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■ 증례보고 ■

Facial paralysis after spine surgery
－A case report－

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Idiopathic facial nerve paralysis after surgery is not common but has clinical significance. We report a case of facial nerve paralysis in the immediate postanesthetic period after cervical spine surgery. A 41-year-old man with cervical herniated disc was scheduled for cervical laminectomy. After uneventful surgery, he suffered from left facial numbness and weakness. Imaging study and audiogram couldn't reveal any anatomic abnormality except Thornwaldt cyst. Conservative treatment with steroids and antivirals resolved his symptoms until 16th day after surgery. **(Anesth Pain Med 2009; 4: 187～189)**

Key Words: complication, facial nerve paralysis, general anesthesia, spine surgery.

Facial nerve paralysis after general anesthesia is not common, but well documented. The most common cause of facial paralysis is Bell's palsy.[1] In regard to nerve injuries related with general anesthesia, there are two basic mechanisms.[2] Stretch injury occurs more commonly in the nerves which course superficially for long distances. On the other hand, compression injury usually develops in the nerves that pass over bony surface. We would like to report a case of facial nerve paralysis after uncomplicated cervical laminectomy and review the related papers to decrease this complication.

CASE REPORT

A 41-year-old man complaining of left fourth and fifth fingers' numbness and neck pain was admitted. Imaging studies revealed herniated cervical disc through C4 to C7. He was scheduled for cervical laminectomy. He denied any previous medical illnesses including diabetes, hypertension and recent infection. Physical examination and preoperative laboratory tests did not reveal any other abnormalities that are not related with his chief complaint. Awake nasotracheal intubation was successfully performed with the assist of fiberoptic bronchoscope. The internal diameter of the nasotracheal tube was 7.0 mm. Intravenous fentanyl 100 μg and topical anesthesia over nasopharyngeal mucosa with 10% lidocaine spray made it easy to work. Anesthesia induction was conducted with 250 mg of thiopental, and 8 mg of vecuronium. Anesthesia was maintained with 50% of nitrous oxide, 50% of oxygen, and 1.5－2.5 vol% of sevoflurane. After the anesthesia induction, the surgery started with placing the patient in left lateral decubitus. The patient's head was put on a soft pillow and adequately padded in the lateral position. The anesthesiologist made sure the patient's left face was not compressed, especially with his eyes and auricles. There was no significant event throughout the anesthesia. Blood loss during the surgery was 800 ml and his body temperature was maintained between 35.5°C and 35.9°C. Anesthesia time was 360 min and operation time was 255 min. The patient was transferred from the post-anesthetic care unit to the general ward after confirming alert mental state and stable vital signs.

In the afternoon of the operation day, the patient started complaining of left facial numbness and weakness. Eye closure and hearing were impaired in the left side. He was referred to otolaryngology department by the neurosurgeon. Phsycial examination revealed impaired grimacing over ipsilateral forehead, incomplete left eye closure, mouth deviation to the contralateral side, severity of House-Brackmann scale IV (Table 1).[3] Skin

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Table 1. House-Brackmann Classification of Facial Nerve Dysfunction

Normal	Normal function in all areas
Mild dysfunction	Gross Slight weakness noticeable on close inspection May have slight synkenesis Normal symmetry and tone at rest Motion Forehead: Moderate to good function Eye: Complete closure with minimal effort Mouth: Slight assymetry
Moderate dysfunction	Gross Obvious but not disfiguring difference between the two sides Noticeable but not severe synkinesis, contracture, or hemifacial spasm Normal symmetry and tone at rest Motion Forehead: Slight to moderate movement Eye: Complete closure with effort Mouth: Slightly weak with maximum effort
Moderately severe dysfunction	Gross Obvious weakness and/or disfiguring asymmetry Normal symmetry and tone at rest Motion Forehead: None Eye: Incomplete closure Mouth: Asymmetric with maximum effort
Severe dysfunction	Gross Only barely perceptible motion Asymmetry at rest Motion Forehead: None Eye: Incomplete closure Mouth: Slight movement
Total paralysis	No movement

lesion, gustatory impairment, hyperacusis were not observed. Brain computed tomogram showed a nasopharyngeal mass and nasal hematoma. Small Thornwaldt's cyst was discovered by further nasopharyngeal magnetic resonance imaging. Both were not assumed to be related with facial nerve paralysis. The pure tone audiometry which was performed on 5th day after the surgery showed bilateral high tone hearing impairments both in bone and air conduction (over 30 decibels at 4 kHz) with preserved hearing ability in middle and low tones

Under the impression of Bell's palsy, the treatment started. Steroid pulse therapy with prednisolone 20 mg bid for 2 days, 10 mg bid for 2 days, 5 mg bid for 1 day and Acyclovir 250 mg tid for 5 days was administered in addition to facial massage. These treatments failed to improve his symptoms until the post-operative day 12. On the 13th day after the surgery, facial weakness was slightly improved, although dysarthria was still present.

His facial weakness was totally resolved to House-Brackmann scale I on the 16th day after the surgery and the patient was discharged without any problem and did not follow up.

DISCUSSION

Nerve injuries associated with the positioning are preventable, but continue to happen.[4] Nerve injury can be classified by three categories.[2] Neurapraxic injury, which occurs most frequently during anesthesia, indicates loss of function without demonstrable anatomic injury. This type of injury resolves completely within 6 weeks. Axonotmesis is anatomic disruption of the axon with preserved nerve sheath and connective tissue. In this injury, function improves gradually as the regeneration progresses. Neurotmesis occurs with complete destruction of axon, sheath, and connective tissue. Surgery may be helpful in this type of injury. In regard to compression injury, there is a report of facial and brachial plexus palsy caused by cervical collar.[5] Facial nerve paralysis which is developed after spinal surgery under general anesthesia has not been reported until now. In this case, presenting symptoms including impaired grimacing over ipsilateral forehead, incomplete left eye closure, mouth deviation to the contralateral side suggest peripheral facial nerve paralysis. The possible causes of peripheral facial nerve paralysis are Bell's palsy, traumatic injury, infectious injury, neoplasm, systemic diseases in the order of frequency. The imaging study reveals nothing significant which rules out traumatic injury and neoplasm. In addition, there were no test results suggesting infection or systemic disease, which leaves us Bell's palsy. If another etiology should be considered aside Bell's palsy, the most probable cause of facial paralysis in this case is compression associated with positioning. Although we paid attention to prevention of compression injury by putting the patient's head on a soft pillow and appropriately padded, ischemia of nerves from compression might contribute to the injury presented in this case. We think that proper padding will reduce, but not totally eliminate the risk of nerve injury. In this case, the nerve injury is thought to be from neuropraxic injury caused by compression as complete recovery of his symptoms without sequelae occurred in three weeks. The patient was positioned left lateral decubitus for 100 min and that was enough time for nerve injury which was demonstrated in a case report of 75 minutes of pressure caused neural

injury.[6]

Nitrous oxide might play a role in injuries of nerves especially that pass through the middle ear. Nitrous oxide is 35 times more soluble than nitrogen in blood and diffuses into air-containing cavity more rapidly than nitrogen is removed, which lead to a hazardous elevation of pressure, contributing to labyrinthine membrane rupture, particularly in patients with prior histories of middle ear surgery.[7] Segal and colleagues proposed that nitrous oxide is a possible cause of sensory neural hearing loss which results from labyrinthine membrane rupture due to its space expanding property.[8] Pure tone audiogram revealed that he had hearing difficulty in high frequency which assumes hearing impairment due to noise exposure. Hearing impairment which emerged first with facial paralysis in this patient may result from tinnitus associated with facila nerve paralysis or increased middle ear pressure due to nitrous oxide.

Among less probable cause, viral infection and microemblism are worth considering. The microembolism was demonstrated as the cause of facial paralysis in a pediatric cardiac surgical patients.[9]

There have been only a few reports of facial paralysis associated with anesthesia. Dorsey and colleaugues[10] reported increased incidence of Bell's palsy related with pregnancy. They presented 36 patients of facial nerve paralysis patients associated with pregnancy and the incidence and distribution of the palsy were similar between patients delivered with and without anesthesia. Although there is no evidence that facial nerve paralysis in pregnancy is different than in nonpregnanat women, altered susceptibility to herpes simplex infection in the third trimester is worth considering. Craniotomy and subdural hematoma removal under general anesthesia has been reported to provoke facial nerve paralysis.[11] Left partial facial nerve paralysis developed after brain surgery and completely resolved over 3 weeks period after parotid swelling receded whose clinical course is pretty similar to the case that we demonstrate. We think this case may present similar etiology of facial nerve paralysis (compression by parotid swelling or face mask/rubber band versus direct pressure by positioning), therefore, showed comparable clinical course.

Peripheral nerve paralysis after general anesthesia usually has very favorable prognosis. In general, it is spontaneously resolved within 3 months. Administration of corticosteroid during the first 7 to 10 days after onset has been beneficial in most trials. Acyclovir, used alone, is no more effective than corticosteroids. The use of both medications together remains under study.[12]

In the light of this case and previous case reviews, we wish to have more concern about positioning and encourage further reports and to recommend early identification and in facial paralysis related to general anesthesia.

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