**Key points**

Whiplash is the most common injury associated with motor vehicle accidents and a major cause of disability and litigation. Whiplash-associated disorders (WAD) can be classified by the severity of signs and symptoms from Grade 0 to 4.

Patients usually complain of neck pain and stiffness in the acute phase, with the majority recovering within 3 months. Depression, anxiety, and mood disorders are common in patients with chronic whiplash. Reassurance, early mobilization, simple analgesic, and physiotherapy are recommended in acute whiplash (WAD I–III).

In chronic WAD, multidisciplinary pain clinic referral followed by cognitive behavioural therapy and cervical radiofrequency neurotomy plays an important role.

An acute whiplash injury follows sudden or excessive hyperextension, hyperflexion, or rotation of the neck affecting the soft tissues. It typically results from rear-end or side-impact motor vehicle collisions. Patients commonly present with pain and stiffness in the neck, headache, and upper backache with or without paraesthesia of the upper limbs. Chronic whiplash syndrome is characterized by symptoms that persist for more than 3 months. With over half a million people making whiplash injury claims per annum in the UK, it has a major impact on the healthcare and legal systems and also the economy.

**Clinical features**

WAD includes all indirect injuries to the cervical spine. The affected individual most commonly complains of neck pain and stiffness in the acute phase. The other symptoms are headaches, mainly occipital, upper back and shoulder pain, and upper limb pain. Patients may also complain of paraesthesias, numbness, and/or weakness of the upper limbs and may occasionally suffer from dizziness, blurred vision, vertigo, dysphagia, and tiredness. Based on the severity of the symptoms, WAD is classified in five grades (Table 1).

**Background**

In 1995, whiplash-associated disorders (WAD) was defined by the Quebec task force as ‘Whiplash is an acceleration–deceleration mechanism of energy transfer to the neck. It may result from rear-end or side-impact motor vehicle collisions, but can also occur during diving or other mishaps. The impact may result in bony or soft-tissue injuries (whiplash-injury), which in turn may lead to a variety of clinical manifestations called Whiplash-Associated Disorders’.2

The incidence of WAD is very variable among countries, averaging to about nine per 1000 people in the UK, the highest in Europe. WAD is usually seen in rear-end, low-impact collisions, in 90% cases at speeds of <14 mph.3 The trunk is forced back on the seat with hyperextension of the neck and then forward recoil (Fig. 1). The risk of whiplash injury is higher in women (as they have thinner necks), people with short necks, car seats with a low neck rest, and if the rear-end collision is with a heavy vehicle. Car seats with an elastic back minimize the risk of neck injury after a car crash. Research indicates that the source of the pain in a majority (~60%) of whiplash patients is the zygapophysial (facet) joints (especially C2/3 and C5/6 levels), rather than the muscles.4

**Chronic whiplash**

Around 40% of patients suffer from symptoms of WAD beyond 3 months (chronic whiplash) and 2–4.5% of patients are left permanently disabled.5,6 Patients who are asymptomatic at 3 months continue to be so even after 2 yr. A worse outcome is associated with severe and rapid onset of neck pain and stiffness after the initial injury, neurological deficit, arm pain, headaches, and acute hospital admission. Older age at the time of injury, pre-existing pain in the neck, low back ache, female gender, part-time employment, lower educational achievement, and lawyer involvement have been established as markers for delayed recovery in patients with whiplash injury.3 Depression, anxiety, and mood disorders are common in patients with chronic whiplash. If one or more of the above adverse indicators are noted, the patients usually need a more intensive treatment and possibly an early referral to a physiotherapist, pain physician, or a specialist with interest in WAD.

Patients who develop chronic pain after whiplash injury also suffer from central hypersensitivity, possibly due to the sensitization of dorsal horn neurones. Studies have shown that these patients experience hyperalgesia to cutaneous and muscular stimulus not only in the neck but also at sites away from the primary site of injury. There is thought to be an alteration of the
afferent nociceptive signal from a focus present deep in the tissues of the neck and an imbalance occurring in the descending pain modulation system.7

Diagnosis
Whiplash or WAD is essentially a diagnosis based on clinical findings. Assessment of a person with an acute whiplash injury includes:1

(i) Confirming a history of sudden or excessive neck extension, flexion, or rotation. Symptoms may be delayed for hours or days after the injury. The two most common symptoms are headache and disabling neck pain, with or without referral to the shoulder or arm.

(ii) Examining for signs of muscular spasm, point tenderness, and neurological problems in the upper or lower limbs. Assess for range of neck movements, if appropriate. Look for ‘red flag’ features suggestive of a serious spinal or other abnormality, including compression of the spinal cord (myelopathy), cancer, severe trauma or skeletal injury, and vascular insufficiency.

(iii) Identifying possible psychosocial barriers to recovery, such as stress, anxiety, or depression (‘yellow flags’). WAD is especially likely to manifest in people with obsessive compulsive behaviour, anxiety, depression, hypochondriasis, and those with high scores of somatization.

Table 1 Classification of WAD1,6

<table>
<thead>
<tr>
<th>Grade</th>
<th>Symptoms</th>
<th>Radiography</th>
<th>Pharmacotherapy</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>Asymptomatic, no complaints of neck pain or stiffness, absent physical signs</td>
<td>Not required</td>
<td>Not required</td>
</tr>
<tr>
<td>I</td>
<td>Pain, stiffness, or tenderness of the neck as the only complaint, absent physical signs</td>
<td>Plain X-ray of cervical spine usually not required</td>
<td>Simple analgesics</td>
</tr>
<tr>
<td>II</td>
<td>Pain, stiffness, or tenderness of the neck along with musculoskeletal signs, e.g. decreased range of movement and point tenderness of the neck</td>
<td>Plain X-ray of cervical spine usually not required unless: <em>Suspected bony injury</em> <em>Impaired consciousness leading to masked pain sensation</em> <em>High speed/high impact collision</em></td>
<td>Non-opioid analgesics NSAIDs</td>
</tr>
<tr>
<td>III</td>
<td>Pain, stiffness, or tenderness of the neck and neurological involvement, e.g. sensory deficits, motor weakness, and/or decreased or absent deep tendon reflexes</td>
<td>Baseline X-ray of cervical spine, anterior–posterior, lateral, and open-mouthed view May need specialized imaging</td>
<td>Non-opioid analgesics NSAIDs Opioids for a limited period in acute severe cases</td>
</tr>
<tr>
<td>IV</td>
<td>Pain, stiffness, or tenderness of the neck along with dislocation or fracture with or without spinal cord injury</td>
<td>Specialized imaging, e.g. CT scan, MRI, myelography, discography</td>
<td>Non-opioid analgesics NSAIDs Opioids Immediate referral to specialist surgeon</td>
</tr>
</tbody>
</table>
Whiplash injury

Treatment

Whiplash injuries are quite difficult to treat because of interactions of various factors such as patient psychology, socioeconomic factors, legal issues, and physical health. The absence of radiological evidence of injury in the symptomatic group further complicates the treatment process for this condition. It was traditionally believed that bed rest and immobilization of the neck with a soft collar was the initial treatment of choice, but this has been refuted. Reassurance, resuming activity as normal, early mobilization, physiotherapy along with isometric exercise with periods of rest intermittently have been associated with better long-term outcomes.

Acute/subacute whiplash injury

Acute whiplash injury is defined as symptoms present up to 4 weeks and subacute whiplash when symptoms persist beyond 4 weeks until 12 weeks. Active physiotherapy and manipulation in the acute phase of the injury has a significant role in improving neck pain at 6 months and also increases range of movement of the neck similar to an uninjured individual. Mobilization is more effective if started within 96 h of the injury. A better range of neck movements at 2 weeks has been reported when non-steroidal anti-inflammatory drugs (NSAIDs) have been used. Patients receiving high-dose methylprednisolone within 8 h of injury have fewer disabling symptoms at 6 months and fewer sick leave days but is not recommended in WAD I–III.6,8 Other physical treatments such as heat and cold packs, pulsed electromagnetic field therapy, hydrotherapy, traction, ultrasound therapy, laser therapy, short-wave diathermy, transcutaneous electrical nerve stimulation, acupuncture, and passive repetitive movements have been tried with variable benefit. There is some evidence that a multimodal treatment approach of postural training, eye fixation exercises, manual treatment, and psychological support may be more beneficial than physical therapy at both symptom improvement and functional improvement.9 Recent work indicates standardized multidisciplinary rehabilitation programme aimed at fostering functional recovery may reduce pain catastrophizing in patients with subacute pain after whiplash injury.10 Various analgesics, antidepressants (especially amitriptyline), and muscle relaxants such as diazepam, azapropazone, epidural local anaesthetics, and epidural corticosteroids have been used. Muscle relaxants are not recommended in the acute phase.

Chronic whiplash injury

Many interventions have been tried for chronic whiplash injuries; of which, cervical radiofrequency neurotomy (CRFN) facet joints have shown reasonable relief from pain for up to 9 months. Some studies have reported a benefit in nearly 70% of the patients from CRFN.11 Other interventions like cervical facet joint injections have been used with short-term benefit. Intra-articular corticosteroids, temporomandibular joint treatment, cervical traction, and botulinum toxin injections have been described in the literature, with evidence lacking to support their use currently. A multimodal approach to treatment including cognitive behaviour therapy along with physical and or mechanical therapy and patient education is more effective at 3 months with a higher percentage of patients being satisfied with their pain management when compared with only physical treatment.9

For symptom management, along with simple analgesics, patients may be commenced on antineuropathic medications according to NICE clinical guideline 96.12 Patients with neurological deficits undergo more aggressive management including surgical treatment. One-third of the people suffering from brachialgia after chronic whiplash injury may benefit from cervical fusion procedures and nearly half of the patients with nerve impingement have reported pain relief from subacromial decompression.

Medicolegal implications

Whiplash injuries cost the economy of the UK approximately £3.64 billion per annum and have increased in number by 25% since 2002, probably the highest in Europe. They constitute 76% of motor-inurance claims. There is a prevalent view that a claimant’s symptoms will improve once litigation has finished, but this is unsupported by the literature. However, claimants who seek therapy from a physician initially or do not seek care at all are more likely to have their claims closed faster than those who look for help from physical therapists or chiropractors, as active manipulation may worsen symptoms.13 Residual pain was slightly higher in the 'Red flag' features or for those with persistent problems of pain down the arms.1 Pre-existing degenerative cervical spine disease (especially spondylosis at the C5/6 level) is the most common finding seen radiologically in patients complaining of whiplash injury. Additionally, they may have a minimal loss of the lordotic curve of the cervical vertebrae. Dynamic X-rays of the neck during flexion and extension initially may show a kyphotic angle possibly due to muscle spasm causing decreased mobility at the cervical region with a resultant increase in mobility at the adjacent vertebral level.5

(iv) Radiological investigations in the acute phase can lead to a large number of false-positive cases. It is important to avoid overinvestigating; these should be held in reserve for patients with ‘Red flag’ features or for those with persistent problems of pain down the arms.1 Pre-existing degenerative cervical spine disease (especially spondylosis at the C5/6 level) is the most common finding seen radiologically in patients complaining of whiplash injury. Additionally, they may have a minimal loss of the lordotic curve of the cervical vertebrae. Dynamic X-rays of the neck during flexion and extension initially may show a kyphotic angle possibly due to muscle spasm causing decreased mobility at the cervical region with a resultant increase in mobility at the adjacent vertebral level.5
Conclusion
Current evidence supports early mobilization, simple analgesics, re-assurance, and active interventions as the best treatment options for WAD I–III. Immediate specialist referral should be made for severe symptomatic WAD III and for all patients with WAD IV. Recovery from whiplash injury can be predictable in most cases. Most patients who are symptomatic at 3 months remain so indefinitely, especially if psychosocial and economic factors are involved.5

Declaration of interest
None declared.

References

Please see multiple choice questions 17–20.