EDITORIALS

Vascular parkinsonism—an important update

The differential diagnosis of parkinsonism changes with the age of onset of the syndrome. In older patients, vascular parkinsonism is an important cause [1]. Recognition of the condition is attributed to McDonald Critchley, who wrote an article on arteriosclerotic parkinsonism in 1929 [2]. There has been debate since then about the existence of the condition. For example, in one study it was found that arterial disease was no more frequent in patients with parkinsonism than in controls [3]. The diagnosis can be difficult to make, partly because many patients with Lewy-body Parkinson’s disease have concomitant cerebrovascular disease [4]. Many patients with vascular parkinsonism do respond to the right treatment so correct diagnosis is important [5].

The article by Thanvi et al. in this issue is a timely and important update to current knowledge of this condition, giving guidance on making the right diagnosis and appropriate management [6]. Geriatricians need to be well informed of this work—not only those running movement disorder clinics. but also those who see patients with mobility problems, falls, cognitive impairment and incontinence—because vascular parkinsonism commonly presents in a non-specific fashion. Many geriatricians will be managing patients with parkinsonism because about 80% of cases are over 65 years of age [7].

Thanvi supports the view that vascular parkinsonism affects the lower body with predominant gait problems [8] and postural instability. However, upper limb symptoms and signs may be more frequent than is traditionally thought to be the case. Tremor affects over half of the patients, though postural tremor is more common than rest tremor [9]. Cognitive dysfunction may be present in about a third of cases [10]. Patients may have other signs of cerebrovascular disease. The mean age of onset is about 10 years later than for idiopathic Parkinson’s disease [9]. Patients suffer from hypertension in as many as 70% of cases [11], and gave a history of stroke, in one study, in 43% of cases which helps the distinction from idiopathic Parkinson’s disease [12]. Prognosis is therefore likely to be worse on average than for patients with idiopathic Parkinson’s disease.

Differentiating vascular parkinsonism from idiopathic Parkinson’s disease is not always easy. One recent suggestion not mentioned by Thanvi is that as the sense of smell is universally affected in idiopathic Parkinson’s disease and not in vascular parkinsonism, testing smell can aid with diagnosis [13]. Investigation is to be recommended if there are any atypical features in someone with possible Parkinson’s disease. MRI is a sensitive way of detecting cerebrovascular disease, though CT scanning in many areas is more readily available and causes less patient distress. DaT scanning—using a gamma camera—which measures dopamine transporter activity in the pre-synaptic nigro-striatal neurons, is becoming more widely available. Though it is a costly test, it is not particularly unpleasant for the patient and can help in many cases to differentiate Parkinson’s disease from essential tremor, drug-induced parkinsonism and vascular parkinsonism [14].

Vascular pathology of the syndrome varies, though patients usually have small-vessel cerebral disease, commonly periventricular ischaemia [10], but the patients who respond best to L-dopa treatment are those with predominant basal ganglia pathology [5]. However, only a few patients with striatal infarcts develop parkinsonism [15]. Another interesting association mentioned by Thanvi is the presence of anticardiolipin antibodies in as many as 40% of patients. If autoimmune vascular disease is part of the cause this may explain the lack of a consistent correlation between the presence of basal ganglia vascular disease and the signs of vascular parkinsonism [16].

Most patients deserve a trial with L-dopa for 3 months, but the response may be disappointing and not sustained. Imaging may help to distinguish those with basal ganglia disease, who are most likely to benefit from treatment. There is no evidence of benefit from other drugs used in idiopathic Parkinson’s disease. Thanvi reports on a study which has shown clinical benefit from drainage of cerebrospinal fluid in vascular parkinsonism [6]. This was tried because of the similar clinical and, in some cases, pathological features as normal pressure hydrocephalus. Patients with hypertension and other vascular risk factors, such as smoking, justify appropriate action to reduce the risk of progression of arteriosclerosis. Antiplatelet agents should be used, though there is no evidence that these affect the progress of the movement disorder. All patients with disability will justify assessment and treatment by the multidisciplinary team as appropriate to their problems.

In summary, there is increasing information about this condition, which is leading to an improved ability to make the correct diagnosis, and the role of treatment. Further research will hopefully lead us to more effective treatment and hopefully enable us to do more to prevent the disease.

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References
Prevention of falls—a time to translate evidence into practice

Over the last 25 years we have seen a substantial rise in the number of publications in the area of falls in older people (Figure 1). It is 10 years since the publication of Tinetti’s seminal paper showing that it is possible to prevent falls in an at-risk population [1]. The recent release of UK NICE guidelines ‘Falls: the assessment and prevention of falls in older people’ summarise the available evidence up until the end of 2003 and give simple guidance on utilisation and serious injury in the intervention group. As with PROFET [6], Davison et al. report trends towards reduction in subsequent bed day utilisation and serious injury in the intervention group.

In the trial, all consenting participants in the intervention group underwent detailed cardiovascular assessment across the world, this is potentially resource intense in the control and intervention group but not in the proportion of fallers in the two groups. The authors openly acknowledge that 1:5 of the control group also received a falls assessment and intervention, and this is likely to reflect normal care in a unit where falls have been high profile for many years. It is therefore possible that the effect size of the intervention was diluted. As with PROFET [6], Davison et al. report trends towards reduction in subsequent bed day utilisation and serious injury in the intervention group.

In the trial, all consenting participants in the intervention group underwent detailed cardiovascular assessment including carotid sinus massage irrespective of the nature of the falls. For many service providers in the UK and across the world, this is potentially resource intense in terms of finance and manpower, notwithstanding the risk to the individual patient of a 1:1000 chance of a neurological event [7].

Whilst successful in a cognitively intact Emergency Department population, this multifaceted approach, when applied with the same rigour, has not been shown to have the same impact on cognitively impaired individuals [8], leaving us with a gap in our current knowledge base as to how we prevent falls in cognitively impaired older people.

There is little doubt that prevention of falls is a complex area given the methods by which we identify and define risk, the heterogeneity of the population at risk and the modes of intervention on offer. Are all falls of equal impact and are all interventions of equal value within and between different at-risk populations? Should the aim of intervention strategies be to prevent all falls or prevent ones which impact on