Concise Report

Does physical trauma lead to an increase in the risk of new onset widespread pain?

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Abstract

Background: Patients with widespread musculoskeletal pain often attribute the onset of their symptoms to a physically traumatic event such as a motor vehicle crash. However there have been few epidemiological studies conducted to examine this hypothesis.

Objective: Firstly to determine, the rate of new onset of widespread pain six months after a motor vehicle crash. Secondly to determine, whether this is in excess of the new onset rate in the absence of such a physically traumatic event.

Methods: A prospective cohort study of persons registered with an insurance company who had, or had not, experienced a motor vehicle crash. All participants were sent a questionnaire to assess pre-crash (or for the non-crash group, prior) psychosocial factors and widespread pain. Participants reporting pre-crash (prior) widespread pain were excluded from follow-up. At six months, participants were sent a follow-up questionnaire to ascertain new prevalent widespread pain.

Results: 597 (51%) of those invited to participate returned a baseline questionnaire (465 crash and 132 non-crash). Amongst the cohort who had experienced a crash, the new onset rate of widespread pain six months later was low – 8%. In comparison to the non-crash group there was an elevated risk (RR 1.9 (95% CI 0.8, 4.8 adjusted for age and gender)) which was however attenuated after adjustment for pre-crash (prior) psychological distress and somatic symptoms (RR 1.4 (95% CI 0.5, 3.2)).

Conclusions: Our findings do not provide strong support for a motor vehicle crash (as an example of a physically traumatic event) having a large impact on the new onset of widespread pain. Any observed relationship may, in part, be explained by psychological distress.
Introduction

Widespread pain (WP) is a common condition affecting approximately 16% of the population[1]. Almost 80% of those with WP experience chronic symptoms[2], representing a considerable burden, both to individuals and their families, as well as to health care resources. A number of adverse psychosocial factors may predict the onset of WP, in particular depression, somatic symptom reporting and specific health behaviours[3-5]. In addition patients often attribute the onset of their symptoms to a precipitating physical trauma[6,7], although there have been very few studies conducted to investigate this potential aetiological factor.

A few case-control and cross-sectional studies have suggested there may be an association between trauma and WP[7,8]. For example a case-control study comparing 136 fibromyalgia hospital out-patients with 152 matched controls found that 39% of fibromyalgia patients reported physical trauma in the previous six months, compared to 24% of controls[8]. However given that such studies measure exposure retrospectively they are potentially subject to recall bias – emphasising the importance of, where possible, validating events. One study has prospectively assessed the association between trauma and fibromyalgia syndrome[9], of which widespread pain is the primary symptom. The authors concluded that fibromyalgia was 13 times more frequent following neck injury than following lower extremity injury. However, many of the tender point sites used to diagnose fibromyalgia are located in the neck area and, therefore, it may be expected that neck injured patients would report more pain and tenderness in these areas.

The aim of the current study was therefore to determine for the first time, prospectively, the rate of new onset widespread pain after a motor vehicle crash, and whether this is in excess of the new onset rate in the absence of such a physically traumatic event.

Methods

The design was a prospective cohort study. Subjects were recruited through a national UK motor insurance company. Individuals aged 17-70yrs, who contacted the company to report a motor vehicle accident, were eligible for participation in the crash cohort. Those reporting other claims, for example theft or damage whilst the vehicle was unoccupied, were eligible for participation in the non crash cohort. Additionally all participants had to be making a claim less than 7 days after their crash/ non-crash. The definition of
crash was that provided by participants i.e. reporting of a motor vehicle accident, in addition to verification by the insurance company operative.

Subjects were asked to participate in the study at the time of telephoning the insurance company. Those who agreed to participate were then sent a questionnaire by post. General health and well-being in the month pre-crash (or prior month for the non-crash group) was assessed using the Health Status Short-Form 8 (SF-8) questionnaire. The Somatic Symptoms Checklist (SSC) measured the number of certain bodily symptoms experienced ever pre-crash (or ever for the non-crash group). General psychological distress was measured during the pre-crash (or prior) month using the General Health Questionnaire (GHQ). Participants' pain status was determined using four full-view body manikins on which they were asked to shade the location of any pain either in the past month (non-crash group) or in the month before the crash (crash group). Individuals with widespread pain (using the American College of Rheumatology definition as part of the criteria for fibromyalgia were excluded.

Six months subsequently, participants were re-contacted by postal questionnaire and asked the same questions on pain. Subjects with widespread pain at follow up (using the body manikins) were labelled as “new prevalent widespread pain”.

Analysis

Poisson regression was employed to examine which factors measured at baseline predicted the presence of widespread pain at six months. Results are expressed as relative risks (RR) with 95% confidence intervals (95%CI).

Results

We invited 1178 individuals to participate in the study, of whom 896 consented to receive a postal questionnaire. Questionnaires were returned by 597 (51%) of those invited to participate. They comprised 465 individuals in the crash cohort, mean age 42.0, 59% female, and 132 individuals in the non-crash cohort, mean age 42.0, 60% female. The prevalence of WP in the month prior to the incident was 4% (n=20) and 6% (n=8) in the crash and non-crash cohorts, respectively. These individuals were excluded from follow-up, and thus 569 were eligible for follow up.

In all 517 (87%) of these individuals completed the six-month follow up questionnaire, of whom 490 provided complete pain data: 376 individuals in
the crash cohort (81%) and 114 individuals in the non-crash cohort (86%). The prevalence of new WP was 8% (n=31) in the crash cohort and 4% (n=5) in the non-crash cohort (RR 1.9 95%CI 0.8, 4.8 adjusted for age and gender). Further adjustment for GHQ score (high vs. low distress) and somatic symptom reporting (0 vs. 1 or more symptoms) attenuated this risk (RR 1.4 95%CI 0.5, 3.2) (figure 1). Adjustment for SF-8 mental and physical scores did not further alter the risk of new WP.

Although this study was not designed to have sufficient power to examine risk separately in men and women, the data suggests that there may be differential effects by gender, with a slightly higher risk in females (RR 1.5 95%CI 0.5, 5.1) compared to males (RR 1.1 95%CI 0.3, 4.3) (table 1).

<table>
<thead>
<tr>
<th>New onset widespread pain n (%)</th>
<th>Adjusted RR 95%CI*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Crash group</td>
<td>Non-crash group</td>
</tr>
<tr>
<td>All subjects 31 (8.2) 5 (4.4)</td>
<td>1.4 (0.6, 3.3)</td>
</tr>
<tr>
<td>Male 12 (6.9) 2 (4.7)</td>
<td>1.1 (0.3, 4.1)</td>
</tr>
<tr>
<td>Female 19 (9.5) 3 (4.2)</td>
<td>1.5 (0.5, 5.1)</td>
</tr>
</tbody>
</table>

* Adjusted for age, psychological distress and somatic symptom reporting

Discussion

This is the first study to examine prospectively the rate of onset of widespread pain after physical trauma, namely a motor vehicle crash. It suggests that the rate of onset after a motor vehicle crash is low and at most there is a modest increase in risk, particularly after adjusting for levels of psychological distress.

There are a number of caveats that need to be considered to put this study into context. Firstly, although the study was large, the number of new onset pain episodes was small. This is interesting as an observation in itself but does limit the power of the study. Secondly, the initial response rate was low; of those invited to participate just over 50% returned a questionnaire, raising questions about the external validity of the study. The available data on those who did not return a questionnaire is limited, however there were no significant differences between those who returned a completed questionnaire and those who were invited to participate but refused or did not reply, in either
age (Z m-w: p=0.08) or gender (chi²: p=0.08). It is also unlikely that those subjects who had experienced a crash but did not take part would be at a greater increased risk of developing WP, thus causing us to underestimate the risk. Thirdly the precise role of psychological distress is difficult to determine in this study. It could be hypothesised that psychological distress is a pathway variable, that trauma leads to distress and then WP, and therefore should not be adjusted for. However, the measure of psychological distress was taken pre-trauma. Therefore psychological distress could be considered a confounding factor, i.e. distress leads to an increased likelihood of experiencing a motor vehicle crash, in addition to being associated with the onset of pain. A recent study by Lagarde et al (2004)¹⁵ found that stressful life events were associated with a subsequent increase in serious motor vehicle accidents, lending support to the hypothesis that psychological distress may be a confounding factor. Adjusting for psychological distress does attenuate the relationship between trauma and WP, but it is unknown to what extent the reporting of pre-crash psychological distress was influenced by the post-crash psychological distress experienced. Current work has suggested that prior distress, in particular depression, leads to a poor adjustment to stressful events.¹⁶ Additionally there are interactions between affective states, stressors and pain.¹⁷ It will be important for future studies to explore not only the effect of trauma but modifiers of its effect.

This study does have a number of strengths. The design is prospective, and therefore we have been able to determine the new onset of WP in persons who were initially WP free. An inherent problem of prospective studies is loss to follow-up, however loss in this study is minimal. Further it may be expected that any such loss to follow up may result in an over estimation of the new onset of widespread pain. The setting was population based, therefore overcoming the potential issue of selection bias associated with samples selected from clinic populations.

The current study has suggested that the new onset of widespread pain after a motor vehicle crash is low, and only marginally above that experienced by a non-crash group. The rate of new onset over a six-month period in the non-crash group (4%) is very similar to the rate reported (5%) over a twelve-month period in a study of 1658 adults in the general population of one area of North West England.¹⁸ This provides additional support for the study’s external validity.

Previous studies have reported that trauma is associated with WP, however methodological limitations, such as recall bias and inability to measure the onset of pain, have restricted the interpretation of these findings. We have added to the literature by demonstrating a weak relationship
between trauma and the onset of WP. Studies have suggested that prior trauma is reported by 23%[7] and 39%[8] of fibromyalgia patients. These studies, however, have been conducted in specialist clinics amongst a selected population of fibromyalgia patients. Nevertheless the latter study although showing an association between fibromyalgia and trauma found that in both case and control subjects the report of a motor vehicle crash was uncommon. It is possible that an association does exist but with other and more severe forms of trauma.

In summary, the results of this preliminary study do not provide strong support for physical trauma (at least as exemplified by a motor vehicle crash) having a large impact on the new onset of WP. It suggests a more modest estimate of the effect of trauma than those previously reported.

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The study received ethical approval from The University of Manchester Committee for the Ethics of Research on Human Beings.

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Figure 1: Risk of new onset WP at six months

Accident versus non-accident cohort

<table>
<thead>
<tr>
<th>Reference</th>
<th>Adjusted for age &amp; gender</th>
<th>Adjusted for age, gender, psychological distress &amp; somatic symptoms</th>
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