Pathological Mechanism and Clinical Practice of Whiplash-Associated Disorders

Yiqin Peng*

Kingswood Oxford School, Connectict, West Hartford, 06119, USA

*Corresponding author's e-mail: pennypyqtt@gmail.com

Abstract. This review discusses the pathological mechanisms and clinical manifestations of Whiplash-Associated Disorders (WADs, a common clinical term), which result from sudden acceleration-deceleration forces affecting the neck, commonly seen in traffic accidents. Clinical studies and follow-up data demonstrate that many patients continue to experience symptoms such as neck pain, headaches, and upper limb pain long after the initial injury, potentially involving damage to the cervical spine and associated nerves. The etiology of WADs is complex, involving injuries to cervical facet joints, intervertebral discs, and cervicothoracic nerves. Due to the diagnostic challenges, treatment of WADs requires a multimodal approach, including medications, rehabilitation, and psychological interventions. For chronic pain cases, surgical intervention may be necessary. This review emphasizes the importance of a comprehensive understanding of the pathology and clinical presentation of WADs to effectively manage both acute and chronic pain in affected patients.

Keywords: Whiplash-Associated Disorders (WADs), Cervical spine anatomy, Chronic pain management, Multimodal treatment

1 Introduction

The term "whiplash injury" describes damage to the neck brought on by a machine that accelerates and decelerates quickly, causing sudden movements of the neck's extension and flexion [1]. Dr. Harold Crowe observed in 1928 that rear-end crashes in automobiles cause a "whiplash effect" on the cervical spine. It wasn't until 1945 that the name "whiplash" was used in a paper that it was formally recognized [2].

Due to the frequent occurrence of traffic accidents, whiplash injuries are a prevalent form of unintentional injury, contributing to a substantial economic burden on health systems. Approximately $29 billion is spent annually in the United States on whiplash injury treatment, diagnosis, insurance, and litigation.

Patients suffering from whiplash may exhibit a range of symptoms categorized as whiplash-associated disorders (WADs) [3]. A follow-up study conducted by Radanov and colleagues tracked 117 patients with WADs over two years, revealing that many continued to experience symptoms in the long term. Specifically, the data indicated that 44% of patients had symptoms at three months, 30% at six months, 24% at one

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year, and 18% at two years post-injury. Figure 1 presents what is considered a conservative estimate of long-term morbidity compared to findings from other research.

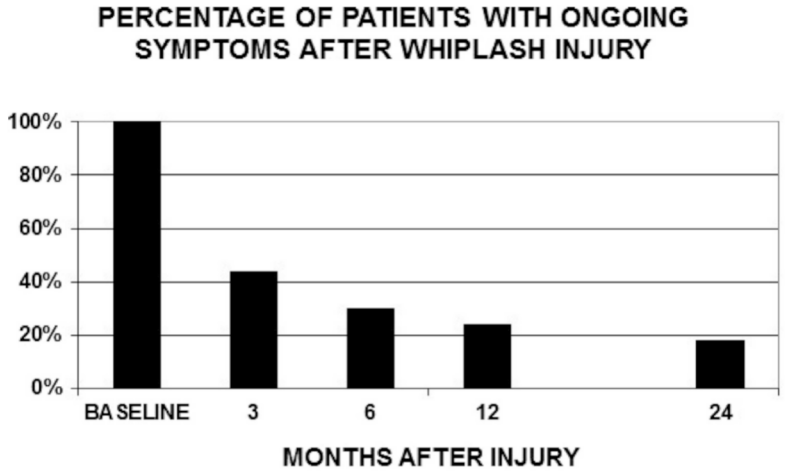

Fig. 1. Percentage of patients with ongoing symptoms after whiplash injury [3].

2 Anatomy of Whiplash-Associated Disorders (WADs)

A comprehensive knowledge of the cervical spine's anatomy and its associated regional spinal cord is essential for accurately evaluating patients who present with neck pain. The cervical spine comprises seven vertebrae, with specific articulations that are pivotal for its range of motion. About a third of the neck's flexion and extension as well as half of its lateral bending are made possible by the atlantooccipital joint, which is located between the occipital bone and the first cervical vertebra. Around half of the rotational mobility of the neck, on the other hand, is controlled by the atlantoaxial joint, which is situated between the first and second cervical vertebrae. About 50% of rotational and lateral bending actions, as well as two-thirds of flexion and extension, are produced by the joints connecting the second to seventh cervical vertebrae.

The area between the C4 and C7 vertebrae, where the nerve roots that travel through the intervertebral foramina correspond to C5, C6, and C7, is where degenerative changes are most commonly seen. Luschka's joints, also called uncovertebral joints, are located at the posterolateral border of the intervertebral disc and the anteromedial side of the intervertebral foramen between C3 and C7. foramen between C3 and C7. These structures can experience hypertrophic alterations in response to intervertebral disc degeneration, while not being considered real synovial joints. The intervertebral foramen may shrink as a result of this hypertrophy, which is commonly linked to cervical radiculopathy. It is crucial to understand that although the symptoms brought on by these degenerative alterations may resemble those of whiplash-associated disorders (WADs), there are significant anatomical differences at play.

There is a dorsal root and a ventral root for each of the eight spinal nerve pairs that comprise the cervical spine. The dorsal root contains primary sensory afferent fibers from neurons in the dorsal root ganglia, whereas the ventral root distributes efferent fibers from alpha motor neurons in the ventral horn of the spinal cord. Cervical radiculopathy, a condition caused by degenerative changes in the spine, compresses the nerve roots and, depending on which nerve root is affected, produces a variety of clinical symptoms. Myotomal and dermatomal distributions are usually evident, but they are not always precisely aligned.

The dorsal primary ramus and the ventral primary ramus are the two divisions of the spinal nerve that results from the junction of the dorsal and ventral roots. The muscular, cutaneous, and articular components of the posterior neck are innervated by the dorsal ramus. Conversely, the brachial plexus, which supplies the upper limb, is built with the assistance of the ventral ramus, which also innervates the prevertebral and paravertebral muscles. Compression of the brachial plexus, discussed later in this paper, is often attributed to upper limb pain associated with whiplash-associated disorders (WAD).

3 Clinical Manifestations and Mechanisms of Whiplash-Associated Disorders (WADs)

Whiplash injuries are categorized based on the accompanying signs and symptoms [4]:

Grade 1: The existence of stiffness or soreness in the neck without any physical symptoms.

Grade 2: Musculoskeletal symptoms including restricted range of motion and localized discomfort are present along with neck pain or stiffness.

Grade 3: stiffness or pain in the neck combined with neurologic symptoms such muscle weakness, sensory deficits, and diminished or nonexistent deep tendon reflexes.

Grade 4: A fracture or dislocation accompanied by stiffness or pain in the neck.

3.1 Cervicothoracic Pain

Neck pain is the most commonly reported symptom after a whiplash injury. Frequently, patients report stiffness and nonradicular neck pain. During a thorough physical examination, the cervical spine usually has a restricted and painful range of motion, and the paracervical area is tender to the touch.

Neck pain associated with whiplash-associated disorders (WAD) is frequently accompanied by pain in the mid to upper thoracic region and/or post-traumatic headaches. Current literature indicates that a considerable proportion of upper thoracic pain in WAD patients may arise from referred pain originating in the cervical area.

After conducting a comprehensive literature study, we have concluded that the main causes of whiplash injuries most likely go beyond the well-known phenomenon of muscle guarding. Patients with chronic WAD frequently exhibit decreased cervical

range of motion long after the expected recovery period for muscle injuries has passed, despite the common assumption that the restricted range of motion is predominantly caused by muscle guarding. This study suggests that the persistent neck stiffness that this patient population experiences may be greatly influenced by additional factors, such as intervertebral disc damage, cervical facet joint injuries, and central nervous system sensitivity of pain.

3.2 Cervical Facet Joint

In patients with persistent injuries from auto accidents, cervical facet joint degeneration is the main cause of headaches and neck pain, according to research published in peer-reviewed journals. Previous studies [5] have reported that approximately 60% of cases of chronic non-radicular neck pain following such injuries can be attributed to injuries of the cervical facet joints, particularly at the C2-C3 level, although involvement at other cervical levels has also been noted. However, these investigations employed a double-blind experimental design utilizing analgesic and placebo injections, rather than relying on direct anatomical or imaging evidence.

Researchers studied isolated cervical spine specimens under various force conditions in early biomechanical investigations. The mechanism of whiplash damage was captured by high-speed photography, which revealed that the chin moves toward the chest and the occiput shifts posteriorly in relation to the trunk in a split second following a rear hit. This movement pattern challenges the conventional wisdom that uniform hyperextension in the neck occurs after rear-end crashes by producing a curve in the shape of a S of the neck in the sagittal plane [6].

3.3 Cervical Intervertebral Discs

Internal disruption of the intervertebral disc is a less commonly identified cause of cervicogenic headaches and neck discomfort reported in the literature on whiplash-associated disorders (WAD) [7]. Notably, whiplash-induced cervical disc injuries are not the same as common degenerative cervical spine disorders. Axial pain and referral patterns, such as headaches above the neck or upper back pain below the neck, are more common in patients with cervical discogenic pain than radiculopathy. In these cases, radiculopathy is rare, and as will be covered in the next sections, cervicobrachial symptoms are more frequently ascribed to thoracic outlet syndrome (TOS) or other contributory causes.

A hemorrhagic tear of the disc annulus at the adjacent vertebral endplate is linked to certain post-traumatic symptoms, as illustrated in Figure 2, which also illustrates the process of disc injury. An underlying cause of discogenic injury linked to whiplash-associated disorders (WAD), this injury is most likely caused by high tensile pressures. We call this condition RIM LESIONS.

It is important to note that disc injuries are relatively infrequent and can be difficult to confirm through auxiliary imaging techniques, such as cervical MRI. An early study conducted in the last century demonstrated that MRI was not effective in accu-

rately identifying sources of pain following cervical spine trauma [8], displaying limited sensitivity and specificity.

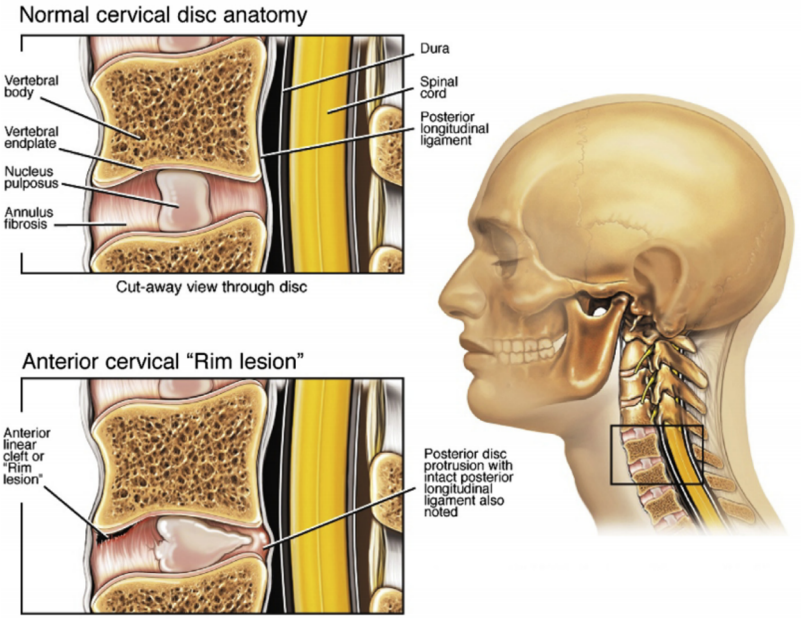

Fig. 2. Mechanism of disc injury

3.4 Cervical Intervertebral Discs

Acute muscular pain usually goes away quickly, but chronic pain can develop for a number of reasons. Direct strain from muscle attachments on the facet joint capsule, whiplash-induced chronic muscle atrophy that reduces neck strength and function, changes in proprioception brought on by damage to muscle spindles, and other causes of aberrant neuromuscular control are some of these factors.

3.5 Headache

Patients with whiplash-related disorders (WAD) most frequently describe headaches as their most common pain complaint. A few potential reasons for these headaches are as follows: ① headaches resulting from internal disturbances of the cervical intervertebral discs, specifically at the C2-3 level; ② headaches associated with posttraumatic temporomandibular joint (TMJ) disorder; ③ headaches caused by damage to the atlantoaxial (C1-2) joint and the greater or lesser occipital nerves, which are structures located above the C2-3 facet joint; ③ headaches caused by internal disturbances of the cervical intervertebral discs, specifically at the C2-3 level; and headaches associated with thoracic outlet syndrome (TOS), which may be caused by hy-

pertonic scalene muscles that apply abnormal forces to the facet joints (more on this later).

3.6 Cervicobrachial Pain

The bone components of the spinal column, first ribs, and sternum define the thoracic outlet. Patients with whiplash-associated disorders (WAD) may experience symptoms that spread into one or both shoulders and, in certain cases, into the upper extremities from the cervicothoracic area. When symptoms spread below the elbow, they frequently come with neurological problems that impact the hand, wrist, and forearm in many people. According to available research, degenerative cervical disc diseases like cervical spondylosis or radiculopathy are usually not the cause of this kind of discomfort in WAD patients. As an alternative, posttraumatic thoracic outlet syndrome (TOS) might be connected to it.

Inside the small region of the thoracic outlet, several structures situated just above the first rib and posterior to the clavicle compress the neurovascular bundle, causing a range of symptoms known as thoracic outlet syndrome (TOS). To refer to the range of conditions involving this anatomical area, the phrase "thoracic outlet syndrome" was employed.

Neurovascular impairment in the thoracic outlet occurs in three key anatomical locations: the pectoralis minor space, the costoclavicular area, and the scalene triangle. The main location of brachial plexus compression and the most frequently affected area in thoracic outlet syndrome (TOS) is the scalene triangle. This triangle's anterior border is formed by the anterior scalene muscle, which originates from the transverse processes of the third through sixth cervical vertebrae (C3–C6) and joins to the inner margins and superior surfaces of the first rib. The middle scalene muscle forms the posterior boundary. The transverse processes of the second through seventh cervical vertebrae (C2-C7) are where it begins and ends, attaching to the back of the first rib. The scalene triangle's upper limit is indicated by the superior border of the first rib. This little opening between the anterior and middle scalene muscles is traversed by the brachial plexus and subclavian artery trunks, while the subclavian vein travels anteromedial to this area. Abnormal cervical and first ribs may also contribute to greater compression inside the scalene triangle.

Although anomalies of ribs[9], muscular variations, and injuries are significant mechanisms of thoracic outlet syndrome (TOS), chronic inflammatory changes resulting from trauma are the most frequently observed mechanism in patients experiencing whiplash-associated pain[2]. The most common injury associated with neurogenic thoracic outlet syndrome (TOS) is a neck hyperextension-flexion injury, which can happen in a car crash.

TOS can be classified based on the affected structures into neurogenic TOS (nTOS), resulting from brachial plexus compression; venous TOS (vTOS), brought on by the subclavian vein being compressed; and arterial TOS (aTOS), stemming from subclavian artery compression[9]. Neurogenic TOS is the most prevalent, accounting for over 95% of TOS cases, while venous and arterial types represent 3% and 1%, respectively. Symptoms of nTOS may include pain, sensory disturbances, numbness,

and muscle weakness that are not confined to specific peripheral nerve distributions. Activities requiring arm elevation or sustained use frequently exacerbate these symptoms. Diagnosing TOS-related pain can be challenging due to the limited sensitivity of imaging and electrodiagnostic tests. While non-invasive manual therapy and rehabilitation are the mainstays of current treatment, severe instances may benefit from muscular trigger point stimulation, scalene muscle chemodenervation, or surgery. Additionally, intrinsic shoulder injuries should be considered as a potential source of pain, particularly after other mechanisms have been excluded.

4 Recent Advances in Treatment

For whiplash injuries, there are currently no extremely specialized treatments available. Depending on whether the patient is experiencing acute, subacute, or chronic pain, doctors should adjust the course of treatment. Recommended interventions may include encouraging an early return to activities, engaging in active exercises, and utilizing short-term analgesics along with nonsteroidal anti-inflammatory drugs (NSAIDs). It is crucial for patients to avoid prolonged use of cervical collars and to refrain from extended periods of bed rest unless a severe spinal injury is present [4].

Administration of corticosteroids for acute whiplash injuries has also been studied. If methylprednisolone sodium succinate is administered within eight hours of an accident, it has been demonstrated to enhance neurologic outcomes up to a year later. According to the suggested dosing regimen, a 30-mg/kg bolus is given over 15 minutes, followed by a maintenance infusion of 5.4 mg/kg every hour for 23 hours. According to recent research, further improvements in motor neurologic function and overall functional status may be possible if methylprednisolone treatment is prolonged by an additional 24 hours, for a total of 48 hours. The treatment of whiplash injuries has also demonstrated the effectiveness of this methylprednisolone regimen.

For managing chronic pain, a multimodal approach is currently advocated, consistent with the biopsychosocial model of chronic pain management. For instance, acute-phase management focuses on controlling inflammation and maintaining mobility (e.g., using NSAIDs and early exercise), whereas chronic cases often require more comprehensive strategies, such as combining physical therapy with psychological interventions (e.g., cognitive-behavioral therapy) to address persistent and complex pain. According to the majority of research, individuals with persistent neck pain benefit from a mix of exercise and other therapy methods [10]. In cases of chronic whiplash pain related to specific conditions such as Thoracic Outlet Syndrome (TOS), more advanced treatment options are available. For recalcitrant cases of TOS, these could involve surgical examination, chemodenervation of the scalene muscles, and muscle trigger point therapy.

5 Conclusion

The review is limited by the relatively small number of high-quality, evidence-based studies and emerging treatment modalities for whiplash-associated pain in recent

years. Consequently, it incorporates a limited selection of new research from around the globe. Much of the existing literature focuses on specific populations, with a significant gap in data pertaining to Asian and African groups, potentially introducing selection bias in the study conclusions. Future research should prioritize large-scale, multicenter prospective studies involving diverse populations affected by whiplash injuries to yield more robust evidence-based data.

Whiplash injuries are prevalent cervical spine injuries resulting from events that cause hyperextension and flexion of the head and neck. This review summarizes essential anatomical knowledge related to Whiplash-Associated Disorders (WADs) and investigates the potential pathophysiological mechanisms that underlie their various clinical manifestations, including cervicothoracic and cervicobrachial pain. The intricate pathogenesis of WADs poses significant challenges in managing both acute and chronic pain associated with these injuries. Therefore, to offer complete, long-term treatment for patients with WADs, doctors may need to work in conjunction with specialists in psychology, rehabilitation, and other related professions.

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