Drooping After Scoping: A Rare Case of Peripheral Facial Nerve Palsy After Routine Esophagogastroduodenoscopy

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ABSTRACT

Peripheral facial nerve palsy is a prevalent type of mononeuropathy that can have a variety of etiologies. Facial nerve damage because of esophagogastroduodenoscopy, however, is exceedingly rare and has only been reported in 1 patient. We report the first case in the United States of a patient who developed left-sided facial nerve palsy after a routine esophagogastroduodenoscopy, with little meaningful recovery of nerve function. We hope to bring awareness to gastroenterologists of this rare complication with potential long-term detrimental effects that can be avoided with the adjustment of equipment and patient position before the procedure.

INTRODUCTION

Peripheral facial nerve palsy (FNP) is a common type of mononeuropathy that can be idiopathic or a result of secondary causes, such as bacterial or viral infections, trauma, stroke, intracranial hemorrhage, malignancy, diabetes, autoimmune diseases, and medication side effects.1 Idiopathic FNP is also known as Bell palsy. It is most commonly unilateral and results in the inability to make voluntary facial movements.2 This disorder is usually self-limited with spontaneous resolution normally observed within 3–5 months.3 Nonetheless, in some cases, it can lead to severe long-term outcomes, such as facial disfigurement and psychological trauma.2 FNP because of external compression has been reported in multiple case reports where a patient was undergoing general anesthesia for surgery.4,5 However, FNP from endoscopic procedures is rare and has only been documented in 1 case report from South Korea.6 We report the first case in the United States of a patient who developed left-sided FNP after a routine esophagogastroduodenoscopy (EGD).

CASE REPORT

A 49-year-old woman with a history of alcoholic cirrhosis, hepatitis C, diffuse large B-cell lymphoma, hypothyroidism, diabetes, and hypertension presented to our hospital for routine EGD for esophageal varices screening. On examination, her blood pressure (BP) was 150/80 mm Hg, temperature 36.6°C, pulse 84 beats per minute, oxygen saturation of 99% on room air, weight 80.7 kg, height 1.63 m, and body mass index of 30.55 kg/m². The procedure was performed with monitored anesthesia care with no intubation required. Propofol was given at 100 mg bolus followed by a rate of 150 mcg/kg/min. Other medications included 50 mcg of fentanyl, 150 mg of lidocaine, 0.2 mg of glycopyrrolate, 200 mcg of phenylephrine, and 4 mcg of ondansetron. The total anesthesia time was 32 minutes. Vitals during endoscopy showed systolic BP range of 75–200 mm Hg and diastolic BP range of 50–100 mm Hg, pulse of 75–100 beats per minute, and oxygen saturation of 97%–100% with FiO₂ of 58%–98%. Three columns of large varices were banded with incomplete eradication. An antral nonbleeding erosion was biopsied. The procedure was without any complications, and the patient was discharged home (Figures 1 and 2).

Five days later, she presented to an outside hospital with swelling, numbness, and drooping of her left face, along with dryness and inability to close her left eye. The symptoms started on the same day as her EGD and had gotten progressively worse since then. She...
denied any fever, malaise, speech or visual changes, deafness, tinnitus, dizziness, gait abnormalities, headaches, cough, cold symptoms, mouth sores, or skin vesicles. Physical examination was significant for left facial droop, loss of nasolabial fold, inability to wrinkle the left side of her forehead, and inability to close her left eye (Figure 1). A blood test was negative for human immunodeficiency virus antigen and antibody and herpes simplex virus (HSV) on a polymerase chain reaction test. No electrolyte abnormalities were noted. Varicella-zoster virus (VZV) antibodies and Lyme disease serology were not checked because of low clinical suspicion. Head computed tomography was declined by the patient. She was diagnosed with idiopathic FNP and managed conservatively with gentamicin ophthalmic ointment to reduce dryness. Two days later, the patient followed up with her primary care doctor and was started on a 10-day prednisone taper because of persistent facial droop. At the 3-month follow-up, only mild symptom improvement was noted. A second round of prednisone was prescribed for 14 days along with a course of empiric valacyclovir for 7 days. Despite the 2 courses of prednisone and valacyclovir, the patient’s peripheral neuropathy persisted with minor improvement at the 5-month follow-up (Figure 2).

**DISCUSSION**

Idiopathic FNP or Bell palsy accounts for approximately 75% of all cases of peripheral FNP, with an incidence rate of 20–25 per 100,000 and a propensity toward those between the ages of 15–45 years. Pregnancy and diabetes are 2 of the risk factors associated with it. It is a diagnosis of exclusion that requires ruling out secondary causes, such as infections, trauma, diabetes, autoimmune diseases, malignancy, stroke, intracranial hemorrhages, and medications. Patients with FNP, fever, malaise, and painful vesicular eruptions should raise suspicion for HSV and VZV. In addition, Lyme disease can present with FNP, rash, and arthralgias and is most commonly found in the Northeastern United States associated with tick bites. Autoimmune conditions such as Guillain-Barre syndrome should be suspected in someone with concurrent ascending paralysis and polyneuropathy. Medication-induced FNP is rare but has been reported with interferon and linezolid.

Our patient had no recent travel, new sexual partners, trauma, or new medication use. She had no systemic or neurological symptoms to suggest autoimmune disease. She had no painful skin lesion, fever, or malaise to suggest HSV or VZV. She denied any tick bites and did not live in a region endemic for Lyme disease. Her laboratory tests showed no signs of infection or electrolyte derangement, and her head computed
tomography was unremarkable. Given the acute onset of her symptoms hours after her EGD, the most likely etiology of her FNP was believed to be mechanical compression of the facial nerve during EGD in the background of diabetes that puts her at a higher risk for FNP. It is possible that the patient’s head was not aligned properly with her body, compounded by the fact that her oxygen mask and/or mouthpiece band were too tight, subsequently leading to compression of the left facial nerve when she was lying in a left lateral decubitus position during the procedure. To the best of our knowledge, there has only been 1 case report in the literature that documented facial paralysis after therapeutic endoscopy due to extrinsic compression, where the patient had complete recovery in 2 weeks with only conservative management. A few surgical case reports also showed that mechanical compression from a forward digital pressure at the mandible with a tight mask seal can cause facial paresis in surgical patients undergoing general anesthesia.

Overall, 80%–85% of the patients have complete spontaneous recovery within 3 months; however, full recovery is not as likely if there has been no improvement after 6 months of symptom onset. This small subset of patients may experience permanent sequelae, such as persisting facial weakness, contractures, facial spasm, and synkinesis. There is a high likelihood that our patient will have some degree of permanent nerve damage given that her facial paresis has not completely resolved after 5 months despite being administered prednisone and valacyclovir. This is the first reported case, to the best of our knowledge, of a patient developing FNP from mechanical nerve compression during a procedure with little meaningful recovery of nerve function. Therefore, it is important for gastroenterologists to be aware of this rare complication of peripheral FNP from endoscopic procedures that require the use of an oxygen mask and mouthpiece and maintaining a body position that may compress the facial nerve. Misalignment of the head and a tight mask/mouthpiece band are elements that can be easily adjusted and avoided to prevent the development of FNP along with its long-term detrimental complications.

DISCLOSURES

Author contributions: All authors contributed equally to this article. Y. Tu is the article guarantor.

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