A 28-year-old man was brought to the emergency department with quadriparesis of acute onset after a bout of binge drinking. Evaluation revealed a mid-cervical myelopathy and magnetic resonance imaging (MRI) showed an acute compressive cervical myelopathy. He also developed rhabdomyolysis, and cervical paraspinal muscles showed MRI hyperintensities. After resolution of rhabdomyolysis and acute kidney injury, he underwent cervical spine fixation. He was found to have acute dropped head syndrome with secondary compressive myelopathy.

KEYWORDS: acute myelopathy, intoxication, intervertebral disc disruption, neck flexion

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Case presentation

A 28-year-old man was brought to the emergency department after a New Year’s Eve party. He had binge-drunk 750 mL of brandy and had slept it off sitting against a wall, with his head slumped onto his chest for over 7 h. When he was woken by his brother the next morning, he complained that he could not move his hands and legs. He also complained of an inability to pass urine despite severe lower abdominal pain.

In the emergency department, abdominal palpation revealed a massively overdistended urinary bladder and he was catheterized. Neurological examination revealed flaccid quadriparesis (grade 0/5 power in all four limbs) with extensor plantar responses. Sensory examination was within normal limits. Magnetic resonance imaging (MRI) of spine showed a C5–6 central intervertebral disc prolapse with cervical cord compression and spinal cord oedema at this level. The 5th and 6th cervical vertebral bodies showed marrow hyperintensities. The posterior paraspinal muscles and interspinous ligament were also hyperintense (more on the right side). His serum creatine kinase (30,000 IU/L; 24–95) and serum creatinine (159 μmol/L; 60–110) were elevated.

He was treated with forced alkaline diuresis and a nephrology consultation was obtained. A Philadelphia cervical collar was provided. After his biochemical parameters normalised, he underwent an anterior cervical disectomy and fusion on day seven after admission.

Postoperatively, he was transferred to inpatient rehabilitation and improved to grade 3/5 power within 4 days. He was discharged on day 17. Eight months later, he presented to the neurology outpatient department with twitching and weakness of both arms.

On examination, he had a pectoral crease, wasting of the shoulder girdle muscles bilaterally, and fasciculations over the deltoid and biceps muscles. Upper limb deep tendon reflexes were absent, lower limb reflexes were brisk and plantar responses were extensor. MRI of spine showed cervical cord myelomalacia at C5–6 levels with some susceptibility artifacts resulting from spinal implants at the C5–6 bodies. A retrospective diagnosis of acute dropped head syndrome (DHS) with secondary compressive myelopathy (ADHM) was made. He was offered alcohol rehabilitation services.

Discussion

A chronic DHS or ‘head ptosis’ is seen in association with isolated neck extensor myopathy (INEM), nemaline myopathy, inclusion body myositis, multiple system atrophy, amyotrophic lateral sclerosis, inflammatory myositis, myasthenia gravis and a variety of other neuromuscular illnesses. The chronically abnormal flexed neck posture can lead to compressive myelopathy. The usual duration from the onset of DHS to myelopathy averages 1–2 years. Compressive myelopathy in these cases occurs secondary to a combination of factors, including cervical segmental instability, accelerated degenerative changes, compression of the cervical spinal cord by anterior structures such as the vertebral bodies or intervertebral discs, as well as spinal microcirculation impairment. The anterior horn cells are particularly predisposed to ischemia and microcirculatory dysfunction. Similarly, a ‘tight’ dural canal can cause selective anterior horn cell degeneration during neck flexion by pinching the cervical cord between the posterior border of the vertebral body and the posterior dura, resulting in Hirayama disease or chronic brachial amyotrophy. The treatment of choice in chronic DHS with myelopathy is cervical fixation, which leads to gradual improvement of symptoms in most patients.
Conversely, head ptosis itself can occur following acute central spinal cord injury. In such cases, MRI shows cervical cord compression and paraspinal muscle hyperintensity. Prolonged abnormal postures following alcohol or substance intoxication are known to produce compressive neuropathies or myelopathies. Well-known compressive neuropathic syndromes include toilet seat neuropathy, and bilateral sciatic neuropathy after prolonged lotus posture, prayer posture and seated forward bend.\textsuperscript{1–4} Toilet seat neuropathy is a constellation of sciatic neuropathy and rhabdomyolysis with gluteal compartment syndrome.\textsuperscript{2} The sciatic nerve is damaged from direct compression of the nerve at the sciatic notch or gluteal compartment syndrome and is worsened by prolonged hip flexion from sitting on the toilet seat. Severe cases can be accompanied by perineal gangrene (resulting from perineal and internal pudendal artery compression) or renal failure.

Similarly, acute mid-cervical myelopathies have been described following prolonged flexion or hyperextension of the cervical spine during surgical procedures and following substance abuse.\textsuperscript{5} These are accompanied by quadriplegia and rhabdomyolysis with MRI changes in the spinal cord and deep cervical muscles.

Conclusion

I describe an unusual case of acute mid-cervical compressive myelopathy or acute head ptosis with secondary cervical myelopathy and rhabdomyolysis following alcohol intoxication. Unlike previous reports, the patient developed a compressive mid-cervical myelopathy resulting from acute intervertebral disc disruption following prolonged neck flexion. The patient’s ‘acute ptotic myelopathy’ resembled a ‘toilet seat neuropathy’ because he developed it following an alcoholic blackout with prolonged neck flexion (head ptosis). The temporal relationship of head ptosis to the clinical syndrome of myelopathy and rhabdomyolysis, MRI findings of vertebral body marrow oedema, intervertebral disc protrusion at the site of compression, and posterior cervical paraspinal oedema were consistent with an acute ptotic myelopathy. Acute prolonged neck flexion must have led to the cervical vertebral body, disc, spinal cord, and paraspinal muscle injury as a result of a local ‘compartment’ syndrome. Although surgical cervical spine fixation is the cornerstone of treatment, the additional complications associated with this syndrome should be recognised and treated. It is important to recognise this as

![Fig 1. Radiological and clinical features of the patient.](image-url)

(a) Sagittal T2 weighted magnetic resonance imaging (MRI) showing intervertebral disc protrusion at the C5–6 level with marrow changes in C5 and C6, along with a short segment spinal cord hyperintensity (arrow). (b) Sagittal T2 weighted MRI demonstrating cervical paraspinal oedema on the right side (arrow). (c) Sagittal T2 weighted MRI demonstrating cervical paraspinal oedema (of lesser intensity) on the left side (arrow). (d) Sagittal T2 weighted MRI 8 months after the event showing myelomalacia at the C5–6 level with postoperative vertebral body changes (arrow). (e) Axial T2 weighted MRI showing oedema in the lamina and spinous process of the C5 vertebra with paraspinal oedema (right > left) (arrows). (f) Axial T2 weighted image at 8 months, showing anterior horn cell hyperintensities (arrow). (g) Photograph of the patient showing bilateral shoulder girdle wasting and an oblique pectoral crease on the right side (arrow) indicative of periscapular, trapezius and pectoralis muscle weakness.
an accompaniment of an alcohol use disorder associated with binge drinking. Presenting patients should be offered alcohol rehabilitation services.

References


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