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Population-Based Parkinson's Study Cohort to Confirm Pain Diagnosis and Subtypes

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Abstract

Intracellular deposits of aggregated α synuclein, ubiquitin, and other proteins found in many surviving neuronal populations are considered to be the pathologic characteristic of Parkinsons Disease. Whether Lewy bodies are themselves neurotoxic, or represent the end product of cellular defence mechanisms to sequester toxic abnormal proteins, remains to be determined.

Keywords: Causal mutations; Dominant early-onset; Leucine-rich; Non-familial disease; Response rates; Exercise prescription

Introduction

Similar to Alzheimer's disease, epidemiologic differences in early-onset and late-onset Parkinson's disease have been described. Genetic factors, especially specific causal mutations, appear to be more prominent in early-onset Parkinsons Disease, although the distinctions are by no means absolute. Kindred studies of heavily affected families have identified at least five genetic loci for Parkinson's disease. The initial discoveries were mutations of the gene encoding the α-syncline protein that have been related to autosomal dominant early-onset Parkinson's Disease, typified by rapid disease onset and progression. The functional consequences of mutations in these genes are incompletely understood, although abnormal brain protein aggregation and clearance appears to be a common feature [1]. Mutations in the leucine-rich repeat kinase 2, first identified from kindred studies in Japan and subsequently confirmed in Europe and North America, have also been associated with typical late-onset Parkinson's disease, and thus may also contribute to risk for non-familial Parkinson's disease. Identified mutations in other genes include, parkin, Phosphatase and tension homolog on chromosome 10-induced putative kinase I and DJ-1, all of which follow a recessive inheritance mode [2]. Candidate gene studies for late-onset non-familial Parkinson's disease have explored associations with the same genes related to familial Parkinson's disease. In general, the rare causal mutations observed for familial Parkinson's disease have not been associated consistently with non-familial disease.

Methodology

Extensive efforts have also been undertaken to identify common variants of biologically-based candidate genes that may confer Parkinsons Disease susceptibility, either independently or in combination with host or environmental factors [3]. These include variants of genes related to the metabolism of dopamine and toxic environmental chemicals, and to presumed Parkinson's disease pathogenesis mechanisms [4]. Perhaps not surprisingly, numerous associations have been observed, yet attempts at replication have been largely disappointing. An illustration is the inconsistent pattern of results for the gene encoding the enzyme monoamine oxidase B that catabolizes dopamine [5]. Apart from older age, the most consistent epidemiologic observation has been an inverse relation between cigarette smoking and Parkinson's disease, with smokers having approximately half the rate as never smokers, and strong evidence for an inverse dose response effect with duration and pack-years smoked as shown in Figure 1. The reduced risk among smokers does not appear to be due to selective survival bias. A biochemical basis may be the lowering of MAO-B enzyme activity in the brain, and consequent reduced dopamine catabolism [6]. Alternatively, aversion to novelty-seeking behaviour, such as smoking, by persons who ultimately develop Parkinson's disease may explain the relation with smoking. Inverse Parkinson's Disease risk associations have also been reported for caffeine and non-steroidal anti-inflammatory medications, although the evidence is less consistent than for smoking. Additionally, family history of Parkinson's disease and history of severe head trauma have been related to elevated Parkinson's disease risks [7]. No studies examined the explanations underlying the reported attitudes so it is difficult to draw strong conclusions from the data regarding attitudes. However, published discussions suggest factors that may negatively affect General Practitioner's attitudes: exposure to contradictory information, concern about lack of efficacy and potential for harm. Clinical and research implications resulting from this study Small response rates and use of specialist groups of General Practitioner's limited the generalizability of the results of many studies.

Figure 1: Epidemiologic observation being an inverse relation between smoking and Parkinson's disease.

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Such samples may provide over-estimates of exercise behaviour [8]. Given the apparent under-use of exercise by General Practitioner's for patients with Chronic Knee Pain, it is possible that the true pattern of practice is even further from exercise recommendations in available guidelines. The negative clinical effect of this apparent under-use of exercise use may be further exaggerated if patients are unable to translate advice or instructions into correctly executed and frequently performed exercises. Dexter noted that of those that had been advised to exercise for hip and/or knee osteoarthritis only did so [9]. In addition, only patients who were undertaking strengthening and/or stretching exercises of the hip or knee were performing these correctly and regularly. Individual studies suggested potential reasons for the apparent under-use of exercise by General Practitioners. These include, uncertainty about the role of General Practitioners in relation to exercise for Chronic Knee Pain and appropriate types of exercise, uncertainty of the correct exercise prescription, lack of awareness about the guidelines, the belief that patients will not exercise, the presence of comorbidities, increasing patient age, and limited access to services [10]. Barriers imposed by healthcare systems such as unclear referral criteria, limited onward referral to other healthcare professionals and limited consultation time may prevent General Practitioners from providing their desired management. Ostbye reinforced the latter issue by identifying that provision of comprehensive management for ten common chronic diseases, including arthritis, exceeds the total time General Practitioners have for all patient care. Future research should focus on consistent investigation of attitudes, beliefs and behaviours of General Practitioners regarding the use of exercise for Chronic Knee Pain. Research should identify, or confirm suggested, barriers to the use of exercise for Chronic Knee Pain and thus full implementation of national guidelines. System barriers and General Practitioners attitudes and behaviours, may vary within and between countries due to local and national differences in healthcare provision. Therefore, further research should utilise large, nationally representative samples of General Practitioners. The role of General Practitioners in initiating exercise for Chronic Knee Pain was not outlined in studies or guidelines, including the recent NICE guidelines [11]. Primary care guidelines recommend exercise as a core management approach for Chronic Knee Pain but provide no explicit expectations about whether General Practitioners should refer patients for exercise therapies, advise general or specific exercises, or prescribe exercises. The expected roles of General Practitioners in initiating and supporting exercise in patients with Chronic Knee Pain thus require clarification.

Discussion

Work must also identify the optimal means of supporting and educating General Practitioners at the clinical, educational and service level, to improve certainty and confidence about the value of exercise and to use the exercise recommendations in practice. Our systematic review has highlighted a paucity of studies investigating, and variability in, the attitudes, beliefs and behaviours of General Practitioners regarding the use of exercise for Chronic Knee Pain [12]. However, this treatment modality appears to be underused by General Practitioners. Future work should investigate the attitudes, beliefs and behaviours of General Practitioners regarding exercise for Chronic Knee Pain and clarify the expected roles of General Practitioners to help support the translation of best practice recommendations into everyday clinical care. Appropriate diagnosis of knee osteoarthritis is an essential pre-requisite to treatment, in this respect [13]. Virtually all existing practice guidelines agree that combination of treatment modalities, including non-pharmacological and pharmacological intervention, is strongly recommended. It was the firm opinion of the task force that this basic

Figure 2: Initial measures and interventions.

principle is valid and makes the effort to develop a treatment algorithm an absolute priority in order to prevent physicians from being confused as to how treatments should be prioritized and possibly added on for combination therapy. The core set proposed was adopted and expanded by the task force to represent the initial measures and interventions that every patient with knee osteoarthritis should undergo as shown in (Figure 2). In particular, Information access and education consists in providing to the patient the necessary knowledge about the nature of the disease and the objectives of treatment [14]. If necessary, the physician should prompt changes in the patient's lifestyle toward behaviours that may have a beneficial impact on joint protection or at least not worsen the progression of the disease or of its symptoms. It is recognized that these measures have minimal effect on osteoarthritis symptoms, but they are essential for treatment adherence. European Alliance of Associations for Rheumatology has recently published comprehensive recommendations for the non-pharmacological management of hip and knee osteoarthritis that provided extensive guidance on the principles of information and education, as well as of lifestyle changes. Analysis of the available evidence indicates that at least weight loss within months induces a small but well-substantiated symptomatic benefit, more evident on physical function than on pain, where the effect is less predictable. The task force strongly felt that a threshold should be indicated and, based on previous evidence and recent high quality trials, weight loss should be targeted to at least to achieve significant symptom benefit. A similar degree of weight loss has also been indicated to improve the quality and thickness of medial femoral compartment cartilage. Exercise program [15].

Conclusion

Education should include information about exercise and physical activity since exercise produces benefit on both pain and function in patients with knee osteoarthritis by different delivery modes. Although the optimal exercise dosing and rate of progression in its application remains unclear, expert opinion suggests that the intensity and/or duration of exercise should be increased over time. There is good evidence that water-based exercise is effective on both pain and function. However, specific quadriceps strengthening exercises or strength training for the lower limb, together with aerobic training such as walking, remain the best documented exercise approaches, experts suggest that mixed programs should be recommended as long as minimal intensity requirements are met. Recent evidence suggests that tai chi is also effective in relieving symptoms.

Acknowledgement

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Conflict of Interest

None

References

1. Weiser M (2004) The Homeopathic Preparation Traumeel® S Compared With NSAIDs For Symptomatic Treatment Of Epicondylitis. J Musculoskelet Res EU 8:119-128.
2. Moattar F (2009) Comparison of effects of ginger, mefenamic acid, and ibuprofen on pain in women with primary dysmenorrhea. J Altern Complement Med US 15:129-132.
3. Dahl V (2009) Clinical application of glucocorticoids, antineuropathics, and other analgesic adjuvants for acute pain management. CUP UK: 398-731.
4. Prystupa A, Drozd M (2013) Assessment of pain: types, mechanism and treatment. Ann Agric Environ Med EU 1:2-7.
5. Kruse RJ (2004) The physiologic basis and clinical applications of cryotherapy and thermotherapy for the pain practitioner. Pain Physician US 7:395-399.
6. Trout KK (2004) The neuromatrix theory of pain: implications for selected non-pharmacologic methods of pain relief for labor. J Midwifery Wom Heal US 49:482-488.
7. Mao J (2014) Neuropathic pain: mechanisms and their clinical implications. BMJ UK 348:1-6.
8. Mello RD (2008) Spinal cord mechanisms of pain. BJA US 101:8-16.
9. Weidner MS, Andersen LA (2000) A randomized, placebo-controlled, cross-over study of ginger extracts and ibuprofen in osteoarthritis. Osteoarthr Cartil EU 8:9-12.
10. Ross NA (2006) Natural anti-inflammatory agents for pain relief in athletes. Neurosurg Focus US 21:1-13.
11. Kahn LH (2006) Confronting zoonoses, linking human and veterinary medicine. Emerg Infect Dis US 12:556-561.
12. Smith HV (2000) Emerging parasite zoonosis associated with water and food. Int J Parasitol EU 30:1379-1393.
13. Macpherson CNL (2014) Zoonoses and one health: a review of the literature. J Parasitol 2014:1-8.
14. Cooper GS (2004) Occupational and environmental exposures as risk factors for systemic lupus erythematosus. Curr Rheumatol Rep EU 6:367-374.
15. Costenbader KH (2016) Environmental exposures and the development of systemic lupus erythematosus. Curr Opin Rheumatol US 28:497-505.