P1: KWW/KKL P2: KWW/HCN QC: KWW/FLX T1: KWW
GRBT050-107 Olesen- 2057G GRBT050-Olesen-v6.cls August 17, 2005 1:41

Chapter 107

Headache Attributed to Whiplash Injury

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INTRODUCTION

International Headache Society (IHS) code and diagnoses:

5.3 Acute headache attributed to whiplash injury
5.4 Chronic headache attributed to whiplash injury

World Health Organization (WHO) code and diagnoses:

S13.4 Disorders of the cervical column
S13.4C Whiplash syndrome

Short description: The term *whiplash* as such includes both the mechanism of a trauma and a collection of symptoms found after exposure to a certain type of injury (8,59). Immediate exposure to a whiplash-type neck distortion is consistent with the S13.4 diagnosis, which is a nonspecific neck distortion diagnosis. The chronic whiplash syndrome S13.4C is described by a collection of symptoms and plausible exposure to a whiplashlike injury of the neck.

In the IHS International Classification of Headache Disorders (ICHD)-II, a distinction is made between acute headache attributed to whiplash injury (ICHD-II code 5.3) and chronic headache attributed to whiplash injury (ICHD-II code 5.4) (see Table 107-1).

Whiplash injury is produced when acceleration and deceleration forces act on the neck, resulting in hyperextension, hyperflexion, and, occasionally, hyperrotation of the cervical spine. Rear-end or side-impact car collisions (39) or other types of events can cause whiplash injury (58).

The Quebec Task Force (58) proposed the whiplash-associated disorder (WAD) criteria to grade the severity of a whiplash injury (Table 107-1). The criteria resemble previous classifications (43) and have been widely adopted. The term *chronic WAD,* defined as persisting WAD symptoms after 6 months postinjury, is acknowledged, and it is essentially identical with the widely used terms *chronic whiplash syndrome* and *late whiplash syndrome*.

EPIDEMIOLOGY

A Canadian epidemiologic survey indicated that the incidence of compensated whiplash was 0.70 per 1000 inhabitants per year in 1987 (0.85 per 1000 women and 0.54 per 1000 men) in Quebec and 10 times higher in Saskatchewan (58). Notably, Saskatchewan changed from the tort system to the no-fault system in 1995 (14), resulting in a reduced number of insurance claims and a significant decline in the incidence of long-term pain and disability: a 43% reduction of claims in men and a 15% reduction in women during a 1-year period. This result was contrasted by an increase in the number of vehicle damage claims and number of kilometers driven. A Swedish study reported an incidence of one cervical strain injury per 1000 inhabitants per year (7). At present, the prevalence of whiplash injury is unknown.

In some Western countries, but not in other, the incidence of whiplash injury has increased remarkably during the last decades (5). A 25-year Dutch retrospective study documented a 12-fold increase between 1970 and 1994 in the occurrence of neck sprain after motor vehicle accidents in persons immediately treated and diagnosed at the unit (63). The frequency of neck sprains was highest (0.28 per 1000) among 25- to 29-year-old people. A 1.3-fold increase in number of cars and a 1.7-fold increase in number of kilometers driven were among the proposed explanations for the rise in frequency of neck sprains (63). Also, there was a fourfold increase in neck sprains after noncar accidents: 25% accidental falls, 24% sport traumas, 14% bicycle accidents, and 8% motorcycle accidents during the observation period. Noncar accidents were most frequently reported in young persons (15 to 19 years old: 39 per 100,000; 10 to 14 years old: 34 per 100,000). The increase in neck sprains after noncar accidents was explained by an increase in sport and leisure activities and by mandatory participation in school gymnastics. The largest increase was observed in concert with intensive focusing on whiplash in the media.

P1: KWW/KKL P2: KWW/HCN QC: KWW/FLX T1: KWW
GRBT050-107 Olesen- 2057G GRBT050-Olesen-v6.cls August 17, 2005 1:41

TABLE 107-1 Whiplash Associated Disorders (WADs)

Grades 0:	No complaints and no clinical signs
Grade 1:	Complaints of neck pain, neck stiffness, or neck tenderness only
Grade 2:	Neck complaints as in grade 1 + musculoskeletal signs with reduced neck mobility and/or tender muscle points at examination
Grade 3:	Neck complaints and musculoskeletal signs as in grade 1 and 2 + neurologic signs (absent or decreased tendon reflexes or weakness, sensory deficits)
Grade 4:	Fracture or dislocation

Grades 0 and 4 are not considered whiplash injuries.
Patients with reported amnesia and/or loss of consciousness do not fulfill the whiplash diagnosis criteria.

From ref. 58.

A British study found a remarkable increase in road traffic accidents in the years 1982–1991, and the proportion of accidents associated with a diagnosis of neck sprain increased correspondingly (7). For example, 8% of 929 traffic injury victims sustained a neck sprain in 1982–1983 as compared to 46% of 6149 victims in 1991–1992 (7).

CLINICAL FEATURES

IHS ICHD-II diagnostic criteria are as follows:

5.3 Acute headache attributed to whiplash injury
A. Headache, no typical characteristics known, fulfilling criteria C and D.
B. History of whiplash (sudden and significant acceleration/deceleration movement of neck) associated at the time with neck pain.
C. Headache develops within 7 days after whiplash injury.
D. One of the other of the following:
  1. Headache resolves within the following 3 months after whiplash injury.
  2. Headache persists, but 3 months have not yet passed since whiplash injury.

5.4 Chronic headache attributed to whiplash injury
A. Headache, no typical characteristics known, fulfilling criteria C and D.
B. History of whiplash (sudden and significant acceleration/deceleration movement of neck) associated at the time with neck pain.
C. Headache develops within 7 days after whiplash injury.
D. Headache persists for >3 months after whiplash injury.

Principal symptoms: Neck pain, restricted neck movement/neck stiffness, and headache are the most commonly reported symptoms after acute whiplash injury. High initial pain intensity (56) and initial restriction of cervical mobility (32,50) are related to bad outcome after whiplash injury.

Other more inconsistently reported symptoms are shoulder–arm pain; numbness in the shoulder, arm, and hand; dysphagia and globulus; dizziness; and visual and auditory disturbances (26,30,47,60).

HEADACHE IN ACUTE WHIPLASH INJURY

Headache occurs in 40 to over 80% of acute whiplash patients (16). In one study, 34% of all responders reported persistent troublesome neck pain within the last year, and 14% suffered neck pain for more than 6 months (12). A lifetime history of headache is associated with moderate or severe whiplash headache (15,26,33).

Proposed causes of headache in acute whiplash injury are zygapophyseal arthropathy at the cervical C2–C3 level (64), segmental spreading (31), central (36) sensitization of nociceptors giving rise to chronic pain condition, or dysfunction in brainstem or upper cervical medulla (34).

Types of Headache After Whiplash Injury

Headache is most frequently classified as a unilateral or even bilateral cervicogenic type (2,17), as occipital or tension-type (51), or, more rarely, as orofacial pain or temporomandibular disorder–related pain (49,19,37). Retrospectively, Balla and Karnaghan examined headache in 122 late whiplash syndrome patients, seen 6 months to 3 years after injury (3). Headache was present in 82%: 46% occipital headache, 34% generalized headache, 20% in other regions, and constant in more than 50% (3).

Natural History of Headache and Other Symptoms in Whiplash

Whether acute whiplash injury produces long-term headache, neck pain, other spinal pain, or musculoskeletal pain remains unclear. High frequencies of various types of headache (52), neck pain (12), musculoskeletal neck pain, intrascapular pain, and low back pain have been reported (13,25,38). The high frequency of these painful conditions emphasizes the need for control groups when examining headache and other pain conditions related to acute whiplash injury and the so-called late whiplash syndrome.

P1: KWW/KKL P2: KWW/HCN QC: KWW/FLX T1: KWW
GRBT050-107 Olesen- 2057G GRBT050-Olesen-v6.cls August 17, 2005 1:41

PATHOPHYSIOLOGY

Animal Models

Retropharyngeal hematomas and lesions in anterior longitudinal ligaments are observed during acceleration injuries in monkeys (39,40). Others have reported membrane leakage in cervical spinal ganglia in pigs sustaining experimental hyperextension and hyperflexion trauma of the neck (66).

Human Studies

Strain of anterior structures and compression of posterior structures (spinous processes, apophyseal joints) may occur (e.g. anterior cervical muscles, anterior part of intervertebral discs, anterior longitudinal ligament, and pharyngeal structures) during forced extension of the neck. On the other hand, there is a potential strain of posterior structures (nuchal ligament, posterior neck muscles, and apophyseal joint capsules) and a potential compression of anterior structures (intervertebral discs and vertebral bodies) during forced flexion (5,8). A lesion of cervical apophyseal joints has been proposed as a possible pain source (8) and may theoretically be causative for eye dysfunction. Painful stimulation of superficial nociceptors in the neck and head induces ipsilateral pupil dilation (ciliospinal reflex). Deep nociceptors near apophyseal joints may have the same properties (8,9). Indeed, pain relief is obtained in whiplash patients after diagnostic blockade to apophyseal joints and following radiofrequency medial branch neurotomy (4,55).

Good or excellent pain relief can be achieved in over 70% of patients with whiplash injury and deep occipital aching pain by means of occipital nerve release (41). These observations are explained anatomically by the convergence of deep and superficial nociceptive information to the dorsal horn (27,42) or the trigeminal subnucleus caudalis (the caudal part is situated at C2–C3 level) (57). Furthermore, some anatomic studies support the theory that injuries to the second cervical ganglion and the nerve cause occipital headache in whiplash (35). Other possible sources for damage/dysfunction after whiplash injury are strain of cervical (39) and alar ligaments (18), disc protrusions (28,29), and reduced cerebral blood flow in posterior regions (44,45,6).

In a prospective study, Hildingsson and colleagues reported a pathologic oculomotor test (smooth pursuit test) initially in 8 out of 40 patients (26). All eight patients and further five more patients at 15 months follow-up had abnormal test results. All patients with abnormal oculomotor mobility had persisting symptoms.

The smooth pursuit test and the modified smooth pursuit neck torsion test can be abnormal in chronic whiplash patients both with or without complaints of dizziness and visual disturbance (24,62). Dysfunction of proprioceptors in the neck is a proposed cause of the dizziness and eye motility deficiency. However, some argue that the abnormal eye movements result from hyperventilation when performing the test (21,22).

Biomechanical and Pathophysiologic Considerations on Pain Development After Acute Whiplash Injury

At the time of acceleration of the trunk and shoulders when a car is rear-ended, there are no direct forces acting on the head, which is in a greater inertial state than the neck, shoulder, or trunk. This results in neck hyperextension. After the head's inertia is overcome, the head accelerates, probably facilitated by decompression of previously compressed neck structures and with the neck acting as a lever for the head. Deceleration is due to a difference in inertia, which is slower in the head than neck and trunk, resulting in neck hyperflexion (5). Cadaver specimens (46,28,65) indicate that structures at risk are cervical discs, facet joints, and soft tissue structures in the neck (8,61). However, computed tomography (CT), magnetic resonance (MR), positron emission tomography (PET), and single-photon emission computed tomography (SPECT) scans have not revealed any visible structural changes after common whiplash lesions (1,11,30,48,53).

Treatment of Acute and Chronic Whiplash Patients

Treatment of Headache and Neck Pain in Acute Whiplash Injury

No universally accepted treatment guidelines have been published. Nevertheless, short-term active exercise and physiotherapy (10,23,54) and short-term use of acetaminophen, acetylsalicylic acid, or a nonsteroidal antiinflammatory drugs (20,58) are recommended. Application of a soft collar, immobilization, and more than 3 to 5 days of rest should not be advised (11,20,58).

Treatment of Chronic Whiplash Syndrome

At present, no studies have described the long-term effect of physiotherapy, chiropractic treatment, interdisciplinary approach, massage, ultrasound, acupuncture, or other passive treatments (20,58).

Initial relief with a diagnostic block followed by radio frequency medial branch neurotomy may benefit both local neck pain and psychologic distress, but long-term results have not been published (4,55,64).

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GRBT050-107 Olesen- 2057G GRBT050-Olesen-v6.cls August 17, 2005 1:41

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GRBT050-107 Olesen- 2057G GRBT050-Olesen-v6.cls August 17, 2005 1:41

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