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The efficiency of electromagnetic field treatment in Complex Regional Pain Syndrome Type I

ABUBEKIR DURMUS†, AYSEGUL CAKMAK†*, RIAN DISCI‡ and LUTFIYE MUSLUMANOGLU†

† Istanbul University, Istanbul Medical Faculty, Department of Physical Medicine and Rehabilitation, Istanbul, Turkey
‡ Istanbul University, Istanbul Medical Faculty, Department Public Health (Biostatistics and Demography Section), Istanbul, Turkey

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Abstract

Introduction: Complex Regional Pain Syndrome Type I is a pathological condition that occurs without evident nerve injury and follows a course characterized by severe pain.
Purpose: The aim of this study is to assess whether or not electromagnetic field treatment administered with calcitonin and exercise has positive effects on clinical improvement, scintigraphic assessment and bone markers compared to calcitonin and exercise administration.
Method: In this randomized double-blind, placebo-controlled study, 40 patients with Complex Regional Pain Syndrome Type I, that developed after a Colles fracture were included in the assessments and were administered calcitonin and exercise treatment for 6 weeks. In addition to this treatment, half the patients received electromagnetic field treatment, and the other half received placebo treatment. The patients were evaluated at the beginning and end of treatment with clinical parameters, scintigraphic assessment and biochemical markers.
Results: Although we found some significant improvements in our evaluation criteria, we could not find a significant statistical difference between groups.
Conclusions: The absence of a significant difference between the two groups in the assessment parameters has been interpreted as evidence that electromagnetic field treatment does not provide additional benefit to calcitonin and exercise treatment.

Introduction

The great majority of people are exposed to a range of physical or psychological traumas during their lives. An individual's reaction to trauma, however, may vary greatly. Whilst there may be a quick recovery without complication, depending on the severity of the trauma and the psychological and physical condition of the individual, the trauma may also result in a condition called Complex Regional Pain Syndrome (CRPS) Type I, which may lead to clinical manifestations eventually causing physical disability.[1]

Type I CRPS is a disease characterized by severe spontaneous pain that is not related to the distribution of a peripheral nerve and that is excessive in comparison with the initial complaint, oedema, changes in skin blood flow, abnormal sudomotor activity in the painful region, allodynia and hyperpathia. CRPS usually develops as a consequence following a harmful stimulus, yet infectious agents such as ureaplasma urealyticum have been implicated in the aetiology. Most often it is observed in the distal parts of the extremities, but it may spread to the proximal parts as well.[2–6]

The disease, first described by Ambroise Pare in the 16th century, was later called causalgia after the detection of patients with a severe burning complaint.[7] In 1900, Sudeck described spotted osteoporosis observed in the direct X-ray of a group of patients with stiffness, swelling and painful joints, and the term 'Sudeck atrophy' became commonly used.[8–10] In later years, the term 'reflex sympathetic dystrophy (RSD)' was suggested, with the concept that there was a sympathetic system involvement in the pathogenesis. In 1986, the International Association for the Study of Pain (IASP) described the pathology as pain developing as a result of sympathetic hyperactivity in some part of the extremity without major nerve injury. Causalgia, however, was deemed to result from major nerve injury, and the two pathologies were distinguished from each other.[8, 11] In 1994, RSD and causalgia were combined under the

* Author for correspondence; Istanbul University, Istanbul Medical Faculty, Department of Physical Medicine and Rehabilitation, 34390 Sehremini-Istanbul, Turkey. e-mail: cakmakaysegul@hotmail.com

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name of CRPS by the International Association for the Study of Pain taxonomy committee, CRPS Type I was defined as the disease without major nerve injury that was previously called RSD, and Type II was defined as pain with nerve injury (causalgia).[12]

Type I CRPS, although observed in all age groups, is encountered more frequently in patients between the ages of 40 and 60, and it is also found more frequently in women. In 10–25% of the patients, no aetiological reason can be identified.[13] It is reported in various studies to be observed at ratios of 5–20% in patients with coronary artery disease, 12–21% in patients with hemiplegia, 0.2–35% after a Colles fracture, and 1–2% after general fractures.[13–16] There are also studies indicating that it can be observed after pacemaker implantation and after herpes zoster infections.[17, 18] Although generally observed in a single extremity, it is reported that there can be bilateral involvement at a ratio of 23%.[13, 19]

Making the diagnosis and commencing the treatment at an early stage is important in Type I CRPS for the prevention of muscle atrophies, formation of contractures and the progression of functional loss. Yet, considering that the definition and aetiology are controversial in this syndrome, the absence of conclusive treatment criteria is not that surprising.[20] Various medical and physical treatment methods such as sympathetic blockade, steroids and pamidronate are used in this disease, and calcitonin is a drug used for its bone mineralization prevention, analgesic and probable anti-inflammatory effects.[21–24] While there are some studies that recommend the use of calcitonin because of its efficacy, there are also others that recommend limited use due to its side effects.[25, 26] The use of some analgesic substances such as guanethidine, however, are reported to prolong the recovery period of vasomotor instability and have been proven not to have greater analgesic effects than placebo.[27] In a study by Kemler *et al*. it was shown that physical treatment methods do not contribute to recovery and that patients with better assessment criteria at the beginning of treatment respond better to treatment.[28]

Electromagnetic field treatment is a method that has been demonstrated to increase osteoblastic activity and to inhibit osteoclastic activity in experimental studies.[29] It is recognized that it also has anti-inflammatory effects and that it stimulates tissue-repair agents.[30] Sert *et al*. stated, 'the use of a low-frequency electromagnetic field (EMF) in the treatment of osteoporosis has been investigated as a non-invasive alternative method. Experimental and clinical studies have shown that EMF treatment prevents the bone loss in osteoporosis. EMF treatment has some biological effects on the behaviour of the cell populations of bone. For example, it increases the maturation of the bone trabecula, bone volume, and bone formation'.[31] Pilla stated, 'EMF (short-wave radio frequency) signals are often employed for the reduction of pain and oedema in strain, pain, and contusion injuries, as well as for wound repair'.[32]

PURPOSE

We expect positive improvements of EMF treatment in CRPS, based on the results of EMF in experimental studies of bone formation, reduction of pain and oedema, as well as for wound healing. (Which are the main characteristics of CRPS.)

The aim of this study is to assess whether or not electromagnetic field treatment administered with calcitonin and exercise has positive effects on clinical improvement, scintigraphic assessment and bone markers compared to calcitonin and exercise administration.

Materials and methods

Forty patients diagnosed as having Type I CRPS subsequent to trauma (Colles Fracture), who consulted the Physical Medicine and Rehabilitation Department of Istanbul University, Istanbul Medical Faculty between 1999 and 2001 were included in the study. The diagnosis was made clinically according to the criteria determined by 'Committee on taxonomy of chronic pain conditions of the International Association for the study of pain'.[26, 33] The patients' being in the 18–55 age group, the development of pathology after a trauma, the presence of phase I CRPS diagnosis in three phase bone scintigraphy, the absence of any known hypersensitivities to calcitonin and the patients' signing the informed consent form were determined as the criteria for inclusion in the study. Having been previously treated for this diagnosis, using pacemakers, the presence of an infectious or malignant disease, being in a pregnancy or menopausal state constituted exclusion from the study. There were no refusals or drop-outs from the study. The patients were divided into two groups with the random numbers table. Both groups of patients were administered 100 units of calcitonin ampule for 6 weeks. The patients received this treatment through the intramuscular route daily 1 × 1 for the first 3 weeks and once every other day during the second 3 weeks. Both groups of patients performed the active and active assistive Range of Motion (ROM) exercises (flexion, extension, inversion and eversion direction) and the stretching programme (to wrist and fingers) three times a day (for a period of 30 min per session).

While the first group of patients received the pulsed electromagnetic field treatment of 100 Gauss intensity and 50 Hz frequency five times a week for 6 weeks (30 sessions), the second group of patients received placebo treatment by being placed in the same device without it being switched on (for a period of 60 min per session). The Magnetic-Therapy Mg Port Cosmogamma® (coil) device was used for this treatment.

The patients were assessed at the beginning of a 6 week course of treatment and on the final week of treatment by a physician who did not know which group received the applied magnetic field treatment. A 6 week period was chosen in reference to former studies of EMF. The method of assessment was conducted by clinical questioning, examination, bone scintigraphy and biochemical markers indicating bone formation and resorption.

Pain at activity and rest was assessed using a visual analogue scale (VAS) graded between 0 and 10;[34] with verbal pain scale graded over 4 points,[35] and with pain on palpation graded over 5 points (0—no pain, 4—hyperesthesia).[34] In addition, stiffness[15] and change in colour[36] were also rated. The change in oedema in the extremity was monitored by recording the amount of water (ml) overflowing after immersion of the extremity in a container filled to the brim with water. The change in the range of movement of the joint was determined by the evaluation of the standard movement range (ROM) with a goniometer.[34] Laboratory assessment was carried out using three-phase bone scintigraphy and biochemical markers.[33] Three-phase bone scintigraphy was carried out at the Department of Nuclear Medicine. Abnormal blood flow, increase at vascularization and increase of activity at late phase were accepted as phase I CRPS. Numeric assessment (bone-to-soft tissue ratios were calculated from static (delayed) images and scintigraphic evaluations were done both visually and quantitatively) was carried out during scintigraphy and performed before and after the treatment. Since one of the aims of our study was to assess whether the localized osteoporosis observed in CRPS was treated or not, the biochemical markers of bone formation (bone alkaline phosphatase, osteocalcin, procollagen 1) and bone resorption (pyridinoline, deoxypyridinoline and hydroxyproline) were evaluated before treatment and 2 weeks after treatment in the same laboratory.

SPSS 7.5 was used for statistical analyses. The tests applied were Cross-tab Correlation, Independent Student *t*-test, Wilcoxon Matched Paired Signed Test and Mann–Whitney *U*-test.

Results

No significant difference with respect to demographic data was observed between the two groups at the beginning of treatment as shown in table 1.

All patients had a Colles fracture. In the treatment group 60% sustained a right wrist fracture, 40% sustained a left wrist fracture (95% dominant). In the control group 55% sustained a right wrist fracture, and 45% sustained a left wrist fracture (90% dominant). The dominant hands and traumatized hands of the patients, time elapsed until the emergence of symptoms after the trauma, time elapsed after the trauma and the duration of placement in plaster casts were equal between the two groups as shown in table 1. Making an early diagnosis and starting the treatment in an early phase after the occurrence of Type I CRPS is one of the most important principles for the regression of the disease. All the patients we included in our study were in phase I.

The patients in Group 1 (EFT = electromagnetic field treatment) had reported 113.70 ± 93.81 days after the trauma. This time period was 102.30 ± 58.49 days in patients in Group 2 (Placebo = placebo electromagnetic field treatment). The patients' complaints, such as pain, swelling and a change in colour, began on average 48.80 ± 28.63 days before the patients in Group 1 attended our out-patient clinic; in the second group, the complaints began 54.55 ± 36.24 days before they applied to our out-patient clinic. There was no significant difference between the groups with respect to the two parameters ($p > 0.05$).

As shown in tables 2 and 3, in the pain assessment with VAS, the pre-treatment pain at rest was 3.60 ± 2.50 in Group 1 and 2.65 ± 1.84 in Group 2. Though there was no statistically significant difference between the groups ($p > 0.05$), the values in Group 1 were slightly higher. At the end of treatment the pain at rest value dropped to 0.80 ± 1.15 in Group 1 and to 1.00 ± 1.38 in Group 2. The pain at rest decreased considerably in both groups ($p < 0.01$). Considering the pre-treatment values, although the decrease in Group 1 was greater, no statistically significant difference was detected between the two groups ($p > 0.05$).

Before treatment, the pain score at activity was found to be 7.15 ± 1.95 in Group 1 and 5.60 ± 2.41 in Group 2. There was no statistically significant difference between the groups ($p > 0.05$). After treatment, pain at activity dropped to 2.90 ± 1.80 in Group 1 and to 2.60 ± 2.04 in Group 2. Although a clear decrease was observed in both groups ($p < 0.001$), the reduction in pain in Group 1 was interpreted as more significant

Table 1 Demographics and details of the patients before treatment

	EFT	*Placebo*	t	χ^2	p
Age[a]			− 0.797		0.430
Mean ± SD	37.65 ± 12.33	40.60 ± 11.05			
(min-max)	(18 – 55)	(18 – 55)			
Gender[b]				0.100	0.752
Male	50.00%	55.00%			
Female	50.00%	45.00%			
Education level[b]				1.310	0.520
Primary	55.00%	65.00%			
Secondary	30.00%	15.00%			
High	15.00%	20.00%			
BMI[a]			− 0.066		0.948
	0.251 ± 0.038	0.251 ± 0.029			
Duration after trauma[a]			0.461		0.647
Days	113.70 ± 93.81	102.30 ± 58.49			
Duration of pain before treatment[a]			− 0.557		0.581
Days	48.80 ± 28.63	54.55 ± 36.24			
Duration of plaster cast[a]			0.813		0.422
Days	45.72 ± 11.89	41.56 ± 18.21			

[a]Independent student *t*-test.
[b]Cross-tab correlation.

Table 2 Comparison of evaluation criterias before and after treatment between groups

	EFT	*Placebo*	z	p
VAS-rest				
Before treatment	3.60 ± 2.50	2.65 ± 1.84	− 1.167	0.243
Post treatment	0.80 ± 1.15	1.00 ± 1. 38	− 0.212	0.832
VAS-activity				
Before treatment	7.15 ± 1.95	5.60 ± 2.41	− 1.888	0.059
Post treatment	2.90 ± 1.80	2.60 ± 2.04	− 0.345	0.730
Measure of swelling				
Before treatment	87.40 ± 69.50	117.55 ± 92.26	− 0.771	0.441
Post treatment	13.65 ± 29.27	37.35 ± 45.52	− 1.314	0.189
Bone alkaline phosphatase				
Before treatment	25.65 ± 1.72	25.18 ± 2.29	− 0.015	0.988
Post treatment	25.25 ± 1.01	25.41 ± 1.09	− 0.382	0.702
Osteocalcin				
Before treatment	8.99 ± 1.22	9.39 ± 1.63	− 0.549	0.583
Post treatment	8.93 ± 1.32	8.16 ± 1.01	− 2.111	*0.035*
Procollagen				
Before treatment	7.25 ± 1.75	7.26 ± 1.67	− 0.096	0.924
Post treatment	6.98 ± 1.46	6.17 ± 1.67	− 2.179	*0.029*
Pyridynoline				
Before treatment	781.25 ± 46.25	765.83 ± 49.47	0.836	0.403
Post treatment	746.84 ± 40.52	743.75 ± 53.99	− 0.067	0.947
Deoxypyridynoline				
Before treatment	746.50 ± 91.67	730.00 ± 71.62	− 0.424	0.671
Post treatment	683.15 ± 86.03	676.87 ± 73.93	− 0.464	0.642
Hydroxyproline				
Before treatment	22.22 ± 1.06	21.02 ± 2.20	− 1.104	0.269
Post treatment	20.71 ± 2.50	20.68 ± 1.23	0.000	1.000
Scintigraphic evaluation				
Before treatment	2.93 ± 2.67	1.83 ± 0.55	− 1.469	0.142
Post treatment	2.18 ± 0.40	1.57 ± 0.39	− 3.210	*0.001*

z = Mann – Whitney *U*-test.

Table 3 Comparison of evaluation criterias before and after treatment in groups

	Before	*Post*	z	p
VAS-rest				
EFT	3.60 ± 2.50	0.80 ± 1.15	− 3.526	*0.000*
Placebo	2.65 ± 1.84	1.00 ± 1.37	− 3.169	*0.002*
VAS-activity				
EFT	7.15 ± 1.95	2.90 ± 1.80	− 3.838	*0.000*
Placebo	5.60 ± 2.41	2.60 ± 2.04	− 3.786	*0.000*
Measure of swelling				
EFT	87.40 ± 69.50	13.65 ± 29.27	− 3.920	*0.000*
Placebo	117.55 ± 92.25	37.35 ± 45.52	− 3.921	*0.000*
Bone alkaline phosphatase				
EFT	25.65 ± 1.72	25.25 ± 1.01	− 1.552	0.121
Placebo	25.18 ± 2.29	25.41 ± 1.09	− 0.028	0.977
Osteocalcin				
EFT	8.99 ± 1.22	8.93 ± 1.32	− 0.705	0.481
Placebo	9.39 ± 1.63	8.16 ± 1.01	− 1.979	*0.048*
Procollagen				
EFT	7.25 ± 1.75	6.98 ± 1.46	− 0.825	0.409
Placebo	7.26 ± 1.67	6.17 ± 1.67	− 1.508	0.132
Pyridynoline				
EFT	781.25 ± 46.25	746.84 ± 40.52	− 2.660	*0.008*
Placebo	765.83 ± 49.47	743.75 ± 53.99	− 1.819	0.069
Deoxypyridinoline				
EFT	746.50 ± 91.67	683.15 ± 86.03	− 2.255	*0.024*
Placebo	730.00 ± 71.62	676.87 ± 73.93	− 2.483	*0.013*
Hydroxyproline				
EFT	22.22 ± 1.06	20.71 ± 2.50	− 1.693	0.090
Placebo	21.02 ± 2.20	20.68 ± 1.23	− 0.730	0.465
Scintigraphic evaluation				
EFT	2.93 ± 2.67	2.18 ± 0.40	− 0.384	0.701
Placebo	1.83 ± 0.55	1.57 ±0.39	− 1.067	0.286

z = Wilcoxon matched paired signed test.

since the pain at activity was greater in Group 1 in the pre-treatment period. When both groups were compared with each other no significant difference was observed after treatment ($p > 0.05$).

In the pre-treatment swelling assessment, the difference between the two extremities in patients in Group 1 was found to be 87.40 ± 69.50 ml on average. In Group 2, the difference between the two extremities was 117.55 ± 92.26 ml. After the treatment, these values dropped to 13.65 ± 29.27 ml in Group 1 and to 37.35 ± 45.52 ml in Group 2. The swelling decreased considerably in both groups ($p < 0.001$). When both groups were compared with each other no significant difference was observed post-treatment ($p > 0.05$).

At the beginning, the groups were equivalent with respect to clinical, scintigraphical and laboratory values as well ($p > 0.05$). The rate of radionuclide substance uptake in the affected extremity was 2.93 ± 2.67 in patients in Group 1 and 1.83 ± 0.55 in patients in Group 2. There was no statistically significant difference between the groups ($p > 0.05$). In post-treatment assessment, the rate of uptake dropped to 2.18 ± 0.40 in Group 1 and to 1.57 ± 0.39 in Group 2. The reduction in both groups was not statistically significant ($p > 0.05$). There was a statistically significant difference between the groups at post-treatment evaluation ($p < 0.001$).

Before treatment, in Group 1, the bone alkaline phosphatase was found to be 25.65 ± 1.72, osteocalcin 8.99 ± 1.22, procollagen I 7.25 ± 1.75, urinary pyridinoline 781.25 ± 46.25, deoxypyridinoline 746.50 ± 91.67, and hydroxyproline 22.22 ± 1.06. In Group 2, bone alkaline phosphatase was found to be 25.18 ± 2.29, osteocalcin 9.39 ± 1.63, procollagen I 7.26 ± 1.67, urinary pyridinoline 765.83 ± 49.47, deoxypyridinoline 730.00 ± 71.62, and hydroxyproline 21.02 ± 2.20. There was no statistically significant difference between the groups ($p > 0.05$).

After treatment, there was no significant change in bone alkaline phosphatase, osteocalcin, procollagen I and urinary hydroxyproline levels in Group 1 ($p > 0.05$). A statistically significant decrease was observed in urinary pyridinoline and deoxypyridinoline levels ($p < 0.05$). In Group 2, the changes in bone alkaline phosphatase, procollagen I, pyridinoline and hydro-

Table 4 Comparison of differences (post–pre) of evaluation criteria between groups

	EFT	*Placebo*	z	p
VAS				
VAS-rest	2.80 ± 2.17	1.65 ± 1.63	− 1.580	0.114
VAS-activity	4.25 ± 2.10	3.00 ± 2.20	− 2.132	*0.033*
Swelling				
Measure of swelling	73.75 ± 53.96	80.20 ± 64.51	− 0.162	0.871
Lab. evaluation				
Bone alkaline phosphatase	0.54 ± 1.60	− 0.19 ± 1.91	− 1.094	0.274
Osteocalcin	0.17 ± 1.52	1.22 ± 2.15	− 1.632	0.103
Procollagen	0.38 ± 1.98	0.88 ± 2.55	− 0.875	0.382
Pyridynoline	34.21 ± 49.70	24.69 ± 49.58	− 0.298	0.765
Deoxypyridynoline	56.84 ± 100.49	53.75 ± 80.49	− 0.083	0.934
Hydroxyproline	1.53 ± 1.88	0.62 ± 1.43	− 0.954	0.340
Scintigraphic evaluation	0.75 ± 2.70	0.25 ± 0.59	− 0.027	0.978

z = Mann–Whitney *U*-test.

xyproline levels were not statistically significant ($p > 0.05$). However a statistically significant reduction in osteocalcin and deoxypyridinoline levels was found ($p < 0.05$). When the post-treatment results of both groups were compared, there was only a significant difference between them ($p < 0.05$) in osteocalcin and procollagen I.

Because there were conflicting results between groups, we tried to resolve the situation by comparing the differences (post–pre-treatment results). We could not find any statistically significant results as seen in table 4.

Discussion

In their studies, Forouzanfar *et al.*[37] reported that there was no difference between pain assessment carried out different intervals throughout the day and pain assessment done only once in patients followed up with the diagnosis of CRPS Type I with respect to validity and reliability. In our study, the patients too were assessed once only.

In another study Atkins *et al.* assessed 60 patients with a Colles fracture 9 weeks after trauma and found that Type I CRPS had developed in 25% of the patients. It was stated that the duration of placement in plaster cast in patients in whom Type I CRPS developed was 5.6 weeks.[14]

Similarly, Bickerstaff *et al.* reported that Type I CRPS developed in 77 of 274 patients with a Colles fracture and that the duration of placement in plaster cast was 38.9 ± 0.9 days in these patients.[15]

In our study, the average duration of placement in plaster cast was 45.72 ± 11.89 days in patients in Group 1 and 41.56 ± 18.21 days in patients in Group 2; this period is comparable with the relevant literature. The difference between the two groups was not statistically significant ($p > 0.05$).

There are varying studies exploring a variety of medical and physical treatment methods in CRPS treatment and comparing their effectiveness with each other.

In a study by Gobelet *et al.* in which they examined the effectiveness of calcitonin, they found that in the group in which they administered the calcitonin and physical treatment modalities together, the duration of the disease was 50.4 days, and in the group in which they administered only physical treatment, they found the disease duration to be 51.2 days.[35] In another study by Gobelet *et al.* 100 IU of salmon calcitonin per day was administered along with active and passive physical treatment in a painless range of movement, passive pressure treatment, lymphatic drainage or whirlpool bath and TENS. The pain severities at rest and activity of 33 patients were evaluated with palpation, and a highly significant reduction in pain severity at rest was detected during the assessments conducted at 3 weeks and 8 weeks. However, for pain severity at activity, only a moderately significant reduction was reported.[38]

In a placebo-controlled study by Fialka *et al.* 14 patients had received classical acupuncture treatment 5 days a week for 3 weeks, and when pain was assessed with VAS (0–100), pain severity was observed to have decreased from an average of 57.8 ± 1.8 to 17.9 ± 2.4. In the other group, which received placebo acupuncture, pain severity had dropped from 55.4 ± 1.9 to 28.6 ± 1.9. They reported classical acupuncture to be an effective method in RSD treatment.[39]

In a study by Rabah *et al.* assessing pain severity by VAS, they applied stellate ganglion blockade and standard physical treatment separately to stage I, II and

III patients (21 patients in each group). At the end of treatment they reported excellent results in 76% and good results in 23% of the patients at stage I.[40]

Jayalakshmi *et al*. studied 38 patients with Type I CRPS, applied stellate ganglion blockade and active physiotherapy to 20 patients and reported a statistically significant reduction in VAS.[41]

In a study carried out by Davidof *et al*. to develop a simple and systematic method for assessing patients with RSD, swelling with distal extremity volume was evaluated.[35] Stenberg *et al*. used the water overflow method in order to objectively assess hand swelling in patients with rheumatoid arthritis.[42]

Buttler *et al*. conducted a study of 23 patients in whom Type I CRPS developed subsequent to calcaneus fracture and found abnormal swelling in 15% of the patients.[43]

In patients developing Type I CRPS after tibia fracture oedema assessment was done by measuring the bimalleolar circumference in the affected and unaffected extremities and when compared with the control group, a highly significant oedema was found in the patient group with Type I CRPS ($p < 0.001$).[44]

Bickerstaff *et al*. carried out a study to explore the efficacy of 400 IU nasal calcitonin in CRPS; they evaluated the swelling by the amount of overflowing water and reported that swelling decreased considerably at the end of treatment.[45]

We evaluated the swelling with the same method, and although there was improvement in both groups, there was no difference between groups.

Early mobilization is the essence of Type I CRPS treatment.[13, 46] It has been reported that active assistive joint movement range (ROM) exercises and strengthening exercises are beneficial while constraining ROM exercises may aggravate symptoms and signs.[46]

Jayalakshmi *et al*. applied stellate ganglion blockade and active physiotherapy in 20 patients with Type I CRPS, and reported that they observed a considerably significant increase in joint movement range at the end of treatment ($p < 0.01$).[41]

In our study there was evident limitation in joint movement range in both group patients before treatment. There was no significant difference between the two groups ($p > 0.05$). However, in patients in Group 1, the restriction in wrist extension was more prominent. In Group 1 there was a moderately significant increase in thumb abduction ($p < 0.01$); in all other joints' ROMs, there was a highly significant increase ($p < 0.001$). In Group 2, however, there was a prominent increase in wrist extension and flexion and in thumb abduction and flexion ($p < 0.001$).

In our study, we have tried to discover the efficiency of electromagnetic-field treatment, but we were unable to achieve any significant statistical results.

In Type I CRPS, three-phase bone scintigraphy yields an earlier diagnosis than radiography.[47] In the late phase (bone phase), an increase in radionuclide substance uptake in the periarticular region is characteristic.[7, 22, 28] The views expressed in the literature on the sensitivity and specificity of scintigraphy are contradictory. In the studies conducted, the sensitivity ranges between 44% and 100% and the specificity ranges between 80% and 98%.[48]

Zyluk *et al*. conducted pre-treatment and post-treatment assessments of 12 patients they treated with calcitonin for 30 days with scintigraphy; in the beginning they found the rate of involvement in the metacarpophalangeal region to be 1.99 on average (the ratio of the affected extremity to the unaffected extremity). At post-treatment, this value dropped to 1.28. However, in this study, the post-treatment scintigraphies were done after 6–18 months, and no information was provided about the duration of the disease.[49]

We have also found a decrease in scintigraphic results post-treatment in both groups, but there was no significance, most probably because of the short duration of evaluation.

One of the most frequently encountered signs in Type I CRPS is osteoporosis. In radiographies, widespread and spotted osteoporosis is observed in small bones and in the periarticular region.[4, 7, 22] It is reported that treatment of the disease can prevent the progression of osteoporosis but may not be able to reverse it completely.[13] It is reported that serum alkaline phosphatase and osteocalcin levels may increase as well as the urinary hydroxyproline excretion in the acute stage.[16, 47]

In a study by Bickerstaff *et al*. they assessed the efficacy of 400 IU nasal calcitonin along with its effect on serum calcium and urinary hydroxyproline levels. At post-treatment they reported a significant reduction in serum calcium in the group treated with calcitonin while they could not detect any significant change in urinary hydroxyproline levels.[45]

Mudun *et al*. compared the efficiency of 100 IU nasal calcitonin with indomethacin and observed that there was no significant change in serum calcium, phosphate, alkaline phosphatase and urinary calcium values post-treatment.[23]

Gobelet *et al*. explored the efficiency of calcitonin; they assessed the calcium, phosphorus and alkaline phosphatase levels in the blood and calcium, phosphorus and hydroxyproline levels in the urine. They reported that, of the 22 patients, five had an increase

in the hydroxyproline level before treatment, and 10 patients had an increased hydroxyproline level after treatment. No significant change was detected in other biochemical parameters between pre-treatment and post-treatment values.[35]

In our study, we made pre-treatment and post-treatment assessments of bone alkaline phosphatase, osteocalcin and procollagen I, urinary pyridinoline, deoxypyridinoline and hydroxyproline levels. There was no significant difference between the two groups before treatment. At post-treatment there was no significant change in bone alkaline phosphatase, osteocalcin, procollagen I and urinary hydroxyproline levels in Group 1 ($p > 0.05$). A statistically significant reduction was observed in urinary pyridinoline and deoxypyridinoline levels ($p < 0.05$). However in Group 2, there was a statistically significant reduction in osteocalcin and deoxypyridinoline levels ($p < 0.05$). These results can be explained by calcitonin usage.

Conclusions

The absence of a significant difference between the two groups in the assessment parameters has been interpreted as electromagnetic field treatments not providing additional benefit to calcitonin and exercise treatment.

This result was interpreted as suggesting that longer follow-up studies with possibly larger patient groups and different dosage and type (coil, mattress) are needed to establish the effect of electromagnetic field treatment.

References

1 Loonvander L, Veldman PHJM, Goris RJA. Severe complications of reflex sympathetic dystrophy: Infection, ulcers, chronic edema, dystonia and myoclonus. *Archives of Physical Medicine and Rehabilitation* 1998; **79**: 424–429.

2 Palazzi C, D'Amico E, Izzo F, Pace-Palitti V, Petricca A. Ureaplasma urealyticum as a possible cause of reflex sympathetic dystrophy syndrome. *Scandinavian Journal of Rheumatology* 2002; **31**(2): 97–99.

3 Schwartzman RJ, Popescu A. Reflex sympathetic dystrophy. *Current Rheumatology Reports* 2002; **4**(2):165–169.

4 Geertzen JHB. A study in reflex sympathetic dystrophy patients introduction. *Acta Orthopedica Scandinavica* 1998; **69**: 1–3.

5 Rowbothom MC. Complex regional pain syndrome type I (Reflex sympathetic dystrophy). *Neurology* 1998; **51**: 4–5.

6 Wasner G., Backonja MM., Baron R. Traumatic neuralgias: Complex regional pain syndrome (Reflex sympathetic dystrophy and causalgia): Clinical characteristics, pathophysiologic mechanisms and therapy. *Neurologic Clinics* 1998; **16**: 851–868.

7 Lankford LL. Reflex sympathetic dystrophy. In: JM Hunter, EJ Mackin, AD Callahan (eds) *Rehabilitation of the Hand*. St Louis: Mosby, 1995; 779–815.

8 Dhar S., Klenerman L. Reflex sympathetic dystrophy (algodystrophy). *British Journal of Rheumatology* 1993; **32**: 2–3.

9 Goris RJA. Reflex sympathetic dystrophy: Model of a severe regional inflammatory response syndrome. *World Journal of Surgery* 1998; **22**: 197–202.

10 Schott GD. Bisphosphonates for pain relief in reflex sympathetic dystrophy? *Lancet* 1997; **350**: 1117.

11 Dhar S, Klenerman L. Reflex sympathetic dystrophy (algodystrophy). Eular Bulletin. *Journal for Education and Information in Rheumatology* 1993; **22**(3): 81–82.

12 Allan L. Interrupting the sympathetic outflow in causalgia and reflex sympathetic dystrophy. Terminology is outdated- 1994 taxonomy should be used. *British Medical Journal* 1998; **317**: 752.

13 Kozin F. Painful shoulder and the reflex sympathetic dystrophy. In: WJ Koopman (ed) *Arthritis and Allied Conditions. Textbook of Rheumatology*. Philadelphia: Williams & Wilkins, 1997; 1887–1922.

14 Atkins MR, Duckwort I, Kanis JA. Features of algodystrophy after Colles fracture. *Journal of. Bone and Joint Surgery* 1990; **72-B**: 105–110.

15 Bickerstaff DR, Kanis JA. Algodystrophy: An under- recognized complication of minor trauma. *British Journal of Rheumatology* 1994; **33**: 240–248.

16 Veldman PHJM, Goris RJA. 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.

17 Okada M, Suzuki K, Hidaka T, Shinohara T, Kataharada K, Takada K, Tanaka H, Ohsuzu F. Complex regional pain syndrome type I induced by pacemaker implantation, with a good response to steroids and neurotropin. *Internal Medicine* 2002; **41**(6): 498–501.

18 Querol I, Cisneros T. Reflex sympathetic dystrophy syndrome following herpes zoster. *Cutis* 2001; **68**(3): 79–82.

19 Goldsmith DP, Vivino FB, Eichenfield AH, Athreya BH, Heyman S. Nuclear imaging and clinical features of childhood reflex neurovascular dystrophy: comparison with adults. *Arthitis and Rheumatology* 1989; **32**: 480–484.

20 Forouzanfar T, Koke AJ, van Kleef M, Weber WE. Treatment of complex regional pain syndrome type I. *European Journal of Pain* 2002; **6**(2): 105–122.

21 Kubalek I, Fain O, Paries J, Kettaneh A, Thomas M. Treatment of reflex sympathetic dystrophy with pamidronate: 29 cases. *Rheumatology (Oxford)* 2001; **40**(12): 1394–1397.

22 Perez RS, Kwakkel G, Zuurmond WW, de Lange JJ. Treatment of reflex sympathetic dystrophy (CRPS type 1): a research synthesis of 21 randomized clinical trials. *Journal of Pain Symptom Management* 2001; **21**(6): 511–526.

23 Mudun A, Bursali A, Oklu T, Araci A, Silahci H, Cantez S. Scintigraphic evaluation of the effectiveness of intranasal calcitonin therapy in Sudeck's atrophy. *Nuclear Medicine Communications* 1993; **14**: 805–908.

24 Rico H, Merono E, Gomez-Castresana F, Torrubiano J, Espinos D, Diaz D. Scintigraphic evaluation of reflex sympathetic dystrophy: Comparative study of course the disease under two therapeutic regimens. *Clinical Rheumatology* 1987; **6**: 233–237.

25 Schurmann M, Vogel T, Gartner A, Andress HJ, Gradl G. Experiences with calcitonin treatment of patients with type I complex regional pain syndrome (CRPS I–Sudeck disease) *Zeitschrift fur Orthopadie und ihre Grenzgebiete* 2001; **139**(5): 452–457.

26 Harden RN. A clinical approach to complex regional pain syndrome. *The Clinical Journal of Pain* 2000; **16**(2): S26–S32.

27 Livingstone JA, Atkins RM. Intravenous regional guanethidine blockade in the treatment of post traumatic complex regional pain syndrome type 1 (algodystrophy) of the hand. *Journal of Bone and Joint Surgery British* 2002; **84**(3): 380–386.

28 Kemler MA, Rijks CP, de Vet HC. Which patients with chronic reflex sympathetic dystrophy are most likely to benefit from physical therapy? *Journal of Manipulative Physiological Therapy* 2001; **24**(4): 272–278.

29 Cruess RL, Kan K, Bassett CAL. The effect of pulsing electromagnetic fields on bone metabolism in experimental disuse osteoporosis. *Clinical Orthopedics and Related Research* 1983; **173**: 245–250.

30 Mizushima Y, Akaoka I. Effects of magnetic field on inflammation. *Experientia* 1975; **31**: 1411–1412.

31 Sert C, Deniz M, Düz MZ, Akşen F, Kaya A. The preventive effect on bone loss of 50-Hz, 1-mT electromagnetic field in ovariectomized rats. *Journal of Bone and Mineral Metabolism* 2002; **20**: 345–349.

32 Pilla AA. Low-intensity electromagnetic and mechanical modulation of bone growth and repair: are they equivalent? *Journal of Orthopaedic Science* 2002; **7**: 420–428.

33 Stanton-Hicks M. Complex regional pain syndrome (Type I, RSD, Type II, Causalgia): Controversies. *The Clinical Journal of Pain* 2000; **16**(2): S33–S40.

34 Davidoff G, Morey K, Amann M, Stamps J. Pain measurement in reflex sympathetic dystrophy syndrome. *Pain* 1988; **32**: 27–34.

35 Gobelet C, Meier JL, Schaffner W, Bischof-Delaloye A, Gerster JC, Burckhardt P. Calcitonin and reflex sympathetic dystrophy syndrome. *Clinical Rheumatology* 1986; **5**: 382–388.

36 Geertzen JHB, de Bruijn H, de Bruijn-Kofman AT, Arendzen JH. Reflex sympathetic dystrophy: Early treatment and psychological aspects. *Archives of Physical Medicine and Rehabilitation* 1994; **75**: 442–446.

37 Forouzanfar T, Kemler M, Kessels AG, Koke AJ, Van Kleef M, Weber WE. Comparison of multiple against single pain intensity measurements in complex regional pain syndrome type I: analysis of 54 patients. *Clinical Journal of Pain* 2002; **18**(4): 234–237.

38 Gobelet C, Woldburger M, Meier JL. The effect of adding calcitonin to physical treatment on reflex sympathetic dystrophy. *Pain* 1992; **48**: 171–175.

39 Fialka V, Resch KL, Ritte-Dietrich D, *et al*. Acupuncture for reflex sympathetic dystrophy. *Archives of Internal Medicine* 1993; **153**: 6661–6665.

40 Rabah E, Souyet H. Stellate blocks in different ebolutive stape 5 reflex sympathetic dystrophy, 8th World Congress on Pain, 1996, P:408.

41 Jayalakshmi TS, Dreja GP. Evaluation of repeated sympathetic blockade and oral nifedipine in the sympathetically maintained pain and reflex sympathetic dystrophy of upper extremity, 8th World Congress on pain, 1996, P:408.

42 Stenberg VI, Tveter K, Maxwell J. Accurate measurement of extent of hand swelling in patients with rheumatoid arthritis. *Arthritis and Rheumatism* 1980; **23**: 1411–1412.

43 Butler SH, Galer BS, Benirschke S. Sympathetic nervous system and pain: Clinical disease as a cause of sign and symptom of CRPS(I), 8th World Congress on Pain, 1996, P:401.

44 Sarangi PP, Ward AJ, Smith EJ, Staddon GE, Atkins RM. Algodystrophy and osteoporosis after tibial fractures. *Journal of Bone and Joint Surgery* 1993; **75-B**, 450–452.

45 Bickerstaff DR, Kanis JA. The use of nasal calcitonin in the treatment of post traumatic algodystrophy. *British Journal of Rheumatology* 1991; **30**: 291–294.

46 Walsh MT. Therapist's management of reflex sympathetic dystrophy. In: JM Hunter, AD Callahan (eds) *Rehabilitation of the Hand*. St Louis: Mosby, 1995; 817–833.

47 Doury P, Dequeker J. Regional bone diseases, Algodystrophy/Reflex sympathetic dystrophy syndrome. In: JH Klippel, PA Dieppe (eds) *Rheumatology*. London: Mosby, 1994; 7, 38.1–7.

48 Staton-Hicks M. Upper and lower extremity pain. In: PP Raj (ed) *Practical Management of Pain*. St Louis: Mosby, 1992; 312–327.

49 Zyluk A, Birkenfeld B. Quantitative evaluation of three-phase bone scintigraphy before and after treatment of post traumatic reflex sympathetic dystrophy. *Nuclear Medicine Communications* 1999; **20**: 327–333.