REPETITIVE STRAIN INJURY (RSI)
DIAGNOSIS AND TREATMENT

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Abstract. Repetitive strain injury (RSI) is a form of neuropathic pain which closely mimics carpal tunnel syndrome (CTS). The incidence and the recognition of RSI are on the rise. Due to lack of training regarding RSI, and lack of familiarity with the syndrome, the clinicians usually do not include the RSI in their list of differential diagnosis. Careful history taking, neurological evaluation, along with such tests as quantitative sensory testing (QST) and infrared thermal imaging (ITI) is helpful to differentiate RSI from other causes of complex regional pain syndrome (CRPS). Proper treatment with epidural and paravertebral nerve blocks, I.V. Mannitol for inflammation, and physical therapy spared these patients from unnecessary surgery.

Descriptors. carpal tunnel syndrome (CTS), complex regional pain syndrome (CRPS), repetitive strain injury (RSI)

INTRODUCTION

Repetitive strain injury (RSI) also known as "Cumulative Trauma Disorders"(CTD), has been recognized by the International Labor Organization as an occupational Cumulative Trauma Disorder resulting from working in mechanized occupations as far back as 1960 (1-4). It affects the factory workers processing, machining, and fabricating jobs, as well as construction workers. Recent literature shows the disease to occur more commonly among typists, musicians, computer operators, and word processors (Table I) (5-12). The risk of occupational RSI is greater for women in all jobs (4).

The incidence and the recognition of RSI are on the rise. The sharp increase in the number of personal computers (PC), and the rapidly increasing demand for meeting deadlines, has exposed the computer operators to RSI with acceleration of medical care cost on these patients (13-19).

REPETITIVE STRAIN INJURY (RSI)

Repetitive strain injury (RSI) is a form of neuropathic pain which closely mimics carpal tunnel syndrome (CTS) (20-22). RSI is seen among keyboard operators, such as transcribers, court reporters, and data entry processors. The major risk factors are meeting deadlines and rushing through the data entry. The symptoms consist of

frequent cramps in the flexor muscles of the hand, cold hyperhydrosis, neuropathic pain, and weakness of grip. These patients are invariably mistaken for CTS.

According to Allen et al, standard sympathetic nerve blocks are not helpful in these patients (23). Sharma et al, have applied five minute typing stress test in 21 RSI patients. All 21 had pain after 5 minutes of typing. The mean temperature reading after typing was significantly reduced to 2.11º C with 95% confidence interval 1.35 to 2.26°C ($P<0.001$)(24).

CARPAL TUNNEL SYNDROME (CTS)

Carpal tunnel syndrome (CTS) was first reported by James Paget in 1854(25). Marie and Foix first performed surgical release for CTS (26). Rothnagel in (1867-quoted by Marie and Foix), and later Sudeck reported "vasomotor neurosis" inflammatory form of median nerve entrapment (26, 27). This inflammatory form is frequently seen as a complication of complex regional pain syndrome (CRPS) (28). When properly applied, nerve conduction velocity (NCV) test easily identifies the CTS. Unfortunately, surgeons frequently skip the NCV test, and proceed with surgery - in spite of neuroinflammatory manifestation at the wrist and hand. Surgery on the inflammatory form of CTS invariably aggravates this condition.

CTS is the most commonly diagnosed neurological syndrome after migraine. The true CTS with characteristics of sensory loss limited to the median nerve distribution, and with delayed distal nerve conduction at the wrist, are essential minimal diagnostic requirements. Other diseases such as cervical spondylosis, cervical sprain, and diabetic neuropathy, RSI, and mononeuropathy multiplex, should not be mistaken for CTS. Otherwise, the patient will end up with surgery as an additional iatrogenic pathology.

The large majority of chronic pain patients suffer from somatic type nerve dysfunction, with no neurovascular involvement, such as seen in CTS. In a small minority of chronic pain patients, neuropathic pain with neurovascular pain, such as seen in CRPS patients, the disease mimics the CTS. The surgeon tends to explain away the neuropathic aspect of the pain even in the face of normal electromyography EMG/NCV tests, abnormal asymmetric hyperhydrosis, and purplish discoloration of the hand. The loser is the patient who develops more pain and neuroinflammation after surgery.

According to the occupational studies by US West Communications, 15% of all employees had tendon injuries and disorders vs. 1% suffering from CTS. This severe discrepancy between clinical diagnosis of CTS and the unbiased statistics by US West Communications, explains the poor results of CTS treatment in RSI patients (29).

MATERIALS AND METHODS

From January 1996 through August 2002, Twenty-one patients were identified as suffering from RSI (Table II). The patients were evaluated by: *(i)* careful detailed history taking; *(ii)* detailed neurological examination; *(iii)* dynamic and static evaluation of muscle strength and range of motion using a jamar dynamometer (30); *(iv)* nerve conduction velocity (NCV) not limited to only median nerve conduction, but to include ulnar nerve conduction test; *(v)* Phelan's and Tinel's tests at the wrist; *(vi)* quantitative sensory testing (QST); *(vii)* infrared thermal imaging (ITI)(31); *(viii)* pain threshold and strength measurement by Lovett and Martin scale of 0-5 and measurement of strength ½ hour before and ½ hour after reproduction of rapid performance of the job duty (32).

Table I. Sources of reported causes of Repetitive Stress Injury (RSI).

1. Editors 1a. Journalist 1b. Keyboard Operators 1c. Musicians 1d. Programers 1e. Telephone Operators 1f. Typesetters 1g. Word Processors 1h. Writers
2. Game "Keeper's Thumb" or "Skiers Thumb"
3. Compartment Syndrome 3a. Repeated cast application or surgical closure of fascial defects.
4. Sports activities 4a.Occupational RSI leading to tennis elbow or TOS.

Table II. Causes reported in our patients.

Job Description	Number of Cases
Computer Operator	7
Court Reporter	2
Dairy Farmer	1
Heavy Equipment Operator	1
Massage Therapist	1
Nurse	5
Pathologist (using a microscope)	1
Plumber	1
Stock Trader	1
Telephone Operator	1

SYMPTOMS AND SIGNS

The patients suffered from neuropathic pain, which were hyperpathic and allodynic (18 of 21 patients), burning pain (13 of 21 patients), stabbing or sharp pain (4 of 21 patients) which was present as one pain modality, or two or more modalities, on the same patient.

The pain was regional (21 of 21 patients) involving not only the extremity, but showing regional spread to the rest of the extremity and craniocervical regions (19 of 21 patients).

The spread of pain, edema, tremor, vasoconstriction, and flexor spasm was present in all surgical and cryotherapy patients (33).

DIAGNOSIS

Due to cursory training regarding RSI and lack of familiarity with the syndrome, the clinicians usually do not include the RSI in their list of differential diagnosis. As a result, RSI is commonly diagnosed as CTS, fibromyalgia (FM), tendinitis, thoracic outlet syndrome (TOS), or rotator cuff syndrome (7, 34). The other common syndromes mistaken for RSI are, Raynaud's Phenomenon, and CRPS (12, 20, 35).

REPETITIVE STRAIN INJURY (RSI) VS CARPAL TUNNEL SYNDROME (CTS): Careful history taking, neurological evaluation, QST and ITI are helpful to differentiate RSI from other causes of CRPS. In our study of 824 CRPS patients, we found a tendency for over - and under - diagnosis of CTS (35). In spite of the fact that NCV test was normal, the patients showed the typical four principles of CRPS, i.e., *(i)* Neuropathic and hyperpathic pain in a regional pattern rather than exclusively involving the median sensory nerve distribution; *(ii)* Neurovascular dysfunction; *(iii)* Neuroinflammation in the form of edema or skin rash; and *(iv)* Dysfunction of Limbic system. Unfortunately, surgery on the 93 patients mistaken for CTS resulted in marked aggravation of the disease. In the study of 824 CRPS patients, 93 underwent CTS surgery. Only 32 patients had undergone NCV studies and only 8 of the 32 had undergone comprehensive comparison of right and left ulnar and median NCV studies (35). Clinical correlation and comprehensive neurophysiological tests are the keys to the diagnosis of CRPS due to RSI.

REPETITIVE STRAIN INJURY (RSI) VS COMPLEX REGIONAL PAIN SYNDROME (CRPS): In the 21RSI patients, the clinical picture was very similar in both diseases. The consistent major difference was the fact that RSI patients, while staying away from work, showed no constant pain, no skin discoloration, no asymmetrical sweating, and no neuroinflammation. This was in contrast to CRPS patients who had practically constant neuropathic pain, neurovascular dysfunction, and tendency for flexor spasms (Table III). The RSI patients invariably showed recurrence of neuropathic pain when they tried to go back to the same job without having had proper treatments (such as ergonomic changes and nerve blocks).

REPETITIVE STRAIN INJURY (RSI) VS FIBROMYALGIA (FM): This differentiation was quite easily due to the fact that the RSI patients suffered from regional pain and sensory loss in the distribution of branches of brachial artery below the elbow in contrast to FM patients who had multiple areas of pain and tenderness with no circulatory changes. The FM patients did not suffer from hyperpathic and allodynic pain. In addition, the FM patients showed no neurovascular instability due to sympathetic nervous system (SNS) dysfunction.

REPETITIVE STRAIN INJURY (RSI) VS RAYNAUD'S PHENOMENON: Raynaud's Phenomenon causes skin discoloration usually due to female hormonal imbalance. There is no hyperpathia or allodynia, no weakness of grip, no flexor spasms, no mottling which is the sign of sympathetic neurovascular insatiability, and no asymmetrical sweating (35, 36).

CARPAL TUNNEL SURGERY

In our series of 21 RSI patients, 6 out of 21 had undergone CTS surgery with marked aggravation and spread of the disease to the contralateral extremity. Only 2 of the 6 patients had undergone NCV. Neither one showed any evidence of CTS.

Kleinert et al, noted that CRPS followed CTS surgery in 1/4 of their post-traumatic CRPS cases (37, 38). In our experience, usually the original trauma causing CRPS precedes the CTS surgery (35). Then the disease flares up secondary to surgical trauma resulting in a new source of neuropathic pain in the surgical scar, and flexion deformity of the hand and spread of CRPS (39-42) (Table III). The CTS and surgery share the blame, resulting in ultimate delay in diagnosis of the CRPS which originated the symptoms of CTS (12).

TREATMENT

Excluding the RSI patient who have undergone invasive treatment such as application of ice (Cryotherapy), surgery, or repetitive cast applications, the three forms that cause damage to myelinated nerves (ice), as well as damage to sensory nerves (surgery), and ischemia (cast application). The rest of RSI patients faired better, and were less prone to suffer from long standing disability. The neuropathic pain was different from the CRPS pain prior to surgical procedures, and in the cases that did not undergo surgery, the RSI patients did not demonstrate the full clinical picture seen in CRPS.

The difference was the fact that the patients would go into remission after stopping their repetitive strain work duties. The RSI would recur if the patient returned to work without correcting the extrinsic (i.e., improper key board, or other types of equipments), and intrinsic (i.e., improper position of the extremity, or perusing repetitive and rapid cumulative trauma) defective ergonomics.

In the non-invasive RSI patients, while in remission, there was no constant pain, no skin discoloration, no asymmetrical sweating, and no neuroinflammation. This was in contrast to CRPS patients who had practically constant neuropathic pain, neurovascular dysfunction, and tendency for flexor spasms (Table III).

Treatment with epidural and paravertebral nerve blocks, I.V. Mannitol for inflammation, and physical therapy spared these patients from surgery (35, 43).

Table III. Somatic RSI vs. Neuropathic RSI

The neuroinflammation due to CRPS may lead to entrapment neuropathies. Surgery may aggravate the CRPS. Somatic entrapment is usually helped with surgery.

	A-Somatic RSI 69 Patients	**B- Neuropathic RSI 21 of 824 Cases (35)**
Pain	1. Proportionate to impingement 2. Limited to sensory branches of median nerve	Allodynic and regional hyperpathic pain, and edema
Trophic changes	Thenar atrophy in late stages	Edema, dystrophy (hair and skin changes); neurovascular instability (Blotching)
Tinel's sign	Limited to carpal tunnel	Indiscriminative: Entire wrist Hyperpathic
Movement Disorder (Tremor)	None	Completely seen in late stages of the disease
Muscle tonus	Weak, atrophic in advanced stages	Dystonic flexor spasm and deformity of digits
NCV	Delayed sensory and motor distal latency	Normal or borderline sensory delay
Thermographic Changes	Usually no change. In advanced stages with over 6 millisecond distal delay, mild hyperthermia in the first three fingers distribution pointing to vasa neuronum damage.	Thermographic changes are in thermatomal (distal vascular) distribution involving the entire hand and wrist, but not limited to median nerve distribution.
Neurovascular instability : Mottling, color changes: (Chameleon sign)	None	Present off and on
Surgical findings	No inflammation	Diffuse inflammation; edema; osteonecrosis

RESULTS

Careful examination revealed the presence of trophic and inflammatory changes, manifested by hyperthermia on ITI. NCV studies showed normal or borderline test results. There was a major contrast between the somatic, classical CTS versus neuroinflammatory entrapment of the median, radial, and ulnar nerves in CRPS. The CTS patients showed normal thermographic distribution. Only in advanced, severe, and atrophic CTS patients with distal sensory latency on nerve conduction velocity of 6ms or longer the ITI showed mild hyperthermia in sensory median nerve distribution pointing to a dermatomal temperature change. This was in contrast to the CRPS group of patients who showed moderate hypo-or hyperthermia in the entire hand and wrist pointing to a thermatomal change in the distribution of brachial nerves and arterioles. Ammer et al have also shown similar thermal changes in late stage CTS patients with distal latency of 6 ms (44). The inflammatory nature of the CRPS mimicking CTS is best treated conservatively with physiotherapy, epsom salt bath, and alpha blockers such as terazosin, catapres patch, calcium channel blockers, and I.V. Mannitol, rather than surgical treatment (12).

SUMMARY AND CONCLUSION

We studied a series of 21 patients: Age range 26-43; Sex: 17 females and 4 males; Occupation: Computer operator 7; Court reporter: 2; Dairy farmer: 1; Heavy Equipment operator: 1; Massage Therapist: 1; Nurse: 5; Pathologist using a microscope: 1; Plumber: 1; Stock trader: 1; Telephone operator: 1. The NCV was normal in all but two patients. These two patients had borderline slowing of the median nerve distal latency. The ITI showed moderate bilateral hypothermia from the wrist down in 17 of the 19 patients with mean delta-T of forearm minus palm being 1.8± .91.

Treatment with epidural and paravertebral nerve blocks, I.V. Mannitol for inflammation, and physical therapy spared these patients from unnecessary surgery (35, 43).

References

1. Schwartz RG. Cumulative trauma disorders. *Orthopedics* 1992; 15: 1051-1053.

2. Schwartz RG, Weinstein SM. Getting a handle on cumulative trauma disorders. *Patient Care* 1996; 118-142.

3. Frederick LJ. Cumulative trauma disorders- an overview. *AAOHN J* 1992; 40:113-116.

4. Ashbury, FD. Occupational repetitive strain injuries and gender in Ontario, 1986 to 1991. *J. of Occupational and Environmental Medicine* 1995; 37: 479-485.

5. Fry HJM. Overuse syndrome of the upper limb in musicians. *Med J. Aust* 1986; 144: 182-185.

6. Pascarelli EF, Kella JJ. Soft-tissue injuries related to use of the computer. A clinical study of 53 severely injured persons. *J. of Occupational Medicine* 1993; 35: 522-532.

7. Sheon RP. Repetitive strain injury. 2. Diagnostic and treatment tips on six common problems. The Goff Group. *Postgrad Med* 1997; 102: 72-78, 81, 85.

8. Yassi A. Repetitive strain injuries. *Lancet* 1997; 349: 943-947.

9. Brogmus GE, Sorock GS, Webster BS. Recent trends in work-related cumulative trauma disorders of the upper extremities in the United States: An evaluation of possible reasons. *J. of Occupational and Environmental Medicine* 1996; 38: 401-411.

10. Chatterjee DS. Repetitive strain injury– a recent review. *J. Soc Occup Med* 1987; 37:100-105.

11. Verdon ME. Overuse syndrome of the hand and wrist. Primary Care; Clinics in Office Practice. 1996; 23: 305-319.

12. Hooshmand H. Chronic Pain: Reflex Sympathetic Dystrophy: Prevention and Management. CRC Press, Boca Raton FL. 1993.

13. Freudenheim M. Cost soar for on the job injuries. The New York Times. April 11, 1991; D5-6.

14. Cumulative trauma disorders in the workplace: costs, prevention and progress. Washington, DC: The Bureau of National Affairs Inc. 1991.

15. Kantrowsky B, Crandall R. Casualties of the keyboard. Newsweek. August 20, 1990; pg 57.

16. Kassler J. Job ailments in offices rising. The New York Times. February 24, 1991; 1, 4.

17. Vicklund B. Industrial relations in Sweden in the 1990's. International Industrial Relation Association (IIRA) Conference Report. December 29, 1988; 1-13.

18. Feder JB. As hand injuries mount, so do the law suits. The New York Times. June 8, 1992; D1, D10.

19. Miller MH, and Duncan TJ. Chronic upper limb pain syndrome (repetitive strain injury) in the Australian work force: a systematic cross sectional rheumatological study of 229 patients. *J. Rheumatol* 1988; 1705-1712.

20. Pascarelli EF, Quilter D. Repetitive strain injury. A computer user's guide. John Wiley & Sons New York. 1994; 1-200.

21. Bureau of Labor Statistics. Work injuries and illnesses by selected characteristics. 1992: Washington, D.C.:Bureau of Statistics, U.S. Department of labor publication. 1994; 94-213.

22. England JD. Entrapment neuropathies. *Current opinions in Neurology* 1999; 12: 597-602.

23. Allen TH, Peng MT, Chen KP, et al. Prediction of total adiposity from skinfold and the curvilinear relationship between external and internal adiposity. *Metabolism* 1956; 5: 346-352.

24. Sharma SD, Smith EM, Hazleman BL, Jenner JR. Thermographic changes in keyboard operators with chronic forearm pain. *BJM* 1997; 314: 118.

25. Paget J. Lectures on Surgical Pathology, Ed 3, Ed by Turner W, Lindsay and Blakistan. 1865.

26. Marie P, Foix CH. *Rev Neurol* 1913; 26: 647.

27. Sudeck P. Die sogen akute Knochenatrophie als ntzudndengsvorgang. *Der Chirurg* 1942; 15:449-458.

28. Bennett GJ. Neuropathic pain: In Melzack and Wall- Text book of pain, 3rd Edition. 1994; 201-224.

29. Hales T, Sauter S, Petersen M, et al. "Health hazard evaluation report, HETA 89-299-2230, US West Communications," National institute of Occupational Safety and Health, July 1992.

30. Schmidt R, Toews J. Grip strength as measured by the jamar dynamometer. *Arch Phys Med Rehab* 1970; 51; 231.

31. Hooshmand H, Hashmi M, Phillips EM. Infrared thermal imaging as a tool in pain management - An 11 year study, Part I of II. ThermologyInternational 2001; 11: 53-65.

32. Lovett R, and Martin EG. Certain aspects of infantile paralysis: with a description of a method of muscle testing. *JAMA*1916; 66: 729-733.

33. Maleki J, LeBel AA, Bennett GJ, Schwartzman RJ. Patterns of spread in complex regional pain syndrome, type I (reflex sympathetic dystrophy). *Pain* 2000; 88: 259-266.

34. Thorson EP, Szabo RM. Tendonitis of the wrist and elbow. *Occ Med: State Art Rev* 1989; 4: 419-431.

35. Hooshmand H, Hashmi H. Complex regional pain syndrome (CRPS, RSDS) diagnosis and therapy. A review of 824 patients. *Pain Digest* 1999; 9: 1-24.

36. Bej MD, Schwartzman RJ. Abnormalities of cutaneous blood flow regulation in patients with reflex sympathetic dystrophy as measured by laser Doppler fluxmetry. *Arch Neuro1* 1991; 48: 912-915.

37. Kleinert HE, Cole NM, Wayne L, Harvey R, Kutz JE, Atasoy E. Post -traumatic sympathetic dystrophy. *Orthop Clin North Am* 1973; 4: 917-927.

38. Wilson RL. Management of pain following peripheral nerve injuries. *Orthop Clin North Am* 1981; 12: 343- 58.

39. Jimenez DF, Gibbs SR, Clapper AT. Endoscopic treatment of carpal tunnel syndrome: A critical review. *J. Neurosurg* 1998; 88: 817-826.

40. Schwartzman RJ, McLellan TL. Reflex sympathetic dystrophy. A review. *Arch Neurol* 1987; 44: 555-561.

41. Veldman PH, Goris RJ. Multiple reflex sympathetic dystrophy which patients are at risk for developing a recurrence of reflex sympathetic dystrophy in the same or another limb. *Pain* 1996; 64: 463-466.

42. Kozin F, McCarty DJ, Sims J, Genant H. The reflex sympathetic dystrophy syndrome. I. Clinical and histologic studies: evidence of bilaterality, response to corticosteroids and articular involvement. *Am J Med* 1976; 60: 321- 331.

43. Hooshmand H, Dove J, Houff S , et al. Effects of diuretics and steroids in CSF pressure, a comparative study. *Arch Neurol* 1969; 21: 499-509.

44. Ammer K, Engelbert B, Melnizky, P, Schartelmüller T. The thermal image of patients suffering from carpal tunnel syndrome with a distal latency higher than 6.0 msec. *Thermology International* 1999; 9: 15-19.