INTRODUCTION

Vascular dementia is the second most common form of dementia after Alzheimer Disease. The condition is not a single disease, it is a group of syndromes relating to different vascular mechanisms.

It is heterogeneous in terms of both clinical phenotype and pathogenetic mechanisms. It may result from multiple cortical infarctions due to cerebral large vessel pathologies or to subcortical ischemic changes such as leukoaraiosis or lacunar infarction due to cerebral small artery disease.
• A sign of a vascular cause for a dementia syndrome is a sudden onset after stroke with a fluctuating or stepwise course. On the other hand, dementia associated with severe white matter lesions (still widely known as Binswanger’s disease) has a gradual onset and is slowly progressive. An additional history of gait disturbance, frequent falls and urinary incontinence is evidence for this diagnosis.

• Typical symptoms of vascular dementia are focal neurological signs such as hemiparesis, hemianopia and pseudobulbar symptoms (swallowing disturbance, spastic dysarthria, uncontrollable laughing or crying).
• **Vascular disease** produces either focal or diffuse effects on the brain and causes cognitive decline. Focal cerebrovascular disease occurs secondary to thrombotic or embolic vascular occlusions.

• Common areas of the brain associated with cognitive decline are the white matter of the cerebral hemispheres and the deep gray nuclei, especially the striatum and the thalamus.

• Hypertension is the major cause of diffuse disease, and in many patients, both focal and diffuse disease are observed together.
Subtypes of Vascular Dementia

- Mild vascular cognitive impairment
- Multi-infarct dementia
- Vascular dementia due to a strategic single infarct
- Vascular dementia due to lacunar lesions
- Vascular dementia due to hemorrhagic lesions
- Binswanger disease
- Subcortical vascular dementia
- Mixed dementia (combination of AD and vascular dementia).
Mild vascular cognitive impairment can occur in elderly persons. It is associated with cognitive decline that is worse than expected for age and educational level, but the effects do not meet the criteria for dementia. These people have subjective and objective evidence of memory problems, but their daily functional living skills are within normal limits.

In multi-infarct dementia, the combined effects of different infarcts produce cognitive decline by affecting the neural nets.

In single-infarct dementia, different areas in the brain can be affected, which may result in significant impairment in cognition. This may be observed in cases of anterior cerebral artery infarct, parietal lobe infarcts, thalamic infarction, and cingular gyrus infarction.
Small vessel disease affects all the small vessels of the brain and produces 2 major syndromes, Binswanger disease and lacunar state. Small vessel disease results in arterial wall changes, expansion of the Virchow-Robin spaces, and perivascular parenchymal rarefaction and gliosis.

Binswanger disease is a progressive neurological disorder caused by arteriosclerosis and thromboembolism, affecting the blood vessels that supply the white-matter and deep structures of the brain.

Most patients experience progressive loss of memory and intellectual abilities, urinary urgency or incontinence, and an abnormally slow, shuffling, unsteady pattern of walking.
Lacunar disease is due to small vessel occlusions and produces small cavitary lesions within the brain parenchyma secondary to occlusion of small penetrating arterial branches. These lacunae are found more typically in the internal capsule, deep gray nuclei, and white matter. Lacunar state is a condition in which numerous lacunae, which indicate widespread severe small vessel disease, are present.

Leukoaraiosis greater than 25% is considered to be pathological. Subcortical vascular dementia is a diffuse small vessel disease with minimal or absent infarction with homogenous pathological and clinical features. White matter ischemic changes affect executive function and cause slower processing speed, rather than memory and language impairment.
Mixed dementia is diagnosed when patients have evidence of Alzheimer’s disease and cerebrovascular disease, either clinically or based on neuroimaging evidence of ischemic lesions. Growing evidence indicates that vascular dementia and Alzheimer dementia often coexist, especially in older patients with dementia. Autopsy studies have shown an association between Alzheimer disease and vascular lesions.

Recent evidence suggests that the vascular processes in both disorders may mutually induce each other. Apolipoprotein E may play a role in Alzheimer disease and vascular dementia. Apolipoprotein E4 also increases the risk of dementia in stroke survivors and is a strong risk factor for the development of cerebral amyloid angiopathy in patients with Alzheimer disease. In elderly individuals, many cases of dementia may be caused by the cumulative effect of cerebrovascular and Alzheimer pathology.
**RISK FACTORS**

Risk factors for vascular dementia include hypertension, smoking, hypercholesterolemia, diabetes mellitus, and cardiovascular and cerebrovascular disease.

Vascular dementia development after stroke can be influenced by many factors. Some of the important factors that can lead to the development of dementia are older age, lower education level, family history of dementia, left-sided lesions, larger lesions, larger periventricular white matter ischemic lesions, and strokes in thalamic artery territory, inferomedian temporal lobes, hippocampus, and watershed infarcts involving superior frontal and parietal regions.
MEDICAL CARE

• The mainstay of management of vascular dementia is the prevention of new strokes. This includes administering antiplatelet drugs and controlling major vascular risk factors. Aspirin has also been found to slow the progression of vascular dementia.

• Drug treatment is primarily used to prevent further worsening of vascular dementia by treating the underlying disease such as hypertension, hyperlipidemia, and diabetes mellitus.
• A common feature of most dementias is lack of acetylcholine in the brain, and evidence is accumulating that such a deficit may also play a role in the cognitive symptoms in vascular dementia. One possible way of increasing the level of cerebral acetylcholine is by inhibiting its degradation by acetylcholine esterase. This therapeutic strategy has some effect in Alzheimer’s disease but there is no data available in vascular dementia.

• In recent years, acetylcholinesterase inhibitors have attained wide use in the treatment of Alzheimer’s disease. Drugs such as Donepezil may improve memory and other cognitive measures among Alzheimer’s disease patients. These drugs also may have beneficial effects on associated behaviors such as apathy and degree of cooperativeness.
• **Donepezil**, which was approved for use in late 1996 has effects on the cognitive and behavioral symptoms of patients with Vascular Dementia.

• **Galantamine** is a novel cholinergic drug that both inhibits acetylcholine esterase activity and stimulates the acetylcholine receptor. In a recently completed, large, randomised trial, there were beneficial effects of 24 mg daily on cognition, global function, functional abilities and behavioural symptoms in patients with probable vascular dementia. The beneficial effects appeared to last for at least 6 months and may even be sustained for months.
PROGNOSIS

In patients with dementia who have had a stroke, the increase in mortality is significant. The 5-year survival rate is 39% for patients with vascular dementia compared with 75% for age-matched controls. Vascular dementia is associated with a higher mortality rate than Alzheimer’s disease, because of the coexistence of other atherosclerotic diseases. Study on causes of death in patients with dementia showed that circulatory system disorders (e.g., ischemic heart disease) is the most common immediate cause of death in vascular dementia, followed by respiratory system diseases (e.g., pneumonia). A study of hospitalization rates in patients with dementia showed that persons who developed different types of incident dementia, including vascular dementia, were found to have an increased risk of hospitalization, including hospitalization for ambulatory care-sensitive conditions.
CONCLUSIONS

• Researchers think that Vascular Dementia will become more common in the next few decades because it is generally caused by conditions that occur most often in older people, such as atherosclerosis, heart disease, and stroke. Another aspect is that people are living longer with chronic diseases such as heart disease and diabetes.

• If the conditions that cause vascular dementia go untreated, the prognosis is not good. A person with vascular dementia may seem to improve for periods of time until another stroke takes away more brain function, memory, and independence. Eventually, untreated vascular dementia usually ends in death from stroke, heart disease, or infection.

• Although vascular dementia is a serious condition, catching it early and preventing further damage are the best medicine. People with vascular dementia can work with their doctors and families to detect and manage the condition.
THANK YOU!