CASE REPORT

Nasogastric Tube Syndrome
—a Potentially Fatal But Treatable Complication—

Makoto Hibino1; Motoki Ohe1; Tetsuri Kondo1

ABSTRACT — Background. Nasogastric tube syndrome is a potentially fatal but treatable complication of an indwelling nasogastric tube, which causes vocal cord abductor paralysis and upper airway obstruction. Case. We report a case of a 51-year-old woman who developed this syndrome after hospitalization for putaminal hemorrhage. Vocal cord abductor paralysis was caused by inflammation of the posterior cricoarytenoid muscle, as confirmed by computed tomography. The patient was successfully treated with antibiotics, corticosteroids, and proton-pump inhibitors. Conclusion. While a nasogastric tube is commonly used in various settings, the syndrome itself is rare. Clinicians should learn more about this complication to avoid underdiagnosis and administer prompt treatment.

KEY WORDS — Nasogastric tube, Nasogastric tube syndrome, Vocal cord abductor paralysis

BACKGROUND

In 1939, Iglauer and Molt described patients with bilateral vocal cord paralysis secondary to postcricoid ulceration from rubber nasogastric tubes. In 1981, Sofferman et al. came up with the concept of nasogastric tube syndrome; a potentially fatal but treatable complication characterized by the following clinical triad: the presence of (i) a nasogastric tube; (ii) sore throat or stridor or both; and (iii) vocal cord abductor paralysis, usually bilateral. Recently, nasogastric tube syndrome has been rarely reported; however, it is a rare syndrome that can be easily overlooked and possibly underdiagnosed, which suggests a need for readdressing this issue.

CASE PRESENTATION

A previously healthy 51-year-old Japanese woman presented to the emergency department of our hospital with a sudden onset of unconsciousness. On admission, a neurological examination revealed right-sided hemiparesis and altered mental status with a Glasgow Coma Score of 5/15 (E1V1M3). Head computed tomography (CT) revealed left putaminal hemorrhage, and the patient underwent external decompressive craniotomy with the removal of a hematoma. After the operation, she was nursed in an intensive care unit with mechanical ventilation via endotracheal intubation. On day 2 after admission, a nasogastric tube was inserted for feeding and administration of a calcium channel blocker for hypertension because of dysphagia, total aphasia, and right-sided hemiparesis. Her consciousness improved and she was extubated on day 15. After discharge from the intensive care unit, her respiratory condition remained stable. On day 43, a new nasogastric tube was inserted smoothly and without difficulty because the previous one was accidentally pulled out. Six hours later, inspiratory stridor suddenly developed. The nasogastric tube was immediately pulled out and the arterial blood gas analysis was performed but no abnormalities were noted. Next, a detailed examination of the larynx by direct endoscopy was performed, which revealed an ulceration on the reddish and remarkably swollen left aryepiglottic fold and mildly swollen right arytenoid and cuneiform tubercles (Figure 1a). In addition, a markedly narrow glottal space was noted be-

1Department of Respiratory Medicine, Shonan Fujisawa Tokushukai Hospital, Japan.
Correspondence: Makoto Hibino, Department of Respiratory Medicine, Shonan Fujisawa Tokushukai Hospital, 1-5-1 Tsujido Kandai, Fujisawa, Kanagawa 251-0041, Japan (e-mail: m-hibino@ctmc.jp).
Received August 15, 2014; accepted December 6, 2014.
© 2015 The Japan Society for Respiratory Endoscopy

cause of the abductor paralysis of the bilateral vocal cords; the left vocal cord was fixed in the mid position and the right vocal cord could abduct slightly. A contrast-enhanced CT image of the neck showed a swollen posterior cricoarytenoid muscle without low-density area, suggesting an abscess (Figure 1b). The patient fulfilled all 3 clinical criteria for nasogastric tube syndrome: 1) the presence of a nasogastric tube, 2) stridor, and 3) the presence of vocal cord abductor paralysis. Following diagnosis, we introduced a conservative treatment with methylprednisolone (80 mg/day), cefmetazole (3 g/day), and omeprazole (20 mg/day) intravenously. Inspiratory stridor gradually improved and resolved completely on day 49, following which the steroid dose was gradually reduced. On day 68, direct endoscopy of the larynx showed a marked improvement of mucosal inflammation and bilateral vocal cord abduction (Figure 2a). Moreover, noncontrast CT of the
neck showed a reduction in the swelling of the posterior cricoarytenoid muscle (Figure 2b). Once the clinical improvement had been confirmed, the treatment with corticosteroids, antibiotics, and proton-pump inhibitors was discontinued. The patient temporarily received central venous hyperalimentation after removing the nasogastric tube, and underwent percutaneous endoscopic gastrostomy for enteral nutrition on day 71. Following this, the patient was eventually transferred for rehabilitation to a chronic care hospital.

DISCUSSION

Vocal cord abductor paralysis in nasogastric tube syndrome occurs mostly bilaterally, but, occasionally, also unilaterally. In the present case, we observed bilateral vocal cord abductor paralysis; it was complete on the left side and incomplete on the right side. In addition, considering the presence of a nasogastric tube and stridor, a diagnosis of nasogastric tube syndrome was made.

Vocal cord movement in the phonation, respiration, and sphincter activity is directly controlled by the intrinsic laryngeal muscles. Among these muscles, vocal cords are abducted only by the posterior cricoarytenoid muscle, whose motor nerve supply is via the branches of the recurrent nerves on each side. The other muscles affect the adduction of the vocal cords. Therefore, the dysfunction of the posterior cricoarytenoid muscle leads to vocal cord abductor paralysis.

Some mechanisms underlying nasogastric tube syndrome have been proposed in previous studies. Sofferman et al. described three mechanisms that could account for the syndrome, either alone or in combination. The first mechanism is the dynamic nature of the larynx, sliding up and down against a semirigid nasogastric tube as the patient coughs and swallows. The second one is the normal tonic contractile state of the cricopharyngeus muscle pressing the tube against the posterior cricoid cartilage lamina. The third mechanism involves gravity pulling the larynx posteriorly, pinching the nasogastric tube between the two rigid structures of the cricoid cartilage and anterior cervical spine, when the patient is in the supine position. A possible fourth mechanism might be gastroesophageal reflux associated with the use of a nasogastric tube. Gastric acid stimulation secondary to gastroesophageal reflux and mechanical pressure associated with the above three mechanisms against the posterior cricoid lumina lead to the focal edema and/or ulceration, which is occasionally followed by bacterial infection and abscess. These four mechanisms cause inflammation, neurogenic atrophy, and ischemic and congestive injuries of the posterior cricoarytenoid muscle. This multifactorial pathophysiology including myogenic, neurogenic, and vasculogenic processes eventually leads to vocal cord abductor paralysis. In the present case, inflammation of the posterior cricoarytenoid muscle was thought to be the main pathophysiological factor on the basis of swollen aryepiglottic fold and posterior cricoarytenoid muscle, which was confirmed by bronchoscopy and contrast-enhanced CT, respectively.

As revealed in a previous report, the symptoms of the syndrome may develop from 12 hours after intubation to 2 weeks after extubation. Moreover, another study reported that the period between tube placement and tracheostomy needed for airway maintenance was between 2 and 52 days (mean, 24.6 days). In the present case, symptoms occurred on day 42 after the first insertion of the nasogastric tube and 6 hours after the second insertion. Therefore, we believe that mucosal edema and muscle inflammation had been partially induced by the initial placement of the tube, and mechanical stimulation during the second insertion exacerbated the lesions and directly triggered the onset of the syndrome.

Once nasogastric tube syndrome is suspected, it is most important to remove the nasogastric tube. In addition, treatment with antibiotics, corticosteroids, and proton-pump inhibitors has been reported to be useful. Moreover, if vocal cord abductor paralysis is complete and the glottal space is markedly narrow and closed, tracheostomy or cricothyroidotomy are needed for airway maintenance, and the use of those procedures has been reported in 47% to 77% of the cases. In the present case, bilateral and complete vocal cord abductor paralysis was confirmed on the left side, while incomplete on the right side. The glottal space was not completely closed and the patient's respiratory condition was not so bad as to require tracheostomy.

It is very important to apply preventive measures in patients with a nasogastric tube to avoid nasogastric tube syndrome. To prevent the onset of the syndrome, clinicians should, first of all, have adequate knowledge about the syndrome in order to be able to diagnose it; second, they should reassess the necessity of using the
Nasogastric Tube Syndrome: a Potentially Fatal But Treatable Complication—Hibino et al

CONCLUSION

Nasogastric tube syndrome is a very rare complication of nasogastric tube placement, which occasionally may be life-threatening. However, it is curable if it is diagnosed and treated in the early phase. Recently, a nasogastric tube has been commonly used in various settings ranging from acute care in the intensive care unit to chronic care in home health care. To decrease the rate of underdiagnosis and administer prompt treatment, clinicians should have adequate knowledge about this rare complication and perform endoscopy of the larynx as soon as the syndrome is suspected.

No potential conflicts of interest are disclosed.

REFERENCES