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Cervical Myelopathy Caused by Injections into the Neck

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Abstract

Three cases of longitudinally extensive cervical myelopathies temporally associated with neck injections are presented. The spinal cord injury was similar radiographically, despite a number of different needle approaches and substances injected. In recent years, there have been reports of an acute cervical myelopathy immediately following an injection procedure in the neck. Various explanations have been offered for this unfortunate complication, including (1) direct injection into the cord leading to traumatic injury, (2) injection of particulate matter into the arterial supply of the cord causing microvascular embolism and spinal cord infarction, and (3) intraneural injection of the chemical with centripetal spread of the injectant from the nerve trunk to the substance of the cord. The merits of each of these 3 mechanisms in explaining these cases are discussed. Albeit rare, acute cervical myelopathy should be considered a potential complication from any deep injection of chemicals into the neck.

Keywords

brachial plexus neuropathies, neuromuscular diseases, spinal cord injuries, spinal cord diseases, EMG/nerve conduction velocity, techniques

Introduction

In the 1950s, Moore and colleagues[1,2] described cases of acute-onset clinical "transverse myelitis" following paravertebral blocks with Efocaine. These patients developed immediate, severe bilateral leg weakness with only minimal improvement after many months of observation. Since then, there have been other case reports of cervical myelopathy following brachial plexus blocks and cervical epidural steroid injections. The spinal cord syndrome may present immediately upon injection or within minutes.[3] The clinical spectrum ranges from weakness and numbness of the ipsilateral arm and hand[4] to a Brown-Séquard syndrome[5]; bilateral motor deficits have also been described.[6-9] Magnetic resonance imaging (MRI) usually shows longitudinally extensive T2-hyperintense lesions, located eccentrically within the cord, ipsilateral to the injection site. Syrinx-like intramedullary fluid accumulations have also been described.

The literature on this subject consists of single case reports in which a number of different procedures were performed and outcome studies (case series) involving the same procedure. We present these three cases to highlight that this complication is not restricted to a single type of procedure. It may be that any deep injection in the neck carries some risk of this complication. Another purpose of our article is to raise awareness that nerves may serve as potential conduits for injected substances to reach the spinal cord. In the cervical epidural steroid injection literature, the possibility that an intraneural injection could cause this complication is rarely mentioned.

Case 1

A 28-year-old right-handed woman underwent a right brachial plexus block in preparation for right rotator cuff surgery. The needle was introduced posteriorly and parasagittal to the spine. Immediately upon injection of the anesthetic, she complained of severe right shoulder pain. Within a few minutes, she lost consciousness and had a respiratory arrest, prompting endotracheal intubation and mechanical ventilation; her blood pressure remained stable. The shoulder procedure was then completed without additional complications. Upon awakening, she reported transient deafness followed by tinnitus. Approximately 30 minutes later, she noticed severe pain in her left lateral hand and forearm. Her right upper extremity was diffusely numb and weak. She denied urinary or bowel symptoms.

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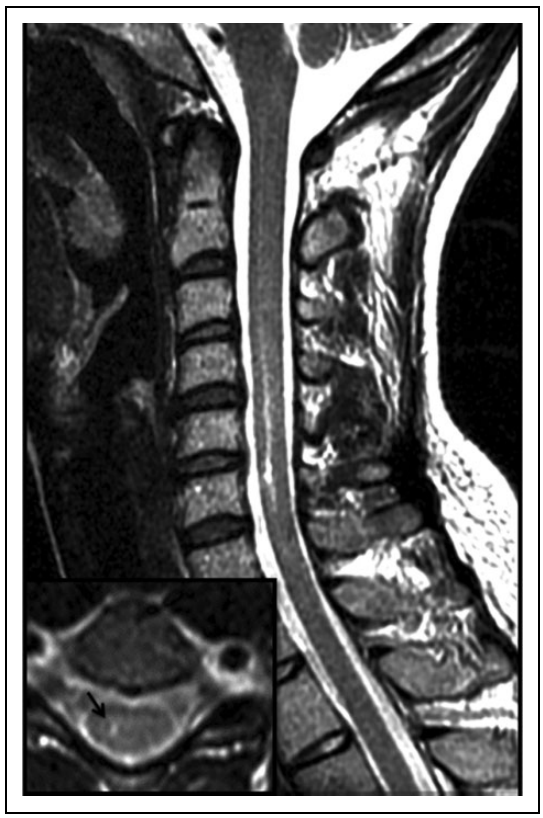

Figure 1. Cervical magnetic resonance imaging (MRI) with sagittal T2-weighted sequence demonstrating a linear hyperintensity from C3 to C7. Axial image (inset) shows abnormal signal in the right paracentral cord (arrow).

Magnetic resonance imaging of the cervical spine obtained 1 month after the injury (Figure 1) showed a linear T2-hyperintense lesion in the right paracentral region of the cord, extending from C3 to C7. The caliber of the cord was normal. An magnetic resonance (MR) neurogram of the brachial plexus demonstrated mild enlargement and high signal intensity in the right C8 nerve root and lower trunk. Magnetic resonance angiography of the neck did not show evidence of dissection.

She was referred to neurology 2 months after the injury. On examination, there was severe weakness, Medical Research Council (MRC) grade 2, of the right infraspinatus, deltoid, and triceps and moderate weakness (MRC grade 4) of all muscle groups distal to the elbow, including abductor pollicis brevis (APB) and abductor digiti minimi (ADM). Muscle tone was normal. Reflexes were depressed in the right upper extremity, except for a brisk triceps reflex. Reflexes in the left upper and bilateral lower extremities were normal. Light touch, vibration, and pain sensation were mildly and diffusely impaired in the right upper extremity.

Nerve conduction studies performed 2 months after the surgery revealed normal amplitudes for the right median (89 μV; normal > 15 μV), ulnar (61 μV; normal > 12 μV), and radial (70 μV; normal > 15 μV) sensory nerve action potentials as well as right median (APB; 13 mV; normal > 5 mV) and ulnar (ADM; 8 mV; normal > 5 mV) compound muscle action potentials. All motor and sensory nerve conduction velocities were normal. Concentric electromyography (EMG) of the right deltoid, biceps, triceps, and first dorsal interosseous muscles did not reveal evidence of denervation. There was evidence of mild to moderate suprasegmental weakness of the right deltoid, biceps, and triceps muscles due to either CNS injury or poor effort related to pain.

Her neuropathic pain improved with gabapentin. After several months, she had a modest improvement in the strength; perception of pain and vibration remained mildly impaired.

Case 2

A 61-year-old woman underwent a transforaminal cervical epidural steroid injection (TFCESI) at an outside hospital for chronic left shoulder and neck pain. The left C4 to C5 neural foramen was entered with a 25-gauge needle and injection of contrast material (1 cm^3 Omnipaque 300) confirmed that the needle tip was in the epidural space. A 1.5-cm^3 solution of 40 mg methylprednisolone acetate in 0.25% bupivacaine hydrochloride was then injected. There was no immediate adverse reaction to the injection. Several minutes after the procedure, the patient experienced severe burning pain in her right shoulder and paralysis and loss of pain and temperature sensation in her left upper extremity and left side of the head.

An MRI (Figure 2), obtained 6 days after the procedure, showed a T2-hyperintense lesion from C2 to C6 involving the left central and dorsal aspect of the cervical cord. An EMG study was not performed for this patient.

On examination 2 months after the injury, there was full strength in the limbs except for moderate weakness (MRC grade 3) of left supraspinatus, infraspinatus, deltoid, and biceps muscles. Muscle tone was normal. Reflexes were absent at the left biceps, trace at the left triceps, 1+ at left brachioradialis but 3+ at the left finger flexors, and 2 to 3+ in the right upper and bilateral lower extremities. Plantar reflexes were flexor bilaterally. Perception of pain and temperature was diminished on the left side, in the C2 to C8 dermatomes, most severely from C4 to C6. A year after the injection, she reported a modest improvement in her strength. Pain was fairly well controlled on gabapentin and celecoxib.

Case 3

Assisted by a friend, a 25-year-old drug user received an injection of methamphetamine into his neck. A 2- to 3-cm long needle was inserted superior to the right clavicle near the sternoclavicular joint. The goal was to inject the substance into a major blood vessel in the neck. Immediately after the injection, the patient noticed a burning sensation in his tongue and felt momentarily confused. After 30 minutes, he noticed tightening of his right facial muscles, progressive weakness of his right arm and leg, and dysesthesia of his entire right side.

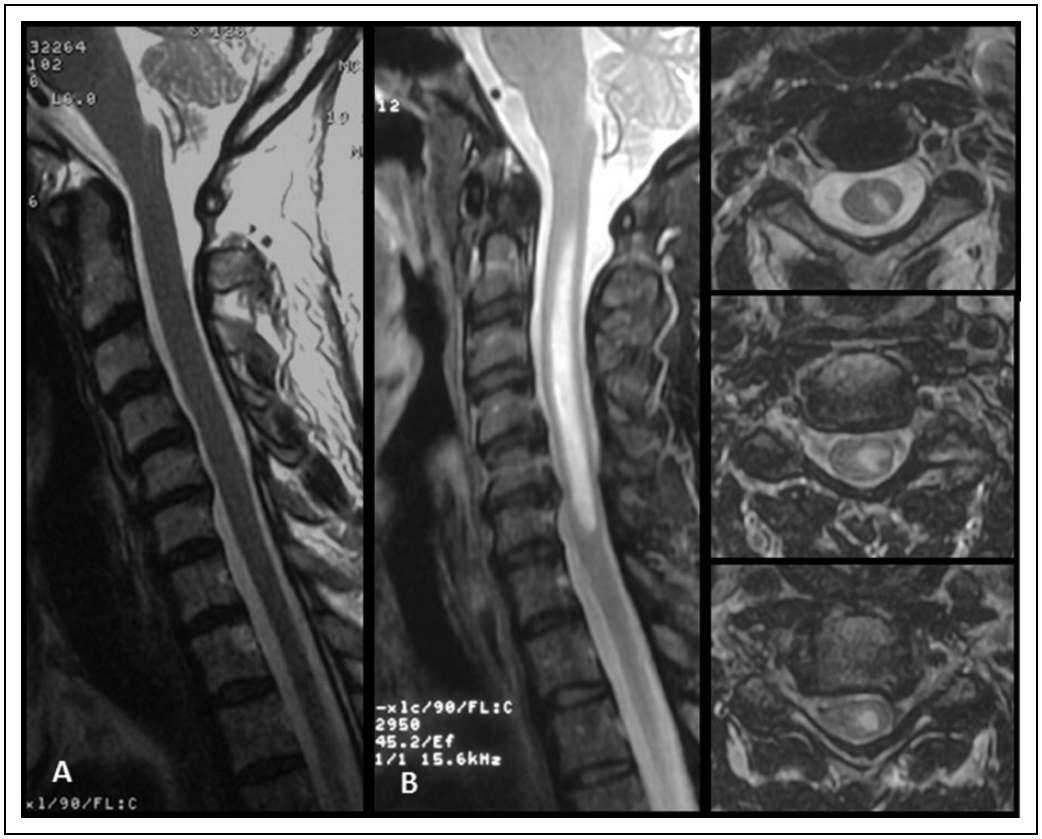

Figure 2. Cervical magnetic resonance imaging (MRI) pre (A) and post (B) cervical injection demonstrating development of a T2-hyperintense lesion from C2 to C6. Selected axial images (right panel) demonstrating involvement of the left central and dorsal aspect of the cord at C2, C4, and C5, top to bottom, respectively.

Neurologic examination demonstrated mild right eyelid ptosis but no miosis, right hemiplegia, right-sided allodynia sparing the face, and loss of pinprick and temperature sensation below the C4 dermatome on the left side; proprioception was intact bilaterally. An MR scan showed T2-signal prolongation and mild expansion of the cervical cord from the C3 to C5 levels, mainly located in the gray matter of the right side of the cord. During his hospitalization, he experienced severe neuropathic pain in the right arm and leg, which responded better to lorazepam than to gabapentin and morphine. After 1 month, he was able to walk, albeit unsteadily, and had recovered some use of his right hand. This case was previously reported by one of the authors.[5]

Discussion

In all 3 cases, cervical myelopathy immediately followed injections into the neck region. In the first 2 cases, clinical findings were largely restricted to the upper limb ipsilateral to the injection site. We suspect that the myelopathy could have been confined to the gray matter sparing the corticospinal tracts so that the arm weakness was lower motor neuron in type. The depressed reflexes in the upper limbs in the first 2 cases were likely caused by injury to the spinal circuitry involved in the reflex arcs. In the first case, an MR neurogram showed some abnormal signal in the inferior brachial plexus and C8 root, but there was no electrophysiologic evidence of a brachial plexopathy, so any injury to those structures was probably mild. In light of the electrodiagnostic results, we conclude her strength was limited mainly by spinal-mediated neuropathic pain, although upper motor neuron weakness related to the myelopathy could not be excluded. An electrodiagnostic study was not performed for the second case because of the difficulty in electrophyisologically differentiating root versus cord pathology. In case 3, eyelid ptosis was probably caused by injury to the ipsilateral descending spinal sympathetic fibers in the cervical spinal cord.

In earlier reports, direct injection into the cord has been postulated as the mechanism of injury, resulting in injury due to mass effect, local anesthetic or adjuvant toxicity, edema, or hematoma formation.[10] In our 3 cases, however, direct puncture of the cord seems unlikely. In case 1, the needle was directed in an orientation (posterior, parasagittal approach) in which vertebral bone would have blocked entry into the central canal. In case 2, needle was guided fluoroscopically and a test injection with contrast material confirmed the epidural location of the needle tip. In case 3, the needle was too short to reach a neural foramen.

A second possible mechanism is vascular, either by injury of a radicular or vertebral artery leading to vasospasm or dissection or by injection of particulate matter resulting in thromboembolism. Inadvertent arterial injection during an epidural steroid procedure is surprisingly common. A prospective study of 337 patients undergoing TFCESI revealed a 19% incidence of penetration of the adjacent radicular artery.[11] If arterial injection occurs, the particulate nature of the injected material could cause embolic infarction. This theory has become popular based on case reports in which patients undergoing TFCESIs sustained infarctions of the cord and posterior brain without evidence of arterial dissection. Animal studies showing that arterial injections of particulate steroids can cause cord infarcts are lacking.

In case 1, an arterial injection of the anesthetic into a vascular branch feeding the vertebral artery could have explained the tinnitus. Given that this was a nerve block procedure, there was presumably no particulate matter in the injectant, so it seems unlikely that embolism and infarction of the cord could have occurred. Inadvertent injection of a radicular artery could have occurred in case 2, although contrast injection immediately prior to the therapeutic injection did not show arterial penetration. In case 3, a vascular mechanism of injury also seems possible; the injection occurred in the vicinity of the vertebral artery origin, and the immediate burning sensation in the tongue and the occurrence of confusion suggest that some of the injected material may have passed into the arterial supply of the brain. It is possible that particulate matter was admixed with the illicit drug.

A third possible mechanism is injection of a peripheral nerve or nerve root with intraneural, centripetal spread of the injectant to the cord. This mechanism has been demonstrated in animal studies. Moore and colleagues demonstrated central spread of Efocaine, colored with methylene blue, following intraneural injection into the lumbar and brachial plexus of a killed monkey.[1,2] The colored Efocaine reached the spinal cord in 2 to 5 minutes, initially spreading up and down the surface of the cord subpially. After 10 to 15 minutes, the spinal fluid became tinged, suggesting that the Efocaine had crossed the pia mater. The fluid became heavily stained after 35 to 40 minutes. These experiments demonstrated that substances injected into the nerves distal to the intervertebral foramen could spread to the cord and, later, the spinal fluid. Moore concluded that the perineurial spaces could serve as highways to the CNS.

In a series of additional experiments, the same investigators injected 0.5 to 1.5 cm^3 of Efocaine into the lumbar nerves of 11 monkeys under pentobarbital sodium anesthesia.[1] Respiratory paralysis developed in 2 monkeys after the procedure leading to death. Respiratory paralysis occurred in a third monkey, but it was successfully kept alive with mechanical ventilation. After this monkey recovered from the procedure, a clinical transverse myelitis developed. In the other monkeys, leg weakness developed ipsilateral to the site of injection. Elevated anesthetic levels were seen in most of the monkeys' CSF. The levels were the highest when the resistance to the injection was the least. These studies demonstrated that a myelopathy (and sometimes brain stem anesthesia) could occur after a peripheral injection.

Selander and Sjostrand[12] demonstrated that intrafascicular injections were associated with the rapid centripetal spread of the injectant to the cord, whereas extrafascicular injections led only to local fluid accumulations. Certain local anesthetics can produce dose-related neural toxicity. Central cord necrosis and subpial vacuolation can occur with intrathecal injections of 8% tetracaine or 32% lidocaine.[13] In addition to a direct toxic effect, the injected substances may increase the interstitial pressure enough to embarrass tissue perfusion pressure leading to ischemic injury of the cord. However, experimental evidence supporting this latter mechanism of injury is lacking.

Direct injection into the plexus or nerve root may have occurred in case 1. Magnetic resonance neurography demonstrated enlargement and increased signal intensity in the C8 nerve root and lower trunk, indicating that the needle tip may have entered those structures. The occurrence of arm pain during the injection also supports this explanation. Interestingly, the patient had a respiratory arrest during the procedure, which has been reported in some intraneural injections. In the second case, there was no local pain upon injection, so an intraneural injection may not be likely. It is conceivable, however, than an injection into the motor portion of the root may not be associated with pain. In the TFCESI literature, the possibility that the needle tip could enter the root and cause neurological complications is rarely entertained.

Unfortunately, it is difficult to be certain which of these 3 mechanisms applies to our cases. Nevertheless, the neurohospitalist benefits from recognizing that the needle tip during transforaminal injections ventures very close to the radicular vessels that supply the cord. Also, chemicals inadvertently injected into a nerve root or plexus can spread directly to the spinal cord. Owing to the dense vascular and neural anatomy of the region, we feel that acute cervical myelopathy is a potential complication from any chemical injection into the neck.

Certain practices can be adopted to reduce the chances for unintended intraneural or intravascular injections. These include minimizing sedation during regional anesthesia procedures, immediate cessation of the procedure if the patient reports pain, and the use of test injections and fluoroscopic or computed tomography guidance for transforaminal injections.

Declaration of Conflicting Interests

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