Acta Derm Venereol (Stockh) 1996; 76: 52–54

Lack of Sympathetic Involvement in Dermatitis Confined to the Median Nerve Territory

A Case Report

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Both decreased and increased sympathetic nerve activity has been suggested as a possible underlying mechanism in inflammatory skin lesions. Modulation of sympathetic function has been proposed in the treatment of dermatitis. This case report describes the investigation strategy and normal findings in a case of dermatitis strictly confined to the median nerve territory, illustrating the need for specific tests of sympathetic function when pharmacological as well as physical sympatho-modulatory therapies are considered. ***Key words: skin sympathetic activity (SSA); microneurography; skin temperature.***

(Accepted August 15, 1995.)

Acta Derm Venereol (Stockh) 1996; 76: 52–54.

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Dermatitis confined to the median nerve territory has been described in association with local compression of the median nerve in the carpal tunnel and attributed to impaired function of sympathetic vasoconstrictor nerve fibres (1). On the other hand, Bjorna & Kaada (2) in 1987 suggested that increased sympathetic activity to skin may generate and/or maintain dermatitis. They demonstrated a beneficial effect of transcutaneous electrical nerve stimulation (TENS) on dermatitis, which was attributed to improved cutaneous microcirculation due to TENS-induced sympathetic inhibition (2). Thus, both decreased and increased sympathetic nerve activity has been suggested as a possible underlying mechanism for dermatitic lesions. Inflammatory skin lesions are also found in the chronic stage of the reflex sympathetic dystrophy syndrome (RSD), suggesting the possibility of neural involvement in localized dermatitis (3). Therapies modulating sympathetic function should then be considered in cases with localized dermatitis. However, given the complex relation between sympathetic nerve activity and skin sympathetic effector organ function, especially after nerve injury (4), such therapeutical decisions should be based on a thorough investigation of sympathetic neural function.

The present report illustrates clinical and neurophysiological investigation strategy and findings in a case of dermatitis localized within the innervation area of a peripheral nerve.

CASE REPORT

A 49-year-old man complained of spontaneously occurring recurrent eczematous dermatitis, mainly affecting digits I-III of the left hand, with a duration of 10 years. Physical examination showed hyperkeratotic and erosive eruption confined to the classical cutaneous territory of the median nerve, including the volar surface of digits I-III, a small part of the volar surface of the IVth digit, the thenar eminence and the skin area between the first and second digit (Fig. 1). Subjectively the patient did not report any abnormal sensation or pain from the affected region except a 'thick skin' feeling in the dermatitic area and transient pain in association with frequent skin cracks close to the finger joints. Direct microscopic examination and cultural procedure proved negative for mycosis. A skin biopsy specimen revealed a spongiotic dermatitis with a mild perivascular lymphomonocytic infiltrate. Laboratory investigations were within normal limits, IgE serum level was normal. The patients had no other systemic disease and no hemorrheological problem. One hyperglycemic episode was reported but no established diabetes mellitus or other metabolic diseases. Prick tests for common allergenes (pollen, inhalants and food allergens) and patch test with standard series (GIRDCA) were negative. Repeated trials with topical (corticosteroids, antibiotics, vitamin F, emollients) as well as systemic (steroids) therapies were unsuccessful in permanently relieving the dermatitis. TENS and regional sympathetic block with guanethidine (Bier's block technique) had also been performed, based on hypothetical sympathetical involvement, but did not improve the condition. Physical examination did not reveal any motor impairment of the muscles supplied by the median nerve in the left hand. Sensory testing with von Frey hairs revealed no negative or positive sensory abnormalities except a light increase in sensory perception threshold in hyperkeratotic skin areas. Tinel or Phalen signs of median nerve entrapment in the carpal tunnel could not be provoked. The left hand was consistently warmer than the right hand, also outside the skin area with dermatitic lesions, as shown by repeated thermographies (AGA Infrared Telethermographic

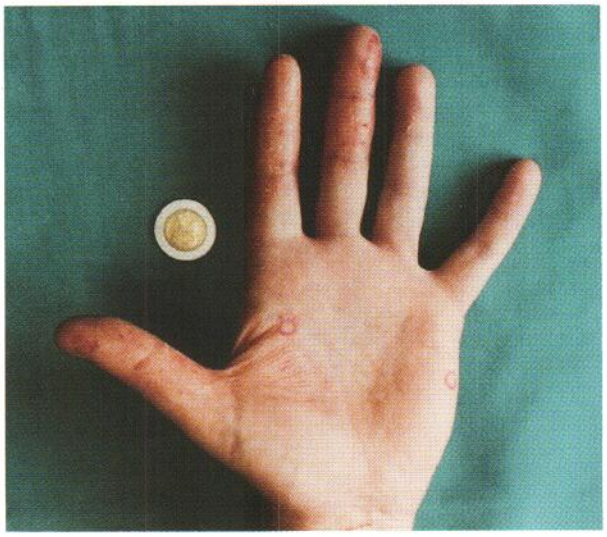

Fig. 1. Photograph showing the dermatitis distribution corresponding to the median innervation area of the left hand.

 ISSN 0001-5555

System) (Fig. 2). Neurophysiological testing was designed to investigate the following pathophysiological possibilities:

(a) entrapment of the median nerve in the carpal tunnel;
(b) subclinical polyneuropathy;
(c) regional neurovegetative disturbance.

a) Macroneurography in left median and ulnar nerves showed normal sensory and motor conduction velocities, distal latencies and response amplitudes (Table I). Electromyography in the median and ulnar muscles of the left hand was normal. b) In addition to a), macroneurography also included recording of motor and sensory conduction velocities in the lower limb which were normal (Table I). c) Indirect evaluation of sympathetic nerve function included recording of sympathetic skin response (SSR: skin potential changes indicating neurally mediated sweat production) (5) and fingertip photoplethysmography (PL; changes in capillary pulse volume indicating neural vasomotor function) (6). Direct record of multi-unit sensory afferent and postganglionic sympathetic nerve activity was performed (for details on methodology see Vallbo et al. (7)). Eight skin nerve fascicles with innervation territories which together covered the region of dermatitis were impaled (Fig. 3). In all fascicles normal sensory action potentials were recorded during tactile stimulation (i.e. stroking or stretching the skin within the receptive field) (Fig. 4). In three out of eight fascicles sympathetic activity was recorded, showing the normal characteristics of sympathetic discharges in skin nerves (Fig. 5). Stimuli known to transiently increase skin sympathetic neural discharge (i.e. arousal, mental stress, deep inspiratory gasps) induced normal sudomotor and vasomotor responses in the affected skin region (Fig. 5). Resting skin sympathetic activity was low, whereas inspiratory gasps or arousing stimuli (i.e. sudden noises or painful stimuli) evoked bursts of sympathetic discharge which were consistently followed by sudomotor and/or vasomotor reactions (cf. above).

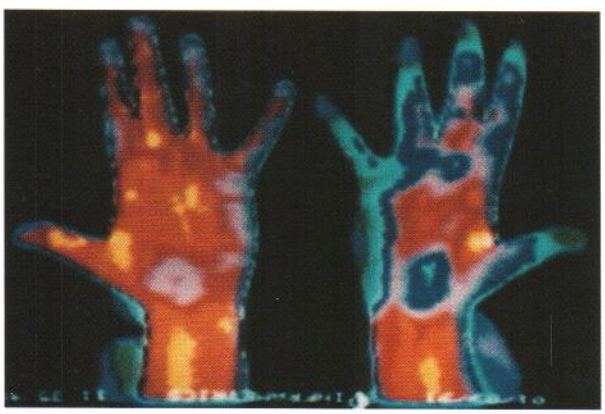

Fig. 2. Thermography showing the asymmetry in skin temperature between left and right hand (AGA Infrared Telethermovision System).

Table I. *The normal macroneurographic findings of sensory and motor conduction velocities recorded in the upper (median, ulnar) and lower (peroneal, sural) left limbs*
Distal latency is measured in milliseconds (msec). Maximum conduction velocity is measured as meters per seconds (m/s). The amplitude of the evoked responses has been reported in mVolt for motor responses.

Nerves		Dist. Lat. (msec)	Ampl. (uV/mV)	Cond. Vel. (m/s)
Median	motor	3.5	12.8	56.3
	sensory	3.1	9.5	41.9
Ulnar	motor	2.7	16.4	53.1
	sensory	2.4	7.7	43.7
Peroneal	motor	3.5	11.6	46
Sural	sensory	4	15	36.2

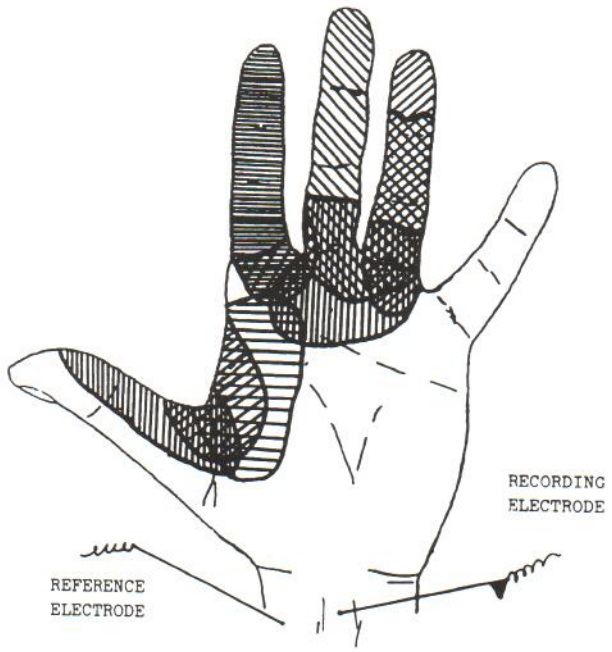

Fig. 3. Receptive fields of multi-unit sensory afferents recorded in eight fascicles of the left median nerve.

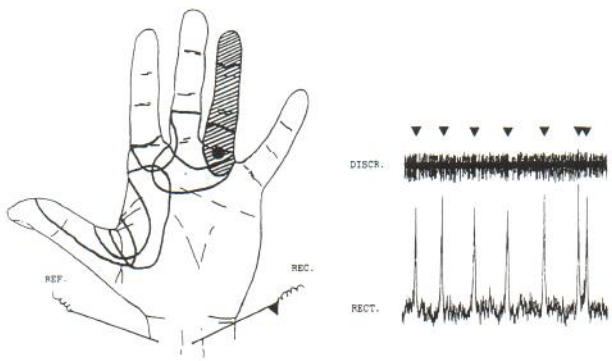

Fig. 4. Record showing a spontaneously active sensory afferent fibre responding to slight stretching of the skin (▼) in the area indicated by ●. The original nerve signal was fed through a band pass filter with a band width of 700–2000 Hz (Discr.). A mean voltage neurogram was obtained by passing the filtered neurogram through an RC integrating network with a time constant of 0.1 s (Rect.). The hatched area indicates the multi-unit sensory receptive field of the impaled fascicle.

DISCUSSION

Although the distribution of the dermatitis suggests a neurological involvement, the normal findings with macroneurography and electromyography make a local compression of the median nerve, and/or a subclinical polyneuropathy affecting large diameter myelinated nerve fibres, highly improbable. Macroneurography does not evaluate the function of thin unmyelinated (i.e. sympathetic) nerve fibres, and since experimental models of neuropathy have shown complete loss of sympathetic fibres also after mild compression of a peripheral

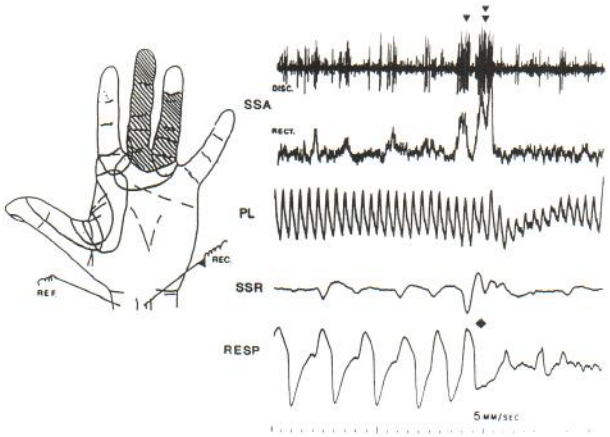

Fig. 5. Record showing spontaneous and stimulus-evoked bursts of skin sympathetic activity (SSA) and corresponding sudomotor (SSR) and vasomotor (PL) reactions. (▼) indicates demand for and (▾) the performance of a deep inspiratory gasp (◆). The hatched area indicates the multi-unit sensory receptive field of the impaled fascicle.

nerve (4), the normal macroneurographic findings do not rule out the possibility of sympathetic neural dysfunction. Hence the need for a more direct evaluation of sympathetic function. Although the degree of skin sympathetic nerve activity is difficult to quantify, due to the constantly changing influence from thermoregulatory and mentally arousing factors, the fact that skin sympathetic nerve activity was easily found and showed normal characteristics, including a low level of resting discharge, speaks against a sympathetic hypo- or hyperactivity in the left arm. A putative sympathetic dysfunction distal to the carpal tunnel could not be evaluated by direct recording since this was performed proximal to the wrist. However, the finding that not only strong stimulus-evoked bursts of sympathetic discharge but also weak spontaneous bursts were consistently followed by SSR and/or PL amplitude reductions indicates functioning distal sympathetic fibres and a normal neuroeffector transfer in the median area. Thus the alteration of skin temperature in the left hand is in all probability locally generated, possibly by the dermatitis per se, and not caused by a change in sympathetic neural control. Sympathetic involvement was clinically suspected in the present case because of markedly increased skin temperature in the left hand, also in regions not affected by the dermatitis. In RSD and related syndromes regional changes of skin temperature in the affected region is a common finding and generally considered to indicate alterations in sympathetic neural activity (8). However, animal studies indicate that the relation between sympathetic nerve activity and skin temperature abnormalities in experimental neuropathy is complex and that for instance skin vasoconstriction may be marked despite complete lack of sympathetic innervation (4).

Despite this controversy, regional sympathetic blocks are often performed on patients with pain syndromes associated with regional changes in skin temperature, also regardless of the direction of temperature change (cf. ref. 9). Since inflammatory skin lesions can occur in RSD and a sympathetic involvement was clinically suspected in the present case, both TENS and sympathetic blockade were performed in analogy with the traditional treatment strategy of RSD, both without beneficial effects and thus supporting a lack of sympathetic involvement in skin temperature changes found in this case. In summary, the present case did not show any sign of sympathetic or other nerve fibre dysfunction, arguing against the use of pharmacological or physical treatments modulating sympathetic function. The findings also underline the fact that regional alterations in skin temperature cannot be taken as evidence for altered sympathetic neural drive. Thus more specific tests of sympathetic function are necessary when planning therapeutic strategies in patients with localized pain, itch or other dysfunction such as dermatitis where a neurological disorder can be suspected.

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