Segmental neuromyotherapy: a novel system for diagnosis of pain at the foot and ankle

Neuromioterapia segmentar: um novo modelo para o diagnóstico da dor no pé e no tornozelo

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Abstract

Many mechanisms may contribute to the pathogenesis of chronic pain at the foot and the ankle. Activation of nociceptive nerve endings by mechanical or chemical painful stimuli are local factors that can produce significant neurochemical changes and reorganizations within the dorsal horn of the spinal cord. These changes include an increased excitability of dorsal horn neurons which produces pain hypersensitivity and allodynia, that may follow a segmental distribution. Improved diagnosis techniques allow the diagnosis of spinal segmental sentitization (SSS). Once these clinical findings are present, the rationale for treatment should also target desensitization maneuvers to control peripheral and central sensitization.

Keywords: Pain/diagnosis; Pain/physiopathology; Foot diseases

Resumo

Vários mecanismos podem contribuir para a patogênese da dor crônica no pé e no tornozelo. Estímulos dolorosos, de etiologia mecânica ou química, são fatores locais que ativam terminações nervosas livres nociceptivas capazes de produzir alterações neuroquímicas significantes e novas organizações no corno posterior da medula espinal. As alterações incluem o aumento da excitabilidade dos neurônios do corno posterior da medula espinal que produz hipersensibilidade dolorosa e alodínea com padrão de distribuição segmentar. Técnicas diagnósticas melhoradas permitem o diagnóstico da sensibilização segmentar espinal. Quando estes achados clínicos estiverem presentes, o tratamento da dor deve também contemplar manobras desensibilizadoras para controlar a sensibilização periférica e central.

Descritores: Dor/diagnóstico; Dor/physiopathology; Doenças do pé

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INTRODUCTION

The Joint Commission on Accreditation of Healthcare Organizations (JCAHO) requires that physicians consider pain as "the fifth vital sign", together with temperature, respiration, pulse, and blood pressure[1]. According to the standard conventional assessment and management of pain is considered difficult because of its subjective nature influenced by emotional, cognitive and psychosocial factors[1]. It is necessary to understand its pathophysiological basis and to introduce more objective and quantitative ways to diagnose and to better manage it. The International Association for the Study of Pain (IASP) defines pain as an unpleasant sensation, which is associated with tissue damage[2]. Pain is usually classified as nociceptive, neuropathic, mixed or psychogenic[2].

Contemporary pain management has shifted from symptom control to a mechanism based approach[3], associated with improvement of function and health-related quality of life with avoidance, if possible, of therapeutic toxicity. This paper is designed to provide readers with experience in novel and improved clinical diagnostic assessment of pain of different etiologies in patients with foot and ankle diseases. This approach is of particular value, when pain persists after the possible mechanical and anatomical causes of pain were ruled out and already taken care of.

Pathophysiology of pain

The clear understanding of the complex mechanisms involved in pain generation, modulation, amplification and perpetuation plays a critical role in any comprehensive pain control program. Recently, it has been recognized that constant and intense nociceptive sensory information generated by painful and inflamed deep somatic structure produces significant neurochemical and metabolic activity changes and reorganizations within the spinal cord segments[4,5]. These changes include an increased excitability of dorsal horn neurons, which produces pain hypersensitivity in a segmental distribution[4,6-10]. Diagnosis of central and peripheral sensitization (Table 1) is very important, because spinal cord neurons, that normally would only be activated by noxious stimuli, are now activated by normally non-noxious stimuli (allodynia)[9]. Together, these neurochemical changes suggest that pain induces and is partially maintained by the state of central sensitization[10-11], in which an increased transmission of nociceptive information allows normally non-noxious input to be amplified and perceived as noxious stimuli. Once these complex mechanisms are present, the rationale for treatment approaches should also target to promote desensitization mechanisms. It is important that these spinal cord changes may sometimes not be attenuated by blocking the original tissue damage and pain[5]. Spinal segmental sensitization (SSS) is a hyperactive state of the spinal cord caused by irritative foci sending nociceptive impulses from a sensitized damaged tissue to dorsal horn neurons[6-8].

Table 1 - Diagnosis of peripheral and segmental (central) sensitization.

Findings Methods	Quantification
A. Peripheral sensitization	
1. Hyperalgesia of skin: sharper feeling on scratch and pinch and roll 2. Tenderness: decreased pressure pain threshold: a. skin b. subcutaneous c. deep tissues, muscles 3. Micro-tropho edema: indentation by nail persists	Algometer (Figure 3)
4. Subcutaneous edema: thicker skin fold	Caliper, ultrasound
5. Electric skin conductance: increased	Micro amp meter
B. Segmental (central) sensitization	
1. Dermatomal distribution of peripheral sensitization findings	See part A
2. Temporal summation of repetitive stimuli Pin prick or electric stimuli repeated 1 per second causes gradually increasing pain reaction	Pinprick or electrical stimulation
3. Myotomal distribution of muscle sensitization: a. tender spots/trigger points b. taut bands c. muscle spasm (tender hypertonicity)	Algometer (Figure 4) Tissue compliance meter
4. Sclerotomal inflammation: a. enthesopathy at insertions of taut bands b. tendonitis, bursitis, epicondylitis c. joint changes, capsulitis d. ligament tenderness	Algometer
5. Viscerotome: segmental organ irritation and dysfunction	Palpation/auscultation of target organs
6. Spatial irradiation: spreading of segmental sensitization proximally and distally	All of the above tools are used to identify spinal spinal segmental sensitization

The clinical manifestation of dorsal horn sensitization includes hyperalgesia or allodynia of the dermatome, pressure pain sensitivity of the sclerotome and myofascial trigger points within the myotomes, which are supplied by the sensitized spinal segment[6-8]. Hyperalgesia is increased sensitivity to noxious stimulation. Allodynia is pain due to a stimulus that does not normally provoke pain. Detailed physical examination of patients with plantar fasciitis (Figure 1), for example, revealed tender areas over the calf muscles, especially in the proximal portion of the medial gastrocnemius muscle (Figure 2) in all evaluated patients[12-13], when compared to the contralateral uninvolved muscle. Plantar fasciitis patients present a lower pressure threshold[13] at the origin of the plantar fascia (Figure 3) and over the medial gastrocnemius muscles (Figure 4). In previous studies, we could speed up recovery, providing patients relief of pain and gain in function 80% faster than the conventional treatment by using needling and infiltration of the myofascial trigger point at the proximal portion of the medial gastrocnemius muscle with local anesthetics[12-14]. Results were compared to a control group of patients who received conventional treatment performed with ultrasound and electrical stimulation at the origin of the plantar fascia, followed by stretching exercises of the gastrocnemius muscle and plantar fascia[12-14]. Shah et al.[15] found that active myofascial trigger points present lower pressure pain threshold when compared to people with no pain or the presence of only latent trigger points. They also demonstrated the distinct *in-vivo* biochemical milieu of muscle with significant elevated levels of substance P, calcitonin gene-related peptide (CGRP), bradykinin, tumor necrosis factor-α (TNF-α) and interleukin-1β (IL-1β), serotonin, and norepinephrine in the vicinity of the active myofascial trigger point at the upper trapezius muscle, when compared to patients with latent or no myofascial trigger points. Overall, pH was significantly lower in the active trigger point. Treatment rationale and techniques may evolve from this information and should be taken into account, when dealing with chronic patients with amplified pain responses.

Failure to recognize and diagnose the peripheral and central sensitization of the dorsal horn neurons in patients with foot and ankle pain may lead to transient and temporary analgesic effects rather than long-term relief[6-8], since spinal neuronal plasticity is shown to be a key contributor to pathologic pain hypersensitivity[3]. Pain treatment strategies should target both the disease and the injury process in the periphery and the changes it induces or triggers in the central nervous system[16]. Prevention of central sensitization will eliminate the hyperalgesia and allodynia that are so distressing to some patients[16].

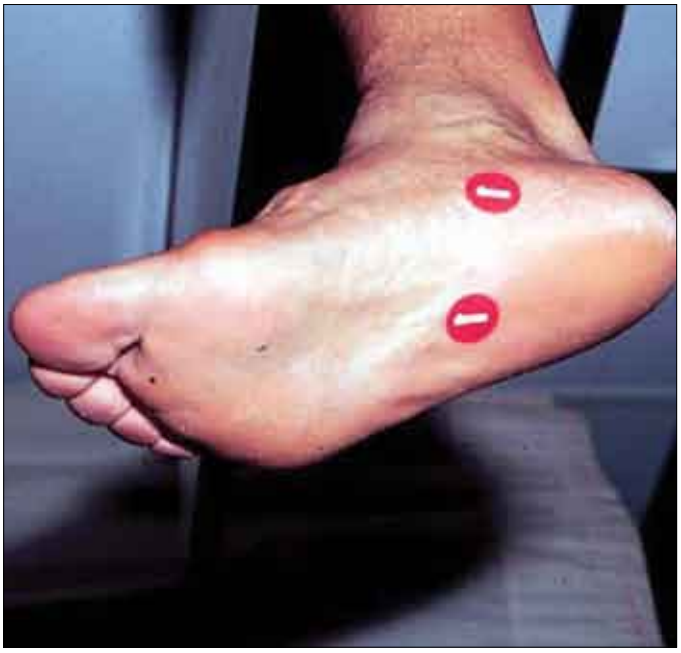

Figure 1 - Point of maximum tenderness in patient with plantar fasciitis at the medial tubercle of the calcaneous.

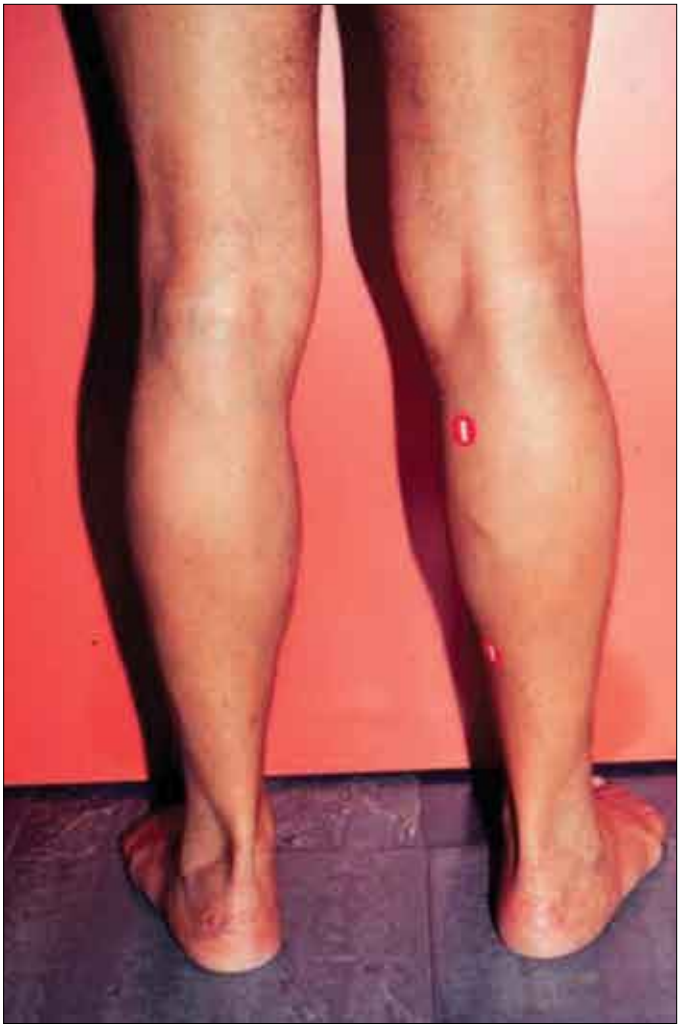

Figure 2 - Point of tenderness at the proximal portion of the medial gastrocnemius muscle.

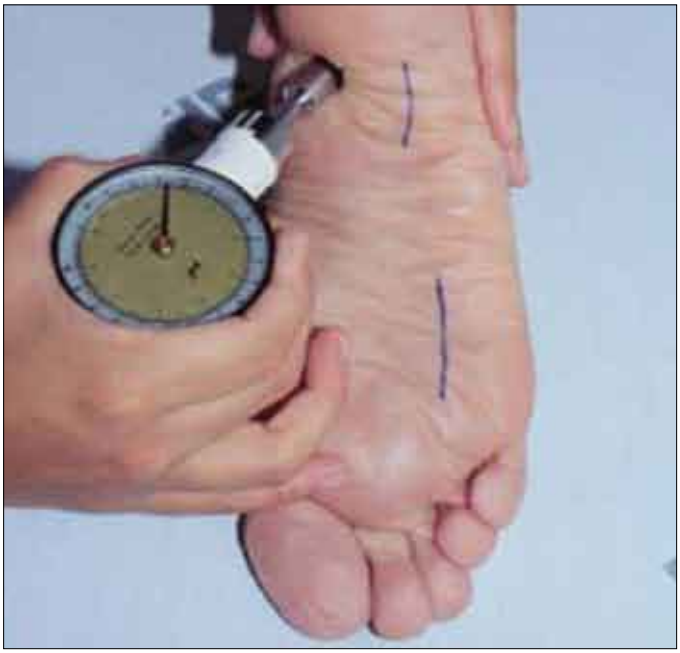

Figure 3 - Measurement of the pressure pain threshold at the origin of the plantar fascia using a pressure algometer.

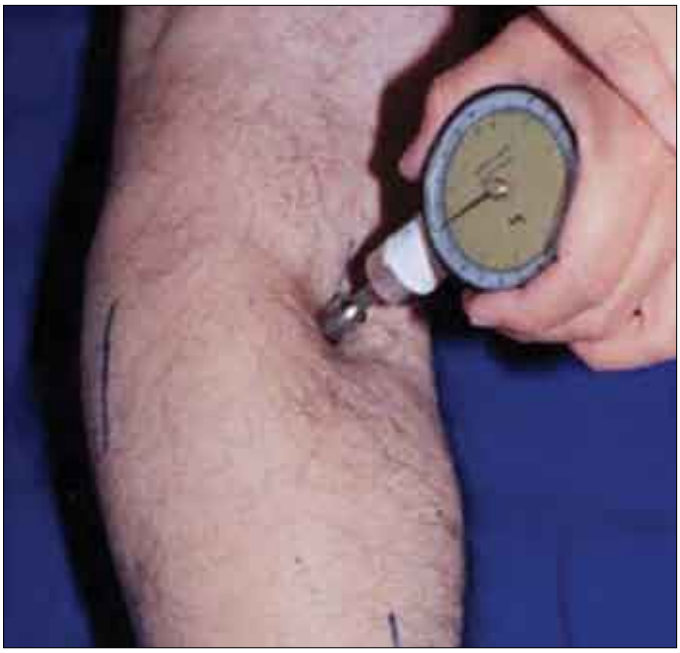

Figure 4 - Measurement of the pressure pain threshold over the medial gastrocnemius muscles using a pressure algometer.

Algorithm for diagnosis of foot and ankle pain based on spinal segmental sensitization (segmental neuromyotherapy) – Fischer[6-8]

1. Identification of the immediate cause of pain:
   A. Ask the patient to point with one finger where the most intensive pain is
   B. Find the point of maximum tenderness
   C. Reproduction (recognition) of pain: press over the maximum tender point and ask: is this the pain you are complaining about?
   D. Quantify the tenderness (degree of sensitization) by algometer
2. Diagnosis of sensitized spinal segment (SSS):
   A. Dermatomal Hyperalgesia (Keegan, Garrett's dermatonal chart)[17], (Figures 5, 6)
      1. Pain diagram
      2. Paper clip scratch test (use of sensory diagnostic tracks[6,8])
      3. Subcutaneous pinch and roll: test sensitization of subcutaneous tissue[6-8] (Figures 7, 8). After this maneuver, one can note the area of hyperemia that can be observed only at the sensitized level (Figure 9)
      4. Electric skin conductance: objective quantitative testing[6-8]

   Sclerotomal hyperalgesia (Figure 10):
      1. Palpation for tenderness of supraspinous/interspinous ligaments
      2. Palpation for tender spots (TsP) and MTrPs at at tachment sites, and enthesopathies

   B. Myotomal distribution of:
      1. Trigger points/tender spots within taut bands by palpation and algometry[7,18,19]
      2. Taut bands by palpation and tissue compliance meter which renders quantified, objective results
      3. Muscle spasm/reduced stretch range by palpation

   C. Sympathetic hyperactivity[6,8]:
      1. Microedema (Figure 11)
      2. Increased electrical skin resistance
      3. Orange peel skin
3. Diagnosis and removal of perpetuating factors:

   Physical examination reveals:
      1. Mechanical overload of body parts, overuse, obesity
      2. Deficiency of muscle function (loss of flexibility, weakness)
      3. Postural deficiencies such as loss of lumbar lor dosis (Robin McKenzie)

   Laboratory results:
      1. Endocrine disorders, particularly, low thyroid or estrogen supply to the muscles (normal blood levels are sometimes insufficient)
      2. Metabolic, rheumatologic or electrolyte disorders
      3. Vitamin deficiencies

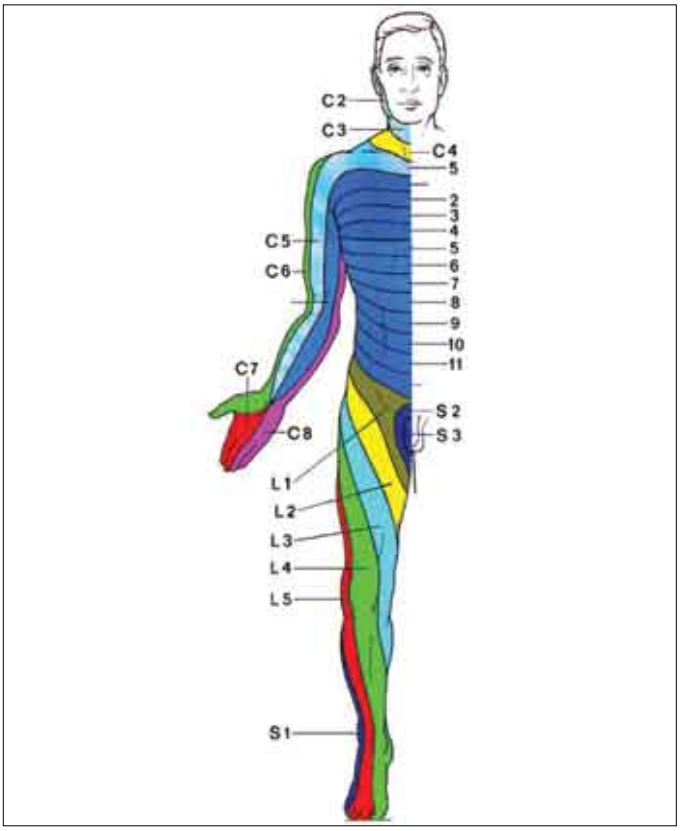

Figure 5 - Graphic representation of the anterior view of the dermatomal chart described by Keegan and Garret.

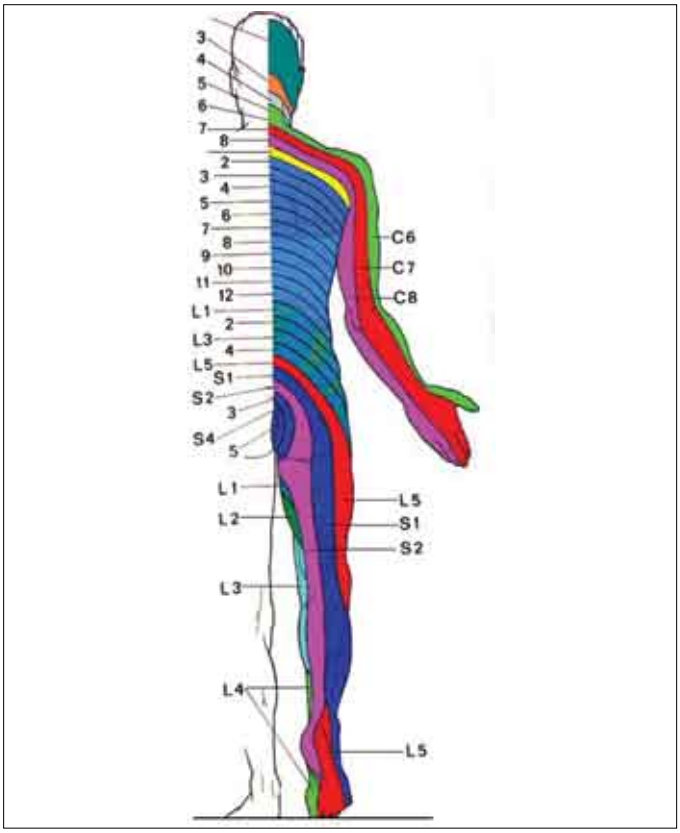

Figure 6 - Graphic representation of the posterior view of the dermatomal chart described by Keegan and Garret.

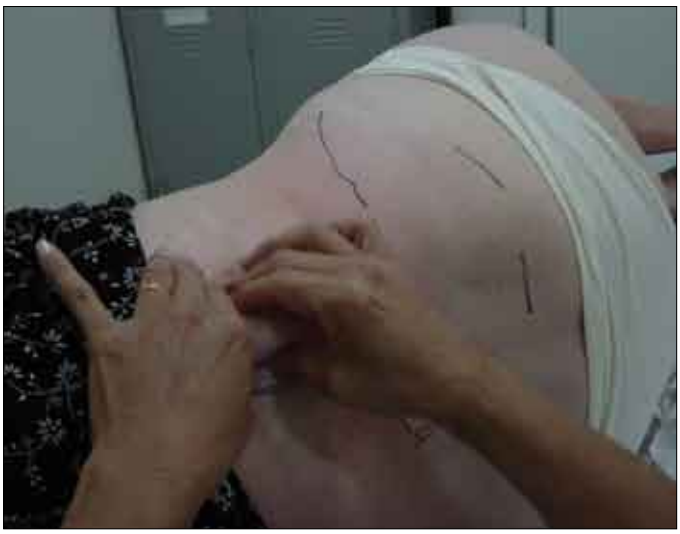

Figure 7 - Subcutaneous pinch and roll maneuver at L1 level.

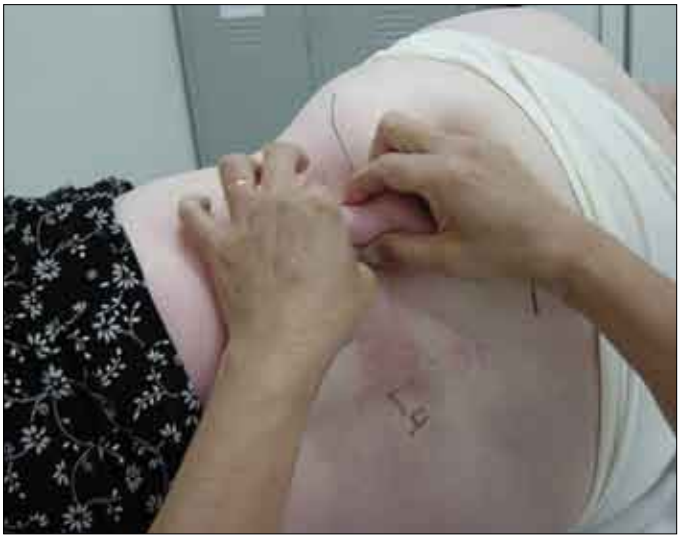

Figure 8 - Subcutaneous pinch and roll maneuver at L4 level.

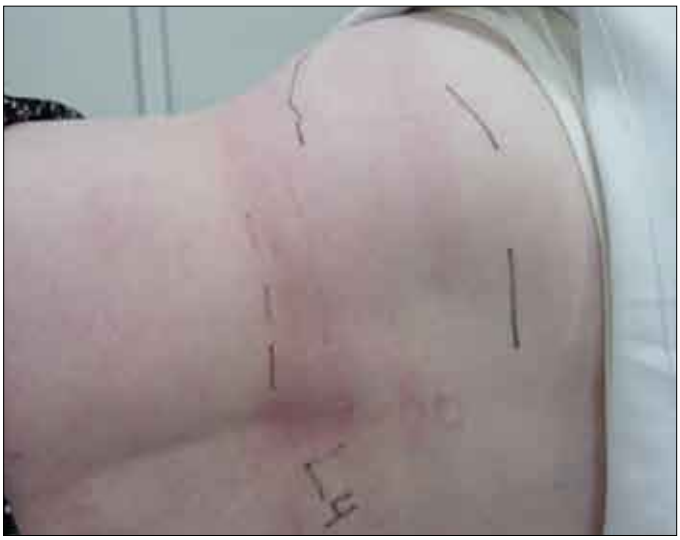

Figure 9 - Skin hyperemia at L4 spinal level after the pinch and roll maneuver.

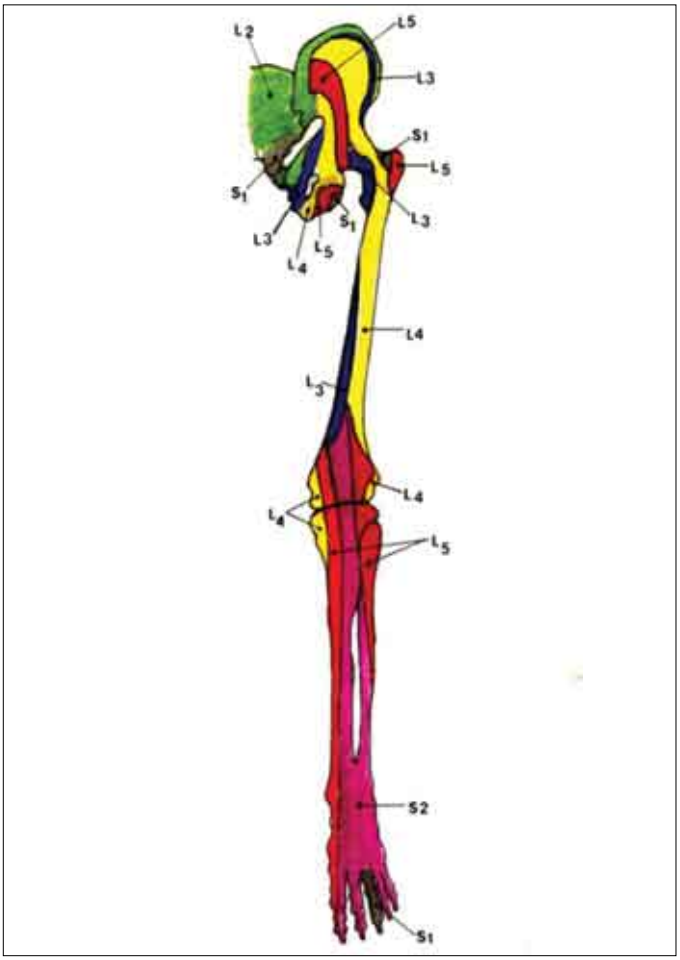

Figure 10 - Graphic representation of the sclerotomal distribution at the lower limbs.

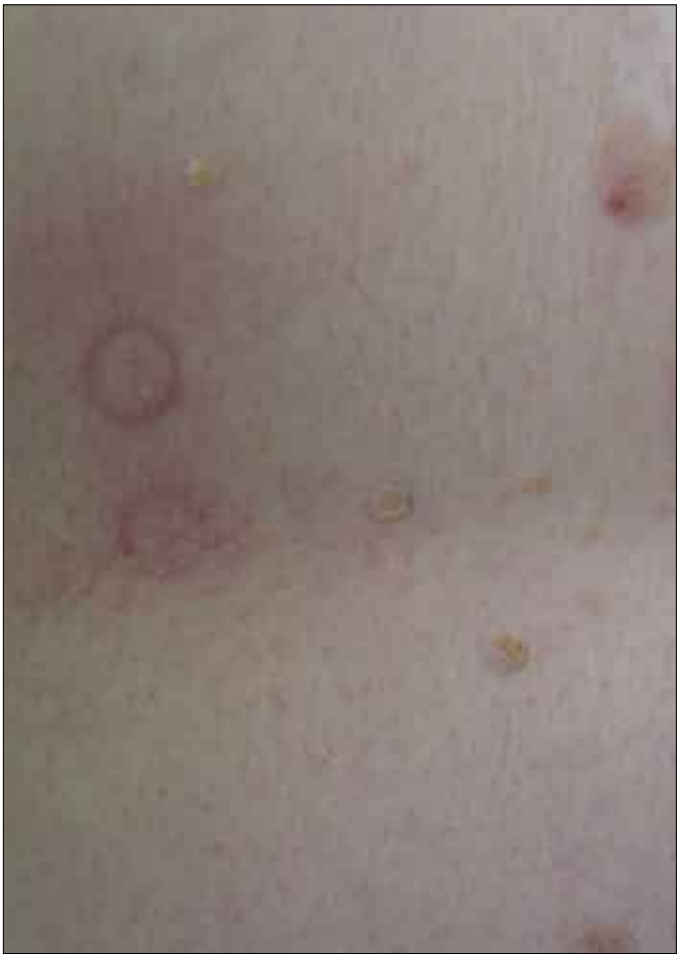

Figure 11 - Microedema and skin hyperemia at L4 sensitized segmental level.

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