# Diagnosis and management of vascular dementia

**Vascular dementia is one of the most common causes of dementia in the elderly population. Intervention in the early stages may help to prevent progression towards more severe cognitive impairment.**

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Dementia is a multifaceted decline in cognitive functioning that impairs daily living. Traditionally, memory impairment has been regarded as mandatory for diagnosis, along with demonstrated decline in one or more other cognitive domains (frontal executive function, language, praxis, gnosis or visuoconstructive function). However, the main cognitive feature of vascular dementia may be impaired organisation and judgement, rather than impaired memory. Vascular dementia is diagnosed if a patient has cerebrovascular disease that is judged to be causally relevant to the cognitive impairment.

## How common is vascular dementia?

Vascular dementia is considered to be the second most common type of dementia in Western societies; cross-national studies suggest that it is more common than Alzheimer's disease in Japan, China and Russia.[1] In a European epidemiological sample, estimates of the lifetime risk of vascular dementia have been made for men (34.5%) and women (19.4%).[2]

The incidence of vascular dementia in people who have been hospitalised after ischaemic stroke is of particular relevance to general practice. In two longitudinal European studies, dementia

**IN SUMMARY**

- **Stroke is a major cause of vascular dementia; therefore, risk factors for stroke are risk factors for vascular dementia.**
- **Vascular disease and Alzheimer's disease co-occur in a significant proportion of patients. Cerebrovascular disease is known to promote clinical expression of Alzheimer's disease.**
- **In general, cognitive impairments secondary to vascular dementia are more varied than those seen in Alzheimer's disease. Memory deficit may not be as marked in patients with vascular dementia.**
- **Impairment of frontal lobe executive function is more prominent in vascular dementia than in Alzheimer's disease, and is characterised by impaired organisation or judgement, and by behavioural changes. These frontal lobe deficits may significantly impair independent living before memory deficits are manifest.**
- **Control of hypertension is one of the most established interventions for reducing the risk of vascular dementia. However, it is crucial to also make lifestyle changes that lower blood pressure. Modification of other cardiovascular risk factors and antiplatelet therapy may also reduce the risk of progression.**
- **Management of psychiatric complications can greatly improve quality of life for patients and carers.**
- **Liaison with specialists and aged care assessment teams can augment the care provided by the GP.**

was diagnosed in 26.3% and 31.8% of patients after acute stroke.[3,4]

## Risk factors

Sociodemographic, cerebrovascular and cardiovascular risk factors for vascular dementia are listed in the Table.

### Stroke

Stroke is a major cause of vascular dementia; therefore, risk factors for stroke are risk factors for vascular dementia. Not all stroke patients develop dementia, but the majority develop a level of cognitive impairment that depends on the site of the lesion. Due to hemispheric specialisation, it is more common for stroke in the left hemisphere to produce a greater level of cognitive impairment.

Certain strategic areas of the brain are crucial (for example, the frontal subcortical white matter, thalamic regions, cortex and angular gyrus) – infarction in these areas may result in dementia.[3,4] Furthermore, vascular dementia may develop in the absence of macroscopic infarction, occurring instead as a result of silent or lacunar infarction or because of small vessel disease affecting the deep white matter.

### Hypertension

Hypertension is a major risk factor for vascular dementia by means of cerebrovascular disease

**Table. Risk factors for vascular dementia**

**Sociodemographic factors**

- Age (especially after 60 years)
- Race (higher rates in Asian and African-American populations)
- Male sex
- Lower level of education

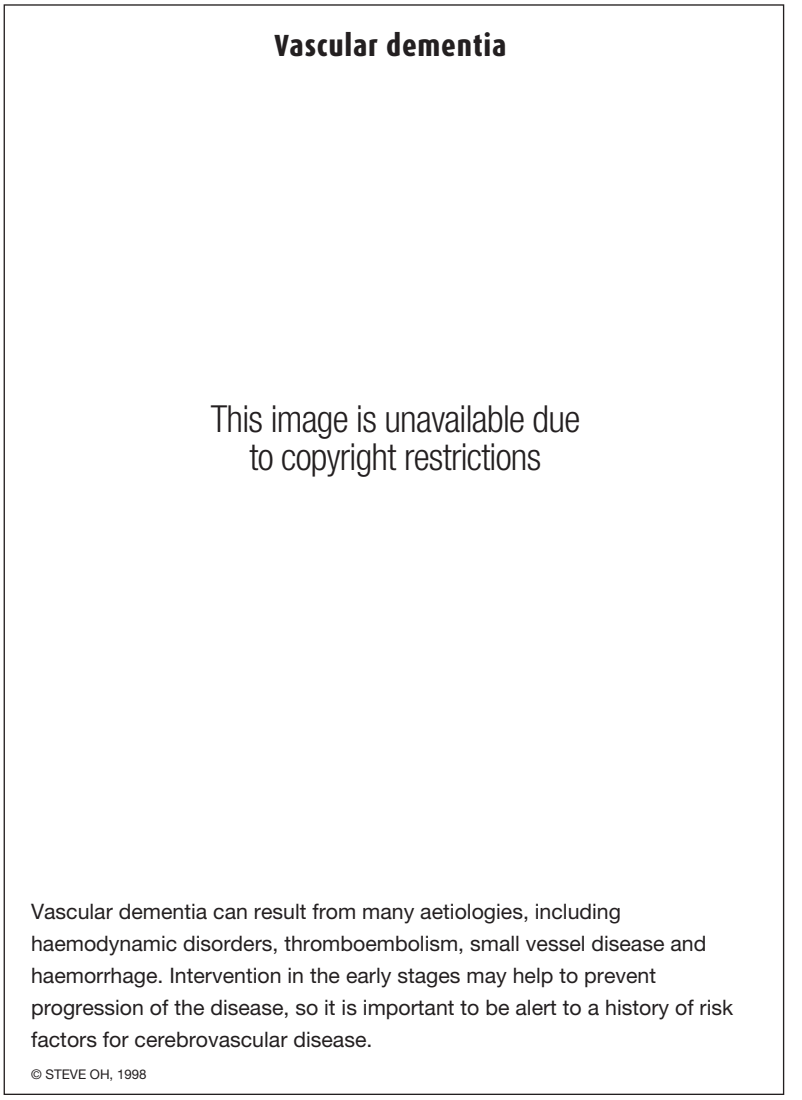
**Cerebrovascular disease**

- Stroke (number, volume and location)
- Pre-existent atrophy
- Periventricular white matter changes on CT or MRI
- Strategic single infarction
- Genetic factors:
  – cerebral autosomal dominant arteriopathy with subcortical infarction and leucoencephalopathy (rare)
  – autosomal dominant hereditary cerebral haemorrhage with amyloidosis of the Dutch type (rare)
  – apolipoprotein E and homocysteine (possible risk factors)

**Atherogenic and other cardiovascular factors**

- Hypertension (a major risk factor)
- Diabetes mellitus
- Cigarette smoking
- Hypercholesterolaemia
- Atrial fibrillation (risk of cerebral embolism)
- Mitral valve prolapse (risk of cerebral embolism)

**Vascular dementia**




Vascular dementia can result from many aetiologies, including haemodynamic disorders, thromboembolism, small vessel disease and haemorrhage. Intervention in the early stages may help to prevent progression of the disease, so it is important to be alert to a history of risk factors for cerebrovascular disease.

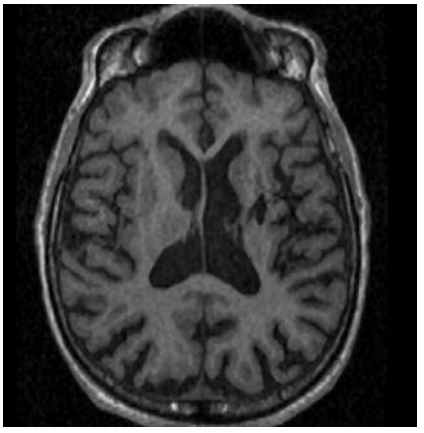


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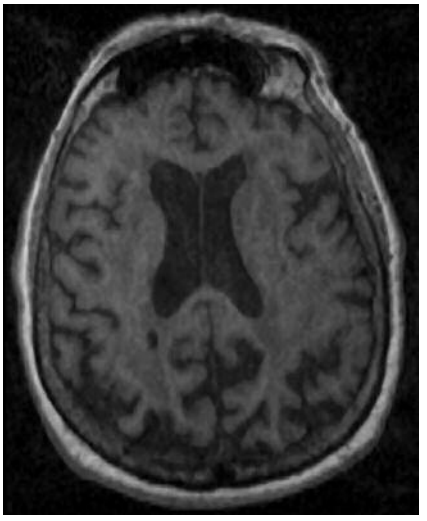
Figure 1. T1-weighted MRI showing a macroscopic infarction in the internal capsule adjacent to the left lateral ventricle.

(stroke- and non-stroke-related). Antihypertensive drug treatment may reduce the incidence of dementia.[5] Relative hypotension in a patient who was previously hypertensive may contribute to vascular cognitive impairment by hypoperfusion.[4]

### Genetic factors

The role of genetic factors for cerebrovascular disease and, consequently, vascular dementia is controversial. The evidence remains inconclusive at this stage, but recent investigations are focusing on apolipoprotein E and homocysteine.



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## Reviewer's comment

This is an important article because vascular dementia is perhaps the only form of dementia for which there is some inferential evidence, based on stroke recurrence prophylaxis studies, for which there is potential for halting or at least slowing the progression of this devastating disease.

With current demographic projections of the increased individual longevity and ageing of the Australian population, at least through to 2030, the need to address this treatable form of dementia is imperative. Dementia is predicted to increase in incidence and prevalence faster than the growth in the aged population. It is hoped that by aggressive delineation of the cause(s) of dementia in the individual patient that some downward trend in the appalling predictions for the first half of the 21st century can be made. It is most appropriate that attention be once again drawn to this form of dementia which previously was regarded in the 1960s as the principal cause of dementia before Alzheimer's disease emerged as the leading cause and the vascular aetiopathogenesis was somewhat neglected.

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## What causes vascular dementia?

The vascular lesions underlying vascular dementia may be produced through ischaemia or haemorrhage (Figure 1). White matter lesions secondary to small vessel ischaemia (indicated by hypodensities on CT or hyperintensities on T2-weighted MRI) may contribute to ischaemic subcortical dementia (Figures 2a and b).

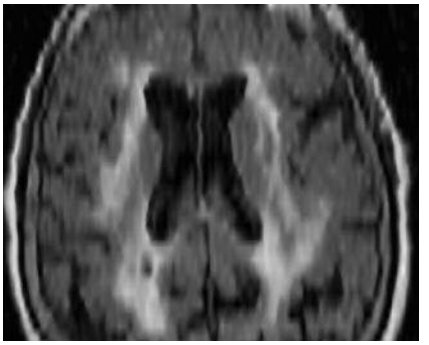

Figure 2a (left). Extensive, deep, white matter disease shown by T1-weighted MRI. Figure 2b (above). The white matter changes shown in Figure 2a are highlighted in a FLAIR MRI sequence.

Vascular dementia may therefore result from single or multiple causes. The main causes are:

- haemodynamic disorders – thrombotic infarction (arterial, watershed or lacunar) and venous, arteriopathic or genetic disorders
- thromboembolism – cardiac, carotid or arterial emboli
- small vessel disease – subcortical white matter lesions
- haemorrhage – subdural, subarachnoid or intracerebral haemorrhage.

In a significant proportion of patients, the pathological changes of vascular dementia and Alzheimer's dementia co-occur.[6] Cerebrovascular disease is also known to promote clinical expression of Alzheimer's dementia.[7]

## Clinical features and diagnosis

Vascular dementia should be evaluated appropriately, with the clinician alert to a history of risk factors for cerebrovascular disease. The clinical assessment of a patient with possible vascular dementia is outlined in the box on page 20.

## Vascular dementia or Alzheimer's disease?

### Types of onset

The onset of vascular dementia is variable in presentation, including sudden stroke, gradually progressive onset with some degree of fluctuation, and stepwise deterioration. Dementias that are particularly associated with large infarction follow the traditional pattern of sudden onset, with or without stepwise deterioration. However, a subgroup of people with predominantly small subcortical lesions have gradual onset of cognitive impairment and slow progression that may be difficult to distinguish from Alzheimer's disease.

### Key differences in cognitive deficits

Clinical features that may help to distinguish vascular dementia from Alzheimer's disease are:

- relative preservation of personality until the later stages of the disease
- depression
- emotional lability[8]
- somatic symptoms
- impaired judgement and organisation.[9]

In general, cognitive impairments secondary to vascular dementia are more varied than those seen in Alzheimer's disease. A particular distinguishing factor is that memory deficit may not be as marked, and there may be a discrepancy in performance between verbal and nonverbal memory (verbal memory being relatively preserved in vascular dementia).[9]

Impairment of frontal lobe or executive function is more prominent in vascular dementia than in Alzheimer's disease, and may be evident as difficulties with planning and organising, mental flexibility, behavioural dysregulation and emotional lability.[9] These frontal lobe deficits may markedly impair judgement and insight in the relative absence of memory deficits, resulting in significant impairment in activities of daily living.

Other associated features of vascular dementia include visuospatial dysfunction, dysphasia and cognitive slowing. As a consequence of language impairment, assessment may be complicated in a patient with left hemispheric stroke. A significant proportion of patients have mixed symptoms of Alzheimer's disease and vascular dementia and, therefore, mixed patterns of cognitive impairment.

## Other dementias

Other dementias may figure in the differential diagnosis. These include:

- other subcortical dementias (with characteristic neurological signs), such as Parkinson's disease or

Huntington's disease
- Lewy body dementia (recurrent episodic confusion, hallucinations and spontaneous parkinsonism)
- frontotemporal dementia (prominent frontal lobe and language dysfunction).

## Reducing the risk of vascular dementia

The control of risk factors is the mainstay of specific management for vascular dementia, representing an opportunity to reduce the incidence of vascular dementia, halt the progression of the disease, and achieve partial amelioration of symptoms. Strategies for preventing vascular dementia are outlined in the box on page 23.

### Controlling hypertension

The control of hypertension is one of

## Clinical assessment of the patient with vascular dementia

The clinical assessment of a patient with possible vascular dementia includes a history and examination to document the evidence for cerebrovascular disease. Functional status and resultant disability should also be evaluated, and investigations may be required.

### History

Document the onset, course and nature of cognitive deficits. Include information from the carer on possible subtle personality or behavioural changes.

### Examination

#### Cognitive examination

Screen for cognitive impairment. The Folstein Mini Mental State Examination may be useful, and can be supplemented by clock drawing and clinical assessments of frontal lobe functioning, such as verbal fluency, design fluency, and alternating shape drawing.

If available, a full neuropsychological evaluation should be conducted by a neuropsychologist.

#### Functional assessment

Ask the carer about levels of function. The GP's assessment may be augmented by a home assessment by an occupational therapist, if available.

#### Psychiatric evaluation

Perform a psychiatric evaluation. Depressive disorder is common, and anxiety and psychotic disorders may be present. Remember that behavioural disturbances (such as disinhibition, aggression and wandering) may occur.

#### Physical examination

In the general examination, focus on cardiovascular status and evidence of hypertension. Assess pulse irregularity and the presence of bruit. An examination of the fundi and measurement of blood pressure should also be conducted.

Look for focal neurological symptoms and signs that suggest a possible cerebrovascular aetiology for cognitive impairment, including:
- gait abnormalities
- pseudobulbar palsy (dysarthria, dysphagia, spastic tongue, brisk hyperreflexia and plantar extensor responses)
- brainstem abnormalities (diplopia, nystagmus, vertigo, ataxia and cranial nerve palsies)
- sensorimotor symptoms
- visual disturbance or field deficits
- hemiparesis
- extrapyramidal signs (rigidity, dyskinesia and tremor).

### Investigations

Routine investigations include:
- a full blood count
- erythrocyte sedimentation rate (ESR)
- blood glucose
- cholesterol and triglyceride levels
- vitamin $B_{12}$
- folate
- thyroid stimulating hormone (TSH)
- ECG
- chest x-ray.

#### Brain imaging

Structural brain imaging (CT or MRI) provides information on the extent, type and distribution of possible vascular lesions, and can exclude other causes of dementia.

The absence of vascular lesions on CT and MRI would militate against a possible vascular aetiology. If a rare cause is suspected, consultation with a geriatrician, neuropsychiatrist or neurologist may be helpful.

#### Specialised investigations

Specialised investigations that can be arranged on the advice of a specialist and are relevant to the aetiology of stroke or cerebrovascular disease include echocardiography and carotid Doppler ultrasound.

Rarer causes, such as autoimmune disease causes or vasculitides, may be investigated with antibody testing and serum protein electrophoresis.

the most established interventions for reducing the risk of stroke and, hence, vascular dementia. Patients who have a diastolic blood pressure greater than 110 mmHg are treated, but there is evidence that patients with a diastolic blood pressure between 90 and 100 mmHg and a systolic blood pressure greater than 160 mmHg may also benefit from treatment.[10]

It is crucial to also make lifestyle changes that can lower blood pressure. Modifiable lifestyle factors include:

- cigarette smoking
- excessive alcohol consumption
- obesity
- lack of exercise.

If possible, hypotension should be avoided because failure of blood flow autoregulation in patients with vascular dementia may increase the detrimental effect of cerebral hypoperfusion.

### Managing other cardiovascular risk factors

Modification of other cardiovascular risk factors such as atrial fibrillation and carotid arterial stenosis is important in the prevention of stroke and, potentially, vascular dementia. For those with atrial fibrillation, anticoagulant therapy is beneficial in reducing the risk of stroke – warfarin (Coumadin, Marevan) is recommended for these patients. In symptomatic, severe carotid artery stenosis (that is, a greater than 70% occlusion), carotid endarterectomy has been successful.

Antiplatelet therapy may reduce the risk of further events only in patients with a history of coronary heart disease, previous TIA or nonhaemorrhagic stroke. The dosage of aspirin recommended for a patient in this group is between 75 and 325 mg per day (commonly available formulations in Australia range between 100 and 300 mg).[11] If aspirin therapy is unsuccessful, an antiplatelet agent may be indicated, such as clopidogrel (Iscover, Plavix), ticlopidine (Ticlid, Ticlopidine Hexal, Tilodene) or dipyridamole (Persantin); in addition, a combination therapy of aspirin and dipyridamole (Asasantin) is available and may be used. Antiplatelet treatment may be planned in consultation with a geriatrician or neurologist.

There is less evidence that control of hyperlipidaemia and diabetes mellitus is efficacious, but appropriate management is warranted in any case. Diabetes mellitus itself may result in cognitive impairment as a result of impaired glucose metabolism.[12]

## Managing psychiatric complications

After diagnosis, identifying and treating psychiatric complications of cerebrovascular disease (such as depression) will be necessary. Care can be planned in consultation with a specialist in geriatric psychiatry.

Depression associated with vascular dementia usually responds to treatment with selective serotonin reuptake inhibitors (SSRIs) or other antidepressants, but SSRIs may be better tolerated in an elderly patient. Depression needs to be distinguished from apathy secondary to a stroke. Atypical antipsychotics such as olanzapine (Zyprexa) or risperidone (Risperdal) may reduce the burden of side effects of psychosis related to dementia, but their use in vascular dementia should be negotiated with the PBS. Behavioural disturbances may occur, such as disinhibition, aggression and wandering.

## Practical and legal issues

The principles of general treatment are prevention and support (see the box on page 24). After assessment and diagnosis, the patient and carer will need to be counselled about the nature of the dementia. Convening a family interview is advisable to educate the family about the issues involved in the patient's care and to assess the need for supports.

### Strategies for preventing progression of vascular dementia

#### Control of hypertension

- Treat hypertension
- Counsel patient to make lifestyle changes that lower blood pressure (such as ceasing smoking and reducing alcohol intake)
- Recommend lifestyle changes (for example, weight loss, exercise, stress reduction and reduced salt intake)

#### Management of other cardiovascular risk factors

- Introduce anticoagulant therapy in patients with atrial fibrillation
- Consider carotid endarterectomy for severe symptomatic carotid stenosis (more than 70% occlusion)
- Introduce antiplatelet therapy for high risk patients with a history of nonhaemorrhagic stroke, TIA or coronary heart disease
- Control hyperlipidaemia
- Treat diabetes
- Introduce dietary control for diabetes, obesity and hyperlipidaemia

#### Stroke-related interventions

- Implement early interventions for stroke and TIA with neuroprotective agents
- Implement intensive rehabilitation after stroke, including a neuropsychological assessment and advice on managing deficits

### Legal planning

Legal planning for power of attorney and guardianship is an important area of management. These issues are relevant given possible frontal lobe dysfunction (impaired judgement) secondary to stroke

## General management of patients with vascular dementia

- Counsel the patient and carer about the nature of the dementia
- Educate and involve the family through a family interview
- Provide information about support agencies such as the National Stroke Foundation and the Alzheimer's Association
- Consider the use of vitamin E or C supplements
- Treat psychiatric complications of vascular dementia
- Treat physical complications of vascular dementia related to stroke (which involves physical rehabilitation, provision of aids and additional home supports)
- Address legal and financial issues
- Assess fitness to drive or work (particularly an issue for younger patients)

or microvascular disease, and are best addressed in the early stages when the patient is able to give informed consent.

### Financial issues

Finance and work also need to be addressed. Financial support may be sought if the patient has had to stop work; capacity and ability to continue working may need to be assessed in conjunction with the deficits.

### Driving ability

In the interest of public health, the issue of driving should be assessed carefully, especially if the patient has concomitant possible physical stroke complications, such as visuospatial deficits. Mandatory reporting may require the practitioner to report to the appropriate licensing body; otherwise, it is reasonable to suggest that the patient take a test for fitness to drive.

### Accommodation

The issue of respite care or placement in hostel or nursing home accommodation should be considered for the longer term.

### Other issues

The aim of dementia management will be to preserve quality of life as much as possible for both the patient and the family, and other issues will arise in the early, middle and late stages of the disease.

A recent article by Professor Brodaty provides a comprehensive overview of the principles of dementia management.[13] (The article discusses management of patients with Alzheimer's disease, but most of the principles described apply equally to vascular dementia.)

## Further help and information

GPs should avail themselves of aged care assessment teams based at public hospitals and community health centres. These teams provide links to neuropsychological assessment, home help, food services, allied health services, community nursing, transport and accommodation options. Further education for the patient, family and carer can be provided by support agencies such as the Alzheimer's Association (toll-free HelpLine, phone 1800 639 331) or the National Stroke Foundation (freecall 1800 657 007).

## Developments in treatment

Specific drug treatments for vascular dementia have been investigated, and no drug can be recommended at this stage. Some drugs that improve cognitive function in Alzheimer's dementia may be potentially useful, such as the cholinergic agents rivastigmine (Exelon) and donepezil (Aricept); trials of donepezil are in progress for vascular dementia.

Vitamins E and C have demonstrated a significant protective effect against vascular dementia in a high risk Hawaiian group.[14] Potentially neuroprotective drugs (such as propentofylline, calcium channel antagonists and *N*-methyl-D-aspartate receptor antagonists) may also have a role in reducing damage in acute stroke, and are undergoing preliminary investigation.[15] These agents may reduce the long term development of cognitive impairment.

Future developments may include individualised neuropsychological rehabilitation to assist patients with vascular dementia and their families.

## Prognosis

Longitudinal studies of vascular dementia suggest that mortality rates are greater than those for Alzheimer's disease. Overall, rates of admissions into nursing homes are comparable for the two disorders.[16]

## Concluding remarks

The GP's role in assessing and managing a patient with vascular dementia includes:

- establishing the diagnosis
- documenting the evidence for cerebrovascular disease
- determining the aetiological role of cerebrovascular disease
- evaluating functional status and resultant disability
- assessing interpersonal community supports, and
- determining modifiable risk or protective factors.

Also, GPs can perform a key role in the prevention of secondary complications and in ongoing patient management. MT

## Acknowledgements

The preparation of this paper was supported in part by an NHMRC project grant. Thanks to P. Lynch, D. Gorman and V. Earnshaw.

*A list of references is available on request to the editorial office.*

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MEDICINE TODAY November 2000 pp. 16-27

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