OCULAR MANIFESTATIONS OF WHIPLASH INJURY

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ABSTRACT

Introduction: Whiplash or indirect injuries to the neck are a common occurrence because of motor vehicle collisions, in which the frequency of ocular complications is largely unknown.

Aim: We aim to review the ocular manifestations, the utility of relevant diagnostic tests, the management, prognosis, and sequelae of whiplash-related ocular complications.

Methods: A comprehensive literature search was conducted on Medline®, PubMed®, Google Scholar™, and Cochrane databases. Only articles published or translated into the English language from 1985–2015 were considered and key data from each paper were extracted and evaluated.

Results: The authors’ search yielded 41 articles. Blurring of vision was the most common presenting symptom. Other common presenting symptoms included words and objects moving within the visual field as well as difficulty in concentration. Ocular findings involved: disturbed eye movements; vestibulo-ocular reflex and cervico-ocular reflex dysfunction; anterior segment abnormalities such as accommodative and convergence dysfunctions and relative afferent papillary defect; and posterior segment abnormalities such as macular oedema and foveal lesions. The treatment for ophthalmic complications of whiplash injuries is mainly conservative and revolves around neck physiotherapy and oculomotor rehabilitation. The prognosis of the ocular injury depends on the ocular findings as well as the interval between whiplash injury and treatment initiation.

Conclusion: Whiplash injuries occur commonly in motor vehicle accidents. While patients may present asymptptomatically, a myriad of ophthalmic complications may be detected. Prognosis for ocular complications depends mainly on the severity of complication(s), the time lapse between injury and detection of complication(s), and the initiation of treatment. Ophthalmologists and physicians should be aware and vigilant towards patients following whiplash injury as a small minority of patients may have poor visual outcome and quality of life without treatment and follow-up.

Keywords: Whiplash, closed head injury, flexion-extension neck injury, cervical spine injury, ocular, eye, visual.
INTRODUCTION

Whiplash injuries are the most common injuries encountered in motor vehicle accidents. The term ‘whiplash’ has been described in medical literature as the mechanism in which the head of an occupant is forced backwards with forward recoil, predominantly in a rear-end or side-on collision.1,2 The acceleration-deceleration transfer of energy to the neck can result in extensive soft tissue and/or bone injury which in turn may lead to a variety of clinical manifestations known as whiplash-associated disorders (WADs). They can be classified by the severity of signs and symptoms into Grades 0–4 according to the Quebec Task Force Classification (QTFC) (Table 1).3 Patients commonly complain of pain or stiffness in the neck, headache, vertigo, backache, or paraesthesia in the limbs. Although uncommon, ophthalmic and oculomotor complications may also present.

Common ocular manifestations include cervico-ocular reflex (COR) and vestibulo-ocular reflex (VOR) dysfunction, reduced smooth eye pursuit, and nerve palsies. Ocular and oculomotor complications arising from WADs can lead to long-term deficits in visual and systemic functioning. The purpose of this article is to review the commonly reported whiplash-associated ocular complications, their management, prognosis, and sequelae.

MATERIALS AND METHODS

A comprehensive literature search was conducted using Medline®, PubMed®, Google Scholar™, and Cochrane databases using the keywords ‘whiplash’, ‘ocular’, ‘eye’, ‘visual’, ‘closed head injury’, ‘flexion-extension neck injury’, and ‘cervical spine injury’. Only studies with abstracts and full-texts published in English from 1985–2015 were included. A hierarchical approach was adopted when selecting articles; relevant articles were initially selected based on their titles and abstracts. The full texts of these articles were then obtained and reviewed in more detail. Articles published before 1985, whiplash injuries because of non-accidental injuries in children, and whiplash injuries associated with penetrating eye injury were excluded from our study to ensure that the mechanism of injury was not confounded by another source. Forty-one studies were eventually collated, comprising 7 observational studies, 1 review article, 21 case series/reports, and 12 case-control studies.

DISCUSSION

Diagnosis of Whiplash Injury

The diagnosis of whiplash injury is made clinically based on the patient’s or witness’ account of the vehicle accident. Frequently, the mechanism of injury can predict the possibility of a whiplash injury. A high-impact, rear-end, or side-on collision is likely to result in a whiplash injury. The common symptoms of whiplash injury include neck pain, neck stiffness, headache, and memory or concentration disturbances. Radiological investigations usually do not identify abnormalities in the acute setting.4

Common Ocular Symptoms of Whiplash Injuries

Ocular symptoms of whiplash injuries may be related to oculomotor control deficits, problems with gaze stability, and head-eye co-ordination disturbances (Table 2). Treleaven and Takasaki5 compared ocular symptoms reported by individuals with WAD and neck pain, and those with idiopathic neck pain. It was found that individuals with WAD had a higher visual complaints index of mean 32.8±26.0 compared to a visual complaints index mean of 18.8±20.0 in those with idiopathic neck pain.

Table 1: The Quebec Task Force Classification (QTFC) of whiplash-associated disorders.3

<table>
<thead>
<tr>
<th>Grade</th>
<th>Classification</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>No complaint about neck pain and no physical signs</td>
</tr>
<tr>
<td>1</td>
<td>Neck complaint of pain, stiffness, or tenderness only, no physical signs</td>
</tr>
<tr>
<td>2</td>
<td>Neck complaint and musculoskeletal signs; musculoskeletal signs include a reduced range of movement and point tenderness</td>
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<tr>
<td>3</td>
<td>Neck complaint and neurological signs; neurological signs include decreased or absent deep tendon reflexes, weakness, and sensory deficits</td>
</tr>
<tr>
<td>4</td>
<td>Neck complaint and fractures or dislocations</td>
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</tbody>
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Individuals with WAD also had a significantly greater magnitude of complaints of blurred vision compared to individuals with idiopathic neck pain (55% versus 32%), words or objects moving (48% versus 24%), and needing to concentrate (72% versus 60%). Other symptoms include visual fatigue, sensitivity to light, double vision, difficulty judging distance, red eyes, and dizzy reading. The report also confirmed that double vision and red eyes did not appear to be associated with neck pain as there were no differences in reported prevalence and magnitude of these symptoms in subjects with neck pain when compared to asymptomatic individuals. Burke et al.⁶ also reported three cases of decreased stereocuity, of which two were asymptomatic. These symptoms can greatly impact the quality of life of individuals.

**Ocular Complications Involving Eye Movements**

**Nerve palsies**

Out of the 41 articles, 13 documented cranial nerve palsies, especially those that control the eye movement, namely the oculomotor (CN3), trochlear (CN4), and abducens (CN6) nerves. Abducens nerve palsy was the most common injury sustained, as described in 13 articles.⁷-¹⁹ One retrospective study⁷ identified 17 (28.3%) cases of CN3 and 20 (33.3%) cases of CN4 palsies in the 60 cases examined. Additionally, combined nerve palsies constituted five cases (8.3%). In the 13 articles, bilateral abducens palsies were present in 12 patients,⁸,⁹,¹¹-¹⁵,¹⁷-¹⁹ unilateral left abducens palsy occurred in 1 patient¹⁰ while the rest were unspecified.¹⁰,¹⁶ The abducens nerve is most commonly involved as it runs a long intracranial course and is vulnerable to injury. It can be injured at the following areas during its course:¹⁰

- As it crosses the petrous part of the temporal bone
- Stretched as it passes from the brainstem to its entry to the dura at the basilar process by downward and forward displacement of the brainstem during whiplash injury
- Fracture of the cranial floor
- Meningeal oedema
- Inflammation within the skull base

<table>
<thead>
<tr>
<th>Common ocular symptoms</th>
<th>Common ocular signs</th>
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<tbody>
<tr>
<td><strong>Blurred vision</strong></td>
<td><strong>Nerve palsies</strong></td>
</tr>
<tr>
<td><strong>Words or objects moving</strong></td>
<td><strong>Superior oblique muscle deficit</strong></td>
</tr>
<tr>
<td><strong>Needing to concentrate</strong></td>
<td><strong>Internuclear ophthalmoplegia</strong></td>
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<tr>
<td><strong>Common ocular signs</strong></td>
<td><strong>Saccadic eye movement deficits</strong></td>
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<tr>
<td><strong>Common ocular symptoms involving eye movements</strong></td>
<td><strong>Smooth pursuit deficits</strong></td>
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<tr>
<td><strong>Nerve palsies</strong></td>
<td><strong>Ocular complications involving the anterior segment of the eye</strong></td>
</tr>
<tr>
<td><strong>Superior oblique muscle deficit</strong></td>
<td><strong>Hyphema</strong></td>
</tr>
<tr>
<td><strong>Internuclear ophthalmoplegia</strong></td>
<td><strong>Traumatic mydriasis</strong></td>
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<tr>
<td><strong>Saccadic eye movement deficits</strong></td>
<td><strong>Relative afferent papillary defect</strong></td>
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<tr>
<td><strong>Smooth pursuit deficits</strong></td>
<td><strong>Horner’s syndrome</strong></td>
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<tr>
<td><strong>Ocular complications involving the anterior segment of the eye</strong></td>
<td><strong>Increased intraocular pressure</strong></td>
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<tr>
<td><strong>Hyphema</strong></td>
<td><strong>Ocular complications involving the posterior segment of the eye</strong></td>
</tr>
<tr>
<td><strong>Traumatic mydriasis</strong></td>
<td><strong>Macular oedema</strong></td>
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<tr>
<td><strong>Accommodative and convergence dysfunction</strong></td>
<td><strong>Foveal lesions</strong></td>
</tr>
<tr>
<td><strong>Relative afferent papillary defect</strong></td>
<td><strong>Commotio retinae</strong></td>
</tr>
<tr>
<td><strong>Horner’s syndrome</strong></td>
<td><strong>Macular detachment</strong></td>
</tr>
<tr>
<td><strong>Increased intraocular pressure</strong></td>
<td><strong>Optic disc oedema</strong></td>
</tr>
<tr>
<td><strong>Ocular complications involving the posterior segment of the eye</strong></td>
<td><strong>Retinal detachment</strong></td>
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<tr>
<td><strong>Macular oedema</strong></td>
<td><strong>Retinoschisis</strong></td>
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<tr>
<td><strong>Foveal lesions</strong></td>
<td><strong>Foveomacular retinitis</strong></td>
</tr>
<tr>
<td><strong>Commotio retinae</strong></td>
<td><strong>Retinal epithelial tears</strong></td>
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<tr>
<td><strong>Macular detachment</strong></td>
<td><strong>Vestibulo-ocular reflex and cervico-ocular reflex dysfunction</strong></td>
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Table 2: Common ocular clinical symptoms and signs associated with whiplash-associated disorders.
Delayed presentation of abducens nerve palsy was reported in two papers\textsuperscript{17,18} with an interval of 3 days to 6 weeks. Delayed presentations have been postulated to be because of vasospasm or injury to branches of the meningodorsal artery causing nerve ischaemia.\textsuperscript{20} The other cause could be due to surrounding tissue oedema, a mechanism akin to delayed traumatic facial palsy.\textsuperscript{21} Associated injuries to the cervical spine, diagnosed using imaging studies, were also reported in six cases.\textsuperscript{8,13-15,18} Abducens nerve palsy can also relay false localising signs due to raised intracranial pressure. In the 13 articles studied, raised intracranial pressure causing bilateral abducens palsy was only suggested in one patient,\textsuperscript{17} with the majority reporting unremarkable computed tomography (CT) brain findings. Of the 79 cases of nerve palsies reported, 67 reported diplopia while the rest were asymptomatic or had unspecified ocular symptoms.

**Other cranial nerve involvement**

One article documented the presence of unilateral hypoglossal nerve palsy.\textsuperscript{19} Steroids were used as treatment in three cases with isolated bilateral abducens palsy. Of these three cases, one resolved completely, one resolved partially, and one remained persistent after 1 year, 6 months, and 3 months, respectively. Six cases received treatment for associated cervical spine injury, all of which had abducens palsy that resolved. Others had no treatment or treatment that was unspecified.

Only 12 patients\textsuperscript{8-15,17-19} were reported to be on regular follow-up, with the duration of follow-up ranging from 3 months to 1 year post-injury (mean: 6.5 months). Of these 12 patients, 7 had abducens palsy completely resolved (mean follow-up: 7.3 months), 3 had abducens palsy partially resolved (mean follow-up: 7 months), and 2 patients did not improve over time (mean follow-up: 3 months). Mutyala et al.\textsuperscript{22} reported a spontaneous improvement rate of 27\% in unilateral traumatic sixth-nerve palsy and 12\% in bilateral traumatic sixth-nerve palsy. However, Holmes et al.\textsuperscript{23} reported an overall spontaneous recovery rate of 73\%. Spontaneous recovery was more frequent in unilateral cases (84\%) than in bilateral cases (38\%).\textsuperscript{7}

**Extraocular Muscle Involvement**

Burke at al.\textsuperscript{6} described three cases of superior oblique muscle involvement. Two cases had superior oblique muscle paresis while one case had an underaction of the superior oblique muscle. The latter case was also described to have other ocular signs such as hypometric horizontal saccades, a reduction in convergence, and cogwheel pursuits. The aetiology of the cases is uncertain but has been suggested to be because of axonal shearing of the trochlear nerve and stretching forces at the moment of impact. All three cases recovered within 1 year of the accident.

**Other Ocular Complications**

Jammes\textsuperscript{24} reported a case of internuclear ophthalmoplegia occurring immediately following a whiplash injury in a 58-year-old lady. Extensive recovery was achieved after a 1-year follow-up.

**Oculomotor Dysfunction**

Deficits in saccadic eye movements and smooth pursuit eye movements were noted.\textsuperscript{25-34} Electro-oculography, electronystagmography, and a smooth pursuit neck torsion test were some of the common investigations conducted to detect the deficits. Storaci et al.\textsuperscript{25} reported that oculomotor rehabilitation conducted within 3 months of injury could help to improve pursuit eye movement deficits. Kongsted et al.\textsuperscript{33} suggested that other symptoms, such as persistent neck pain, could help to predict prognostic outcome of oculomotor dysfunction. Persistent neck pain was found to be associated with reduced smooth pursuit performance at 1-year follow-up.

**Ocular Complications Involving the Anterior Segment of the Eye**

**Hyphema and traumatic mydriasis**

Mustafa et al.\textsuperscript{35} in their case report accounted for the presence of hyphaema in a 25-year-old gentleman who presented with a visual acuity of light perception. Visual acuity improved to counting fingers after 4 months. The gentleman also presented with extensive commotio retinae with associated gross macular detachment, fine vitreous haemorrhage, and traumatic mydriasis.

**Pupillary dysfunction**

Stimulation to the sympathetic pathway due to injury to the cervical neck can result in pupillary dysfunction. Relative afferent papillary defect was also described in one case by Williams et al.\textsuperscript{36} Accommodation and convergence dysfunction were described in 19 and 10 cases respectively by Burke et al.\textsuperscript{6} and Brown.\textsuperscript{37}
Horner’s syndrome

Horner’s syndrome was noted in two case reports. Uzan et al.\(^8\) accounted for the presence of a left Horner’s syndrome associated with bilateral abducens nerve palsy in a 42-year-old gentleman. Jammes\(^24\) presented a case of right Horner’s syndrome in a 58-year-old lady who also suffered bilateral internuclear ophthalmoplegia. Due to the relationship between the anatomy of structures of the neck and the cervical sympathetic trunk, any injury to the cervical region following a whiplash mechanism can account for Horner’s syndrome.

Increased intraocular pressure

Wei and Spaeth\(^38\) accounted for four cases of transient increase in intraocular pressure in patients with an established background of open angle glaucoma. Intraocular pressure increased by the range of 5–19 mmHg. All four patients required an increase in dosage of topical medication or an additional topical medication. Intraocular pressure decreased to pre-accident levels in all cases within 1 year of the accident.

Ocular Complications Involving the Posterior Segment of the Eye

Complications involving the posterior segment of the eye may be attributed to shearing stresses generated from the acceleration-deceleration forces, raised intracranial and intraocular pressures, and the breakdown in the blood-retinal barrier.

Macular oedema

Macular oedema has been described immediately after whiplash injury\(^39,40\). The swelling can be symptomatic and persistent at 6-month follow-up sessions\(^39\) or resolve completely after 1 year.\(^40\)

Foveal lesions

Foveal lesions, which include perifoveal lesions with paracentral scotoma, have been described immediately following a whiplash injury. The effectiveness of optical CT to detect lesions at the vitreoretinal interface related to whiplash maculopathy has been emphasised.\(^39\) Small yellowish lesions over the fovea with loss of normal foveal reflex were found in fundoscopy on a patient who had complained of decreased central vision 1 year after a whiplash injury.\(^41\) There were also persistent signs and symptoms at 6 months and 20 months later. Williams et al.\(^36\) described swelling of the foveal zone and a foveal pit. It is proposed that the foveolar pit is due to selective destruction of the central photoreceptors, which may occur secondary to physical or toxic agents, and that no treatment had been proven effective.

Other posterior segment complications

Other less common posterior segment complications include vitreous detachment,\(^6\) vitreous haemorrhages,\(^35\) commotio retinae,\(^35\) macular detachment,\(^35\) Terson syndrome (vitreous haemorrhage associated with subarachnoid haemorrhage),\(^36\) optic disc oedema,\(^36\) relative afferent papillary defect,\(^36\) retinal detachment,\(^42\) retinoschisis,\(^42\) foveomacular retinitis,\(^43\) dot-blot haemorrhages,\(^14\) and retinal epithelial tears.\(^45\)

Vestibulo-ocular reflex and cervico-ocular reflex dysfunction

The COR works with the VOR and optokinetic reflexes to control the extra-ocular muscles creating clear vision with head movement.\(^46,47\) Damaged cervical afferent input, both proprioceptive and nociceptive, from injury to the cervical region or injury to the brainstem during a whiplash injury can result in deficits. Neck pain resulting in restricted neck movements has also been described to affect oculomotor performance.\(^30\) Four articles\(^48-51\) have described VOR and COR dysfunction, specifically an increased gain in COR. Bexander and Hodges\(^48\) also reported a decreased co-ordination between the COR and VOR. VOR and COR can be evaluated using electro-oculography.

Ocular Complications Due to Similar Mechanisms of Head Injury

In extrapolation, we have found ocular injuries due to a similar mechanism of head injury, specifically bungee jumping. Ocular injuries due to bungee jumping have been reported in several case reports. The most common ocular symptom is blurring of vision\(^52-54\) which spontaneously resolves after a few weeks. Common ocular signs include subconjunctival haemorrhage,\(^52-54\) periorbital bruising,\(^53\) and retinal haemorrhages,\(^53,54\) Less common but reported symptoms include horizontal diplopia,\(^54\) nystagmoid jerks on versions,\(^54\) vitreous haemorrhage,\(^53,54\) retinal detachment,\(^52\) and macular oedema.\(^53,54\) The mechanism of injury has been postulated to be because of the rise in intrathoracic pressure during the sudden deceleration phase of the jump, consequently increasing intravenous pressure
and hydrostatic pressure in the intraocular circulation.\textsuperscript{52-54} Breath holding and tensing of the abdominal muscles during the jump are also contributors to the increase in intrathoracic pressure.\textsuperscript{52-54} A gravitational force of -3.0 G has been shown to be sufficient to cause ocular injuries.\textsuperscript{52-54}

### Treatment

The treatment of ocular complications of whiplash injury should be based on the deficits identified during physical examination and investigation outcomes. Commonly, treatment is conservative and revolves around neck physiotherapy and oculomotor rehabilitation, which involves oculomotor convergence and motility exercises. Early rehabilitation can predict a better outcome. Controlling pain and inflammation may also help to improve outcomes.

## REFERENCES


## CONCLUSION

Whiplash injuries occur commonly in motor vehicle accidents. While patients may present asymptptomatically, a myriad of ocular complications may be detected. This possibly highlights the need for ophthalmologists and physicians to conduct basic ophthalmological examinations for all patients who present with whiplash injury. Prognosis for ocular complications depends mainly on the severity of complication and the time lapse between injury, detection of complication, and initiation of treatment. Ophthalmologists and physicians should be aware and vigilant towards patients with ophthalmic complaints following whiplash injury as a small minority of patients may have poor visual outcome and increased morbidity without treatment and follow-up.
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