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Timely diagnosis of vascular dementia key to management

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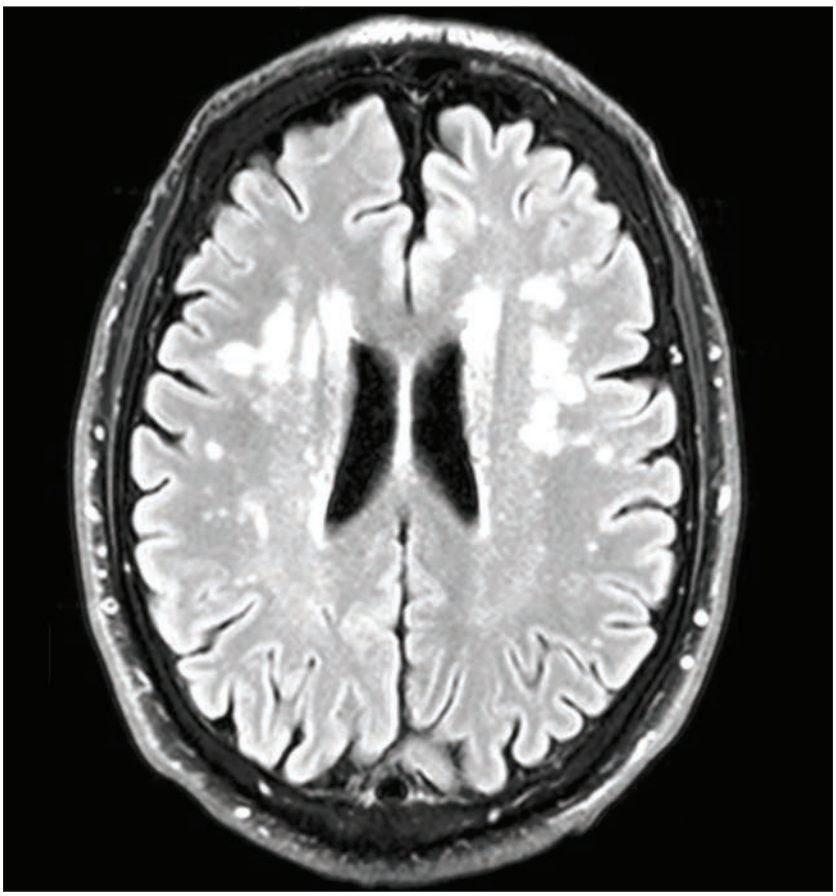

What are the risk factors for vascular dementia?

How should patients be assessed?

What are the management approaches?

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

VASCULAR DEMENTIA IS THE SECOND MOST COMMON TYPE OF DEMENTIA, AFTER Alzheimer's disease, and accounts for 15% of dementia cases.[1]

Several sets of diagnostic criteria have been developed, and this reflects the heterogeneous nature of the condition in terms of its clinical manifestation and severity of symptoms.[2]

The core diagnostic features include cognitive impairment in at least two domains (orientation, attention, language, visuospatial function, executive function, motor control and praxis), which affect social or occupational function, together with evidence of cerebrovascular disease (focal neurological signs or neuroimaging), see table 1, p13. Crucially there should be a temporal relationship between cerebrovascular disease and the onset of cognitive changes.[2,3] Vascular dementia typically affects executive function more than it affects memory.

RISK FACTORS

Vascular dementia occurs when damage to cerebral blood vessels is great enough to cause cognitive problems. Therefore, physical health conditions affecting the cardiovascular system, or those which may cause strokes, are risk factors for vascular dementia. These can broadly be divided into modifiable and nonmodifiable.

Demographic nonmodifiable risk factors are older age, male gender, and ethnicity (there is a higher rate of stroke in Asian populations). Genetic risk factors include a family history of cardiovascular or cerebrovascular disease and rarer conditions, such as CADASIL (cerebral autosomal dominant arteriopathy with subcortical infarcts and leukoencephalopathy).

A further nonmodifiable risk factor is previous stroke, with an estimated one in three stroke patients at risk of developing vascular dementia within five years.[4]

Modifiable risk factors are mainly arteriosclerotic such as hypertension, type 2 diabetes, atrial fibrillation and hypercholesterolaemia. Related to these are lifestyle factors such as smoking, excess alcohol intake, obesity, a high-fat diet and low physical activity levels.

Hypertension has been the most extensively studied modifiable risk factor. Chronic hypertension particularly in midlife has been associated with executive dysfunction, slowing of mental processing speed, and memory; it has been shown to increase the risk of vascular dementia.[5] Findings regarding late-life hypertension remain inconclusive.[5] »

Table 1

Summary of diagnostic criteria for vascular dementia

1 Cognitive impairment in at least two domains e.g. aphasia, apraxia, agnosia and executive dysfunction
2 Cognitive changes cause significant impairment in social or occupational function
3 Evidence of a temporal relationship between the clinical symptoms and cerebrovascular disease e.g. focal neurological signs and symptoms, neuroimaging changes
4 No evidence of delirium

UNDERLYING MECHANISMS

The pathology and mechanisms underlying vascular dementia are yet to be fully understood. Clinical signs and symptoms may vary depending on the cause and type of the vascular dementia, as well as site and size of the infarction or damage.[6]

Onset may occur following a single (major) stroke, a series of multiple small strokes (multi-infarct dementia) including silent strokes, small vessel disease, CADASIL, and also in combination with other dementias, particularly Alzheimer's disease, where the vascular component may exacerbate the symptoms.[7]

Vascular dementia occurs when chronic hypoperfusion, hypoxia and oxidative stress trigger inflammatory responses and an ischaemic cascade. The periventricular white matter, basal ganglia, and hippocampi are highly susceptible to hypoperfusion-induced lesions. If the resulting damage is significant and leads to disruption of the prefrontal-basal ganglia circuitry, this may induce cognitive deficits.[6]

PRESENTATION

Presentation in primary care may be very varied, given the range of pathophysiological processes (e.g. haemorrhages in single or multiple areas of the brain) that are relevant to the aetiology. The most common cognitive symptoms are changes in language (particularly nominal aphasia), executive function (planning) and visuospatial skills. Vascular dementia should be suspected if there is a decline in at least two cognitive domains over at least six months, particularly if there is a characteristic stepwise progression in symptoms.

There may be associated clinical symptoms such as: early gait disturbance, history of unsteadiness or falls, urinary frequency or urgency, pseudobulbar palsy, personality change and mood changes.[3,8]

Cognitive changes may be detected incidentally following a review of vascular disease or risk factors e.g. atrial fibrillation, hypertension, diabetes mellitus and hypercholesterolaemia. Patients may have multiple cardiovascular risk factors, including previous transient ischaemic attacks (TIAs) or strokes and, on examination, may have focal neurological signs.

It is worth noting that the presence of vascular risk factors increases the risk of vascular dementia, mild cognitive impairment,[9] and Alzheimer's disease.[10] Given the significant overlap in these conditions, the presence of multiple vascular risk factors should prompt clinicians to review cognition, even if the presentation is not typical for vascular dementia.

'Vascular risk factors increase the risk of vascular dementia, Alzheimer's disease and mild cognitive impairment'

ASSESSMENT

A thorough clinical history and physical examination, together with collateral history is vital. Any pertinent areas of concern, particularly in relation to driving, home safety or finances, need to be identified. If there are significant concerns, this will help guide onward referrals.

Brief cognitive screening should be performed using a validated tool that rapidly assesses multiple cognitive domains. Screening tools form only part of the cognitive assessment process, and should be supplemented by history taking and further investigations.

The General Practitioner assessment of Cognition (GPCOG), Mini Mental State Examination (MMSE) and Montreal Cognitive Assessment (MoCA)[11,12] are all good screening tools. The GPCOG has a positive predictive value of 71.4% and a misclassification rate of 14%.[13] The MoCA has been developed particularly with stroke populations in mind and covers more domains of cognition, so is therefore preferable.

Cognitive impairment can be caused, or exacerbated, by depression or anxiety. A brief review of mental health is therefore important; screening for depression and anxiety can be supported by use of common tools such as the PHQ9 or GAD7.[14] If these indicate significant symptoms, then patients should be treated according to standard guidelines.[15]

It is important to rule out acute or reversible causes of cognitive impairment with simple investigations. Routine blood tests including a full blood count, thyroid function, B_{12}, folate and calcium levels along with a midstream urine test can help identify reversible causes of cognitive decline (e.g. delirium, hypothyroidism and vitamin deficiencies).

A brief medication review is vital to ensure that cognitive decline is not iatrogenic, see table 2, p13. Common medications known to affect cognition include: anticholinergics (e.g. oxybutynin and amitriptyline), antiemetics (e.g. cyclizine and metoclopramide) and opiates; alternatives should be sought where possible.[16]

Neuroimaging can help distinguish the type of dementia and exclude other cerebral pathologies. A CT head is generally the most cost effective, well tolerated and easily accessible scan, though not all GPs are able to obtain this, and it is more commonly requested in secondary care. CT head scans are primarily used to exclude a treatable cause for dementia (e.g. hydrocephalus) and may also provide supportive evidence for a diagnosis of vascular dementia (e.g. old stroke, or white matter changes which are evidence of small vessel ischaemia).

REFERRAL

Patients may require referral for assessment, management of complexity in vascular dementia, or palliative care.[17]

In general, there should be a low threshold for referral to a memory clinic for assessment for possible dementia. GPs should consider a referral if patients, their families, or other professionals have raised concerns, particularly if these are having an adverse impact on the patient's ability to live independently.

It is important that GPs seek the

patient's consent prior to referral, and that relevant background information in relation to their physical and mental health is included, see table 3, below.

There are circumstances when a referral to a memory clinic may not be appropriate, and other services may be better placed to meet the patient's needs. For patients with comorbid depression, anxiety or alcohol dependence, treatment of these conditions should be the priority, before a more detailed memory assessment. If there is a physical explanation for cognitive changes (e.g. infection in the context of delirium) or if there are new neurological signs then a referral to neurology or another medical specialty may be needed. Patients with learning disability or a history of head injury may be better served by a referral to specialist learning disability/head injury services.

As vascular dementia progresses, many patients will develop noncognitive and behavioural symptoms such as anxiety, psychosis, and agitation.[18] If these symptoms cause concern then a referral (back) to specialist services should be made to assist with management. If there are significant risk issues, then a referral to social services may also be needed.

If there is concern that the patient is severely disturbed and could be an immediate danger then an urgent referral to mental health services may be needed, with a view to possible inpatient admission.

Finally, GPs should ensure that patients with dementia and their families have access to palliative care services, for example to discuss artificial feeding and cardiopulmonary resuscitation. This alone may be a reason for referral to old age psychiatry.

MANAGEMENT

A timely diagnosis of vascular dementia provides the opportunity to assess and treat comorbidities, provide information and support, and help to maintain an individual's independence.[1] NICE recommends that people with mild to moderate cognitive symptoms should be offered a place on a group cognitive stimulation programme, although the current evidence base for cognitive interventions is poor.[17,19,20] There are currently no nationally recommended pharmacological treatments to improve cognitive function in vascular dementia.[17] Small improvements have been noted using the acetylcholinesterase inhibitor galantamine[21,22] and the calcium channel blocker nimodipine,[23] however neither improved functioning. A randomised placebo-controlled trial of amlodipine for vascular dementia has recently completed recruitment.[24]

For the management of noncognitive and behavioural symptoms, NICE recommends an individual care plan and trials of nonpharmacological interventions such as cognitive stimulation therapy,[25] structured exercise[26] and animal-assisted therapy,[27] although the evidence base is weak. Patients should be offered treatment for comorbid anxiety or depression, which are more common in people with dementia. If patients become severely distressed or a risk to themselves or others, they should be reassessed to exclude any new physical symptoms (e.g. infection, pain, constipation). Sometimes a short course of antipsychotic medication may be required if the risk of such behaviour is high. While atypical antipsychotics, such as risperidone and olanzapine, reduce aggression and psychotic symptoms in dementia, these should be prescribed cautiously following a discussion about the risks and benefits including the increased risk of cerebrovascular events and mortality.[28] Initial doses should be low and the need for medication reviewed regularly.

PREVENTION

Vascular dementia is both modifiable and preventable. Diagnosing and treating comorbidities and risk is the mainstay of prevention and treatment strategies. This should focus on modification of risk factors such as: obesity, smoking, cardiovascular disease, hypertension, atrial fibrillation, hypercholesterolaemia, diabetes (type 1 or 2) and chronic kidney disease. Modifying risk factors in midlife will help »

Table 2

Common medications known to impair cognition

Condition	Drug class	Drugs to avoid in cognitive impairment	Recommended alternatives
Urinary frequency	Anticholinergics	Oxybutynin Tolterodine	Darifenacin Trospium Solifenacin
Nausea/vomiting	Antiemetics	Cyclizine Metoclopramide Prochlorperazine	Domperidone Serotonin 5-HT3 antagonists
Irritable bowel	Antispasmodics	Atropine sulphate	Mebeverine
Pain	Analgesics	Codeine Tramadol Fentanyl patches	Paracetamol Topical NSAIDs Buprenorphine
Allergy	Antihistamines	Chlorpheniramine	Cetirizine

Table 3

Information to be included when referring patients to a memory clinic

- Patient's consent to the referral
- Named carer and consent to contact the carer if available
- Any special communication needs e.g. interpreter, sign language
- A short description of the history and impact on functioning of the presenting memory problem
- A full physical screening
- Blood and urine tests as per dementia protocol
- Mental health check: screen for depression and anxiety, and consider using an appropriate measure (e.g. PHQ9, GAD7)
- Cognitive screen (e.g. MMSE or GPCOG)
- Physical and mental health history and current medication
- Any known risk factors, for example relating to driving

key points

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Vascular dementia is the second most common type of dementia, after Alzheimer's disease, and accounts for 15% of cases. The core diagnostic features include cognitive impairment in at least two domains (orientation, attention, language, visuospatial function, executive function, motor control and praxis), which affect social or occupational function, together with evidence of cerebrovascular disease (focal neurological signs or neuroimaging). Crucially there should be a temporal relationship between cerebrovascular disease and the onset of cognitive changes.

Onset may occur following a single (major) stroke, a series of multiple small strokes (multi-infarct dementia) including silent strokes, small vessel disease, CADASIL, and also in combination with other dementias, particularly Alzheimer's disease, where the vascular component may exacerbate the symptoms.

Presentation may be very varied. The most common cognitive symptoms are changes in language (particularly nominal aphasia), executive function (planning) and visuospatial skills. Vascular dementia should be suspected if there is a decline in at least two cognitive domains over at least six months, particularly if there is a characteristic stepwise progression in symptoms.

A thorough clinical history and physical examination, together with collateral history is vital. Clinical cognitive assessment should include the use of a validated tool which rapidly assesses multiple cognitive domains. The MoCA is preferable as it has been developed particularly with stroke populations in mind and covers more domains of cognition.

In general, there should be a low threshold for referral to memory clinics for assessment for possible dementia. GPs should consider a referral if patients, their families, or other professionals have raised concerns, particularly if these are having an adverse impact on the patient's ability to live independently. As vascular dementia progresses, many patients will develop noncognitive and behavioural symptoms such as anxiety, psychosis, and agitation. If these symptoms cause concern then a referral (back) to specialist services should be made to assist with management.

There are currently no nationally recommended pharmacological treatments to improve cognitive function in vascular dementia. Patients should be offered treatment for comorbid anxiety or depression. If patients become severely distressed or a risk to themselves or others, they should be re-assessed to exclude any new physical symptoms (e.g. infection, pain, constipation). Diagnosing and treating comorbidities and risk factors is the mainstay of prevention and treatment strategies. Modifying cardiovascular risk factors in midlife will help reduce the risk of developing vascular dementia, and may slow progression of vascular dementia.[9]

CONCLUSION

Vascular dementia is a common cause of dementia, yet still lacks effective, evidence-based treatments. Intervention should focus on the modification of vascular risk factors (hypertension, diabetes, obesity, smoking cessation and exercise) to reduce the incidence and progression of vascular cognitive impairment.[29]

'Vascular dementia is both modifiable and preventable'

Although the number of pharmacological treatments is limited, referrals for further assessment of cognition can yield significant benefits for patients and their families, and should be actively considered. Assessment can aid understanding about the presence of any significant cognitive concerns, and enables access to other support and services which can greatly improve quality of life, and promote independence.

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Useful information

The following organisations provide information about all types of dementia, including vascular dementia:

Age UK
www.ageuk.org.uk

Alzheimer's Society
www.alzheimers.org.uk

Alzheimer's Research UK
www.alzheimersresearchuk.org

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