

I Can't Move My Face! A Case of Bilateral Facial Palsy

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The authors present a case of bilateral facial palsy in a 52-year-old man. The patient presented to an emergency department in Pennsylvania, describing left-sided neck pain and headache from “sleeping wrong,” symptoms which eventually progressed to facial diplegia by his fourth visit in 2 weeks. His admitting diagnosis was Bell palsy; he was ultimately tested for and found to have Lyme disease. Delay in treatment of patients with Lyme disease may lead to bilateral facial paralysis and disease progression. Thorough history taking, physical examination, and scrutiny of prior records are important elements of identifying and treating patients such as these (ie, whose vague symptoms progress to facial diplegia) appropriately.

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Bilateral facial palsy is extremely rare, comprising 0.3% to 2% of all facial paralysis cases.¹ Idiopathic Bell palsy is a common cause, but other reasons—such as Lyme disease, Guillain-Barré syndrome, neurosarcoidosis, meningitis, leukemia, viral infections, syphilis, and basilar skull fractures—must be considered and ruled out by diagnostic testing or clinical presentation.¹ We report a case of a 52-year-old man who initially presented with cervical pain, then with bilateral facial palsy, which led to a diagnosis of Lyme disease.

Report of Case

A 52-year-old man with a history of cigarette smoking presented to a Pennsylvania emergency department (ED) with a chief complaint of left neck pain (“stiff neck”) and headache, which he stated had occurred from “sleeping wrong” 2 days earlier. He expressed concern regarding a tick being on his left ear 2 weeks before initial presentation, but he also reported that it never implanted. He displayed no rash, and his medical history was otherwise unremarkable. His clinical examination was consistent with cervical strain. He was discharged from the hospital and was given cyclobenzaprine 10 mg every 8 hours and naproxen 500 mg every 12 hours for management of his symptoms.

The patient returned 6 days later with persistent cervical pain and muscle spasm; the pain had gotten worse and was now radiating throughout his left arm and shoulder. His neck pain was reproduced with movement of the trapezius and deltoid muscles. At this visit, a nonspecific skin rash was noted on the patient’s left forearm along with a small bug bite. He continued to deny any other symptoms besides the constant pain. Naproxen was continued with the addition of diazepam and a narcotic. He was told not to work for 2 days and given the recommendation for outpatient magnetic resonance imaging if his pain did not improve.

Nine days after the initial visit, the patient returned to the ED complaining of difficulty swallowing and continued left-sided neck pain that had progressed to the left side of his head and his left ear. In addition, he complained of a hoarse voice. He was able to swallow water without difficulty, and neurologic examination showed no evidence of abnormalities. He denied any fever, chills, numbness, difficulty breathing, chest pain, sputum production, visual disturbances, dizziness, blackouts, migraines, or head trauma. The patient was concerned that the lump he felt in his throat was from a possible allergic reaction from the prescribed narcotic medication (Percocet [oxycodone/acetaminophen], 5 mg/325 mg every 6 hours as needed) that he had taken the previous day. On this presumption, he was given diphenhydramine and prednisone in the ED. The treating physicians were not aware of the patient's first visit with a history of tick exposure, as those medical records were not reviewed. Subsequently, because of his persistent neck pain and progression of symptoms, computed tomographic scans of the head and neck were obtained, which revealed left palantine tonsillar asymmetry. The patient's chart indicated he had a 40-pack-year smoking history, so the asymmetry was presumed to be malignant and the patient was referred to an otolaryngologist. He was discharged with a 5-day course of amoxicillin/clavulanate 875 mg every 12 hours and prednisone 20 mg twice daily.

Two weeks after his initial visit (his fourth visit), the patient presented with complete bilateral facial paralysis; he reported having trouble smiling, eating, and closing his eyes. Interestingly, his pain had completely resolved. He described his face as feeling "heavy" and how he had to push food to the back of his mouth when chewing. Despite these symptoms, he was still able to swallow. On examination, he exhibited some ptosis bilaterally and minimal ability to raise or close his eyelids, as well as orbicularis oris weakness with puffing in his cheeks. There was also asymmetry of the nasolabial folds, with weakness more pronounced on the right side. His gag reflex was unaffected and his tongue remained midline with minimal weakness to lateral movement. Sensation was intact throughout the patient's face, and the remainder of the examination was unremarkable. His vital signs were within normal limits; the patient had no leu-

kocytosis. He had attempted to make an appointment with the otolaryngologist, but he stated that the office told him he may have a tumor and to return to the ED.

The patient's Lyme titers were obtained, and both IgM and total levels were elevated. A neurologist was consulted and additional tests were performed. A magnetic resonance image revealed extensive abnormal enhancement of cranial nerves III, V, VII, and XI. A lumbar puncture exhibited an elevated white blood cell count of $78 \times 10^9/L$, which was predominantly lymphocytes, and an elevated total protein level of 173 g/L. We ordered additional tests for demyelinating diseases—such as multiple sclerosis and Guillain-Barré syndrome, as well as myasthenia gravis and other autoimmune diseases—and the results were negative for each disease. There was also concern for possible cancer given the patient's history of smoking; no evidence of malignancies or sarcoidoses, however, was identifiable on computed tomographic scans of the chest, abdomen, and pelvis. The patient was eventually admitted to the hospital's general medicine service, received a diagnosis of Lyme disease, and given intravenous ceftriaxone IV—all of which improved his condition during the remainder of his hospital visit.

Comment

Bilateral facial paralysis (diplegia) should be identified and managed early because of its various life-threatening causes. Nonetheless, diagnosing diplegia accurately can be challenging. For the condition to be considered simultaneous-bilateral—as opposed to unilateral-recurrent—both sides of the face must be involved within 30 days of each other.² Bilateral involvement usually implicates a systemic process or disease as opposed to the most common cause (ie, idiopathy) in unilateral facial paralysis.² Patient history and results of examination should include recent illnesses and rashes (eg, erythema chronicum migrans of Lyme disease, herpes simplex virus lesions).

The differential diagnosis for facial paralysis can be separated into infectious and noninfectious. Infectious causes include bacterial (eg, tuberculosis, tetanus, otitis media, meningitis, brain stem encephalitis, Lyme dis-

ease, mycoplasma, syphilis, Hansen disease [leprosy], mastoiditis), fungal (eg, cryptococcal), and viral (eg, herpes simplex virus type 1 and type 2, varicella zoster virus, influenza, polio, human immunodeficiency virus, Epstein-Barr virus [ie, mononucleosis], cytomegalovirus, Guillain-Barré syndrome) diseases.³⁻⁵ Noninfectious causes include stroke, pre-eclampsia, trauma, lymphoma, Melkersson-Rosenthal syndrome, Möbius syndrome, tumor, myasthenia gravis, diabetes mellitus, vasculitis, sarcoidosis, benign intracranial hypertension, leukemia, idiopathic cranial neuropathy, and adverse reaction to medication.^{4,5} Diagnostic blood work should include a complete blood cell count, fluorescent treponemal antibody test, human immunodeficiency virus test, fasting glucose level, erythrocyte sedimentation rate, Lyme titer, and antinuclear antibody level. A lumbar puncture can be performed to evaluate cell counts, as well as bacterial or viral content.

Although its incidence varies geographically, generally facial paralysis is caused by Lyme disease 36% of the time, followed by Guillain-Barré syndrome (5%), trauma (4%), sarcoidosis (0.9%), and AIDS (0.9%, particularly at the time of seroconversion).² Persistent misconceptions about available diagnostic tools and nervous system involvement for Lyme disease continue.⁶ People of any sex or age can contract Lyme disease; however, the disease occurs primarily in men, with the greatest incidence in the age groups of 5 to 9 years and 55 to 59 years.⁷ Furthermore, initial symptoms can begin as late as 30 days after a tick bite, although this presentation is uncommon.⁷ Lyme disease can manifest with various constitutional, cardiac, musculoskeletal, and neurologic manifestations, and erythema chronicum migrans is not always diagnostically predictive of the disease.⁸ Common and often nonspecific symptoms that accompany Lyme disease are joint swelling, headache, fever, diffuse aches and pains (particularly pain on neck flexion), malaise, cognitive slowing, and cranial nerve palsies (usually facial nerve palsy).^{6,7,9} Interestingly, out of the approximately 30,000 reported US Lyme disease cases annually, approximately 8% manifest as facial nerve palsy.¹⁰

As with the patient in the present report, vague complaints without the typical bull's-eye rash (ie, erythema chronicum migrans) may be the only clues and symptoms of Lyme disease early on. The patient denied a tick

bite, even though he had reported a tick on his ear—which corresponds to the 75% of US patients who do not recall the actual tick bite.^{8,9} Had the treating providers been suspicious enough from his initial exposure to order a Lyme disease test, his progression to bilateral palsy may have been preventable. Early recognition of this patient's Lyme disease would have meant a more appropriate use of medication therapy and fewer ED visits.

Conclusion

A thorough history and physical examination, as well as careful review of existing records, is critical in leading the clinician in the direction of diagnosing Lyme disease and excluding Guillain-Barré syndrome, human immunodeficiency virus, meningitis, tuberculosis, neoplasms, and other illnesses. Such an approach to the differential diagnosis will also help contain costs by eliminating unnecessary testing. Most patients can recover uneventfully from Lyme disease if it is diagnosed and managed early.

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