Case Reports

VENIPUNCTURE COMPLEX REGIONAL PAIN SYNDROME TYPE II

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Abstract. *Venipuncture Complex Regional Pain Syndrome Type II (VP-CRPS II) is a rare and unpredictable complication of venipuncture. It is the manifestation of a minor injury leading to a severe form of CRPS. It should not be mistaken for benign forms of hematoma or phlebitis without CRPS. There is no definite causal relation with the type of needle used, nor with number of attempts at IV insertion. It is usually a rare complication of the needle accidentally injuring the microscopic microvascular C-thermoreceptor sensory nerve. Lack of experience and severity of the trauma are not proven risk factors, and there are no known preventive measures. Accidental infiltration of chemical irritants can instigate the VP-CRPS, unless the injection is discontinued immediately. Early diagnosis and proper treatment provide significant pain relief. Multimodal treatment is essential. Surgical procedures, especially sympathectomy, may exacerbate the condition and lead to irreversible therapeutic failure.*

Descriptors. *causalgia, CRPS Type I and II, neuroinflammation, sympathectomy, venipuncture*

AJPM 2001;11:112-124. Received: 1-30-01; Accepted: 5-23-01

INTRODUCTION

Venipuncture Complex Regional Pain Syndrome Type II (VP-CRPS II) is a rare complication of the most commonly practiced invasive procedure in medicine IV insertion (1). In both medical and dental literature, this condition has been recognized as a sudden onset of a painful and persistent neuropathic pain due to attempted venipuncture (1-23). It is the complication of a minor injury causing one of the most severe forms of CRPS. VP-CRPS II should not be mistaken for common benign and short-lived complications of venipuncture, such as focal IV infiltration, hematoma, transient phlebitis, vasovagal syncopal attacks, or transient neuropathic pain (1-7). The term VP-CRPS II should be associated with just those patients suffering from the full picture of CRPS after venipuncture. VP-CRPS II is due to the traumatic disturbance of microcirculation generated by thermoreceptor sensory nerves in the wall of blood vessels, resulting in causalgic pain and neuroinflammation (CRPS II) (8-11).

The occurrence of venipuncture complications has been estimated at one in 25,000 patients in the general

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population (12). However, because of difficulty in diagnosis, the exact incidence cannot be accurately assessed. On the other hand, the more common benign complications have been reported to occur far more frequently than the VP-CRPS II. The incidence of the benign complications has been reported to be as frequent as 14.2% of 4,050 venipunctures (1). This is in contrast to the reported 3.4 % incidence of VP-CRPS II in venipunctures (1). The occurrence of benign symptoms has been reported in 18% of 40,000 blood donors, phlebitis in 15%, bruises and hematomas in 9-16%, and vasovagal symptoms in 5% (1-4,6,13,14).

The stereotypical VP-CRPS II is easily distinguishable from the benign complications (Table I). The pain is sudden, instantaneous, intolerable, and causalgic in nature (15). The pain is described by patients as severe, burning, lightning-like, piercing, deep, searing, stabbing, electric shock-like, or crushing (16-23). The patient routinely demands immediate removal of the needle. The causalgic pain is usually accompanied by regional neuroinflammation in the form of edema, erythema, and, infrequently, bulbous lesions and skin ulcers (17,24-30).

Table I. Clinical characteristics of venipuncture injuries in 22 patients.

Pain	
a. causalgia	22 patients*
b. hyperpathia and allodynia	18 patients
Vasomotor dysfunction	22 patients*
a. flexor spasm and vasospasm	19 patients
Neuroinflammation - (after venipuncture injury)	22 patients*
a. neurodermatitis	13 patients*
b. skin eruptions	5 patients*
c. bulbous skin ulcers	3 patients*
Remote effects	
a. pelvic inflammation, spread of CRPS	12 patients*
b. frozen shoulder (shoulder hand syndrome)	11 patients
c. vascular headaches, vertigo, blurred vision	10 patients
d. joint pain aggravated by drop in barometric pressure	9 patients

* There were more than one complication in each patient.

The disease has a tendency for rapid deterioration in a matter of a few weeks to months. With passage of time, flexor spasm, muscle weakness, tremor, myoclonic jerks, and vasoconstriction (cold extremity) develop (Table I) (16-23). VP-CRPS II deteriorates from a Stage I to Stage III (I - dysfunction; II - dystrophy; III - atrophy) in a span of a few weeks to a few months rather than the extended several months to years duration seen in other forms of CRPS (Table II) (17).

Table II. Average duration of development from stage I to stage III.

Types of CRPS	Duration
VP CRPS II (22 patients)	4 1/2 months
CRPS treated with amputation	10 months
CRPS after carpal tunnel surgery	14 months
CRPS in electrical injury	25 months
CRPS treated with no surgery	29 months

DIAGNOSIS AND PATIENT IDENTIFICATION

From 1989 through 2000, a total of 22 VP-CRPS II patients were referred to Neurological Associates Pain Management Center for evaluation. The diagnostic tests consisted of electromyography (EMG), nerve conduction velocity (NCV), somatosensory evoked potentials (SSEP), infrared thermal imaging (ITI), and quantitative thermal sensory test (QST). At the time of the first visit, each patient had already been afflicted by the disease for months to years.

Anatomical structures at risk. The areas at risk in order of occurrence were antecubital regions (9 patients), hand (10 patients), wrists (2 patients), and abdominal wall (1 patient) (Table III). With VP-CRPS II, other less common areas of the body also may be involved (1,12,16,31-34).

Table III. The nerves and blood vessels injured in VP CRPS II.

Case Number	Age/Sex	Site of Injection Nerve Damage	Blood Vessel Punctured	Type of Procedure	Chemical Injected
1 AC	43/F	median nerve	median vein	blood test	
2 CM	49/F	L. antecubital median nerve	median basilic	blood test	
3 LK	44/F	dorsal sensory of hand radial	posterior ulnar vein	IV infiltration	
4 JAR	45/F	dorsal sensory of hand radial	radial vein	IV diazepam	diazepam
5 DS	37/F	superficial sensory radial	median cephalic	IV thiopental	thiopental attempt
6 MT	49/F	superficial radial	median cephalic	IV thiopental	thiopental attempt
7 AR	23/F	L. antecubital median nerve	median basilic	levonorgestrel IV Norplant® implant	levonorgestrel Norplant®
8 LW	37/F	L. antecubital median nerve	median basilic	IV infiltration	diazepam
9 TE	29/F	ulnar nerve	ulnar artery	nerve block attempt at wrist	corticosteroid attempt
10 SP	23/F	dorsal sensory of hand radial	radial vein	IV infiltration	
11 SC	25/F	superficial radial	cephalic	arteriol blood gas test	
12 CB	48/F	L. antecubital median nerve	median basilic	IV diazepam	diazepam
13 BC	45/M	L. antecubital median nerve	median basilic	blood test	
14 PB	42/F	superficial radial	median cephalic	nerve block attempt at wrist	corticosteroid attempt
15 JG	40/F	L. antecubital median nerve	median basilic	IV infiltration	IV colchicine
16 JH	46/M	dorsal sensory of hand radial	posterior ulnar vein	blood test	
17 DB	34/F	L. antecubital L. lateral antocubital cutaneous	median cephalic	blood test	
18 JJ	13/F	R. antecubital ulnar nerve	median basilic	blood test	
19 CB	51/F	dorsal sensory radial	radial vein	IV infiltration	
20 KB	42/F	dorsal sensory radial	radial vein	IV infiltration	
21 PM	38/F	L. T-10 intercostal nerve	L. superficial abdominal vein	IM analgesic injection	morphine
22 SW	42/F	L. antecubital ulnar nerve	median basilic	blood test	

Diagnosis. Patients suffering from benign complications of venipuncture (*e.g.*, syncopal attack, transient phlebitis, bruises or hematomas not accompanied by CRPS symptoms and signs) were excluded from this study.

CRPS is a clinical diagnosis with four main diagnostic criteria including (*i*) neuropathic pain, (*ii*) flexion deformity and vasospasm, (*iii*) neuroinflammation, and (*iv*) disturbance of the limbic system function (*e.g.*, insomnia, agitation, poor memory). The first three criteria have been used at the Mayo Clinic (35,36). The authors added a fourth to arrive at a more restricted diagnosis (17,37). All 22 VP-CRPS II patients met the four diagnostic criteria in varying severity.

The neuroinflammation fluctuates from day to day depending on the level of the stress. It is not consistently present at the time of physical examination. According to previous reports, neuroinflammation has been observed in one-third of patients undergoing examination; the other two-thirds may present with a history of neuroinflammatory changes (35). The neuroinflammation presents in the form of edema, skin lesions, neurodermatis, or bulbous ulcers (Table I) (17,24,26,27,37-46).

No single laboratory test can be expected to diagnose CRPS in 100% of patients (17). As is usually the case with CRPS patients, NCV may be normal in VP-CRPS II patients (47). The EMG could not be performed on 12 of 22 patients in the present study due to severe allodynic pain. The other

ten patients had normal EMG. These findings should not be misconstrued as no nerve damage or dysfunction in CRPS. The NCV measures the conduction velocity of myelinated somatic motor or sensory nerve fibers such as median, ulnar, or peroneal nerves. In contrast, the nerve dysfunction in CRPS involves the thermal sensory microscopic nerve fibers in the wall of the blood vessels (48-51). Expecting such unmyelinated C-thermoreceptor nerve damage to reflect itself on NCV tests is tantamount to expecting a standard microscope not an electron microscope to identify a virus.

The QST was abnormal in 15 of the 22 patients, pointing to C-thermoreceptor microcirculatory sensory nerve dysfunction in the involved extremity. The infrared thermal imaging (ITI) showed bilateral hypothermia more severely on the injured extremity in all 22 patients. The area of the venipuncture trauma was too small to manifest itself in the background of hypothermic extremity in 11 of 22 patients. In one patient (Case 1, Table III), the attempt at venipuncture injured the median nerve in the antecubital region. The lesions were too small to be detected with EMG and NCV.

The scintigraphic triphasic bone scan test was performed on 15 of 22 patients prior to referral to the clinic. Only 8 of the 15 were diagnosed as CRPS by the scintigraphic bone scanning. This is compatible with the lack of diagnostic sensitivity of this test in CRPS patients as reported in the literature. A meta-analysis has documented the test to be diagnostic in 55% of the patients (52), while another study determined the bone scan to be positive in no more than 25% of patients (35).

TREATMENT PROTOCOL

The aim of treatment is to improve the patient's quality of life by relieving pain and suffering while avoiding any harmful surgical procedures, hence preventing any further deterioration and spread of CRPS to other regions (*i.e.*, other extremities) (28,53-58). The treatment protocol included multimodal disciplines including detoxification from multiple narcotics. Treatment was with tramadol, analgesic antidepressants (*e.g.*, trazodone, doxepin, desipramine), and anticonvulsants (non-generic carbamazepine for causalgic pain, gabapentin for burning pain, and non-generic clonazepam for myelogonic myoclonus due to spinal cord sensitization) (17,25), and nerve blocks (epidural, brachial plexus, and paravertebral nerve blocks containing methylprednisolone).

VP-CRPS II is a rapidly deteriorating disease; in a matter of a few months, it changes from sympathetically maintained pain (SMP) to sympathetically independent pain (SIP). By the time the patient is referred to the clinic, it is too late to get any benefit from sympathetic ganglion blocks. Physical therapy, hydrotherapy, avoidance of prolonged inactivity (such as hospitalization), and avoidance of application of cast to the extremity are the additional minimum required treatments.

For severe neuroinflammation causing water retention and pelvic inflammation, treatment with IV mannitol, bumetanide, or zaroxolyn proved effective. For advanced autoimmune neuroinflammation causing bulbous lesions, treatment with IV immunoglobulin has been reported to help healing and clearance of the skin lesions *(Figure 1C)* (17,25-27).

RESULTS

Multidisciplinary treatment with analgesic antidepressants (*e.g.*, trazodone, desipramine, doxepin) and anticonvulsants (*e.g.*, carbamazepine), proper physical therapy, and nerve blocks (*e.g.*, epidural, plexus nerve blocks) improved and reversed the stages of the condition, from Stage III to II to I, in 16 patients. The intractable tendency of VP-CRPS II should not discourage the treating physician from assertive treatments.

The complex regional vasoconstriction in the extremities has a tendency to spread regionally to craniocervical vasculature, leading to symptoms of vertebral basilar artery insufficiency and brain stem dysfunction (*i.e.*, vertigo, ataxia, diplopia, and blurred vision) (59-61). This phenomenon was noted in 15 of the 22 patients.

Pain management. CRPS is a disease characterized by

intolerance to stressors such as inactivity, insomnia, emotional stress, or excessive activity. The stress accelerates the deterioration due to the disease. Monotherapy, such as gabapentin alone or only opioid treatment, cannot be expected to provide any significant relief for the multiple complications of VP-CRPS II. The treatment should be multidisciplinary in the form of physical therapy, nerve blocks, analgesic antidepressants, and anticonvulsants. For the management of pain, the use of antidepressants such as trazodone or desipramine in therapeutic doses provides analgesia and natural sleep. Anticonvulsants play a major role in the treatment of CRPS II. Eight VP-CRPS II patients developed myoclonic jerks of the extremities due to spinal cord sensitization (49-51). All eight patients recovered from the myoclonic seizures after treatment with Klonopin®. Tegretol® proved to be more effective for CRPS II causalgic pain (16), while gabapentin was the treatment of choice for burning pain and allodynia (17). The neuroinflammation responded best to cervical or lumbar epidural nerve blocks.

Stellate and other sympathetic ganglion blocks usually provide a few hours or days of relief. The blocks are usually performed with the use of local anesthetics. With this technique, they are more diagnostic than therapeutic. Repetitive ganglion blocks may lead to repetitive damage to the sympathetic ganglion nerve cells, leading to "virtual sympathectomy" (17,62,63). Other types of nerve blocks, especially cervical or lumbar epidural, caudal, paravertebral, and brachial plexus blocks containing local anesthetic and corticosteroids such as methyl prednisolone acetate, or an equivalent dose of Celestone, were found to be effective in reducing the pain and neuroinflammation, as well as increasing the extremity temperature (up to 2-6°C). The therapeutic effect of these blocks usually lasted up to 2 to 4 months, rather than a few hours or weeks which is the therapeutic duration achieved with ganglion blocks.

RESULTS OF SURGICAL TREATMENT

Two of the 22 patients had undergone sympathectomy which caused a severe flare up, as well as spread of CRPS to other regions, and development of bulbous skin ulcers in the extremities which eventually improved with the nerve blocks (*Figure 1A*) (54). Review of the literature has shown, in the long run, that sympathectomy has not been successful in CRPS patients (37,64,65). Two patients had undergone carpal tunnel surgery in spite of the regional nature of the neuroinflammation and normal NCV tests which did not confirm median nerve entrapment. Another two patients had undergone skin biopsies of the bulbous lesions. Immediately after the biopsies, the lesions spread to other parts of their bodies and even confinement to wheelchair (Table IV). All six patients who underwent surgery or skin biopsies had severe neuroinflammation and marked clinical deterioration (Table IV).

Neuroinflammation. Treatment with magnesium sulfate soaks or oral milk of magnesia acts as an osmotic gradient (66-68). Calcium channel blockers provide similar benefits. IV mannitol therapy is effective as an intracellular dehydrant (17,37,69-71). In addition, it prevents the need for surgical procedures for entrapment neuropathies or thoracic outlet syndrome secondary to neuroinflammation (17).

Prevention. There is no known measure to prevent VP-CRPS II. However, avoidance of bed rest, ice application, immobilization, biopsy, and other surgical procedures help prevent deterioration and spread of the medical problem. The present-day therapies do not cure the patient; rather they help to prevent and slow down the deterioration.

As previously stated, VP-CRPS II is the manifestation of a minor injury causing a severe form of CRPS (16,18-23). A minor trauma is more likely to cause CRPS than a major injury such as fracture (11,72,73). Major trauma is more likely to stimulate large myelinated somatic sensory nerve fibers which tend to overshadow the neuropathic type of pain — pain originating from the unmyelinated perivascular sympathetic sensory nerve fibers (48-51). The inhibition and overshadowing of the neuropathic pain due to a major trauma reduces the likelihood of development of CRPS (74).

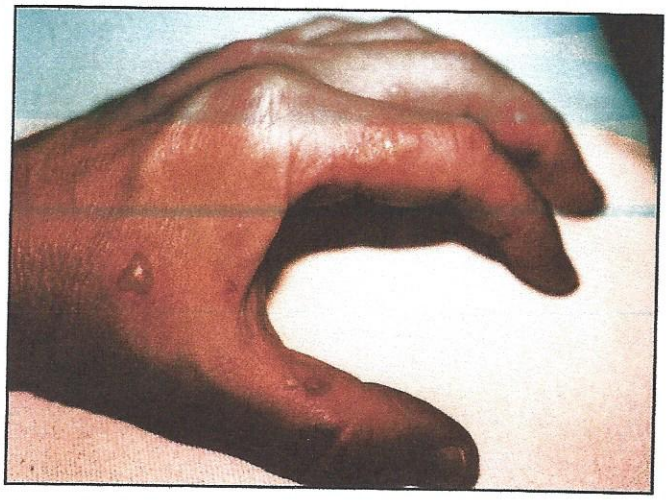

Figure 1A (Case # 13, Table III): 9/22/96: Venipuncture CRPS II, five months after blood test resulted in neuroinflammatory bulbous lesions.

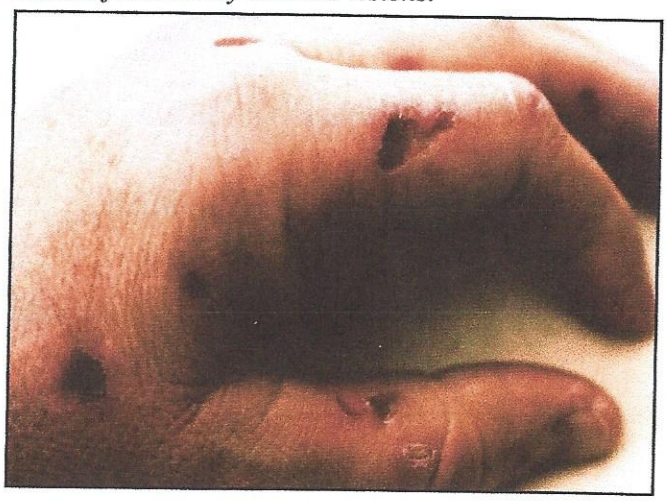

Figure 1B (Case # 13, Table III): 9/27/96: Lesions become ulcerated.

The therapeutic mode played a major role in the outcome of the disease in the patients studied. Lack of proper diagnosis and lack of proper treatment results in a poor prognosis. Inactivity and chronic application of ice (11 patients) to the area of injury led to cryogenic damage to the sensory nerves (17,37,75-80). The long-term application of ice in CRPS has the tendency for Valerian degeneration and segmental demyelination of the sensory nerves (76,77,80). Another aggravator was application of cast to the extremity (4 patients) which increased the neuroinflammation and secondarily caused edema and poor circulation in the extremity surrounded by the cast.

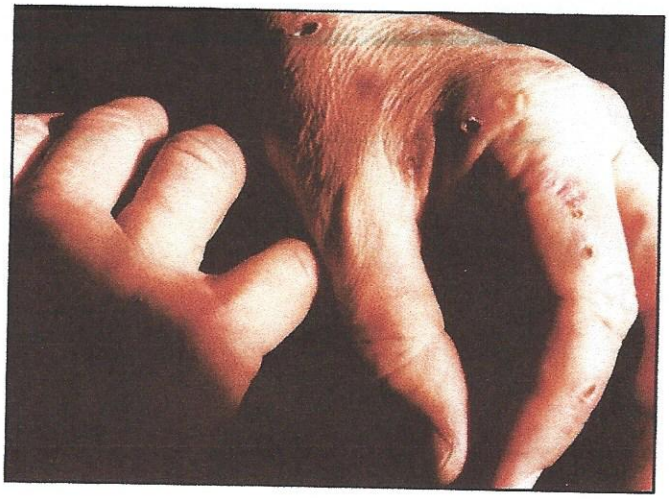

Figure 1C (Case # 13, Table III): 11/1/96: The lesion healed after treatment with IV mannitol and IV immunoglobulin treatment.

Treatment with extensive bed rest during hospitalization or excessive bed rest partially due to treatment with strong narcotics and sedatives (12 patients) aggravated and accelerated the deterioration and led to spread of the VP-CRPS II (54,55). Elective, non-emergent surgical procedures, especially sympathectomy (2 patients), carpal tunnel surgery (2 patients), and skin biopsies (2 patients) played major roles in further deterioration of the disease (Table IV) (17,37).

DISCUSSION

The VP-CRPS II is an example of a minor injury selectively damaging the thermoreceptor nerves in the wall of venules or arterioles, leading to severe complications such as neuroinflammation. The neuroinflammation in VP-CRPS II has been mistaken for phlebitis. Whereas earlier publications use the phlebitis model to explain the pathologic changes, more recent research has demonstrated neuroinflammation to be the main catalyzer

(1,43,44,81). Thrombosis, as a complication of neuroinflammation, has been noted as the originator of neuropathic pain including CRPS (82). Neuropathic pain, including CRPS, is usually complicated by neuroinflammation in varying degrees (82). The neuroinflammation in neuropathic pain was first reported by Mitchell in 1864 as "shiny skin" and, later by Sudeck in1942 (26,27,38,39,83). The neuroinflammation may lead to bulbous and ulcerative lesions (Cases 2, 13, and 20, *Figure 1* and Table III), sterile abscess, and edema mistaken for entrapment neuropathy. The neuroinflammation is a manifestation of sympathetic modulation of the immune system (84). In one patient (Case 13, *Figure 1C*), the bulbous and ulcerative lesions were successfully treated with IV immunoglobulin and IV mannitol (17).

The review of the venipuncture literature reveals no specific, preventable, and predictable precipitating factor (12,16,19-21,32,33,85-87); however, the injection of 50% dextrose or anticoagulant drugs may represent added risks (33). IV diazepam injection is another risk factor (20,32,43,88). However, the majority of patients have developed VP-CRPS II during blood test and blood transfusion without the injection of any chemical (1,2,16).

Preexisting CRPS seems to be a probable risk factor. Five of the 22 VP-CRPS II patients referred to the clinic already had history of prior CRPS I which was aggravated by IV insertion into the dorsum of the hand. Two other patients had undergone carpal tunnel nerve blocks for treatment of CRPS, and one patient had undergone multiple trigger point injections around the wrist with instantaneous flexor spasm and aggravation of the preexisting CRPS.

Ten patients developed VP-CRPS II during the injection of potentially irritant chemicals and devices such as IV diazepam (3 patients), IV colchicine injection (1 patient), thiopental attempt (2 patients), corticosteroid attempt (2 patients), morphine (1 patient), and levonorgestrel implants (Norplant®) insertion (1 patient) in manners similar to previous reports (15,20,32,43,88-90).

Etiologic factors: trauma. Traumatic injury due to needle insertion has been cited as an etiologic factor in VP-CRPS (56,91). However, the review of this subject cites, but does not document any proof that the needle size or the intensity of the needle insertion play any role in the development of VP-CRPS II (1,33,34,81,92). The short beveled needle has been found to be more traumatic then the long beveled needle (81,92); however, the short beveled needle insertion pain does not last longer then 28 days (92).

Etiologic factors: thermoreceptor injury. In VP-CRPS II, the haphazardous C-thermoreceptor nerve fiber stimulation due to needle insertion, or any other minor trauma, is left uninhibited by simultaneous somatic nerve stimulation (such as a coincidental fracture). This results in a pure and uninhibited sympathetic norepinephrine (NE) sensitization (48-51,93). The purely uninhibited stimulation of C-thermal receptors can also lead to severe neuroinflammation and bulbous and ulcerative lesions (26-28,37,39,41). However, this phenomenon is extremely rare, and there is no specific precaution to prevent it. The chance of injuring such nerves with needle insertion is remote (48-51).

Etiologic factors: inexperience. The inexperience of a venipuncture operator has been considered by some to be a factor (21,33). However, there is no definite evidence to prove such a claim. VP-CRPS II appears to be as likely to develop in the hands of a novice as in the hands of an expert, because no human can visualize or feel the microscopic C-thermal receptors in the perivascular or intravascular region.

Etiologic factors: hematoma. The development of a blood clot in the area of needle insertion in VP-CRPS II patients has been blamed as the pathologic factor in CRPS II (7,16,81). In the present series of 22 VP-CRPS II patients, hematoma developed several minutes after the causalgic pain, heralding the onset of VP-CRPS II complications. This makes it very unlikely for the hematoma to be the causative factor in CRPS II. In addition, Horowitz has reported hematoma developing hours after the venipuncture pain and injury in 6 patients, suggesting it more unlikely to be the causative factor (16).

Table IV. The influence of surgical versus non-surgical treatment on CRPS stages during 1 year or longer follow-up of 824 (including the VP CRPS II) patients (17). Note high percentage of late stage CRPS in surgically treated patients.

CRPS I and II Surgical Group			
Surgical Treatments (% of 824 patients)	Stage I † number of patients	Stage II number of patients	Stage III-IV ± number of patients
Amputation * 11 patients (1.3%)	0 (0%)	2 (19%)	9 (81%) (P = 0.025)
Chemical sympathectomy 13 patients (1.5%)	0 (0%)	2 (15.4%)	11 (84.6%)
Surgical sympathectomy 22 patients (2.6%)	0 (0%)	3 (13.6%)	19 (86.4%)
Other surgical treatments ‡ 295 patients (36%)	24 (8%)	106 (36%)	165 (56%) (P < 0.001)
CRPS I and II Non-Surgical Group			
	Stage I † number of patients	Stage II number of patients	Stage III-IV ± number of patients
No surgery 528 patients (64%)	164 (31%)	190 (36%)	174 (33%) (P < 0.001)
VP CRPS II Group (At first visit to clinic) (The surgical procedures on 6 patients preceded their first visit)			
	Stage I † number of patients	Stage II number of patients	Stage III-IV ± number of patients
Venipuncture CRPS II 22 patients	0 (0%)	4 (18%)	18 (82%)
VP CRPS II Group (After last visit to clinic)			
	Stage I † number of patients	Stage II number of patients	Stage III-IV ± number of patients
Venipuncture CRPS II 22 patients	6 (27.5%)	9 (41.3%)	7 (31.2%) §

* Many patients had more than one treatment modality which change the total percentage.
‡ Rotator cuff surgery; thoracic outlet surgery (rib resection); epicondylectomy; nerve exploration; nerve decompression.
± Stage I = dysfunction; Stage II = dystrophy; Stage III = atrophy; Stage IV = autonomic system failure.
† Depending on treatment modality, stage III may reverse to stage I and vice-versa.
§ Six of seven irreversible stage III patients had undergone surgical procedures.

Etiologic factors: chemical infiltration. Injections of diazepam, colchicine, tetracycline, other antibiotics, and chemical block with alcohol in the soft tissues have been reported to cause chemical damage and neuroinflammation (17,20,32,43,88-90,94). Such complications may be avoided if the IV injection is discontinued immediately after the first burning causalgic pain develops. While the size of needle and excessive volume of blood transfusion have been blamed as causative factors (2,16,21,92,94,95), a detailed review of the literature shows no proof that excessive blood transfusion plays a role in the development of VP-CRPS II (1).

In either needle or chemical injury to the blood vessel, the mechanism is quite similar. The chemical irritant, be it diazepam or IV colchicine (Case 15), causes the same injury and stimulation of the perivascular unmyelinated C-fibers as the rare incidence of the needle traumatizing the same nerve (20,32,43,88-90). In either case, the sudden nerve stimulation starts the process of neuroinflammation typical of neuropathic pain with microvascular involvement (16,17,43).

Etiologic factors: phlebitis. Phlebitis due to needle insertion is a part of the neuroinflammatory response. In contrast to the common form of phlebitis due to trauma, immobilization, or infection, the VP-CRPS II inflammation and phlebitis usually begins instantaneously at the time of venipuncture in absence of any other precipitating factor (3,7,24-30). The biopsy sample of the involved area reveals inflammatory pathologic changes in venules (3). However, due to the fact that neuroinflammation is not commonly recognized as a cause of phlebitis and ulcerative lesions, the condition is undiagnosed.

Finally, there has been only one case report of neuroma formation due to needle trauma, making it very unlikely to be any significant factor (34).

SUMMARY AND CONCLUSION

Venipuncture Complex Regional Pain Syndrome Type II (VP-CRPS II) is the manifestation of a minor injury causing a severe form of CRPS. It is due to the IV needle accidentally injuring the C-thermoreceptor sensory nerve branches in the wall of microcirculation (venules and arterioles). There is no known preventive measure. Lack of experience and severity of trauma have not been proven as risk factors. Early diagnosis and proper multidisciplinary treatment provide good pain relief.

Disclosure. The authors have no fiduciary interest in any patient, medical supplies, or any medications discussed in this paper.

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