Reflex sympathetic dystrophy of the upper extremity: a new diagnostic approach using Flexi-Therm liquid crystal contact thermography

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A case of reflex sympathetic dystrophy syndrome (RSDA), diagnosed by liquid crystal contact thermography (LCT) is presented. The pathogenesis, diagnosis and treatment of the syndrome are reviewed having regard to liquid crystal thermography.

KEY WORDS: sympathetic dystrophy, thermography, chiropractic, manipulation.

Un cas de syndrome dystrophique sympatique réflexe (SDSR), diagnostiqué par thermographie de contact par cristaux liquides (TCL) est présenté. La pathogénèse, le diagnostic, et le traitement du syndrome sont revus, en vue de la thermographie par cristaux liquides.

MOTS CLÉS: dystrophie sympatique, thermographie, chiropractique, manipulation.

Introduction

The reflex sympathetic dystrophy syndrome (RSDS) is the current diagnostic term applied to a constellation of signs and symptoms in one or more extremities due to sympathetic dysfunction.[1,2] This term includes shoulder-hand syndrome, Sudeck's atrophy, causalgia, algodystrophy, post-traumatic dystrophy/pain/edema, and other related diagnoses.[3-5] Recently there seems to be an increasing interest in the diagnostic[2,5,6,7] and therapeutic[1,3,4,8,9] dilemma of RSDS. Though it was first recognized as an entity in wounded soldiers more than a century ago, the diagnosis of RSDS remains elusive, to the point of its frequent misdiagnosis as a psychiatric disorder.[5,6] The key features of RSDS include:

Pain

Though its intensity and duration are variable, the pain of RSDS is characteristically severe and unremitting for prolonged periods of time. Its frequently burning nature spawned the early diagnostic term, causalgia. Alternately, the pain may be sharp, lancinating, or a dull, throbbing ache.

Hyperpathia (hyperalgesia and hyperesthesia) is associated with the pain of RSDS. The slightest sensory or emotional stimulation may cause excruciating exacerbation of pain. The patient's universe consists of his pain, which affects every aspect of his life. Psychological and behavioural problems frequently result, with some patients being driven to suicide.

Since even dependence of the extremity or a gust of air can exacerbate pain, the only (non-therapeutic) relieving factors are isolation and immobility of the affected extremity, and application of wet cloths.

Swelling

The affected extremity may be constantly swollen. Alternately, swelling may be induced or increased by motion of the extremity or even by atmospheric changes. In association with the swelling, the overlying skin may become cyanotic. Additionally, the (manual) interphalangeal joints become swollen.

Autonomic Instability

Dramatic changes in skin colour (varying from erythema to mottling to cyanosis) and temperature (often cooler, but at times warmer, than the unaffected extremity), and in sudomotor activity (varying from anhidrosis to hyperhidrosis) are often experienced during different stages[3] of RSDS. Such signs of vasomotor and sudomotor instability indicated the potential for sympathetic nervous system dysfunction to early investigators. Decreased vascular pulsatility in the affected region has been interpreted as indicating that arterial spasm is involved in the generation of RSDS.[6]

Trophic Changes

All tissues comprising the affected extremity atrophy if successful therapy is not instituted at an early stage of the reflex sympathetic dystrophy. The skin becomes hairless, tight, shiny and pale or cyanotic. The subcutaneous swelling progresses from a pitting to a brawny edema. The loss of fat pads causes marked tapering of the digits. The nails become brittle and grooved.

The muscles atrophy from neurovascular dysfunction and disuse. Marked weakness and contractures develop. Sudeck's atrophy (regional osteoporosis) of the bone becomes evident on roentgenographic evaluation. Even the intra-articular synovial tissue shows signs of atrophy and chronic inflammation.[1]

Stiffness

Joint stiffness develops because of the trophic changes affecting all of the peri- and intra- articular structures, and because of the painful limitation of all motion of the involved extremity. Bony ankylosis has been known to result.

Precipitating Event

Historically, trauma to the distal or proximal part of the affected extremity – even if trivial in nature – has figured prominently in the pathogenesis of RSDS. Other causes include ischemic heart disease, stroke, thrombophlebitis, cervical spondylosis, infection, carcinoma, and neuropathy (eg, disc prolapse, herpes zoster). However, RSDS has been diagnosed in the absence of a precipitating event in the patient's history.

Thermography is a non-irradiating diagnostic imaging method which yields a "heat map" of the body's surface. In turn, this map of the skin's infrared emissions can be interpreted to indicate the presence or absence of peripheral neurovascular disturbances, more so those associated with sensory nerve dysfunction.[10] Recently, liquid crystal contact thermography (LCT) was found to be a highly sensitive evaluator of back

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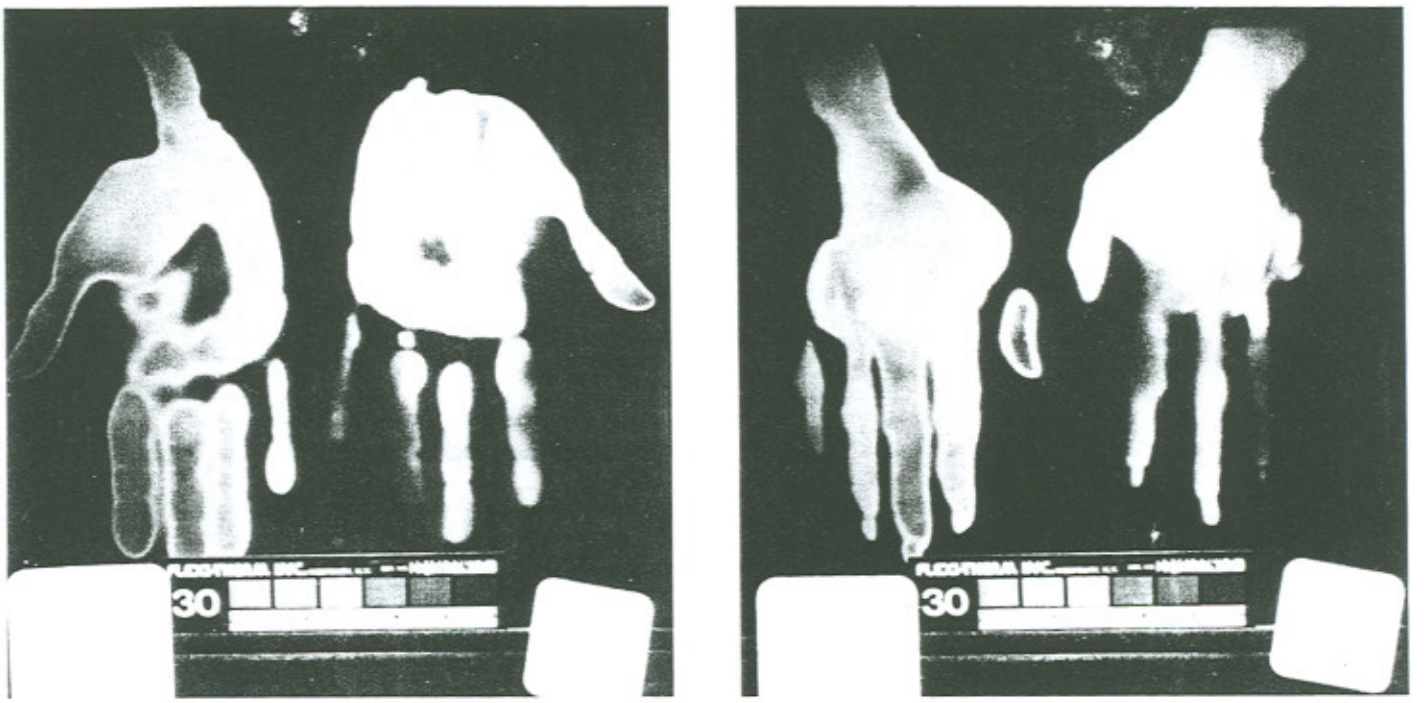

A B

Figure 1: Thermographs of patient's a) palmar, b) dorsal hands. There is a relative decrease of 2.5–3.5°C in the vascular heat emission pattern of the left hand with respect to the right. Figures are black and white photographs of original colour prints.

pain which was better correlated with more established functional (clinical neurological examination, electromyography) rather than structural (myelography, computerized tomography) diagnostic modalities.[1]

The following case report reflects the growing popularity of LCT in clinical diagnosis – especially in RSDS – and demonstrates the potential for effective conservative therapy.

Case Report

R.E. is a 42-year-old woman who was involved in a motor vehicle accident in which her vehicle was hit with great force from behind. Her left lower arm was severely bruised and several hours later she began to experience neck pain. She has continued to experience left-sided neck, occipital and left shoulder pain ever since, in spite of multiple injections of cortisone into the shoulder and considerable physiotherapy. Sensory, motor and reflex examination was unremarkable. X-rays and CT scan of the cervical spine were normal.

Flexi-therm®* liquid crystal thermography of hands and forearms revealed a 2.5-3.5° temperature decrease of the left hand and distal forearm (Figure 1). The non-dermatomal pattern was consistent with RSDS.

*Flexi-Therm Inc., Westbury, N.Y.

Discussion

Pathogenesis

It is beyond the scope of this paper to detail the various hypotheses of the pathogenesis of RSDS, which have been reviewed elsewhere.[3,6,8] Given the enigmatic nature of RSDS from both the diagnostic and therapeutic points of view, one may appreciate the difficulty investigators have experienced in agreeing upon its origins. However, the main mechanisms that have been considered, following a traumatic injury of an extremity, include:

1 consequent local inflammation and ischemia result in increased sensitivity of the peripheral vasculature to norepinephrine (both circulating and from sympathetic innervation), resulting in further ischemia (and pain, trophic changes), etc.;
2 consequent increased sensory nerve activity results in aberrant internuncial pool activity, with consequent aberrant increase in efferent sympathetic and somato-motor nerve activity, thus increasing local vasospasm, ischemia, pain, etc.;
3 formation of ephapses (artificial synapses) between the injured regional sensory and efferent sympathetic nerves; i.e., a vicious, self-perpetuating short-circuit.

Predisposing factors which may determine whether trauma induces RSDS in any given patient include subsequent disuse

and immobility; psychological and behavioural factors (e.g., pain avoidance); and physiological factors (e.g., plasminogen activator levels). The latter factor figures prominently in Ecker's[12] recent hypothesis of RSDS pathogenesis, which proposes that RSDS is the analogous result in the upper extremity of the mechanism of development of deep vein thrombosis in the lower extremity.

Diagnosis

Before the advent of LCT, the diagnosis of RSDS was based on clinical, radiological and therapeutic modalities. Clinically, the history, symptomatology, and presence of trophic changes, hypomobility and signs of autonomic instability in an affected extremity could lead to the correct diagnosis (assuming the clinician's awareness of the entity of RSDS). However, there are many possible precipitating events which, if obtained in the patient's history, may not be obviously related to the presenting complaint. Furthermore, the variable nature of the signs and symptoms during the earlier stages of RSDS (when treatment is most effective) may obscure the correct diagnosis.

Part of the difficulty in diagnosing RSDS arises from the overlap between its clinical signs and symptoms and those of peripheral vascular disease, cervicogenic brachialgias, peripheral entrapment neuropathies, and psychogenic pain syndromes. Thus ancillary diagnostic tests, such as radiological examination of the extremities or scintigraphy,[2] which could help to diagnose RSDS may not be considered. To date, the pathognomonic test for RSDS has been the dramatic relief of symptomatology following a therapeutic trial of sympathetic ganglion block or sympathectomy, even if the relief is of short duration.

Because it is sensitive, non-invasive and harmless, LCT is a diagnostic modality whose use at an early stage in the sequence of a diagnostic work-up may obviate the need for more invasive procedures while facilitating the accurate diagnosis of RSDS. The diagnosis of RSDS by LCT is based upon a non-neuroanatomical pattern of temperature differences of 1°C or greater between the affected region of an extremity, its contralateral analogous part, and the more proximal, asymptomatic region of the ipsilateral limb. However, there appears to be no correlation between the magnitude or direction (i.e., warmer or cooler) of the temperature difference and the duration or severity of symptomatology.[6] The pattern of temperature change in RSDS may reflect "vascular zones".[13]

Treatment

All investigators agree that the best prognosis in RSDS is predicated on accurate diagnosis and prompt institution of therapy. The fundamental principles underlying medical and surgical treatment are the control of pain and inflammation and the complete elimination of sympathetic innervation of the affected extremity. Physiotherapy has been considered indispensible for restoring and maintaining optimal flexibility and strength (i.e., deep massage, passive and active exercise).[4,8] TENS is gaining respect as an effective modality for pain control in RSDS.[5,9] Varying rates of success have been reported.[3,4,8]

As outlined above, one prominent hypothesis of the pathogenesis of RSDS involves a somato-visceral (i.e., sensory nerve – vascular sympathetic innervation) reflex mediated by aberrant internuncial pool activity, or a central excitatory state.[14] Chiropractors and osteopaths have long maintained that, by interrupting the vicious cycle between pain and muscle spasm associated with spinal motion segment dyskinesia, spinal manipulation is an effective therapy for dysfunction arising from a central excitatory state at the spinal cord level.[14,15] Thus, RSDS appears to be an ideal clinical entity for investigating the rationale for spinal manipulation.

Summary

Though occurring in a minority of cases of traumatic injury of the (upper) extremities,[12] RSDS is not as rare as previously thought.[16] The relatively recent use of liquid crystal contact thermography in its diagnosis is partly responsible for the increased awareness of this clinical entity, and may become an indispensible diagnostic modality in such cases. There may be a great potential for chiropractic treatment of this severe, debilitating condition.

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