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Khalid Malik, K. C. Eseonu, D. Pang, B. Fakouri and J. R. Panchmatia

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Is Preexisting Cervical Degeneration a Risk Factor for Poor Prognosis in Whiplash-Associated Disorder?

KHALID MALIK, BSC, MB CHB, MRCS,[1] K. C. ESEONU, BSC, MSC, MB CHB, FRCS,[2] D. PANG, MB CHB, FRCA,[3] B. FAKOURI, MD, FRCS,[4] J. R. PANCHMATIA, MA, MPH, MB BCHIR, FRCS[5]

[1]*Speciality Registrar, Trauma and Orthopaedics,* [2]*Senior Spinal Fellow,* [3]*Consultant Pain Physician,* [4]*Consultant Spine Surgeon and Lead for Spine Surgery,* [5]*Consultant Spine Surgeon, Guy's & St. Thomas' Hospitals, London, United Kingdom*

ABSTRACT

Background: The term *whiplash* describes the acceleration-deceleration mechanism of injury to the cervical spine. Whiplash injuries present with a variety of clinical and psychological manifestations, collectively termed as *whiplash-associated disorders* (WADs). Although largely self-limiting, some patients may experience long-lasting symptoms. This review aimed to summarize the current literature regarding the predictive value of cervical degeneration in the prognosis of patients with WAD.

Methods: A comprehensive search of the literature was performed. Nine studies were identified, including 894 patients, with an age range between 16 and 76 years.

Results: A statistically significant association was found between moderate facet joint degeneration and nonrecovery. Although no association was established between isolated disc degeneration and nonrecovery, total cervical degeneration (facet joint + disc degeneration) was shown to correlate with nonrecovery.
All included studies demonstrated the lack of correlation between preexisting disc degeneration and clinical outcomes. Four studies showed a significant correlation between cervical degeneration and poor prognosis following whiplash injury. A significantly higher proportion of patients who remained symptomatic at 2 years following a whiplash injury had preexisting degenerative changes.

Conclusions: This review highlights the presence of significant variability in the existing literature concerning WAD in terms of study methodology, definitions of cervical degeneration, and outcome measures. Degenerative changes of the facet joint lead to alterations in its biomechanics. Several cadaveric, biomechanical, and clinical studies have demonstrated facet joints as a source of pain in patients with chronic WAD. We present moderate evidence to suggest that preexisting facet joint degeneration is a negative prognostic indicator for long-lasting symptoms in WAD. Conversely, preexisting disc degeneration is not associated with chronicity of WAD symptoms. We propose facet joint instability due to facet joint capsule rupture as a potential mechanism for nonrecovery. Further studies are needed to inform our knowledge of the long-term sequelae of WAD among patients with preexisting cervicalspine degeneration.

Cervical Spine

Keywords: whiplash, whiplash associated disorder, WAD, cervical facet joint, medial branch block

INTRODUCTION

The term *whiplash* is used to describe the acceleration-deceleration mechanism of injury to the cervical spine (c-spine), most frequently caused by rear-end or side impact in motor vehicle accidents (MVAs).[1] Patients with whiplash injuries present with a variety of clinical and psychological manifestations which may include neck pain and stiffness, paresthesia, dizziness, deafness, tinnitus, depression, sleep disturbance, and posttraumatic stress disorder. This group of symptoms was collectively termed as *whiplash-associated disorders* (WADs) by the Quebec Task Force in 1995.[1]

WAD is associated with a substantial socioeconomic burden worldwide, with a significant increase in incidence reported over the last 4 decades.[2] The incidence of WAD in Western Europe and North America is approximately 300 per 100 000 inhabitants each year.[3] The annual economic cost of WAD is estimated to be around £3.1 billion in the United Kingdom, US$3.9 billion in the United States and A$950 million in Australia.[4–6] Additionally, rehabilitation of patients with WAD is associated with the highest financial cost among all musculoskeletal injuries.[7]

It has been hypothesized that the sudden impact in whiplash injuries causes the c-spine to develop an

abnormal S-shaped curve due to simultaneous extension in the lower c-spine (LCS) and relative flexion in the upper c-spine (UCS).[8,9] Under physiological conditions, movement in the neck is initiated from the UCS to the LCS in an antegrade fashion. This pattern of movement is reversed in whiplash injuries, as the c-spine is forced to initiate movement from the LCS upwards.[10] Clinical studies have suggested that the mechanism of whiplash injuries may result in damage to the intervertebral discs in the LCS and injury to the facet joints in both the UCS and LCS.[11–15] Furthermore, injury to the muscles of the c-spine due to lengthening contraction has also been implicated in WAD.[16] Although WAD is largely a self-limiting condition, some patients may experience long-lasting and occasionally disabling symptoms.[17] The recovery rate among patient with WAD is variable, with over 60% reporting symptoms at 3 months following the initial injury and 50% experiencing neck pain at 12 months.[17–19] The etiology behind the persistence of symptoms in WAD remains largely unknown. Previous studies have identified the severity of initial symptoms, psychological factors, and medicolegal involvement as predictors for poor prognosis in WAD.[17,20–22]

Cervical spondylosis is reported as the most frequent radiological finding in WAD, yet its role as a predictive factor for nonrecovery in WAD remains unclear.[23] The terms cervical spondylosis and cervical degeneration are often used interchangeably. Cervical spondylosis is the most common disorder of the c-spine caused by age-related degeneration of the intervertebral discs and facet joints.[24] Although cervical spondylosis is seen in 95% of the population by the age of 65 years, the majority of these individuals remain asymptomatic.[25] Symptomatic patients may present with neck pain, cervical radiculopathy, and very rarely with cervical myelopathy.[26] In this review, we aim to summarize the current literature regarding the predictive value of cervical degeneration in the prognosis of patients with WAD.

METHODS

A comprehensive search of PubMed, MEDLINE, and Embase was conducted using the terms *whiplash*, *whiplash associated disorder*, *WAD*, *cervical spondylosis*, *cervical degeneration*, *disc degeneration*, and *facet degeneration*. We included all studies published in the English literature that investigated the association between preexisting cervical spondylosis (intervertebral disc or facet joint degeneration) and outcomes following whiplash injuries.

RESULTS

Nine studies were identified: 6 prospective cohort and 3 retrospective comparative studies.[27–35] These studies included 894 patients, with an age range from 16 to 76 years. The characteristics of the included studies are summarized in the Table.

Facet Joint and Disc Degeneration

Rydman et al[27] studied the association between preexisting cervical degeneration (facet joint and disc degeneration identified on computed tomography [CT] scan) and self-perceived nonrecovery at 6 months following whiplash injury. Outcome measures included self-reported nonrecovery on a binary scale and level of neck pain on a scale of 0 to 10. A statistically significant association was found between moderate facet joint degeneration and nonrecovery, odds ratio (OR) 6.7 (95% confidence interval [CI]: 1.9–24.3). Although no association was established between isolated disc degeneration and nonrecovery, total cervical degeneration (facet joint + disc degeneration) was shown to correlate with nonrecovery, OR 6.2 (95% CI: 2.0–19.0). Both facet joint and disc degeneration were associated with a higher level of residual neck pain at follow-up ($P = .01$).

Disc Degeneration

Three studies investigated the relationship between preexisting disc degeneration and clinical outcomes in whiplash injuries.[28–30] All included studies demonstrated the lack of correlation between preexisting disc degeneration and clinical outcomes in WAD. In the study by Chung et al,[28] outcomes in patients with advanced disc disease were compared to those with no or mild disc degeneration. Radiological assessment was undertaken using magnetic resonance imaging (MRI). Clinical outcomes were measured using the visual analog scale, neck disability index and short form-36 (physical and mental) at 3 months, 6 months, and 12 months following the initial injury. There were no statistically significant differences in the clinical outcomes between the 2 groups at each visit.

In the prospective trial by Kongsted et al,[29] the authors evaluated the correlation between abnormal

Table. Summary of studies included.

Study	Study Type	No. of Patients	Age Range	Follow-up	Imaging	Type of Degeneration	Clinical Outcome	Findings
Rydman 2019[27]	Prospective cohort	121	16–70 y	6 mo	CT	Facet joint degeneration	Self-perceived nonrecovery	• (16/25) 69.6% Nonrecovery with moderate facet joint degeneration versus 23.6% (13/54) if no degeneration • Increased risk of nonrecovery with total cervical degeneration (facet joint degeneration + disc degeneration) (adjusted OR 6.2 [95% confidence interval: 2.0–19.0]) • Mean pain level was associated with facet joint degeneration ($P = .01$) and disc degeneration ($P = .01$)
Chung 2017[28]	Retrospective comparative	97	18–60 y	3 mo, 6 mo, 12 mo	MRI	Disc degeneration	VAS, neck disability index, SF-36 physical, SF-36 mental	• Advanced disc degeneration = 45, no or mild disc degeneration = 52 • No statistically significant difference between groups at 3, 6, or 12 mo • Clinical presentation and prognosis of WAD were not affected by preexisting disc degeneration
Kongsted 2008[29]	Prospective cohort	178	18–70 y	3 mo, 12 mo	MRI	Foraminal spinal stenosis Disc degeneration	Neck pain, Copenhagen neck functional disability scale, working ability	• Moderate-severe preexisting degeneration = 56 • Intensity of neck pain and radiating arm pain at baseline did not differ between the participants with preexisting degeneration • Preexisting degeneration was not associated with the 3-mo outcome • At 1 y, moderate-severe preexisting degeneration was associated with reduced risk of lasting pain • Preexisting degeneration not associated with prognosis
Borchgrevink 1995[30]	Prospective cohort	52	18–66 y	3 mo, 6 mo, 12 mo, 24 mo	MRI	Disc + spondylosis	Neck pain, neck stiffness, headache	• 12 Disc changes, 7 spondylosis • Preexisting spondylosis reported significantly more headache at the 3- and 6-mo follow-ups ($P < .05$ and $P < .01$, respectively) • Disc abnormalities and spondylosis had more neck pain at 3 mo • No difference in neck pain at 12 mo and 24 mo
Radanov 1995[31]	Prospective cohort	117 Asymptomatic at 2 y = 96, symptomatic at 2 y = 21	19–51 y	24 mo	XR	Degenerative changes (no specifics)	Neck pain, headache, shoulder pain, fatigue, anxiety, sleep disturbance, back pain, noise sensitivity, impaired concentration, blurred vision, irritability, dizziness, forgetfulness, dysphagia, jugular pain,	• 18/96 (19%) Asymptomatic patients had degenerative changes • 9/21 (43%) in symptomatic group had degenerative changes , $P < .017$
Hildingsson 1990[32]	Prospective cohort	93	17–67 y	25 (6–43) mo	XR	Degenerative spondylosis	Continuous pain and inability to return to previous work	• 7 had degenerative spondylosis on presentation. • No correlation between XR and symptoms

Table. Continued.

Study	Study Type	No. of Patients	Age Range	Follow-up	Imaging	Type of Degeneration	Clinical Outcome	Findings
Maimaris 1988[33]	Retrospective comparative	102 (67 symptomatic, 35 asymptomatic)	17–72 y	26 mo (18–30 mo)	XR	Osteoarthritis	Neck pain, neck stiffness, shoulder pain, headaches, interscapular pain, referred symptoms	• 48% of symptomatic patients had degenerative changes versus 12% asymptomatic
Miles 1988[34]	Retrospective comparative	73	17–73 y	24 mo	XR	Degenerative changes (no specifics)	Neck pain, abnormal neurology, neck movement	• 15/73 (20.6%) had degenerative changes at the time of injury • 8 (53.3%) had symptoms ($P < .025$) and 5 (33.3%) had abnormal neurology ($P < .01$) • Significant reduction in neck ROM ($P < .01$)
Norris 1983[35]	Prospective cohort	61	19–76 y	Group 1 = 19.7 ± 6 mo, group 2 = 23.9 ± 5.5 mo, group 3 = 24.7 ± 7.3 mo	XR	Degenerative spondylosis – no specifics	Neck pain, Headache, Paresthesia, reduced neck ROM, neurological signs	Degenerative changes found in • 26% Group 1 – symptoms but normal clinical exam • 33% Group 2 – symptoms, reduced ROM, no neurology • 40% Group 3 – symptoms, reduced ROM, objective neurology

Abbreviations: CT, computed tomography; MRI, magnetic resonance imaging; ROM, range of motion; SF-36, short form-36; VAS, visual analog scale; WAD, whiplash-associated disorder; XR, x-ray.

MRI findings and persistent symptoms at 1 year following injury. They defined *mild preexisting degeneration* as "reduced disc height and/or signal" and *moderate-severe preexisting degeneration* as "foraminal stenosis and/or nontraumatic bulge/protrusion with or without compression of the spinal cord and/or modic changes." Clinical outcomes included neck pain, radiating arm pain, headache, Copenhagen neck functional disability scale measurements, and self-reported working ability. Moderate-severe preexisting changes were identified in 56/178 (31.5%) of participants. No association was found between preexisting degeneration and clinical outcomes at 3 months. Moderate-severe preexisting disc degeneration was associated with reduced risk of persistent neck pain at 12 months. It is important to note that in their study, patients with significant neck pain before the MVA were excluded from the study leading to a significant risk of selection bias.

In the prospective MRI-based study of 52 patients by Borchgrevink et al,[30] disc abnormalities and cervical spondylosis were considered as separate entities. *Disc abnormalities* were defined as "disc protrusions or reduced disc signal" on the MRI, whereas a clear definition for cervical spondylosis was not provided. Patients were split into 4 groups based on the MRI findings: no abnormal findings, postural abnormalities, preexisting spondylosis, and disc abnormalities. Patients with disc abnormalities and those with preexisting spondylosis reported significantly more pain at 3 months. However, at 12 months and 24 months, there were no significant differences between the groups in terms of neck pain, neck stiffness, or headaches.

Cervical Degeneration, Spondylosis, and Osteoarthritis

Radanov et al[31] showed that a significantly higher proportion of patients who remained symptomatic at 2 years following a whiplash injury had preexisting degenerative changes identified on the initial plain radiographs: 43% (9/21) in the symptomatic groups compared to 19% (18/96) in the asymptomatic group. Similarly, Maimaris et al[33] reported that at a 2-year follow-up, the proportion of patients with osteoarthritis of the c-spine seen on the initial radiographs in the symptomatic group was 48% (17/35), compared to 12% (8/67) in the asymptomatic group ($P < .001$).

Miles et al[34] found that 21% (15/73) of patients who sustained soft tissue injury to their c-spine had preexisting degenerative changes on the initial radiographs. When compared to those with normal radiographs, these patients had significantly reduced range of movement ($P < .01$) and higher rate of ongoing symptoms ($P < .025$) at 2 years. Norris and Watt[35] showed that following rear-end MVA, presence of objective neurology, neck stiffness, muscle spasm, and preexisting degenerative spondylosis were associated with poor prognosis. In their prospective study, patients were allocated to 3 different groups based on the severity of their symptoms. (group 1 = symptomatic, normal clinical examination; group 2 = symptomatic, reduced range of movement of neck, normal neurology; group 3 = symptomatic, reduced range of movement of the neck, abnormal neurology). The more symptomatic groups had a higher number of patients with preexisting degenerative spondylosis on radiographic examination (group 1 = 26%, group 2 = 33%, group 3 = 40%).

Conversely, Hildingsson and Toolanen[32] failed to show an association with cervical spondylosis and clinical outcomes following whiplash. They analyzed the relationship between 17 factors (including radiographic examination of the c-spine) and presence of chronic symptoms in 93 patients who sustained soft tissue injury of the c-spine following an MVA. At an average follow up of 2 years, persistent pain and inability to return to previous work were not associated with any of the studied factors. Seven patients had degenerative spondylosis on the initial radiographs.

DISCUSSION

This review highlights the presence of significant variability in the existing literature concerning WAD in terms of study methodology, the definition of cervical degeneration, and outcome measures. Disc degeneration was assessed with MRI in 3 studies, whereas only 1 study used CT scanning to assess facet joint arthrosis and disc degeneration.[27–30] All included studies published before 1995 used plain radiographs and did not explicitly define cervical spondylosis, using the terms *degeneration*, *spondylosis*, and *osteoarthritis* synonymously.[31–35] Furthermore, there was significant heterogeneity among the included studies in accounting for confounders such as smoking history, body mass index, occupation, level of education, and variables relating to MVAs.

The process of cervical degeneration begins with desiccation of the nucleus pulposus.[36] With increasing age, the nucleus pulposus loses its intradiscal pressure and hence its elasticity.[37] As the nucleus pulposus becomes more fibrous and smaller in size, more of the mechanical load is transferred to the annulus fibrosus leading to the development of clefts and fissures, resulting in disc bulging and loss of intervertebral height.[38] A kyphotic deformity of the c-spine may occur due to the initial loss of intervertebral height ventrally, which creates a positive-feedback cycle as more forces are applied to the ventral aspect of the vertebral bodies under physiological load.[24] These changes cause the posterior longitudinal ligaments and peripheral fibers of the annulus fibrosus to be dissected away from the vertebral endplates leading to degenerative intervertebral instability (degenerative spondylolisthesis).[24]

Loss of intervertebral disc height leads to a greater load transfer to the uncovertebral and facet joints leading to facet joint hypertrophy and osteophyte formation. Facet joints (also known as zygapophyseal joints) are true synovial joints. Although facet joint degeneration is typically considered as a secondary process associated with disc degeneration, it may also occur independently.[39] Additionally, disc degeneration and intervertebral instability may also cause the ligamentum flavum to become hypertrophic.[40] The combination of these degenerative changes may result in the narrowing of the central spinal canal, lateral recess, and intervertebral foramina. Neck pain in cervical spondylosis is thought to arise from ligamentous laxity, disc herniation, and degenerative changes of the synovial facet joints.[24]

Several cadaveric, biomechanical, and clinical studies have demonstrated facet joints as a source of pain in patients with chronic WAD.[10,12,41,42] During the pathological movement of the c-spine in the whiplash mechanism, the forceful impact of the inferior articular process of the upper vertebra against the superior articular process of the inferior vertebra may lead to damage to the articular cartilage, intra-articular hemorrhage, and rupture of the intra-articular meniscoids.[10] During whiplash, the facet joints undergo compression that exceeds physiological limits followed by abnormally high strains in the facet capsule.[12] Simulated whiplash in animal studies has demonstrated that

activation of the nociceptive pathway occurs secondary to stretch of the facet joint capsule.[41,42] Clinical studies using medial branch blocks as a diagnostic test found that the prevalence of pain originating from the zygapophysial joint in chronic WAD was about 50%.[13,43] Similarly, percutaneous radiofrequency neurotomy of the cervical zygapophyseal joints in symptomatic patients following whiplash injury showed successful outcome in 70% and pain relief (greater than 50%) lasting for a median of 263 days following treatment.[44] Furthermore, repeat neurotomy procedures are also proven to provide pain relief in recurrent cases.[45,46]

Only 1 study (Rydman et al[27]) evaluated both the intervertebral discs and facet joints in the context of cervical degeneration. The same study used CT scanning for the radiological assessment of the c-spine, which is shown to be superior to MRI and plain radiography in detecting facet joint degeneration and has relatively higher levels of interrater agreement in the assessment of facet joint degeneration.[47] This well-executed study demonstrated that moderate facet joint arthrosis was an independent risk factor for nonrecovery in WAD.[27] Additionally, 4 x-ray based studies also found that a significantly higher proportion of patients with preexisting cervical degeneration remained symptomatic following whiplash injuries, though these studies did not explicitly define cervical degeneration or spondylosis and did not differentiate between degenerative disc disease and facet joint degeneration.[31,33–35]

Degenerative changes of the facet joint lead to alterations in its biomechanics. Thinning of the articular cartilage and hypermobility of the spinal segments result in increased laxity of the facet joint capsule.[48] The collagen fibers of the facet joint capsule may rupture while resisting the higher tensile stress due to hypermobility of the diseased spinal segment. These ruptured fibers are replaced by scar tissue, hence resulting in loss of their mechanical properties.[49] It is plausible that whiplash injuries to a c-spine with preexisting degenerative changes may result in increased axial and rotational movement, resulting in a more severe articular and capsular injury compared to a normal c-spine. The existing literature suggests that whiplash trauma in the degenerative c-spine may represent a catalyst for clinical manifestations of WAD, potentially leading to a higher risk of nonrecovery. Although Rydman et al[27] showed a significant association between moderate facet joint degeneration and nonrecovery in WAD, no such association was demonstrated for severe facet joint degeneration. This may be explained by the fact that in advanced degeneration, the vertebral bodies may undergo ankylosis, resulting in a paradoxical increase in c-spine stability. This phenomenon may also explain why some patients with moderate myelopathy experience improvement in symptoms during the advanced stages of cervical spondylosis.[24,50]

Although cervical disc degeneration is considered to be an important source of neck pain, it affects 80% of the population over the age of 60 without causing any symptoms.[51] Cervical discs are innervated by sinuvertebral nerves posteriorly, the vertebral nerve laterally, and sympathetic trunks anteriorly.[52,53] In disc degeneration, annular tears may stimulate nociceptors in the disc tissue. Disc degeneration also leads to elevated levels of proinflammatory cytokines secreted by the intervertebral discs that facilitate matrix degradation, chemokine production, and changes in cell phenotype.[54] These cytokines promote nerve growth factor expression, resulting in nerve fiber ingrowth into an otherwise aneural tissue.[54] Loss of structural integrity due to matrix degradation in the degenerative disc may also cause mechanical stimulation, which in turn could generate an exaggerated response to innocuous stimuli, resulting in peripheral sensitization.[55]

It has been previously hypothesized that preexisting cervical disc degeneration may be associated with increased severity and duration of symptoms in WAD due to sensitization of nociceptors in the cervical disc.[56] In the present review, all 4 studies that investigated the impact of disc degeneration on prognosis in WAD failed to show a significant correlation between preexisting cervical disc degeneration and chronicity of WAD symptoms.[27–30] In another cross-sectional study conducted by Fujimura and Matsumoto[57] between 1993 and 1996, MRI of the c-spine was performed in 506 patients after whiplash injury. The authors found no statistically significant difference in disc space narrowing, reduced signal of the intervertebral disc, and posterior disc protrusion between WAD patients and healthy volunteers.[57] Their 10- and 20-year follow-up study showed that although progression of the cervical disc degeneration was seen on MRI in over 95% of patients, it was not associated with clinical symptoms of WAD. Fujimura and Matsumoto concluded that following whiplash injuries, the progression of cervical disc degenera-

tion on MRI was attributable to physiological ageing, rather than posttraumatic sequelae.[58,59]

CONCLUSION

This review highlights the paucity of good-quality studies on this topic and significant heterogeneity in the definition of cervical degeneration and study methodology. The importance of the facet joint in the pathogenesis of WAD is well established. We present moderate evidence to suggest that preexisting facet joint degeneration is a negative prognostic indicator for long-lasting symptoms in WAD. Conversely, preexisting disc degeneration is not associated with chronicity of WAD symptoms. We propose axial and rotational facet joint instability due to facet joint capsule rupture as a potential mechanism for nonrecovery in WAD in diseased spinal segments. The traumatic nature of this injury makes prospective study design problematic. Further longitudinal studies are needed to inform our knowledge of the long-term sequelae of WAD among patients with preexisting c-spine degeneration.

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Corresponding Author: K. C. Eseonu, Guys and St Thomas' Hospital, Westminister Bridge Road SE1, London, UK. Phone: 02071887188 Email: Kelechi.eseonu@doctors.org.uk.

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