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Aims & Scope of the Journal

Pan Arab Journal of Neurosurgery publishes peer reviewed original articles only and highlights the current advances in Neurosurgery and related sciences throughout the Arab world. It is also hoped to stimulate associated scientific research and communication between hospitals and universities in the Arab World. Original manuscripts from all over the world will be published, as well as summaries of any MD thesis successfully completed in any Arab University. An Academic Calendar is published to co-ordinate symposia throughout the region and abroad and inform readers of dates of international meetings.

Inside This Issue

"Differential diagnosis of complex regional pain syndrome," This review article by a world leader in pain management explicitly outlines the differential diagnosis of complex regional pain syndrome and other nerve entrapment syndromes that are commonly seen in clinical practice.

"Petroclival tumour surgery – A personal experience of 20 cases," A considerably large series of the surgical management of one of the most difficult lesions to surgically resect. The shortcomings faced by the neurosurgeons have been outlined.

"Subdural empyema, diagnostic difficulties and surgical treatment controversy," The experience of the authors about one of the most treacherous forms of intracranial sepsis often ignored and confused with milder forms of intracranial collections. Importance of conscientious search for the sources of infection have been mentioned.

"Preoperative prognostic value of magnetic resonance imaging in cervical spondylitic radiculomeylopathy," A series comprising of 10 cases of cervical spondylity radiculo-meylopathy highlighting the value of MRI findings in predicting the outcome of surgical intervention. This according to the author can help in selecting the appropriate candidates for surgical intervention.

"Bilateral dilated unreactive pupils in paediatric patients following severe head injury – Long term outcome," This paper reviews authors experience of aggressive management of children with severe head injuries and fixed dilated pupils. Factors adversely affecting survival have been highlighted.

"Intracranial hypertension revealing lumbar neurinoma," A case review of this unusual presentation. Pathogenesis and mechanism for intracranial hypertension resulting from a lumbar pathology have been mentioned.

"Cerebral infection by candida albicans," Case review highlighting the importance of suspecting an intracranial pathology even in the absence of any indolent systemic infection.

"Infantile spinal cord tumour presenting as hydrocephalus," This case review is an interesting presentation of hydrocephalus from an intraspinal tumour. Surgical management

Review Article

Differential diagnosis of complex regional pain syndrome, Type I (RSD)

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Abstract

Of 38 patients referred to Mensana Clinic with the diagnosis of complex regional pain syndrome, Type I (CRPS Type I, formerly called RSD), 27/38 (71%) of the patients were found not to have clinical and diagnostic studies to support this diagnosis. Before referral to Mensana Clinic, 16/38 patients never received a sympathetic block (42%), which is considered one of the essential diagnostic tests needed to confirm the presence of CRPS I. After diagnostic evaluation at Mensana Clinic, only 1/38 (3%) of the patients actually had CRPS I exclusively, while 10/38 (26%) had a mixture of both CRPS Type I and nerve entrapment syndromes, thoracic outlet syndrome, disrupted disc, and/or radiculopathies. The largest category of missed diagnoses was nerve entrapment syndromes, which were verified at Mensana Clinic in 37/38 (96%) of the patients, followed by thoracic outlet syndrome found in 16/38 (42%). A simple diagnostic framework is reported, to assist in the differential diagnosis of CRPS I and nerve entrapment syndromes.

Note: Throughout this article, for the sake of consistency, earlier references, that used the terms of reflex sympathetic dystrophy, or RSD will be referenced or quoted as CRPS, Type I, despite the original nomenclature. This same approach will be used for references using the term causalgia, which will be changed, for the sake of continuity, to CRPS, Type II. *(p1-9)*

Introduction

Complex regional pain syndrome, Type I (CRPS I) (formerly known as RSD, or reflex sympathetic dystrophy) and complex regional pain syndrome, Type II (CRPS II) (formerly known as causalgia) are symptom complexes that evoke a great deal of confusion. Very often, physicians do not recognise that these are separate and distinct entities, and commonly assume that they are disorders of the same aetiology, as well as responsive to the same treatment. Clinically, this has not proven accurate.

Both Payne and Long make the distinction between CRPS Type II and CRPS Type I.[15,19] CRPS Type II is secondary to partial injury to major mixed nerves, caused by low- or high-velocity missiles, and manifests as trophic changes in the distribution of the nerve associated with

extreme hypersensitivity. The pain is diffuse and burning and true CRPS Type II almost always responds to sympathectomy.[15] Long suggested performing three or more sympathetic blocks, sometimes every day for up to a week or longer, with the expectation that longer relief should follow each subsequent block. With positive responses to sympathetic blocks he would suggest a sympathectomy.[15]

On the other hand, according to Payne, one considers CRPS Type I as the result of minor trauma; inflammation following surgery, infection, or lacerations resulting in some degree of swelling in the affected limb; infarctions, degenerative joint disease; frostbite and burns.[19] CRPS Type I usually follows a minor injury and does not involve a major nerve root.[15] Frequently, the site of injury is the knee, ankle or wrist and the pain seems to get worse with cold but not with emotional upset, unlike CRPS Type II. Demineralisation of the bone occurs with fibrosis of tendons and sheaths and spasm of the muscle.[15] Dysesthesia suggests that there will be less success with sympathectomy.[15]

The epidemiology of CRPS I and CRPS II is vague. A number of figures have been cited, but not often substantiated. Epidemiological research has been hampered in the United States by the absence of a ICD -9 code for these disorders until the efforts of Audrey Thomas (RN) led to the issuance of codes 337.21, and 337.22 for CRPS I of the arms and legs (personal experience). Epidemiological data from Sweden, a country with a population of 8.6 million people indicated that there were 27 cases of CRPS II in 1990, 40 cases in 1991, 38 cases in 1992, and 29 cases in 1993, while there were 67 cases of CRPS I in 1990, 44 cases in 1991, 40 cases in 1992, and 80 cases in 1993.[25] These data suggest that the incidence (number of new cases a year) for CRPS II is 0.31 to 0.47 per 100,000 while the incidence for CRPS I is between 0.46 to 0.92 per 100,000. However, this is data from only hospitalised patients.

The relative percentage of CRPS II (as the result of high velocity bullets or missile injuries) to all peripheral nerve injuries was reported from Iran.[6] The authors found 54 cases of CRPS II in 1,564 cases of peripheral nerve injury (3.4%).[6] The data from hospitalised patients in Sweden, with pain in an extremity regardless of its origin compared to patients diagnosed with CRPS II ranged from 27 per 1,249 cases, or 2%, to 29 per 2,458, or 1%. For CRPS I the numbers ranged from 67 per 1249, or 5%, to 80 per 2,458 or 3%.[25]

Many of the patients referred to Mensana Clinic with the diagnosis of CRPS I did not have the appropriate diagnostic studies to confirm this diagnosis yet were assigned the diagnosis anyway. Others had none of the signs and symptoms of CRPS I, yet were given this diagnosis. Therefore, there seems to be a need to clarify the criteria for the diagnosis of CRPS I. In this article, we report the diagnosis of these same patients established at Mensana Clinic and the process for establishing the differential diagnosis in patients thought to have CRPS I.

Materials and method

The subjects in this study were patients referred to Mensana Clinic, by treating physicians, attorneys, insurance companies, other patients, and a non-profit organisation formed to provide support for patients with CRPS I. Of the 38 patients in this study, 3 were from New Jersey, 12 from Maryland, 2 from Nevada, 5 from Pennsylvania, 2 from Mississippi, 3 from West Virginia, 2 from New York, 2 from Texas, 2 from Delaware, and one each from Montana, Washington, Louisiana, and Connecticut. There were 7 males and 31 females. The average age was 40.6 and the average length of the pain problem was 3.3 years. All were referred to the clinic with the only diagnosis being CRPS I. No patients were referred with the diagnosis of CRPS II, nerve entrapment, thoracic outlet syndrome, radiculopathy nor disc herniation or degeneration. All have signed consent forms

permitting their medical cases to be reported in the medical literature, as long as anonymity was preserved. A summary of their treatment before and after evaluation at Mensana Clinic is shown in Table 1.

Results

After evaluation, at Mensana Clinic, the patients received a discharge diagnosis based on clinical observations and diagnostic studies. The various diagnoses established at Mensana Clinic, and the basis for the diagnosis are shown in Table 1. The rational for establishing diagnoses are shown in Table 2. Twenty-seven of the 38 patients (71%) of the patients referred with the diagnosis of CRPS Type I were not found to have clinical and diagnostic studies to support this diagnosis. Other diagnoses such as nerve entrapment syndromes, disrupted discs, thoracic outlet syndrome, and radiculopathy were established based on clinical observations, objective laboratory findings and testing. Only 1 (3%) of the 38 patients referred with the diagnosis of CRPS Type I actually had it exclusively, while 10 (26%) had a mixture of both CRPS Type I and nerve entrapment syndromes, thoracic outlet syndrome, disrupted disc, and/or radiculopathies. By far, the largest category of missed diagnoses was nerve entrapment syndromes, which were verified in 37 (96%) of the 38 patients. This was followed by thoracic outlet syndrome, which had been overlooked in 16 (42%) patients.

Even though all of the 38 patients referred to Mensana Clinic had been diagnosed with RSD by their treating doctors, only one had received a chemical sympathectomy, using phenol, and none had received a surgical sympathectomy. Of the patients treated at Mensana Clinic 6 of the 11 people diagnosed with CRPS Type I have gone on for surgical sympathectomies, with good results in 4 of the 6 people, and the fifth would have had a good result had the patient received decompression of a concomitantly trapped nerve. She was lost in the follow-up. Finally, of the 38 patients who received the diagnosis of CRPS I, before referral to Mensana Clinic, 16 of them never had received a sympathetic block (42%), which is considered one of the essential diagnostic tests needed to confirm the presence of CRPS I.[25] Prior to admission, none of the 38 patients (0%) diagnosed by other physicians as having CRPS I had received a phentolamine test. This test consists of an I.V. injection of an alpha 1 post-synaptic blocking agent, which provides a response analogous to a sympathetic block and can serve as another diagnostic test for CRPS I.[22]

Discussion

A number of authors have advanced the notion that there are other types of sensory mechanism, other than hyperalgesia evident in CRPS I and II.[4,12-14] Clinically, hyperalgesia is defined as a more intense response to a normally painful stimulus, which is seen in the early phases of nerve entrapments and radiculopathies. In counter-distinction allodynia, which is defined as a painful response to a normally non-painful stimulus, is seen in CRPS Type I and II.[4,12-14] The clinical features most often seen in CRPS I are shown in Table 3.

It is important to make a distinction between cold hyperalgesia, heat hyperalgesia and mechanical hyperalgesia.[18,21] Both cold and heat hyperalgesia are rarely seen in CRPS II.[18,21] Moreover, it is important to make a distinction between cold allodynia and mechanical allodynia. Cold (thermal) allodynia is most often seen in CRPS Type I and II, while mechanical allodynia is seen commonly in CRPS Type I and II nerve entrapment syndromes and radiculopathies.[8] This clinical distinction has led to the use of the 'Hendler Alcohol Drop and Swipe test' to make a distinction between a) CRPS Type I and II, which have cold allodynia manifesting as a painful response to an alcohol drop on an affected limb (allodynia), and b) CRPS Type I and II nerve entrapment syndromes, and radiculopathies which have mechanical allodynia

Table 1 - Treatment summary of patients referred to Mensana Clinic with the diagnosis of CRPS I, N= 38

SUMMARY OF PATIENTS REFERRED TO MENSANA CLINIC WITH CRPS I					
Numbers show number of patients: numbers in parenthesis show range of procedures per patient					
Prior to Mensana Clinic admission					
Referring diagnosis		**Diagnostic studies**		**Treatments**	
CRPS I	38	Sympathetic blocks	22 (1-350)	DCS	7 (1-6)
Nerve entrapment	0	Bone scans	7 (0-3)	Sympathectomy	1 (1)
Radiculopathy	0	Phentolamine I.V.	0	Physical therapy	33 (8-432)
		EMG/Nerve conduction	2 (1)	Acupuncture	6 (2-12)
		Peripheral nerve blocks	0		
After Mensana Clinic admission					
Discharge diagnosis		**Diagnostic studies performed at Mensana Clinic**		**Treatments**	
CRPS I	10	Sympathetic blocks	11 (1-5)	Sympathectomy	6 (1-5)
CRPS II	2	Bone Scans	38 (1-1)	Nerve decompression	34 (1-4)
Disrupted Disc	9	Phentolamine I.V.	7 (1-3)	Discectomy/Fusion	9 (1-6)
Nerve entrapment	37	EMG/Nerve conduction	38 (1-2)	Thoracic outlet	16 (1-2)
Radiculopathy	9	Neuromoter studies	38 (1-2)		
Thoracic outlet	16	Peripheral nerve blocks	35 (1-6)		
		Nerve root blocks	10 (3-10)		
		Provocative discograms	10 (3-10)		
		Doppler flow of arms	17 (1-2)		

After diagnosis and treatment at Mensana Clinic, many of the patients had multiple diagnoses confirmed. Therefore, the number of diagnoses from Mensana Clinic exceeds the total number of patients.

demonstrated by lightly stroking the affected limb with the used swab.[10] Concisely stated, mechanical allodynia is of less use diagnostically since it may be present in CRPS Type I and II nerve entrapment syndromes and radiculopathies while thermal allodynia is a more useful clinical feature, usually being limited mostly to CRPS Type I and occasionally to CRPS II.[18,21]

Table 2 - Diagnostic considerations

	Positive response to Phentolamine IV[22]	**No response to Phentolamine IV[22]**	**Partial response to Phentolamine IV[22]**
EMG/nerve conduction velocity/ somatosensory- evoked potential: All negative	CRPS Type I	Microvascular damage with swelling and mechanical hyperalgesia; Sensory nerve entrapment	Mixed injury
EMG/nerve conduction velocity/somatosensory-evoked potential: at least one positive	CRPS Type II	Neuroma or nerve entrapment at site of injury	Both CRPS I and nerve entrapment
Positive response to alcohol drop test	CRPS Type I	Too low a dose phentolamine	Too low a dose phentolamine
Positive response to alcohol swab swipe	CRPS Type I CRPS Type II	Nerve entrapment Radiculopathy	CRPS Type II Nerve entrapment Radiculopathy
Positive response to a local nerve block (radial, ulnar, median, peroneal, saphenous, tibial) with 100% relief of all symptoms	Nerve entrapment syndrome with sympathetic component	Nerve entrapment syndrome without any sympathetic component	Nerve entrapment syndrome with any sympathetic component
Positive response to sympathetic block (a warm limb and 100% relief of all symptoms)	CRPS Type I	Too low a dose of phentolamine or too slow infusion	Too low a dose of phentolamine or too slow infusion
Partial relief of pain with local nerve block	Both nerve entrapment and CRPS I	Poor nerve block	Both nerve entrapment and CRPS I

Additionally, a cool limb is not diagnostic of CRPS I and II, despite many reports in the literature to that effect.[5,20] Uematsu, Hendler, Hungerford, Ono and Long, reviewed 803 cases at Johns Hopkins Hospital, and found that, as expected, patients with CRPS I and II had cold limbs, most of the time, with ranges of 0.5°C to more than 3°C coldness being reported for over 79% of the cases diagnoses with CRPS I.[26] However, in 89% of the cases in which there were abnormal EMG or nerve conduction velocity studies, the affected limb was also cold, although not to the same severity as the patients with CRPS I. These figures included cases of CRPS II as well as patients with radiculopathies and nerve entrapment syndromes.[26]

Table 3 - Comparison between nerve entrapment syndromes and CRPS Type I

Nerve entrapment syndromes	CRPS, Type I
SYMPTOMS	
Pain in distribution of a peripheral nerve, ie. ulnar, tibial, peroneal, radial, etc.	Circumferential pain, around the entire limb or distal portion of limb
Worsened by use, pressure on a certain spot	Worsened by use, light touch, temperature change, touch anywhere on the affected limb
Onset with sprain, direct trauma broken bone, electrical injury	Onset with sprain, direct trauma or minor event like light tap, or after broken bone
Pain reduced by rest or comfortable position	Constant pain unaffected by change of position
No history of spread to opposite limb	May spread to opposite limb
No history of spread to ipsilateral limb	May spread to ipsilateral limb
SIGNS	
Positive tinel at common nerve entrapment spots, with pain radiating in distribution of a well known nerve pathway	Touch at any spot on the affected limb worsens the pain
No swelling	May have distal swelling, total limb pitting oedema, even a "ligature sign"
Negative Hendler alcohol drop test (no thermal allodynia)	Positive Hendler alcohol drop test (thermal allodynia)
Usually negative Hendler swipe test (no mechanical allodynia)	Usually positive Hendler swipe test (mechanical allodynia)
TESTS	
Negative bone scan	Positive bone scan
Positive SSEP	Negative SSEP
Positive response to peripheral nerve blocks	No response to peripheral nerve blocks
No response to sympathetic block	Positive response to sympathetic blocks
No response to I.V. phentolamine[22]	Positive response to I.V. phentolamine[22]

Anatomical distribution of the pain is another important feature to consider. Sympathetic fibres travel with the sensory nerves, so an injured sensory nerve may have a component of sympathetic damage reported, such as coldness, or hyperalgesia. However, the actual location of the pain is a critical factor. If the pain is in the distribution of a peripheral nerve, even if all the sensations for CRPS Type I are present, then the clinical syndrome is really a nerve entrapment with the sympathetic sensory components of it coming from the sympathetic fibres travelling with the sensory nerve. CRPS Type I has a circumferential pain distribution, ie. it is all around the limb in the pattern of the blood flow, not in a discrete nerve distribution nor radicular distribution.

A differential diagnosis between nerve entrapments and CRPS I is critical and based on the distribution of the pain, which follows nerve pathways for nerve entrapments and is circumfrencial for CRPS I. Failure to recognise the clinical features of nerve entrapment, thoracic outlet syndrome and radiculopathy has led to overlooking these disorders 40%- to 67% of the time.[7,9] The data in this article suggests that patients with these disorders constitute the majority of the patients mistakenly called CPRS Type I.

Sympathetic blocks have always been the mainstay of diagnosis and treatment for CRPS I. After a clinician determines a block is effective then the patient should have a series of six to ten blocks. After this series of blocks, several results are possible: a) the CRPS Type I or II may go away b) the CRPS Type I or II may temporarily go away for weeks or months only to return c) the CRPS Type I or II may temporarily go away for hours or days after the blocks only to return or d) the blocks did not produce a warm limb or had no effect on pain relief. If scenario (a) occurs the diagnostic blocks have also provided the cure. If scenario (b) or (c) occurs then the patient is a candidate for surgical sympathectomy since direct visualisation of the sympathetic chain and pathology reports on the tissue are more reassuring than blind ablation techniques.[1] Despite the obvious benefit of direct visualisation for sympathectomy there are still some physicians, mostly anaesthesiologists who continue to use blind chemo-ablative techniques, with neurolytic agents, such as phenol or radiofrequency lesions which even done under fluoroscopic control, do not provide the confirmation of a complete ganglionectomy that is accomplished by the direct visualisation of surgery.[25] If scenario (d) occurred, then a trial with I.V. phentolamine, if positive, may provide a more reliable sympathetic blockade.[22] Then a, b, or c can be determined using phentolamine instead of a sympathetic block. However, if there is a negative response to phentolamine in addition to a previous negative response to a sympathetic block (d), then the physician can feel very confident in dismissing CRPS I as a diagnostic consideration.

Other factors leading to the failure to accurately diagnose CRPS Type I are based on the confusion that exists in the literature about the diagnosis of CRPS Type I.[2,17,19,24] Lack of accurate history taking, the absence of a complete description of the symptoms in the history, failure to adequately appreciate that both the clinical and radiological picture of CRPS I changes over time, ordering inappropriate tests, or not ordering the appropriate tests, inability to properly perform the correct tests and inadequate knowledge permitting the appropriate interpretation of various test results.[11,16]

Finally, another reason for poor outcome is the failure to recognise that both CRPS Type I and nerve entrapment syndromes can coexist in the same patient. The failure to treat both, even though one or the other has been treated properly still leave an input of pain to the wide dynamic range neurons of the spinal cord, which lowers the threshold to painful stimuli resulting in continued hyperalgesia, or allodynia on a spinal cord basis.[3,23]

In order to combat these problems Mensana Clinic has created a patient administered automated history-taking instrument. The patient completes the 1,100 question clinical questionnaire in 12-45 minutes, depending on the various symptoms the patient experiences. The answers to the questions are computer analysed and then diagnoses as well a differential diagnoses are generated. Subsequently the computer generates a treatment algorithm specific for the various diagnoses reported by the computer interpretation. This diagnostic instrument has been retrospectively tested on 31 chronic pain cases and correlates with the clinical diagnosis given to patients by the author of this paper 93.5% of the time. Prospectively, in a series of 32 patients, the correlation was 97%. The diagnostic paradigm was designed to be overly inclusive so it does generate a great many false positive results, which were 44% in the retrospective study, and 39% in the prospective study. However, the inverse result is more positive: there were 6.5% false dismissal or failure to diagnose in the retrospective study and 3% in the prospective study.

When analysing the cases reported in this article, the most commonly missed diagnosis was a peripheral sensory nerve entrapment mistakenly diagnosed as CRPS Type I. Therefore, comparisons between the clinical symptoms signs and diagnostic test results for peripheral nerve entrapment and CRPS I are shown in Table 3.

Conclusion

In conclusion, by following the guideline for the diagnosis of CRPS Type I, as well as the guidelines for the diagnosis of nerve entrapment syndromes offered in this paper the ability to more accurately diagnose both of these disorders hopefully will be enhanced.

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