Widespread Pain Following Whiplash-Associated Disorders: Incidence, Course, and Risk Factors

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ABSTRACT. ***Objective.*** To investigate the incidence and course of widespread pain (WP) subsequent to localized pain in subjects with whiplash-associated disorders (WAD); and to investigate the influence of depressive symptoms, neck pain intensity, number of whiplash-associated symptoms, and number of painful body areas on such conditions.

Methods. From a large prospective cohort of injury claimants who reported WAD after motor vehicle collision (MVC; n = 7462), we identified a subgroup with only localized head/neck/back pain, and who responded to one or more followup questionnaires mailed at 6 weeks and 4, 6, and 12 months after the MVC (n = 266). Pain drawings were distributed at the followup, and we defined WP as having 9 or more painful areas, including posterior neck, at any of these occasions. Depressive symptoms were assessed with the Center for Epidemiological Studies Depression Scale and pain intensity with a visual analog scale (VAS).

Results. The cumulative incidence of WP was 21%, and it occurred early after the injury. Continuous WP over the 12 months was rare. The odds for developing WP were greater in those with depressive symptoms (OR 3.2, 95% CI 1.6–6.3), VAS pain intensity 55–100 (OR 3.2, 95% CI 1.3–8.0), reporting ≥ 3 pain-associated symptoms (OR 1.9, 95% CI 0.9–3.8), and those reporting 4 or 5 painful body areas (OR 2.6, 95% CI 1.3–5.4).

Conclusion. WP occurred early in the course. Even though the cumulative incidence was 21%, continuous WP was rare. Subjects with WAD who report early depressive symptoms and more severe neck injury symptoms are at risk of developing WP after MVC. (First Release Dec 1 2006; J Rheumatol 2007;34:193–200)

Key Indexing Terms:
WHIPLASH WIDESPREAD PAIN NECK PAIN DEPRESSIVE SYMPTOMS

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Whiplash injury is a common sequelae after motor vehicle collision (MVC)[1-3], but may also occur after other types of traumatic events, such as falls and bicycle or diving accidents. Whiplash is defined by the Quebec Task Force as "an acceleration/deceleration mechanism of energy transfer to the neck"[1]. This force may result in soft tissue injury to the neck, which in turn may lead to a variety of symptoms. Since whiplash is a description of a mechanism of injury, rather than a diagnosis, the Quebec Task Force coined the term whiplash-associated disorders (WAD), which are the clinical signs and symptoms associated with the injury. The main symptom is neck pain, but other symptoms such as limited range of cervical motion, headache, dizziness, or numbness in arms might also be present.

Widespread pain. Chronic or persistent widespread pain (WP) is often operationally defined according to the American College of Rheumatology (ACR) 1990 criteria[4]. Criteria include: duration of pain for at least 3 months, pain in the axial skeleton, in addition to pain above and below waist and left and right side of the body. This definition has been commonly used in population surveys by identifying pain areas from pain drawings[5-8]. Studies on prevalence of chronic WP using the ACR criteria indicate a prevalence of 3–13% depending on country and study population[6]. The natural course of WP is

complex and it is likely to persist or recur to a large extent, but may in some cases resolve to no pain[5,8].

The relationship between a physical trauma and later onset of persistent WP is not well established. Some have suggested that WAD may lead to WP or fibromyalgia[9-11]. However, the data are from cross-sectional studies so temporal associations are unclear. Despite numerous experimental studies and case studies suggesting lower pain threshold as a result of an alteration in the pain processing mechanism within the central nervous system[12,13], or that fear and anxiety are important factors for pain threshold[14,15], a clear etiology has not been presented. In a recent prospective study of uninjured and injured car occupants, the authors report a frequency of 7.8% of new onset of WP in subjects at 12 months post-MVC[16]. That study focused on the effect of a traumatic event and not of the presence of WAD per se. When investigating the temporal relation between WAD and subsequent onset of WP, one must clearly exclude those who report WP at baseline. To our knowledge there are no prospective studies where such a relation has been investigated. There is also little knowledge about how common WP is after WAD in a clearly defined population of localized pain.

One known risk factor for poor prognosis of recovery in WAD is high initial intensity of neck pain[2,17]. Another suggested risk factor for poor prognosis is depressive symptoms[18]. Whether neck pain intensity and depressive symptoms are also risk factors for subsequent WP in subjects reporting WAD has to our knowledge not been studied. In their study, Wynne-Jones, *et al* suggest that apart from having any physical injury, precollision health-seeking behavior and precollision somatization were associated with new onset of WP[16]. Large prospective studies investigating exposures other than MVC found that psychological factors[19], mechanical work-related factors (e.g., repetitive movements or physically heavy workload), and psychosocial work-related factors[20,21] are associated with the development of WP. Thus the current literature suggests that both physical and psychosocial factors might be involved in the development of WP.

We have not identified any studies investigating the incidence and natural course of WP after WAD. Nor have we found any studies focused on investigating risk factors for the onset of subsequent WP in such populations. Therefore the objectives of this study are: (1) to investigate the incidence and course of WP in subjects with WAD with localized pain after MVC; and (2) to investigate factors associated with the onset of subsequent WP, including depressive mood, neck pain intensity, number of pain-associated symptoms, and number of painful body areas after the injury.

MATERIALS AND METHODS

Study population. The study population was a cohort of traffic injury claimants to Saskatchewan Government Insurance (SGI) in the Canadian province of Saskatchewan[2]. Data collection was between July 1, 1994, and December 31, 1995. All car occupants who filed an insurance claim or were treated for a traffic injury and reported neck pain after the MVC were eligible.

Data collection. Information with personal identifiers removed was obtained from the insurance application form, a questionnaire that included questions about the collision, previous health including general health, neck pain and headache before MVC, and signs and symptoms after the MVC. The form also included a pain drawing, asking claimants to shade in areas of the body where they felt any present pain since injury. This type of pain drawing has been used in pain research over the past decades and has been shown to be a valid assessment tool[22-25]. All those who gave informed consent to participate in a followup study were asked to respond to an additional questionnaire as soon as possible after the initial one. Participants received mailed questionnaires at 6 weeks and 4, 8, and 12 months after the MVC or until they indicated a decision to withdraw from the study. Pain drawings were included in each followup questionnaire.

Inclusion criteria. For the purpose of this study, we included those who reported localized injury-related head/neck/back pain since injury, at entry into the study, responded to the initial questionnaire within 6 weeks of the injury, consented to participate in the followup, and completed at least one of the followup questionnaires. Localized injury-related head/neck/back pain was operationally defined as (1) having answered "yes" to both of the following questions: "Did the accident cause neck or shoulder pain?" and "Have you felt neck or shoulder pain or have you felt reduced or painful neck movement since the accident?"; and (2) on the initial questionnaire, shaded the following body areas on a pain drawing: posterior neck pain with or without posterior shoulder pain, head pain, thoracic pain, or low back pain. The pain drawings were coded according to 45 topographic areas as suggested by Margolis, *et al* (Figure 1)[22,23]. We arbitrarily restricted the criteria for the included subject to have a maximum of 5 of 11 areas, in order to exclude widespread pain at entry into the study. These 11 areas correspond to areas 1, 2, 23–27, and 34–37 on the manikin template (Figure 1). Subjects who had pain in other areas of the body at baseline were excluded from the study.

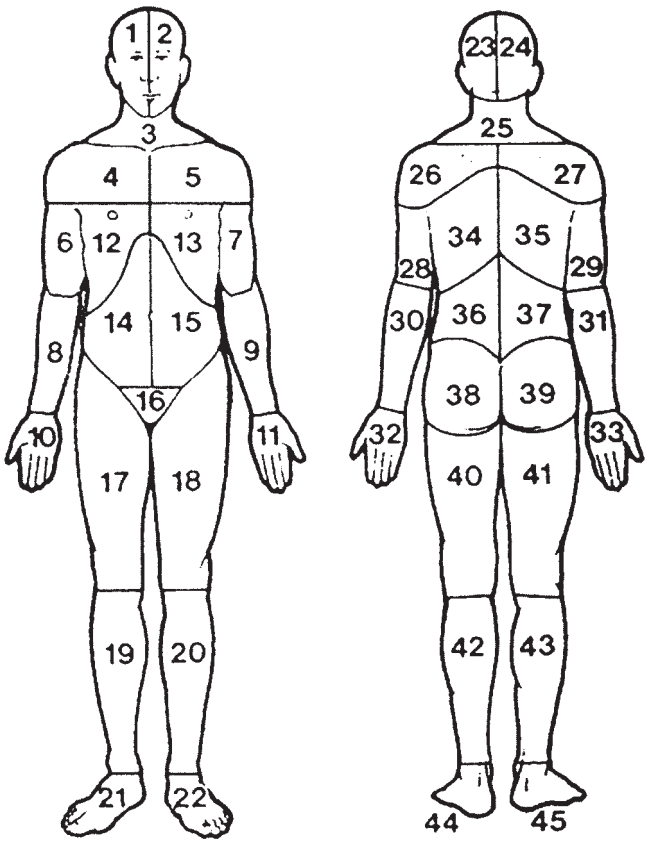

Figure 1. The manikin template.

The original WAD cohort included 7462 individuals, of whom 7419 had pain at the time of making the claim and completed the initial pain drawing. Of these, 845 (11%) had localized pain as defined above, and were thus eligible for the study. One hundred nine did not respond to the initial questionnaire within 6 weeks and 470 did not respond to any of the followups. Thus 266 subjects were enrolled into the study.

The study was approved by the University of Saskatchewan's Advisory Committee on Ethics in Human Experimentation and by the Regional Committee on Ethics at Karolinska Institutet, Stockholm.

Outcome: Widespread pain. WP at followup was operationally defined as having shaded 9 or more of the 45 body areas, including posterior neck, at any of the 4 followups.

Investigated factors. Depressive symptomatology during the past week was assessed on the additional baseline questionnaire, using the Centre for Epidemiological Studies Depression Scale (CES-D). It is a valid and reliable instrument, and has a possible range of scores from 0 to 60, with a cutoff score of 16 to identify depressive mood[26-30]. Other factors were neck pain intensity at baseline, number of self-reported symptoms at baseline, and number of painful body areas at baseline. Pain intensity was assessed with a 100 mm visual analog scale (VAS), which is a valid and reliable pain instrument[31,32]. We categorized the VAS scores into mild pain (0–30 mm), moderate pain (31–54 mm), and severe pain (54–100 mm), according to Collins, *et al*[33]. Symptoms of reduced/painful neck movement, jaw movement, numbness, tingling or pain in arms/hands or in legs/feet, dizziness or unsteadiness, nausea, vomiting, difficulty swallowing, ringing in the ears, memory problems, concentration problems, and vision problems were assessed by self-report on the initial questionnaire. We dichotomized the 2 variables "number of painful body areas at baseline" and "number of symptoms at baseline" using the median of the distribution.

Analysis. We present the natural course of WP using frequencies and proportions. To investigate factors associated with the onset of WP, we used logistic regression. Odds ratios (OR) and their 95% confidence intervals (CI) were calculated for depressive mood and the injury-related factors in bivariate models[34]. Potential colinearity between each of the factors was assessed with Spearman rank correlation coefficient (r^s). If r^s was ≥ 0.8, colinearity was considered present. We then entered all factors simultaneously into a multivariable model. Finally, we adjusted for age, sex, and prior health status by entering them into the model one at a time, to assess whether they changed the regression coefficients by 10% or more, and if so, they remained in the model[35]. Health status before collision was measured using a modification of the Medical Outcome Study Short-Form 36 (SF-36) "general health" subscale question[36]. The modification consisted of a change aiming to record health during the month prior to collision and was phrased, "How was your health the month before the accident?", but offering the same response options as in the original SF-36 question. We also considered the frequency of precollision neck pain and headache.

As for the definition of WP, we undertook a sensitivity analysis by recalculating the crude OR for the association between depressive mood and the onset of WP using 6 or more, 8 or more, and 10 or more painful body areas at each of the followup periods. Since time between injury and response to the questionnaires varied between subjects, and was likely to have an influence on the recovery process, we analyzed its potential influence by comparing the number of days from the collision to date of response to each of the questionnaires, between those that did and those that did not develop WP. We also compared the response rate during the followup mailings between the 2 groups.

To investigate attrition, we used multivariable logistic regression to assess associations between baseline subject characteristics and participation. Complete baseline data were available on all 845 eligible subjects. Potential explanatory factors were age, sex, education level, combined family income, baseline neck pain intensity (by VAS), number of symptoms, and number of painful body areas. All analyses were carried out using SPSS[37].

RESULTS

The median time between the collision and response to the initial and additional baseline questionnaires was 10 days (25th percentiles, 5 and 17 days) and 13 days (25th percentiles, 7 and 22 days), respectively. There was no difference in the number of days since collision and response to the questionnaires between those who developed WP and those who did not. A total of 167 (63%) of the 266 participants responded to all 4 followups, 49 (18%) responded to 3 followups, 20 (8%) to 2 followups, and 30 (11%) to 1 followup. There was no difference in followup rate between the subjects who developed WP and those who did not. Of the 167 subjects who responded to all followups, only 4 (3 women and 1 man) fulfilled the criteria for WP at all followups.

Incidence and course. Baseline characteristics of the study sample and those subjects who had developed WP at any of the followups are presented in Table 1. The cumulative incidence of WP over the 4 followup periods was 21%. Onset was more frequent among women than men, among those reporting poor health before the injury, a greater number of painful body areas at baseline, greater initial neck pain intensity, a greater number of whiplash-associated disorders, and more depressive symptomatology.

In individuals who developed WP, over half (63%) experienced onset before the first followup point (mean 52 days, range 36–133) and another 20% by the second followup (mean 117 days, range 77–179).

Figure 2 illustrates the variation of the course of WP from becoming a case until the last followup. In most instances (64%), WP was followed by an improvement, and the majority of these also remained improved over the followup period. Of the 36 subjects who reported improvement at any followup after becoming a case, 24 no longer fulfilled the case definition at the last followup, and of these, 7 were pain-free (data not shown). However, 20 cases still had WP and did not improve at any time, and indeed some got worse.

The prevalence of subjects classified as cases of WP was 50–60% at different followups, except for the 8-month followup, where 36% fulfilled the case definition (Table 2). The response rates were lower at the last 2 followups, and it is therefore difficult to determine if the proportion of WP differed over time.

Risk factors. The crude and adjusted OR for the association between the investigated factors and onset of WP are presented in Table 3. In the unadjusted analysis, depressive mood, higher baseline neck pain intensity, reporting 3 or more symptoms, and 4 or 5 painful body areas at baseline were all associated with the onset of WP. Adjustment for all factors changed the estimates only slightly. Age, gender, or prior health status did not change any of our estimates, and were not included in the final regression model. There was no colinearity between the risk factors.

The sensitivity analysis of the definition of WP did not greatly change our estimates. Depressive mood was still

Table 1. Baseline characteristics of the study population, stratified on those who developed widespread pain (WP) at any followup and those who did not.

Variable	Total	WP, n (%)	No WP, n (%)
All	266	56 (21)	210 (79)
Demographic and socioeconomic factors			
Sex			
Male	109	18 (17)	91 (83)
Female	157	38 (24)	119 (76)
Age, yrs			
≥ 40	100	21 (21)	79 (79)
30–39	83	18 (22)	65 (78)
18–29	83	17 (20)	66 (80)
Education			
Less than high school	66	13 (5)	53 (95)
High school	64	12 (5)	52 (95)
Post-secondary or university	136	31 (23)	105 (77)
Prior health-related factors			
General health the month before MVC			
Excellent	113	18 (16)	95 (84)
Very good	85	20 (24)	65 (76)
Good, fair, or poor	68	18 (26)	50 (74)
Neck pain before MVC			
Never or almost never	194	33 (17)	161 (83)
Every month	62	22 (35)	40 (65)
Every week or every day	10	1 (10)	9 (90)
Headache before MVC			
Never or almost never	157	28 (18)	129 (82)
Every month	97	24 (25)	73 (75)
Every week or every day	12	4 (33)	8 (67)
Symptoms after the MVC			
No. painful body areas at baseline			
5	108	32 (30)	76 (70)
4	43	11 (26)	32 (74)
3	87	11 (13)	76 (87)
2	17	2 (12)	15 (88)
1	11	0	11 (100)
Neck pain VAS			
0–30	79	7 (9)	72 (91)
31–54	80	17 (21)	63 (79)
55–100	105	32 (30)	73 (70)
Symptoms* other than neck pain			
0–1	123	16 (13)	107 (87)
2–3	98	22 (22)	76 (78)
4 or more	43	18 (42)	25 (58)
CES-D			
Below 16	190	25 (13)	165 (87)
≥ 16 (depressive mode)	76	31 (41)	45 (59)

* Number of symptoms includes any of the following 12: reduced/painful neck movement or jaw movement, numbness, tingling or pain in arms or hands or in the legs or feet, dizziness or unsteadiness, nausea, vomiting, difficulty swallowing, ringing in the ears, memory problems, concentration problems, vision problems (missing value in 2 cases). MVC: motor vehicle collision. VAS: visual analog scale 0–100 mm (missing value in 2 cases). CES-D: Center for Epidemiological Studies — Depression Scale.

strongly associated with onset of WP when we used a cutoff of 6 painful areas (OR 3.0, 95% CI 1.7–5.2). When we used 8 and 10 body areas in pain as our outcome criterion, the OR for depressive symptoms increased as expected (OR 3.7, 95% CI 2.0–6.5, and OR 4.7, 95% CI 2.3–9.6, respectively).

The result of the attrition analysis is presented in Table 4. The participants were more likely to be female, have higher education level, and have a combined family income of $20,001–$60,000 Cdn/year. Age was of only minor importance and there was no association between the baseline neck pain intensity, number of pain areas or associated symptoms, and participation.

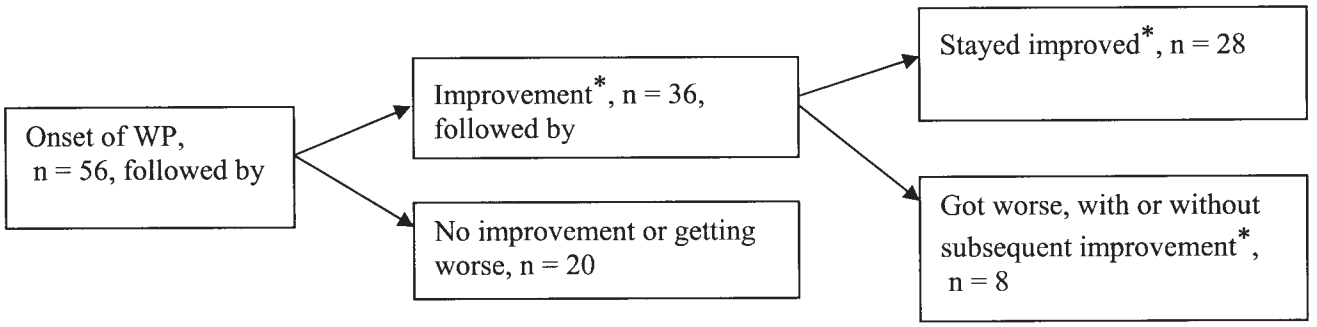

Figure 2. Course of pain extension in subjects who developed widespread pain (WP) ≥ 9 body painful areas, including posterior neck, at one or more of 4 followups (6 weeks, 4, 8, and 12 months). *Fewer painful areas than when became a case.

Table 2. Distribution of pain at baseline and at followups in subjects who fulfilled the criteria of widespread pain (WP) at any followup (n = 56).

Body Area(s) in Pain	Baseline, n = 56	6-Week Followup (%)	4-Month Followup (%)	8-Month Followup (%)	12-Month Followup (%)
No. of cases responding to followups	—	53	50	47	45
No. of subjects with WP	—	35 (66)	28 (56)	17 (36)	23 (51)
(%) Painful body area					
Posterior neck pain	56 (100)	35 (100)	28 (100)	17 (100)	23 (100)
Anterior neck pain	—	16 (46)	14 (50)	11 (65)	13 (57)
Head pain	38 (68)	30 (86)	22 (79)	15 (88)	22 (96)
Posterior shoulder pain	40 (71)	34 (97)	27 (96)	17 (100)	23 (100)
Anterior shoulder pain	—	16 (46)	13 (46)	13 (76)	16 (70)
Upper extremity pain	—	9 (26)	11 (39)	5 (29)	8 (35)
Chest pain	—	2 (6)	1 (4)	2 (12)	5 (22)
Thoracic pain	29 (52)	28 (80)	24 (86)	16 (94)	20 (87)
Lumbar pain	13 (23)	26 (74)	20 (71)	12 (71)	17 (74)
Buttock pain	—	0	19 (68)	9 (53)	10 (43)
Lower extremity pain	—	3 (9)	9 (32)	3 (18)	3 (13)
Abdominal pain	—	0	1 (4)	3 (18)	2 (9)
Groin pain	—	0	1 (4)	0	1 (4)

DISCUSSION

To our knowledge, this is the first original study to describe the incidence and course of WP after WAD. It is also the first time that the associations between initial depressive symptoms, neck pain intensity, number of symptoms, and onset of such condition have been assessed in a population with WAD.

Surprisingly, only 11% of participants in the original WAD cohort had localized pain at baseline according to our definition. The others either had different pain patterns or already had more widespread pain when making their insurance claim. Whether these painful areas were due to other injuries, such as bruises or fractures, has not been investigated, but associated injuries seemed to be common and should be considered in future studies of widespread pain after WAD. Of the 266 subjects included in the study, 21% developed WP at some point during the course of the one-year followup. Over half the subjects with WP improved, of which some experienced subsequent deterioration, suggesting that WP is a recurrent condition in some cases. We also cannot rule out the possibility that some of these subjects had occurrence of WP at some point in time before the MVC. Since only 4 subjects reported WP at all 4 followups, continuous or chronic WP after WAD is likely to be rare.

During the past few years there has been a focus on the role of psychological and social factors for the prognosis of recovery in WAD[2,38-42]. Our study is the first to assess the role of psychological and injury related factors for the development of WP after WAD. In the study by Wynne-Jones, *et al*, the research objective was slightly different from ours, in that they included subjects who had been exposed to a MVC independent of subsequent injury or extent of injury[16]. They found associations between precollision factors such as health-seeking behavior and precollision somatization and new onset of WP, but as well, having reported a physical injury after the collision was associated with WP. Results from a general population sample indicated that somatic symptoms and illness behavior are risk factors for developing WP[19]. Further, results from various cross-sectional studies show associations between psychological and/or social factors and WP[7,43-45]. Our finding that depressive mood as well as injury-related factors in this setting of WAD are associated with the onset of WP is therefore not surprising.

Table 3. Logistic regression analysis of the associations between baseline psychological and injury-related factors and the onset of widespread pain (≥ 9 painful areas including posterior neck, reported at any of 4 followups (n = 266).

Risk Factors	Crude OR (95% CI)	Adjusted OR* (95% CI)
Depressive mood		
CES-D < 16	Referent	Referent
CES-D ≥ 16	4.6 (2.4–8.5)	3.2 (1.6–6.3)
Neck pain intensity		
VAS 0–30	Referent	Referent
VAS 31–54	2.8 (1.1–7.1)	2.4 (0.9–6.5)
VAS 55–100	4.5 (1.9–10.9)	3.2 (1.3–8.0)
Symptoms†		
0–2	Referent	Referent
3 or more	2.6 (1.4–5.0)	1.9 (0.9–3.8)
No. painful body areas		
1–3	Referent	Referent
4-5	3.1 (1.6–6.1)	2.6 (1.3–5.4)

* Adjusted for all other factors in the regression model (no confounding by age, gender, or prior health status). † Symptoms other than pain (1 or more of the following: reduced/painful neck movement or jaw movement, numbness, tingling or pain in arms/hands or in the legs/feet, dizziness or unsteadiness, nausea, vomiting, difficulty swallowing, ringing in the ears, memory problems, concentration problems, vision problems). CES-D: Center for Epidemiological Studies-Depression Scale, VAS: visual analog scale.

Table 4. Logistic regression analysis of the association between subject characteristics at baseline and being a responder to the initial questionnaire within 42 days and to at least 1 of the followup questionnaires (n = 845).

Variables	Adjusted OR* (95% CI)
Sex	
Male	Referent
Female	1.6 (1.1–2.1)
Age	
18–29	Referent
30–39	1.2 (0.8–1.9)
40 and over	0.9 (0.3–1.3)
Education level	
Less than high school	Referent
High school	1.0 (0.6–1.5)
Post-secondary or university	1.6 (1.1–2.4)
Combined family income ($ Cdn/year)	
0–20,000	Referent
20,001–40,000	2.1 (1.4–3.1)
40,001–60,000	2.4 (1.5–3.9)
Above 60,000	1.4 (0.8–2.4)
Neck pain intensity (VAS)	
0–30	Referent
31–54	1.0 (0.7–1.5)
55–100	0.8 (0.6–1.2)
No. painful body areas (1–5)	1.0 (0.9–1.1)
Symptoms (1–9)†	1.0 (0.9–1.2)

* Adjusted for all other factors in the model. † Symptoms other than pain (1 or more of the following: reduced/painful neck movement or jaw movement, numbness, tingling or pain in arms/hands or in the legs/feet, dizziness or unsteadiness, nausea, vomiting, difficulty swallowing, ringing in the ears, memory problems, concentration problems, vision problems). VAS: visual analog scale.

We cannot determine to what extent the depressive mood was a preinjury state or had occurred as a consequence of the collision. It is unlikely, though, that the depressive symptoms reported in our study are due to long-lasting pain, since the CES-D was completed early after the onset of injury (for 56% of the participants, depressive mood was assessed within 14 days, and for 73% within 21 days after the MVC). In a recent study, depressive symptoms after WAD were also shown to be present early after the injury[30].

One of the strengths of our study is the prospective design. Another is that this was a well defined cohort, including only those who had localized pain and no other pain sites at baseline. A third strength is the use of validated questionnaires to measure factors associated with the onset of WP. Both the CES-D and VAS are frequently used in pain populations, as is the pain drawing. The information about symptoms was collected by a simple symptom score list (answered yes or no), which is also commonly used in studies of WAD[40,46]. Although the selection criteria of including only those who responded to at least one of the followup mailings may have had an influence on external validity, the internal validity within the study group was high because of low dropout rate (over 80% responded to at least 3 followups).

A limitation of our study might be our definition of localized head/neck/back pain at baseline (maximum of 5 painful body areas including posterior neck with or without pain areas including other parts of the spine) and our case definition of WP (≥ 9 pain areas including posterior neck pain). We determined these definitions before the analysis based on consultations with experts. However, there are other possible case definitions, for example, definitions based on recommendations from the ACR, which require pain to be present in contralateral body quadrants[4,47]. Our data did not allow use of the ACR definition, since it was not possible to clearly identify the 4 body quadrants suggested by the ACR. Nor did we include duration of WP in our definition, since a question about this was not explicitly asked. One would expect the case definition used has influence on the incidence rates, and we may have overestimated the incidence in comparison with other, more conservative definitions. However, our sensitivity analysis suggests that our findings from logistic regression are robust to modifications in the case definition, and our finding that WP resolves or improves over time in most cases is in accord with other studies based on general populations with no specific pain etiology[8,48].

In addition, selective participation may be present since 69% of eligible subjects did not give their consent to participate in the followup part of the data collection, or did not respond to any of the followup mailings. Those included in the study were more likely to be female, have higher education, or have an annual family income > $20,000 Cdn. This possible

selection bias might have affected the incidence of WP. However, there were no differences in the 3 neck injury-related factors between those participating in the followup and those who did not, suggesting that there was no or low selective participation bias for the results of the multivariable logistic analysis. Nevertheless, we cannot completely rule out the possibility that there are differences in physiological or psychological factors that may confound the relations we found.

Misclassification is always a concern in epidemiological studies and may be a potential source of bias. First, there may be misclassification of the exposures. The categorization of the VAS is based on one study comparing verbal and numerical pain rating, and the optimal cutoff may be difficult to assess. We did a sensitivity analysis of the crude association between terciles of VAS and WP, but we could find no major influence on the estimates compared to the cutoffs we used for the multivariable model. There might also be misclassification of the outcome of WP. If the respondents did not mark or shade the pain drawing thoroughly enough and if this systematically varied over time, it may be an explanation why WP changed over time. An alternative explanation may be that the pain drawing is supposed to reflect the pain experienced in the past week, and it is well known that pain varies over time. Finally, unmeasured mediators or interactions may also be present. Neck pain intensity, for instance, has been shown to be affected by preinjury health status and social factors[49], which in turn may influence or be influenced by the presence of depressive mood. To explore these complex patterns, a larger study would be necessary, and it was therefore not possible for us to disentangle these relations.

In summary, only 11% of a large cohort of persons with WAD due to a MVC had localized head/neck/back pain. In this subgroup, the cumulative incidence of WP within 1 year of the injury was 21%. The state occurred early in the course of WAD. Continuous WP over the 12 months was rare. Subjects with WAD who report early depressive symptoms, high neck pain intensity, many symptoms, and greater initial spread of pain are at higher risk of developing WP after MVC. Further large studies are needed to determine the influence of preinjury and injury-related conditions to make conclusions about causal relations.

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