



Diagnosis and management of vascular dementia

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Abstract

Vascular dementia describes chronic progressive impairment of cognitive function arising from cerebrovascular injury. Although overlap exists with Alzheimer's disease (around 20% of dementia patients have a mixed form) vascular dementia is regarded as the second most common cause of dementia in the UK, a reasonably pure form constituting 15-20% of dementia cases in Europe and North America. Vascular cognitive impairment (VCI) refers to the full spectrum from mild cognitive impairment to dementia associated with, and presumed to be caused by, cerebrovascular disease. The most common cause of VCI is cerebral small vessel disease. The presentation of vascular dementia is often insidious, with disorganisation, loss of drive, irritability, mental rigidity, difficulties with planning and problem solving, emotional lability and other mood changes, and sometimes inappropriate or disinhibited social behaviour. Slowed gait is common in those with advanced disease. Physical examination may identify gait impairment, bradykinesia, rigidity, and focal neurological signs. Screening tools are insensitive in vascular dementia, in which memory can be relatively preserved. All patients should have their BP checked. Vascular risk factors are not essential for the diagnosis but should be identified, as should any reversible medical causes. Patients with impairment of sufficient severity to classify as dementia will require input from a dedicated dementia service or community mental health team. Objective evidence of cognitive impairment is required for diagnosis: the cognitive profile is dominated by executive dysfunction, slowed processing speed and impaired retrieval. Imaging is a prerequisite for diagnosis.

Dr Killian Welch
BSc MBChB MPhil MD MRCPsych
Consultant Neuropsychiatrist

Dr Parvez Thekkumpurath
MB BS MRCPsych
Consultant Old Age Psychiatrist
Royal Edinburgh Hospital, Edinburgh, UK



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AUTHORS

Dr Killian Welch
BSc MBChB MPhil MD
MRCPsych
Consultant
Neuropsychiatrist

Dr Parvez Thekkumpurath

MB BS MRCPsych
Consultant Old Age
Psychiatrist

Royal Edinburgh
Hospital, Edinburgh, UK

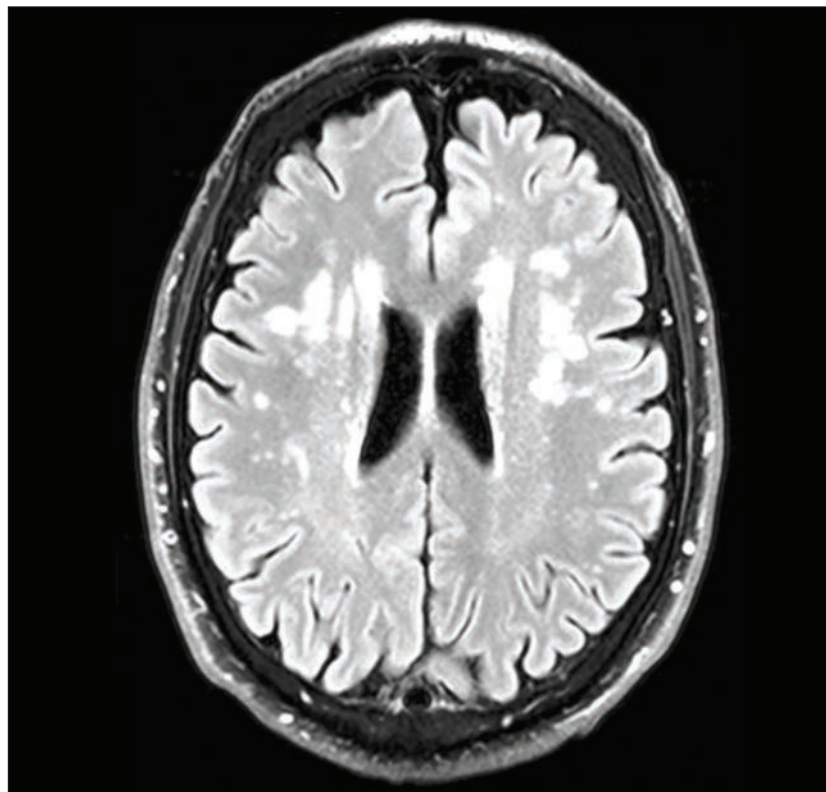


FIGURE 1

Axial MRI scan showing evidence of periventricular and deep white matter hyperintensities, both common in vascular cognitive impairment



VASCULAR DEMENTIA (VaD) DESCRIBES CHRONIC PROGRESSIVE IMPAIRMENT OF COGNITIVE FUNCTION

arising from cerebrovascular brain injury.

Although overlap exists with Alzheimer's disease (AD) (around 20% of dementia patients have a mixed form¹) VaD is regarded as the second most common cause of dementia in the UK. A reasonably pure form constitutes 15-20% of dementia cases in Europe and North America.²

As with AD, the incidence of VaD increases steeply with age, contributing to the growing epidemic of dementia in ageing populations worldwide.³

The estimated incidence in people aged 65-69 years is 0.3-2.2 per 1,000 per annum, rising to 9.3-15.9 per 1,000 per annum in those aged over 90.⁴ It has been argued that, owing to improvement in the management of vascular disease, the incidence of vascular dementia may have decreased in recent decades. Indeed, the age-specific risk of all-cause dementia in the USA and Europe has declined by about

20% per decade since the late 20th century, paralleled by reductions in the burden of cerebral small vessel disease.⁵

AETIOLOGY

Dementia, renamed major neurocognitive disorder in DSM-5, is the loss of cognitive functioning (thinking, remembering, and reasoning) to such an extent that it interferes with a person's daily life and activities.⁶

Vascular cognitive impairment (VCI) is a broader concept. It covers the full spectrum of cognitive impairment from mild cognitive impairment to dementia associated with, and presumed to be caused by, cerebrovascular disease.

Diagnostic criteria for VaD have been refined in recent years and currently require establishing the presence of dementia syndrome with clinical features indicative of vascular aetiology. These include onset after stroke, decline in complex attention and frontal executive function, and evidence of cerebrovascular disease from history, examination or imaging.⁶

However, establishing a link between

What are the presenting symptoms?

How should patients be investigated?

What are the treatment strategies?

onset of dementia and the presence of cerebrovascular disease can be challenging. Vascular brain changes are common in people with other dementias such as AD as well as in older people without dementia.⁷ These considerations help explain why for many patients, especially older patients in their eighth or ninth decade, labelling their dementia as having a mixed aetiology is more appropriate.

The most common cause of VCI is cerebral small vessel disease, such as arteriolosclerosis and cerebral amyloid angiopathy. Other less common causes include CADASIL (cerebral autosomal dominant arteriopathy with subcortical infarcts and leukoencephalopathy) a type of subcortical small vessel disease with a genetic basis.⁸ Typical features of CADASIL are shown in table 1, p12.⁸

Arteriolosclerosis predominantly affects subcortical brain regions, including the corona radiata and basal ganglia, and patients typically have multiple lacunar infarcts (cystic lesions < 1.5 cm) or extensive, confluent white matter lesions,⁹ (see figure 1, above).

Table 1

Typical presentation of CADASIL. Many features are shared by other less common forms of hereditary small vessel disease, adapted from Di Donato et al³⁴

- Migraine, usually with aura, as the first symptom in the third decade of life
- Recurrent subcortical ischaemic events (transient ischaemic attack/stroke) in adulthood
- Mood disturbances, apathy and depression among other psychiatric symptoms
- Progressive cognitive decline, especially of executive functioning
- Seizures in a small but well defined proportion of patients
- MRI shows bilateral, symmetrical abnormalities

Cerebral amyloid angiopathy, the second most common cerebral small vessel disease, is caused by beta-amyloid deposition in small arteries. Vascular integrity is lost leading to haemorrhage, most commonly in lobar locations while sparing the basal ganglia and brainstem.⁸ Table 2, right, lists the specific vascular dementia syndromes.¹⁰

PRESENTATION

Though vascular dementia can present with the classical stepwise decline, insidious deterioration is more common, with disorganisation, loss of drive, irritability, mental rigidity, difficulties with planning and problem solving, emotional lability and other mood changes, and sometimes inappropriate or disinhibited social behaviour.¹¹

The cognitive profile is dominated by executive dysfunction, slowed processing speed and impaired retrieval (memory difficulties that improve with cueing, e.g. being able to identify a word when given multiple choice options).

Slowed gait is common in those with advanced disease.¹¹ Impaired balance, falls and incontinence can also occur.

The extent of vascular disease may be unrecognised until revealed by imaging. Imaging is a prerequisite for diagnosis. The presence of vascular damage is not sufficient for a vascular dementia diagnosis and, as mentioned above, the number of patients for whom vascular pathology is a contributor to their dementia likely exceeds those with a pure vascular dementia.¹²

If a single ischaemic or haemorrhagic stroke is large enough, or simply damages a crucial brain region such as the thalamus or language cortices, (i.e. a strategic infarct), it can be associated with substantial cognitive impairment. There may be stroke-related symptoms and signs, such as aphasia or motor weakness, meaning the association between vascular brain damage and cognitive impairment should be clear.

Though cognitive impairment may be

static or improve if vascular risk factors are treated and there are no further strokes, stroke is a strong and independent risk factor for incident dementia.¹³ At any point following stroke, more than one in five people will have dementia; in around a quarter of these dementia will have predated stroke, but in three-quarters it will be a new diagnosis.¹⁴

This relationship between stroke and dementia is complex and variable. Stroke may lead to a clear picture of VaD, may trigger Alzheimer's pathology or exacerbate prestroke cognitive decline of either vascular or Alzheimer's pathology.

ASSESSMENT

Insidious or stepwise cognitive deterioration, potentially in association with overt stroke pathology, should prompt consideration of vascular dementia as a potential diagnosis. Equally, executive dysfunction, cognitive slowing, mood and behavioural changes along with gait and balance problems in the absence of overt memory problems should also prompt consideration of vascular dementia. Collateral history should be sought, including a timeline of cognitive and functional changes. Vascular risk factors should be identified but are not essential for the diagnosis (see table 3, right).

Potentially reversible medical causes which can masquerade as dementia should be considered. This could include depression, but neuropsychiatric disorders are particularly common in vascular dementia, so comorbidity is also likely.

Other possibilities are brain lesions (including subdural haematoma following recent head injury), nutrient or vitamin deficiencies (B1, B6, B12, folate), endocrine conditions (including hypothyroidism), chronic infection and inflammation, medication (including anticholinergics, benzodiazepines, opioids), alcohol, sensory impairment (loss of hearing or vision).

Table 2

Specific vascular dementia syndromes, adapted from Stewart¹⁰

Genetic disorders

- CADASIL
- Amyloid angiopathy
- Homocystinuria
- Sickle cell disease
- Fabry's disease
- HERNs (hereditary endotheliopathy with retinopathy, nephropathy and stroke)
- MELAS (mitochondrial myopathy, encephalopathy, lactic acidosis, and stroke)

Strategic infarct dementia

- Usually in thalamic structures

Conditions associated with cerebrovascular pathology

- Subdural haematoma
- SLE (systemic lupus erythematosus)
- Polyarteritis nodosa
- Buerger's disease
- Polycythaemia rubra vera
- Neurosyphilis

Table 3

Risk factors for vascular cognitive impairment in the general population, adapted from Zanon Zotin et al²³

Stronger risk factors

- Hypertension (especially midlife hypertension)
- Obesity (especially midlife obesity)
- Smoking

Weaker risk factors

- Diabetes
- Dyslipidaemia (may have greater impact on AD)
- Sleep apnoea

Physical examination is particularly important if vascular dementia is suspected. It may identify gait impairment, bradykinesia, rigidity, and focal neurological signs.

Objective evidence of cognitive impairment is required for diagnosis. Screening tools for dementia may be particularly insensitive in vascular dementia, in which memory can be relatively preserved. There is data to support the use of the Montreal Cognitive Assessment in separating vascular dementia from controls;¹⁵ its relative sensitivity to executive deficits makes it intuitively a good choice.

key points

SELECTED BY

Dr Phillip Bland

Former GP, Dalton-in-Furness, UK

Vascular dementia describes chronic progressive

impairment of cognitive function arising from cerebrovascular injury. Although overlap exists with Alzheimer's disease (AD) (around 20% of dementia patients have a mixed form) vascular dementia is regarded as the second most common cause of dementia in the UK, a reasonably pure form constituting 15–20% of dementia cases in Europe and North America.

Vascular cognitive impairment (VCI) refers to the full

spectrum from mild cognitive impairment to dementia associated with, and presumed to be caused by, cerebrovascular disease. The most common cause of VCI is cerebral small vessel disease. Arteriolosclerosis predominantly affects subcortical brain regions: patients typically have multiple lacunar infarcts or extensive, confluent white matter lesions. Cerebral amyloid angiopathy is caused by beta-amyloid deposition in small arteries.

Most commonly the presentation of vascular dementia

is insidious, with disorganisation, loss of drive, irritability, mental rigidity, difficulties with planning and problem solving, emotional lability and other mood changes, and sometimes inappropriate or disinhibited social behaviour. Slowed gait is common in those with advanced disease.

Physical examination may identify gait impairment,

bradykinesia, rigidity, and focal neurological signs. Screening tools are insensitive in vascular dementia, in which memory can be relatively preserved. There is data to support the use of the Montreal Cognitive Assessment but normal performance does not exclude vascular dementia. All patients should have their BP checked. Vascular risk factors are not essential for the diagnosis but should be identified, as should any reversible medical causes.

Young patients (aged under 65), atypical or rapidly

progressive cases will likely be referred to neurology. Cases arising after a clear stroke will likely be under the care of stroke services. Patients with impairment of sufficient severity to classify as dementia will require input from a dedicated dementia service or community mental health team. Objective evidence of cognitive impairment is required for diagnosis: the cognitive profile is dominated by executive dysfunction, slowed processing speed and impaired retrieval. Imaging is a prerequisite for diagnosis. However, vascular brain changes are common in people with other dementias such as AD as well as in older people without dementia.

Expert consensus suggests treating hypertension in

overt VCI but it is important to be vigilant for orthostatic hypotension. Statins have not been shown to prevent cognitive decline or dementia in people at risk of vascular disease. The effects of cholinesterase inhibitors and memantine are small and of uncertain clinical benefit in dementia of solely vascular aetiology. Many patients have a mixed dementia, with coexisting AD, and are more likely to benefit.

As ever, normal performance on a screening tool does not exclude dementia.

All patients should have their blood pressure (BP) checked and a urine dipstick. Blood tests should include full blood count, urea & electrolytes, thyroid function tests, vitamin B12 and folate, liver function tests, calcium, lipid profile and glucose/HbA_{1c}. Substance use should be enquired about and gamma-glutamyl transferase tested if alcohol excess is suspected.

Lupus anticoagulant, antiphospholipid, and antinuclear testing should be performed in selected patients with atypical presentations or at increased risk for these conditions (e.g. young people with recurrent strokes) and syphilis serology if indicated.

It is useful to distinguish patients with atherosclerotic ischaemic disease from those with cardioembolic disease (e.g. atrial fibrillation, vascular heart disease); ECG will obviously be helpful in identifying an arrhythmia. Imaging will likely be deferred until review by secondary care but is central to diagnosis. Magnetic resonance imaging (MRI) is more sensitive than computed tomography for signs of cerebral small vessel disease, including microbleeds.

MANAGEMENT OF VASCULAR RISK FACTORS

Since risk factors for VCI are modifiable, targeting these would be expected to reduce the incidence and progression of VCI/VaD. In patients who have experienced a stroke there is clear guidance on the use of antithrombotics, antihypertensives, lipid lowering agents and consideration of endarterectomy (depending on patient characteristics).¹⁶

Hypertension

The situation with VaD is more complex. Hypertension, especially high BP during midlife, is associated with a higher risk of developing cognitive impairment and dementia.¹⁷ This has implications beyond pure VaD and may help to explain why so many patients have a mixed aetiology dementia.

It is thought, for example, that one of the main mechanisms underlying cognitive dysfunction in hypertension involves cerebral vessel remodelling, leading to changes in cerebral autoregulation and perfusion, and thus affecting the ability to clear beta-amyloid;¹⁸ this has obvious implications for the development of AD pathology as well as VCI.

Studies examining the impact of tight BP control on the development of

dementia have reported mixed results. The SPRINT-MIND study, for example, reported reduction in the risk of MCI in intensive compared with standard BP control (i.e. BP target < 120 mmHg vs < 140 mmHg) by 20%, with a 16% risk reduction in both MCI and probable dementia (though no statistically significant reduction was seen for probable dementia alone).

While midlife hypertension undoubtedly has a deleterious effect on later life cognitive function,¹⁹ intervention for high BP in those aged 80 years or older has not been shown to reduce incidence of dementia and orthostatic hypotension is associated with an increased risk of dementia and cognitive decline.²⁰

Studies examining the impact of hypertension control on progression of vascular dementia are lacking. Nonetheless, given the strong evidence that lowering BP with antihypertensive drugs was associated with a reduced risk of stroke,²¹ and the likelihood that it reduces incidence of mild cognitive impairment and dementia,²² effectively treating hypertension in primary care is a public health priority.

While vigilance for orthostatic hypotension is important (i.e. check sitting and standing BP in these patients; if there is a postural drop ≥ 10 mmHg, record the standing BP), expert consensus suggests treating hypertension in overt VCI.²³ Guidance in this specific population is lacking, but NICE advises a target BP of 140/90 mmHg for those younger than 80 years and 150/90 mmHg for people 80 or older.²⁴ Any advantages of aggressive treatment that allows BP to reach below 120 mmHg are still unclear and may be associated with harm in some.²⁵

Other risk factors

The main role of statins in the treatment of vascular dementia is in preventing further ischaemic events. Despite good evidence for secondary stroke prevention, they have not been shown to prevent cognitive decline or dementia in people at risk of vascular disease.²⁶

Blood glucose control in patients with diabetes should be optimised given it can reduce macrovascular and microvascular disease risk. Assistance should be given to stop smoking, alcohol intake minimised and weight loss advised and supported if indicated.

REFERRAL

The appropriate secondary care referral route will depend on local services. »

Young patients (under the age of 65), atypical or rapidly progressive cases will likely be referred to neurology. Cases arising after a clear stroke will likely be under the care of stroke services.

Patients with impairment of sufficient severity to classify as dementia will require input from a psychiatric team, either a dedicated dementia service or community mental health team.

SPECIFIC TREATMENTS

General issues in the management of dementia were discussed in our previous article on AD in this journal.²⁷ The focus is on ensuring the safety of the patient and others, giving reassurance and support to the patient and carers, and identifying and treating comorbidity. A multidisciplinary team is often required, including nurses, physiotherapists, occupational therapists, psychologists, and social workers.

The effects of cholinesterase inhibitors and memantine are small and of uncertain clinical benefit in patients with dementia with a solely vascular aetiology.²⁸ Many patients have a mixed dementia however, with coexisting AD, and are more likely to benefit.

COMORBIDITY

Neuropsychiatric comorbidity is common in vascular dementia. Apathy is reported in more than 50%,²⁹ agitation in 40%,³⁰ and depression in 25% (compared with 15% in AD).³¹ Though evidence is weak, antidepressant treatment should be considered if depression is suspected. SSRIs, with their lower side effect burden are favoured. These generally well tolerated drugs are, however, still associated with risks, including postural hypotension, falls, hyponatraemia and bleeds, so treatment must be individualised. They may also, with careful patient selection and open discussion of risks, be trialled in agitation.

Though there is little data in vascular dementia, post-stroke psychosis (delusions and/or hallucinations with poor insight) has been reported to occur in around 5% of stroke patients.³² Given the concerns about metabolic syndrome, adverse cardiac effects and association with stroke, antipsychotics are used with caution in this population. In the presence of frank psychosis use will likely be justified, but this would necessitate psychiatric input, and unfortunately a Cochrane review suggests effects can be disappointing.³³

Competing interests: None

FURTHER READING

For healthcare professionals

Zanon Zotin MC, Sveikata L, Viswanathan A, Yilmaz P. Cerebral small vessel disease and vascular cognitive impairment: from diagnosis to management. *Curr Opin Neurol* 2021;34:246-57

This paper provides a good overview of the aetiology and clinical presentation of vascular dementia as well as a helpful discussion on the complex area of managing risk factors to try and slow progression.

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Useful information

For patients

Alzheimer's Society

www.alzheimers.org.uk/about-dementia/types-dementia/treatment-and-support-vascular-dementia

Royal College of Psychiatrists

www.rcpsych.ac.uk/mental-health/problems-disorders/memory-problems-and-dementia

NHS

www.nhs.uk/conditions/vascular-dementia

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