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Developments in the Diagnosis and Treatment of Vascular Dementia

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It has been forecasted that more than 152.8 million people will be living with dementia by 2050. (Morgan AE, Mc Auley MT.) With vascular dementia accounting for around 20% of all cases, the need to identify and treat this condition will only intensify over time.

Recent advances in both the diagnosis and treatment of vascular dementia point to some encouraging signs, but more work remains for professionals on the front lines of long-term care to prepare for this rising population of vascular dementia sufferers.

An emerging picture

The GuideStar Eldercare blog post Understanding Vascular Dementia provides a comprehensive overview of how vascular dementia is understood and categorized. As evident by the name, vascular dementia is a cognitive impairment brought on by a vascular injury. The journal Arteriosclerosis, Thrombosis and Vascular Biology lists a number of these injuries: (Wolters FJ, Ikram MA.)

- Stroke
- Cerebral small vessel disease (atherosclerosis and arterio(lo)sclerosis)
- (Micro) Infarcts
- Cerebral amyloid angiopathy

Vascular dementia remains difficult to diagnose and treat, due to diverse factors. It can co-occur with other forms of dementia, such as Alzheimer's disease. Also, the symptoms exhibited by vascular dementia patients can present differently depending on where the brain injury exists.

Dementia remains a serious risk for stroke patients, as its effects may still occur even after the inciting injury is treated successfully. The Journal of stroke and cerebrovascular diseases noted, "It is unfortunately well recognized that "good" functional recovery... does not necessarily translate into good cognitive recovery." (Bir SC et al.)

Advances in diagnostics

As the journal Stroke and vascular neurology has observed, the classification of vascular dementia has evolved over time. Specifically, vascular cognitive impairment (VCI) has been introduced as a term to include a broader spectrum of cognitive impairment, with vascular dementia (VaD) classified as the most severe form. (Ng S et al.)

This broader view of VCI is good news for patients. According to the Journal of stroke and cerebrovascular diseases, "Identification of vascular cognitive impairment, preceding actual vascular dementia, may allow... for halting or delaying progression of the cognitive deficit." (Bir SC et al.)

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Researchers are leaving no area unexplored in the pursuit of better diagnostics for VCI. Stroke and Vascular Neurology has cited "a growing interest in biochemical markers to distinguish VaD patients from other dementia patients." (Ng S et al.)

Their research noted that lipocalin 2 (LCN2) can be found at elevated levels in vascular dementia patients. LCN2 is generally secreted in response to injury and inflammation. This elevated cerebrospinal fluid level was useful in distinguishing vascular dementia patients from those with Alzheimer's disease—with sensitivity of 82% and specificity of 87%. (Ng S et al.)

Another biomarker that shows promise is the so-called "anti-aging" Klotho protein. Ng S et al recorded that in VaD patients, this protein has been found at lower levels. However, this lower level of Klotho has not been associated with late-onset Alzheimer's disease.

Neuroimaging has also proven to establish a baseline of difference in the presentation of vascular dementia versus Alzheimer's disease. Damage to the brain is widespread within the white matter in VaD scans, especially in thalamic radiations and in the corpus callosum. In Alzheimer's patients, the damaged regions appear mostly in the parahippocampal pathways and the splenium. (Ng S et al.)

Experimental treatments

Addressing underlying risks has proven effective in holding off vascular dementia. As observed in Continuum, "Control of cardiovascular risk factors, including… blood pressure, cholesterol, and blood sugars, remains the mainstay of prevention." (Chang Wong E, Chang Chui H.) Indeed, Wolters and Ikram have also noted that "control of cardiovascular risk factors… is likely to have contributed to the reported decline in the age-specific incidence of dementia over the past decades."

Understanding these risks involves deepening our understanding of cerebral small vessel disease (cSVD), one of the chief causes of vascular dementia. Experimental data published in Molecular degeneration has identified a number of potential etiologies for cSVD: (Inoue Y et al.)

- Lack of oxygen to tissues due to decreased blood flow (hypoperfusion/hypoxia)
- Dysregulation of blood-brain barriers
- Brain fluid drainage disturbances
- Vascular inflammation

The role inflammation appears to play in vascular dementia presents a positive avenue for potential treatment. In fact, daily doses of N-acetylcysteine (an antioxidant and anti-inflammatory) have shown to improve the Dementia Rating Scale for some patients with mild cognitive impairment. (Ng S et al.)

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Serotonin also seems to play a role. Experimental treatment with Pimavanserin, a serotonin receptor modulator, has been found to reduce relapse rates for vascular dementia patients. Stem-cell infusion shows promise, too, leading to improved Dementia Rating Scale scores in some patients. (Ng S et al.)

Another treatment to be explored further involves stimulation of the brain through electrical and/or magnetic means. This type of non-invasive neuromodulation was conducted in a prospective pilot study and led to improved cognitive performance and lower cytokine levels. (Ng S et al.)

Help for vascular dementia patients

As diagnosis and treatment improves, more can be done to improve quality of life for vascular dementia patients in your facilities. GuideStar Eldercare specializes in bedside neurology for long-term care patients, and our clinical service teams can assist you in diagnosing and treating dementia and its co-occurring conditions. Contact us today and see how we can help.

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