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Symptomatic approach to posttraumatic headache and its possible implications for treatment

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Abstract We investigated 112 patients [mean age 39.5 ± 10.5 years, 59% women ($n=66$)] with chronic posttraumatic headache following cranio-cervical acceleration/deceleration trauma after an average time interval of 2.5 ± 1.9 years from trauma. Headache following minor head injury or whiplash is one of the most prominent problems in neurotraumatology. Previous research is inconclusive regarding the symptomatic approach of this type of headache. Details of the phenomenology of posttraumatic headache in the previous literature are inconclusive. This may lead to inappropriate treatment strategies, because recent advances in therapy of different headache types may be neglected. Patients were investigated at the outpatient service of the Department of Psychiatry. Headache was analyzed according to its principal location, laterality, projection, quality, precipitation or aggravation and possible additional symptoms. For this analysis,

headache was diagnosed according to the classification of the International Headache Society. The results showed that 42 patients (37%) had tension-type headache, 30 (27%) were identified as suffering from migraine, whereas 20 patients (18%) had cervicogenic headache. An additional 18% of patients suffered from headache that did not fulfill criteria of a particular category. In 104 patients (93%), neck pain was associated in time with headache. Each of the diagnosed headache types in this study may require specific treatment strategies based upon empirical studies of non-traumatic headache types. For these reasons a detailed analysis of headache following cranio-cervical acceleration/deceleration trauma is necessary.

Keywords Head trauma · Whiplash injury · Chronic posttraumatic headache · Migraine · Tension headache

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Introduction

According to the International Headache Society, posttraumatic headache is classified as acute or chronic [15]. Both of these types may occur with or without relevant clinical findings. In addition, to be recognized as posttraumatic headache, it has to be different from pre-existing headache, if such is reported by the patient. However, in the headache classification [15], details of the phenomenology of posttraumatic headache are lacking, and the

previous literature is inconclusive with respect to this issue. Furthermore, regarding posttraumatic headache, the headache classification emphasizes that “the interrelationship between organic and psychosocial factors...is difficult to assess” [15]. This classical understanding encourages overestimation of the relevance of psychosocial factors in this type of headache, while neglecting other possible factors of interest. This is particularly true for posttraumatic headache following minor head injury (i.e., concussion) or whiplash, both being among the most prominent problems in neurotraumatology [2, 5, 10].

Patients with posttraumatic headache frequently undergo extensive investigations, which usually yield negative findings. As a consequence of this, in their treatment of post-traumatic headache, practitioners may include a cocktail of strategies, most of which are not supported by empirical studies. This results in high costs without evidence of increased quality of life of the suffering patient. For these reasons, a symptomatic approach to headache after cranio-cervical acceleration/deceleration injury may be more likely to indicate underlying pathophysiological mechanisms, and aid to the development of treatment strategies.

There are inconclusive results regarding provocation of symptoms in volunteers exposed to low-speed rear-end collisions [8, 20]. However, in volunteer experiments there is usually a well-defined biomechanical mechanism loading on the cervical spine [8, 20], which cannot be properly simulated in the laboratory or analyzed in traffic accident injuries [27, 33]. The following mechanisms may be of relevance in cranio-cervical acceleration/deceleration trauma under traffic conditions:

- **Acceleration/deceleration.** Acceleration/deceleration forces lead to a high load particularly on the upper cervical spine [1, 25, 30]. Depending on the position of the head at the time of impact flexion/extension, translation, rotation, or a combination of these mechanisms may occur [1, 30]. Rotation or translation occur in the upper cervical spine, and both are limited by the anatomical properties of this part of the axial organ [27, 33]. Consequently damage of soft tissue of the upper cervical spine may occur. Such lesions may be uncovered using advanced diagnostic tools [19, 29]. It is considered that these lesions may introduce pain [19, 29, 35]. In addition, the relevance of the cervical spine in headache following cranio-cervical acceleration/deceleration trauma has been clinically documented [15];
- **Rotation.** Rotation of the accidentally accelerated/decelerated head may lead to brain tissue damage [11, 22]. This is indicated by a loss of consciousness, its duration being considerably related to the trauma severity [2, 9]. Lower degrees of trauma severity, as indicated by a shorter loss of consciousness, do not frequently leave brain lesions detectable by routine diagnostic tools (e.g., magnetic resonance imaging) [2, 9]. However, experimental studies demonstrated microscopically detectable lesions in the more superficial areas of the frontal cortex (i.e., between the gray and white matter, which is referred to as tissue shearing) even at lower acceleration/deceleration forces [11, 22]. Furthermore, recent studies showed that mild or moderate head trauma (i.e., loss of consciousness shorter than 5 min) [17] or whiplash [4] may lead to perfusion deficits in the frontal cortex, in which lesions due to cranio-cervical acceleration/deceleration trauma are most likely to occur [22]. According to these findings, different types of lesions may occur in patients after cranio-cervical

acceleration/deceleration trauma, and on this basis different types of posttraumatic headache may be expected to be found and, eventually, explained.

In this study, we investigated patients who suffered cranio-cervical acceleration/deceleration trauma (i.e., mild to moderate head trauma or whiplash) in order to specify the phenomenology of headache in these patients as a possible rationale for treatment strategies, particularly in primary care.

Materials and methods

We analyzed 112 patients [mean age 39.5 ± 10.5 years, 59% women ($n=66$)] with chronic posttraumatic headache (frequency at least twice a week) after cranio-cervical acceleration/deceleration trauma. Nineteen of the patients (17%) were in litigation. Trauma in these patients had occurred on average 2.5 ± 1.9 years previously. Twenty-five patients (22%) had suffered traumatic loss of consciousness (range 0–20 min, median 0 min) and the remaining 87 patients had suffered whiplash injury, as defined previously [27, 33]. Objectively verified traumatic intracranial sequelae (e.g., subdural or intracerebral hematoma), injuries to other parts of the body, epileptic seizures or neurological signs were exclusion criteria in this study. Detailed analysis of headache in all patients included principal location (e.g., frontal, occipital), laterality (e.g., uni- or bilateral, alternating), projection (e.g., arising from the neck projecting to frontal), quality (e.g., pulsatile, pressing/tightening), precipitation or aggravation (e.g., physical or mental activity, tiredness) and possible additional symptoms (e.g., nausea, sensitivity to light or noise). Based on this analysis, diagnosis of headache was made according to the headache classification [15]. In addition, patients were interviewed to establish whether they had suffered from headache before the trauma, and if so, details of its type and frequency. Furthermore patients were asked about medication prescribed for their headache following trauma, and how they were following prescriptions.

Results

Forty-two patients (37%) fulfilled criteria for tension-type headache, 30 (27%) were identified as suffering from migraine, whereas 20 patients (18%) had cervicogenic headache. An additional 18% of patients suffered from headache overlapping between different groups, and could not be classified as a particular headache category. There was no significant gender difference between patients diagnosed with migraine and the rest. In addition, migraine was not significantly more frequent in patients who had suffered traumatic loss of consciousness. Twenty-eight patients (25%) had strictly unilateral pain, and an additional 18 patients (16%) had unilateral pain that alternated

Table 1 Location of headache in the investigated sample

	<i>N</i>
Always bilateral	66
Strictly unilateral	28
Alternating unilateral	18

Table 2 Quality of headache

Additional sensation	Principal sensation			
	Pressing-tightening (<i>n</i> =66)	Pulsatile (<i>n</i> =21)	Stitching (<i>n</i> =21)	Burning (<i>n</i> =4)
Pulsatile	16	–	–	–
Burning	6	4	11	–
Stitching	2	1	–	–
Pulsatile and stitching	3	–	–	–
Pulsatile and burning	1	–	–	–
Burning and stitching	–	1	–	–

Table 3 Precipitation or aggravation of headache

Principal mode	Additional mode	<i>N</i>
Physical activity (<i>n</i> =101)	Mental effort and tiredness	26
	Climate	7
	Tiredness and climate	3
	Emotions	3
	Tiredness and emotions	1
	Tiredness and alcohol	1
Climatic changes (<i>n</i> =6)	Alcohol	1
	Food	1
	Emotions	1
Mental effort (<i>n</i> =5)	Tiredness	5
	Sleeplessness	2

in side, but was limited to one side of the head during each attack (Table 1). In 104 patients (93%), neck pain was associated in time with the headache. A remarkable finding was that many patients indicated one prominent quality of the headache (e.g., pressing/tightening), but this was frequently accompanied by one or more additional painful sensations (e.g., pulsatile, burning) (Table 2). The increased number of adjectives to describe quality of headache was not in significant correlation with the length of the posttraumatic symptom-free interval. One further observation was that many patients specified one principal mode of precipitation (e.g., physical activity) but also different additional modes of either precipitation or aggravation of their headache (e.g., mental effort, climatic changes) (Table 3). During headache attacks, many patients suffered additional symptoms such as sensitivity to light (*n*=43), sensitivity to noise (*n*=29), nausea (*n*=35), and vomiting (*n*=16) (Table 4).

With regard to the existence or type of the pretraumatic headache, the data were very inconclusive. Most of the patients, however, insisted that their headache had occurred exclusively as a consequence of trauma. Based on the limited reliability of these data, we did not include them in statistical analysis. Furthermore, there was a great variability in both prescribed and self-administered med-

Table 4 Additional symptoms during headache attack

	<i>N</i>
Sensitivity to light	43
Sensitivity to noise	29
Nausea	35
Vomiting	16

ication for posttraumatic headache, including the substances used or the frequency with which they were applied (e.g., only now and then or during each attack). Moreover, many patients (if in treatment or being prescribed medication) did not strictly follow the physicians' instructions on how to use their medication, mainly because of lack of effect. These factors precluded a sound statistical analysis of the currently used medication as a possibly relevant factor. Most patients in this sample suffered from a broad spectrum of psychological symptoms frequently referred to as dysphoric mood. However, none of the patients fulfilled the diagnostic criteria of an episode of major depression. This prevented any analysis of different types of headaches in this sample in relation to depression.

Discussion

Headache is one of the most prominent symptoms after cranio-cervical acceleration/deceleration trauma. It represents a considerable problem in neurotraumatology, particularly in patients after minor head trauma or whiplash, and is reasonably well correlated with high costs for assessment and treatment [2, 5, 10]. As yet, little attention has been paid to the phenomenology of posttraumatic headache, that is, the headache itself. Understanding the nature of posttraumatic headache may help us to understand its underlying pathophysiology, and knowledge of both could be helpful for indications of treatment. This may be particularly useful in primary care, where patients usually receive medical help after trauma, and where disorderly sequences of treatment strategies are implemented, usually with very poor response rates. Disillusion may then lead to the belief that psychosocial factors or financial claims are primarily responsible for this type of headache [5]. However, recent studies have indicated that psychosocial problems of patients after cranio-cervical acceleration/deceleration trauma emerge secondary to as yet undetectable lesions [27, 35], and the primary relevance of the compensation issue in posttraumatic headache has been rejected on the basis of empirical research [23].

We investigated a selected sample, which nevertheless presented with problems typical for patients suffering from chronic posttraumatic headache. The following results should be highlighted.

Neck pain

Neck pain was documented in over 90% of patients in the present study. In accordance with previous research, this result suggests a considerable involvement of the neck even in patients diagnosed with head trauma (e.g., concussion) [34]. This result likely reflects damage of the upper cervical spine, where high load may be expected due to acceleration/deceleration forces [1, 25, 30]. Damage to the soft tissue of the upper cervical spine may lead to central sensitization [18], which is considered to occur in the cervical dorsal horn or nuclei of the trigeminal nerve, as may be concluded from experimental studies [12]. As pointed out earlier, this sensitization may trigger the trigeminovascular efferents, the pivotal mechanism of the pathophysiology of migraine attacks, according to current experimental data [18]. Furthermore, the suggested involvement of extracerebral structures, particularly the neck, in migraine [6], has been supported by experimental data [21]. In addition, central sensitization may be responsible for muscle contraction, a mechanism found to be involved in tension-type headache [18], while recognized lesions to structures of the cervical spine may be responsible for cervicogenic headache [19, 29]. Given the possibility of central sensitization as a basis for developing chronic headache following cranio-cervical acceleration/deceleration trauma, the important preventive measure may be adequate initial analgesia in these patients. In this regard, considerably higher initial doses of analgesics than are usually administered may be required. In addition, the concept of central sensitization may provide grounds for a better understanding of the different types of the headache found in this study.

Headache type

The majority of patients in this study suffered from tension-type headache. Present results further indicate that about one-third of patients who suffer cranio-cervical acceleration/deceleration trauma may suffer from migraine, which is similar to previously published results [13]. In previous research, microscopic lesions detectable by nuclear medicine techniques have been found in the frontal cortex in mild to moderate head trauma or whiplash [4, 17]. Such lesions may establish the basis for a biochemical cascade in brain tissue that is similar to events said to occur in migraine, and may help to explain migraine as a result of cranio-cervical acceleration/deceleration trauma [24]. Furthermore, about 20% of the present sample suffered from what is referred to as cervicogenic headache [15]. Finally, there was a sizable minority of patients in whom headache fulfilled several criteria between two or more categories, indicating that following cranio-cervical acceleration/deceleration injury a continuum of different headache types may occur. It may be argued that a part of

this latter group may suffer from headache due to overuse of different types of medication, some of which may lead to a rebound headache, as pointed out previously [36]. Despite considerable reservations about posttraumatic headache as an analgesic rebound phenomenon [14, 31], it may be assumed that patients who do not strictly follow physicians' medication recommendations (one possible conclusion from this sample) may do so because medication prescribed by the physician is not effective. This inefficacy may be due to lack of recognition of the given type of headache before initiating treatment.

Psychological problems

Psychological problems were quite common in this sample, and mainly fulfilled criteria of dysphoric mood, which is in line with previous studies [35]. In addition, in accordance with previous research [28], results here indicated that clinical depression is uncommon in patients who have suffered cranio-cervical acceleration/deceleration trauma. This, as stressed previously [28], should be considered in deciding on treatment strategies, in particular when initiating therapy with antidepressants.

Pretraumatic headache

One unresolved problem that emerged from the retrospective nature of this study was the relationship between pretraumatic and trauma-related headache. Most of the patients in this study indicated that their headache was exclusively due to the trauma. This may be disputed, because previous studies have found pretraumatic headache to be a significant risk factor for headache following cranio-cervical acceleration/deceleration trauma [26, 27]. The proposed relationship between pretraumatic and trauma-related headache may be based on pre-existing central sensitization and triggering by the injury, again explaining chronic headache as a consequence of cervical soft-tissue injury. In contrast, in individuals without this risk factor, soft-tissue injury of the cervical spine may more likely be tolerated without headache.

Conclusions

Based on present results, treatment strategies for chronic posttraumatic headache should be appropriate for the type of headache, always keeping in view the results of empirical studies. This may appear to make sense for headache specialists. However, the very poor response rates usually found in clinical routine indicate that treatment of posttraumatic headache by practitioners is usually not appropriate for the symptomatology. In the case of tension-type headache, tricyclic antidepressants have the best empirically supported effectiveness [3]. In consideration of side

effects, prophylactic treatment of migraine following cranio-cervical acceleration/deceleration trauma may include beta-blockers, calcium channel blockers, anticonvulsants, ergotamine alkaloids, or serotonin-1-D-receptor agonists [29, 31]. Patients suffering from cervicogenic headache may primarily benefit from a combination of

analgesics, well-directed physical therapy, or blockade of the greater occipital nerve [7]. In carefully selected cases of posttraumatic cervicogenic headache, botulinum toxin or manipulative therapy may be considered [16]. Treatment studies using a randomized allocation of patients will be needed to clarify the main points mentioned here.

References

- Adams MA (1998) Biomechanics of the cervical spine. In: Gunzburg R, Szpalski M (eds) Whiplash injuries: current concepts in prevention, diagnosis, and treatment of the cervical spine whiplash syndrome. Lippincott-Raven, Philadelphia, pp 13–20
- Alexander MP (1995) Mild traumatic brain injury: pathophysiology, natural history, and clinical management. *Neurology* 45:1253–1260
- Bendtsen L, Jensen R, Olesen J (1996) A non-selective amitriptyline, but not a selective (citalopram), serotonin reuptake inhibitor is effective in the prophylactic treatment of chronic tension-type headache. *J Neurol Neurosurg Psychiatry* 61:285–290
- Bicik I, Radanov BP, Schäfer N, Dvorak J, Blum B, Weber B, von Schultness GK, Buck A (1998) FDG-PET and HMPAO-SPECT in patients suffering from late whiplash syndrome. *Neurology* 51:345–350
- Binder LM, Rohling ML (1996) Money matters: meta-analysis review of the effects of financial incentives on recovery after closed head injury. *Am J Psychiatry* 153:7–10
- Blau JN, MacGregor EA (1994) Migraine and the neck. *Headache* 34:88–90
- Bovim G, Bovim S, Bovim T (1992) Cervicogenic headache, migraine without aura and tension-type headache. Diagnostic blockade of greater occipital and supraorbital nerves. *Pain* 51:43–48
- Brault JR, Wheller JB, Siegmund GP, Brault EJ (1998) Clinical response of human subjects to rear-end automobile collisions. *Arch Phys Med Rehabil* 79:72–80
- Esselman PC, Uomoto JM (1995) Classification of the spectrum of mild traumatic brain injury. *Brain Inj* 9:17–24
- Evans RW (1992) The postconcussional syndrome and the sequelae of mild head injury. *Neurol Clin* 10:815–847
- Gennarelli TA (1993) Mechanisms of brain injury. *J Emerg Med* 11[Suppl 1]:5–11
- Goadsby PJ, Knight YE, Hoskin KL (1997) Stimulation of the greater occipital nerve increases metabolic activity in the trigeminal nucleus caudalis and cervical dorsal horn of the cat. *Pain* 73:23–28
- Haas DC (1996) Chronic posttraumatic headaches classified and compared with natural headaches. *Cephalalgia* 16:486–493
- Haas DC (1997) Posttraumatic headache. *Neurology* 48:1735
- Headache Classification Committee of the International Headache Society (1998) Classification and diagnostic criteria for headache disorders, cranial neuralgias and facial pain, 1st edn. *Cephalalgia* 8 [Suppl 7]
- Hobson DE, Gladish DF (1997) Botulinum toxin injection for cervicogenic headache. *Headache* 37:253–255
- Ichise M, Chung D-G, Wang P, Worzmann G, Gray BG, Franks W (1994) Technetium-99m-HMPAO-SPECT, CT and MRI in the evaluation of patients with chronic traumatic brain injury. *J Nucl Med* 35:217–226
- Jensen R, Olesen J (1996) Initiating mechanisms of experimentally induced tension-type headache. *Cephalalgia* 16:175–182
- Lord SM, Barnsley L, Wallis BJ, Bogduk N (1996) Chronic cervical zygapophysial joint pain after whiplash: a placebo-controlled prevalence study. *Spine* 21:1737–1745
- Meyer S, Weber M, Castro W, Schilgen M, Peuker C (1998) The minimal collision velocity for whiplash. In: Gunzburg R, Szpalski M (eds) Whiplash injuries: current concepts in prevention, diagnosis, and treatment of the cervical spine whiplash syndrome. Lippincott-Raven, Philadelphia, pp 95–115
- Moskowitz MA (1993) Neurogenic inflammation in the pathophysiology and treatment of migraine. *Neurology* 43 [Suppl 6]:16–20
- Ommaya K, Gennarelli TA (1975) Experimental head injury. In: Vinken P, Bruyn G (eds) Handbook of clinical neurology. North-Holland, Amsterdam, pp 67–90
- Packard RC (1992) Posttraumatic headache: permanency and relationship to legal settlement. *Headache* 32:496–500
- Packard RC, Ham LP (1997) Pathogenesis of posttraumatic headache and migraine: A common pathway? *Headache* 37:142–152
- Penning L (1992) Acceleration injury of the cervical spine by hypertranslation of the head. II. Effect of hypertranslation of the head on cervical spine motion: discussion of literature data. *Eur Spine J* 1:13–19
- Radanov BP, Sturzenegger M, Schnidrig A, Di Stefano G, Aljinovic M (1993) Factors influencing recovery from headache after common whiplash. *BMJ* 307:652–655
- Radanov BP, Sturzenegger M, Di Stefano G (1995) Long-term outcome after whiplash injury – a two years follow-up considering features of accident mechanism, somatic, radiological and psychosocial findings. *Medicine* 74:281–297
- Saper JR (1997) Diagnosis and symptomatic treatment of migraine. *Headache* 37 [Suppl 1]:1–14
- Schellhas KP, Smith MD, Gundry CR, Pollei SR (1996) Cervical discogenic pain. Prospective correlation of magnetic resonance imaging and discography in asymptomatic subjects and pain sufferers. *Spine* 21:300–312
- Shea M, Edwards WT, White AA, Hayes WC (1991) Variations of stiffness and strength along the human cervical spine. *J Biomech* 24:95–107
- Silberstein SD, Lipton RB (1994) Overview of diagnosis and treatment of migraine. *Neurology* 44 [Suppl 7]:6–16
- Sternberg PE (1997) Posttraumatic headache. *Neurology* 48:1735
- Sturzenegger M, Radanov BP, Di Stefano G (1995) The effect of accident mechanisms and initial findings on the long-term course of whiplash injury. *J Neurol* 242:443–449
- Treleaven J, Jull G, Atkinson L (1994) Cervical musculoskeletal dysfunction in post-concussional headache. *Cephalalgia* 14:273–277
- Wallis BJ, Lord SM, Bogduk N (1997) Resolution of psychological distress of whiplash patients following treatment by radiofrequency neurotomy: a randomised, double-blind, placebo-controlled trial. *Pain* 73:15–22
- Warner JS, Fenichel GM (1996) Chronic posttraumatic headache often a myth? *Neurology* 46:915–916