Vascular Dementia

Synonym: vascular cognitive impairment

See related separate articles Dementia, Supporting the Family of People with Dementia and Alzheimer's Disease.

Vascular dementia (VaD) is not a single disease but a group of syndromes of cognitive impairment caused by different mechanisms causing ischaemia or haemorrhage secondary to cerebrovascular disease (multiple infarcts, single strategic infarct or small vessel disease.) Increasingly the term vascular cognitive impairment (VCI) is used to encompass the spectrum of deficit, in which VaD is the most severe form of the disease.\[1\]

Aetiology

The main subtypes of VaD are:

- Stroke-related VaD. This incorporates multi-infarct dementia, the result of a series of small strokes, which in themselves may not be recognised, and single-infarct dementia, which occurs after a larger stroke.
- Subcortical VaD (small-vessel disease or Binswanger's disease).\[2\]
- Mixed dementia. Changes of both VaD and Alzheimer's disease are found together in the brain. The distinction between VaD and Alzheimer's dementia is becoming increasingly blurred because vascular risk factors play a role in both diseases and both types of dementia may co-exist in the same patient. Similar patterns of biochemical abnormalities are also seen on proton magnetic resonance spectroscopy.\[3\] However, where they are seen mostly in white matter in VaD, in Alzheimer's dementia they predominate in cortical grey matter.

Most VaD is sporadic, but some cases display familial traits. The most common form of inherited VaD is CADASIL (= cerebral autosomal dominant arteriopathy with subcortical infarcts and leukoencephalopathy).\[4\] This is rare, and occurs in younger individuals.

Risk factors for VaD include:\[1\]

- History of stroke or transient ischaemic attack (TIA).
- Atrial fibrillation.
- Hypertension.
- Diabetes mellitus.
- Hyperlipidaemia.
- Smoking.
- Obesity.
- Coronary heart disease.
- Family history of stroke or cardiovascular disease

Epidemiology

VaD is the second most common form of dementia in the West after Alzheimer's disease. It is the most common form in some parts of Asia. Incidence increases with age.

VaD is thought to account for around 17% of dementia in the UK.\[5\]

Prevalence of dementia following a first stroke varies depending on location and size of the infarct, definition of dementia, interval after stroke and age among other variables. Overall, stroke doubles the risk of developing dementia.\[1\]

Presentation

Characteristically, VaD is a progressive disease where deteriorations may be sudden or gradual but tend to progress in a stepwise manner. In contrast to acute confusional state (which is usually of recent onset and may have a reversible cause), in dementia the history should go back at least several months and usually several years.

NINDS-AIREN criteria for the clinical diagnosis of PROBABLE vascular dementia (VaD) - as recommended by the National Institute for Health and Care Excellence (NICE)\[6, 7\]

- Presence of dementia - cognitive decline from higher level of functioning. This can be demonstrated as memory loss plus impairment in two or more different cognitive domains (see 'Diagnosis', below). This should be established by clinical examination and neuropsychological testing. Deficits should be severe enough to interfere with activities of daily living - not secondary effects of the cerebrovascular event alone.
Cerebrovascular disease, defined by the presence of signs on neurological examination and/or by brain imaging. A relationship between the above two disorders inferred by:
- Onset of dementia within three months following a recognised stroke.
- An abrupt deterioration in cognitive functions.
- Fluctuating, stepwise progression of cognitive deficits.

Presentation varies significantly, as does speed of progression. Presenting features which may suggest a vascular cause include:
- Focal neurological abnormalities: visual disturbances (eg, field defects), sensory or motor symptoms (eg, dysphasia, hemiparesis, visual field defects) or extrapyramidal signs (eg, dystonias and Parkinsonian features).
- Difficulty with attention and concentration.
- Seizures.
- Depression and/or anxiety accompanying the memory disturbance.
- Early presence of disturbance in gait, unsteadiness and frequent, unprovoked falls.
- The patient has bladder symptoms (eg, incontinence) without a demonstrable urological condition.
- Features of pseudobulbar palsy
- Emotional problems - eg, emotional lability, psychomotor retardation or depression.

For objective evidence, carry out a test of cognitive functioning (see under 'Diagnosis', below).

Also consider dementia with Lewy bodies (DLB) in elderly patients presenting with hallucinations, lucid periods, movement disorders, falls or syncope. Making this diagnosis will have important implications for treatment, as the use of neuroleptics in these patients is associated with an increased risk of adverse reactions, and may cause an increase in mortality. [7]

Diagnosis
The diagnosis of dementia requires: [7]
- Comprehensive history and physical examination. The key to diagnosis is a good history of progressive impairment of memory and other cognitive functioning (usually requiring the help of a spouse, relative or friend).
- A formal screen for cognitive impairment - see separate article Screening for Cognitive Impairment. Specific notes should be made on the following:
  - Memory - both short- and long-term.
  - Individual cognitive domains:
    - Orientation - time, place, person.
    - Attention and concentration ability.
    - Language function (usually evident during questioning).
    - Visuospatial functions.
    - Executive function - problem solving, etc.
    - Motor control.
    - Praxis - whether they can get dressed, lay a table, etc.
- Medication review to ensure cognitive decline is not due to medication.
- Other reversible organic causes to have been excluded.
- MRI scanning may show evidence of infarcts, cortical lacunae, and extensive white matter changes. [8] Appearances will vary depending on the pathogenesis. It may help distinguish VaD from Alzheimer's disease.

Note: it is important to identify depression and treat appropriately. Sometimes it is difficult to distinguish between depression and dementia and depression is quite common in dementia. If in doubt, treat.

General management
Like other dementias the treatment is symptomatic, addressing the individual's main problems and supporting the carers. Detecting and addressing cardiovascular risk factors is also very important to try to slow progression. See separate article Cardiovascular Risk Assessment.

General principles of management of people with dementia are addressed in the separate article 'Dementia', and in National Institute for Health and Care Excellence (NICE) Quality Standards. [9, 10]

Non-pharmacological interventions should be tailored to the individual person's preferences and abilities as well as to local resources, and adapted depending on response. These include:
- Cognitive stimulation programmes.
- Multisensory stimulation.
- Music and art therapy.
- Dancing.
- Massage.
- Aromatherapy.
- Structured exercise programmes.
- Animal-assisted therapy.

Comorbid emotional or psychiatric disorders should be addressed by non-pharmacological means (as above) and pharmacological methods as appropriate.

**Community and hospital care**

Patients should be cared for in the community as much as possible. However, if they become severely disturbed and need to be contained for their own safety or the safety of others, inpatient care should be considered (this might include those liable to be detained under the **Mental Health Act 1983**). Inpatient admission would also be justified for patients with complex physical and psychiatric problems who could not be properly assessed in the community.

**People with challenging behaviour**[7, 9]

This patient group is given a specific mention. They should be offered early assessment which includes:

- Physical health.
- Depression and any psychosocial issues.
- Possible undetected pain or discomfort.
- Adverse effects of medication.
- Life history, including spiritual, cultural and religious identity.
- Physical environment.
- Behavioural and functional analysis by a skilled professional.

Factors which may exacerbate violent or aggressive behaviour, or increase the risk of harm to self or others, include:

- Overcrowding.
- Lack of privacy.
- Boredom or lack of activity.
- Poor communication.
- Conflict.
- Weak clinical leadership in care home settings.

Staff should identify, monitor and address factors such as these, and be trained in managing aggression or agitation.

**Pharmacological management**[7]

There is no specific pharmacological treatment approved for the treatment of vascular dementia. Acetylcholinesterase inhibitors and N-methyl-D-aspartate (NMDA) antagonists such as memantine are not currently recommended by NICE for use in non-Alzheimer's dementia. Studies have shown donepezil improves cognitive function for some patients with VaD.[11] It has less consistent effects on global function and activities of daily living. [11, 12] Galantamine has been shown to improve cognition (including executive function) in patients with VaD. It has good safety and tolerability. [14] However, it was shown to provide no improvement in activities of daily living compared to placebo. There have also been studies on rivastigmine and memantine, but again so far there has not been significant benefit demonstrated, and none has been recommended for use in vascular dementia. [15]

Medication for non-cognitive symptoms (eg, emotional symptoms) and challenging behaviour

This should only be used if there is severe distress or immediate risk of harm to the patient or others. NICE does not recommend the use of antipsychotics for mild-to-moderate non-cognitive symptoms in dementia with VaD or mixed dementia because of the increased risk of cerebrovascular adverse events and death. For severe symptoms (eg, psychosis and/or agitated behaviour causing significant distress), antipsychotics should only be prescribed once the risks and benefits have fully been considered and discussed with carers, risk factors have been assessed and a regular assessment has been made of changes in cognition. Treatment, when considered appropriate, should be time-limited. Comorbid conditions such as depression should be considered and treated.

**Urgent treatment of challenging behaviour**

If intramuscular agents are required for behavioural control, NICE recommends lorazepam, haloperidol or olanzapine.

If possible, a single agent should be used. If rapid tranquilisation is required, lorazepam and haloperidol should be used in combination. The patient should be monitored for dystonia and other extrapyramidal effects. Anticholinergic drugs may be used if side-effects become distressing, but monitor for deteriorating cognitive function.

Diazepam or chlorpromazine should be avoided.

**Prevention**[1]

VaD is modifiable and preventable. Modifying vascular risk factors in midlife may help to prevent stroke and VaD. The single most important modifiable risk factor in mid-life is hypertension, but the value of treating this is more debatable as age increases. [1] There is not yet any convincing evidence that the treatment of hypertension reduces the incidence of dementia, although it appears likely that this is the case in mid-life treatment. There is evidence that lowering high blood pressure after stroke decreases the risk of post-stroke dementia.
Complications

- Behavioural problems, including wandering, delusions, hallucinations, and poor judgement.
- Depression.
- Falls and gait abnormality.
- Aspiration pneumonia.
- Decubitus ulcers.
- Caregiver burden and stress: this should be considered a complication of any dementia, including vascular dementia. This can lead to increased psychiatric and medical morbidity in the caregiver.
Prognosis

The prognosis of VaD is thought to be worse than that of Alzheimer’s disease, carrying an average life expectancy of three to five years. [16]

Further reading & references


2.Binswanger's Disease; National Institute of Neurological Disorders and Stroke
5. Dementia UK, 2nd Edition - Overview; Alzheimers Society, September 2014
7. Dementia: Supporting people with dementia and their carers in health and social care; NICE Clinical Guideline (November 2006, last updated September 2016)
8. Dementia; NICE OCS, March 2010 (UK access only)
9. Dementia; NICE Quality Standard, June 2010
10. Dementia: independence and wellbeing; NICE Quality Standard, April 2013

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