

Available online at www.rajournals.in

RA JOURNAL OF APPLIED RESEARCH ISSN: 2394-6709

DOI:10.33826/rajar/v6i1.01 Volume: 06 Issue: 01 January-2020 International Open Access



Impact Factor- 6.295

Page no.- 2601-2609

Complex Regional Pain Syndrome Type I: Update on Therapeutic Coverage

Saloua Khalfaoui¹, El Mustapha El Abbassi²

^{1,2}Department of Physical Medicine and Rehabilitation of the Military Instruction Hospital Mohammed V, Rabat, Morocco

ARTICLE INFO	ABSTRACT
Published Online:	Management of the algodystrophy is not limited to the management of pain. It can turn out to be
06 January 2020	difficult, in front of pathology with an evolution which is known by severe pains, incapacitating and
	most of time refractory to usual analgesic treatments. The functional echo is often severe; the
	persistence of pains is besides frequently responsible of unfavorable psychological echo. In the
	absence of a definite curative treatment of the algodystrophy, every doctor has to consider
	immediately, facing a trauma, an illness or a therapeutic act may become complicate of an
	algodystrophy, early coverage as soon as possible and efficient of the pain to put the patient in best
	conditions in order to prevent such a complication.
	The best approach is then multidisciplinary; especially for patients with treatment failure referred to
Corresponding Author:	the pain centers. It associates with different degrees a medicinal and / or infiltrative treatment, a
Saloua Khalfaoui	psychological approach, a reeducation, and an irregular reevaluation in a view of a vocational
GSM: 212612608298	integration.

KEYWORDS: Algodystrophy; Medicinal treatment; Psychological coverage; Reeducation.

Introduction:

Chronic pain syndrome is a whole somatic, psychological and behaviors involved in the maintenance and exacerbation of chronic pain [1]. The model psychosocial bio of pain readily applies to complex regional pain syndrome. Dysregulation of the sympathetic nervous system often follows physical and / or psychological trauma, with an inability to cope, a behavioral deficit adapting to this stressful event.

Thus, psychological factors participate the etiopathogenesis of CRPS [2, 3]. They go mostly to grow with time. The pain, the impotence functional and disability that occur stress, willingly associated with anxiety or even depression. Coping strategies are usually liabilities, in particular because of the increase pain by movement and fear of movement. Avoidance pipes are happy to use reinforced by the family environment and even the context socioprofessional, especially in the case of an accident working. The frequent occurrence of an existential loss, real symbolic, with feelings of abandonment these dysfunctional strategies.

Definition

In 1993, the IASP (International Association for the Study of Pain) proposed the term Painful Syndrome

Regional Complex (SDRC), which excludes any reference to a physiopathological mechanism. Spontaneous pain permanent is the obligatory condition of the diagnosis.

The term regional recognizes the possible spread of these pains, which can thus spill over the territory of the initial trauma. The varied and dynamic nature of clinical expression for the same patient, as well as a patient to another is recognized on the complex term.

Classification and diagnostic criteria for CRPS established by IASP 1994, there is a distinction between CRPS type I (algodystrophy), which occurs after a nociceptive traumatic event, and type II CRPS (causalgia), which follows a proven nervous lesion.

Other clinical features are common for the two types [4-6].

Historical

Until the early 1990s, the diagnosis was based on clinical signs of the 3 progressive stages (inflammatory: hot, dystrophic: cold, atrophic: sequellar), the confirmation being supported by the X-ray, the

3-stage bone scintigraphy, and possibly I.R.M. in T2 mode [7]. The supposed physiopathology was for the most part about the deleterious role of the nervous system sympathetic [8, 9] because of the symptomatology

clinical than that of efficiency, at the time indisputable, different locoregional block techniques.

The similarities of the pains of the algodystrophie with that of the causalgie, syndrome occurring sometimes after a nervous lesion, led IASP to propose in 1994 a new terminology [10]. Since then, we talk about complex regional pain syndrome (CRPS, at RC PS type I (algodystrophy) and CRPS) type II (causalgia), the sympathetic nervous system can (sympathetic dependent pain, DDS or SMP: sympathetically maintained bread or not (pain independent of the sympathetic DIS or SIP: sympathetically independent bread contribute to the painful constellation CRPS. CRPS is defined by pain and functional disorders extensive and disproportionate to look at the initiator event.

Epidemiological data

The occurrence of a dysphasystrophy is possible at any age. Forms of children, adolescents, young adults are quite common. A single epidemiological study concerning the RSD [11]. She has included the inclusion criteria (sympathetic dystrophy Reflex), then (CRPS) when this term was created (in excluding cases of transient regional osteoporosis or migratory). This study revealed an incidence and prevalence of RSD 5.4% per 100,000 and 20.5% per 100,000. The sex ratio woman / man was, in this study, 4/1. Reaching upper limbs was twice as frequent as that of the lower limbs [11].

Clinical data

A- The three classic stages of dystrophy [12]

1- Stage I: High stage of hyper permeability transient locoregional

It lasts from a few weeks to a few months. The pain begins in the area of the trauma, increases willingly to mobilization, pressure, heat, to emotions. Autonomous signs are present: redness, local hyperthermia, edema, increased growth of appendages. Osteoporosis begins.

2- Stage II: Dystrophic stage

It starts at the end of stage I and lasts several months, sometimes almost a year or more.

The pain gets worse in extension, in intensity; cold, rather than the hot, increases the pain. In the distal region, the signs are: cyanosis, cold skin, cloudiness dander, hypersudation. The fibrosis settles. Osteoporosis, when she is present, is clear. The atrophy begins.

3- Stage III: Atrophic Stage

It starts at the end of stage II and lasts 2 years or more after the beginning of the disease. The pain can remain important and then diminish. The swelling has disappeared. The stiffness and limitation of movements predominate. At the ends, the skin is bright, fine, and pale, the subcutaneous tissue is atrophic.

Osteoporosis is possibly present in a clear way on the member segment or the member.

B- <u>CRPS: Modified Clinical Diagnostic Criteria</u> proposed by Harden and Bruehl [12]

The following four criteria must be validated:

- 1- Continuous pain, disproportionate compared to any triggering element;
- 2- Presence reported by the patient of at least one of symptoms in 3 of the following 4 categories:
- -Sensory disturbances: hyperalgesia and / or allodynia
- Vasomotor disorders: thermal asymmetry and / or change in skin color, and / or asymmetry of skin color,
- Sudoromotor disorders or edema: edema and / or modified sweating and / or asymmetric sweating,
- Motor / trophic disorders: decreased mobility and / or motor disorders (weakness, tremor, dystonia) and / or trophic disorders involving hair, nails or the skin;
- 3- Presence found on examination at the time of the evaluation of at least one sign in 2 or more following categories:
- Sensory disturbances: hyperalgesia (with puncture) and / or allodynia (light touch or somatic pressure deep)
- Vasomotor disorders: Thermal asymmetry and \slash or change in skin color, and \slash or asymmetry of skin color,
- Sudoromotor disorders or edema: edema and / or modified sweating and / or asymmetric sweating,
- Motor / trophic disorders: decreased mobility and / or motor disorders (weakness, tremor, dystonia) and / or trophic disorders (hair, nails, skin).
- 4- The signs and symptoms are not better explained by another diagnosis.

The choice of imaging exams depends on the consultation period and diagnostic doubts.

C- Some radiological aspects

Comparative standard radiographs show local and regional bone hypertransparency after a delay of at least 3 to 4 weeks, heterogeneous. Heterogeneous demineralization results in a hypertransparency trabecular speckled micro or macropolygéodique.

Increased bone fixation in the algodystrophy in the warm phase in the pathological area is a expected scintigraphic phenomenon, early, sensitive, sustainable but not specific.

Objective MRI on the same pathological area algodystrophic, transient tissue edema superficial, periarticular, synovial and effusion transient synovium. The results of the MRI are based on stages of dystrophy.

Bone densitometry offers the possibility of quantify bone mineral loss in the bone area algodystrophic, more marked on the trabecular bone than cortical.

Prevention

The concept of preventive analgesia, largely altered in anesthesiological practices [13], postulates that any analgesic action reducing as early as possible and as long as

necessary the activity of the afferences generated within a tissue damage will have beneficial effects in terms of prevention of possible secondary chronic pain. This concept made the proof of its effectiveness for the prevention of pain post-zosterian and for the prevention of algohallucinosis.

We could therefore imagine that early detection post-traumatic algodystrophy, both an assessment of sympathetic activity at the bedside patient [14] only by complementary examinations, can start a specific treatment under preventive. More simply, the effective treatment of any pain related to a circumstance likely to give a period of dysplasia should reduce the incidence of occurred. Postoperative preventive measures have suggested (the least unpleasant intervention possible, treatment of any superinfection, facilitation of return venous, effective analgesic [15]), but the exact role remains to prove.

Treatment

A- Goal

The treatment aims to fight against pain, vasomotor abnormalities, and to prevent the installation possible capsular, synovial, tendinous retraction, fascial. But there is no cure currently defined algodystrophy [16-19].

ALS is sometimes not the only cause of pain. The underlying disease must be taken into account of the initial injury, psychological data, associated chronic diseases such as diabetes, medico-legal problems. Support in physiotherapy and psychotherapy is necessary, just enough time spent on information algodystrophic patient data.

B- Means

Drug treatment uses analgesics and physiopathological drugs, see to infiltrations (sympathetic chain, perimedullary, plexus). The psychological approach aims to identify limited control factors (depression, anxiety, neurosis post-traumatic, symbolic of the initiating event) is to start a care, often in the long run course (cognitive-behavioral therapy, biofeedback, hypnosis, psychotherapy). A recent trend of the algological literature wanted the painful chronicles were, more than others, victims of abuse, even abuse in childhood: Now, it seems although it is not so. Non-painful rehabilitation, aims to restore functional abilities through different means: orthoses, mobilizations and desensitization proprioceptive ... [9]

1- Drugs

a- Corticosteroids

Only two controlled studies concerning the administration have been published [20, 21]. They mention a benefit but note the lack of long-term follow-up.

Classically, joint and periarticular infiltrations corticosteroids are recommended at the stadium evolved from dystrophy, where they lead to improvement, without healing.

Prednisolone per os, has been proposed without evidence of a specific benefit [22] .It is used in infiltration intra articular in advanced forms.

b- Calcitonin

Classical treatment, its benefit is controversial, especially in advanced stages [23, 24]. At the early stage, injection subcutaneous daily of 100 IU is more effective than placebo (60% versus 25% of relieved patients).

c- Bisphosphonates

Effect of their osteoclastic action and their action inhibitory on the production of pro-inflammatory cytokines, they should, in the physiopathological hypothesis inflammatory, have a favorable effect. This is confirmed by a recent randomized study [25].

Different products were evaluated: Clodronate (300mg IV), Alendronate (7.5 mg IV), Pamidronate (60 mg IV), Neridronate (100 mg IV), especially in one relatively evolved, with benefits on pain and edema in the short and medium term, as well as functional abilities. In fact, they currently constitute one of the rare indications validated according to some authors [22, 26-28].

d- Analgesics

They are and remain the basis of treatment. At the early stage, minor analgesics (including NSAIDs) and major alleviate spontaneous pain, but have little effect on mechanical hyperalgesia. It is at the late stage that the use of major analgesics is problematic [29]. They are most often offered orally in the form of 24-hour background processing, with the possibility of manage the possible painful accesses, by interdoses. Thus, beyond a daily adapted support, it is sometimes necessary to know how to use certain level 2 or opioid analgesics, before rehabilitation or according to daily activities [30].

The opioids or tier 3 are used if the pain is very intense, after failure or intolerance of others levels, rarely from the outset, and with information from precaution because even if useful to reduce the pain, they have the disadvantage of causing side effects cognitive.

The prescription in CRPS must be limited in the time, and she would be more effective associated with an antagonist NMDA receptor [30].

The only opioid that could, perhaps, be of interest because of its NMDA antagonistic action is methadone, currently reserved for substitution treatments. Anticonvulsants, currently and primarily Gabapentin and pregabalin, are proposed if component of neuropathic pain is present or predominant (DN4 questionnaire) [30].

Benzodiazepines are also sometimes prescribed for their anxiolytic and myorelaxant action, taking into account recent recommendations regarding clonazepam.

The benefit of certain short-acting opioids must encourage them to know how to use them before the re-education sessions.

Similarly, some local painkillers (such as lidocaine compress) can be used as complement, rather in forms associating pains neuropathic and possibly before some care.

e- Other current and potential drugs

Beta-blockers and calcium channel blockers did not subject of controlled studies, but published series, no controlled, report a profit. This does not mean that these products are not superior to placebo, but this remains to be proved.

In the same vein, local anesthetics intravenous are analgesic with no net effect on mechanical hyperalgesia [31], have been prescribed (mexiletine, flecaine) without controlled studies can not come to prove the correctness of this attitude.

Sympatholytics (phentolamine, prazocine ...) have a beneficial effect, superior to placebo, in the DDS, with however, binding side effects.

2- Sympathectomy, regional blocks, epidural

Sympathectomy L2-L4 or lower third of ganglion stellar to T3 according to the locations respectively to

lower and upper limbs was proposed, but is quite rarely practiced in the algodystrophy. A great controversy remains about the interest and the place of the blocks motors, epidural anesthetics, regional blocks [32, 33]. Many products [16, 34] have been tested in regional blocks, intravenous: clonidine, phentolamine, labetalol, reserpine, guanethidine, droperidol, ketanserin, methylprednisolone, bretylium, lydocaine.

One of the difficulties comes from the recognition of mechanisms involved, which vary from patient to patient and for the same patient as a function of time [12].

In the hot phase of dystrophy, current data are in favor of a dual system response friendly: in the first place a locoregional defect of response, but which is opposed by hypersensitivity locoregional adrenergic receptors to circulating monoamines, whose secretion is a function of stress, pain and the general condition of the patient.

Blocks designed to block the system sympathetic to this phase could not be effective on this second anomaly (aggravating even the first component) [12].

Flexion dystonia of the upper limb, or extension to the lower limb are being tested treatment with intrathecal baclofen or many local injections of botulinum toxin [12].

3- Neurostimulation

Neurostimulation is widely proposed for its analgesic action. Most often in the form of transcutaneous neurostimulation, it is interesting in particularly in essentially neuropathic forms, on well-localized areas. It is often proposed early, but requires patient education.

Self-control of pain contributes psychologically to the management of pain and function by the patient.

However, the validity of its use in the context of SRDC was not well determined [35].

Medullary stimulation, whose analgesic efficacy has been confirmed in neuropathic pain peripherals, has been used for the treatment of CRPS type II (causalgia) since 1982 by Broseta [36] but Barolat's type I as early as 1987 [37], with encouraging results.

Since then, a number of complications have confirmed the interest of the use of this analgesic technique.

The different studies show favorable results for treatment of CRPS by medullary stimulation for pain relief and quality improvement of life.

Efficiency on functional improvement is not clearly demonstrated [6].

Medullary stimulation is being considered by some in the rebel algodystrophies with for Sears et al., a certain benefit for patients [38] and for Kumar et al. a long-term benefit according to certain criteria predictive [39], and even an economic benefit. It is a technique presenting certain risks, expensive, not to propose that for rebellious forms but several authors are currently considering it as one of the therapeutic proposals benefiting from a good validity of clinical efficiency [27, 40].

Brain stimulation has sometimes been proposed in excessively rebellious forms of SRDC, in particular vis-à-vis motor or sensory symptoms [41,42].

Finally some data on the use of stimulation repetitive transcranial magnetic (rTMS) high frequency are reported by Picarelli et al. [43] out of 23 SRDC type I upper extremity patients with positive on pain during the stimulation period of ten sessions offering perspectives for forms refractory.

4- Other treatments

a- Ketamine

General anesthetic of the NMDA type (N-methyl-Daspartate) antagonist, is increasingly used in the treatment of pain, with an analgesic effect and antihyperalgesic at subanesthetic dose. She has benefited from two studies highlighting his effectiveness in the SRDC, particularly where there is a neurological component [44-46].

In a recent review, Azarie et al. report that Ketamine is a promising treatment for CRPS, but whose procedures for use remain to specify as well as efficiency by studies better conducted to clarify its long-term effect and its safety [47].

b- Botulinum toxin (TB)

Some recent data make use of TB in the management of intramuscular injection SRDCs.

The use of intradermal TB has been reported for its action in chronic neuropathic pain. In the context of CRS, the analgesic effect of TB may be to associate with the muscle relaxant effect. A retrospective study, rather limited Karkar et al. [48], seems to evidence a benefit on pain, in the short term, taking into account certain forms of CRPS with signs dystonia or localized spasms in the neck and the scapular belt. This author reports 5 other small studies or case studies

previously conducted with the limited and inconsistent results [48].

5- Psychological care

Prescribing tranquilizers, or anti-depressants antidepressant analgesic action is to be adapted according to patients. Interference psychic state- algodystrophy are complex, but the prolonged pain has, by definition, always a psychological repercussion, he do not take for the initial disease. The patient, once the pain is gone, find a personality normal [12].

Patients still sadly suffering algodystrophy are really handicapped. A psychotherapy can achieve support for the patient algodystrophic who continues to suffer, especially close entourage, friends, coworkers, employer, and sometimes doctors may mistakenly consider the a reflex patient as a person who is complains exaggeratedly. In the same vein, patient associations exist in different countries as well as several news websites and on the subject of dystrophy, but which group together often patients with chronic and severe forms [12].

6- Cognitive Behavioral Therapy

The high frequency of inappropriate behavior and associated mistaken beliefs justify the use of cognitive behavioral therapies within the socket in therapeutic charge. There are however some peculiarities in CRPS compared to others chronic pain. The natural history of CRPS is evolving in general towards healing with, sometimes, after-effects moderate (in particular capsular retractions). The recurrences exist and warrant that patients be warned and adopt a preventive attitude.

Unlike other painful conditions, usually finds a rough start with an accident inaugural.

The effectiveness of CBT management in chronic pain is well established [49, 50].

There are some studies highlighting the effectiveness of such supported in CRPS. They are most often isolated clinical cases [51] and often relate to children and adolescents. The techniques used are in general relaxation [52], biofeedback and sometimes hypnosis. These techniques can lead to regression complete symptoms or an improvement in pain, motor skills or even skin temperature.

7- Rehabilitation and Rehabilitation

Rehabilitation and rehabilitation are always considered essential in the management of CRPS, associated with that of pain, with, most often, equal importance both the fear of losses functional is present: occurrence of stiffness, retraction, "segmental pseudo-negligence or neglect-like syndrom", disorders of the body diagram, related to pain and immobilization [53].

Yet, the place and type of rehabilitation techniques (physiotherapy, occupational therapy, psycho motility, etc.), in a multidisciplinary care so often praised, remains poorly

codified and poorly evaluated [22, 30, 35, 54]. He there is indeed little or no scientific data on the effectiveness of the rehabilitative care.

Rehabilitation aims to prevent, limit or reduce disability that may occur in the evolution of CRPS. It must therefore be adapted to the evolution, to the symptoms and to the clinical forms, reasoned and prudent not to not be a factor of aggression and exacerbation painful, prolonged, and must be part of a internal and multidisciplinary therapeutic strategy coordinate.

The objectives are:

- Maintaining a voluntary mobilization, allowing maintenance of joint amplitudes and trophicity muscular
- The contribution to analgesia and reduction of edema;
- Maintaining a functional capacity. This rehabilitative care is essential to limit the risk of a disabling chronic evolution, with a strong functional impact, making sure to reduce fear of movements or potentially activities painful for the patient [55].

Physiotherapy and occupational therapy must be able to associate essentially:

- analgesic techniques: massages, physiotherapy, balneotherapy with short-term benefit but facilitate progressive active mobilization and rehabilitation;
- methods of discharge and / or resting: in the early phase, so-called warm phase immobilization / discharge is often necessary (support discharge for the lower limb with canes, rest splint for foot, knee, upper limb, mobilization balneotherapy allowing a segmental discharge), but it must be limited in time with evaluation very regular of its indication, explained to the patient, because it can contribute to the loss of mobility probably at the risk of "segmental pseudo-negligence";
- techniques for reducing edema: at the phase hot the reduction of edema should be sought by posture techniques, touch drainage, by the muscular contractions that favor the return venous and lymphatic. This step is important because facilitator of mobilizations. She can be temporarily helped by a restraint, especially to members lower;
- active mobilization techniques (see passive and posture): Active mobilization is essential for the maintenance of muscle trophicity and amplitudes joints, venous return, the fight against phenomena of segmental pseudo-negligence. Active, and less painful, but it must be controlled for be exercised in the fullest possible range and for all articular segments, in those above and underlying to the affected area. According to evolution, passive techniques and posture must be combined to limit the tendon-muscular contractions and regain the amplitudes

joint. The use of some dynamic orthoses may be necessary but with the requirement to be properly performed, monitored and adapted;

- functional exercises, more global occupational therapy will allow to continue the use of the member and thus to avoid the apprehension behaviors of movements. This global functional approach is fundamental.

All of this support must be accompanied by of a patient education, so that some exercises can be performed outside the care sessions by the patient himself, and for information from the patient and his family on behavioral adaptation and psychological to pain and the negative effects of not using the painful area for fear of movements [56].

This rehabilitation can be done in a liberal cabinet of physiotherapy for simple forms with a good evolution. It will be carried out in a unit of medicine physical therapy and rehabilitation, often in a ambulatory (part-time hospitalization), as soon as multidisciplinary support with specific follow-up will be necessary. This modality allows for a more high intensity of care (longer sessions allowing a fragmented and varied work), a more global approach, especially psychological and socioprofessional is often necessary.

In this context of maintaining mobility, according to new concepts integrating the neurobehavioral dimension

CRPS (disruption of the cortical integration of sensory motor information, reorganization somesthesia, cognitive disorders and body schema), other rehabilitative technical approaches can be evoked:

- The mirror therapy that is proposed in the management of CRS, as in stroke and phantom limb syndromes [35]. A recent analysis of literature [57], however, reports a low level of evidence for this technique in this clinical setting, no doubt to moderate according to the evolutionary stage;
- The mental imagery, developed by Moseley, which allows, with laterality recognition exercises and virtual movements, to have an effect on the intensity of the pain [58]; a specific sequential program associating mirror exercises could be proposed [59, 60];
- Prismatic visuo-motor adaptation has been proposed, in parallel to these effects in negligence after injury cerebral symptoms, symptoms of neglect found in the CRPS [61] by action of modification of visual-spatial attention. It is in this framework of a technique offering new perspectives therapeutic.

Beyond this motor rehabilitation, the management must associate psychotherapeutic techniques (psychotherapy, relaxation, hypnosis, sophrology ...), and take into account the whole social context, in particularly socio professional post-traumatic CRPS.

Recent data on rehabilitation programs more specific, considering that pain leads to adverse effects by non-use are proposed to "force" the use, without specifically account for the pain, to favor a restoration functional and introduce a reversal of the vicious circle of non-use, with results reporting good feasibility of these programs [62].

Rehabilitation should be considered early, early to avoid non-use of the limb, atrophy, stiffness and it needs to be evaluated regularly. She is of set up easy at the start phase, but can become more complex in rebellious and sometimes diffuse forms.

The regular evaluation of the patient must make it possible to know offer a multidisciplinary support associating center of pain and unit of physical medicine and rehabilitation.

C- Therapeutic Strategy and Indications [63]

In the absence of recommendations, standards therapeutically, given the often changing unpredictable, lack of recognized efficiency data certain therapies in the proposed arsenal, and the need not to harm in this clinical context complex, it is nevertheless useful to propose a strategy graduated is not "trying everything and everything at the same time".

This strategy is part of the characterization of the phase evolutionary (warm, cold, trophic phase ...), type I or type II, the overall consequences of CRPS and a progression of proposed therapies evaluated.

We can thus propose:

- 1- At first: This is most often the so-called "hot" phase justifying analgesia (treatment medicinal analgesic background I or II-infusion bisphosphonates-transcutaneous neurostimulation), a resting (controlled discharge and immobilization discussed), rehabilitation focused at least on a active mobilization and education (at least physiotherapy, sometimes occupational therapy) with a rather "cognitive" objective aimed at minimizing and reassuring the patient; it is important not to neglect the suffering possible psychological (antidepressant treatment and / or psychotherapist) and neuropathic component (anticonvulsants);
- 2- In a second step, if the evolution is more severe, with intense pain and functional impact: multidisciplinary management associating a therapy analgesic more important (analgesics palliers II and III, antidepressants, anti-epileptics, ketamine, block nervous...). Rehabilitation should be, at this time, more intensive, more
- Rehabilitation should be, at this time, more intensive, more global and adapt with the different analgesic techniques that have been proposed, multidisciplinary, with functional approach (physiotherapy and occupational therapy-analytical technique and global-remobilization / place mirroring techniques or motor imagery), psychological and social;
- 3- In a third step: Analgesia techniques can be more invasive (stimulations) and taking the rehabilitation treatment is intended instead to compensate the disability.

The care requires some attention. It is necessary to establish early the diagnosis for treat pain early, fight edema and restore the function. We must be wary of any mobilization

painful (physiotherapy should not be harmful), uncontrolled and unexplained capital aggressive techniques not explained, climbing treatment without evaluation and without concerted several specialists.

It is important to keep in mind that evolution CRPS can be spontaneously and naturally favorable, with a sometimes long evolution, hence the gradualness and adaptability of different approaches therapeutic.

Conclusion

The management of CRPS remains difficult, despite often favorable evolution, in the absence of modalities therapeutically validated. For the majority authors, pain management (evaluation and therapeutic adaptation) and rehabilitation (limitation, prohibition of immobilization) constitute the two pillars of treatment that must be initiated at the earliest. The neurological component must be evaluated even in the absence of neurological lesion taking into account the risks of cognitive repercussions and on the body diagram.

Finally, the psychosocial approach must always be associated in the care.

Currently, on a purely Evidence Based Medicine, the specific therapeutics appearing the most validated are the use of bisphosphonates and the medullary neurostimulation [63], and promising ketamine.

Références

- 1. Bureau F. Pratique du traitement de la douleur. Paris, Doin, 1988.
- 2. Van Houndenhove B., Vasquez G. Ethiopathogenesis of reflex sympathic dystrophy: a review and biopsychosocial hypothesis. The Clinical Journal of Pain 1992; 8:300-6.
- Gatchel RJ (Eds). Psychological approaches to pain management. 2nd edition, New-York, Guilford, 2002.
- 4. Merskey H., Bgduk N. Classification of Chronic Pain Syndromes and Definitions of Pain Terms. 2nd Ed. Seattle, wash: IASP Press; 1994.
- 5. Stanton-Hicks M. Spinal cord stimulation for the management of complex regional pain syndromes. Neuromodulation 1999; 2: 193-201.
- AllanoG., Cavagna R., Vilain N. La stimulation médullaire dans le traitement des syndromes douloureux régionaux complexes. Douleurs Evaluation- Diagnostic- Traitement 2009; 10:19-22
- Doury PCC. Algodystrophy. Aspectrum of disease, historical perspectives, criteria of diagnosis, and principles of treatment. In: COONEY WP, SCHUIND F (Eds) Hand Clinics. Upper extremity pain dysfunctions: somatic and sympathetic disorders. WB Saunders Company, Philadelphia, 1997:327-37.

- 8. Baron R, Janig W. Schmerwsyndromemitkausaler Beteilung des Sympathikus. Anaesthesist 1998, 47: 4-23.
- 9. Muller A., Kopferschmitt J., Dupeyron JP. Thérapeutique «algologiques» des algodystrophies. Douleurs, 2003; 4:2.
- 10. Merskey H., Bogduk N. Classification of chronic pain. (2nd Ed). IASP Press, Seattle, 1994.
- Sandroni P, Benrud-Larson LM., McClelland RL., Low PA. Complex regional pain syndrome type I: incidence and prevalence in Olmsted County, a population-based study. Pain 2003; 103:199-207.
- 12. Masson C. Algodystrophie: syndrome douloureux regional complexe type I. EMC (Paris), Appareil locomoteur, 14-286-A-10, 2011.
- 13. Mc Quay HJ., Analgésie préventive, pp 253-61. In : Brasseur L., Chauvin M., Guilbaud G (Eds) Douleurs. Maloine, Paris, 1997.
- 14. Schumann M., Gradl G., Andress HJ. Assessment of peripheral sympathetic nervous function for diagnosing early post-traumatic complex regional pain syndrome type I. Pain 1999; 80: 149-59.
- 15. Schuind F., Burny F. Can algodystrophy be prevented after hand surgery? Pp 455-76. In: Cooney WP, Schuind F (Eds) Hand Clinics. Upper extremity pain dysfunctions: somatic and sympathetic disorders. WB Saunders Company, Philadelphia, 1997.
- 16. Masson C, Andran M. Algodystrophie. EMC (Paris), Appareil Locomoteur, 14-286-A-10, 1999.
- 17. Berthelot JM., Current management of reflex sympathetic dystrophy syndrome (complex regional pain syndrome type I). Joint Bone Spine 2006; 73: 495-9.
- 18. Perez RS., Kwakkel G., Zuurmond WW., de Lange JJ. Treatment of reflex sympathetic dystrophy (CRPS type I): a research synthesis of 21 randomized clinical trials. J Pain Symptom Manage 2001; 21: 511-26.
- 19. Tran de QH. Duong S., Bertini P., Finlayson RJ. Treatment of complex regional pain syndrome: a review of the evidence. Can J Anaesth 2010; 57: 149-66.
- 20. Christensen K., Jensen EM., Noer I. The reflex sympathetic dystrophy syndrome response to treatment with systemic corticosteroids. ActaChir Scand 1982; 36: 653-5.
- 21. Braus DF., Krauss J., Strobel J. The shoulder-hand syndrome after stroke: a prospective clinical trial. Ann Neurol 1994; 36:728-33.
- 22. Tran D., Duong S., Bertini P., Finlayson RJ. Treatment of complex regional pain syndrome: a review of the evidence. Can J Anaesth 2010; 57(2): 149-66.

- 23. Gobelet C., Meier JL., Schaffner W. Calcitonin and reflex sympathetic dystrophy syndrome. Clin Rheum 1986; 30: 291-4.
- 24. Gobelet-Waldburger M., Meier JL. The effect of adding calcitonin to physical treatment of reflex sympathetic dystrophy. Pain 1992; 48: 171-5.
- Varenna M., Zucchi F., Ghiringhelli D. Intravenous clodronate. The treatment of reflex sympathetic dystrophy syndrome. A randomized, double blind, placebo controlled study. J. Rheumatol 2007; 27: 1471-83.
- Chauvineau V., Codine P., Hérisson C. What is the place of diphosphonates in the treatment of complex regional pain syndrome. Ann Readapt Med Phys 2005; 48 (3): 150-7.
- Van Ejis F., Stanton-Hicks M., Van Zundert J. Evidence-based interventional pain medicine according to clinical diagnoses. 16. Complex regional pain syndrome. Pain Pract 2011; 11(1): 70-87.
- 28. Varenna M., Adami S., Rossini M. Treatment of complex regional pain syndrome type Iwith neridronate: a randomized, double-blind, placebocontrolled study. Rheumatology, 2012.
- 29. Kalso E., Mc Quay HJ., Wiesenfeld-Hallin Z (Eds) Opiopidsensitivy of chronic noncancer pain, 379 pp. IASP Press, Seattle, 1999.
- Palazzo C., Poiraudeau S. Actualités dans le traitement du SDRC. Lett Med PhysRéadpt, 2012; 28:64-9.
- 31. Wallace MS., Ridgeway BM., Leung AY. Concentration-effet relationship of intravenous lidocaine on the allodynia of complex regional pain syndrome types I and II. Anesthesiology 2000; 92: 75-83.
- 32. Hord ED., Stojanovic MP., Vallejo R., Barna SA., Santiago-Palma J., Mao J. Multiple bier blocks with labetalol for complex regional pain syndrome refractory to other treatments. J Pain Symptom Manage 2003; 25: 299-302.
- 33. Cepeda MS., Carr DB., Lau J. Local anesthesic sympathetic blockhade for complex regional pain syndrome. Cochrane Database Syst Rev 2005 (4): CD004598.
- 34. Masson C., Ballard M., Vigneron AM. Algodystrophies: actualités en 1986. In: Kahn MF., Bardin T., Meyer O., Lioté F., editors. L'actualité rhumatologique. Paris: Elsevier; 2006: 241-56.
- 35. Maihofner C., Seifert F., Markovic K. Complexregional pain syndromes: new pathophysiological concepts and therapies. Eur J Neurol 2010; 17(5): 649-60.
- 36. Broseta J., Roldan P., Gonzalez Darder J., Bordes V., Barcia Salotio JL. Chronic epidural dorsal

- column stimulation in the treatment of causalgic pain. ApplNeurophysiol 1982; 45:190-4.
- 37. Barolet G., Schwartzman R., Woo R. Epiduralspinal cord stimulation in the management of reflex sympathetic dystrophy. Procedings of the meeting of the meeting of the Americain Society for Stereotactic and Fonctionnal Neurosurgery. Montreal ApplNeurophysiol 1987; 50:442.
- 38. Sears NC., Machado AG., Nagel SJ. Long-term outcomes of spinal cord stimulation with paddle leads in the treatment of complex regional pain syndrome Neuromodulation. 2010; 14(4): 312-8.
- 39. Kumar K., Rizvi S., Bnurs SB. Spinal cord stimulation is effective in management of complex regional pain syndrome I: fact or fiction. Neurosurgery 2011; 69(3):566-78.
- 40. Hyatt KA. Overview of complex regional pain syndrome and recent management using spinal cord stimulation. AANA J 2010; 78(3):208-12.
- 41. Velasco F., Carrillo-Ruiz JD., Castro G. Motor cortex electrical stimulation applied to patients with complex regional pain syndrome. Pain 2009; 147(1-3): 91-8.
- 42. Fonoff ET., Hamani C., Ciampi de Andrade D. Pain relief and functional recovery in patients with complex regional pain syndrome after motor cortex stimulation. StereotactFunctNeurosurg 2011; 89(3): 167-72.
- 43. Picarelli H., Teixeira MJ., de Andrade DC. Repetitive transcranial magnetic stimulation is efficacious as an add-on to pharmacological therapy in complex regional pain syndrome (CRPS) type I. J Pain 2010; 11(11): 1203 10.
- 44. Sigtermans MJ., Van Hilten JJ., Bauer MC. Ketamine produces effective and long-term pain relief in patients with complex regional pain syndrome type I. Pain 2009; 145(3): 304-11.
- 45. Schwartzman RJ., Alezander GM., Grothusen JR. The use of ketamine in complex regional pain syndrome: possible machanisms. Expert Rev Neurother 2011; 11(5): 719-34.
- 46. Sorel M., Lefaucheur JP., Beatrix JC. Sydrome régional douloureux complexe : intérêt du couplage de la phase vasculaire de la scintigraphie osseuse au technétium 99 avec l'efficacité de la kétamine. Lett Med PhysRéadapt 2012 ; 28 : 83-92.
- 47. Azari P., Lindsay DR., Briones D. Efficacy and safety of ketamine in patients with complex regional pain syndrome: A systematic review. CNS Drugs 2012; 1-26(3):215-28.
- 48. Khardar S., Ambady P., Venkatesh Y., Schwartzman RJ. Intramuscular botulinum toxin in complex regional pain syndrome: case series and literature review. Pain Physician 2011; 14(5): 419-24.

- 49. Moulin JF., Boureau F., Ferreri M. La prise en charge de la douleur par la médecine comportementale. In : Samuel-Lajeunesse B. Manuel de thérapie comportementale et cognitive. Paris, Dunod, 1998.
- Turk DC., Gatchel RJ (Eds). Psychological Approaches to Pain Management, 2nd edition, New York, Guilford, 2002.
- Bruehl P. Psychological Interventions. In Wilson P., Stanton-Hicks M., Harden NH. CRPS: current diagnosis and therapy. Progress in pain research and management. IASP, Press Seattle 2005; 201-16.
- 52. Fialka V., Korpan M., Saradeth T. Autogenic training for reflex sympathetic dystrophy: a pilot study. Complement Ther Med 1996; 4: 103-5.
- Frettloh J., Huppe M., Maier C. Severity and specificity of neglectlike symptoms in patients with complex regional pain syndrome (CRPS) compared to chronic limb pain of other origins. Pain 2006;
- 54. 124(1, 2): 184-9.
- 55. Albazaz R., Wong YT., Homer-Vanniasinkam S. Complex regional pain syndrome: a review. Ann VascSurg 2008; 22(2): 297-306.
- 56. De Jong JR., Vlaeyen JW., De Gelder JM., Patijin J. Pain-related fear, perceived harm-fulness of activities, and functional limitations in complex regional pain syndrome type I. J Pain 2011; 12(12): 1209-18.
- 57. Bruehl S., Chung OY. Psychological and behavioral aspects of complex regional pain

- syndrome management. Clin J Pain 2006; 22(5): 430-7.
- 58. Rothangel AS., Braun SM, Beurskens AJ. The clinical aspects of mirror therapy in rehabilitation: a systematic review of the literature. Rehabil Res Int J 2011; 34(1): 1-13.
- 59. Moseley GL. Imagined movements cause pain and swelling in a patient with complex regional pain syndrome. Neurology 2004; 62:1644.
- 60. Moseley GL. Is successful rehabilitation of complex regional pain syndrome due to sustained attention to the affected limb? A randomized clinical trial. Pain 2005; 114: 54-61.
- 61. Riout B., Bouc D., Leruyer M. Syndrome douloureux régional complexe et les signes centraux, le « programme de Moseley» d'imagerie motrice. Lett Med PhysRéadapt 2012; 28: 99-104.
- 62. Legrain V., Bultitude GH., DePaepe A., Rosetti Y. Pain, BBody and Space: what do patients with complex regional pain syndrome really neglect? Pain 2012; 153(5):948-51.
- 63. Van de Meent H., Oerlemans M., Bruggeman A. Safety of « pain exposure» physical therapy in patients with complex regional pain syndrome type I. Pain 2011; 152(6): 1431-8.
- 64. 63. Cossins L., Okell RW., Cameron H. Treatment of complex regional pain syndrome in adults: A systematic review of randomized controlled trials published from June 2000 to February 2012. Eur J Pain 2012.