Unilateral Hypoglossus Nerve Palsy Following Intubation

M. Güven Güvenç¹, A. Sanem Özata², Türker Şengül³, Sibel Şener⁴

ABSTRACT

Some neurological diseases, malignant tumors, trauma and surgery might cause hypoglossal nerve palsy. However XIIth nerve palsy following intubation is unusual.

A 40 year-old woman underwent surgery for nasal and right phalangeal fractures. The patient had a left hypoglossal nerve palsy which was detected on the first postoperative day. Her neurological and otorhinolaryngological evaluation showed no other pathology. The palsy resolved completely in the 6th postoperative week.

Post-intubation hypoglossal nerve palsy is very rare. Appropriate cuff pressure, uneventful oro-tracheal intubation, and avoiding malpositioning of the head during surgery are of considerable importance in avoiding this complication. A meticulous neurologic and otorhinolaryngologic evaluation and follow-up of these patients is critical. The majority of cases recover without sequela.

Key words: unilateral hypoglossal palsy, intubation

The hypoglossal nerve is a pure motor nerve innervating all muscles of the tongue; it is in a distal position to skull base. Vascular aneurysms, local infections, some surgical procedures, trauma and tumors might cause hypoglossal nerve injury (1). Nevertheless, XIth nerve palsy following intubation is very rare.

Case report

A 40 year-old female patient was admitted to the emergency department with nasal deformity, swelling and pain on the right hand which she had after a trauma.
An orthopedic and otorhinolaryngologic consultation and radiologic evaluation revealed nasal fracture and a phalangeal fracture in her right hand. Subsequently, the patient underwent a closed nasal fracture reduction, an open right phalangeal fracture reduction and fixation using a K wire under general anesthesia. A number 7 armored tube was used for the intubation. The intubation and extubation of the patient were uneventful. On the first postoperative day, the patient mentioned that she felt an abnormality when she moved her tongue. Her examination revealed a left hypoglossal nerve palsy (Figure 1). Her laryngoscopic examination was normal. On her neurologic examination, there was no additional finding. The patient was subsequently discharged, and her hypoglossal nerve palsy recovered without any sequela during the sixth postoperative week (Figure 2).

**Discussion**

Sore throat, laryngeal edema, hoarseness, nerve injury, superficial laryngeal ulcers, laryngeal granuloma, glottic and subglottic granulation tissue, laryngeal synechiae, vocal cord paralysis and aspiration, laryngotracheal membrane, tracheal stenosis, tracheomalacia, tracheoesophageal fistula, trachea-innominate artery fistula are common post-intubation complications (2). However, very few cases of hypoglossal nerve palsy secondary to intubation have been reported. Una et al reported a bilateral hypoglossal palsy case after orotracheal intubation (1). This clinical picture’s etiology has been suggested to be a neuropathy due to compression of the nerve. Difficult or complicated oro-tracheal intubation, hyperextension or excessive lateral extension of the head during surgery, prolonged oro-tracheal intubation, tight throat pack, and inappropriate cuff pressure of the intubation tube might contribute to this compression mechanism (1,3,4). Nitrous oxide might increase the cuff pressure by diffusing into the cuff (5). The normal cuff pressure is between 20-30 cm H₂O (6,7). In our case, the intubation was uneventful, no throat pack was used. The cuff pressure was checked and found to be within normal limits, and no nitrous oxide was used. Neither hyperextension nor excessive lateral extension of the head was present. However the endotracheal tube was placed on the left side, and our patient had a left sided hypoglossal palsy. This finding is suggestive of a compression neuropathy, and shows that the palsy can occur in susceptible patients even in the absence of the other risk factors.

Tapia’s syndrome is defined as isolated recurrent and hypoglossal nerve palsy. Tesei at al reported a Tapia’s syndrome case occurring after a rhinoplasty was performed with orotracheal intubation (3). Anatomically the hypoglossal nerve is placed on the most lateral prominence of the anterior surface of the transverse process of the first cervical vertebra (C1), and crosses the vagus nerve. It has been suggested that the Xth and XIIth nerves might be stretched and pressed against this prominence due to the hyperextension of this joint (3). Boisseau et al reported Tapia’s syndrome following a shoulder surgery. They also assumed that marked lateral flexion of the head might cause damage to several cranial nerves by the prolonged stretching mechanism, and that the tracheal tube might press on a localized area at the crossing of the hypoglossal and vagus nerves (8). In our case, the recurrent nerve was not affected. The progressive recovery of function in the majority of cases reported in the literature, as seen in our case, is also suggestive of a neuropraxic type of nerve damage (1,3). The other reported
causes of Tapia’s syndrome are direct trauma to the Xth and XIIth nerves, carotid artery dissection involving the ascending pharyngeal artery, nasopharyngeal fungal infection, and neurofibromatosis involving the vagal and hypoglossal nerves (9-12). Rotondo et al. reported a Tapia’s syndrome after cardiac surgery in a patient undergoing anticoagulant therapy. They argued that rhinopharyngeal hematoma, occurring after placement a nasogastric tube, was exacerbated due to heparin therapy, and led to symptoms (13).

Appropriate cuff pressure, uneventful oro-tracheal intubation, and good positioning of the head during surgery are of considerable importance in avoiding XIIth nerve palsy. A meticulous neurological and otorhinolaryngological evaluation and follow-up of the patients with this complication is important. The majority of the cases recover without sequela.

Acknowledgement
Published with the written consent of the patient.

References