ORIGINAL PAPERS

Adv Clin Exp Med 2007, **16**, 6, 785–791
ISSN 1230-025X

Paweł Reichert[1], Roman Rutowski[1, 2], Tomasz Greczner[1], Jerzy Gosk[1],
Krzysztof Zimmer[1, 3], Witold Wnukiewicz[1]

Treatment of Algodystrophic Syndrome of the Upper Extremity in Own Material

Leczenie zespołu algodystroficznego kończyny górnej w materiale własnym

[1] Department of Traumatology and Hand Surgery, Silesian Piasts University of Medicine in Wrocław, Poland
[2] Chair and Department of Sports Medicine, Wrocław University of Physical Education, Poland
3 Unit of Sport Medicine, Silesian Piasts University of Medicine in Wrocław, Poland

Abstract

Background. In spite of the development of new diagnostic methods and treatment possibilities, algodystrophic syndrome (Sudeck's disease, CRPS I) still constitutes a challenge for the treating surgeon. Its etiopathogenesis is still not fully explained, diagnostic criteria are not uniform, and treatment results are unsatisfactory. Estimation of the treatment results of CRPS I of the upper extremity in own material was the purpose of this study.
Material and Methods. Between 2000 and 2005, 38 patients were treated because of algodystrophic syndrome at the Department of Trauma and Hand Surgery. Diagnosis was based on clinical examination, X-ray, and scintigraphy. Rehabilitation and tricyclic antidepressants, anticonvulsants, vasodilators, Dexaven and Mannitol, brachial plexus blocks were used in the treatment depending on the phase of disease.
Results. The best results were achieved in patients in the first, posttraumatic phase of disease. Amelioration was achieved in most patients after use of brachial plexus blocks in the second and third phases of disease, but recurrence of full function was achieved in less that 30% patients.
Conclusions. Effective treatment and rapid rehabilitation in the posttraumatic phase of disease prevent its further progression. Use of a brachial plexus block and then rehabilitation is an efficient method of treating patients with CRPS (**Adv Clin Exp Med 2007, 16, 6, 785–901**).

Key words: algodystrophic syndrome, CRPS I, scintigraphy, brachial plexus blocks.

Streszczenie

Wprowadzenie. Pomimo rozwoju nowych technik diagnostycznych i możliwości leczenia, zespół algodystroficzny (choroba Sudecka, CRPS I) nadal jest wyzwaniem dla leczącego chirurga. Etiopatogeneza nie jest do końca wyjaśniona, kryteria diagnostyczne niejednolite, a wyniki leczenia niezadowalające.
Cel pracy. Ocena wyników leczenia zespołu CRPS I kończyny górnej w materiale własnym.
Materiał i metody. W latach 2000–2005 w Klinice Chirurgii Urazowej i Chirurgii Ręki leczono 38 chorych z powodu zespołu algodystroficznego. Rozpoznanie opierało się na badaniu klinicznym, radiologicznym i scyntygraficznym W leczeniu w zależności od okresu choroby stosowano rehabilitację oraz trójpierścieniowe leki antydepresyjne, leki przeciwpadaczkowe, leki rozszerzające naczynia, Dexaven i Mannitol, blokady splotu ramiennego.
Wyniki. Najlepsze wyniki uzyskano u chorych w pierwszym pourazowym okresie choroby. Stosując blokady splotu ramiennego w II i III okresie choroby, uzyskano poprawę u większości chorych, powrót do pełnej funkcji uzyskano jednak u niespełna 30% chorych.
Wnioski. Skuteczne leczenie i szybka rehabilitacja w pourazowym okresie choroby zapobiegają dalszej progresji choroby. Zastosowanie blokady splotu ramiennego i następnie rehabilitacja jest skuteczną metodą w leczeniu chorych z zespołem CRPS (**Adv Clin Exp Med 2007, 16, 6, 785–901**).

Słowa kluczowe: zespół algodystroficzny, CRPS I, scyntygrafia, blokady splotu ramiennego.

Despite great efforts all over the world, CRPS I (or reflex sympathetic dystrophy, Sudeck's atrophy) is still an enigma. Its pathophysiology is unknown, diagnostic criteria are still debatable, and the results of treatment are poor [1, 2]. CRPS is a potentially incapacitating syndrome occurring in an extremity usually after a minor injury or operation [3–5]. Key symptoms in the acute phase include signs and symptoms of inflammation within the affected extremity and are listed in Table 1 [6]. These alterations are present in an area larger than and including the distal part of the extremity [7, 8]. In later stages one can observe osteoporosis, pseudomotor changes, temperature changes, vasomotor instability, palmar fascitis, and trophic changes [6].

There is no consensus on the treatment of CRPS I [4] and it depends on the stage of disease. Clinical symptoms differentiating particular stadia of disease are presented in Table 2 [6]. In the acute phase one can use anti-inflammatory agents (scavengers or steroids), optimize peripheral circulation (vasodilators or sympathetic blocks) if the skin

Table 1. Primary signs and symptoms of CRPS I

Tabela 1. Główne objawy i symptomy zespołu CRPS I

Sign (Objaw)	Features (Opis)
Pain (Ból)	paramount feature, often with burn ing, throbbing, aching, stabbing, bursting, pressure, or crushing sensations
Swelling (Obrzęk)	first physical sign: initially local soft edema, then extensive and hard
Stiffness (Sztywność)	progressively worsens due to increased fibrosis
Discoloration (Zaburzenia koloru skóry)	red, cyanotic, or pale to grayish; related to vasomotor instability

Table 2. Clinical course of CRPS I

Tabela 2. Kliniczne objawy w kolejnych okresach CRPS I

Primary signs (Pierwotne objawy)	Early CRPS I: 0–3 months (Początkowy ostry okres: 0–3 m-ce)	Intermediate CRPS I: 3–9 or 12 months (Pośredni dystroficzny okres: 3–9 do 12 m-cy)	Late CRPS I: 9–12 months-years (Końcowy atroficzny okres: 9–12 m-cy, lata)
Pain (Ból)	paresthesia	notable increase, pain with motion	diminished pain, but severe with motion
Swelling (Obrzęk)	soft local edema	hard edema over extremity	periarticular (minimal)
Stiffness (Sztywność)	pain-related	increased stiffening due to fibrosis	peak stiffening, contractures common
Discoloration (Zaburzenia koloru skóry)	red then cyanotic	cyanotic, with redness over joints	pallor

Table. 3. Pharmacological targets and relate treatment of CRPS I

Tabela 3. Grupy leków i ich wykorzystanie w leczeniu CRPS I

Target (Cel)	Therapy against target (Ukierunkowane leczenie)	Advantages (Korzyści)
Inflammatory process (Proces zapalny)	naproxen p.o. [10], prednisone p.o. [11], methyloprednisolon + lidocaine [12]	no effectiveness of naproxen effective in CRPS good response to treatment
Reactive oxygen species (Reaktywne formy tlenu)	dimethylsulfoxid [13], vitamine C [14]	recommendable in CRPS could be a prophylactic method
Pain sensitization (Nadwrażliwość na ból)	carbamazepine [15], nifedipine [16], lidocaine [17]	useful, initial increase of pain possible side effects limit application effective in early CRPS
Descending control of pain (Ograniczanie bólu)	amitriptyline [18]	effective in neuropathic pain
Sympathetically maintained pain (Nadczynność układu współczulnego)	reserpine [19]	effective in early CRPS
Others (Inne)	calcitonin [20]	improvement of pain

temperature of the affected extremity is lower than that of the contralateral extremity, treat all local causes of pain (trigger points), apply systemic pain medication, adapt skeletal muscle work to the limited possibilities aided by a physiotherapist and/or ergotherapist, or splint the affected extremity if required [5]. In the late phase the procedures should include optimizing pain medication, providing splints for comfort and protection, for the upper extremities training for one-handed activities and adapting the home, and for the lower extremities providing crutches or a wheelchair and adapting the home for wheelchair use. The most often used medications and their mechanisms of action are presented in Table 3 [9]. Birklein [21] recommends the four basic steps in CRPS treatment presented in Table 4.

The differentiated treatment modes and the lack of satisfactory results of treatment presented by different authors encouraged the present authors to evaluate and analyze the methods of CRPS I treatment used in their clinic. Determining the results of treatment of CRPS of the upper extremity in own material was the purpose of this study.

Material and Methods

Between 2000 and 2005, 38 patients were treated because of CRPS I of the upper limb in this clinic. In the tested group, women constituted the majority (29 patients, 76%) and the patients' ages ranged from 31 to 76 years (average: 61 years). Eighteen had ambulatory treatment and 20 were hospitalized. CRPS was a consequence of fracture of the distal radius in 26 patients, surgery of carpal tunnel syndrome in 3, 3 patients had surgery for Dupuytren's contracture, and there were several single cases of contusion of an upper limb, contusion of the wrist, and severe hand injury. Most of the patients had been treated for their trauma outside this clinic.

CRPS I was diagnosed when four of the five following signs and symptoms were present: pain, altered skin color, altered skin temperature, edema, and reduced range of motion, which were present in an area much larger than and also distal to the primary injury. X-ray and scintigraphy were performed as additional tests. The patients had neurological and psychiatric consultation. Figure 1 presents a clinical example of the disease in the atrophic phase. Twenty-two of the patients were treated in the first (posttraumatic, group I) period, 12 in the second (dystrophic), and 4 patients in the third (atrophic) phase. The time from fracture/injury to starting treatment was 4–6 weeks for the patient group in the first phase (I), 12–16 weeks for the group in the second (II), and 6–9 months for those in the third phase (III). In the posttraumatic period (I), vasodilators, tricyclic antidepressants, tranquilizers, and non-steroid anti-inflammatory medications were used for six weeks and the so-called Szczecin method of Dexaven and Mannitol in intravenous infusion for one week. Brachial plexus blocks were used in periods II and III of disease. Brachial plexus block was established with a bolus of 15 ml of 0.25% bupivacaine and then, depending on the degree of motor blockade, the concentration and volume of the anaesthetic were reduced. A continuous analgesia was provided by regular application of 5–10 ml of 0.25–0.125% bupivacaine every 6 h or 12 h for one week. The aim was to achieve good sensory

Table 4. Symptom-oriented treatment options for posttraumatic CRPS I

Tabela 4. Poszczególne etapy w leczeniu pourazowego CRPS I

1	All patients should receive physical therapy for neuropathic pain (Leczenie bólu neuropatycznego u wszystkich pacjentów)	Antineuropathic pain therapy should be selected according to pain characteristics and concomitant symptoms (sleeplessness, fear, secondary depression). Best evidence exists for TCA and CA 2+ channel blockers. If side effects are unbearable but the drugs work, serotonin-noradrenalin re-uptake inhibitors are worth trying (Leki przeciwdepresyjne i blokujące kanały wapniowe. W przypadku niezadowalającej poprawy inhibitory wychwytu serotoniny-noradrenaliny)
2	Patients in an acute stage with edema, increased skin temperature (W ostrym okresie z obrzękami i ociepleniem skóry)	systemic steroids + local 50% dimethyl sulfoxide (steroidy systemowe i miejscowo 50% dimetylosulfoxyd)
3	Repeated sympathetic blocks should be performed in all patients under suspicion of SMP (sympathetically maintained pain-primary) cold CRPS, cold allodynia, positive effect of single sympathetic block (Powtarzalne blokady układu współczulnego)	
4	In severe chronic stage (Przewlekły okres)	spinal cord stimulation (stymulacja rdzenia kręgowego)

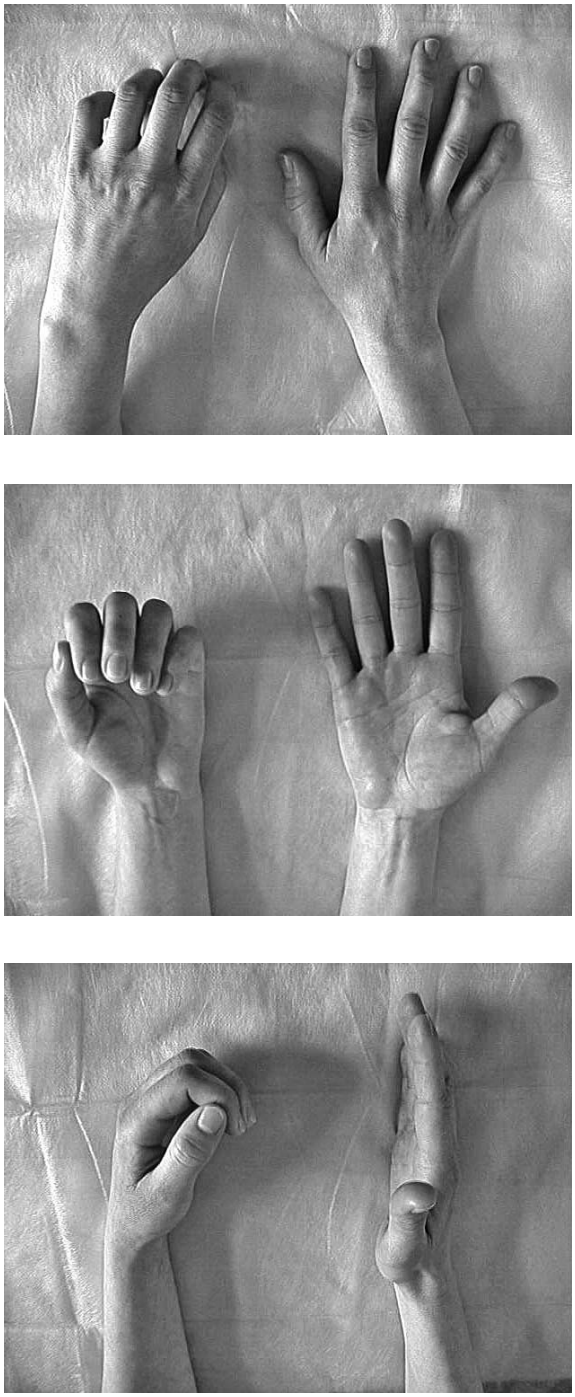

Fig. 1. Patient age 41, atrophic period of CRPS I – compulsory position of left arm: a) dorsal image of both hands, b) palmar image of both hands, c) lateral image of both hands

Ryc. 1. Chora lat 41, atroficzny okres choroby – przykurcz palców lewej ręki: a) strona grzbietowa obu rąk, b) strona dłoni obu rąk, c) zdjęcie boczne obu rąk

analgesia without motor blockade. Since motor function was unaffected, an active and painless exercise program was possible [5]. All patients had rehabilitation with calcium ionotophoresis, centrifuging massage, cryotherapy, and kinesitherapy. The follow-up time was 12 months.

Pain, mobility of the fingers and wrist, strength of the extremity, and function of the sympathetic nervous system were taken into account in estimating the results. Pain was measured using a 0–10 numeric rating scale (correlating with the result of the Visual Analogue Scale, VAS). Range of movement was measured in degrees (excellent; lack of extension up to 15% when the fingers are flexed the distance from the pulp to the distal palmar crease has to be equal to or less than 1 cm; lack of extension equal to or less than 30% with flexed fingers than can touch the palm; greater lack of extension or flexion). Strength of grip and pinch were measured with a dynamometer (excellent; result higher than 75%; result higher than 50%; result less than 50% of opposite hand). Autonomic (sympathetic) symptoms we determined by measurement of skin temperature (difference between affected and unaffected side exceeds 1.0 C°) and others autonomic testing. At the end the function of the extremity was assessed using the Quick-DASH (Disabilities of the Arm, Shoulder, and Hand) questionnaire. Excellent results meant a completely normal hand (0–15 score in Quick-DASH). Good results were pain up to a level of 2, no signs of dysfunction of the sympathetic nervous system, and limitation of range of movement (15–30 score in Quick--DASH). A poor result was pain of more than 5 points and stiffness (> 60 score in Quick-DASH). A satisfactory result meant amelioration compared with the initial state (less pain, better mobility of the fingers) requiring, however, further treatment.

Results

The best results were achieved in patients in the first period of disease. An essential difference was not observed in the treatment results between the group of patients treated with local anti-inflammatory drugs, anticonvulsants, and vasodilators and the group of patients in which the anticonvulsant was replaced by antidepressants. The effectiveness of the so-called Szczecin method (dexaven and mannitol) was similar to that of the methods presented above. Treatment results in periods II and III of disease were less satisfying than in period I, but amelioration was achieved in most patients.

Discussion

When describing the results of treatment, almost all authors use similar terminology: diminished pain and improved mobility. There are no

Table 5. Results of treatment of algodystrophic period

Tabela 5. Wyniki leczenia w zależności od okresu CRPS

Method of treatment (Zastosowane leczenie)	Number of patients (Liczba chorych)	Period of disease (Okres choroby)	Result (Wynik)			
			Very good (Bardzo dobry)	Good (Dobry)	Satisfactory (Zadowalający)	Poor (Zły)
Anti-inflammatory, anti-convulsant, vasodilator (Leki przeciwzapalne, przeciwdrgawkowe, rozszerzające naczynia)	10	I	6	3	1	
Anti-inflammatory, anti-depressants, vasodilator (Leki przeciwzapalne, przeciwdepresyjne, rozszerzające naczynia)	9	I	6	2	1	
Dexaven + Mannitol	3	I	2	1		
Brachial plexus blocks (Blokady splotu ramiennego)	12	II	2	6	4	
Brachial plexus blocks (Blokady splotu ramiennego)	4	III		1	1	2

exact and comparable criteria in the literature. Goris [4] writes that in 90% of patients a few problems may remain, such as some persistent pain, limited active range of motion, and certain decreases in skeletal muscle strength and endurance of the affected extremity. Compounding the significant variation due to individual differences, it is well known that these signs and symptoms also vary with the time course of the disease [22]. Stiffness is predominantly related to the pain upon movement and becomes progressively worse throughout stage I [24]. The most important principle is to start treatment as soon as possible before irreversible changes in the affected limbs occur [9, 24].

It is likely that CRPS is a disease of the central nervous system, but there are also numerous indications that point to peripheral inflammatory processes, abnormal sympathetic-afferent coupling, and adrenoreceptor pathology [6]. A real humoral inflammation could never be proved. However, the coincidence of signs of inflammation with trophic changes and mechanical hyperalgesia in CRPS strongly resembles neurogenic inflammation. Activation of primary afferent nerves leads to the release of calcitonin gene-related peptide (CGRP) and substance P (SP) from nerve endings [23]. It has been hypothesized that CRPS I may be a condition of psychogenic origin, may be psychologically mediated, and/or that psychological/psychiatric disturbances can be facilitating factors [24]. A significant number of psychiatric disorders and personality abnormalities were diagnosed in patients with CRPS I in other studies [24, 25].

The anti-inflammatory treatment of CRPS with steroids is based on controlled studies [6–8, 10–12]. Steroids have multiple effects: they inhibit the production of inflammatory mediators, reduce the transcription rate in dorsal root ganglia cells and thereby reduce neuropeptide content of sensory neurons, and they facilitate the degradation of neuropeptides [24]. Dimethyl sulfoxide 50% in a fatty cream applied four times daily is effective in reducing free oxygen-derived radicals in CRPS limbs [9, 13].

The most important class of substances being used for neuropathic pain are tricyclic antidepressants (TCAs). The best studies have been on amitriptyline and imipramine [14, 18]. The analgesic effect of TCAs is based on serotonin and noradrenaline re-uptake inhibition in the CNS and peripheral blockade of sodium channels. Newer substance classes such as combined serotonin/noradrenaline re-uptake inhibitors may be an alternative [26, 28].

Antiepileptic drugs are also very important in the treatment of neuropathic pain. The best evidence for analgesic properties are for gapentin and pregabalin, calcium-channel blocking agents [27]. There are less convincing data on carbamazepine, a sodium-channel blocker [15]. Calcitonin and diphosphonate effect bone turnover, a beneficial effect of both substances as Gobelet showed [9, 20].

Brachial plexus block as a method of treating CRPS was successfully used in some cases [28, 29]. Intermittent or continuos block of the sympathetic nervous system was successfully used on a few

occasions [30]. The sensory block of the brachial plexus was maintained for 6–7 days. Then the analgesia was stopped, but the catheter was left in place in case the patient needed some more analgesia in the following days

Stellate ganglion blocks can be applied both therapeutically and diagnostically. Stellate blocks inhibit efferent impulses to the extremities, interrupting the abnormal sympathetic reflex without blocking normal somatic nerve function. Technically, stellate blocks are much more demanding to perform than IV regional blocks and employ either bupivacaine (Marcaine) and lignocaine (Xylocain). Amelioration of the symptoms is usually noted after about 30 min and may last up to a few hours. Three to ten sessions can be performed at 10- to 14-day intervals. The block is considered successful when a Horner's sign develops and warming of the affected area is observed [6].

Local anaesthetic blocks of somatic nerves can be performed using lignocaine. Somatic nerve blocks are aimed at interrupting the abnormal reflex through the somatic nerves by blocking afferent nerve impulses. Blocks can be administered two to three times per week without producing local irritation.

The physical therapy management of patients with CRPS mimics most other conditions for which physical therapy interventions remain empirical and symptom based. Physical therapy appears to be a useful adjunctive therapeutic approach for patients with CRPS, particularly in an interdisciplinary setting. Physical therapy should include at least a gentle range of motion exercises, inactivation of myofascial trigger points, desensitization interventions, aquatic physical therapy, posture training, and movement retraining [31].

It can be concluded that correct treatment and quick rehabilitation in the posttraumatic period of disease prevent disease progression to the dystrophic and atrophic forms and the use of a brachial plexus block and then rehabilitation is an effective method in treatment of patients with CRPS.

References

[1] Galer B, Bruehl S, Harden R. IASP diagnostic criteria for complex regional pain syndrome: a preliminary empirical validation study. International Association for the Study of Pain Clin J Pain 1998, 14, 48–54.

[2] Janig W, Stanton-Hicks M: Reflex Sympathetic Dystrophy: A Reappraisal. Progress in Pain Research and Management. IASP Press, Seattle 1996.

[3] Harden R: A clinical approach to complex regional pain syndrome. Clin J Pain 2000, 16, 2, 26–32.

[4] Goris R, van der Laan L: Reflex Sympathetic Dystrophy-Another View. European J Trauma 2001, 3, 99–103.

[5] Margić K, Pirc J: The treatment of complex regional pain syndrome (CRPS) involving upper extremity with continuous sensory analgesia. Eur J Pain 2003, 7, 43–47.

[6] Soucacos P, Johnson E: Upper extremity reflex sympathetic dystrophy. Current Orthop 2001, 14, 356–364.

[7] Veldman P, Reynen H, Arntz I, Goris R: Signs and symptoms of reflex sympathetic: prospective study of 829 patients. Lancet 1993, 342, 1012–1016.

[8] Stanton-Hicks M: Complex regional pain syndrome (type I, RSD; type II, causalgia): controversies. Clin J Pain 2000, 16, 2, S33–40.

[9] Ludwig J, Baron R: Complex regional pain syndrome: an inflammatory pain condition? Drug Discovery Today: Disease Mechanism 2004, 4, 449–455.

[10] Kingery WS: A critical review of controlled clinical trials for peripheral neuropathic pain and complex regional pain syndrome. Pain 1997, 73, 123–139.

[11] Christensen K: The reflex dystrophy syndrome response to treatment with systemics corticosteroids. Acta Chir Scand 1982, 148, 653–655.

[12] Żyluk A: Results of the treatment of posttraumatic reflex sympathetic dystrophy of the upper extremity with regional intravenous blocks of methylprednisolone and lidocaine. Acta Orthop Belg 1998, 64, 452–456.

[13] Zuurmond W, Langendijk P, Bezemer P, Brink H, de Lange J, van Loenen AI: Treatment of acute reflex sympathetic dystrophy with DMSO 50% in a fatty cream. Acta Anaesthesiol Scand 1996, 40, 364–367.

[14] Cazeneuve J, Leborgne J, Kermad K, Hassan Y: Vitamin C and prevention of reflex sympathetic dystrophy following surgical management of distal radius fractures. Acta Orthop Belg 2002, 68, 481–484.

[15] Harke H, Gretenkort P, Ladleif H, Rahman S, Harke O: The response of neuropathic pain and pain in complex regional pain syndrome I to carbamazepine and sustained-release morphine in patients pretreated with spinal cord stimulation: a double randomized study. Anest Analg 201, 92, 488–495.

[16] Muizelaar JP.: Complex regional pain syndrome (reflex sympathetic dystrophy and causalgia): management with the calcium channel blocker nifedypine and/or the alpha-sympathetic blocker phenoxybenzamine in 59 patients. Clin Neurol Neurosurg 1997, 99: 26–30.

[17] Wallace MS, Ridgeway B, Leung A, Gerayli A, Yaksh T: Concentration-effect relationship of intravenosus lidocaine on the allodynia of complex regional pain syndrome types I and II. Anesthesiology 2000, 92, 75–83.

[18] Pawl RP: Controversies surrounding reflex sympathetic dystrophy: a review article. Curr Review of Pain 2000, 4, 259–267.

[19] Chuinard R, Dabezies E, Gould J, Murphy G, Matthews R: Intravenosus reserpine for treatment of reflex sympathetic dystrophy. South Med J 1981, 74, 1481–1484.
[20] Gobelet C, Meier J, Schaffner W, Bischof-Delaloye A, Gerster J, Burckhardt P: Calcitonin and reflex sympathetic dystrophy syndrome. Clin Rheumatol 1986, 5, 382–388.
[21] Birklein F: Complex regional pain syndrome. J Neurol 2005, 252, 131–138.
[22] Soucacos P, Diznitsas L, Beris A, Xenakis T, Malizos N: Reflex sympathetic dystrophy of the upper extremity. Clinical features and response to multimodal management. Hand Clinics 1997, 13, 339–354.
[23] Birklein F, Handwerker H: Complex regional pain syndrome: how to reesolve the complexity? Pain 2001, 94, 1–6.
[24] Monti DA, Herring CL, Schwartzmann RJ, Merchese: Personality assessment of patients with complex regional pain syndrome type I. Clin J Pain 1998, 14, 295–302.
[25] Puchalski P, Żyluk A: Complex regional pain syndrome type I after fractures of the distal radius: a prospective study of the role of psychological factors. J Hand Surg 2005, 30B, 6, 574–580.
[26] Sindrup SH, Jensen TS: Efficacy of pharmacological treatments of neuropathic pain: an update and effect related to mechanism of drug action. Pain 1999, 83, 389–400.
[27] Mellick GA, Mellick LB: Reflex sympathetic dystrophy treated with gabapentin. Arch Phys Med Rehabil 1997, 78, 98–105.
[28] Ribbers GM, Geurts AC, Rijekn RA, Kerkkamp HE: Axillary brachial plexus blockade for the reflex sympathetic dystrophy syndrome. Int J Rahabil Res 1997, 20, 371–380.
[29] Wang L-K, Chen H-P, Chang P-J, Kang F-C, Tsai Y-C: Axillary brachial plexus block with patient controlled analgesia for complex regional pain syndrome type I: a case report. Reg Anesth Pain Med 2001, 26, 68–71.
[30] Gibbons JJ, Wilson PR, Lamer TJ, Elliot BA: Interscalene blocks for chronic upper extremity pain. Clin J Pain 1992, 8, 264–269.
[31] Dommerholt J: Complex regional pain syndrome-1: history, diagnostic criteria and etiology. J Bodywork Mov Ther 2004, 8, 167–177.

Adres do korespondencji:

Paweł Reichert
Department of Traumatology and Hand Surgery
Silesian Piasts University of Medicine
R. Traugutta 57/59
50-417 Wrocław
Poland
Fax: +48 71 733 27 06
E-mail: chiruraz@churaz.am.wroc.pl

Conflict of interest: None declared

Received: 6.06.2007
Revised: 13.08.2007
Accepted: 28.11.2007