopelessness, emotional variability, and thoughts of suicide (Janneke et al., 2013).

Discharge/Patient Education and Health Promotion

Discharge Plan

The patient progressed significantly with improved right sided hemiparesis and aphasia after administration of IV rtPA; NIHSS score was stable eight. No hemorrhage was identified in the follow-up CT of head without contrast at 24 hours and anticoagulation and antithrombotic therapy was initiated. Discharge to an inpatient rehabilitation center was recommended by physical and occupational therapy. The goal of the aggressive (three hours a day, five days a
Inpatient rehabilitation therapy is to assist the patient to regain, maintain, or prevent further deterioration of functional ability. Inpatient CVA rehabilitation centers are recommended for post-CVA survivors because the centers provide access to a multidisciplinary team consisting of nurses, physical, occupational, and speech therapists, physicians, psychologists, social workers, and rehabilitation assistants that can assist with the complex care required for CVA patients. The rapid assessment and intervention for a recurrent CVA or related complications is feasible in this environment because of 24-hour supervision (National Institute for Health and Care Excellence [NICE], 2013).

**Patient Education**

When educating CVA survivors, practitioners must consider the potential hearing, vision, language, and memory deficits that may affect understanding and retaining the learned information. Additionally, the level of literacy of the patient is also important during educational interventions. Only 20% of patients are at the fifth to sixth grade reading level (Cameron, 2013). Family and caregivers care for approximately 74% of CVA survivors after discharge and play a vital role in patient’s recovery process (Hakkennes et al., 2012). Thus, it is also important to educate and support family members during all stages of rehabilitation.

Approximately 25% of the 795,000 CVAs that occur each year are recurrent incidents (AHA/ASA, 2013). Patients with atrial fibrillation have an average annual risk of CVA of 5-10% (Longo et al., 2012). Other major modifiable risk factors for a recurrent CVA are hypertension, diabetes, and hyperlipidemia (AHA/ASA, 2011). Secondary prevention measures are a key to prevent long-term complications. A variety of strategies such as patient education and lifestyle modification are adopted for affected CVA survivors and their families. The CVA education must be reiterated at every opportunity by multiple staff members before discharge and at
follow-up outpatient appointments in order to illustrate the importance of adequately managing CVA-related risk factors.

**Modifiable personal risk factor: Hypertension.** Hypertension is an important modifiable risk factor for risk reduction of CVA. Lowering BP to <120/80 mmHg is associated with a 30-40% lower incidence of CVA (Quan et al., 2013). The patient will be instructed to maintain the target BP goal of <120/80 mmHg by taking anti-hypertensives as prescribed. Lifestyle modifications associated with a lower BP consist of salt restriction, weight loss/management, eating a diet low in fat and rich in fruits and vegetables, and regular aerobic exercise (30 minutes at least three times a week or a supervised therapeutic physical therapy regimen for those who cannot tolerate intense exercise) (Class IIa, Level of Evidence C) (AHA/ASA, 2011). Herbal medications, licorice, bayberry, and blue cohosh, may worsen hypertension and should be avoided (Lexi-Comp, 2013).

Monitoring of home BP and heart rate at least daily will be emphasized in order to evaluate trends for BP management. The patient will be instructed to continue to take home medications metoprolol 25 mg PO BID and lisinopril 20 mg PO daily. Education on the side effects of metoprolol such as hypotension, bradycardia, dizziness, fatigue, and decreased libido will be provided. Lisinopril side effects include hypotension, cough, angioedema, acute renal failure, increased risk of bleeding (due to thrombocytopenia), and hyperkalemia. The patient will be monitored for renal impairment, hyperkalemia, and thrombocytopenia with routine lab work (i.e. CBC and CMP) during lisinopril therapy (Katzung, Masters, & Trevor, 2012). The effects of lisinopril may be decreased by nonsteroidal anti-inflammatory agents and these drugs should be avoided. Potassium supplements and potassium-containing salts may worsen hyperkalemia and should also be restricted. Avoidance of herbal medications such as shepherd’s purse, black
cohosh, and hawthorn with metoprolol and lisinopril is essential to prevent severe hypotension (Lexi-Comp, 2013).

**Modifiable personal risk factor: Diabetes.** The prevalence of diabetes is 15-33% in patients with ischemic CVA (AHA/ASA, 2011). Instruction on obtaining a blood glucose level three times a day and at bedtime will assist with monitoring of complications such as hypoglycemia or hyperglycemia. The patient will be instructed on maintaining the desired fasting blood glucose of < 100 mg/dL. Exercise, low-fat and glucose diet, stress reduction, and oral home hypoglycemic drugs (glyburide 10mg PO BID) will be suggested to maintain normal glucose levels (Yong & Kaste, 2008). The patient will be instructed on the side effects of glyburide such as hypoglycemia, dizziness, angioedema, blurred vision, and increased risk of infection (due to agranulocytosis). Concurrent use of ethanol and herbal supplements such as alfalfa, aloe, ginseng, and bilberry with glyburide is necessary in order to avoid enhancement of the hypoglycemic effect (Lexi-Comp, 2013). An annual (or bi-annual) hemoglobin A1C level will aid in the management of therapy. Target hemoglobin A1C in recurrent CVA prevention is <7% (*Class IIa, Level of Evidence B*) (AHA/ASA, 2011). The patient will be instructed to follow-up with primary care provider (PCP) for long-term management of diabetes.

**Modifiable personal risk factor: Hyperlipidemia.** Elevated total cholesterol or low-density lipoprotein cholesterol (LDL) is associated with an increased risk of ischemic CVA (Burns et al., 2012). The National Cholesterol Education Program (NCEP) expert panel recommends reducing LDL to < 70 mg/dL as the primary target in patients with a history of CVA or coronary heart disease (CHD) (*Class IIa; Level of Evidence B*). Other desired goals include: total cholesterol < 200 mg/dL, triglycerides < 150 mg/dL, and high density lipoprotein (HDL) > 40 mg/dL (AHA/ASA, 2011; NCEP, 2002). Statin therapy has demonstrated
effectiveness in reducing LDL to less than 70 mg/dL in CHD, and thereby decreasing the occurrence of ischemic CVAs by 37% (RR 0.63 [95% CI 0.49-0.81], \( p = 0.001 \)) (Amarenco et al., 2007). The patient will be instructed to continue the home medication simvastatin 40 mg PO daily and to follow-up with PCP for monitoring of liver enzymes and lipid studies every four to six months during therapy (NCEP, 2002).

Side effects of simvastatin such as myalgia, arthralgia, hepatic failure, and headache, are vital to incorporate in patient education (Lexi-Comp, 2013). The patient will be educated to avoid grapefruit juice and red yeast rice as these agents can increase drug effect of simvastatin. Alternatively, St. John’s wart should be avoided because it can decrease simvastatin drug levels (Katzung, Masters, & Trevor, 2012). Therapeutic lifestyle modification therapies to maintain LDL in the target range include reduction of saturated fat and cholesterol intake, increasing fiber intake, and promotion of physical activity. Administration of plant stanols/sterols and omega-3 polyunsaturated fatty acids may also be considered (NCEP, 2002).

**Non-modifiable personal risk factor: Atrial fibrillation.** About 75,000 cases of CVA are attributed to atrial fibrillation every year (AHA/ASA, 2011). Risk factors of atrial fibrillation include advancing age, hypertension, diabetes, valve disease, and heart failure (Longo et al., 2012). Warfarin is the treatment of choice for overall risk reduction of 68% of CVA rate in atrial fibrillation (Deitelzweig et al., 2013). The calculated CHA\(_2\)DS\(_2\)-VA score of the patient is six (presence of hypertension, diabetes, recent CVA, between the age of 65-74 years, and female gender), signifying the patient at a high-risk for a thromboembolic event (Deguchi et al., 2013). Bridging therapy with heparin will be initiated prior to discharge and the patient will be instructed to continue to take home medication warfarin daily. The patient will require multiple
follow-up visitations with PCP for frequent INR testing and dose adjustments to achieve the INR target range of 2-3 *(Class I, Level of Evidence A)*.

Education will be provided on the medication’s narrow therapeutic margin and numerous related food and drug interactions. Large amounts of consumption of foods high in vitamin-K (e.g. spinach, kale, green tea, turnip greens) reduces the effectiveness of warfarin, and thereby resulting in sub-therapeutic INR levels (Deitelzweig et al., 2013). On the other hand, vitamin-E and cranberry, acetaminophen, omeprazole, and certain antibiotics (fluroquinolones and macrolides) may increase the effect of warfarin (and risk of bleeding) and should be avoided (Lexi-Comp, 2013). The patient will be instructed to take warfarin at the same time daily, keep a consistent vitamin-K diet, and avoid supplements such as gingko, licorice, St. John’s wort, and feverfew (Deitelzweig et al., 2013). Education on other adverse reactions of warfarin will be provided such as dermatitis, abdominal pain, diarrhea, and osteoporosis (Lexi-Comp, 2013).

Since the patient is at high-risk for recurrent thromboembolic event (CHA₂DS₂VA score six), bridging therapy with low-molecular-weight-heparin will be utilized rather than abruptly temporarily interrupting warfarin therapy prior to future surgical procedures *(Class IIa; Level of Evidence C)* (AHA/ASA, 2011; Mazighi et al., 2012).

**Stroke warning signs.** The patient and family will be educated about the five “sudden” signs of CVA for early detection of a possible recurrent event. One or more of the following signs and symptoms are present in 90-100% of all CVAs and TIAs: sudden weakness, sudden, severe headache, sudden visual loss, sudden speech difficulty, and sudden dizziness (Nazarko, 2013). The FAST (face, arm, speech, time) exam will be introduced to the patient and family to assist with quick recognition of a CVA. The assessment includes facial weakness, arm weakness,
speech difficulty, and “time is brain” (the importance of calling 9-1-1 to save valuable time for treatment interventions) (AHA/ASA, 2013).

Emergency activation. Family members and patients are educated about activating the emergency medical services (EMS) in a CVA by calling 9-1-1 instead of self-transporting a patient to the hospital. Benefits of an EMS transfer can be life-saving. Not only will patients get immediate triage and stabilization in the field, but pre-hospital delays are avoided and brain imaging studies (i.e. CT or MRI) can be obtained sooner if patients are transported by an ambulance (AHA/ASA, 2011). When possible, patients will also be selectively transferred to a CVA-certified hospital with an EMS transport. Hospitals can also be notified prior to patient arrival so that the ED physicians are prepared to assess the patient soon after admission. Additionally, ED-care protocol for rtPA can be activated sooner, thus minimizing the door-to-needle time (Fox-Wilson & Cruickshank, 2012).

General Health Promotion

The patient will be instructed on overall medication management and the importance of adherence to control modifiable and non-modifiable CVA risk factors. Other possible post-CVA complications will be discussed such as bowel or bladder incontinence, skin ulceration, and contractures (Hakkennes et al., 2012). Importance of maintaining a diet high in fiber, keeping the skin dry and clean, and performing active or passive range of motion will be stated. The patient and family will be offered to participate in CVA support groups and community-based counseling programs. Complementary therapy that is suitable for CVA survivors, such as Tai Chi, will be encouraged to maintain a regular exercise program or to implement relaxation strategies (AHA/ASA, 2011). Fall-prevention safety methods, such as adequate lighting and
removal of objects in walkway, will be provided to minimize the risk of falls due to possible CVA residual deficits (Dworzynski et al., 2013).

The patient will be educated to maintain an up-to-date vaccine status. Since the patient has not received a flu vaccine this season, a flu vaccine will be administered before discharge. An annual physical examination by a PCP is crucial for follow-up monitoring of co-morbidities: atrial fibrillation, hypertension, diabetes, and hyperlipidemia. Eating a healthy diet with low in sodium, fat, glucose, and cholesterol will be reiterated. Caregivers will be offered to participate in patient care planning and will be provided with guidance, support, and encouragement. Special consideration of family members is of essence in comprehensive CVA care to prevent caregiver-exhaustion.
References


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