Black Sea Journal of Health Science
doi: 10.19127/bshealthscience.803290

Open Access Journal
e-ISSN: 2619 – 90410

Case Report
Volume 4 - Issue 2: 153-155 / May 2021

A CASE REPORT: PROBABLY CONCURRENT NASOGASTRIC TUBE SYNDROME AND CEREBROVASCULAR DISEASE IN A POSTOPERATIVE PATIENT

Emel YILDIZ[1]*, Özlem ÖNER[2], Özlem KOÇAK[1], Nadir YILDIRIM[3]

[1]*Kütahya Health Sciences University, Evliya Celebi Education and Research Hospital, Department of Anesthesiology, 43040, Kütahya, Turkey*
[2]*Dokuz Eylül University, Department of Intensive Care, 35330, İzmir, Turkey*
[3]*Kütahya Health Sciences University, Evliya Celebi Education and Research Hospital, Department of Otolaryngology, 43040, Kütahya, Turkey*

Abstract: Nasogastric tube syndrome (NTS) is characterized by acute upper airway obstruction due to bilateral vocal cord paralysis. Although NTS is a rare cause of vocal cord paralysis, it has the potential of causing serious morbidities even mortality. We here with present a case of concurrent NTS and cerebrovascular disease patient. 73year-old male was admitted to our university hospital emergency department with acute respiratory distress and stridor days after his cholecystectomy operation and laryngoscopy showed bilateral vocal cord paralysis. The patient was admitted to the intensive care unit (ICU) and urgent tracheotomy was performed. On the day after his discharge from the hospital, his vocal cord movements fully recovered state. Nasogastric tube syndrome can be considered in patient who complains because of acute upper airway obstruction with nasogastric tube inserted. Thinking NTS early may be reducing the complication rate.

Keywords: Vocal cord paralysis, Bilateral, Brainstem infarction, Nasogastric tube syndrome, Tracheotomy

***Corresponding author:** Kütahya Health Sciences University, Evliya Celebi Education and Research Hospital, Department of Anesthesiology, 43040, Kütahya, Turkey
E mail: dremelyldz@gmail.com (E. YILDIZ)
Emel YILDIZ https://orcid.org/0000-0003-4493-2099
Özlem ÖNER https://orcid.org/0000-0001-6171-2114
Özlem KOÇAK https://orcid.org/0000-0001-9322-9776
Nadir YILDIRIM https://orcid.org/0000-0002-4916-3885

Received: October 01, 2020
Accepted: January 06, 2021
Published: May 01, 2021

Cite as: Yıldız E, Öner Ö, Koçak Ö, Yıldırım N. 2021. A case report: probably concurrent nasogastric tube syndrome and cerebrovascular disease in a postoperative patient. BSJ Health Sci, 4(2): 153-155.

1. Introduction

Nasogastric tube syndrome (NTS) was first identified by Iglauer and Molt (1939) in a series of 12 patients in 1939. Although NTS is rare, it may lead to severe respiratory distress requiring emergency airway management in some cases including tracheotomy. To our knowledge, a total of 20 case reports and two reviews of NTS have been published in the medical literature until 2018 (Sofferman et al., 1990). Two of these cases were the results of long intestinal tube (LIT) application and the results of the cases were caused by nasogastric tube (NT) (Sanaka et al., 2004). Thirteen of these cases in the literature were reported to have fully recovered. While there meaning five of them did not survive (Sofferman et al., 1990; Nehru et al., 2003). There meaning five patients' processes could not be described (Apostolakis et al., 2001). If upper airway obstruction occurs in patients with NT or LIT, thinking of NTS would belief saver act. We presented a patient who developed NT Sand cerebrovascular disease (CVD) at the same time and followed up in the intensive care unit (ICU).

2. Case Report

73 year-old male patient was admitted to our emergency department with the complaints of sore throat, acute inspiratory stridor, straining in the intercostal muscles and oxygen failure. The patient had a conscious cooperation and orientation. Glasgow Coma Scales (GCS) 15 and pulse oximetry (SpO_2) were 94%, blood pressure 100/60mmHg.

His breathing difficulty started at previous night and accompanied by vomiting, dysphagia and slurring of speech. The patient had a history of laparoscopic cholecystectomy operation under general anesthesia one week ago. During the operation, a nasogathric tube was inserted and removed 2 days later. The patient was evaluated in the emergency service by an otolaryngologist and anesthesiologist. Direct laryngoscopy revealed that supraglottis was diffusely and mildly edematous and vocal cords were immobile in the midline. The patient was hospitalized in the intensive care unit and 1mg/kg methylprednisolone and 2 mg midazolam was administration. In spite of noninvasive mechanical ventilation to improve oxygenation, respiratory distress persisted and emergency

tracheotomy was performed to maintain airway flow. His vital signs were stable on the following days in the ICU. 2 hours after the patient was admitted to intensive care, her consciousness was asleep, cooperation and orientation were slightly limited, and the GCS was E3M6V1 (tracheotomy status).

Mechanical ventilator support was started. On the first day of treatment, the patient was separated from the mechanical ventilator. The neurological evaluation of the patient did not show any definitive signs of CVD. Hence, slightly pharyngeal reflexes and mild disorientation of the patient along with vocal fold paralysis man dates to rule out a possible CVD. Eternal and central seizures of the lacunar infarction in the left capsule in cranial magnetic resonance imaging (MRI) taken to the patient in the emergency department. Signal enhancement at the flair and T2 sequences in bilateral periventricular and subcortical white matter was consistent with ischemic gliotic lesions. At the level of the right inferior pons and right thalamuslacunary infarctions appeared to be new lesions. The whole cerebrum was found mildly edematous. Echocardiography confirmed mild left cardiac insufficiency (EF: 50%, left ventricular hypertrophy, diastolicdys function, aortic valve calcification). Bilateral carotid Doppler ultrasonography showed that 70% and 48% stenosis of the right and left carotid arteries, respectively. The patient was put on the medical treatment of klopidogrel, acetylsalicylic acid treatments started for the diagnosis of CVH. On the 5th day of the treatment, his general condition was stabilized and he was discharged from ICU to Otorhinolaryngology (ENT) clinic. Within the following weeks, the patient was regularly followed by ENT department with direct laryngoscopy. Laryngoscopy had showed that the edema of the larynx was gradually decreased, and then the movements of right and left vocal cords returned respectively. The patient was decannulated within the 4 weeks. His control MRI showed regression in the cerebraledema. Finally, the patient fully recovered in one month after the onset of NTS and was decannulated. One patient was enrolled in the study and informed consent forms were signed by this patient.

2.1. Ethical Consideration

After fully explaining the purpose of the study to the patient, consent was obtained for the study and case presentation.

3. Results and Discussion

Nasogastrictube syndrome was first described by Soffermanin at 1990 to describe the triad of throat pain, nasogastric intubation, and vocal cord paralysis (Sofferman et al., 1990). According to our knowledge, there are very few reported cases in the medical literature. Nevertheless, because of the unawareness of this clinical entity, it is believed that there are fewer cases reported than the actualones. Brousseau and Kost (2006) reported that 71% of the NTS cases were men, and 29% of them women. Vielva del Campo et al. (2010) reported that this syndrome may develop both in children and adults. Brousseau and Kost (2006) also estimate the range of NTS onset from 12th hour of intubation to 2 weeks after extubation. Time to recovery from respiratory symptoms and vocal cords dysfunction has been reported between 1 day to 3 months. Possible mechanism of NTS is as follows; mobile laryngeal tissues rub against the fixed NT. Whilst the patient is supine, the cricoid bone compresses the tube against the supine. Eventually, tonic contraction of the cricopharyngeus muscle pulls the tube against the delicate and thin post-cricoid mucosa. It is believed that these three mechanisms are caused local tissue edema and ulcerationas to result in NTS (Sofferman et al., 1990; Marcus et al., 2006). The patient presented can be branded as “NTS”, because the patient had a synchronous cerebrovascular accident (CVA), and NTS due to the nasogastrictube insertion at a recent cholecystectomy operation he had undergone. Bourossou et al. (2007) have reviewed 33 such cases reported so far and identified two characteristics of this syndrome which also comply with the findings and history of our patient: the paralysis occurs within 12 hours to 2 weeks after the insertion of the tube and vocal cords recovered full motion spontaneously within a period of up to two months. Mild edema of his laryngeal structures also suggested a local cause.

Nayak et al. (2018) published Sofferman syndrome (nasogastric tube syndrome) developing after nasogastric tube in 2 pediatric acute lymphoblastic leukemia (ALL) cases in 2018 (Nayak et al., 2018). The syndrome is largely attributed to the mechanical compression of the tube against posterior laryngeal structures. However, neurological signs and symptoms that our patient displayed such as confusion, vomiting, speech difficulties and sudden onset of the paralysis hinders this explanation. On the other hand, his clinical picture was not consistent of a full scale brainstem stroke, which is usually characterized with contralateral hemiplegia and hemianesthesia along with ipsilateral cranial nevre findings that includes vertigo, nystagmus and oculomotor signs (Singh et al., 2006). Furthermore, it is very uncommon that bilateral vocal cord paralysis constitutes the chief manifestation of a brainstem syndrome, which requires much more massive infarction of the brainstem. Nevertheless, we could not underestimate the role of CVA, he suffered; it was also indicated by brain stem infarction and associated cerebral edema at MRI. Therefore, we speculated that both potential etiologic factors might have contributed to this incident. It is likely that, he had tolerated mild laryngeal edema caused by nasogastric tube, and that was worsened by intervening CVA, which led to circumscribed infarction and edema in the brainstem that resulted in dysfunction of the motor nuclei of both nucleus ambiguous (NX). Considering that the infarction occurred on the right brainstem, the earlier recovery of the ipsilateral vocal cord function with the lessening of

cerebral edema also supports this hypothesis. In our case, the symptoms were presented one week after NT insertion, and a month was required for complete recovery.

If NTS is suspected in a patient, immediate removal of NT or LIT can redress respiratory failure. Nevertheless, if removal of the NT does not remedy respiratory failure, trachea to my will be required. Nehru et al. (2003) reported that 77% of all NTS cases required tracheotomy. Tracheotomy is preferred for long-term endotracheal intubation because the latter may delay the healing of vocal cord function for several months. With regard to the other treatments, parenteral corticosteroids should be used to reduce inflammation and antibiotics should be used to prevent the formation of retro cricoid abscesses. In addition, the patient should stay away from the oral route for a few days with a daily laryngeal examination to monitor changes in arythenoid edema. Additional intravenous fluids or gastrostomy may be required throughout this interval. Tracheotomy was performed in our case.

To prevent the onset of NTS, the adaptation of the placement of the NT or the LIT should be carefully determined. In addition, a narrower pipe diameter should be selected to reduce the pressure that the pipe presses against local structures.

3. Conclusion

In summary, we have reported a very rare but life-threatening case of the NTS induced by NT insertion. NTS should be considered in all patients who present with sore throat, hoarseness and/or shortness of breath following asogastricoral or gastrointestinal intubation. It is preventable by careful insertion of these tubes. NTS requires rapid treatment such as removal of the tube, tracheotomy when required and close follow-up with bronchoscopy. If the patients are diagnosed early and treated appropriately, the chances of recovery is good.

Author Contributions

Authors have obtained the necessary data by evaluating our case in detail

Conflict of Interest

The authors declare that there is no conflict of interest.

References

Apostolakis LW, Funk GF, Urdan LF, Mc Culloch TM, Jeyapalan MM. 2001. The nasogastrictube syndrome: two case reports and review of the literature. Head Neck, 23: 59–63.

Brousseau VJ, Kost KM. 2006. A rare but serious entity: nasogastric tube syndrome. Otolaryngol Head Neck Surg, 135: 677–679.

Iglauer S, Molt WF.1939. Severe injury to the larynx resulting from the indwelling duodenal tube. Ann Oto Rhinol Laryngol, 48: 886–904.

Marcus EL, Caine Y, Hamdan K, Gross M. 2006. Nasogastric tube syndrome: a life-threatening laryngeal obstruction in a 72-year-old patient. Age Ageing, 35: 538-539.

Nayak G, Virk RS, Singh M, Singh M. 2018. Nasogastric tube syndrome: a diagnostic dilemma. J Bronchol Interv Pulmonol, 25(4): 343-345.

Nehru VIN, Shammari HJA, Jaffer AM. 2003. Naso gastrictube syndrome: the unilateral variant. Med Princ Pract, 12: 44–46.

Sanaka M, Kishida S, Yoritaka A, SasamuraY, Yamamoto T, Kuyama Y. 2004. Acute upper airway obstruction induced by an indwelling long intestinal tube: attention to the nasogastrictube syndrome. J Clin Gastroenterol, 38: 913.

Singh D, Jagetia A, Sinha S. 2006. Brain steminfarction: a complication of microvascular decompression for trigeminal neuralgia. Neurol India, 54(3): 325-326.

Sofferman RA, Haisch CE, Kirchner JA, Hardin NJ. 1990. The nasogastric tube syndrome. Laryngoscope, 100: 962–968.

Vielva del Campo B, Moráis Pérez D, Saldaña Garrido D. 2010. Nasogastric tube syndrome: a case report. Acta Otorrinolaringol Esp, 61: 85–86.