A Clinical Approach to Complex Regional Pain Syndrome
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Among the most often discussed issues in chronic pain management is that which is currently termed “chronic regional pain syndrome” (CRPS), previously reflex sympathetic dystrophy syndrome (RSDS)/causalgia. Despite intensive efforts over the last 15 years to build consensus on its definition and development of objective criteria for its diagnosis, uncertainty persists regarding the pathophysiology that accounts for the heterogeneity of clinical presentations. Most agree on the challenge that these patients may present on the diagnostic or treatment sides or both. This article presents a review of the definition, classification, and epidemiology, followed by current diagnostic criteria. The article concludes by outlining principles and a basic approach to patients with suspected CRPS, including initial and long-term treatment goals.

Chronic regional pain syndrome definition

Attempting to define CRPS in a concise manner presents a challenge similar to the complexity of the disorder itself. CRPS may be defined as a multifaceted (complex) pain syndrome, usually (but not always) having an initiating noxious event in the periphery, not limited to a single nerve (regional, nondermatomal), disproportionate to the inciting event, with altered sudomotor and vasomotor function, often causing trophic alterations and loss of function [1,2]. It is important to realize that CRPS may vary “from relatively mild and self-limiting to a chronic disease with a high impact on daily functioning and quality of life” [3]. CRPS I (previously RSDS) refers to a syndrome involving a given region, unrelated to injury of a specific nerve. CRPS II (previously causalgia) refers to pain that develops after an identifiable nerve injury [4].
Epidemiology

Until there is wider consensus on the definition of CRPS, it is impossible to estimate its “true” incidence. Relative to historic literature review, estimates have varied by a 10-fold factor, from 1 in 2000 to 1 in 20 after discrete accidents, although most current literature suggests less than a 5% overall incidence after extremity injury or surgery [5]. Insofar as there have been efforts to study surgical patients in a longitudinal fashion, typical estimates for CRPS I postorthopedic surgery, for example, reveal 2.3% to 4% after arthroscopy, 2.1% to 5% after carpal tunnel surgery, and 13.6% after ankle surgery [1]. Because of changes (more inclusive) in the International Association for the Study of Pain (IASP) definition, the incidence reported subsequent to 1995 may be higher than earlier studies [6].

Other sources of incidence data have been hospital based; for example, a retrospective cohort study from the 1996-2005 Integrated Primary Care Information Project was based on a research electronic record database review of 600,000 patients in the Netherlands. This project identified 26.2 cases per 100,000 person years with a female/male ratio of 3:1 (versus Allen and colleagues [7] report a 2:1 ratio female/male with an average disease duration of 2.5 years). Women aged 61 to 70 years demonstrated the highest incidence. Upper extremity CRPS presented with a greater frequency than lower extremity CRPS, with fractures accounting for 44% of the inciting events. This incidence is four times higher than the only other population-based study in Olmsted County, Minnesota, believed to be caused by variance in the definition of CRPS [3]. Multiple studies point to certain genotypes (HLA DQ 1, A3, B7, DR 2, DRS15) as being associated with the development of CRPS [8,9].

Many authors have pointed to psychiatric comorbidities in patients with CRPS. Monti and colleagues [10] compared patients who had CRPS with a group of patients with chronic discogenic pain. Their study revealed that both groups have a significant amount of major psychiatric comorbidity, in particular major depressive disorder, and a high incidence of personality disorders.

Pediatric chronic regional pain syndrome

CRPS does not spare the pediatric population, but current concepts recognize differences relative to adult CRPS. In children, the lower limb is more commonly affected than upper extremities, with a predominance involving the foot [11]. Low and colleagues reported on a series of 20 children in a large children’s hospital over a 4-year period. Their study revealed that 90% were girls in later childhood and adolescence who had frequent initiation by way of minor trauma and lengthy time to diagnosis (with the average number of specialists having been consulted before pain management consultation being 2.7). Most of their patients required amitriptyline or
gabapentin for analgesia to allow participation in physical therapy. Most of the children responded to this combination therapy, but 40% required inpatient treatment and 20% experienced a relapse that required treatment [11].

**Initiating events**

The list of inciting events leading to the development of CRPS is impressively broad. Recognizing the frankly traumatic event or surgical procedure that preceded gross, unilateral limb alterations is easy; identifying more subtle initial presentations after no discrete event or what would otherwise be considered a trivial event is considerably more difficult. We make this statement based on the frequent delay in diagnosis and chronicity of symptoms of many patients with the ultimate diagnosis of CRPS [7].

For cases in which a surgical procedure was performed or an injury incurred and the patients develop CRPS after being followed in a longitudinal fashion, a “cause and effect relationship” has been inferred. The incidence of CRPS after orthopedic surgery has been noted [1]. Other inciting events related in the literature range from “paper cuts” to ovarian cancer, dental extraction, venipuncture, shingles, and myocardial infarction [5]. This varied list of potential triggers makes it imperative that we, as evaluating physicians, remain open minded to this diagnosis within our list of differentials for any patient who presents with atypical pain.

**Pathophysiology**

Various causes for CRPS, including exaggerated inflammatory response, genetic predisposition, and sympathetic, peripheral, or central nervous system dysfunction, have been proposed. As early as 1939, Leriche hypothesized a “vicious circle,” when foci of small nerve fibers yield self-perpetuating reflex loops in the dorsal horn of the spinal cord with central processing, resulting in exaggerated pain perception (Fig. 1) [4]. This is opposed to the so-called “neurogenic inflammation,” which is evidenced as pain, hyperemia, edema, and calor [12]. This hypothesis maintains that there is ectopic nociceptive activity, with or without a sympathetic-mediated pain mechanism, that results in the abnormal release of neurotropic factors, producing a local neuroimmune-inflammatory process. Temporary or long-term central nervous system alterations, such as central sensitization and reorganization of processing at the dorsal horn level, may result (see Fig 1; Fig. 2) [4].

Pathology specimens of patients who have CRPS have been shown to exhibit a microangiopathic process with C-fiber axonal sprouts suggestive of a true small fiber neuropathy. In summary, a traumatic injury to the extremity of genetically disposed individuals may initiate a process that results in a neuropathic stalemate in the affected limb. In several instances CRPS
seems to be maintained by “plastic” mechanisms that can be switched off by early and aggressive therapeutic interventions [8].

**Relationship of sympathetic-mediated pain and sympathetic-independent pain to chronic regional pain syndrome**

The relationship between CRPS and sympathetic dysfunction has been re-defined in the last decade. Rather than direct causality, sympathetic-mediated pain is believed to be a component of a significant percentage—but not all—of CRPS cases. The concept of differentiation of sympathetic-mediated pain (SMP) from sympathetic-independent pain (SIP) is widely accepted. SIP implies a more central alteration of function, often more refractory to care. SMP generally responds to sympathetic blockade by way of epidural, intrathecal, lumbar plexus, or peripheral routes [13]. In particular, vasomotor and thermoregulatory instability that responds to sympathetic blockade is thought to be diagnostic of SMP [13]. It is also true, however, that placebo-controlled studies demonstrate sympathetic blockade to be no more effective than placebo (although the benefit from “placebo blocks” tends to be of shorter duration) [14]. One hypothesis that may explain and reconcile both factors as contributing to the cause of CRPS is that SMP may predominate in the early stage of the disorder. Over time, SMP and SIP mechanisms may be present together and eventually be replaced by SIP alone (if left untreated) [15].
Understanding the subjective signs of patients with atypical pain is critical to the foundation of ruling out or confirming the diagnosis of CRPS. We wish to emphasize that ultimately, CRPS is a clinical entity and that no single test is sufficient to make a diagnosis. Because there has been a great deal of ambiguity and lack of consensus about diagnosis, our recommendation is that the criteria of IASP as modified by Bruehl and colleagues [16] be highly considered because they have been derived from an international consensus group whose criteria are reported as having high sensitivity and specificity and they have undergone formal validation study.

In addition to diagnostic criteria that define the disorder, CRPS may be staged by the predominant physiologic alterations that characterize its evolution. The early, acute/hyperemic stage of CRPS (stage I) is characterized by hyperalgesia, vasomotor and sudomotor dysfunction, and edema. The dystrophic stage (stage II), which is said to occur 3 to 6 months after the onset, is characterized more by increasing pain and sensory dysfunction, with the addition of motor and early trophic alterations. Stage III (atrophic stage) is characterized by decreased pain but increasing motor and trophic
alterations [6]. The most current studies suggest that these three stages may not of necessity develop in a sequential fashion but may be three distinct subtypes. This hypothesis suggests that subtype 1 is a relatively limited syndrome, with vasomotor alteration predominating. Subtype 2 is relatively limited with neuropathic pain/sensory alterations, whereas subtype 3 is a more profound disorder with osteopenia and motor and trophic signs [6].

Box 1 summarizes the subjective and objective clinical findings of the modified IASP criteria. Please note that these findings are divided into symptoms reported by the patient and findings evaluated and observed by the examiner. Table 1 summarizes decision rules, sensitivity, and specificity for the criteria.

This part of the article provides further description and explanation of the subjective symptoms and objective tests that may support the diagnosis of CRPS.

Subjective symptoms

The first half of the IASP criteria requires subjective description from patients. Pain out of proportion to the inciting event is historically the criterion that almost all clinicians agree is prerequisite to the diagnosis. A discrete injury or surgery is not essential for the diagnosis. Hyperesthesia/allodynia by history may present with patients describing light touch or thermal stimulation as being intolerable (Fig. 3). They describe vasomotor alterations as involving changing color or volume of the involved extremity. Early in the course of the disorder this may involve subjective swelling, whereas later in the disease course reduced muscle bulk and relative atrophy may be evident. Patients may describe sudomotor alterations with edema and altered sweating of the part. Motor/trophic alterations may present as subjective weakness or tremor and atrophic alterations present within the skin or nails.

Physical examination

The second half of the IASP criteria requires direct examiner observation corresponding to the categories mentioned previously. Allodynia (defined as innocuous stimuli perceived as painful) may be assessed with light, manual touch or cutaneous stimulation with a soft brush (mechanical allodynia), whereas temperature allodynia may be assessed with warm and cool test tubes applied to the skin. Vasomotor alterations may be assessed with infrared thermography, which has been cited as being significant when temperature differences between contralateral limbs are $\pm 1.5^\circ$C. Normals may vary by $0.5^\circ$C for contralateral asymmetries, and normal posttraumatic asymmetry often varies by $1.0^\circ$C [17]. Sudomotor/edema alterations may be assessed with the Quantitative Sudomotor Autonomic Reflex Testing. This test measures resting skin temperature, resting sweat output, and stimulated sweat.
Box 1. Proposed clinical diagnostic criteria for chronic regional pain syndrome

General definition of the syndrome:
CRPS describes an array of painful conditions that are characterized by a continuing (spontaneous and/or evoked) regional pain that is seemingly disproportionate in time or degree to the usual course of any known trauma or other lesion. The pain is regional (not in a specific nerve territory or dermatome) and usually has a distal predominance of abnormal sensory, motor, sudomotor, vasomotor, and/or trophic findings. The syndrome shows variable progression over time.

To make the clinical diagnosis, the following criteria must be met:
1. Continuing pain that is disproportionate to any inciting event
2. Must report at least one symptom in three of the four following categories:
   - Sensory: reports of hyperesthesia and/or allodynia
   - Vasomotor: reports of temperature asymmetry and/or skin color changes and/or skin color asymmetry
   - Sudomotor/edema: reports of edema and/or sweating changes and/or sweating asymmetry
   - Motor/trophic: reports of decreased range of motion and/or motor dysfunction (weakness, tremor, dystoria) and/or trophic changes (hair, nail, skin)
3. Must display at least one sign at time of evaluation in two or more of the following categories
   - Sensory: evidence of hyperalgesia (to pinprick) and/or allodynia (to light touch and/or temperature sensation and/or deep somatic pressure and/or joint movement)
   - Vasomotor: evidence of temperature asymmetry (>1°C) and/or skin color changes and/or asymmetry
   - Sudomotor/edema: evidence of edema and/or sweating changes and/or sweating asymmetry
   - Motor/trophic: evidence of decreased range of motion and/or motor dysfunction (weakness, tremor, dystonia) and/or trophic changes (hair, nail, skin)
4. There is no other diagnosis that better explains the signs and symptoms

output (via iontophoresis) [18]. In terms of motor findings, guarding and limitation of range of motion in the involved extremity may be subtle or prominent while delay in diagnosis and increasing chronicity may be associated with muscular atrophy. Likewise, the early hyperemia may be replaced later in the disease progression by subcutaneous loss and trophic integumentary findings.

**Laboratory testing**

No hematology or serologic tests are specific for CRPS. The ESR, C-reactive protein, and complete blood count results are usually normal.

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**Table 1**

Summary of decision rules for CRPS, sensitivity, and specificity

<table>
<thead>
<tr>
<th>Criteria/decision rules for proposed criteria</th>
<th>Sensitivity (%)</th>
<th>Specificity (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>2 + Sign categories &amp; 2 + symptom categories</td>
<td>0.94</td>
<td>0.36</td>
</tr>
<tr>
<td>2 + Sign categories &amp; 3 + symptom categories</td>
<td>0.85</td>
<td>0.69</td>
</tr>
<tr>
<td>2 + Sign categories &amp; 4 + symptom categories</td>
<td>0.70</td>
<td>0.94</td>
</tr>
<tr>
<td>3 + Sign categories &amp; 2 + symptom categories</td>
<td>0.76</td>
<td>0.81</td>
</tr>
<tr>
<td>3 + Sign categories &amp; 3 + symptom categories</td>
<td>0.70</td>
<td>0.83</td>
</tr>
<tr>
<td>3 + Sign categories &amp; 4 + symptom categories</td>
<td>0.86</td>
<td>0.75</td>
</tr>
</tbody>
</table>


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Fig. 3. A 67-year-old woman with infected left saphenectomy site 2.5 years before with intractable right foot and leg pain, edema, allodynia, and sudomotor alterations.
These normal values, however, may be of benefit in ruling out coexistent inflammatory rheumatic disease and infectious causes.

**Radiologic imaging studies and chronic regional pain syndrome**

Complementary to the diagnostic criteria, radiologic imaging studies are useful as adjunct procedures to demonstrate objective abnormalities, especially if the clinically relevant pain is unilateral.

*Plain radiography*

Plain radiographs of the involved part, although not usually diagnostic in isolation, are essential in the evaluation and care of patients with suspected CRPS. If “normal” at baseline, serial examinations may reveal significant change over time and aid in confirmation of the diagnosis. Likewise, other disorders that may also result in periarticular osteopenia may be revealed with simple, high-quality plain films (eg, inflammatory or endocrine disorders).

*Nuclear medicine scanning*

The reports of sensitivity and specificity of three-phase bone scanning for the diagnosis of CRPS type I vary to a considerable degree, likely because of study design and the duration of the disorder (sensitivity 14%–100%, specificity 50%–100%) [17,19]. What does seem consistent across multiple studies is that the sensitivity of three-phase bone scans decreases and the specificity increases with disease duration. It has been hypothesized that the peripheral pathophysiology is eventually replaced by a centralized process, accounting for reduced sensitivity of this nuclear medicine technique late in the disease course [17]. Although not uniformly diagnostic, there is considerable value to bone scanning. If, for example, an isolated stress fracture is identified without other significant tracer localization, then CRPS may be less likely the source of pain that correlates to the tracer localization. On the other hand, if a positive (“pathognomonic”) scan is obtained and other clinical criteria are met, the diagnosis may be supported long before plain radiographs reveal significant trophic skeletal alterations. Accelerated blood flow into the affected limb with increased periarticular activity during the blood pool and delayed static phases is thought by some to be pathognomonic for CRPS I (Figs. 3–6) [17].

*Single positron emission tomography*

The role of single positron emission tomography in CRPS has not been established. Alterations of regional cortical blood flow are reported in all types of pain syndromes, and cortical and subcortical structures may be abnormal. It has been hypothesized that abnormal thalamic regional cortical blood flow contralateral to the affected limb may be suggestive of CRPS [19].
MRI

MRI findings, like three-phase bone scans, seem to evolve with time. Skin thickening and effusion of adjacent joints seem to be related to acute/early phase of CRPS I, yet conclusive sensitivity and specificity are unknown. Schurmann and colleagues [17] reported that sensitivity decreased from

Fig. 4. Same patient’s flow phase of Tc MDP scan reveals asymmetric flow study.

Fig. 5. Blood pool images reveal increased flow to symptomatic right lower extremity.
43% to 14% between the eighth and sixteenth weeks of their study. Images obtained are T1 and T2 pre- and postgadolinium-diethylene triamine pentaacetic acid. In summary, the reported specificity of this imaging study is not of sufficient magnitude to recommend it as a favored technique in patients with suspected CRPS.

Treatment

Although many authors propose various treatment approaches for patients with CRPS, few randomized controlled trials have tested these suggested treatment schemes. Several randomized controlled pharmacologic trials support given medical interventions. It is generally accepted that a multidisciplinary approach is most likely to optimize clinical outcomes in CRPS patients. This may include initiation of physical medicine orders by a referring specialist or primary physician, pain management consultation, local or sympathetic nerve blocks, oral pharmacologic agents, and behavioral medicine consultation. It is generally agreed that early initiation of treatment is better than later; the avoidance of advanced, trophic limb alterations is a high priority. Toward this end, we believe that physical therapy is among the most important measures and should be instituted at the inception of care. The discussion that follows includes consensus recommendations and data from the few controlled trials published.

Physical medicine

Physical medicine and therapy are regarded by most to be necessary for functional rehabilitation of patients who have CRPS. Of the few prospective,
controlled studies published, Birklein and colleagues [20] demonstrated significantly less pain in patients receiving physical therapy (level 3 evidence).

**Pharmacologic treatment**

Many medications have been advocated in the treatment of patients who have CRPS, but few randomized controlled clinical trials have tested them. Best practice may result from recognition of the pathophysiology presumed to be etiologic for a given finding and then matching medications to the specific sources. For example, it is believed that tricyclic antidepressants and anticonvulsants may inhibit descending noradrenergic pathways and provide sodium channel blockade resulting in a reduction of hyperalgesia, whereas clonidine is postulated to provide relief of sympathetically mediated pain by reducing sympathetic outflow from preganglionic sympathetic neurons in the spinal cord or by decreasing nociceptive transmission in the dorsal horn [21,22]. Likewise, bisphosphonates inhibit bone resorption and presumably exert antinociceptive effects.

*Tricyclic antidepressants*

The analgesic effect of these medications (eg, amitryptiline and nortryptiline) may be related to inhibition of norepinephrine and serotonin reuptake; however, there are anticholinergic and arrhythmogenic effects. They are also potent sodium channel blockers that may allow local anesthetic agents (in the significant doses used for nerve blocks) to exhibit additive toxicity [4,23,24]. In a study of 41 children (open label), improvement was complete in 2 patients, significant in 21 patients, and poor in 18 patients [24]. Because of the prominent side effects of this class of medications, it is probably best to be familiar with several in the class to optimize or avoid certain effects.

*Antiepileptics*

This class of medications is among the best studied for treatment of neuropathic pain, and there is significant evidence of their effectiveness in randomized controlled trials (level 1 evidence) [25]. Many authors point to gabapentin as first drawing attention in the research community for its efficacy in the treatment of a patient who had CRPS. Gabapentin is a gamma-aminobutyric acid analog, a neurotransmitter known to modulate pain transmission and release of serotonin [5]. Other “membrane stabilizing” sodium channel blockers, such as phenytoin and carbamazepine, seem to be effective in treating patients who have CRPS [25].

*Opioids*

Care in prescribing opioids (especially short-acting medications such as propoxyphene and oxycodone) should be exercised because the duration
of therapy often requires increases risk of tolerance or dependence and central nervous system toxicity [5].

Corticosteroids

Experimenters have demonstrated short- and long-term benefit with inflammatory signs, greater than the effect on pain [5]. Christensen and colleagues [26] studied 23 patients who had CRPS and demonstrated that prednisone, 30 mg/d, was significantly better than placebo.

Nonsteroidal anti-inflammatory drugs

This group of medications may be initiated for their analgesic and anti-inflammatory effects, but little experimental evidence exists to support their use as primary agents. The benefit that is achieved is believed to be by way of prostaglandin inhibition [5].

Nifedipine

This agent is not a true sympatholytic but exerts its effect by reduction of vascular smooth muscle tone to improve arterial flow into the extremity. It may be useful in reversing vasoconstriction associated with CRPS in doses up to 60 mg/d [5,24].

Calcitonin

Calcitonin is a hormone that inhibits osteoclastic activity and results in reduced serum calcium and phosphate. The results of several randomized controlled trials have suggested pain relief and improved range of motion in patients who have CRPS, although other studies have failed to show statistical significance in this regard [25].

Bisphosphonates

The bisphosphonates are analogs of pyrophosphate and are among the only class of medications that has survived placebo-controlled studies showing statistically significant improvement with therapy. Their primary mechanism of action is by way of inhibition of bone resorption, although some researchers have hypothesized that their antinociceptive effect is by way of their ability to inactivate osteoclasts and inhibit prostaglandin E2 [25]. In particular, alendronate, pamidronate, and clodronate have shown efficacy regarding pain reduction in patients who have CRPS [25].

Alpha-adrenergics

This group of medications has enjoyed widespread use in patients who have despite a scarcity of controlled studies to demonstrate significant benefit. Among the agents in this group are phentolamine, clonidine (oral and
transdermal), reserpine, and phenoxybenzamine. Experimental data are mixed on their effectiveness [5,23]. One case series demonstrated transdermal clonidine as being effective in reducing or eliminating local CRPS-associated hyperalgesia and allodynia (level 4 evidence), although a recent systematic review was less compelling for significant benefit (level 1 evidence) [27,28].

**Topical agents**

Topical lidocaine and dimethyl sulfoxide have been reported to be of benefit in uncontrolled studies [23].

**Interventional treatments**

*Local anesthetic sympathetic blockade*

The value of local anesthetic sympathetic blockade has been long appreciated in a subset of patients who have CRPS; however, few randomized controlled trials have documented statistical significance over placebo. Two systematic reviews grouped patients with pooled data, with 29% of patients reporting more than 75% improvement in pain reduction and 41% reporting partial relief (25%–75%). They attributed these results to a placebo effect, inferring that these data are inconclusive and that further randomized controlled trials are needed [29,30].

*Spinal cord stimulation*

There has been considerable interest in this modality in treating chronic pain patients, particularly patients who have CRPS. To date, only one randomized controlled trial has been reported in patients with severe, refractory CRPS. Patients were randomized into spinal cord stimulation and physical therapy versus physical therapy alone groups; significant reduction in pain intensity was reported in the spinal cord stimulation and physical therapy group. At 2 years this group reported reduced pain and improved health-related quality of life, but there was no difference in functional outcome between the two groups [31]. The overall value of spinal cord stimulation remains an open question, with the understanding that there is considerable interest in future research in this area.

*Transcutaneous electrical nerve stimulation*

Few data exist on the efficacy of peripheral nerve stimulation in patients who have CRPS, but several authors point to the reasonableness of its use in patients with more localized pain (who fulfill criteria for CRPS II) in relation to pain reduction and enhancing rehabilitation efforts [31]. Patients who have a history of CRPS and must undergo surgery represent a special
circumstance relative to prophylaxis against recurrence. Several authors have pointed to adequate resolution of the initial episode before undertaking any further surgery. Katz and colleagues [32] reported on a group of patients undergoing knee surgery with a mean time between resolution of CRPS symptoms and their surgery of 5 months with a 47% postoperative recurrence rate. Reuben [1] provided an excellent review of considerations relative to prevention of CRPS in patients with previous diagnosis and in unselected populations. The concept regarding delaying of surgery until maximum resolution of CRPS findings was emphasized, as were perioperative regional nerve blocks, which produce chemical sympathectomy. Many other approaches have been studied, largely in nonrandomized fashion, suggesting the potential benefit of perioperative calcitonin, ketanserin (a serotonin antagonist), mannitol, and vitamin C [32].

**Essentials to the approach of patients who have chronic regional pain syndrome**

The algorithm presented in Fig. 7 represents our recommended approach to a patient who has suspected CRPS with respect to history and physical examination, diagnostic testing, and initiation of treatment. The narrative that follows serves as further explanation in addressing the needs of these patients:

1. Listen to patients and take a thorough history, allowing them to describe what they perceive may have been the inciting event or stimulus. Do not discount any part of their history or subjective symptoms as exaggeration or embellishment of their relative discomfort, especially before objective testing, which may or may not support alternative diagnoses. Remember that CRPS is ultimately a clinical diagnosis and one that is made by fulfilling certain criteria and excluding any other diagnosis that better explains a patient’s disorder. “It should be reasserted that the diagnosis of CRPS no longer rests on the response to a sympathetic block alone” [8].

2. Document, in detail, history and physical findings, diagnostics, treatment modalities, and plans for timely follow-up. It is important to document not only findings that suggest CRPS in patients with the disorder but also negative findings in patients in whom the diagnosis is excluded. Because of the potential gravity of the outcome and because a subset of these patients may have workman’s compensation claims or litigation pending, contemporaneous documentation is required for benefit of a patient’s timely care and the medicolegal protection of the provider.

3. Note a patient’s affect. Patients who have CRPS tend to exhibit significant emotional distress that is out of proportion to patients whose pain is more consistent with the antecedent injury or surgery. The following quotations are excerpted from correspondence written by a patient in
Dear Doctor:
I am a new patient. I want to tell you my medical history to save time of discussion when I see you.

I had a heart bypass surgery in 2003. My left leg was infected with MRSA and could not walk for about four months after the surgery. Now I have pains in both legs and ankles. My legs and feet are so sensitive to touch and feel cold always. Walking is hard, getting up from a chair, climbing the stairs, getting into a van are equally hard. I have been seeing (a vascular surgeon) for a few years now.

Suspected CRPS

Fulfills IASP research criteria, “classic” CRPS I or II

Atypical pain but criteria not met

Initiate care: analgesics, PT, peripheral nerve blocks as appropriate

Diagnostics: 3-phase Tc scan, +/- thermography, +/- QSART, CBC/ESR/CRP and treat with PT, analgesics, antiinflammatories as indicated

Improved

No improvement or

Progressive pain, autonomic dysfunction, regardless of whether all research criteria met

No improvement or

Follow-up to rule-out decline and reconsideration of referral for Pain Mgmt. Consultation PRN

Pain Management Consultation

Sympathetic or regional nerve blocks as indicated

Oral sympatholytics, TCAs, bisphosphonates, corticosteroids as indicated

Physical therapy

Behavioral consultation as indicated

Long-term follow-up for general support

Surveillance regarding reoccurrence of CRPS

this practice in whom CRPS was suspected and confirmed, revealing the significant emotional substrate on which the somatic alterations were noted:
Something they are doing in the therapy does not agree with my body. What is wrong?

“Effective management of CRPS requires that …psychosocial and behavioral aspects be addressed as part of an integrated multidisciplinary team approach” (Fig. 8) [33].

Paying attention to the “basics” of preoperative anesthetic and surgical interviews and optimizing perioperative pain management may go a long way in allaying the fears of a certain subset of our patients who, because of genetic traits, personality disorders, or reasons incompletely understood, may be at increased risk of developing CRPS relative to age-matched individuals.

4. Take note of loss of function because it is often a prominent component of CRPS. In few disorders is it more important to observe a patient’s open kinetic chain function and gait and station. The antalgic gait of a patient who has CRPS likely differs in a significant way from that of a patient with, for example, an uncomplicated healing fracture.

5. Once the diagnosis is suspected or confirmed, act in a timely, deliberate, and aggressive manner to get the patient to appropriate care. This often
involves physical medicine and pain management consultation, although treatment should not be delayed and should be initiated by any provider capable of starting even the most basic modalities, such as local nerve blocks (e.g., ankle, popliteal), narcotics or nonsteroidal anti-inflammatory drugs, and physical therapy, because there are likely numerous instances of “impending” CRPS that can be affected by early intervention. This latter concept is not unique to our individual practice but has been cited as being clinically relevant by numerous authors [1,5]. In regard to Fig. 7, which suggests certain therapies and improvement versus progression of pain and autonomic dysfunction, we have not assigned a “hard and fast” timeframe after which pain management consultation is essential. This is purposeful because patients vary as to response rate to given treatments. Improvement or worsening is most often evident in units of several weeks to 1 month, however. If improvement is unclear, it is probably wise to obtain a consultation. Relative to pain management consultation, develop a relationship with a provider or group of providers who have the interest, ability, and resources to deal with these complex and potentially difficult diagnostic and treatment issues.

6. Once you initiate therapy for CRPS, it is wise to see patients through the remainder of their care. This does not imply that a single provider administers all care, because that is often impractical or impossible. Rather, one provider must take responsibility to ensure that patients follow through with all others who may contribute to their care. (It is not uncommon in our experience for patients to assume a “hopeless” orientation, assuming that they will never be “well,” placing additional burden on us as providers to correspond with other caregivers, ensuring that follow-up has been attended). Even if an initial episode seems resolved, patients who have CRPS are at risk for recurrence and should have easy access to reassessment, preferably by a provider who is familiar with their initial clinical presentation.

7. Once care is initiated, reassessment should be frequent enough to allow patients to communicate the relative success or failure of therapeutic interventions. This regular and timely communication facilitates how “deep” the treatment team may need to proceed into the therapeutic algorithm (relative to pharmacologic versus interventional modalities). “Failure to progress in a reasonable amount of time should cause the clinician to consider pharmacotherapy or stronger medications, blocks, or psychotherapy or more psychotherapy, depending on the situation”[24]. Repeating noninvasive or minimally invasive testing to document objective progress toward normalization of physiologic functions may aid the patient and the provider in moving toward further resolution of the disorder.

8. Once treatment is initiated, ensure that adequate behavioral consultation is available to patients. It cannot be overemphasized how the emotional component of CRPS requires active intervention in many patients.
who require solid support relative to reduction of anxiety, discussion of prognosis, coping mechanisms for activities of daily living, and pain management strategies (Fig. 8).

Summary

The goal of this article is to provide an introductory look into current concepts regarding chronic regional pain syndrome. Although great advances have been made over the last 15 years, we are far from a complete understanding of this disorder. Goals for the reader are clinical recognition and early treatment intervention when presented with a high index of suspicion in the clinical setting. It is highly recommended that the podiatric surgeon keep abreast of this topic in the future as new developments arise.

Further readings


References