Incidence of complex regional pain syndrome type I after fractures of the distal radius

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Abstract

Aim of this study was to analyse the incidence CRPS-I after a fracture of the distal radius and to analyse risk factors. Patients who visited the Emergency Unit of the University Hospital, with a fracture of distal radius were asked to participate. As risk factors for CRPS-I, number of repositions (with or without local anaesthesia), additional cast changes and pain during the cast period, were assessed. In a structured interview social life events (SLEs) and psychological and/or psychiatric history were assessed. The patients filled out the Symptom Checklist-90 (SCL-90). In total 88 patients participated in the study. One female (1%, 95% CI: 0.2 to 6%), age 69 years with the following characteristics developed CRPS-I: one set of local anaesthetics, one repositioning attempt, no additional cast changes, average pain scores, no life events and her total score on the SCL-90 of 117, was slightly above average. Based on the results of this study it is concluded that the incidence of CRPS-I may be low (1%, 95% CI: 0.2 to 6%) after fractures of the distal radius. Further the risk factors described in literature play a minor role in the development of CRPS-I.

CRPS-I may follow a variety of inciting events including soft tissue contusions, fractures, tendon ruptures, and carpal tunnel release or other forms of surgery (Geertzen et al., 1994; Geertzen et al., 1998b). The most common inciting event for CRPS-I is a fracture of the distal radius (Veldman, 1995).

1. Introduction

Complex Regional Pain Syndrome type I (CRPS-I), is a complex clinical syndrome characterised by pain or allodynia or hyperalgesia, edema, changes in skin blood flow, or abnormal sudomotor activity. Additionally the pain is disproportionate to the inciting event (Merskey and Bogduk, 1994). A variety of terms, definitions and names have been used to describe this syndrome and to focus attention on different aspects of CRPS-I, etiology, symptomatology and/or supposed pathophysiology (Veldman, 1995). For this syndrome many different names are found: 79 names in the anglo saxon literature, 33 names in the French literature and 51 names in the German literature (Veldman, 1995). The names most frequently used are: Complex Regional Pain Syndrome type I, Reflex Sympathetic Dystrophy, Sudeck’s atrophy and algodystrophy (Veldman, 1995).
From a theoretical point of view it can be hypothesised that SLE and psychological or psychiatric problems might be facilitating factors for the development of CRPS-I (Van Houdenhove et al., 1992). Social life events may play a role as life stress factors increasing nociception in patients suffering from CRPS-I. In a case-control study it was found that subjects with a SLE in the period of the inciting event had a higher chance of developing CRPS-I as compared to subjects without a SLE in that period (OR 13.1, 95% CI: 3.8 to 44.9) (Geertzen et al., 1998a). Others found that patients suffering from CRPS-I differed significantly from controls with respect to depression, anxiety, interpersonal sensitivity, and somatisation (Hardy and Merritt, 1988). Many of these risk factors, however, have been identified in retrospective studies or have merely been postulated without prospective studies.

Aim of this study was to estimate the incidence of CRPS-I and to describe risk factors for the development of CRPS-I of patients suffering from a fracture of the distal radius.

### 2. Patients and methods

All patients who visited the Emergency Unit (in the period of 1 year) of the University Hospital, with a fracture of distal radius were asked to participate in this prospective study, the day after the accident. Excluded were patients younger than 10 years and patients with a bilateral fracture of the distal radius and patients with multiple traumata. Additionally patients with problems understanding the Dutch language were excluded.

All patients were treated with a plaster of Paris cast during 5 weeks (changed once) or were treated operatively. Pain medication was provided if needed. More than one repositioning attempt, more than one set of local anaesthetics and additional cast changes were scored as risk factors. After giving written informed consent, the patients were interviewed concerning dominance of the affected limb, nature of the accident, occupation, etc.

The weeks following the accident, the patients were seen by the researchers on the same days as they visited the trauma or orthopaedic surgeon. The research follow-up was as follows: the day after the accident (T0); two weeks after the accident, when the cast was changed (T1); five weeks after the accident, when the cast was definitively removed (T2); seven weeks after the accident (T3); 3 weeks later, thus 10 weeks after the accident (T4) and one year after the accident (T5).

At T1, the patients were asked to assess the pain experienced in the last 24 h, on a 100 mm straight line (0 = no pain, 100 = severe pain; VAS Pain) a score of 40 mm or more was scored as risk factor. Further the patients were asked in a structured interview whether social life events (SLEs) were present in the time span 3 months before the accident. All SLE values from the Social Readjustment Rating Scale with a Life Change Unit (LCU) rating of 35 or higher were scored (Rahe, 1972). The Social Readjustment Rating Scale is a hierarchy-list of 42 events, such as divorce, death of a partner or near family member, etc. (Rahe, 1972). Additionally the patients’ psychological or psychiatric history was assessed in the structured interview. At T2, the Symptom Checklist-90 (SCL-90) were completed by all patients older than 17 years (n = 79) (Arrindel and
The SCL-90 is a multidimensional self-reported inventory composed of 90 items that measures the following dimensions: 1. anxiety, 2. agoraphobia, 3. depression, 4. somatisation, 5. inadequacy, 6. sensitivity, 7. hostility, 8. insomnia, 9. and a remaining score. The sum-score is a measure for psycho-neuroticism or emotional instability. A sum-score of 200 or more was scored as a risk factor.

At T2, T3 and T4 the patients were examined and the diagnosis CRPS-I was made if the patient fulfilled the criteria of the IASP: continuing pain or allodynia or hyperalgesia, with which the intensity of the pain is disproportionate to the inciting event, evidence at some time of edema or changes in skin blood flow, or abnormal sudomotor activity was present in the region of the pain (Merskey and Bogduk, 1994). The diagnosis was excluded by the existence of conditions that would otherwise account for the degree of pain and dysfunction (Merskey and Bogduk, 1994). At T5, one year after fracturing the wrist the patients were contacted by telephone and asked if they had experienced any signs and symptoms of CRPS-I in the post-fracture period.

This study was approved by the medical ethical board of our University Hospital.

3. Statistics

Data analysis performed in SPSS-package included descriptive statistics.

<table>
<thead>
<tr>
<th>NonCRPS-I group (n = 87)</th>
<th>CRPS-I patient (n = 1)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Subjects</td>
<td>58, 59: 29</td>
</tr>
<tr>
<td>Age: mean (SD)</td>
<td>47.5 (19.4)</td>
</tr>
<tr>
<td>Fracture at dominant side</td>
<td>36</td>
</tr>
<tr>
<td>Treatment surgical</td>
<td>2</td>
</tr>
<tr>
<td>Risk factors</td>
<td></td>
</tr>
<tr>
<td>&gt;1 set of local anaesthetics</td>
<td>6</td>
</tr>
<tr>
<td>&gt;1 reposition attempt</td>
<td>17</td>
</tr>
<tr>
<td>Additional cast changesa</td>
<td>29</td>
</tr>
<tr>
<td>VAS pain ≥ 40b</td>
<td>2</td>
</tr>
<tr>
<td>LCU score ≥ 35b</td>
<td>14</td>
</tr>
<tr>
<td>Psychological and/or psychiatric history</td>
<td>0</td>
</tr>
<tr>
<td>SCL-90 ≥ 200c</td>
<td>1</td>
</tr>
</tbody>
</table>

Sum of risk factors

| 0          | 40 | 1            |
| 1          | 30 |
| 2          | 13 |
| 3          | 3  |
| 4          | 1  |

a Within the first two weeks after the accident.

b Two weeks after the accident.

c Five weeks after the accident.

4. Results

In the period of one year 91 patients visited the Emergency Unit with a distal radius fracture. Of these 91 patients 88 were seen at T1; these 88 patients all participated in this study. The Symptom Checklist-90 (SCL-90) was completed by 79 patients (older than 17 years).

One female, age 69, (1%, 95% confidence interval: 0.2 to 6%) developed CRPS-I. She had none of the risk factors assessed in this study: one set of local anaesthetics, one repositioning attempt, no additional cast changes. Two weeks after the accident the patient who developed CRPS-I had a pain intensity of 11 on a scale of 100mm, which was slightly above average of the group without CRPS-I (mean 7.2 mm; SD:10.3). She did not have any life events and her total score on the SCL-90 of 117, was slightly above average of the group without CRPS-I (mean: 110.6; SD: 27.2). Risk factors of the patients without and with CRPS-I are summarised in Table 1. Forty-seven patients (57%) had one or more risk factors, whereas 17 patients (20%) had two or more risk factors and 4 (5%) had three or more risk factors.

The diagnosis CRPS-I was made seven weeks after the accident, when severe swelling and discoloration was present and pain was still experienced, which could not be explained by the presence of another condition. She was treated with DMSO 50% in a fatty cream and she was send to a physical therapist. One year after the fracture she was experienced occasionally some pain, 2 on a scale of 100 mm and some slight swelling. The range of motion of hand and shoulder was not
restricted, and the function of the hand was normal. There were no trophic or sudomotor or vasomotor differences between affected and nonaffected side.

Of the other 87 patients 71 (82%) could be reached and none of these patients reported any signs and symptoms of CRPS-I.

5. Discussion

The incidence of CRPS-I after fractures of the distal radius found in this study is low (1%, 95% CI: 0.2 to 6%). This incidence of CRPS-I agrees with the low incidences, 0.9% and 7%, found in previous studies, but in considerable disagreement with high incidences, such as 15–37% (Atkins et al., 1989; Bickerstaff and Kanis, 1994; Cooney et al., 1980; de Bruijn, 1987; Field and Atkins, 1997; Hove, 1995). From the point estimators and the 95% confidence intervals the wide range of incidences can be seen (Fig. 1). Additionally the 95% confidence intervals of several studies do not overlap indicating considerable differences in the estimated incidence. The incidence of CRPS-I seems to be dependent on the criteria used in the different studies (Table 2).

Very high incidences after Colles’ fractures were found in several studies (Atkins et al., 1989; Bickerstaff and Kanis, 1994; Field and Atkins, 1997). However, the diagnostic criteria used in those studies differ considerably from the studies with the lower incidences such as de Bruijn (1987) and ours (Table 2) (de Bruijn, 1987).

According to the criteria used by some authors, the diagnose CRPS-I can be made in the absence of pain (de Bruijn, 1987; Zollinger et al., 1999). This phenomenon is odd since CRPS-I is a pain syndrome (Merskey and Bogduk, 1994). Roumen et al. (1994) referred for diagnostic criteria of CRPS-I to a publication of Goris (1985), however, in that paper no diagnostic criteria for CRPS-I were specified (Goris, 1985; Roumen et al., 1991). Additionally, two papers did not specify clearly their diagnostic criteria (Cooney et al., 1980; Dresing et al., 1994).

The risk factors for CRPS-I identified in literature could not be confirmed in our study. Many patients without CRPS-I needed more than one set of local anaesthetics ($n = 6$), endured several repositioning attempts ($n = 6$) or received more than one cast during the first 2 weeks after the injury ($n = 29$). The patient who developed CRPS-I needed one set of local anaesthetic,

<table>
<thead>
<tr>
<th>Authors</th>
<th>Algorithm for CRPS-I</th>
<th>Symptoms</th>
</tr>
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<tbody>
<tr>
<td>de Bruijn (1987)$^a$</td>
<td>Three or more symptoms present</td>
<td>Severe pain, Swelling, Hyperaemia, Hyperaesthesia, Hyperhydrosis, Restricted range of motion$^b$</td>
</tr>
<tr>
<td>Atkins et al. (1989, 1990), Bickerstaff and Kanis (1994), Field and Atkins (1997)</td>
<td>Four symptoms present</td>
<td>Tenderness of the fingers, Swelling of the hand, Reduced range of motion, Vasomotor instability</td>
</tr>
<tr>
<td>Hove (1995)</td>
<td>No specific algorithm described</td>
<td>Diffuse pain, Loss of function, Autonomic dysfunction</td>
</tr>
<tr>
<td>Zollinger et al. (1999)</td>
<td>Four or more symptoms present$^c$</td>
<td>Unexplained diffuse pain, Change in skin temperature$^d$, Change in skin colour$^d$, Diffuse oedema, Limitation in range of motion$^e$, Increase of symptoms after activity</td>
</tr>
<tr>
<td>Current study: Dijkstra</td>
<td>All criteria required</td>
<td>Continuing pain or allodynia or hyperalgesia, Pain, disproportionate to the inciting event, Evidence at some time of edema, or changes in skin blood flow, or abnormal sudomotor activity in the region of the pain, Diagnosis is excluded by the existence of conditions that would otherwise account for the degree of pain and dysfunction</td>
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$^a$ CRPS-I stage 1.

$^b$ Less than 1/3 of the nonaffected side.

$^c$ In an area larger than the wrist.

$^d$ Compared to the nonaffected side.

$^e$ Not related to the stage of fracture treatment.
endured one repositioning attempt and did not require additional cast changes during the first two weeks after accident. Additionally, the patient who developed CRPS-I, seven weeks after fracturing her wrist, had a pain intensity of 11 on a scale of 100 mm two weeks after the accident. This pain intensity is slightly above average of the group at two weeks. She did not have any life events and her total score on the SCL-90 of 117, was slightly above average of the group without CRPS-I. Thus the risk factors previously identified did not lead to CRPS-I. This might indicate that the influence of the risk factors on the development of CRPS-I is somewhat weaker than is suggested in literature.

Field et al. (1994) described that tightness of the cast after a Colles' fractures could be an inciting factor in development of CRPS-I (Field et al., 1994). As a result of this tightness the patients requested cast changes. Thus the number of cast changes may be an indicator for tightness of the cast and thus might be a predictor (risk factor) for CRPS-I. It was found that that 5 of 6 patients developing CRPS-I had a tight cast whereas 1 out of the 17 patients not developing CRPS-I had a tight cast (Field et al., 1994). However, it can be argued that the swelling accompanying CRPS-I was responsible for the patients experiencing tightness of the cast. Additionally, the excessive pain may be the result of the swelling and resulting in a tightness of the cast but it may also be the first sign of CRPS-I and the extra cast changes can also be interpreted as the result of CRPS-I (Field and Atkins, 1997).

SLE, psychological and psychiatric problems have also been described as risk factors for CRPS-I (Geertzen et al., 1998a,c; Van Houdenho, 1986). More or less from a theoretical point of view Van Houdenho et al. (1992) postulated that psychological or psychiatric problems might be facilitating factors for the development of CRPS-I. Additionally, Geertzen et al. (1998a) found in a cohort of CRPS-I patients a large percentage (60%) of patients with a psychological or psychiatric history. SLE can be seen as a life stressor increasing nociception in patients suffering from CRPS-I. In a case-control study it was found that subjects with a SLE in the period of the inciting event had an higher chance in developing CRPS-I as compared to subjects without a SLE in that period (Geertzen et al., 1998a). But because of the retrospective character of case-control studies selection bias and information bias may be present in that study. Additionally, it can be argued that these psychological and psychiatric problems are the result of CRPS-I and not the cause of it.

A weakness of this study was that the number of patients we could include in this study was rather low. Because of limited finances the study could not be continued for an extended period. This lack of patients may have influenced the outcome of the study and because only one subject developed CRPS-I no 'risk profile' could be identified. However, also other prospective studies have failed to include large samples of patients with fractures of wrist in their studies. Looking at the legends of Fig. 1, it can be seen that of the eight prospective studies, six have a study sample similar to ours.

It can be hypothesised that some subjects might have developed CRPS-I after the follow-up was closed and as a consequence the estimation of the incidence of CRPS-I is biased towards a lower estimation of CRPS-I. However, in our hospital, our department is known for its expertise in CRPS-I and patients would have been referred to our department for diagnosis and therapy if CRPS-I was suspected even if the follow-up was closed. No such patients were referred to our department.

Based on the results of this study it is concluded that the incidence of CRPS-I is after fractures of the distal radius might be low (1%, 95% CI: 0.2 to 6%). Further risk factors may play a less important role in the development of CRPS-I as is suggested in literature. Future, large and prospective studies with uniform diagnostic criteria are needed to confirm or refute our results with respect to incidence and risk factors.

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References


