Combined Cerebral and Spinal Cord Infarction Associated with Cardiac Arrest and Multi-Substance Abuse: A Case Report

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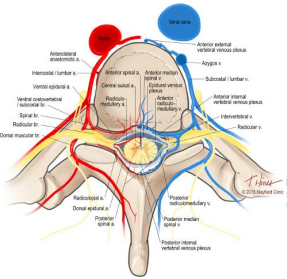
Introduction

Cerebral and spinal cord infarction may have similar etiologies, and indeed may also have overlapping symptoms present on neurologic and musculoskeletal physical examination:
Infarction to the cerebrum may present variably with weakness or flaccidity, loss of sensation, cranial nerve involvement, or disorientation.
Spinal cord infarcts may present with weakness, paraplegia, flaccidity, or loss of sensation depending on the location.
In the less common situation that both infarctions are present, providers are tasked with a sometimes challenging mission of localizing lesions of infarction in order to better monitor and address neurologic symptoms.

Clinical syndromes seen in spinal cord ischemia are dependent on which vessel is affected:
Anterior Spinal Artery: Flaccid tetra or paraplegia with loss of pain and temperature, preservation of proprioception and vibration
Posterior Spinal Artery: Loss of proprioception and vibration, preservation of movement, pain, and temperature
Central Sulcal Artery: Brown-Sequard Syndrome: Ipsilateral spastic paralysis with contralateral loss of pain and temperature



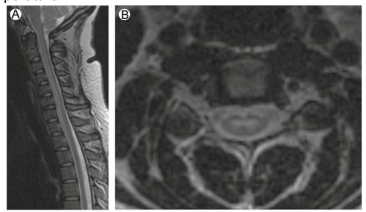


Figure 2 Spinal cord infarction. (A) Sagittal T2-WI and (B) axial T2-WI MR images in a 16-year old with sudden onset neurologic deficit after manipulation of her back. There is long segment T2 signal hyperintensity in the ventral cervical spinal cord most consistent with anterior spinal artery territory ischemia. (*Courtesy:* Dr Rupa Radhakrishnan).

Hospital Course

HD1: Patient is a 57 yo M with Type 2 DM and hx of cocaine and alcohol abuse who collapsed in a casino. CPR was started by bystanders with uncertain efficacy. An AED was obtained and patient was found to be in ventricular rhythm. Cardioversion was attempted twice, but unsuccessful. EMS arrived and administered amiodarone and defibrillated again, resulting in PEA. CPR was administered and the patient achieved ROSC. EKG revealed STEMI and bradycardia. The patient was intubated in the field and transferred to ICU upon arrival to hospital. Interventional cardiology treated.

HD4: Neurology is consulted due to "legs not working" and notes that the patient's legs are plegic and has decreased sensation to pinprick. Light touch sensation is normal. MRI w/o contrast of brain and spine was ordered. Brain MRI obtained revealing an acute global anoxic event. Patient grew agitated and refused spinal cord MRI.

HD11: Patient admitted to inpatient rehab. Notable physical examination findings include:

Orientation to person and place, but not time or situation
Waxing and waning cognition -- inconsistent answers
5/5 upper strength, 0/5 lower strength diffusely
Light touch intact bilaterally, including anal and perianal
Pinprick sensation diminished below left T12 and right L1
Absent anal tone
Truncal instability

ASIA scored as **T12 ASIA B**

Note: Patient's cognition made ASIA exam somewhat unreliable as responses were inconsistent, presumably due to brain injury.

HD13: Spine MRI done revealing abnormal cord signal from T10 to L2, most compatible with a cord infarct.

HD19: Patient was positive for a DVT while on ASA/Plavix, Brilinta, and Lovenox. Patient was also found to have significant rectal bleeding on examination and was subsequently transferred back to ICU.

Discharge: The patient continued to suffer from memory impairment, but eventually achieved wheelchair mobility and was discharged to a group home due to inability to reliably care for self.

Discussion

This case demonstrates the challenges practitioners face when diagnosing a mixed cerebral and spinal cord infarction. Thorough physical exam along with relevant imaging are key to successful diagnosis, monitoring, treatment, and adaptive techniques.
Symptoms of both cerebral and spinal cord infarction are largely dependent on their vascular involvement, but symptoms can sometimes be inconsistent. Imaging can guide--but should not dominate--treatment decisions. Regarding spinal cord infarctions, high resolution MRI is only 67% sensitive. T2 sequence of an ischemic cord presents with more hyperintensity centrally than peripherally, which is a key finding which can aid diagnosis.
Therapeutic treatment will be guided by accurate assessments and observations by physicians, nursing staff, physical therapists, occupational therapists, speech therapists, and hospital staff. A multidisciplinary approach with regular communication can improve outcomes, especially when symptoms are inconsistent as they were with this patient. The eyes of nurses, CNAs, PTAs, sitters, cleaning staff, and food delivery personnel were of great assistance in this patient's care.

Conclusion

Further investigation is warranted when history, physical exam, imaging and/or reported symptoms are not consistent with or do not fully explain the clinical scenario. An understanding of anatomy--particularly cerebral and spinal vascular anatomy--is imperative in localizing cerebral and spinal cord infarcts. Doing so allows for improved outcomes with regard to function and adaptation. An interdisciplinary approach is best to allow for multiple assessments and interventions, with frequent communication between providers.

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