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Cerebellar Stroke

Definition
A cerebellar stroke occurs when the blood supply from one or more of the blood vessels (superior cerebellar artery, anterior inferior cerebellar artery, or posterior inferior cerebellar artery) to the cerebellum is interrupted.

Epidemiology
Strokes are the third leading cause of death in North America. Cerebellar strokes occur in 1.5% to 3.8% of all strokes. The mortality rate associated with cerebellar stroke is 23%.

Pathophysiology of Cerebellar Stroke
The cerebellum is the second largest part of the brain. The main functions of the cerebellum include coordination of posture and gait; coordination of voluntary muscle activity and muscle tone; coordination of location in space and movement; and regulation of fine movements especially in the distal muscles of the hand.

The cerebellum is supplied by three arteries: the superior cerebellar artery (SCA) and the anterior inferior cerebellar artery (AICA) which arise from the basilar artery, and the posterior inferior cerebellar artery (PICA) arises from the vertebral artery.

Hypertension and cardioaortic diseases are the major causes of cerebellar infarction. Cerebellar stroke is mainly related to ischemic events, in which, most cases are resulted from embolism (50%); occlusion or stenosis of the cerebellar artery. Arterosclerotic branches in the cerebellar artery (28%) is the second most common cause. Other risk factors for cerebellar strokes include hyperlipidemia, a previous transient ischemic attack, diabetes mellitus, and a hypercoagulable state.

The posterior fossa is very limited in space. It is unable to accommodate the increased volume if edema occurs after cerebellar stroke. Thirty nine percent of patient with a cerebellar stroke develop mass effect due to edema. The mass effect moves the cerebellar tissue forward and upward, which compresses the 4th ventricle and/or cerebral aqueduct and obstructs the cerebrospinal fluid (CSF) flow. Disturbance of the CSF flow results in obstructive hydrocephalus. The ischemic process of cerebellar stroke can be extended to the brain stem and cause brain stem infarction and locked-in syndrome.

Manifestation
The most common manifestations of cerebellar strokes are a loss in coordination that may affect speech (dysarthria), posture/locomotion (bradykinesia), oculomotor movement (nystagmus), and upper limb movement. Patients with a cerebellar stroke may also present with acute vestibular syndrome which is the rapid onset of vertigo, nausea/vomiting, and gait unsteadiness in association with head motion intolerance and nystagmus.

Rapid deterioration in patients with cerebellar stroke is usually related to the stroke extending to the brain stem, and from obstruction of the 4th ventricle and/or the cerebral aqueduct due to cerebellar edema.

Diagnostic tests
Cerebellar stroke is difficult to diagnose because its manifestations are ambiguous and the initial computed tomography scan (CT scan) may be negative. The most commonly used diagnostic tests include CT scan and magnetic resonance imaging (MRI). CT scan is useful to assess a hemorrhage, however, its sensitivity to detect ischemic strokes is only approximate 40% and it is extremely insensitive to identify cerebellar strokes. MRI is able to provide information about the infraction, vessel status, and the extent of the lesion. Diffusion-weighted MRI can detect ischemic lesions more precisely and in the initial hours after stroke onset.

CT scan shows a large cerebellar stroke (Arrow)

A cerebral angiogram may be performed to visualize the status of the cerebral vasculature. It is not commonly performed because it is invasive, but it can be used for removing blood clots for recanalization of the blood vessels.

Other diagnostic tests may include electrocardiogram, transthoracic echocardiogram, transesophageal echocardiogram, or transcranial doppler to assess the etiology of the diagnosis.

Treatment Options
Intravenous thrombolysis has been used to recanalize the occluded cerebellar artery. Intra-arterial thrombolysis has been demonstrated to be...
effective in improving patients' outcome and may even reverse locked-in syndrome. When patients have severe cerebellar edema, urgent surgical decompression of the posterior fossa is required to reduce the increased intracranial pressure and prevent upward herniation or hydrocephalus.

The use of an external ventricular drain to reduce intracerebral hypertension for patients with cerebellar stroke is controversial because of the risk of upward herniation. However, Kirolos et al. suggest using a ventricular drain and surgical intervention together as an effective way to reduce the mass effect of cerebellar edema.

**Nursing Implications**

Timely recognition of neurological deterioration is essential to detect extension of the infarction and/or cerebellar edema. A cerebellar stroke could cause significant neurological deficits especially in the SCA territory. Early rehabilitation is important to optimize functional recovery.

Motor recovery, especially upper limb function, is important to rehabilitate in the first two weeks after the acute phase. The functional disability from ataxia is able to be rapidly improved or overcome with appropriate physiotherapy.

**Reference**