Lobster claw sign: a new sign depicting central cervical cord injury at or below that level. Case report

Ahmed Ansari

Department of Neurosurgery, Hamdard Institute, Jamia Hamdard, New Delhi, INDIA

Abstract: Traumatic cervical central cord spinal injuries (CSS) are now more readily recognized both clinically and on radiolgical scans. In this article, we introduce a lobster claw sign due to a particular positioning of spinous process of C2 and C3 with cord contusion from C3-C5 following injury.
Key words: traumatic central cervical cord, lobster claw sign

Introduction

McKinley found that 9.2% of all spinal cord injuries were attributed to central spinal cord syndrome.1 When presenting to emergency rooms, typically with greater upper vs. lower extremity neurological deficits, CSS patients first undergo X ray or computed tomography (CT) studies to rule out fracture/dislocation, and secondarily have MR examinations looking for classical central cord contusion /edema / hematomas. We present a typical sign of lobster claw on plain X ray cervical spine with underlying cord contusion on MR.

Case report

A 56 year old man with fall of heavy object overhead presented to us five days after injury with quadriparesis. There was no bowel and bladder involvement. Power in his right upper limb was 1/5 and 2/5 in left upper limb. Bilateral lower limb power was 3/5. CT head suggested linear fracture in occipital bone on right side reaching till foramen magnum. X ray cervical spine lateral view (Figure 1) showed a typical appearance of spinous process of C2 and C3, resembling a lobster claw along with degenerative changes. MRI cervical spine (Figures 2, 3, 4) was performed which showed cord contusion from C3-C5.

Figure 1 - X- ray cervical spine lateral view showing the typical lobster claw sign owing to spinous process of C2 and C3

Figure 2 - Sagittal MRI showing cord contusion from C3- C5 level

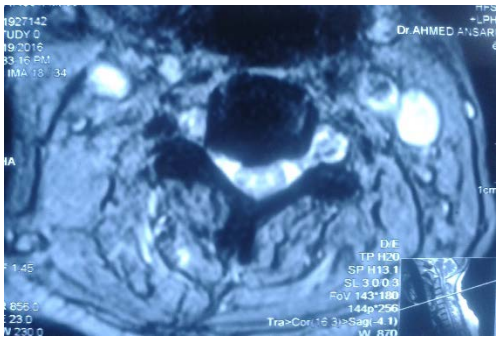

Figure 3 - Axial MRI at C3-C4 level with cord contusion

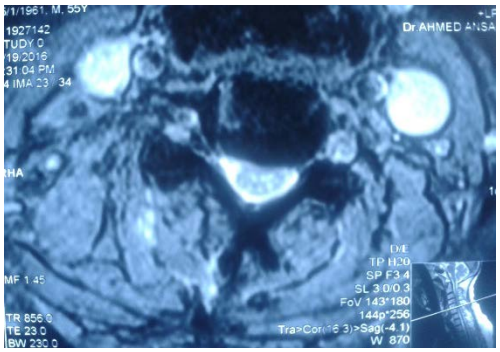

Figure 4 - Axial MRI at C4-C5 level with cord contusion

Discussion

Nowak et al. considered CSS to be the most common type of incomplete spinal cord injury that typically occurs following traumatic hyperextension events in older patients with underlying cervical spondylosis/stenosis. (2) Deficits may vary from weakness in the hands/forearms with relative sensory preservation, to severe quadriparesis (with sacral sparing consistent with an incomplete injury).

Li and Dai assessed the stress distribution to the cervical cord under different injury conditions to better understand the etiology of acute CCSs. (3) Histopathologic studies typically revealed that CSS occurs due to predominant white matter injury. In their feline model, cervical cord injuries were simulated in hyperextension (pinch force anterior (A) or posterior (B); flexion injuries (C)), and vertical compression (pinch force anterior (D) or posterior (E)). Results were analyzed utilizing a representative cross-section analysis. They attributed upper extremity weakness to damage involving the corticospinal tracts and motor neurons in the anterior horn.

Schneideret al. originally defined this syndrome in 1954, citing the predominant hyperextension mechanism in the absence of fracture/dislocation following various traumatic events (e.g. motor vehicle accidents, falls, and diving injuries). His definition included the greater upper vs. lower extremity motor deficits, the presence of bladder dysfunction, and variable sensory below the locus of the trauma. His study included a

review of 15 patients who presented with minor or major cervical injuries, in patients ranging in age from 18 to 85. Most injuries were attributed to acute mechanical compression attributed to hyperextension, resulting in the "pincer compression" of the cord between the spondylotic osteophytes anteriorly and acute inward buckling of the yellow ligament posteriorly.

Aarabi et al. evaluated traumatic central cord syndromes (TCCSs) characterized by incomplete spinal cord injuries. (4) In up to 50% of patients, they are due to hyperextension events in those with underlying congenital/degenerative spinal stenosis.

Song et al. assessed the clinical/prognostic value of dynamic X-rays and MR studies in 23 patients with CCS without fracture/dislocation.5 They evaluated multiple X-rays and MR factors including; prevertebral hyperintensity (HI), cord compression, intramedullary high-signal intensity (IMHSI), with/without instability.

In our patient, we were able to find this new sign on plain X-ray, namely "lobster claw", which we think should raise the suspicion of central cervical cord injury. This, all the more signifies the importance of plain radiology at small centres which are not equipped enough with advanced scanners.

Although, "lobster-claw" is a classic sign in uroradiology, (6, 7) it is a sign of papillary necrosis and it is due to complete sloughing of the central papilla with distortion of a residual "claw-shaped" calyx.

We clearly understand more cases are needed to validate this sign "lobster claw" in neuro radiology.

Correspondence
Dr. Ahmed Ansari
ahmed.ansari2@gmail.com
+91-9997456045

References

1.McKinley W, Santos K, Meade M, Brooke K. Incidence and outcomes of spinal cord injury clinical syndromes. J Spinal Cord Med. 2007;30:215–24.
2.Nowak DD, Lee JK, Gelb DE, Poelstra KA, Ludwig SC. Central cord syndrome. J Am Acad Orthop Surg. 2009;17:756–65.
3.Li XF, Dai LY. Acute central cord syndrome: Injury mechanisms and stress features.Spine (Phila Pa 1976) 2010;35:E955–64.
4.Aarabi B, Koltz M, Ibrahimi D. Hyperextension cervical spine injuries and traumatic central cord syndrome. Neurosurg Focus. 2008;25:E9.
5.Song J, Mizuno J, Inoue T, Nakagawa H. Clinical evaluation of traumatic central cord syndrome: Emphasis on clinical significance of prevertebral hyperintensity, cord compression, and intramedullary high-signal intensity on magnetic resonance imaging.Surg Neurol. 2006;65:117–23.
6.Dunnick NR, Sandler CM, Newhouse JH, Amis ES, Jr. Textbook of uroradiology 3rd ed. Baltimore, Md: Williams & Wilkins,2001; 300.
7.Hartman GW, Torres VE, Leago GF, Williamson B, Hattery RR. Analgesic-associated nephropathy. JAMA 1984; 251:1734-1738