Chronic stroke disease

Despite a welcome decline in stroke-related mortality witnessed in recent decades, stroke remains the second leading cause of mortality worldwide, as well as a major cause of disability, as shown by Feigin et al (2014). Stroke disease occurs on a spectrum from initial presentation with acute stroke to progression to chronic stroke disease. However, it is generally not viewed in such terms, with the focus on acute and hyperacute stroke care often overshadowing that of chronic stroke. Even the traditional definition of stroke as an 'acute neurological event of presumed vascular origin lasting longer than 24 hours' does not accommodate chronic stroke disease. It is not uncommon for major international stroke conferences to overlook chronic stroke disease to such an extent that it is not represented in any form during the conference.

The last 20 years have seen a complete transformation in acute stroke care, with the establishment of stroke units, as well as the widespread introduction of intravenous thrombolysis, and telemedicine to provide 24-hour acute stroke assessment where it would otherwise have been inaccessible. This focus on acute care was welcome, as management in the acute period after a stroke had been underdeveloped until then; however, it must be remembered that interventions such as intravenous thrombolysis are only applicable to 10% of patients in clinical practice and most patients who are thrombolysed will still have some degree of residual disability. Unfortunately these improvements have not been mirrored by similar advances in the management, or even recognition of, chronic stroke or cerebrovascular disease.

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Stroke as a chronic disease

Stroke should be viewed as a chronic disease with acute exacerbations, somewhat analogous to chronic obstructive pulmonary disease or cardiac failure. The manifestations of chronic stroke disease include cognitive deficits, dysphagia and gait disorders rather than breathlessness or cough, while the 'acute exacerbations' can take the form of decompensation in swallow or gait, or a delirium, all of which are more likely to occur in the setting of concurrent illness. As with all chronic disease, the risk of further acute stroke is much higher in patients with chronic stroke disease, with the risk of recurrent stroke around six times greater than the risk of first ever stroke in a general population of the same age and sex, in a study by Hardie et al (2004).

Again much like chronic respiratory or cardiac illness, chronic stroke disease confers significant disability and morbidity. For example, in a study by Cowman et al (2004), one in six nursing home residents in a national survey had previously had a stroke, with over 70% of these residents deemed to have a high level of disability. Despite this, the impact of chronic stroke disease is invariably not captured when the global burden of stroke is assessed. This is a pity as it is difficult to promote improved chronic stroke care if the full burden of chronic stroke disease is not known.

While this chronic disease analogy can be useful, chronic stroke can be complex and nuanced, and experience is often required, for example to separate what represents a further stroke from a decompensated swallow. Also it must be remembered that one does not have to present with an overt acute stroke in order to develop manifestations of chronic stroke disease. Indeed, it is quite common for patients with apparently occult stroke to subsequently present with symptoms such as gait problems and especially memory impairment. Various different terms have been used to describe this including 'silent strokes' or 'strategic infarction'.

The most common manifestations of chronic stroke disease include swallow impairment, gait disorders, depression and cognitive deficits. A combination of some or all of these features can occur in a single person. It is important therefore that when one of these features is present that the physician screens for the others. This is especially vital when we consider that symptoms such as mobility or swallow problems can occur so gradually, and can be compensated for by the patient to such an extent that they are often not mentioned as a symptom in the same way one would report breathlessness or chest pain for example. Stravinsky syndrome, described by O'Neill et al (2014), named after the composer Igor Stravinsky, comprises gait dyspraxia, dysphagia and cognitive impairment, all of which it appeared Stravinsky had endured after an initial stroke in 1956. It is hoped that promoting an eponymous syndrome such as this may highlight the relative lack of diagnostic curiosity among the medical community as to the cause of these syndromes.

Chronic stroke disease can be caused by either large vessel pathology, for example following a middle cerebral artery syndrome, or by small vessel pathology, often termed subcortical ischaemia. The features can differ based on these different pathological processes with large vessel disease often characterized by chronic dense motor deficits, dysphasia or homonomous hemianopia while small vessel disease can lead to vascular gait dyspraxia, depression, cognitive impairment and swallow disorders. Of course, the risk factors for these two processes are similar and it is not uncommon to find a person with chronic stroke disease displaying both 'large vessel' and 'small vessel' sequelae. It must also be noted that features such as hemiparesis can be caused both by a large vessel and lacunar syndrome.

Cognitive deficits

The classical course of vascular dementia, the commonest subtype seen in chronic stroke disease, is that of cognitive deficits acquired in a step-wise fashion, linked to recurrent vascular events. These vascular events occur most commonly in the frontobasal systems involved in judgment, planning and emotion and vascular cognitive impairment causes deficits in these areas.

The overall prevalence of post-stroke dementia in patients after overt stroke is about 30% and Leys et al (2005) found that the incidence of new onset dementia increases from 7% after 1 year to 48% after 25 years. While most of this is vascular dementia, there is also an increased rate of developing Alzheimer's disease, suggesting that the aetiology of post-stroke dementia may be multifactorial and not related to vascular lesions alone.

Cerebral amyloid angiopathy contributes to subcortical disease burden and can co-exist with Alzheimer disease and vascular dementia. It increases the risk of spontaneous lobar intracerebral haemorrhage and is associated with cognitive decline, although the exact mechanism for this remains unclear.

Gait disorders

Gait disorders are also a common manifestation of stroke disease. They are a reliable predictor of increased falls risk and confer a greater likelihood of nursing home admission. While the hemiparetic gait, characterized by the leg swinging outwards in a semi-circle from the hip, with a hyper-extended knee and inverted, plantar-flexed ankle, is the classical gait disorder associated with stroke, higher level gait disorders are more common and less well recognized.

Higher level gait disorders are gait abnormalities that cannot be explained by demonstrable deficits in the pyramidal, extrapyramidal, sensory or cerebellar systems. The gait abnormality is characterized by hesitancy and short, shuffling steps and patients may present with elements of pure dysequilibrium, failure of gait ignition, or both, as described by Nutt (2013). Some may previously have been categorized as Parkinsonism but have no other features of Parkinsonism on clinical assessment. The term vascular gait dyspraxia is helpful, emphasizing the vascular aetiology, with an increasing recognition of the association with white matter changes on neuroimaging.

Swallow disorders

The incidence of dysphagia after acute stroke can be up to 50% depending on stroke severity and the swallow assessment method, as shown by Mann et al (2000); however, recovery rates are generally high, probably as a result of the bilateral cortical representation of bulbar muscles. The prevalence of dysphagia in chronic stroke disease is unknown but it is likely to be significant given that swallow disorders affected about 15% of older people in general and almost 70% of nursing home residents in a study by Sura et al (2012).

Swallow impairment can often go undetected but can cause significant morbidity and mortality. Dysphagia may present as coughing around meals, poor oral intake causing weight loss, or recurrent pneumonia. Once detected, modifications for dysphagia such as chin tucking, thickened diet or volume control beakers can reduce the risk of these complications.

Depression

Psychological distress can be very prevalent in chronic disease in general, but the impact of psychological problems in acute and chronic stroke can be particularly profound. Up to 40% of patients post-acute stroke develop depression, which can have negative consequences on their post-stroke course and rehabilitation, as described by Dafer et al (2008).

The term 'vascular depression' is also now well recognized, with several studies, including that of Mast et al (2004), demonstrating a significant increase in the frequency of depression in patients with increasing vascular risk factors in the absence of acute stroke. It appears that cerebrovascular disease leads to deep white matter ischaemia, visible on magnetic resonance imaging, which confers a vulnerability to vascular depression, often triggered by challenging events or illness in later life, as described by Krishnan and McDonald (1995). There is also an overlap with executive dysfunction. Vascular depression is associated with impaired fluency and naming and more psychomotor retardation, with less agitation and greater lack of insight compared to non-vascular depression in older people.

Other prevalent psychological problems include anxiety and apathy, and access to psychological support is important given the complexities involved in coming the terms with loss of function and independence, which is often sudden and unexpected. Even in the absence of physical disability or depression post-stroke, fatigue can be marked and quite disabling.

Assessment

Initial history may highlight the presence of vascular risk factors or previous stroke. It is important to ask specifically about symptoms of swallow or mobility impairment, as patients will generally not volunteer this information without prompting. Stigmata of vascular disease, such as atrial fibrillation, congestive cardiac failure or peripheral vascular disease, may be present.

Once cognitive impairment is suspected, a sensitive cognitive screening tool such as the Mini Mental State Exam should be used, in conjunction with a focused neurological assessment and collateral history from a relative or carer. Patients with significant vascular memory impairment are more likely to be unaware of their cognitive deficits.

A simple swallow screening test, such as the Toronto Bedside Swallowing Screening Test, can be used to detect dysphagia, although one must be aware that a normal screening test does not rule out significant dysphagia in this setting, and silent aspiration can be common. More formal testing such as videofluoroscopy can be used if silent aspiration is suspected.

A useful brief measure of gait and balance is the timed get up and go test, where a score of less than 10 seconds is considered normal. Generally, if the patient cannot walk as well as one would expect he/she has a gait disorder, and if this cannot be explained by further deficits on a focused neurological exam the likely diagnosis is vascular gait dyspraxia. This diagnosis relies on having a clear sense of the status of the pyramidal, extrapyramidal, sensory and cerebellar systems. This exam can be done rapidly, but the patient needs to be on an examination couch with his/her shoes and socks off, and trousers as well if they can not easily be pulled up to mid-thigh. Classically vascular gait dyspraxia is characterized by failure of gait ignition, freezing, magnetic stepping and shuffling.

As part of this assessment it is also important to consider, and look for signs of neurodegenerative diseases such as Parkinson's disease and motor neurone disease, which can sometimes present with similar features.

Figure 1. Computed tomography of the brain demonstrating marked periventricular hypoattenuation, consistent with small vessel disease, as well as old right frontal infarct.

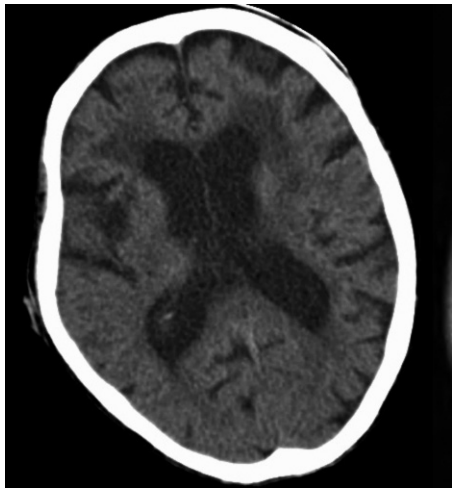
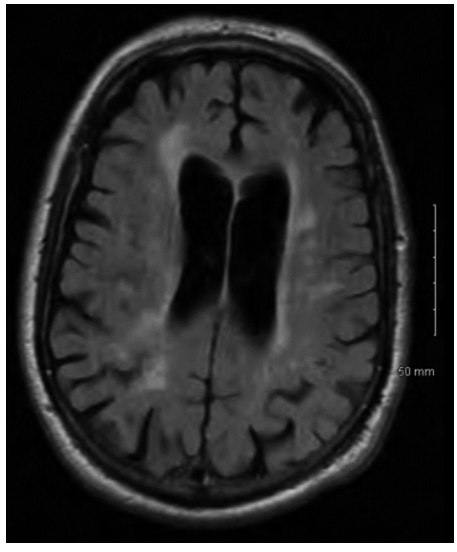


Figure 2. Magnetic resonance imaging fluid-attenuated inversion recovery (FLAIR) demonstrating periventricular and deep white matter hyperintensities.



Management

The diagnosis of chronic stroke disease is predominantly clinical but can be supported by appropriate neuroimaging. Computed tomography of the brain may demonstrate periventricular ischaemia, atrophy, lacunes and mature areas of chronic infarction. Additionally, the burden of small vessel disease is represented by hyperintensities, either periventricular or deep white matter, on magnetic resonance imaging brain T2 and FLAIR (fluid-attenuated inversion recovery) sequences. Magnetic resonance gradient echo sequences can identify cerebral amyloid angiopathy-related microhaemorrhage (*Figures 1* and *2*).

Specific multidisciplinary treatment strategies should be considered in all patients with chronic stroke disease. Gait disorders tend to respond well to gait and balance training and re-education with physiotherapy. For patients with chronic hemiparesis splinting by an occupational therapist or treatment with tizanidine may help in the treatment and prevention of contractures. Use of a modified diet and swallowing strategies, under the direction of speech and language therapy, may help avoid the complications of dysphagia, such as aspiration.

Individualized supports and advance care planning may be of benefit in vascular and post-stroke dementia while treatment with cholinesterase inhibitors may yield modest benefits. The addition of memantine may be beneficial in moderate to severe disease.

The role of secondary prevention therapy to avoid further stroke is well defined, but it is less clear if secondary prevention in chronic stroke can have a positive impact on the incidence or progression of gait disorders and cognitive deficits in chronic stroke. Intuitively one would think that given the likely vascular aetiology of these features that treatment such as blood pressure control or antiplatelet therapy would be beneficial, and some observational studies support this but as yet no randomized controlled trials. This may be a result, in part, of the relative complexities involved in researching this area but the lack of widespread recognition of chronic stroke syndromes must also play a role in this.

The benefit, and specifically the duration, of rehabilitation for chronic stroke syndromes is also controversial. Conventional thinking is that one's rehabilitation potential and functional recovery plateaus on average 2–4 months after acute stroke when a 'new baseline' is deemed to be reached. This is not necessarily backed up with strong evidence, however, and some small studies, including that by Teasell et al (2012), have demonstrated the benefit of more prolonged focused rehabilitation in chronic stroke. While it would be irresponsible and impractical to prolong intensive rehabilitation indefinitely in all chronic stroke patients, one must consider the potential for positively altering function and wellbeing in patients after this early period of rehabilitation.

KEY POINTS

- Stroke is often not considered a chronic disease, with the focus on acute and hyperacute stroke care often overshadowing that of chronic stroke. This is a pity given the significant morbidity associated with chronic stroke.
- The manifestations of chronic stroke disease include cognitive deficits, dysphagia, gait disorders and depression.
- One does not have to present with an overt acute stroke in order to develop manifestations of chronic stroke.
- Features such as mobility or swallow problems in chronic stroke can occur so gradually, and be compensated for by the patient so that they may not be reported as a symptom.
- The classical gait abnormality is vascular gait dyspraxia, characterized by hesitancy and short, shuffling steps, without any evidence of deficits in the pyramidal, extrapyramidal, sensory or cerebellar systems.
- Specific treatment strategies, such as physiotherapy for gait disorders and speech and language therapy for dysphagia, should be considered in all patients with chronic stroke disease.
- The role of pharmacological intervention in chronic stroke is unclear but few studies have been conducted to date.

While coordinated care services exist for management of other chronic diseases such as diabetes and congestive cardiac failure, there is no such service at present for chronic stroke disease. Improved care models for chronic stroke linking primary and secondary care, as well as nursing home care, are required and have been shown to be successful previously, for example by Allen et al (2002).

Conclusions

Audits of stroke care, including that conducted by Hickey et al (2012), continue to show alarming levels of neglect in terms of chronic disease management. Given the recent advances in acute stroke care, the next important step is to increase the organization and coordination of chronic stroke disease management but this will require a sharper focus on awareness, recognition and assessment of this important chronic disease. **BJHM**

Conflict of interest: none.

Allen KR, Hazelett S, Jarjoura D et al (2002) Effectiveness of a postdischarge care management model for stroke and transient ischemic attack: a randomized trial. *J Stroke Cerebrovasc Dis* **11**(2): 88–98 (doi: 10.1053/jscd.2002.127106)

Cowman S, Royston M, Hickey A, Horgan F, McGee H, O'Neill D (2010) Stroke and nursing home care: a national survey of nursing homes. *BMC Geriatr* **10**: 4 (doi: 10.1186/1471-2318-10-4)

Dafer RM, Rao M, Shareef A, Sharma A (2008) Poststroke depression. *Top Stroke Rehabil* **15**(1): 13–21 (doi: 10.1310/tsr1501-13)

Feigin VL, Forouzanfar MH, Krishnamurthi R et al; Global Burden of Diseases, Injuries, and Risk Factors Study 2010 (GBD 2010) and the GBD Stroke Experts Group (2014) Global and regional burden of stroke during 1990-2010: findings from the Global Burden of Disease Study 2010. *Lancet* **383**(9913): 245–54 (doi: 10.1016/S0140-6736(13)61953-4)

Hardie K, Hankey GJ, Jamrozik K, Broadhurst RJ, Anderson C (2004) Ten-year risk of first recurrent stroke and disability after first-ever stroke in the Perth Community Stroke Study. *Stroke* **35**(3): 731–5 (doi: 10.1161/01.STR.0000116183.50167.D9)

Hickey A, Horgan F, O'Neill D, McGee H (2012) Community-based post-stroke service provision and challenges: a national survey of managers and inter-disciplinary healthcare staff in Ireland. *BMC Health Serv Res* **6**(12): 111 (doi: 10.1186/1472-6963-12-111)

Krishnan KR, McDonald WM (1995) Arteriosclerotic depression. *Med Hypotheses* **44**(2): 111–15

Leys D, Hénon H, Mackowiak-Cordoliani MA, Pasquier F (2005) Poststroke dementia. *Lancet Neurol* **4**(11): 752–9 (doi: 10.1016/S1474-4422(05)70221-0)

Mann G, Hankey GJ, Cameron D (2000) Swallowing disorders following acute stroke: prevalence and diagnostic accuracy. *Cerebrovasc Dis* **10**(5): 380–6 (doi: 10.1159/000016094)

Mast BT, MacNeill SE, Lichtenberg PA (2004) Post-stroke and clinically-defined vascular depression in geriatric rehabilitation patients. *Am J Geriatr Psychiatry* **12**(1): 84–92

Nutt JG (2013) Higher-level gait disorders: an open frontier. *Mov Disord* **28**(11): 1560–5 (doi: 10.1002/mds.25673)

O'Neill D, Macsweeney CA, Cornell IA, Moss H (2014) Stravinsky syndrome: giving a voice to chronic stroke disease. *QJM* **107**(6): 489–93 (doi: 10.1093/qjmed/hcu059)

Sura L, Madhavan A, Carnaby G, Crary MA (2012) Dysphagia in the elderly: management and nutritional considerations. *Clin Interv Aging* **7**: 287–98 (doi: 10.2147/CIA.S23404)

Teasell R, Mehta S, Pereira S et al (2012) Time to rethink long-term rehabilitation management of stroke patients. *Top Stroke Rehabil* **19**(6): 457–62 (doi: 10.1310/tsr1906-457)