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The Pathophysiology Of Acute Ischemic Stroke

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Introduction
There are two types of stroke: ischemic and hemorrhagic stroke. Strokes are a type of cerebrovascular accident (CVA) that results from the interruption or obstruction of blood flow to the tissues of the brain. Impairment of the ability of vital brain areas to receive blood as a result of a stroke can lead to neuronal death.

The most common cause of strokes is atherothrombosis, where atheromatous plaques or occlusion of smaller smaller arteries can result in strokes. Other causes include Antithrombotic drugs (e.g., aspirin), Anticoagulants, Thrombolytic therapy, and Antiplatelet therapy.

Risk factors for ischemic stroke include hypertension, hypercholesterolemia, smoking, diabetes, age, race, and a family history of stroke.

Pathophysiological Process

Signs and Symptoms

- **Sudden** onset of numbness or weakness in the face, arm, or leg, especially on one side of the body.
- **Sudden** confusion or trouble speaking or understanding speech.
- **Sudden** trouble seeing in one or both eyes.
- **Sudden** trouble walking, dizziness, loss of balance, or lack of coordination.
- **Sudden** severe headache with no known cause.

Significance Of Pathophysiology

- **Inflammation and Inflammatory markers** are very big part of AIS pathophysiology especially in reference to underlying pathophysiology.

- **Restoration of blood flow** to brain tissue can prevent ischemic damage.

Underlying Pathophysiology

As a neurovascular problem, stroke is considered one of the most devastating diseases that human beings have faced. The pathophysiology of stroke is complex. The pathophysiology process comprises of different collateral path processes, mechanical, oxygenation damages and instabilities. The end result of ischemic stroke is the severe damage of vascular function leading to neuronal death and disability (Yao, 2016).

In the area of the tissue with reduced blood supply or ischemia, extracellular pH (pH) continues in spite of neutral oxygenation, allowing the total ATP level to reduce and induce lactate acidosis, leading to loss of structural homeostasis, and neuronal injury (Yao, 2016).

At the cellular level, the sudden interruption of blood flow to the brain cells is due to lack of blood (thrombus) or a plaque (fatty deposit) that obstructs the brain tissue, from the necessary nutrients and oxygen which leads to intracellular damage.

Hypoxia is caused by ischemic changes. Hypoxia induces inflammation and inflammatory responses. Hypoxia and inflammatory responses are very big part of AIS pathophysiology respectively in reference to reperfusion.

Cocooning collateral formation cascade or clotting. Cocooning of central collateral begins with arterial occlusion and wash with cell death.

Pathological Process

Recognizing the signs and symptoms of acute ischemic strokes is very important because the treatment window to receive tPA is 0–4.5 hours from the onset of symptoms (Anderson, 2016).

Figure 1. The above information is from the American Heart Association. Blood vessel is shown in blue. Eventually, this multitudinous cascade will result in neuronal cell death. To fully grasp the pathophysiological processes, understanding the significance of pathophysiological processes is essential.

Figure 2. Algorithm for the management of patients with acute ischemic stroke to NIH identifies intravenous tissue plasminogen activator (rt-PA) therapy. The above information is retrieved from www.clinicaltrials.gov/ct2/results/NC_01135168

Figure 3. Figure 3 from Reference 1. Name of the reference in parentheses.

Risk factors for ischemic stroke include hypertension, hypercholesterolemia, smoking, diabetes, age, race, and a family history of stroke.

Figure 4. Sudden onset of numbness or weakness in the face, arm, or leg, especially on one side of the body.

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