Psoriasis and Polyneuropathy

Three Case Histories

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Neurophysiological examination of 3 psoriasis patients with symptoms of polyneuropathy revealed varying degrees of both sensory and motor nerve affection and indicated nerve fibre loss as well as demyelination. Previous reports have suggested a connection between peripheral nerves and psoriasis. ***Key words: Demyelination; Nerve fibre loss.***

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The etiopathogenesis of psoriasis is unknown, though in the last decade, cell-mediated immunological mechanisms as well as biochemical abnormalities have been emphasized (1). Furthermore, there appears to be a neural influence on the skin lesion in psoriasis (2, 3).

We present 3 cases of polyneuropathy in selected psoriatic patients admitted to an out-patient clinic for neurophysiological examination.

MATERIAL AND METHODS

Nerve conduction velocities were determined, using orthodromic technique and needle electrodes (4, 5). Warm and cold detection limits were obtained with the use of a Marstock stimulator (6) and compared with detection limits in age-matched healthy subjects (own unpublished data).

REPORT OF CASES

Case 1

A 58-year-old woman with psoriasis since childhood without affection of the joints. Skin lesions were restricted to the extensor side of the elbows and the knees and the patient had received treatment with topical steroids.

For 2 years the patient had suffered bilateral lightning pains and paresthesia in glove and stocking area, with nightly aggravation. There was reduction of both motor and sensory nerve conduction (Table I), as well as low amplitude, polyphasic sensory action potentials. Temperature sensation was impaired on the feet.

Case 2

A 34-year-old woman with psoriasis since childhood and joint complaints during the last 10 years. Her plaque-type psoriasis had been treated with topical steroids and tar ointments. Psoriatic arthritis was treated with NSAID, though neither joint deformity nor tenderness had been present. At the time of investigation, psoriatic lesions were located over knee and elbow joints, on the face, the scalp, and inguinal region.

For 18 months the patient had complained of symmetric paresthesia and lightning as well as deep pain in the extremities. Neurophysiological examination revealed a minor reduction in some sensory conduction velocities, while there was a substantial reduction in motor conduction velocity in the ulnar nerve on the forearm (Table I). Sensory action potentials were of low amplitude and polyphasic. Temperature sensation was normal on the feet, but impaired on the wrists.

NSAID treatment was discontinued, as NSAIDs can produce peripheral neuropathy (7, 8). Two months later the symptoms of neuropathy had worsened. The patient refused a neurophysiological re-examination.

Case 3

A 35-year-old woman with psoriasis, mainly of the plaque type, for the past 20 years. In 1987, psoriatic arthritis was diagnosed. Various joints were affected, causing tenderness, but no deformity. From October 1987 through May 1988 the patient was given aurothiomalate intramuscularly to a total dose of 880 mg, but without clinical effect. Since July 1988, methotrexate had been given in weekly doses of 15 mg, although at irregular intervals, due to gastric complaints. The accumulated total dose was 150 mg, but the efficacy was doubtful. In addition, NSAIDs, paracetamol and dextropropoxiphene had been given. Skin changes had been treated with topical steroids and tar ointments.

Over 2–3 years the patient had developed symptoms of peripheral neuropathy. The patient complained of lightning and deep pain and paresthesia, primarily in the glove and stocking area. At the time of the investigation the patient had not received NSAIDs or methotrexate for several months. Results from the neurophysiological examination of the patient are detailed in Table I. Motor nerve function was almost normal, as only a slightly increased distal motor latency was present in the ulnar nerve. Sensory action potentials were polyphasic in the three nerves tested, but conduction velocity appeared only to be

Table I. *Sensory nerve conduction velocities (SNCV), distal motor latencies (DML), and motor nerve conduction velocities (MNCV)*

	Case 1	Case 2	Case 3
Ulnar nerve			
SNCV, 5th finger–wrist (m/s)	38 (47–65)	56 (46–68)	52 (46–68)
SNCV, wrist–prox. elbow (m/s)	51 (55–69)	56 (57–76)	65 (56–74)
DML, wrist–hypothenar (ms)	3.5 (2.1–3.2)	3.6 (1.9–3.0)	3.2 (1.9–3.1)
MNCV, prox. elbow–wrist (m/s)	52 (53–69)	47 (58–71)	65 (57–71)
Posterior tibial nerve			
SNCV, 1st toe–ankle (m/s)	30 (36–50)	37 (38–52)	41 (38–51)
DML, ankle-abd. hal. m. (ms)	5.0 (3.0–4.8)	5.0 (3.0–4.8)	4.8 (3.0–4.8)
Sural nerve			
SNCV, ankle–"surae" (m/s)	48 (46–60)	–	40 (47–61)

Numbers in parentheses are reference limits (5).

reduced in the sural nerve. Temperature sensation was unaffected.

The 3 patients denied alcohol abuse or exposure to neurotoxic chemicals and blood tests revealed serum glucose, creatinine, thyroid stimulating hormone, vitamin B_{12}, folic acid and gamma glutamyl transferase to be within reference limits.

COMMENT

Concomitant psoriasis and polyneuropathy have not been described previously. The possibility of a random coincidence cannot be ignored. However, our 3 patients were admitted for neurophysiological examination within 3 months. It has been reported that all cutaneous neural elements in patients with psoriasis are altered (2) and that there is a prompt remission of psoriatic plaques following cutaneous nerve sectioning (3). A recent theory indicates substance P as a possible link between the nervous system and the skin lesions (9, 10). Characteristic symptoms of polyneuropathy are a sensation of pain and paresthesia, both being transmitted to the spinal cord by type C nerve fibres. Pain sensation is mediated by substance P (11). Successful treatment of moderate and severe psoriasis with topically applied capsaicin has been reported (12). Capsaicin is an inhibitor of cutaneous vasodilation, probably acting through depletion of substance P from nerve terminals.

The possibility of drug-induced polyneuropathy exists in case 2, as cases of polyneuropathy attributable to naproxen and indomethacin have been reported (7, 8). However, the symptoms of polyneuropathy worsened after discontinuing the NSAID, minimizing the probability of this etiology of the peripheral nerve disease. In case 3, symptoms of neuropathy started before treatment with gold, which is a well-known inducer of neuropathy.

The results of the neurophysiological examination in these 3 cases of concomitant psoriasis and polyneuropathy represent a wide range of abnormalities. Disturbed function of both motor and sensory nerves was seen. Reduced maximum sensory or motor nerve conduction velocity and reduced amplitudes of sensory action potentials indicated both demyelination and axonopathy (nerve fibre loss) of the thick myelinated nerve fibres. Involvement of thin unmyelinated type C nerve fibres is indicated by impaired temperature sensation. In case 1 and 2, all these abnormalities are represented, whereas in case 3, temperature sensation was unaffected and only minor impairment of motor function occurred. These neurophysiological findings are similar to the findings in the majority of polyneuropathies, regardless of the etiology.

The nature of a possible causal relationship between psoriasis and polyneuropathy can of course not be judged from our data, but our case histories of concomitant psoriasis and polyneuropathy support the theory of a relationship between peripheral nerves and psoriasis.

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