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Extensive Cervical Spinal Cord Hemorrhagic Infraction Secondary to Prolonged Hyperflexion of the Neck Causing Quadriplegia

Asad Ullah*, Zeeshan Khakwani, Muzammil Khan

Department of Internal Medicine, Conemaugh Memorial Medical Center, Johnstown, PA, U.S.A.
*Corresponding author: asad-86@live.com

Abstract A young lady was brought to the emergency department after she was found unconscious sitting in her restroom for several hours. She was found to have decreased sensations below her neck and was unable to move any extremity. Multilevel imaging studies were obtained that revealed extensive areas of cervical spinal cord hemorrhagic infarction, postulated to be due to hyperflexion of her neck resulting in vascular compromise, which were inoperable and rendered her quadriplegic. She required intensive physical rehabilitation and was transferred to a specialist center for further treatment.

Keywords: *cervical hyperflexion, spinal cord infarction*

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1. Introduction

Spinal cord infarction is a rare condition, most commonly caused by trauma, iatrogenic causes, vascular malformations, and bleeding diatheses [1]. Pathogenesis remains largely unknown because most clinical studies included patients with ischemic lesions associated with aortic surgery or prolonged arterial hypotension [2]. Variable and overlapping presentation makes the diagnosis very challenging [1], therefore diagnosis of presumed spinal cord infarction requires the appropriate clinical picture and exclusion of other possible etiologies [3]. Conservative management, including administration of large doses of corticosteroids, reversal of anticoagulation, and close monitoring, has been used as bridging for surgical procedure or as the mainstay of treatment for patients with mild or improving symptoms [1]. Long term prognosis depends upon the extent of spinal cord injury and spinal cord sparing, and ranges from complete recovery to various degrees of neurological deficits [4].

2. Case

A young lady was brought to the emergency department (ED) after she was found unconscious sitting in her restroom for almost 12 hours. Her medical history included depression, anxiety and drug abuse. Upon presentation to the ED, she was very drowsy but arousable and was oriented to time, place and person. She was able to speak in full sentences and reported that she fell unconscious after using heroin.

On physical examination, she was hypothermic and bradycardic with temperature of 30.8 C and heart rate of 46 beats per minute. Her Glasgow coma scale was 15. Pupils were equal, round and reactive to light and accommodation. Neck was supple. Detailed neurological examination was limited due to patient's lethargy but she was able to wiggle her toes and make a loose hand grip. Rest of the examination was unremarkable.

Laboratory investigation revealed significantly elevated myoglobin level and acute kidney injury possibly caused by pigment nephropathy due to rhabdomyolysis. Urinalysis (UA) was suspicious for urinary tract infection (UTI) and patient was empirically started on intravenous (IV) ceftriaxone.

After hydration with warm IV fluids and applying warm blankets, patient's core body temperature and mentation improved. She reported pain in her neck and during her neurological assessment, she was unable to move her extremities and had decreased sensations below her neck. Pupils were equal and reactive to light and accommodation. Extraocular muscles, visual acuity and visual fields were intact. She had normal muscle bulk but decreased muscle tone. Strength was difficult to assess. Deep tendon reflexes were sluggish and planter reflexes were silent bilaterally. Vibration sensation was absent in all extremities and joint position was grossly impaired in both hands and feet. Light touch and pinprick were grossly intact in all extremities. Finger to nose, heel to shin and rapidly alternating movements couldn't be assessed. She also developed urinary retention a Foley catheter had to be placed. Fecal tone was diminished. Her American spinal injury association (ASIA) impairment scale was category B.

MRI of the brain, cervical and thoracic spines were obtained which showed severe edema and expansion of

the entire cervical and upper thoracic spinal cord spanning from C1-T4, predominantly involving the central gray and white matter with multiple small foci of petechial hemorrhages concerning for a large hemorrhagic infarct within the cord (Image 1 and Image 2). MRI findings were also significant for severe edema and enhancement within the posterior para-spinous musculature compatible with known diagnosis of rhabdomyolysis (Image 2).

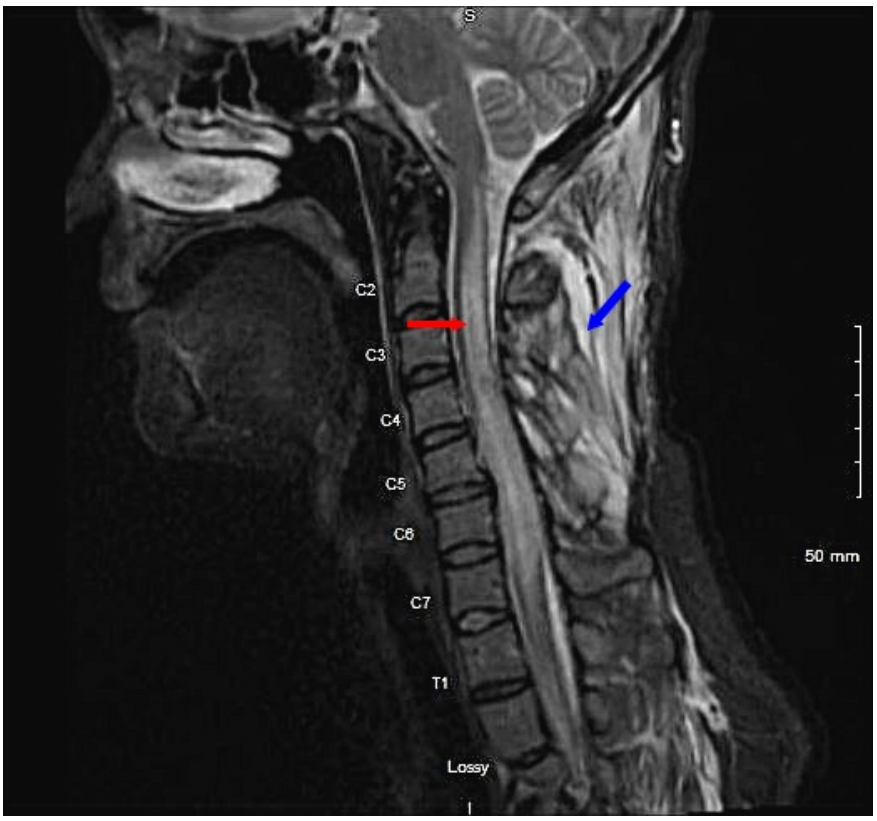

Image 1. MRI cervical spine showing diffuse cervical spinal cord edema (red arrow) and edematous para-spinous muscles (blue arrow)

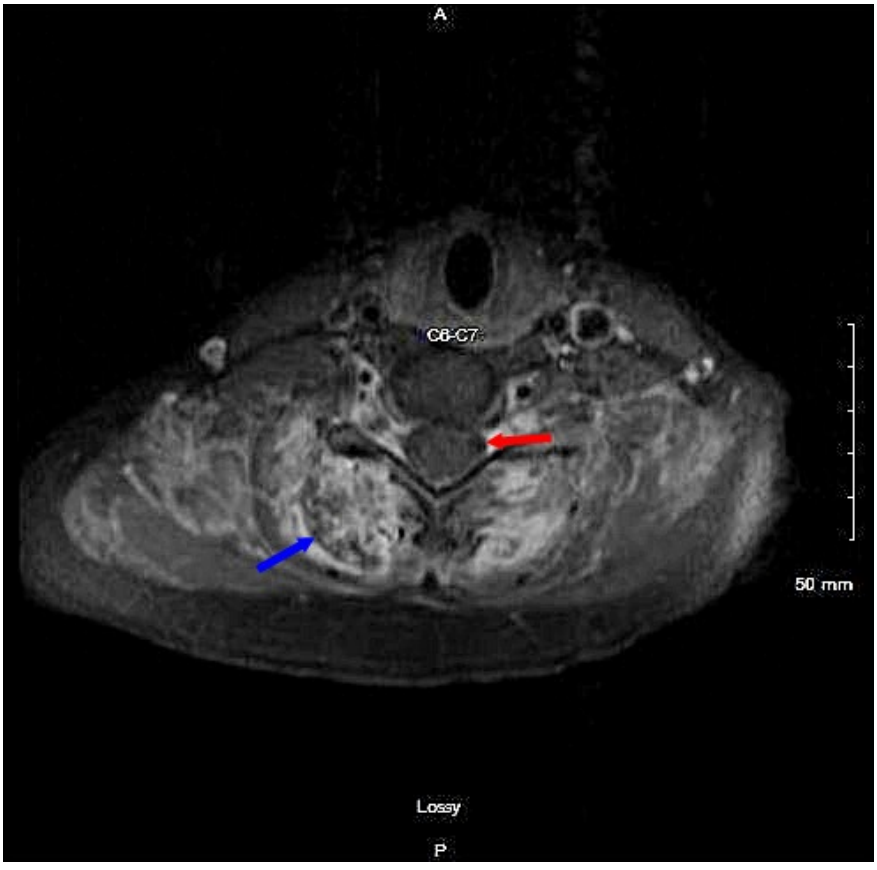

Image 2. MRI cervical spine showing hemorrhagic infarct (red arrow) and edematous para-spinous muscles (blue arrow)

Patient was started on IV decadron to decrease the edema. Neurosurgical team was consulted who evaluated the patient and due to extensive spinal cord damage, patient was unfortunately not amenable for surgical intervention. Physical and occupational therapists were actively managing the patient. Patient developed respiratory distress and hemodynamic instability, and was transferred to intensive care unit where she required endotracheal intubation and mechanical ventilation. Trails of weaning from mechanical ventilation proved futile and patient eventually required tracheostomy tube for adequate ventilation. She was also unable to swallow and a percutaneous gastrostomy tube had to be placed for nutrition. Her hospital course was also complicated by ventilator associated pneumonia and clostridium difficile colitis which were adequately treated.

Patient was provided with comprehensive physical and occupational therapy but she did not regain any sensory or motor function of her body below her cervical region and was unfortunately essentially quadriplegic. She required long term holistic rehabilitation and was thus transferred to a specialist rehabilitation institute for further care.

3. Discussion

Spinal cord infarction is a rare pathology that is either ischemic or hemorrhagic in nature. Ischemic causes include diseases of the spinal vasculature, severe hypotension, trauma or a complication of spinal or vascular surgery. Hemorrhagic infarction is very rare and is mostly caused by trauma or vascular malformation.

Romi et al., [5] reported that majority cases of spinal cord infarction are spontaneous, with no preceding event, and ischemic, while a small number of cases are hemorrhagic. A study by Novy J et al., [2] found two important causes of spinal cord infarction, radicular artery territorial infarct and extensive hypoperfusion. Risk factors associated with atherosclerosis, including hypertension, diabetes mellitus, hyperlipidemia and smoking, are related with spinal cord stroke [5].

Presenting symptoms depend upon the site and severity of spinal cord injury. A through history and physical examination can provide valuable clues to the etiology and diagnosis. In a study, sixty percent of patients with spinal cord infarction reported back or neck pain at the onset of symptoms, usually localized at the level of the lesion [2]. Symptoms generally evolve over several minutes and reach maximal deficit at five hours [6]. Severe impairment on initial examination, absence of Babinski sign, presence of sensory level are associated with poor outcome [6].

MRI is a useful modality for diagnosing spinal cord infarction and associated vascular and bony changes [7]. It can confirm the presence, location and extension of the infarction, but its diagnostic yield depends on the timing and quality (i.e., absence of artifact) of imaging [2]. MRI showing longitudinally extensive lesions are associated with poor prognosis [6].

Our patient was found unconscious in the sitting position for several hours. She was unable to move her extremities and had no sensations below her neck. MRI showed extensive spinal cord edema and hemorrhagic infarction. Novy J et al., reported that abnormal movement of the spine can lead to acute vascular compression [2] and result in spinal cord injury, and thus it was postulated that patient's prolonged unconsciousness and hyperflexion of her neck in the sitting position resulted in the vascular compromise of the cervical spinal cord, causing congestion and extensive hemorrhagic infarction that resulted in devastating neurological deficits.

There are no clear guidelines for the treatment of spinal strokes [2]. Every effort should be made to establish the etiology and treatment should be guided towards the underlying mechanism of spinal cord injury. Risk factors should be identified and managed accordingly. Anticoagulation and corticosteroids are commonly used in appropriate settings [2]. In a study by de Sèze et al., [8] there was no difference in clinical course in patients treated with anticoagulation or corticosteroids. Aggressive physical and occupational therapy should be at the forefront of patient management. Neurosurgical evaluation should be obtained early in cases of hemorrhagic lesions. Other measures for management include the complications of acute paraplegia such as immobility leading to venous stasis and subsequent thromboembolism disease, decubitus ulcer and infections such as pneumonia and urosepsis [9].

Recovery depends upon site and severity of spinal cord injury and is generally favorable, with complete or incomplete recovery [2] in most cases. Robertson et al., [6] concluded from a large retrospective study that severe impairment at nadir is the strongest predictor of poor functional outcome but meaningful recovery is also possible in a substantial minority of these patients.

4. Conclusion

Spinal cord infarction is rare but serious pathology, with recovery depending upon the site and extent of spinal cord damage and ranges from complete recovery to different levels of neurological deficits including complete paralysis. Early intensive physical and occupational rehabilitation is essential for better outcome.

References

[1] Shaban, A., et al., *Spinal Cord Hemorrhage.* J Stroke Cerebrovasc Dis, 2018.

[2] Novy, J., et al., *Spinal cord ischemia: clinical and imaging patterns, pathogenesis, and outcomes in 27 patients.* Arch Neurol, 2006. 63(8): p. 1113-20.

[3] Sandson, T.A. and J.H. Friedman, *Spinal cord infarction. Report of 8 cases and review of the literature.* Medicine (Baltimore), 1989. 68(5): p. 282-92.

[4] de Seze, M., et al., *[Functional prognosis of paraplegia due to cord ischemia: a retrospective study of 23 patients].* Rev Neurol (Paris), 2003. 159(11): p. 1038-45.

[5] Romi, F. and H. Naess, *Characteristics of spinal cord stroke in clinical neurology.* Eur Neurol, 2011. 66(5): p. 305-9.

[6] Robertson, C.E., et al., *Recovery after spinal cord infarcts: long-term outcome in 115 patients.* Neurology, 2012. 78(2): p. 114-21.

[7] Yuh, W.T., et al., *MR imaging of spinal cord and vertebral body infarction.* AJNR Am J Neuroradiol, 1992. 13(1): p. 145-54.

[8] de Seze, J., et al., *Acute myelopathies: Clinical, laboratory and outcome profiles in 79 cases.* Brain, 2001. 124(Pt 8): p. 1509-21.

[9] Patel, S., K. Naidoo, and P. Thomas, *Spinal cord infarction: a rare cause of paraplegia.* BMJ Case Rep, 2014. 2014.