

The Relevance of the Leading Injury in Claiming Whiplash Patients

E Pietsch*

Consultant Orthopaedic Surgeon and Trauma Specialist, Department of Trauma and Orthopaedics, The-Expert-Witness.de, Hamburg, Germany

***Corresponding Author:** E Pietsch, Consultant Orthopaedic Surgeon and Trauma Specialist, Department of Trauma and Orthopaedics, The-Expert-Witness.de, Hamburg, Germany.

Received: June 05, 2020; **Published:** July 29, 2020

Abstract

Low velocity road traffic accidents can produce a wide range of symptoms that are summarised under the term “whiplash-associated-disorder (WAD). The most common symptoms occur to the neck and the lower back. Recovery should usually take place within weeks but can be prolonged. In a medico-legal setting of an orthopaedic clinic, recovery is not seen after 12 months. This changes when claimants sustain additional injuries. We included 382 Clients with WAD and 26 Clients with associated injuries (WADplus) in a retrospective study and compared the recovery from WAD. WADplus Clients recovered on average in 5 months from their WAD symptoms whereas WAD Clients still presented with symptoms at the time of the examination at 10 months. It appears that associated injuries can be a relevant trigger in the recovery from WAD symptoms.

Keywords: Whiplash-Associated-Disorder (WAD); Injury; Road Traffic Accidents (RTA)

Introduction

The most common symptoms in motor vehicle road traffic accidents (RTA) are neck and lower back pain. The mechanism of the injury follows an acceleration/deceleration of the spine, which is known as “whiplash injury” and can coincide with a plethora of other symptoms. While the trauma is defined as “accident without evidence of injury”, the majority of patients usually recover within weeks. However, this appears to be different in a medico-legal setting, as a debatable number of patients start to suffer from ongoing and chronic symptoms. To date, many factors have been found that are associated with the chronification of symptoms. The accident circumstances, however, with well-established crash parameters do not appear to be relevant for the recovery period [1]. This makes it difficult to achieve true prognostic factors for the likelihood of a structural damage or the length of the recovery period. Thus, a multifactorial picture of whiplash recovery with medical and non-injury related factors has been accepted [2,3]. The purpose of this retrospective study was to determine if additional injuries influence the recovery from “whiplash symptoms”.

Methods

We recruited Clients from our population of RTA victims in a medico-legal setting of an orthopaedic clinic that developed neck symptoms as a result of an acceleration/deceleration trauma together with additional injuries. These injuries had to exclude soft tissue injuries like abrasions or minor cuts. Symptoms of these additional injuries had to present themselves within a timely appropriate manner, i.e.

48 hours after trauma. All Clients were seen and examined by the same orthopaedic consultant. If necessary, further investigations were recommended, e.g. imaging or referrals to other non-orthopaedic specialists for further differentiation. A prognosis for each injury was either formed at the time of the interview and examination or after the review of additional medical records, especially the recommended referrals.

Results

In 337 Clients, 26 fulfilled the criteria of additional injuries (WADplus). They were seen 9,7 months (4 to 14 months) after the initial trauma with a final prognosis after 17,3 months (6 to 35 months). The ratio of male: female Clients was equal (13/13 male/female) with an average age of 48 years (20 to 87 years). On average, little more female than male Clients were seen (7/9 male/female) with an average age of 48 years (20 to 87 years). They sustained a severe impact (14/26) with a heterogeneous accident mechanism. All Clients presented with immediate neck pain, and 10 suffered from subsequent lower back pain.

The majority of the clients' additional injuries affected the upper limbs. At the time of impact, only 2 Clients (2/26) anticipated the events and were able to brace themselves on the steering wheel. Injuries involved the shoulder joint in 20% (5/26), elbow in 4% (1/26), wrist and hand in 12% (3/26). 16% (4/26) developed impingement symptoms of the shoulder joint, 8% (2/26) injuries to the AC joint, one sustained a fracture of the olecranon, 8% (2/26) had involvement of the scapholunate ligament, one Client injuries to the scaphoid, and 8% (2/26) an aggravation of thumb OA. One Client sustained rib fractures and one a ligament sprain of her knees. 56% (14/26) of the Clients underwent imaging on primary assessment. 32% (8/26) required further specific investigations (2 X-rays, 7 MRI scans, 1 Ultrasound).

At the time of the examination, symptoms of the additional injuries persisted in 36% of the Clients (9/26). Resolution from neck symptoms had been achieved after 5 months (1 to 13 months) and from lower back pain after 5,8 months (1 to 13 months).

32% (8/26) of the Clients recovered from their additional injury within 14 months after trauma (6 to 30 months). In those, neck symptoms had resolved within one month.

Discussion

Evidence suggests that up to 50% of acutely injured people with WAD can fail to fully recover [4] from their accident, with approximately 25% demonstrating a markedly complex clinical picture including higher levels of neck-related interference [5,6] higher levels of reported pain intensity, muscle composition changes, sensory and motor disturbances, and muscle weakness.

Some prognostic factors with consistency across studies were identified being a risk for poor recovery. These include pain and/or disability levels, restricted neck movements, cold and mechanical hyperalgesia. Socioeconomic factors include pre-injury work status, job satisfaction or social status. Also, there are a number of psychological factors that include little recovery expectations or beliefs, post-traumatic stress symptoms, higher anxiety and depression [7] and pain catastrophising.

Established crash parameters were not associated with the heterogeneity of whiplash injury recovery.

There is an influence of compensation on recovery rates with poorer outcomes if litigation is involved [8,9] or if compensation can be expected. Although studies may be biased, these findings apply to the outcome after treatment of different health issues alike [10-14]. This appears to be in contrast with other studies that conclude that, although claim settlement is the intervention of interest and neck pain at

24 months is the outcome of interest, by removing the financial incentive to over-report symptoms no effect on self-reported neck pain in a fault-based compensation scheme can be observed [15,16]. This does not imply that the Client stops suffering as soon as a verdict has been achieved. Spearing (2012)¹ also argued that there is a potential for reverse causality bias. Although it is commonly believed that claiming compensation leads to worse recovery, it is also possible that poor recovery may lead to compensation claims.

Bearing these aspects in mind, the complexity of the WAD becomes evident and suggests that there is a role for both medical and non-injury related factors [3]. All these considerations apply to those Clients that have developed chronicity in their symptoms. The orthopaedic medico-legal expert will usually see Clients with persisting WAD symptoms after RTAs that do not recover in line with the recommendations of a previous expert. In our Clinic, Clients are usually seen after 10 months following their injury.

The ratio of male to female Clients is almost equal with an average age of 42 years (20 to 83 years). The majority of the Clients suffers from neck pain (220/337) and a considerable number of both neck and lower back symptoms (117/337) mainly as a result of a rear shunt (197/337). Frontal and side crashes occurred equally frequent.

Following Carroll (2009) it can be accepted that the Clients in a medico-legal setting may represent the remaining 50% that fall under the 12 months of unresolved symptoms. In our population of a medico-legal clinic, we have identified a subgroup of Clients (WADplus) that follow a different recovery from WAD symptoms than the rest of the Clients. The accident mechanism in the WADplus group in common was as heterogeneous as in the WAD group and without preference in impact direction. The only difference was that WADplus Clients sustained at least one additional injury the symptoms of which persisted to the date of the examination. It was remarkable that, by the time of the examination, their WAD symptoms had already settled.

The time of recovery from WAD symptoms in the WADplus group was on average 5 months for neck symptoms and 5,8 months for lower back symptoms. For the medical expert, the time for recovery would appear more reasonable than ongoing symptoms beyond 12 months. The additional injuries from the WADplus Clients occurred mainly to the upper limb(s). Their injuries turned out to be significant enough to outlast the WAD symptoms. On average, their pain level was 6/10 VAS and relevant enough to determine the prognosis of the Client's recovery.

A structural damage occurred in 12% of all cases whilst the majority of "injuries" was either an aggravation of symptoms from a pre-dating condition or a minor soft tissue damage. But the impact on the Client could not be less significant. For the examiner, it appeared as if the additional injury had become dominant or "the leading injury" by suppressing the concomitant WAD symptoms.

Recent studies [17] have refuted the thesis that there are body region specific differences in sensory processing, which would explain different chronicity rates between spinal and peripheral sites. Quevedo [18] found that locally mediated inhibitory processes contribute substantially to interactions among afferent inputs from noxious stimuli. It helped to understand that there is a balance between inhibitory processes and facilitatory interactions, which shape the processing of afferent nociceptive information and the subsequent perceptual experience. But there are also central modulators of pain perception, i.e. the periaqueductal gray [19]. The latter one consists of two major descending pathways that involve the rostral ventromedial medulla and the locus coeruleus. They propagate by norepinephrine with an antinociceptive effect in the dorsal horn [20,21] and serotonin, which is considered the key endogenous modulator of pain, constituting a primary target for supraspinal opioid analgesia [22]. The periaqueductal gray can stimulate inhibitory signals to c-fiber pain afferents [23]. Specific serotonin receptors have been implicated for their antinociceptive effects in the PAG, including 5-HT [24].

¹Spearing NM1, Connelly LB, Nghiem HS, Pobereskin L: Research on injury compensation and health outcomes: ignoring the problem of reverse causality led to a biased conclusion. *J Clin Epidemiol.* 2012 Nov;65(11):1219-26.

In this process of pain perception, information is processed with a certain hierarchy and can be inhibited for the sake of the discrimination of pain stimuli. Known as “lateral inhibition”, it is a common neurocomputational function and may occur at multiple levels of the nociceptive neuraxis, ranging from the spinal cord, to thalamus, to SI, and beyond. Spinal cord nociceptive neurons have long been known to have large inhibitory surround receptive fields [25]. These inhibitory fields may occupy nearly the entire body outside of the excitatory zone. However, such large fields would be predicted to exert nearly equal influence on stimuli that were in close proximity as well as those that were widely separated. Thus, these large inhibitory surrounds may account for the sub-additive excitation produced by stimuli that lie far apart [26].

During stimulation from various fields, it suppresses input arising from other areas to enhance single point localisation to equal or exceed that predicted by receptive field organization [27-30]. As such, it keeps pain localised to a given distribution. This helps to explain why symptoms from different areas are not perceived or to a lesser extent.

And finally, pain perception can be modulated by negative emotional states [31], anticipation or expectation of pain [32] even in the absence of a physical pain stimulus or through cognitive modulation of pain by attention [33].

This would help to understand why Clients with a “leading injury” do not develop chronification of WAD symptoms as they get suppressed by a “relevant” injury. Their relevance for the restrictions in the Client’s activities of daily living may be minor than, for example, the loss of upper limb function. In the hierarchy of relevance, the signals from the WAD symptoms may simply get lost.

Conclusion

Whiplash associated symptoms following low velocity road traffic accident can present to the expert with a slow recovery. However, when associated with additional and more relevant injuries, they show a quicker resolution. It can be assumed that this is due to an inhibition of pain symptoms in the complex pain process.

Limitations

This study has limitations. It only includes patients in medico-legal proceedings, which may not truly reflect the general population.

Conflict of Interests

The author declares that there is no conflict of interest.

Bibliography

1. Elliott JM., *et al.* “Motor vehicle crash reconstruction: Does it relate to the heterogeneity of whiplash recovery?” *PLoS One* 14.12 (2019): e0225686.
2. Holm LW., *et al.* “The burden and determinants of neck pain in whiplash-associated disorders after traffic collisions: results of the Bone and Joint Decade 2000-2010 Task Force on Neck Pain and Its Associated Disorders”. *Spine* 33.4-1 (2008): S52-S59.
3. Disorders after traffic collisions: results of the Bone and Joint Decade 2000-2010 Task Force on Neck Pain and Its Associated Disorders”. *Journal of Manipulative and Physiological Therapeutics* (2009).
4. Dufton JA., *et al.* “Delayed Recovery in Patients with Whiplash-Associated Disorders”. *Injury* 43.7 (2012): 1141-1147.

5. Carroll LJ, *et al.* "Course and prognostic factors for neck pain in whiplash-associated disorders (WAD): results of the Bone and Joint Decade 2000-2010 Task Force on Neck Pain and Its Associated Disorders". *Journal of Manipulative and Physiological Therapeutics* 32.2 (2009): S97-S107.
6. Vernon H and Mior S. "The Neck Disability Index: A study of reliability and validity". *Journal of Manipulative and Physiological Therapeutics* 14 (1991): 409-415.
7. Walton DM, *et al.* "Risk factors for persistent problems following acute whiplash injury: update of a systematic review and meta-analysis". *Journal of Orthopaedic and Sports Physical Therapy* 43 (2013): 31-43.
8. Sarrami P, *et al.* "Factors predicting outcome in whiplash injury: a systematic meta-review of prognostic factors". *The Journal of Orthopaedics and Traumatology* 18.1 (2017): 9-16.
9. Carroll LJ, *et al.* "Pain-related emotions in early stages of recovery in whiplash-associated disorders: their presence, intensity, and association with pain recovery". *Psychosomatic Medicine* 73.8 (2011): 708-715.
10. Akkermans AJ, *et al.* "Procedural justice and quality of life in compensation processes". *Injury* 44.11 (2013): 1431-1436.
11. Cassidy JD, *et al.* "Effect of eliminating compensation for pain and suffering on the outcome of insurance claims for whiplash injury". *The New England Journal of Medicine* 342 (2000): 1179-1186.
12. Spearing NM and Connelly LB. "Whiplash and the compensation hypothesis". *Spine* 36.25 (2011): S303-S308.
13. Koljonen P, *et al.* "Difference in outcome of shoulder surgery between workers' compensation and nonworkers' compensation populations". *International Orthopaedics* 33.2 (2009): 315-320.
14. De Moraes VY, *et al.* "Workers' compensation status: does it affect orthopaedic surgery outcomes? A meta-analysis". *PLoS One* 7.12 (2012): e50251.
15. Cheriyan T, *et al.* "Association between compensation status and outcomes in spine surgery: a meta-analysis of 31 studies". *The Spine Journal* 15.12 (2015): 2564-2573.
16. Spearing NM and Connelly LB. "Whiplash and the compensation hypothesis". *Spine* 36.25 (2011): S303-S308.
17. Spearing NM, *et al.* "Are People who claim compensation 'cured by a verdict'? A longitudinal study of health outcomes after whiplash". *Journal of Law and Medicine* 20.1 (2012): 82-92.
18. Holbert MD, *et al.* "Comparison of spatial summation properties at different body sites". *Scandinavian Journal of Pain* 17 (2017): 126-131.
19. Quevedo AS, *et al.* "Lateral inhibition during nociceptive processing". *Pain* 158.6 (2017): 1046-1052.
20. M Mokhtar and P Singh. "Periaqueductal Gray". NCBI Bookshelf. A service of the National Library of Medicine, National Institutes of Health. StatPearls [Internet]. Treasure Island (FL): Stat Pearls Publishing (2020).
21. McGaraughty S, *et al.* "Lesions of the periaqueductal gray disrupt input to the rostral ventromedial medulla following microinjections of morphine into the medial or basolateral nuclei of the amygdala". *Brain Research* 1009.1-2 (2004): 223-227.
22. De Oliveira R, *et al.* "5-Hydroxytryptamine_{2A/2C} receptors of nucleus raphe magnus and gigantocellularis/paragigantocellularis pars α reticular nuclei modulate the unconditioned fear-induced antinociception evoked by electrical stimulation of deep layers of the superior colliculus and dorsal periaqueductal grey matter". *Behavioural Brain Research* 316 (2017): 294-304.

23. Calvino B and Grilo RM. "Central pain control". *Joint Bone Spine* 73.1 (2006): 10-16.
24. Menant O., *et al.* "The benefits of magnetic resonance imaging methods to extend the knowledge of the anatomical organisation of the periaqueductal gray in mammals". *The Journal of Chemical Neuroanatomy* 77 (2016): 110-120.
25. Baptista-de-Souza D., *et al.* "Interplay between 5-HT_{2C} and 5-HT_{1A} receptors in the dorsal periaqueductal gray in the modulation of fear-induced antinociception in mice". *Neuropharmacology* 140 (2018): 100-106.
26. Gerhart KD., *et al.* "Inhibitory receptive fields of primate spinothalamic tract cells". *The Journal of Neurophysiology* 46.6 (1981): 1309-1325.
27. Quevedo AS and Coghill RC. "An illusion of proximal radiation of pain due to distally directed inhibition". *The Journal of Pain* 8.3 (2007): 280-286.
28. Bekesy GV. "Neural inhibitory units of the eye and skin. Quantitative description of contrast phenomena". *Journal of the Optical Society of America* 50 (1960): 1060-1070.
29. Koltzenburg M., *et al.* "The ability of humans to localise noxious stimuli". *Neuroscience Letters* 150.2 (1993): 219-222.
30. Moore CE and Schady W. "Cutaneous localisation of laser induced pain in humans". *Neuroscience Letters* 193.3 (1995): 208-210.
31. Schlereth T., *et al.* "Spatial discrimination thresholds for pain and touch in human hairy skin". *Pain* 92.1-2 (2001): 187-194.
32. Phillips ML., *et al.* "The effect of negative emotional context on neural and behavioural responses to oesophageal stimulation". *Brain* 126 (2003): 669-684.
33. Bushnell MC., *et al.* "Pain perception: is there a role for primary somatosensory cortex?" *Proceedings of the National Academy of Sciences of the United States of America* 96 (1999): 7705-7709.
34. Nakamura Y., *et al.* "Attentional modulation of human pain processing in the secondary somatosensory cortex: a magnetoencephalographic study". *Neuroscience Letters* 328 (2002): 29-32.

Volume 11 Issue 8 August 2020

© All rights reserved by E Pietsch.