PROXIMAL MYOPATHY AS A COMPLICATION OF MYXOEDEMA

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SYNOPSIS

Proximal myopathy has been well documented in several endocrine disorders, notably thyrotoxicosis and Cushing's syndrome. Its occurrence in myxoedema is extremely rare and it was first described by Kugelberg et al. in 1959 and later by Astrom et al (1961) and Nickel et al (1961). It bears no relation to the duration or severity of the primary disease. A similar case is being reported here.

CASE REPORT

A 44 year old Indian man was admitted to the University Hospital because of progressive weakness of the lower limbs over seven years. He initially noticed difficulty in walking up a mild gradient and later his weakness progressed to the point where he had to give up his job as a rubber tapper. At a later stage he could not get up from a squatting position unaided. Together with this, he was noted to be very lethargic and slept an average of fifteen hours a day. He moved his bowels only once in two days and passed out hard stools. Despite a poor appetite, he gained 12 kilograms in weight. He also complained of generalised bodyaches. On direct questioning he said that he preferred hot weather (unusual for this country) and had decreased sexual libido. His past history was unremarkable. There was no family history of thyroid disease.

Examination revealed a dull looking man, slow in thought and movement. His voice was hoarse and it was difficult to catch his words. He had a puffy face and his skin was coarse and thickened; over the back and legs, it was scaly and resembled ichthyosis. His blood pressure was 150/100 mmHg and his pulse rate was 60 per minute. He could only walk leaning heavily on a stick and could not rise unaided from a squatting position. Mild wasting of his thigh muscles was noted. Formal muslce charting revealed a typical proximal myopathy, the muscles of the pelvic girdle being of grade 2 to 3 power mainly. No weakness of the shoulder girdle or distal muscles was detected. His tendon reflexes had a markedly prolonged relaxation phase. The thyroid gland was not enlarged. The rest of the examination was essentially normal.

Initial investigations revealed a mild normocytic normochronic anaemia (Hb 10.8 g/dl). His creatine phosphokinase (CPK) level was 2,150 miu/ml and the serum cholesterol level was 7 mmol/L. His liver function tests, serum electrolytes, calcium, magnesium, phosphate and creatinine were all normal, as was a glucose tolerance test. The serum thyroxine level was less than 1.0 mg/100 ml and the Free

Thyroxine Index was less than 0.43. Thyroid microsomal antibodies were positive in one in a hundred dilution. An electrocardiogram showed generalised low voltages with a heart rate of 60 per minute. A muscle biopsy was done to exclude a myositis in view of the extremely high CPK level. Mucoid degeneration was seen in some muscle fibres, together with crowding of the nuclei in some areas. Some of the nuclei were rounded and larger than normal and were arranged in rows. There was no marked inflammatory response to suggest a myositis. An electromyogram showed only non-specific abnormalities. Nerve conduction studies revealed a bilateral carpal tunnel syndrome although this was not evident clinically. His chest and skull radiographs were normal.

After six weeks of replacement therapy (L-thyroxine 0.2 mg daily) there was subjective and objective improvement. The patient felt more alert. His facial puffiness decreased and his skin texture improved. His voice was less hoarse and his speech could easily be understood. He began to have a daily bowel movement and his appetite improved. He could now walk without a stick but still could not get up from a squatting position unaided. Formal muscle charting showed only slight improvement. A repeat CPK at this time was 155 miu/ml. Three months later, he could get up from a squatting position unaided but had still not returned to work. Fourteen months after therapy was started, he had improved further but had still not recovered full power in his lower limbs. Formal muscle charting showed that his proximal lower limb musculature was between grade 4 to 4+ in strength. His CPK was 69 miu/ml and his TSH was 11 miu/ml. He had given up the idea of returning to rubber tapping and was only doing a few odd jobs which did not require much walking.

COMMENT

This patient undoubtedly had myxoedema and his proximal myopathy appears to be causally linked to his hypothyroid state. The microscopic examination of the segment of muscle biopsied was compatible with the diagnosis of hypothyroid myopathy and significantly, his muscle power returned, although not completely, with treatment, and his CPK level fell at the same time. This case illustrates the fact that although many of the symptoms of hypothyroidism are relieved dramatically by replacement therapy, a myopathy may persist longer. A proximal myopathy, in particular, as noted by Astrom et al (1961) may not even respond completely to treatment as in the case reported. However, further improvement on longer follow up cannot be ruled out at this stage.

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