CLINICAL WORK IN GENERAL PRACTICE 8

Familial Bell's palsy

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SUMMARY. Six attacks of Bell's palsy occurring in five members of one family are described.

Introduction

THE aetiology of Bell's palsy is a matter of conjecture. A familial incidence indicating a genetic component in the aetiology has been reported before (Kakar et al., 1966; De Santo and Schubert, 1969) but it is apparent from the published work that this condition is considered to be uncommon.

Case history

Miss L. M., a 44-year-old spinster of Mediterranean extraction, presented on 16 August 1977 with a rightsided Bell's palsy. She felt well and there had been no prodromal illness, but she was obviously distressed by her facial appearance. She was completely unable to close her right eye and there was a facial deformity typical of Bell's palsy (Miles Foxen, 1970). Physical examination revealed an obese woman with a blood pressure of 150/100. Urinalysis and haematological/biochemical profiles were normal. Examination showed that the ears, nose, and throat were normal, and in particular there were no herpetic lesions. The clinical findings were confirmed by an ENT consultant. An audiogram showed bilateral low-tone deafness.

She gave a strong family history of facial palsy in that her two siblings, mother, and maternal grandmother had had similar attacks in the past (Figure 1). Her brother had a right-sided facial palsy in 1976 when aged 30 from which he had completely recovered. Her sister had a right-sided facial palsy when aged 19 years and a left-sided facial palsy when aged 24. Her mother had a left-sided facial palsy in 1940 when aged 31 and com-

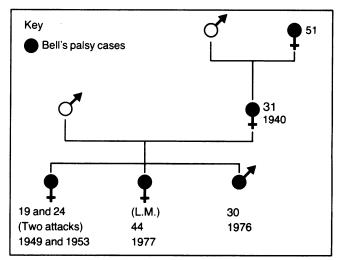


Figure 1. Family genealogy as presented by patient (L. M.). The figures indicate the age at the time of attack and the year.

pletely recovered. She died of a stroke in 1953. The patient's maternal grandmother had a facial palsy (side unknown) when aged 51. There was no family history of diabetes.

The patient made a 90 per cent recovery over a period of nine weeks whilst taking reducing courses of systemic steroids.

Discussion

Singh and Singh (1973) reported a family of facial palsy patients in whom there was a strong suggestion of infectious origin. The temporal and spatial separation of the present cases negate this possibility.

It has been suggested that the predisposition of families to facial palsy is dependent on an inherited peculiarity of the facial nerve or its course (Brackmann, 1974), or to a metabolic oddity of the histidine-histamine decarboxylase enzyme system (Williams, 1963). Such hypotheses have been impossible to prove. The present cases merely add weight to the 'genetic hypothesis'.

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Reference

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