Vascular cognitive impairment (VCI) refers to any severity of cognitive impairment (from subjective complaints only to dementia) caused by vascular pathology. Patients with VCI typically have deficits in memory and executive function, mental slowness and behavioural problems such as anxiety and depression.

Several diagnostic criteria for VCI are available, each of which has different levels of supporting evidence. The two most recent criteria are the Diagnosis and Statistical Manual of Mental Disorders, Fifth edition (DSM-5) classification and the American Heart Association/American Stroke Association scientific statement on Vascular Contributions to Cognitive Impairment and Dementia. Both of these criteria require the presence of, and a temporal link between, cognitive impairment and cerebrovascular disease for diagnosis. Diagnostic work-up of individuals with suspected VCI includes neuroimaging, cognitive testing and laboratory testing. Neuroimaging, preferentially MRI, is essential and can be used to identify the type, severity and location of cerebrovascular pathology.

The mechanisms of VCI are complex and are not fully understood. The best described mechanism is direct tissue injury, such as macroscopic infarcts, microinfarcts, microbleeds and white matter injury. This tissue injury can be caused by intracranial vessel diseases (such as atherosclerosis, arteriolosclerosis and cerebral amyloid angiopathy). In addition, intracranial vessel diseases are independently associated with VCI when infarcts and vascular risk factors are controlled, suggesting that mechanisms other than direct tissue injury are involved.

No symptomatic pharmacological treatments for VCI have been approved by the US FDA. In general, management includes improving control of vascular risk factors and interventions to slow the progression of cognitive decline in individuals with vascular-related mild cognitive impairment. In patients with stroke, antihypertensive, antithrombotic or lipid-lowering drugs, in addition to lifestyle modifications, should be used to prevent recurrent stroke. However, although the prevention of recurrent stroke should, in principle, prevent further cognitive defects, whether conventional therapies actually achieve this is controversial.