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Time Lost is Brain Lost: Impact of Ischemic Stroke

Paula Severns
Otterbein University, paula.severns@otterbein.edu

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Stroke is the leading cause of functional impairment in the United States. Annually, 715,000 strokes occur with a mean lifetime cost estimated at $141,000 per patient (Moucha et al., 2014). Every 40 seconds someone suffers a stroke, while every 2.5 minutes a stroke death occurs (Niem, McIntire, & Tfelt-Hansen, 2015). Stroke is the broad term often used to describe a neurological emergency, of either ischemic or hemorrhagic etiology, affecting blood flow to the brain. Both are medical emergencies requiring timely medical intervention (Elber, Lemon, & Cotello, 2013). Treatment modalities are comprehensively different; therefore, being able to differentiate early signs and symptoms is imperative for healthcare professionals.

Neurovascular imaging is required to differentiate between an ischemic or hemorrhagic stroke. Use of a non-contrast computed tomography (CT) is fast and readily available to rule out hemorrhagic stroke from a major ischemic stroke. Magnetic resonance imaging (MRI) has greater spatial resolution to diagnose minor ischemic strokes.

The topic of ischemic stroke has been chosen in this research to demonstrate the importance and exact location of ischemia. Complete blood deprivation greater than three minutes produce neuron, glial, and vasculature irreversible brain damage. A full symptomatic infarction may take minutes, hours, or dozens of days.

Ischemic stroke degrees: brain metabolic needs of oxygen and glucose. Cerebral ischemia develops from vascular blockage lesions commonly caused by atherothrombotic (from lipid and aggregation) and atheroembolic (from thrombus and cholesterol crystal). According to Davis, Myers, and Dietch (2015), excessive circulating platelets cause endothelial dysfunction by way of circulating within extracellular matrix, altered exposure to free radicals, excess catecholamines, and inflammation increasing endothelial permeability. Combinations of innate and adaptive immune systems, vascular smooth muscle dysfunction, and remodeling of extracellular matrix create atherosclerotic plaque. Plaque rupture leads to a cascade of events:

- Activated platelets adhere to the endothelial cell injury and inhibit excessive blood loss creating a plug of hemorrhage and thrombosis.
- Following adherence to injury site platelets become activated with the assistance of local factor and release agonists thromboxane A2 and adenosine diphosphate (ADP) from platelet membranes causing aggregation.
- G protein contributes by activating glycoprotein Ib/IIa enhancing the thrombotic response.
- Activated platelets promote glycoprotein Ib/IIa receptors recruiting nearby platelets functioning like nets to trap additional blood cells and debris.
- Activated platelets secrete thromboplastin: fibrin, functioning like nets to trap additional blood cells and debris.
- Thrombus growth reduces the lumen diameter restricting blood flow causing ischemia (Figure 2). Ischemic neurons lead cells to ATP loss and glycolysis from sources causing somatic and calcium ion gate ports.
- Reactive oxygen species and inflammation lead to cytotoxicity-mediated cytoxins.
- Microglia produce inflammatory cytokines, including interleukin-1β (IL-1β) and TNF-α (Tumor necrotic immunomodulatory molecule linked to ischemia and neuronal death), causing to cell death and axon activity apoptosis.

Neuronal functioning may alter with a 10-second reduction in blood flow. However, patients can remain asymptomatic until there is a vascular blockage in brain flow created by a complete obstruction. When the brain is not adequately perfused with blood and deprived of oxygen and glucose as well as failure to remove lactate and carbon dioxide, nerve cells begin to die. Localized results compromising capillaries enlivening the ischemic area and reducing neuronal activity. Blood flow is still insufficient to keep tissue alive as well as by symptom recovery as oxygenated blood flow is increased. However, if swelling is severe, symptoms of increased intracranial pressure may occur. Various symptoms include neurological (altering event to reduce morbidity and mortality), and physical (hypertension, arrhythmia, and neurologic symptoms) outcome in patients with acute ischemic stroke. Complete blood deprivation greater than three minutes produce neuron, glial, and vasculature irreversible brain damage. A full symptomatic infarction may take minutes, hours, or dozens of days.

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Paula Severn, RN
Otterbein University, Westerville, Ohio

Introduction

Stroke

Signs & Symptoms

Case Study

Implications for Nursing

References Cited

Stroke affects men and women equally in the United States, predominantly ischemic stroke. Osteoporosis and brain inflammation contribute to the pathophysiology of cerebral injury leading to cellular apoptosis and death (Pasquini et al., 2015). Immune responses within the CNS and systemic inflammatory events play important roles in the progression, initiation, and exacerbation of the disease. Decreasing modifiable risk factors, such as physical activity, smoking, and stress, keep with rapid relief of an occlusion to reduce early death from stroke. Understanding the ischemic stroke pathophysiology (WAD) and improvements in patient education will decrease the morbidity and mortality associated with ischemic stroke. For every minute of treatment delay, 1 million neurons, 14 billion synapses and 12 km of myelinated fibers are destroyed (Moucha et al., 2015, p. 891).

The ISMCA recommends that the door to treatment times continues. According to Adlon (2014), the door to treatment times between hospitals and EMS providers proved beneficial by reducing time to treatment. Current benchmarks have been established to monitor the time the patient arrives at the hospital and the time the patient arrives at the hospital. As an advance practice nurse (APN), a thorough understanding of the sequelae of modifiable risk factors such as long-term cardiovascular disease, smoking, hypertension, atrial fibrillation, diabetes, smoking, dyslipidemia, carotid stenosis, aortic valve disease, hyperlipidemia, and obesity, can be overestimated. Hypertension amplified the degree of severity of ischemic injury contributing to profound neurological recovery and should be closely monitored by routine HSAC testing. Hyperglycemia reduces arterial elasticity, erodes endothelial cell lining and reduces roughness of vessel walls. Making IT the leading risk factor for stroke and should be measured routinely (Moucha et al., 2014). Restoring modifiable risk factors such as HTN, hyperlipidemia, smoking, physical inactivity and obesity, should begin with education.

Education offering a health and mobile nurse took part in a care of recognition of early signs and symptoms of this life-altering event to reduce mortality and morbidity associated with a stroke avoid aversive instructions to call 911. Medical Emergency Services (EMS) (Moucha et al., 2014). Patients arriving within three hours of symptoms achieve better outcomes than those seeking delayed care.

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HANDOUT

Use of Standard Treatment Order Set

According to Elder et al. “Current guidelines for ischemic stroke advocate the use of intravenous (tPA), the early use of thromboembolism (VTE) prophylaxis, anticoagulation in patient with atrial fibrillation (afib), and lipid lowering therapy.” (2015, para. 1) The Joint Commission (JC) requires eight mandatory standards and two optional standards for hospitals to receive Primary Stroke Center (PSC) status:

- Venous thromboembolism (VTE) prophylaxis
- Anticoagulation therapy for afib or flutter
- Thrombolytic therapy
- Antithrombotic therapy by end of hospital day two
- Discharged on statin therapy
- Discharged on antithrombotic therapy
- Receive stroke education
- Assessment for rehabilitation
- Screening for dysphagia
- Smoking cessation services

Order sets ensure compliance with quality indicators, decrease length of stay and decrease overall costs. Order sets created by multidisciplinary stroke experts should follow American Heart Association (AHA)/American Stroke Association (ASA) guidelines. Incorporating order sets in the electronic medical records (EMR) whenever possible will prevent modifications or exclusions. Elder et al. (2015) performed a retrospective study reporting that adherence to national guidelines increased with standard order set use. Current AHA stroke guidelines recommend Emergency Medical Service (EMS) transport suspected stroke patients to the closest PSC unless contraindicated (Jansch et al., 2013).