Gunshot face as a cause of hyperextension central cord syndrome in a young patient

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Abstract
Central cord syndrome (CCS) is a syndrome where the patient's upper limbs are more severely affected than the lower limbs in terms of neurological deficit. This is typically found in an elderly patient with pre-existing spondylosis and a minor hyperextension injury.

This case report highlights an unusual cause of CCS in a young patient with pre-existing congenital cervical canal stenosis and acute hyperextension induced by a facial gunshot.

The aetiology and management dilemmas are discussed.

Key words: central cord syndrome, gunshot, hyperextension, spinal cord injury

Introduction
Acute central cervical cord syndrome is commonly seen in elderly patients with underlying cervical spine stenosis following a fall and subjected to a hyperextension force. Patients present with an incomplete spinal cord injury with predominantly upper limb weakness and relatively spared lower limbs.

We present an unusual case of a young man who was subjected to an acute hyperextension force during a gunshot to the face. In addition to the infrequently encountered aetiology, the management challenges will be discussed.

Case report
A 43-year-old male was admitted to our tertiary hospital Trauma Unit after having sustained a gunshot injury to the face.

He was fully conscious with features of neurogenic shock (BP 93/50 and heart rate 86). Anal tone was present but decreased.

The bullet had entered through the philtrum and exited above the right maxillary sinus. These facial injuries were cleaned and sutured.

His neurological examination confirmed a C4 incomplete lesion with motor weakness but sensory preservation. There was reduced anal tone and he required a urinary catheter due to retention.

On arrival at the Trauma Unit he was screened by low-dose digital X-ray whole body scan (Lodox) which excluded a skull fracture and confirmed that the bullet had not been retained. No cervical spine pathology was identified although these images were of poor quality.

Computerised tomography (CT) scan illustrated a linear undisplaced fracture of the anterior wall of the right maxillary antrum with extension into the alveolar process. There was haemorrhage into the right maxillary sinus.

Magnetic resonance imaging (MRI) demonstrated mild retrolisthesis of C3/C4 with increased signal in the prevertebral tissue as well as interspinous ligaments. Disc osteophyte complexes were present at C3/4 and C4/5 in a congenitally narrowed canal. There was cord compression and contusion from C3–C5 as evidenced by hyperintense cord signal on the T2 MRI sequence as revealed by high signal foci within the cord (Figures 1 and 2).
The patient was stabilised physiologically and referred to the Acute Spinal Cord Injury (ASCI) Unit for supportive ventilation and treatment of atelectasis, bronchopneumonia and shock.

The patient was stabilised with regard to the neurogenic shock, and the bronchopneumonia treated with broad-spectrum antibiotics. Supportive ventilation was required. The neck was initially managed in a Philadelphia collar.

Based on the extensive nature of cord compression from C3–C5, and underlying congenital stenosis, a posterior-based procedure was chosen. A laminoplasty was performed rather than a laminectomy due to his young age.

However, despite an adequate canal enlargement intra-operatively, the patient had no neurological recovery in the subsequent two weeks. Thus a second stage anterior C3/4 disc osteophyte complex decompression was performed via a Smith-Robinson approach (Figure 3).

Following this anterior procedure there was an immediate neurological gain of at least an MRC grade, more so in the lower than upper limbs.

Three weeks later the patient was transferred to the spinal rehabilitation centre.

Figure 1. Pre-operative sagittal MRI demonstrating multilevel stenosis and C3/4 disc protrusion

Figure 2. Pre-operative axial MRI confirming C3/4 disc protrusion

Figure 3. Post-operative X-rays with laminoplasty plates and anterior cervical plate present
At the six-month post-operative visit, his lower limbs had improved from 1/5 to 3 and 4s but due to the severe spasticity he remained non-ambulatory, thus ASIA B to C. His upper limbs remained at 2/5 power. A follow-up MRI confirmed adequate canal decompression with myelomalacia of the cord (Figure 4).

Discussion

Acute traumatic central cervical cord syndrome was defined by Schneider in 1954 as an incomplete spinal cord injury with the upper extremities illustrating a significantly greater motor impairment than the lower extremities with variable bladder dysfunction and sensory abnormality below the affected level. However, Sir Thornburn was the first to describe cervical cord syndrome in literature in 1887 as ‘a case of concussion of the spine’. It is caused by a variety of mechanisms but the most common is a hyperextension force resulting in cord compression and injury to the central part of the spinal cord with some sparing of the peripheral pathways. Three main mechanisms have been postulated:

1. Young patients sustaining a high velocity injury, e.g. motor vehicle accident, diving accident or fall from height
2. Older patients (>50 years) due to a hyperextension force in an already degenerate spine
3. Low velocity trauma in a patient with an acute central disc herniation

Hyperextension of the cervical spine can cause damage to the spinal cord via buckling of the ligamentum flavum or impaction of the posterior elements with rupture of the posterior longitudinal ligament. Hyperextension can be caused by a contact or non-contact force. Direct frontal impact to the head can also cause anterior distraction and posterior compression of the spinal cord, a mechanism reported with the deployment of airbags.

In this case, the young patient had underlying stenosis due to premature C3/4 degenerative stenosis. Despite the bullet not contacting the spine, it is likely to have induced an acute hyperextension force due to its trajectory across the face in an inferior-to-superior direction.

In order for the patient to be classified as a traumatic CCS, Pouw et al. recommended that the upper limb ASIA motor score should be a minimum of 10 points lower than the lower limbs. In our case the differential was 30 points with an initial ASIA B improving to a C.

Radiological features of CCS vary. X-rays may be normal if there is no pre-existing pathology. Underlying congenital narrowing can be assessed with the Pavlov or Torg ratio. This is a ratio of canal size to anterior-to-posterior vertebral body dimension on the lateral X-ray. This should be >0.82 but in our patient was 0.5–0.7 from C3–C5.

These patients are best investigated with an MRI where disc and ligament disruption, spinal canal compromise and degree of spinal cord injury can be assessed. The MRI may indicate cord oedema, cyst formation or, on rare occasions, a haematomyeloma.

With regard to the case study, the MRI was an essential tool in identifying the multilevel cord compression and pre-existing spondylosis.

The treatment of CCS is controversial. Aarabi et al. state that management recommendations in an extensive literature review (1966–2011) is limited to Class III medical evidence.

He separates the treatment of all acute central CCS according to the presenting pathology:

1. Patients with MRI evidence of spinal cord signal change but no radiological abnormality can be treated medically.
2. Patients with skeletal pathology such as fracture must undergo surgery for stabilisation and decompression.
3. Patients with no bony abnormality but who have concomitant spinal stenosis have the option of either surgical or medical treatment.

Timing of surgery in CCS remains controversial. The question remains as to whether there is a role for urgent decompression in order to enhance neurological recovery in patients with no instability. A systemic review by Lenehan et al. reviewed whether there was a need to urgently decompress patients within 24 hours or stage the surgery.
The conclusion was that patients who had ASIA C and below and persistent cord compression benefited from early intervention, but those with ASIA D deficit could be observed and potentially treated surgically later if there was no improvement. Other studies have supported the notion that surgically treated patients for acute cervical syndrome have better outcomes neurologically compared to those receiving only medical treatment. A trend towards decreased length of hospital stay and fewer complications has been illustrated in patients who are treated surgically compared to nonsurgical groups.

There is Class III evidence to support improved patient outcome in acute CCS by aggressive medical support to allow perfusion of the spinal cord. Conservative treatment of patients with acute traumatic cervical spine syndrome may predispose the patient to persistent neuropathic pain and spasticity. The spasticity can be so severe as to hinder recovery, decrease the functional motor grade, prevent a patient from achieving potential ambulation and be the main cause of patient dissatisfaction. Physiotherapy and certain drugs such as baclofen, dantrolene and gabapentin may assist in the reduction of these symptoms. In this study the patient was unable to achieve full ambulation due to the severe spasticity he developed. He underwent physical therapy and medical treatment to control the spasticity, to no avail.

Surgical options vary with regard to the pathology. Most patients present with multilevel pathology due to pre-existing spinal stenosis. Anterior decompression is favoured for focal pathology. This may include discectomy or corpectomy. Zhu et al. performed meta-analysis comparing the anterior versus posterior approach for multilevel cervical spondylotic myelopathy. They found that anterior surgery provided better short-term neurological outcome but higher complication and re-operation rates compared with posterior surgery. Posterior surgery includes laminectomy, laminoplasty, foraminectomy and posterior cervical fusion. Posterior surgery has been associated with increased neck pain and disability, but this was disputed by a study by Seng et al. whereby in a two-year follow-up the study showed no increased neck instability or neck pain post laminoplasty in 52 patients. To date there is no proven superior approach with regard to treating multilevel disease.

Our case highlights the dilemma of both pre-existing multilevel stenosis due to congenital narrow canal and premature spondylosis with a focal disc extrusion. To address this all anteriorly would necessitate multilevel corpectomies with prolonged theatre time and approach-related risks. Posterior decompression allows technically easier multilevel decompression with the laminectomy technique or slightly more demanding laminoplasty. As long as there is lordosis, the thecal sac will migrate posteriorly. However, this posterior migration is kept in check by the nerve roots which run antero-laterally.

Thus large anterior compression such as the C3/4 disc in this case may cause ongoing compression. Therefore an additional focal anterior decompression was performed when there was no initial neurological improvement. Of course, it will never be known whether the subsequent neurological recovery was directly due to the anterior approach or coincidental with delayed recovery from the posterior decompression.

The conclusion was that patients who had ASIA C and below and persistent cord compression benefited from early intervention, but those with ASIA D deficit could be observed and potentially treated surgically later if there was no improvement.

Conclusion

This case reports an unusual cause of CCS in a young patient, via facial gunshot-induced hyperextension with indirect injury to the spinal cord. The management dilemma of anterior focal versus posterior multilevel decompression remains, and the decision is left to the surgeon on a case-by-case basis. In retrospect, with significant disc extrusion, an initial anterior decompression and fusion procedure is probably indicated.

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As this is a case report, our Ethics committee does not require approval.

References


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