Myasthenia gravis and breast carcinoma—a case report

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Summary

A case of true myasthenia gravis, involving brain stem innervated muscles, occurring in a patient with breast carcinoma is described. Treatment with cholinesterase inhibitors gave good sustained improvement despite dissemination of carcinoma.

Introduction

True myasthenia gravis, as distinct from the myasthenic syndrome (Eaton-Lambert syndrome), has not been described in association with neoplastic disease other than malignant thymoma (Simpson, 1958). A case of ocular/bulbar myasthenia gravis occurring in a patient with breast carcinoma is described in which the temporal relationship between the disorders is such that the association seems significant.

Case report

The 64-year-old female patient presented with a mass in the right breast and axillary lymphadenopathy. Simple mastectomy confirmed invasive ductal carcinoma with metastasis to axillary nodes. About 2 weeks after operation she rapidly developed multidirectional diplopia and slurring of speech with difficulty in chewing and swallowing. All symptoms were worse at the end of the day. Clinical examination at the time showed residual axillary lymphadenopathy. Ptosis and diplopia were marked. There was fatigue induced weakness of facial, masticatory and palatal muscles. Tone, power and co-ordination in limb and girdle muscles were normal. Tendon reflexes were well preserved and non-fatiguable. Sensation was normal. Intravenous edrophonium (10 mg) produced dramatic improvement in the affected muscles and this was not repeated after intravenous saline injection. Electromyogram showed no abnormality although bulbar innervated muscles could not be tested. Haemoglobin, white blood cell count, plasma electrolytes and calcium and thyroid function tests were normal. Anti-nuclear factor and thyroglobulin antibodies were detected in the patient's serum but screening for other serum antibodies (including to striated muscle) was negative.

T-cell mediated immunity as determined by antigenic skin tests and *in vitro* lymphocyte studies was normal. Myasthenic symptoms were well controlled by oral neostigmine (total of 120 mg daily) but, unfortunately, further dissemination of carcinoma occurred and no response was obtained with oestrogen and corticosteroid therapy. The patient died with a terminal pulmonary embolus just 3 months after onset of myasthenic symptoms. Permission for post-mortem examination could not be obtained.

Discussion

There seems little doubt that this patient had myasthenia gravis. Only brain stem innervated musculature was involved and no features of the myasthenic syndrome (Lambert *et al.*, 1965) were present. Proximal limb and girdle muscles remained normal clinically and electromyographically. Weakness of affected muscles showed dramatic and sustained response to anticholinesterase therapy. Serum anti-nuclear factor and thyroglobulin antibodies were detected, in keeping with the suggested multi-organ immunologic involvement in myasthenia gravis (Simpson, 1974). Unfortunately, the thymus gland could not be examined after death in search of histopathological changes of the type seen in myasthenia gravis. A trial of cholinesterase inhibitor therapy would seem worth while in any patient with malignant disease developing myasthenic symptoms.

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