Multiple cranial nerve palsies due to a hyperextension injury to the cervical spine

Case report

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The case of a patient with multiple bilateral cranial nerve palsies and spinal cord sparing secondary to a stable hyperextension injury to C-1 is presented.

Key words: cranial nerve, hyperextension, spinal trauma, cervical spine, palsy

Trauma to the upper cervical spine will on rare occasion produce lower cranial nerve palsies.24,9 Case reports where these palsies have been bilateral have always included evidence of damage to the spinal cord extending as high as the cervicomedullary decussation of the motor tracts.12,4,8,10 In reports where no injury to the cord or brain stem has been noted, only the sixth cranial nerve has been identified as being damaged.5,7 The conclusion has thus been that mild neck trauma can stretch the sixth cranial nerve without producing central nervous system (CNS) injury; if the trauma is severe enough to produce lower cranial nerve palsies, then damage to the cord and/or brain stem will result as well.1,8,10 We have recently seen a patient whose clinical presentation runs counter to these conclusions. Without other evidence of CNS injury, this patient presented with bilateral palsy of the ninth through 12th cranial nerves plus a unilateral sixth nerve palsy secondary to hyperextension of the cervical spine.

Case Report

This 28-year-old woman was brought to the emergency room of a local hospital after being involved in a head-on motor-vehicle accident. She reported having struck her chest and abdomen against the steering wheel and her face against the windshield. She was awake, alert, and moving all four extremities. Due to acute respiratory distress, she was intubated. Chest x-ray films revealed bilateral pneumothoraces with air and fluid levels, for which chest tubes were placed. Cervical spine x-ray films suggested the presence of a fracture of the ring of C-1 for which a Philadelphia collar restraint was imposed. Abdominal exploration revealed mesenteric tears and a jejunal laceration. Repeat chest x-ray films suggested mediastinal widening, and aortography revealed an intimal tear of the innominate artery. The patient was then transferred to the Maryland Institute for Emergency Medical Services Systems.

Examination. The intubated patient was noted to be awake, alert, and following commands. She had normal function of all four extremities. Her pupils were equal and reactive, and her face was swollen but with symmetrical movement. There was a left sixth nerve palsy. Pulse was 126/min, and blood pressure was 230/40 mm Hg, although there was no prior history of hypertension. A sternotomy with repair of the tear of the innominate artery was performed without complication. Postoperatively, she remained hypertensive, with systolic pressures consistently greater than 190 mm Hg for 48 hours, and then intermittently elevated for the next week. Tomograms and flexion-extension films were interpreted as indicating a stable fracture of C-1. Neck restraint was maintained by a Yale brace (Philadelphia collar with thoracoabdominal extension).

Following extubation, the patient was found to have significant difficulty handling her oral secretions, and there was a question of aspiration on at least two occasions. Examination revealed dysarthric and nasal
speech, with absent gag reflexes bilaterally. Swallowing was difficult, resulting in the need for frequent suction to clear secretions. The patient was unable to protrude her tongue or move it laterally, and both sternocleidomastoid muscles were weak. Direct laryngoscopy showed paresis of both vocal cords. Long-latency somatosensory and auditory evoked potentials were normal. Computerized tomography (CT) and plain tomography of the skull base were normal.

Due to her neurological deficits, she was fed in the upright position, which helped decrease her swallowing difficulties. The gag reflex showed some return of function over the next 48 hours, and a tracheostomy was avoided. Her subsequent hospital course was one of gradual improvement. At the time of discharge 4 weeks after admission, speech had improved noticeably. She was normotensive, had a stronger gag reflex, but could not protrude her tongue past her teeth (Fig. 1 left). Follow-up review in the outpatient clinic has showed continued recovery. She is now able to protrude her tongue past her lips (Fig. 1 right), moving it toward the left but not toward the right side. Speech has continued to improve and is practically normal. Strength in the sternocleidomastoid muscles is improved, and the sixth nerve palsy has resolved. Blood pressure is stable.

Discussion

This patient showed signs of a unilateral injury to the sixth cranial nerve, as well as bilateral injury to the lower cranial nerves nine through 12. In addition, her hypertension was noteworthy. Hypertension has been shown to result from operative section and/or intraoperative stretching of the ninth cranial nerve, presumably due to the presence of fibers from the carotid sinus. This patient's markedly elevated blood pressure, which gradually returned to a normal level, suggests that stretch may have been the mechanism of injury in this case.

This conclusion is further supported by the presence of the sixth nerve palsy. Due to its long intracranial course and its passage beneath the petrosphenoid (Grüber's) ligament, the sixth nerve is predisposed to stretch injury. The mechanism of injury to the spine in this patient was most likely one of hyperextension, evidenced both by the history and the presence of associated tears of the innominate artery. In addition, there was no CT or tomography evidence of basilar skull fractures, a potential cause of cranial nerve palsies. Therefore, the lower cranial nerve palsies were probably secondary to stretch injury at the cranio cervical junction as a result of the hyperextension injury to the upper cervical spine.

This case is particularly noteworthy due to the absence of associated spinal cord or brain dysfunction. Previous case reports of multiple lower cranial nerve involvement have been associated with nonfatal atlanto-occipital dislocation and hangman's fracture. In all instances, there has been evidence of cord or brain-stem dysfunction. This patient had no clinical or electrophysiological signs of such dysfunction. As in these other cases, however, recovery of the cranial nerve function is usually seen early, and is progressive.

References