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Simultaneous occurrence of neuromyotonia and morphoea: a cause–effect relationship?

We report a case of unusual occurrence of neuromyotonia and morphoea (localised scleroderma) simultaneously in the same limbs. The patient was a 32-year-old man who presented with a 2-year history of twitching muscles in the right limbs with loss of hair and tightness of skin in the same limbs. Examination showed an almost complete loss of hair on the right arm, forearm and hand (fig 1). He also had hair loss on the right lower limb, especially on the back of the thigh and calf (fig 2). The skin in these areas seemed to be stretched and shiny with a parchment-like appearance. The other remarkable feature on examination was myokymia of the right-sided biceps, triceps, deltoid, brachioradialis, hamstrings and calf muscles. The rest of the neurological examination was normal. The clinical diagnosis was morphoea associated with neuromyotonia.

The antinuclear antibody was weakly positive. Anti-SCL-70 and anti-JO-1 antibodies were negative. CT scan of the chest was normal. Results of nerve conduction and repetitive nerve stimulation studies were normal. Examination by needle electromyography of the right deltoid, biceps, triceps and brachioradialis showed increased spontaneous activity, with single, doublet, triplet and long bursts of single motor unit potentials at rest (fig 3). Very long bursts (up to 60 s) of motor unit potentials akin to voluntary contraction were seen at rest in the right vastus lateralis. Needle electromyographic findings of the muscles of the left

Figure 1 Forearms and hands of the patient showing lack of hair on the right side.

Figure 2 Back of thighs and legs of the patient, showing lack of hair on the right side.

Figure 3 Needle electromyographic findings of the right deltoid showing motor unit potentials at rest.

limbs were normal. The result of skin biopsy from the affected area in the right arm showed a thinned out epidermis with hyperkeratinisation, with atrophic changes in the fibrous connective tissue in the dermis. Collagenisation and compression of fibrous tissue into dense compact collagen masses was seen in the dermis. Bundles of thickened nerves with round cell infiltrates were observed. These observations gave an impression of morphoea. A marked improvement in the neuromyotonia was noted within a fortnight of starting treatment with 300 mg/day phenytoin. No immunomodulatory treatment was considered because of the excellent response to phenytoin.

The most remarkable feature in our patient was the spatial and temporal coincidence of morphoea and neuromyotonia. This suggests an arcane relationship between the two conditions. The motor axon being the possible site of origin of neuromyotonia,^{1,2} one possible mechanism could be pathological change of nerves due to scleroderma.³ The appearance of nerve endings in the skin biopsy specimen lend credence to this view.

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Ear dyskinesia

Focal or segmental dyskinesias and dystonic syndromes affecting the cranial muscles are well recognised. Examples include the well known oro-bucco-facial dyskinesia caused by the long term use of neuroleptics, Meige syndrome, oromandibular dystonia, and blepharospasm. Among focal dyskinesias, ear dyskinesias are extremely uncommon and only a few cases have been reported.^{1,2}

Case report

We report a 57 year old woman who complained of involuntary movements of both ears for a year. The movements were initially intermittent but progressively became continuous. She could not control them voluntarily. The movements disappeared during sleep. There was no history of local trauma or neuroleptic treatment. Three months before onset, she had received paroxetine for depression. On examination there was a semirhythmic synchronous elevation and retraction of both ears. Movements were also seen in the skin around the ear. There was an intermittent contraction of frontal muscles as well. On clinical examination, there was amimia, and slow bilateral alternating serial movements. There were no tremor or hypertonias. MRI of the brain was normal. Electromyography of the left auricularis superior muscle showed bursts of normal motor unit potentials lasting 280 ms at a frequency of 2 Hz. The patient responded well to local injections of botulinum toxin type A (Botox) into the auricularis superior and posterior muscles (40 MU). The toxin was injected without EMG monitoring.

Discussion

The clinical appearance of our patient's movement disorder closely resembles dyskinesia because it consists of stereotyped focal or segmental movements affecting the ears and lasting for a few seconds or so. Focal dyskinesia can affect various regions of the body: ear, back, shoulder girdle and upper

extremity, and abdomen.¹ Movements arise spontaneously, following either trauma, surgery, or neuroleptic drug intake. Our patient had been taking selective serotonin reuptake inhibitors (SSRI) for depression for three months before the onset of ear dyskinesias but these persisted despite paroxetine discontinuation. The use of neuroleptics had been excluded before treatment with paroxetine.

Several case reports suggest that SSRI may play an important role in the development of movement disorders.³ A retrospective review reported 71 cases of de novo motor symptoms after SSRI use.³ Akathisia was reported in 32 cases, dystonia in 20, parkinsonism in 10, and tardive dyskinesia-like movements in eight. The interval between treatment initiation and onset of side effects was highly variable, from three days to one year for tardive dyskinesia-like movements. Usually, tardive dyskinesia-like movements consist of orobucco-lingual dyskinesia. So far, no ear dyskinesias have been reported after SSRI use. SSRI-induced dyskinesia may thus be related to serotonergic effects upon dopamine rather than to dopamine-2 receptor blockade.³

The ear movements described here also raise the question of their relation to palatal myoclonus. Others indeed have reported a case of "auricular myoclonus" with irregular clonic movements affecting the antitragus and anthelix at a rate of 70 to 75 per minute and discussed the possibility of a brain stem origin for the myoclonus, analogous to palatal myoclonus.⁴ In contrast, the ear movement in the present case consisted of variable jerky and quite slow elevations (frequency 2 Hz) and posterior rotation of ear muscles, particularly the auricularis superior. Our patient had neither associated palatal movements or ear clicks, as seen in essential palatal myoclonus, nor evidence of a brain stem lesion characteristic of symptomatic palatal myoclonus. Ten cases of "ear wigglers" caused by tics of the ear were described by Keshavan.⁵ However, ear tic is unlikely in this patient because the movements were slow (frequency 2 Hz), semi-rhythmic, and not suppressed by voluntarily effort.

We propose the use of botulinum toxin injections for the treatment of ear dyskinesias.

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BOOK REVIEWS

Psychiatric issues in Parkinson's disease. A practical guide

Edited by Matthew Menza, Laura Marsh. Published by Taylor & Francis, New York, 2005, £75.00 (hardcover), pp 340. ISBN 1-84184-491-8

The non-motor, and particularly psychiatric, aspects of Parkinson's disease are increasingly recognised as difficult and important problems that may impact more on patients' well-being, and be more difficult to manage, than the motor or other physical aspects. This book brings together reviews on theoretical and practical facets of this area from a variety of specialties. It is a useful guide to neurologists and psychiatrists involved in the care of patients with Parkinson's disease, but perhaps its greatest appeal lies in the fact that it is directed towards all those interested and affected by the psychiatric manifestations and complications of Parkinson's disease, including allied healthcare professionals, patients and caregivers. It combines in one book a comprehensive review of the psychiatric aspects of Parkinson's disease as well as providing an overview of motor and other physical aspects of this disorder for healthcare professionals who primarily are involved in the management of the psychiatric problems of patients with Parkinson's disease.

A brief but comprehensive section introduces pathogenesis, diagnosis and treatment of Parkinson's disease, which puts psychiatric symptoms into context and is aimed primarily at non-neurologists. The following two sections represent the heart of the book: Cognitive Dysfunction and Psychiatric Disturbances. The chapter on cognitive impairment includes a lot of detail although one might wish for more practical recommendations on which tests to use. However, this is provided in the following chapter on dementia with a useful approach on how to assess patients in practice and decide on the presence of dementia and its contribution to social and occupational impairment. A further, authoritative chapter in this section is dedicated to the difference between dementia with Lewy bodies and Parkinson's disease with dementia and gives a comprehensive, informative review. The section on Psychiatric Disturbances covers the large areas of depression, anxiety disorders, psychosis, and sleep disturbances, each with appropriate descriptions of the clinical problems and the various pharmacological and non-pharmacological management options. It also includes a chapter on the comparatively rare but fascinating and difficult to manage behavioural disturbances, including deficit disorders (eg apathy and hyposexuality), impulse control disorders (eg dopamine dysregulation, disinhibition, mania, pathological gambling) and repetitive behaviours (eg obsessive-compulsive behaviours and punding), and briefly the effects of neurosurgical interventions on behaviour. Off-period related psychiatric phenomena are discussed in the first section but are briefly mentioned here as well. A particularly interesting and arguably the most useful section is the fourth and last part of the book on "Special Issues". This focuses on the little

studied but important aspects of disability assessment and management and the impact of psychosocial and personal factors on the management and experience of patients with Parkinson's disease. Thus, separate chapters are dedicated to coping skills, personality issues, assessment of disability, rehabilitation and long-term care with a separate chapter on caregivers. Like the entirety of the book, it is directed at all healthcare professionals and also patients and their families, and addresses the holistic approach to managing and coping with Parkinson's disease and its psychiatric symptoms. It is necessarily less evidence-based than the previous sections, but gives many practical suggestions and highlights the need for further research—for example, on the role of rehabilitation.

The list of contributors of this book reflects a wide range of expertise and backgrounds, including Parkinson's disease associations and caregivers. There is a relative North American emphasis in the group of contributors (reflected in the list of patient organisations at the end), but the book is nevertheless likely to be useful for sufferers, caregivers and healthcare professionals in other countries. Technical terms are always explained, often with examples, which makes the information accessible to a wide range of readers while preserving a comprehensive and authoritative knowledge base.

This book will provide informative and practical help to a wide range of readers from patients and caregivers to allied health professional to neurologists interested in the specific psychiatric aspects of Parkinson's disease to psychologists managing these difficult aspects of this chronic and difficult disease.

A Schrag

Oxford textbook of medicine

Edited by David A Warrell. Published by Oxford University Press, Oxford, 2005, £150 (softcover), pp 1085. ISBN 0-19-856978-5

Never before has so much information on medicine been so available to so many.

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