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THE RISK OF CEREBROVASCULAR DISEASE

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Abstract

Cerebrovascular accident, is an emergency medical condition characterized by an acute compromise of the cerebral perfusion or vasculature. The leading cause of ischemic stroke is hypertension whereas clotting disorders, carotid dissection, and illicit drug abuse are common causes in the younger populations. A quick diagnosis followed by prompt management needs to be set in motion by the interprofessional team members to improve outcomes for those with stroke. As noted earlier, the risk of stroke increases with age and doubles over the age of 55 in both men and women. The risk is still increases more when a person already has diseases such as hypertension, coronary heart disease or hyperlipidemia. Almost 60% strokes occur in patients with transient ischemic attack (TIA) in the anamnesis.[2]

Key words

Ischemia, cerebral perfusion, syndromes, prevention.

Stroke is the result of ischemia in an area of the brain. The Na+/K+ ATPase pumps fail mainly because of the poor production of adenosine triphosphate (ATP) and failure of the aerobic mechanism. Ischemia leads to depolarization of cells which results in calcium influx into cells, elevated lactic acid, acidosis, and free radicals. Cell death increases glutamate and leads to a cascade of chemicals (excitotoxicity).[1] Ultimately, blocking integrated mechanisms of neuronal death in the ischemic cascade may be difficult. But this is not because these targets are invalid. Indeed, the fundamental neurobiology of these molecular mechanisms is sound. From a clinical perspective, blocking these early targets is difficult because the therapeutic time windows may be extremely narrow. Furthermore, the majority of these pathways do not necessarily occur during the period of arterial occlusion but when the tissue is reperfused or some significant blood flow still persists[3]. Hence, the clinical rationale herein might not be driven by a stand-alone neuroprotective treatment but instead aimed at a combination therapy to be given along with thrombolytic or mechanical reperfusion. Indeed, a recent powerful study provides proof of concept. Tymianski and colleagues recently showed that a compound that blocks the post-synaptic density protein post-synaptic density protein-95 (PSD-95) was able to reduce infarction in a nonhuman primate model of transient focal cerebral ischemia[6].

The most important piece of historical information that the clinician should obtain is the time of symptoms onset or time last seen normal. This is critical because it determines the eligibility to receive rtPA or endovascular intervention for stroke[7]. Other important information to obtain is risk factors for arteriosclerosis and cardiovascular disease, diabetes, smoking, atrial fibrillations drug abuse, migraine, seizures, infection, trauma or pregnancy.[4]

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The stroke exam is a multi-person coordinated rapid exam. While staff obtain vitals, attach telemetry, and obtain IV access, the physician performs a rapid neurological evaluation.[5] National Institutes of Health Stroke Scale (NIHSS) is routinely used to get the baseline evaluation. The exam has to be rapid as "time is brain." One must examine the following items:

- The level of consciousness (alert and responsive, arouses to noxious stimuli, comatose…)
- Language (fluency, naming, comprehension, repetition)
- Dysarthria (slurring) which may be picked up in the history
- Motor (subtle arm weakness can be picked up by performing a pronator drift)
- Visual field deficits
- Eye movement abnormalities (in general if a gaze preference is present, the eyes deviate towards the side of the lesion)
- Facial paralysis (asking the patient to smile)
- Ataxia (finger to nose)

With a good history and physical exam, we can localize the stroke. There are various stroke syndromes.

Anterior Cerebral Artery (ACA) Infarction

There is significant collateral blood supply in the ACA territory. So, pure ACA strokes are rare. The ACA distribution involves mainly Broca's area, primary motor, primary sensory and pre-frontal cortex. So patients present with motor aphasia, personality issues, and contralateral leg weakness and numbness. Hand and face are usually spared.

Middle Cerebral Artery (MCA) Infarction

The MCA has the main trunk (M1) and it divides into two M2 Branches. The M1 (horizontal branch) supplies the basal ganglia and M2 (Sylvian branches) supplies part of the parietal, frontal and temporal lobes. As MCA supplies a wide territory it is extremely important to rule out MCA occlusion. The MCA syndrome causes contralateral arm and facial numbness and weakness, gaze deviation towards the affected side. Aphasia in the left-sided lesions and neglect in the right-sided lesions.

Posterior Cerebral Artery (PCA) Infarction

The PCA mainly supplies occipital lobe, thalamus and some portion of the temporal lobe. The classic presentation of PCA stroke is homonymous hemianopsia. Apart from this hypersomnolence, cognitive issues, the hemisensory loss can be seen when the deep PCA is involved. Some times there is bilateral infarction of distal PCAs producing cortical blindness and the patient is unaware of the blindness and denies it. This is called Anton-Babinski syndrome.[8]

Cerebellar Infarction

The patients with cerebellar strokes present with ataxia, dysarthria, nausea, vomiting, and vertigo.

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Lacunar strokes are due to occlusion of small perforating vessels and can be a pure motor, pure sensory and ataxic hemiparetic strokes. In general, these strokes don't impair memory, cognition, level of consciousness or speech.

The stroke can be quantified by the NIHSS scale which includes the following:

- Visual function
- Level of consciousness
- Sensation and neglect
- Motor function
- Cerebellar function
- Language

A high score suggests proximal vessel occlusion.[8]

Stroke prevention, either primary or secondary is the primary goal of treatment. Patients at the highest risk should be identified early and counseled on lifestyle changes as well as control of comorbid conditions to prevent this devastating outcome. Conditions that increase a patient's risk for cerebrovascular accidents include uncontrolled diabetes mellitus, uncontrolled hypertension, nicotine abuse, and atrial fibrillation. These are the most common and high prevalence diseases, however, other conditions are also linked to vascular pathologies such as vasculitis and certain autoimmune diseases. Primary care and specialty providers must diligently identify these patients and provide focused counseling as well as aggressive therapy for underlying diseases to prevent this outcome. When conducting primary and secondary prevention of cerebrovascular diseases, special attention should be paid to the correction of such “controllable” risk factors such as chronic psycho-emotional stress, smoking, alcohol abuse, excess body weight, as well as adequate treatment of arterial hypertension, ischemic heart disease and diabetes mellitus.

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