Case Report

Acute cervical-transverse myelitis following intranasal insufflation of heroin

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A B S T R A C T

Irrespective of the route of administration, heroin abuse is attributed to severe medical complications and a high risk for addiction. Complications of acute heroin insufflation vary greatly from epistaxis, anosmia, rhabdomyolysis, stroke, and transverse myelitis. Transverse myelitis is considered a rare but serious complication with associated long-term morbidity. Here we present a case of a 20-year-old male patient who presented with paraplegia hours after nasal insufflation of heroin, consuming Xanax, and smoking marijuana and was incidentally diagnosed with cervical transverse myelitis. Patients with a history of drug abuse who present with acute neurological symptoms such as limb paralysis, and reduced sensation, should raise concern for transverse myelitis. The clinical presentation of heroin associated myelopathy is equivocal and requires prompt recognition and treatment to minimizing long-term sequelae.

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Case presentation

A 20-year-old male presented to the emergency department with a chief complaint of bilateral lower extremity paralysis. The patient denies any history of trauma or a similar previous episode. He reports no significant past medical or surgical history. He is a homosexual male with 1 partner and reports a recent negative HIV test. He discloses that 5 hours prior to noticing his symptoms, he consumed heroin by nasal insufflation, Xanax, and smoked marijuana before falling asleep. He woke up at 3 AM on the floor adjacent to his bed and noticed bilateral lower extremity flaccid paralysis and bilateral distal arm weakness.

On physical examination, the patient was fully alert and oriented, with normal gaze, no facial droop, handgrip ⅔ bilaterally, afebrile, and hemodynamically stable. Neurological examination revealed 1+ patellar reflexes, bilateral negative plantar response, and tingling sensation in the lower extremities. The patient denies head trauma, headache, loss of consciousness, seizures, tongue biting, and incontinence. He reports a 2-pack per year cigarette history, and a 2-year history of heroin inhalation and marijuana use. He denies a prolonged interruption in heroin use. He also inhaled heroin 24 hours prior to the most recent episode. His urine toxicology screen was positive for opiates, benzodiazepines, and tetrahydrocannabinol.
Emergent diagnostic imaging showed no remarkable findings in lumbar and sacral magnetic resonance imaging, thoracic computed tomography (CT), and head/neck CT. A sagittal magnetic resonance (MR) image without contrast indicated diffuse cord enlargement from the levels of C4-C6. An increase in the T2 signal intensity was appreciated with involvement of the central gray-white matter (Fig. 1). There is an abnormal signal on a sagittal, noncontrast T1 weighted image (Fig. 2). An axial T2 weighted MR image revealed an increased T2 signal density involving the central gray matter of C4-C6 (Fig. 3). An axial noncontrast T1 weighted MR image (Fig. 4) and T1 weighted image with IV contrast showed the same lesion to be T1 isointense and without any enhancement. Collectively, these findings represent an inflammatory process, likely transverse myelitis because they affect more than 2 vertebral segments with eccentric enhancement and are significant for cord expansion. The findings on diagnostic imaging rule out an acute ischemic stroke involving the anterior cerebral artery, a spinal cord astrocytoma and multiple sclerosis. Additional workup included a lumbar puncture with cytology that was nondiagnostic for multiple sclerosis and primary neoplasm. Lastly, a CT angiogram study of the neck showed no evidence of abnormalities involving the vertebral arteries. The patient was commenced on a 5-day course of intravenous methylprednisolone sodium succinate. He showed marked improvement of his neurological deficits. He began to ambulate by day 5. He complained of residual weakness in all 4 extremities with 4/5 power in his lower limbs. The Pt was transferred to a long-term drug rehabilitation center.
Discussion

Heroin is notorious for causing various medical and neurological sequelae. Common side effects of acute heroin insufflation vary from epistaxis, anosmia, rhombomyelitis, and stroke. Infrequently reported complications of all routes of heroin toxicity include noncardiogenic pulmonary edema [1,2], endocarditis [2], spongiform leukoencephalopathy, acute inflammatory demyelinating polyradiculopathy, and thoracic cord transverse myelitis [3]. Here we explore an unusual manifestation of heroin toxicity in a 20-year-old repeat drug abuser who presented with cervical cord transverse myelitis within hours of nasal insufflation of heroin. The patient describes a 2 year history of intranasal heroin abuse, intermittent intravenous injection of water dissolved cocaine, and a 2-pack year smoking history. Transverse myelitis is a rare inflammatory condition of unknown etiology that affects isolated segments of the spinal cord, resulting in bilateral motorsensory, and autonomic dysfunction. The suggested mechanisms of heroin-associated myelopathy include hypotension, a direct toxic effect of heroin, vasculitis, and hypersensitivity reaction [6]. Hypersensitivity remains the predominant theory since literature reports, implied that most patients with developed myelopathy had relapsed into heroin use after a period of abstinence [6].

TM causes an acute or subacute spinal cord dysfunction resulting in paresis, and autonomic impairment below the level of the lesion. The literature review highlights only 1 previously documented case of transverse myelitis after nasal insufflation of heroin in which the patient made a full recovery within 7 weeks [4]. There are no cases of cervical level transverse myelitis after consistent insufflation of heroin. The literature implicates most cases of cervical involvement occur after IV heroin abstinent patients’ relapse following an unspecified period of abstinence (incarceration, hospital admission, etc.) [3].

The diagnosis for transverse myelitis requires the exclusion of other definable causes of spinal cord inflammation such as spinal cord astrocytoma, multiple sclerosis, spinal cord infarction, and Lyme disease. The etiologies for transverse myelitis can be broadly classified as parainfectious, paraneoplastic, drug/toxin-induced, systemic autoimmune disorders, and acquired demyelinating diseases [5]. Some differentiating characteristics of a cervical astrocytoma include, homogenous cord expansion, nodular contrast enhancement, and an indolent clinical progression. Multiple sclerosis presents with a relapsing and remitting clinical course, affects 2 or less vertebral segments, and exhibits intracranial lesions.

The implications of a delayed or missed diagnosis are severe. However, treatment is not always efficacious. An evaluation of the entire spinal cord with MR imaging is paramount for diagnosis of transverse myelitis, thought thoracic evolve-

ment is most common. The length of spinal cord involvement classically includes 3 or more vertebral segments [5]. The goal of treatment is to blunt the immune response. Treatment options include 5 cycles of intravenous steroids, plasmapheresis, and intravenous immunoglobulins when refractory to first-line treatment. The goals for long-term management focus on neurorehabilitation and a multidisciplinary approach aimed at managing the various complications of spinal cord damage [5,6].

The annual incidence of TM is 1.34-4.6 cases per million (excluding multiple sclerosis related cases). Transverse myelitis occurrence follows a bimodal distribution with occurrences highest at the second and fourth decade of life [5].

Conclusion

This case presents an opportunity to consider a novel link concerning repeat heroin use and local tissue injury with concurrent inflammation. TM should remain a plausible differential for patients with a history of drug abuse (in particular, opiates), regardless of the route of entry (intranasal or intravenous). Early and timely identification with MR image assessment coupled with the commencement of immunotherapy is crucial to minimize or prevent future disability. Our patient eventually improved and regained motor function.

Consent

The patient provided verbal consent for publication of this case report and for any use of the accompanying images.

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