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Is Parkinson's Disease truly a disease or simply an indication of a long life

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Abstract

Parkinson described paralysis agitans more than 200 years ago but reference to the condition dates back to ancient times, thereby suggesting it is far more common than many appreciate.

While the relationship of Parkinson's Disease to the reduction of dopamine is not disputed, it is accepted that the proteins, involved in Parkinson's Disease, such as α-synuclein, UCH-L1, PINK1 or DJ-1 are also involved in the aging process.

Accepting that there are genetic conditions that predispose to the early expression of Parkinson's Disease, the argument remains that Parkinson's Disease is not a disease entity per se but rather the expression of aging and if one lives long enough one will acquire Parkinson's Disease.

James Parkinson (11/04/1755 - 21/12/1824), after whom Parkinson's Disease was eponymously named, wrote his "Essay of the Shaking Palsy" more than 200 years ago [1], in 1817. He failed the entry criteria to study medicine and came to Australia for the Gold Rush and was a lighthouse keeper near Hobart in Tasmania [1]. He has been described as an "English apothecary surgeon, geologist, palentologist, and political activist [2]. Elements of so-called Parkinson's Disease were identifiable in ancient Chinese, Indian, Babylonian and Greek texts, thereby establishing its relatively broad-based, ubiquitous nature across cultures and societies [2].

Whereas Parkinson identified the characteristic "… resting tremor, stiffness and characteristic station and gait….that he called paralysis agitans…", in 1872 Charcot added bradykinesia as a seminal feature [2]. There remains no absolutely reliable, objective test to clinically diagnose Parkinson's Disease, with the diagnosis being dependent on expert opinion as the benchmark "gold standard" [3]. The diagnosis remains reliant on the cardinal features of bradykinesia, rigidity and resting tremor [4] with a basic requirement of at least two out of these three features. Various primitive reflexes, such as the glabellar tap, grasp reflexes and palmar mental/nasopalpebral reflexes, when present, assist in the clinical diagnosis of Parkinson's Disease [5]. On this basis alone, Parkinson's Disease is a clinical diagnosis that relies on the acumen of the clinician involved. After many years of practice, as a consultant physician/neurologist, it has become apparent that Parkinson's Disease is far more common than most observers accept. With features identified in ancient societies [2] the question arises whether Parkinson's Disease is truly a disease, thereby establishing it as a pathological entity, or whether it merely reflects a pattern of expression that is associated with the normal aging process.

It is accepted that Parkinson's Disease results from reduction of dopamine in the basal ganglia [6] with consequential losses in the thalamus and cortex [6]. This has resulted in enhanced appreciation of the link between degeneration of dopamine neurones in the mid-brain and the development of Parkinsonism [6]. It is widely accepted that those proteins involved in the pathogenesis of Parkinson's Disease, such as α-synuclein, UCH-L1, PINK1 or DJ-1 are also involved in aging [6]. "….Present data suggests that Parkinson's Disease could be the expression of aging on a cell population with high vulnerability to aging…" [6]. It is hypothesised that those neurones which degenerate during the evolution of Parkinson's Disease also degenerate during the normal aging process and that this may represent a local expression of aging on a particularly vulnerable cell population due to an increased number of synaptic terminals, mitochondria and unmyelinated axons [7]. It is acknowledged that one of the dominant determinates of clinical progression of Parkinson's Disease is advancing age, rather than disease duration, and there is a biological interaction between the disease process and aging on non-dopaminergic structures [8]. The prevalence of Parkinsonian features is significantly increased in the older population [9] with an artificial separation and differentiation between Parkinson's Disease and Parkinsonism [9].

Conclusion

Taking all of these features into account, complemented by a long experience of successful management of Parkinson's Disease, predicated on very early introduction of anti-Parkinsonian remedies, particularly L'dopa [10], has led to the supposition that Parkinson's Disease may not be a specific disease entity but rather an expression of aging that

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was initially recognised and documented in ancient times. Accepting one can trace references to the "disease" to the ancient records from China, India, Babylon and Greece [2] and even within the present era of scientific advancement and technological achievement, the diagnosis remains a clinical determination, [3] there exists cogent argument to justify the stance that the so-called entity "Parkinson's Disease" is merely an expression of the aging process. Perhaps it is not a disease at all but simply an indication of a long life with the advancement of medicine realising more people living to the vulnerable age bracket. This does not negate there being genetic predispositions, resulting in premature expression of the Parkinsonian constellation, as associated with UCH-L1 or PINK1. A critical evaluation of both pathophysiology and the clinical picture, over centuries, endorses the position that Parkinson's Disease is more a factor of aging than a specific pathological entity, hence the various types of Parkinsonism. The hypothesis is that if one lives long enough one will develop Parkinsonian features.

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