Acute stress response and recovery after whiplash injuries.
A one-year prospective study

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Abstract

Chronic whiplash-associated disorder (WAD) represents a major medical and psycho-social problem. The typical symptomatology presented in WAD is to some extent similar to symptoms of post traumatic stress disorder. In this study we examined if the acute stress reaction following a whiplash injury predicted long-term sequelae. Participants with acute whiplash-associated symptoms after a motor vehicle accident were recruited from emergency units and general practitioners. The predictor variable was the sum score of the impact of event scale (IES) completed within 10 days after the accident. The main outcome measures were neck pain and headache, neck disability, general health, and working ability one year after the accident. A total of 737 participants were included and completed the IES, and 668 (91%) participated in the 1-year follow-up. A baseline IES-score denoting a moderate to severe stress response was obtained by 13% of the participants. This was associated with increased risk of considerable persistent pain (OR = 3.3; 1.8–5.9), neck disability (OR = 3.2; 1.7–6.0), reduced working ability (OR = 2.8; 1.6–4.9), and lowered self-reported general health one year after the accident. These associations were modified by baseline neck pain intensity. It was not possible to distinguish between participants who recovered and those who did not by means of the IES (AUC = 0.6). In conclusion, the association between the acute stress reaction and persistent WAD suggests that post traumatic stress reaction may be important to consider in the early management of whiplash injury. However, the emotional response did not predict chronicity in individuals.

Keywords: Neck pain; Post-Traumatic stress disorder; Prognosis; Psychology; Whiplash injuries

1. Introduction

Persistent symptoms after an acute whiplash injury known as chronic whiplash-associated disorders (WAD) are characterized by pain in the neck and surrounding areas together with a series of cognitive complaints. The mechanisms underlying WAD are not clear, but a bio-mechanical as well as a psycho-social genesis have been proposed. Injury to cervical facet joints (Lord et al., 1996; McDonald et al., 1999), proprioceptors (Gimse et al., 1996; Treleaven et al., 2005) and upper cervical ligaments (Krakenes et al., 2002;
Krakenes et al., 2004) have been suggested as causes of chronic WAD. Other trials point towards the importance of psychological factors, such as coping strategies (Carroll et al., 2006; Soderlund and Lindberg, 2003) and the emotional reaction to being involved in a traffic accident (Drottning et al., 1995; Sterling et al., 2003; Sterling et al., 2006). Moreover, cultural factors seem to be important since the risk of chronicity is observed to be minimal or absent in cultures in which there are only few factors that potentially accelerate sickness impact, such as focus on disability, access to a wide variety of ineffective treatment options (Cote et al., 2005; Kongsted et al., 2007) and compensation systems (Cassidy et al., 2000). Lithuania and Greece represent such a culture, and studies from these countries have shown no long-term disability due to whiplash (Obelieniene et al., 1999; Partheni et al., 2000; Schrader et al., 1996; Schrader et al., 2000). This contrasts observations in countries with great awareness of chronic consequences due to whiplash in the general public. In Scandinavia, as many as 40–50% of whiplash exposed are reported to develop chronic symptoms (Borchgrevink et al., 1997; Hildingsson and Toolanen, 1990; Karlsborg et al., 1997; Kasch et al., 2001). Studies from Lithuania included participants from police records and these populations are therefore not directly comparable to the Scandinavian populations recruited in emergency units. The latter studies included persons who sought care because of acute symptoms, whereas the former covered cohorts that were included because of the exposure to a car collision without any selection in relation to reported early symptoms. Still it is striking that chronicity rates differ to such great extent.

Musculoskeletal pain, headache and difficulties with short time memory and concentration are described in both WAD and in post-traumatic stress disorder (PTSD) (Norman et al., 2007; Ouimette et al., 2004; Rodriguez et al., 2004; Sterner and Gerdle, 2004), and an overlap between these conditions has been suggested (Kuch et al., 1996; McLean et al., 2005b; McLean et al., 2005a). The diagnosis PTSD is established through a clinical interview, but self-report instruments have been developed as a practical tool to assess the degree of post traumatic stress response. The impact of event scale (IES) (Horowitz et al., 1979; Sundin and Horowitz, 2002) is the most widely used instrument. It has been validated and found to be a useful measure of stress reactions in a large number of populations exposed to various traumatic experiences, including road traffic accidents (Sundin and Horowitz, 2002; Sundin and Horowitz, 2003), and has been used in previous trials regarding whiplash injuries (Drottning et al., 1995; Sterling et al., 2003).

Acute post traumatic stress symptoms were observed to be associated with an increased risk of persistent neck pain and neck disability following a whiplash injury in two trials (Drottning et al., 1995; Sterling et al., 2003; Sterling et al., 2006). However, one of these trials had a follow-up of only 4 weeks (Drottning et al., 1995) and the other studied a relatively small population resulting in only 17 cases with significant long-lasting symptoms (Sterling et al., 2003; Sterling et al., 2006). If it holds true, also from larger trials that an intense post traumatic stress reaction is related to increased risk of chronic WAD, this problem should be specifically dealt with, rather than solely focusing on the neck injury. Since results of conservative interventions after whiplash injury have been discouraging (Kongsted et al., 2007; Verhagen et al., 2004), it is of major importance to identify patients in whom other mechanisms behind the development of chronicity are relevant.

The objective of this study was to investigate whether the acute emotional stress reaction, as assessed by the IES, predicted the development of long-term pain and disability after a whiplash injury.

2. Methods

2.1. Study design

This prospective trial was performed as a two-center study. Part of the study population was allocated to a randomized clinical trial and received one of three interventions: (1) Immobilization in a semi-rigid collar followed by exercises, (2) active mobilization, or (3) advice to act as usual. None of these interventions were directed towards post traumatic stress, acute stress scores did not differ between groups, and there was no significant difference in outcome after 1-year in these three treatment groups (Kongsted et al., 2007). Other participants were randomized to either written or oral advice to act as usual in another randomized trial (Fig. 1). Follow-up was performed 3, 6 and 12 months after the injury. The present study included data only from the 1-year follow-up, since the long-term prognosis was the object of interest. Other prognostic evaluations will be published elsewhere. The trial was approved by the local ethical committee and was carried out in accordance with the Helsinki II declaration. Data collection was permitted by the Danish Data Protection Agency. Recruitment took place between May 2001 and June 2003 and follow-up was completed in October 2004.

Participants were recruited from emergency units and general practitioners in four Danish counties covering approximately 1.7 million inhabitants. Persons who had acute symptoms after a rear-end or frontal car collision were considered potential participants. All eligible participants received a visit by a project nurse in their home. If inclusion criteria were met, written consent to participate was obtained at this visit after verbal and written information about the study. Inclusion criteria were: Age 18–70 years, debut of symptoms within 3 days
after the motor vehicle accident (MVA) and a maximum of 10 days should pass from the MVA to inclusion. Exclusion criteria were: Fractures or dislocations of the cervical spine visualized on X-ray, amnesia connected to the accident, unconsciousness in relation to the accident, injuries other than the whiplash injury, self-reported average neck pain during the preceding 6 months exceeding 5 on a box scale 0–10, where 0 = no pain and 10 = worst possible pain, significant pre-existing somatic or psychiatric disease, and known alcohol- or drug abuse. Subjects were also excluded if they could not read or understand Danish. These criteria resulted in inclusion of WAD grades I–III (Spitzer et al., 1995).

2.2. Variables of interest

2.2.1. Baseline variables

A post traumatic stress score was obtained by the IES. A total sum-score was calculated from all 15 items of the scale (0–8 = subclinical; 9–25 = mild; 26–43 = moderate; 44–75 = severe stress reaction). In addition, an intrusion score (sum of 7 items) and an avoidance score (sum of 8 items) were calculated (Horowitz et al., 1979). Intrusion is symptoms related to reexperiencing the traumatic event. The items covering avoidance disclose whether situations, which remind the subject about the event, are avoided. The total sum-score was the explaining variable of interest. Other questionnaires included assessment of general health by the SF-36 (Bjorner et al., 1998a; Bjorner et al., 1998b), neck pain and headache intensity rated on 11-point box scales (0 = no pain and 10 = worst possible pain) (Jensen et al., 1989), sociodemographic factors, and crash related data. Two summary scores, a physical health score and a mental health score, are obtained from the SF-36 scale. The SF-36 was directed at self-experienced health four weeks prior to the car accident. In addition, participants scored their average neck pain and headache 6 months prior to the accident on 11-point box scales.

2.2.2. Measures of non-recovery

Outcome parameters were chosen so that aspects of self-reported pain, neck disability, general health and work-related consequences one year after the MVA were covered. Follow-up data were collected from a mailed questionnaire. Participants were contacted by phone if the questionnaires were not returned. In case subjects declined to fill in the 1-year questionnaire, they were asked to participate in a short telephone interview. This included whether symptoms related to the accident were still present and information about their working ability the preceding month. The main outcome parameters were self-reported average neck pain and headache intensity the preceding week (11 point box scales; 0 = no pain, 10 = worst imaginable pain), neck disability (15-item Copenhagen Neck Functional Disability Scale; 0 = no neck disability, 30 = extremely disabled) (Jordan et al., 1998), general health (SF-36), and reduced ability to work during the 12th month after the injury (‘‘unaffected workability’’, ‘‘reduced working hours’’, ‘‘off sick’’) marked day by day in a calendar.
2.2.3. Validity

The IES has been widely used (Sundin and Horowitz, 2003) and found to be a valid measure of post traumatic stress reactions (Joseph, 2000; Sundin and Horowitz, 2002), but its reproducibility has been evaluated only in one small trial (Horowitz et al., 1979). The scales for measuring pain and neck disability have been validated in other spinal pain populations (Bolton and Wilkinson, 1998; Jordan et al., 1998). The short-form health survey, SF-36, is considered a valid instrument measuring self-reported general health (Björner et al., 1998a; Brazier et al., 1992), but there is a risk that pre-existing health problems reported at baseline are underreported when using the scale retrospectively as in this study. Crash-related data including impact direction (frontal/rear end) and car damage were self-reported and the validity of this information is unknown. Information regarding car damage should be considered a rough estimate only. Working ability during the 12th month after the injury was registered by marking days with sick listing and reduced working hours in a calendar constructed for this trial. The answers of the calendars were controlled on a spot sample basis by a secretary who checked that participants understood how to fill it in.

2.3. Statistical methods

The IES-score was dichotomized into “mild stress response” (IES < 26) and “distinct stress response” (IES 26–75) (Horowitz et al., 1979) in the analyses. In case of incomplete IES-scores, missing items were replaced by the mean-value of the completed questions to calculate the total IES-score. The variables neck pain intensity and headache intensity were merged into one pain measure by using the higher of the two scores as the pain outcome in the analyses. Pain scores from 0 to 3 and disability scores from 0 to 6 were defined as “minimal” based on previously suggested categories (Jordan et al., 1998) and the distribution of the variables. Missing items in the neck disability scale were replaced by worst case scores if a maximum of 2/15 items were missing, and no disability-score was calculated if more items were missing. Working ability was defined as “affected” if any days with reduced working hours or days off sick were present during the 12th month after the accident, and “unaffected” when a person had not been sick listed or worked reduced hours due to the accident during that month.

Associations between the IES-score and outcome measures were evaluated by means of linear and logistic regression using robust variance estimation (Royall, 1986; White, 1980). Two models were tested: In model I the regression was adjusted for gender. In model II the regression models were furthermore adjusted for baseline pain intensity, since an association between baseline pain intensity and the IES was observed, and initial pain is known to be associated with prognosis (Scholten-Peeters et al., 2003). Self-reported car damage and impact direction were tested as modifiers, but were not associated with outcome and hence not included in the regression model. Results are presented as odds ratios (OR) and regression coefficients with 95% confidence intervals (95% CI). In case of positive associations, the ability of the IES to distinguish between patients with good and poor outcome was tested by means of receiver operator curves (ROC) and presented as area under the curve (AUC) (Metz, 1978). All analyses were performed using STATA 8 (release 8.2, Stata Corp., TX).

3. Results

3.1. Study sample and baseline characteristics

A total of 740 participants were included in the study, and 737 (265 males/472 females), median age 33 years (IQR 26–43), filled in the baseline IES. One year follow-up data were analyzed for 668 participants; 511 who completed the follow-up questionnaires and 157 who did not respond to the mailed questionnaire but participated in a short telephone interview. These 157 were included only in the analysis concerning working ability (Fig. 1).

Baseline data were obtained a median of five days after the accident (IQR 3–6). All participants reported neck discomfort. Neck pain was reported by 99% of the population and headache by 86%. Neck pain or headache intensity of at least 4 was reported by 65% of the population. Baseline characteristics are shown in Table 2.

All items of the IES were completed by 721 participants and 1–4 items were missing in 16 IES-questionnaires. A mild stress response was observed in 87% of the participants and distinct stress was present in 13% (8% males, 15% females) (Fig. 2). Data on the IES are shown in Table 1. A distinct stress response was associated with female gender (OR 1.9; 95% CI 1.2–3.2), and higher initial pain intensities (Fig. 3). An increased IES-score was also associated with self-reported car damage of more than 50% (OR 2.6; 95% CI 1.5–4.6), and with the accident described as severe (OR 5.0; 95% CI 3.0–8.4). Self-reported general health (SF-36), neck pain and headache intensities prior to the accident did not differ significantly between participants with mild and distinct stress responses.

3.2. Measures of non-recovery

At the 1-year follow-up, 43% (95% CI: 39–48) of 510 participants reported considerable neck pain and/or...
headache and 34% (95% CI: 29–38) considerable neck disability. In the 12th month after the MVA, 9% (95% CI 1.8–5.9) were sick listed the entire month and 16% (95% CI: 13–19) had some extent of affected working ability. The average SF-36 scores were slightly lower, denoting worse health, at the 1-year follow-up compared to four weeks preceding baseline (Table 2).

3.3. Prediction of outcome by IES

The baseline IES-score was significantly associated with all outcome measures in the primary analysis (Table 3, model I) (Fig. 4). Significant pain at 1-year follow-up was reported by 68% of the participants with a distinct stress response and by 40% of those with a mild stress response at baseline (OR 3.3; 95% CI 1.8–5.9). Significant disability was reported by 58% (distinct stress) and 31% (mild stress) (OR 3.2; 95% CI 1.7–6.0). In the group with mild stress response, 14% reported reduced working ability in the 12th month after the car collision, whereas this was the case for 31% of participants with a distinct stress response (OR 2.8; 95% CI 1.6–4.9). When baseline pain intensity was taken into account, the association between IES-scores and outcome measures were lowered, and the association with working ability was no longer statistically significant (Table 3, model II). The observed associations were insufficient to distinguish between the patients with considerable pain or reduced working ability from those recovering since ROC analysis revealed quite low AUC for the IES-score (pain = 0.6, work = 0.6, disability = 0.7).

Since baseline pain and gender modified the effect of the early stress reaction, data were analyzed separately in groups with low and high initial pain intensities and within each gender. Regarding initial pain, data were analyzed separately in the 189 participants with low intensity baseline pain (0–3) and those 173 with high initial pain intensity (6–10). In participants with low pain a sevenfold risk of persistent considerable pain in case of a distinct stress reaction was estimated (OR 7.1; 95% CI 2.3–21.8). In participants with high initial pain, the stress score was not significantly associated with outcome measures.

**Table 1**

<table>
<thead>
<tr>
<th>Impact of event score at baseline</th>
<th>All subjects</th>
<th>Females</th>
<th>Male</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total IES n = 737</td>
<td>9 (3–18)</td>
<td>10 (5–21)</td>
<td>6 (2–15)</td>
</tr>
<tr>
<td>Intrusive items n = 727</td>
<td>5 (2–11)</td>
<td>6 (3–12)</td>
<td>3 (1–8)</td>
</tr>
<tr>
<td>Avoidance items n = 730</td>
<td>3 (0–7)</td>
<td>4 (1–8)</td>
<td>2 (0–6)</td>
</tr>
</tbody>
</table>

IES = Impact of event score. Median scores (interquartile range).

**Table 2**

<table>
<thead>
<tr>
<th>Baseline 1-year follow-up</th>
</tr>
</thead>
<tbody>
<tr>
<td>n</td>
</tr>
<tr>
<td>Rear end collision (direct or oblique), %</td>
</tr>
<tr>
<td>Neck pain (0–10), median (IQR)</td>
</tr>
<tr>
<td>Neck pain &gt;3, % (CI)</td>
</tr>
<tr>
<td>Headache (0–10), median (IQR)</td>
</tr>
<tr>
<td>Headache &gt;3, % (CI)</td>
</tr>
<tr>
<td>Neck disability (0–30), median (IQR)</td>
</tr>
<tr>
<td>Neck disability &gt;6, % (CI)</td>
</tr>
<tr>
<td>Physical health (SF-36), median (IQR)</td>
</tr>
<tr>
<td>Mental health (SF-36), median (IQR)</td>
</tr>
<tr>
<td>Sicklisted, % (CI)</td>
</tr>
<tr>
<td>Reduced working ability during the 12th month, % (CI)</td>
</tr>
</tbody>
</table>

* Baseline scores of SF-36 denotes general health prior to the accident IQR = interquartile range. CI = 95% confidence interval.

Fig. 2. Percentage of participants within each stress-reaction category. Percentage of females and males with an IES-score corresponding to the illustrated categories. Moderate and severe were considered a “distinct stress reaction” in the analyses.

Fig. 3. Baseline pain intensity in relation to stress response. Baseline neck pain and headache intensity by IES. IES = Impact of event score.
Table 3

Associations between total baseline IES and outcome measures

<table>
<thead>
<tr>
<th>Explaining variables at baseline</th>
<th>Outcome variables at 1-year</th>
<th>Odds ratio [Robust 95% CI]</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Crude</td>
<td>Model I</td>
</tr>
<tr>
<td><strong>Pain &gt;5</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>IES (distinct stress vs. others*)</td>
<td>3.3 [1.8; 5.9]**</td>
<td>3.0 [1.6; 5.5]**</td>
</tr>
<tr>
<td>Gender (female vs. male*)</td>
<td>2.1 [1.4; 3.1]**</td>
<td>1.9 [1.3; 2.9]**</td>
</tr>
<tr>
<td>Baseline pain intensity (0*-10)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Reduced working ability</td>
<td></td>
<td></td>
</tr>
<tr>
<td>IES (distinct stress vs. others*)</td>
<td>2.8 [1.6; 4.9]**</td>
<td>2.7 [1.5; 4.7]**</td>
</tr>
<tr>
<td>Gender (female vs. male*)</td>
<td>1.6 [1.0; 2.6]**NS</td>
<td>1.5 [0.9; 2.4]**NS</td>
</tr>
<tr>
<td>Baseline pain intensity (0*-10)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Disability &gt;6</td>
<td></td>
<td></td>
</tr>
<tr>
<td>IES (distinct stress vs. others*)</td>
<td>3.2 [1.7; 6.0]**</td>
<td>3.1 [1.6; 5.8]**</td>
</tr>
<tr>
<td>Gender (female vs. male*)</td>
<td>1.5 [1.0; 2.2]**NS</td>
<td>1.3 [0.8; 2.0]**NS</td>
</tr>
<tr>
<td>Baseline pain intensity (0*-10)</td>
<td></td>
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</tr>
</tbody>
</table>

Regression coefficient [Robust 95% CI]

| **Pain (0–10)**                  |                            |                           |                           |
| IES (distinct stress vs. others*) | 2.4 [1.4; 3.4]**           | 2.3 [1.3; 3.3]**          | 1.5 [0.5; 2.4]**          |
| Gender (female vs. male*)        | 0.9 [0.3; 1.4]**           | 0.6 [0.1; 1.2]**          | 0.6 [0.5; 0.7]****        |
| Baseline pain intensity (0*-10)  |                            |                           |                           |
| **Disability (0–30)**            |                            |                           |                           |
| IES (distinct stress vs. others*) | 3.2 [1.7; 6.0]**           | 3.1 [1.6; 5.8]**          | 2.1 [1.1; 4.2]**          |
| Gender (female vs. male*)        | 1.5 [0.9; 2.2]**NS         | 1.3 [0.9; 2.0]**NS        | 1.4 [1.2; 1.5]****        |
| Baseline pain intensity (0*-10)  |                            |                           |                           |
| **SF-36 physical health**        |                            |                           |                           |
| IES (distinct stress vs. others*) | −7.7 [−11.0; −4.3]**       | −7.3 [−10.7; −4.0]**      | −5.1 [−8.3; −1.9]**       |
| Gender (female vs. male*)        | −2.4 [−4.2; −0.6]**        | −1.8 [−3.5; −0.1]**       | −1.6 [−2.0; −1.2]****     |
| Baseline pain intensity (0*-10)  |                            |                           |                           |
| **SF-36 mental health**          |                            |                           |                           |
| IES (distinct stress vs. others*) | −7.6 [−10.9; −4.2]**       | −7.5 [−10.9; −4.1]**      | −6.1 [−9.5; −2.8]****     |
| Gender (female vs. male*)        | −0.7 [−2.5; 1.0]**NS       | −0.4 [−2.1; 1.4]**NS      | −0.9 [−1.4; −0.5]****     |
| Baseline pain intensity (0*-10)  |                            |                           |                           |

Effect of the IES score on pain, working ability and general health after one year. Model I was adjusted for gender. Model II was adjusted for gender and baseline pain intensity (the higher of neck pain and headache).

NS = non-significant, *p < 0.05, **p < 0.01, ***p < 0.001.

4. Discussion

This prospective study on emotional reactions to a whiplash injury demonstrated an association between a post traumatic stress reaction and development of chronic WAD. While this observation is consistent with two previous prospective studies (Drottning et al., 1995; Sterling et al., 2006), this study is by far the largest prospective study with a long-term follow up. In one earlier trial the follow-up was only four weeks (Drottning et al., 1995) and in another trial with long-term follow-up only 65 participants were enrolled (Sterling et al., 2006). The present study represents a verification of the link between psychological response to a car collision and long-lasting physical symptoms which is in demand (Williamson et al., 2007).

Even though the association between the early stress reaction and chronic WAD was statistically significant it was not sufficiently strong to predict prognosis in indi-
individuals. Thus, the acute stress response to a car accident is related to the prognosis, but it is clear that the acute stress response is not the only determining factor. Patients with a severe acute stress response have an increased risk of developing chronicity after whiplash injuries, but still it is unknown whether “correction” of the stress response would improve outcome. Furthermore, this study did not attempt to investigate why the stress response differed between subjects. In theory, a stronger stress response could be related to accidents involving more severe impact but also to greater fear of injury. We observed that heavier car damage was associated with higher IES-scores, which can indicate that the link between post traumatic stress and chronic pain is a car collision with higher impact. Another possible link is that a high expectancy of long-lasting pain after whiplash seems to relate to higher incidence of chronic WAD (Ferrari et al., 2002), and severe stress might be caused by fear of lasting pain itself. It may be too that a marked post traumatic stress is an element of the “cultural factor” playing a part in the development of chronic sequelae following whiplash injury.

Stratification by baseline pain indicated that the IES-score was more strongly related to outcome in participants with lower baseline pain scores. Thus, baseline pain had an effect-modifying influence on IES, which was partly driven by a relatively high risk of chronic WAD in participants with severe baseline pain also without signs of a post traumatic stress reaction. Stratification also showed that the IES was somewhat stronger associated to outcome in men than in women.

The observed frequency of severe post traumatic stress response was lower than previously reported rates of post traumatic stress disorders (PTSD) in traffic accident victims (Blanchard et al., 1996; Drottning et al., 1995; Jaspers, 1998). This might be due to the fact that this cohort consisted solely of relatively mildly injured traffic victims, and that it was not primarily included with the purpose of studying psychological distress.
We believe that this reduced the risk of selection bias. The present study population was recruited consecutively from emergency units and general practitioners, and we consider it to be representative for persons seeking acute care after whiplash-type accidents. Therefore it is of importance that a considerable number of the participants showed signs of a clinically important acute stress response. Moreover, this population was included within a mean of 5 days after the accident, indicating that an undesirable stress reaction can be depicted early after a whiplash injury. Earlier results from longitudinal observations were reported from a population included from emergency units, a number of primary care practices and advertisements within a mean of about 3 weeks after the accident (Sterling et al., 2003).

Participants in the present study were randomized to conservative treatment or information. Since the scope of this study was to illuminate whether post traumatic stress was generally a predictor of poor recovery it was not analyzed whether the association between early post traumatic stress and recovery was related to early treatment. Hence it is undisclosed whether intervention interferes with post-traumatic stress. This will be evaluated in a future sub-group analysis.

A classification system for WAD, which includes the acute stress response in addition to physical risk factors, has previously been suggested (Sterling, 2004). This trial confirmed the importance of considering the acute stress response as one of more risk factors. It also pointed towards it being questionable whether diagnostic categories should be defined by merely adding predictors of the prognosis as in some classification systems (Spitzer et al., 1995; Sterling, 2004). Instead of looking for general risk factors for developing chronic WAD by considering the number of present risk factors, it may be relevant to look for specific risk factors in subgroups of patients. This way the handling of a patient can be tailored towards the individual “main risk factor”.

5. Conclusion

The acute stress response was significantly related to the development of chronic WAD, particularly clear in those with low baseline pain. However, the association was not sufficiently strong to predict chronicity on an individual level.

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