A Variant Form of Nasogastric Tube Syndrome

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Abstract

Nasogastric tube syndrome named by Sofferman et al in 1981 is a laryngeal complication presenting with life-threatening vocal cord abductor paralysis derived from perforation of the NG tube-induced esophageal ulcer. As compared with the previously reported cases of this syndrome, our 4 autopsied patients were so peculiar in the following two points that vocal cord abductor paralysis developed repeatedly and no esophageal ulcer was present in spite of the presence of the laryngeal abductor muscle injury. We hypothesized that the etiology of such a variant form was circulatory injury of the laryngeal abductor muscle which was caused by the compression of the postcricoid blood vessels perfusing this muscle. Nasogastric tube syndrome, which is treatable by decannulation, cannot be ruled out even if no esophageal ulcer is detected by fiberoptic laryngoscopy.

Case Report

Case 1

A 73-year-old man developed bradykinesia and dementia at the age of 68. Two years later, his parkinsonism deteriorated rapidly with no response to L-DOPA treatment. He was almost bed-ridden requiring NG tube feeding. On July 30, 1994, at the age of 72, he developed inspiratory stridor, which was caused by moderately severe VCAP by fiberoptic laryngoscopy. Arterial blood gas analysis under oxygen inhalation showed pH=7.44, pCO2=38 Torr, pO2=89 Torr. In the following month under no treatment, his inspiratory stridor gradually decreased, and he was discharged with subtile inspiratory stridor. On February 6, 1995, he was readmitted to our hospital because of pneumonia. After recovery from pneumonia with antibiotics, mild inspiratory stridor was still continued and fiberoptic laryngoscopy demonstrated mild to moderately severe VCAP. Arterial blood gas analysis on room air showed pH=7.495, pCO2=43 Torr, pO2=76 Torr. On August 4, 1995, he was again admitted to our hospital because of increased stridor and dyspnea. Arterial blood gas analysis at room air showed severe hypoxemia; pH=7.445, pCO2=38 Torr, pO2=40 Torr. The etiology of acute respiratory failure was thought to be deterioration of VCAP since neither inflammatory reaction of the blood sample nor pneumonia shadow on the chest X-ray film was found. He died of respiratory failure on September 6, 1995.

Postmortem pathological examination confirmed the diag-
nosis of Parkinson’s disease. Cryosections of the PCA showed a scattering of a small number of muscle fibers and massive inflammatory cell infiltration with proliferative small blood vessels in the interstitial tissues. Endomysial space was remarkably edematous. No grouped atrophy or target fibers suggestive of neurogenic abnormalities was observed. No ulcer formation was found in any region of the esophagus.

Case 2
A 77-year-old woman with a history of mild left hemiparesis, which had been caused by cerebral infarction at the age of 71, was admitted to our hospital because of complete right hemiparesis with severe consciousness disturbance on April 17, 1996. Brain CT scan showed extensive low density area over the whole region supplied by the left middle cerebral artery. Since she developed apneic respiration of Cheyne-Stokes pattern, mechanical ventilation through an intratracheal tube was required for three days. On April 23, tube feeding through a NG tube was started. On June 3, three days after decannulation of the intratracheal tube, fiberoptic laryngoscopy clarified that vocal cord movement was normal. On July 7, mild inspiratory stridor appeared, becoming loud in the following few days. On July 10, fiberoptic laryngoscopy showed the presence of bilateral severe VCAP with slit-like glottal space (Fig. 1). The arterial blood gas analysis at room air demonstrated acute respiratory

Figure 1. Cryosections of the posterior cricoarytenoid muscle from Case 2 showed sparse muscle fibers (A, HE stain, ×33), associated with severe inflammatory cell infiltration and proliferative capillaries (B, HE stain, ×60).

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Figure 2. Fiberoptic laryngoscopy from Case 2 demonstrated a markedly narrow glottal space because of the abductor paralysis of bilateral vocal cords (VC). Arytenoid mucosa (A) was markedly edematous.
failure; pH=7.35, pCO₂=53 Torr, pO₂=51 Torr. Subsequently, however, her inspiratory stridor became decreased gradually in spite of the lack of treatment. The only medical therapeutic for this period was exchange of the NG tube, which was regularly done every two weeks. On August 15 when the inspiratory stridor almost disappeared, reexamination of fiberoptic laryngoscopy showed normal abduction and adduction movements of the vocal cords. The arterial blood gas analysis at room air became almost normal; pH=7.420, pCO₂=42 Torr, pO₂=80 Torr. However, on September 3 the inspiratory stridor appeared again with gradual exacerbation. Her consciousness level did not change till her death. She was found in cardiorespiratory arrest by a rounding nurse on October 2, 1997.

Postmortem pathological examination revealed bilateral cerebral softening in the areas supplied by the middle cerebral arteries with moderately severe atherosclerotic changes of the internal carotid arteries and the basilar artery. Cryosections of PCA showed sparse muscle fibers with massive inflammatory cell infiltration with proliferative small blood vessels (Fig. 2). Endomysial space was remarkably edematous. No grouped atrophy or target fibers, suggestive of neurogenic origin was observed. No ulcer formation was found in any region of the esophagus including postcricoid area.

Discussion

Since Iglauer and Molt reported severe laryngeal injury resulting from an indwelled duodenal tube in 1939 (1), similar cases have been occasionally reported (2–9). According to the recent review of the literature on this syndrome, it is suspected that the clinical spectrum of severity exists with less severe cases going unrecognized (3). The pathophysiology of this critical illness is considered as follows: The anterior wall of the upper esophagus is pinched between an indwelled NG tube and the posterior lamina of the cricoid cartilage, resulting in the development of the esophageal ulcer. When a NG tube still remains at the same position, the NG tube-induced ulcer becomes further aggravated and then perforates into the laryngeal abductor, PCA, which is located just ventral to the upper esophagus. Consequently, PCA develops a severe myositis-like inflammatory reaction, resulting in the development of VCAP due to the weakness of the muscle in abduction. The present four patients shown in Table 1 were diagnosed as having NG tube syndrome from the findings that VCAP associated with inspiratory stridor developed after the indwelling of a NG tube and that PCA showed severe myositis-like changes, just like those of Sofferman et al.’s cases (6). Compared to the previously reported cases of NG tube syndrome, however, our patients were so peculiar in the following two points: absence of the postcricoid esophageal ulcer and recurrent episodes of VCAP. These features indicate that the mechanism of VCAP in our patients, namely a variant form of NG tube syndrome, is different from that in the previously reported NG tube syndrome having postcricoid esophageal ulcer.

Myopathological findings of PCA seen in our patients have a striking resemblance to those of the rat skeletal muscles after experimental ischemia or ischemia and subsequent venous occlusion (10). The radiographical studies of the postmortem laryngeal specimens with barium injection into the blood vessels clarified that the venous plexus in the esophageal wall covered the dorsal surface of the cricoid cartilage (11–13), as was confirmed by our pathological examination of the autopsied larynx (Fig. 3).

We, therefore, hypothesized that the injuries of PCA in our patients were caused by circulatory failure due to the

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**Table 1. Clinical and Myopathological Findings of Patients with a Variant Form of NG Tube Syndrome**

<table>
<thead>
<tr>
<th>Patients</th>
<th>Age (yr)</th>
<th>Sex</th>
<th>Disease duration (yr)</th>
<th>Number of VCAP episodes</th>
<th>Throat pain</th>
<th>Period 1</th>
<th>Period 2</th>
<th>Cause of death</th>
<th>Postcricoid esophageal ulcer</th>
<th>Histology of PCA</th>
<th>Pathological diagnosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>72</td>
<td>M</td>
<td>6</td>
<td>2</td>
<td>No</td>
<td>2 y</td>
<td>1 m</td>
<td>respiratory failure due to VCAP</td>
<td>No</td>
<td>myositis-like</td>
<td>Parkinson’s disease</td>
</tr>
<tr>
<td>2</td>
<td>77</td>
<td>F</td>
<td>0.5</td>
<td>2</td>
<td>Unknown</td>
<td>2 m</td>
<td>1 m</td>
<td>respiratory failure due to VCAP</td>
<td>No</td>
<td>myositis-like</td>
<td>Cerebral infarction</td>
</tr>
<tr>
<td>3</td>
<td>79</td>
<td>F</td>
<td>5</td>
<td>1</td>
<td>No</td>
<td>2 w</td>
<td>1.6 m</td>
<td>respiratory failure due to VCAP</td>
<td>No</td>
<td>myositis-like</td>
<td>Progressive supranuclear palsy</td>
</tr>
<tr>
<td>4</td>
<td>72</td>
<td>F</td>
<td>1.3</td>
<td>2</td>
<td>Unknown</td>
<td>3 m</td>
<td>1.5 m</td>
<td>respiratory failure due to VCAP, pneumonia, and respiratory center involvements</td>
<td>No</td>
<td>myositis-like</td>
<td>Creutzfeldt-Jakob disease</td>
</tr>
</tbody>
</table>

VCAP: vocal cord abductor paralysis, Period 1: Period from the first NG tube indwelling to the first VCAP episode, Period 2: Period from the last VCAP episode to death, PCA: posterior cricoarytenoid muscle.
compression of the PCA-perfusing veins and arteries by a NG tube. It indicates that postcricoid esophageal ulcer, which is a culprit in typical NG tube syndrome, is not involved in the development of VCAP in the variant form of NG tube syndrome. Concurrently, some of the PCA-innervating nerve branches from the recurrent laryngeal nerves may also be injured in the same manner, causing the neurogenic atrophy of PCA (7).

Another interesting finding in our patients is the “spontaneous recurrence” of bilateral VCAP in spite of the lack of treatment. A similar case with spontaneous recovery but not spontaneous recurrence was recently reported (9). We speculated that recurrence of VCAP was involved in the regular exchanges of a NG tube. Namely, a NG tube is usually exchanged regularly (every two weeks in our hospital) for its luminal smudge, while the possibility of the midline placement of a NG tube is only 6% (5) or 8% (14). Therefore, even if VCAP might happen after unfortunate midline position of a NG tube, it would be relieved after the next exchange of the tube since the possibility of repeated midline placement of a NG tube is so rare. This seems to be the reason why VCAP can develop spontaneous recurrence in our patients and why NG tube syndrome has not been encountered so often despite the popularity of patients receiving NG tube feeding. Considering that no esophageal ulceration was provoked, it is not strange that VCAP ameliorated without antibiotics and that throat pain, which is one of the triad of diagnostic criteria of NG tube syndrome, was lacking in the two patients without consciousness disturbance or dementia. Spontaneous and repeated remission and exacerbation of VCAP is quite unique and different from VCAP observed in the patients with neurodegenerative disorders where VCAP is, as a rule, progressive once it develops.

We speculated that NG tube syndrome is caused by multifactorial mechanisms including neurogenic, vasculogenic, and myogenic processes (Fig. 4). They correspond to the injury of the recurrent laryngeal nerve branches, ischemic and congestive myoinjury, and the postcricoid ulcer-induced myositis-like injury, respectively. More than two processes may participate simultaneously in the development of VCAP.

Since NG tube syndrome can develop in any patient under NG tube feeding, it is of importance to know the existence of NG tube syndrome and to distinguish this treatable NG tube-induced VCAP from the neurodegenerative disorder-related VCAP (15–18). However, the diagnosis of a variant form of NG tube syndrome described here was difficult, because neither postcricoid esophageal ulcer nor throat pain, which are diagnostic clues in the typical NG tube syndrome, was found. At present, midline position of a NG tube on the neck plain X-ray film and recurrent episodes of VCAP, if present, may be some clues to the diagnosis of a variant NG tube syndrome. In addition, a tentative decannulation under intravenous hyperalimentation for a few weeks may be useful as a procedure of the therapeutic diagnosis. To examine the motor function of the vocal cords, laryngeal needle electromyography and sleep load test may be useful. The former examination may disclose neurogenic discharges (19) or prolonged bursts of tonic activity suggestive of laryngeal dystonia (15). The latter may demonstrate sleep-induced exacerbation of VCAP, which is characteristic to VCAP in multiple system atrophy (20). Of importance is to think of the possibility of NG tube syndrome when patients under NG tube feeding develop inspiratory stridor, whatever the underlying disease is.

In conclusion, NG tube syndrome can appear in the patients under NG-tube feeding even in those whom no postcricoid esophageal ulcer is found. They may lack throat pain and repeatedly develop VCAP. We speculated that PCA in the patients with such a variant form of NG tube syndrome was injured by the circulatory failure of the PCA-perfusing
veins and arteries compressed by a NG tube.

References