CASE REPORTS


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Cranial Nerve X and XII Paralysis (Tapia’s Syndrome) after an Interscalene Brachial Plexus Block for a Left Shoulder Mumford Procedure

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IN 1904 the Spanish otolaryngologist Antonio Garcia Tapia described a syndrome of lower cranial nerve palsies in a bullfighter who had sustained a work-related injury. While inserting bandilleras, he was gored in the right side of the neck. He was immediately rendered aphonic and his tongue “dragged” when he spoke. Several hours later left hemiparesis developed. Tapia examined the bullfighter and noted the paralysis of the right vocal cord and deviation of the tongue to the right during protrusion. He hypothesized that the bullfighter suffered direct trauma to cranial nerves X and XII and then an embolus to the brain from a contusion of the nearby carotid artery. Tapia’s eponym subsequently has been associated with various cranial nerve syndromes, with or without hemiparesis, in which cranial nerve X and XII palsies are consistent features.1

Interscalene block of the brachial plexus is overall a safe technique in regional anesthesia, but numerous complications have been described.2–4 Transient hoarseness and a temporary Horner’s syndrome are common.5

We report a case of injury to cranial nerves X and XII—Tapia’s syndrome—in a patient who received an interscalene block for a Mumford procedure.

Case Report

The patient is a 44-yr-old man who presented with a left shoulder injury requiring surgical repair. Before the procedure, he received an interscalene brachial plexus block in the left side of the neck. With the patient’s head turned to the opposite side, a 21-gauge needle was introduced into the interscalene groove at the level of the cricoid, and 50 ml of 1% ropivacaine with 0.2% tetracaine and 1:200,000 epinephrine was injected, aspirating at 5 ml intervals. At some point the patient experienced exquisite left-sided neck pain and perceived a change of sensation in his tongue. At examination he was hoarse and found to have a left Horner’s Syndrome. He retained sensation below the axilla after the first injection; therefore, after several minutes, it was repeated with 20 ml of the anesthetic solution with good results. He was then administered general anesthesia and the operation was performed.

Examination 3 days after the operation revealed deviation of the tongue to the left during protrusion, left vocal cord paralysis, and a persistent partial left Horner’s syndrome. Computerized axial tomography of the head and neck showed only increased prominence of left tonsillar fossa soft tissues. Magnetic resonance imaging of the brain and neck 2 weeks postoperatively showed fatty degeneration of the tongue on the left and tortuous carotid arteries, but otherwise was normal. Four weeks after surgery the patient was evaluated in the neurology clinic. At that time he looked well, but continued to evince pain when elevating the shoulders, and he spoke in a hoarse whisper. There was decreased sensation on the left side of the palate, but the palate elevated symmetrically. The tongue was atrophied on the left and still deviated to the left during protrusion. The remainder of the neurologic examination was normal. Magnetic resonance imaging and magnetic resonance angiography of the brain and neck performed 8 weeks after the operation and carotid ultrasonography performed 10 weeks after

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surgery showed tortuous carotid arteries, but results were otherwise normal.

**Discussion**

Ipsilateral cranial nerves X and XII palsies, the essential ingredients of Tapia’s syndrome, developed in this patient. The sudden onset suggests that it was a vascular event. It was probably not caused by direct injury from the needle to cranial nerves X and XII because the injection was too far caudal to the anatomic crossing of cranial nerve XII at the carotid sheath. However, the ascending pharyngeal branch of the carotid artery provides exclusive blood supply to cranial nerves X and XII, and carotid dissection after minor trauma is a well-described cause of sudden cranial nerve X and XII palsies. Patients with dissections are typically male, are in their mid forties, and have tortuous carotid arteries. Horner’s syndrome is often seen after carotid injury. Therefore, it is very likely that the patient incurred a dissection in the carotid system involving the ascending pharyngeal artery. The possibility that the injury occurred during interscalene injection cannot be excluded. The fact that hoarseness and Horner’s syndrome are common side effects of this anesthetic procedure may have resulted in delay of diagnosis; therefore, even when the concurrence of cranial nerves X and XII palsies and persistence of Horner’s syndrome was confirmed some 5 days later, the full significance was not appreciated. By the time vascular imaging was obtained the dissection either had healed or was too small to be resolved by noninvasive methods.

Tapia’s bullfighter presented with the same cranial nerve deficits, and a hemiparesis developed, presumably from an embolus from the carotid artery to the brain. We believe that our patient was at risk for the same. We recommend that when a patient undergoes an interscalene block and hoarseness develops, tongue protrusion should be examined and documented. If there is deviation of the tongue toward the site of the injection, the procedure should be terminated and carotid angiography should be considered to evaluate the need for anticoagulation.

**References**