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Polyneuropathy in occult hypothyroidism

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Summary

A patient who developed symptoms and signs of a polyneuropathy was found to have hypothyroidism, though this diagnosis was not clinically suspected. Treatment with thyroxine resulted in resolution of his symptoms and restored his nerve conduction studies to normal.

KEY WORDS: polyneuropathy, hypothyroidism, axonal degeneration.

Introduction

The commonest involvement of peripheral nerve in hypothyroidism is thought to be entrapment of the median nerve at the wrist (Dyck and Lambert, 1970). Less commonly, patients with myxoedema may have a more diffuse polyneuropathy, though previously recorded cases have been in patients with clinical evidence of hypothyroidism. We wish to report a patient with hypothyroid polyneuropathy who was symptomatically and clinically euthyroid.

Case report

A 48-year-old man presented in July 1981 with a 6-month history of cramp in the lower limbs and of paraesthesiae in his feet and hands. He also complained of numbness over the dorsum of both feet and a painful dysaesthetic disturbance in both legs which made dressing uncomfortable. There were no complaints referable to the endocrine system and there was no family history of neurological disease.

General examination was normal with a heart rate of 75/min. Higher functions and cranial nerves were normal. Tone in the limbs was within normal limits and there was no weakness. Reflexes were preserved and normal in character, the plantar responses bilaterally flexor and no cerebellar deficit was evident. Sensory examination revealed impaired sensibility to light touch and pin prick as far as the mid tibial level in the legs and the wrist in both arms. Joint position sense was preserved. Investigations including full haematological and biochemical screening (including serum creatine-kinase activity) and chest radiography showed no abnormality. In particular, blood sugar, B_{12}, and protein electrophoresis were normal. Syphilitic serology was negative. Serum thyroxine was <20 nmol/litre (normal 60–140) and basal TSH >60 mu/litre (normal 0–6·0). Thyroglobulin and microsomal haemagglutination tests were both positive at 1 in 320 and 1 in 10 respectively. Nerve conduction studies revealed evidence of a mild sensory axonal neuropathy in the lower limbs (Table 1). Both tibialis anterior muscles were examined with a concentric needle and fibrillation potentials, fasciculations and positive sharp waves were observed. These features are evidence of denervation and indicate involvement of the motor axons as well. Examination of the cerebro-spinal fluid revealed a raised protein of 0·8 g/litre, but was otherwise normal. Treatment was commenced with thyroxine with complete resolution of his symptoms. Nerve conduction studies and electromyography were repeated 1 year later and were normal, except for a mild, persistent reduction in the sural sensory nerve action potential amplitude (Table 1).

Discussion

Disturbances of sensation are common symptoms in myxoedema and occur in about half the patients with established hypothyroidism (Crevasse and Logue, 1959). Although the frequency of these subjective symptoms implies a high incidence of associated neuropathy, there have been few series studied with detailed electrophysiology. Fincham and Case (1968) performed nerve conduction studies on 18 patients with myxoedema and found abnormalities of the sensory nerve action potential in all. Other authors reporting individual cases have also noted slowing of motor and sensory nerve conduction velocities (Dyck and Lambert, 1970; Shirabe *et al.*,

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TABLE 1. Nerve conduction studies in a patient with hypothyroidism (a) before and (b) after 1 years treatment

	MTL		MCV		SNAP Amp.		SCV	
	a	b	a	b	a	b	a	b
Median	3·7 (3·3±0·5)	3·1	55 (58·0±4·9)	59	12* (18·6±8·8)	18*	48* (56·2±6·8)	57*
Ulnar	3·1 (2·4±0·4)	2·5	51 (57·6±4·4)	60	9† (15·4±7·1)	11†	45† (55·1±5·7)	57†
Common peroneal	5·0 (3·9±0·5)	3·9	51 (51·3±3·8)	47	—	—	—	—
Posterior tibial	5·0 (4·0±0·5)	3·9	44 (52·1±5·2)	47	—	—	—	—
Sural	—	—	—	—	2·5 (18·0±5·0)	7	31 (45·9±5·8)	45

Abbreviations: MTL = motor terminal latency (m/s); MCU = motor conduction velocity (m/s); SNAP Amp. = sensory nerve action potential amplitude (μV); SCV = sensory conduction velocity (m/s).
*Digit II; †Digit V.
Figures in parentheses indicate mean normal value ±s.d.

1975; Pollard *et al.*, 1982). Histological examination of peripheral nerves in most of these patients has shown segmental demyelination and remyelination (Dyck and Lambert 1970; Shirabe *et al.*, 1975), although a more recent report suggested that the predominant abnormality was axonal degeneration (Pollard *et al.*, 1982). The electrophysiological abnormalities in our patient were mainly axonal and it may well be that this is the primary abnormality and that demyelination is a secondary phenomenon. Aetiologically, this would place hypothyroid polyneuropathy with other forms of toxic and metabolic neuropathies which are due to primary axonal degeneration.

Since our patient's symptoms resolved completely on treatment with thyroxine and his nerve conduction studies returned to normal, we have no doubt that his polyneuropathy was due to hypothyroidism. The development of a hypothyroid polyneuropathy in a patient who was clinically euthyroid is unusual and emphasizes the need to perform thyroid function tests when screening patients with undiagnosed neuropathies.

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