

〈Case Report〉

Tongue coating and sudden death during hospitalization – A case report of fatal vasovagal reflex (bolus death)

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ABSTRACT Tongue coating is associated with aspiration pneumonia, particularly in long-term inpatients; however, it is not typically recognized as a direct cause of death. We report a recent case in which extensive accumulation of tongue plaque led to exfoliation and subsequent incarceration in the larynx, resulting in sudden death. The patient was a 70-year-old man who was emergently transported to a university hospital due to a right thalamic hemorrhage. Following admission, he remained bedridden and died suddenly on the 13th day of hospitalization. During tracheal intubation, a solid tissue mass was located in the larynx. Autopsy revealed two large, elastic masses measuring $3 \times 2 \times 0.7$ cm and $2.7 \times 1.5 \times 0.5$ cm within the larynx. Notably, there were no typical signs of asphyxiation, such as facial congestion or petechiae. The cause of death was determined to be sudden cardiac arrest due to the vasovagal reflex as a bolus mechanism triggered by the laryngeal incarceration of the exfoliated tongue coating. Sudden deaths can occur in hospitalized or nursing home patients, and a thorough investigation is essential to distinguish between sudden natural deaths and accidental or non-natural causes, such as suffocation. This case highlights that excessive tongue coating can, in rare instances, directly lead to death. Therefore, patients prone to heavy tongue coating require more meticulous oral care than is typically provided to prevent unexpected fatal outcomes.

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Key words : Tongue coating, Sudden cardiac arrest, Vasovagal reflex

INTRODUCTION

Tongue coating is a soft mass that accumulates on the tongue surface and forms normally. It is composed of exfoliated cells of the tongue epithelium, bacterial flora between the papillae, food debris, necrotic substances, inflammatory

cell, and other substances^{1, 2)}. If solid foods are not consumed, masticatory movement may become insufficient because of the disturbance of consciousness or digestive system disease; hence, the friction on the tongue surface is reduced, thereby increasing the grayish white mossy substances^{1–4)}.

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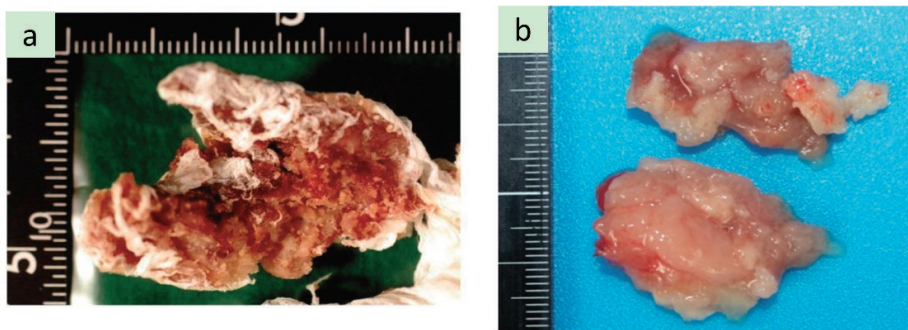


Fig. 1. Solid tissue material was found in the larynx in the hospital (a), and two other elastic solids were found from the pharynx to the entrance of the larynx during the autopsy (b).

Recent studies have revealed a correlation between a large amount of tongue coating and various disease states⁴⁻⁷⁾.

Studies have reported that tongue coating is associated with aspiration pneumonitis in long-term inpatients, older adults in care centers, and especially bedridden patients and that excess accumulation should be prevented^{8, 9)}. They emphasize the importance of oral care via prevention; however, no report has demonstrated the role of tongue coating in directly and immediately leading to death. To address this gap, we report a case of heavy tongue plaque that caused incarceration in the larynx and was a direct cause of death.

CASE REPORT

History

The patient was a 70-year-old man who was emergently transported to the hospital because of left lower extremity paralysis. His clinical history included alcohol consumption and high blood pressure. A diagnosis of right thalamic hemorrhage with ventricular perforation was made using computed tomography. Ventricular drainage and blood pressure control were performed for thalamic hemorrhage on the day of admission, and follow-up was continued.

Subsequently, spontaneous respiration and voluntary exercise were conducted, and the patient

was prescribed constant bed rest and was prohibited from oral ingestion. Nevertheless, oral care was provided every other day during hospitalization. Furthermore, nasal intubation was performed because the patient experienced glossoptosis symptoms after hospitalization.

On the 13th day of hospitalization, a nurse found that the patient had died following cardiopulmonary arrest, and cardiopulmonary resuscitation was unsuccessful. Postmortem CT revealed no expansion of the thalamic hemorrhage and no worsening of cerebral edema. However, a solid tissue slice was found in the larynx during tracheal intubation (Fig. 1a). The solid mass removed from the patient's larynx could not be identified; therefore, the patient was dissected.

Autopsy

The patient's height and weight were 165 cm and 60 kg, respectively. He had good nutrition and exhibited almost no congestion on his face. No obvious petechiae were observed in the conjunctiva, which was pale. No external injuries were discovered.

Two elastic solids, $3 \times 2 \times 0.7$ cm and $2.7 \times 1.5 \times 0.5$ cm, were observed from the pharynx to the entrance of the larynx (Fig. 1b). Many small gelatinous masses with similar characteristics were adhered to the area. An extremely thick tongue

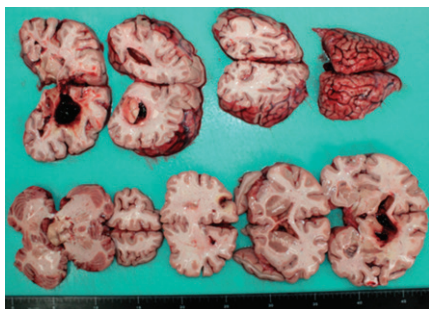


Fig. 2. Right thalamic hemorrhage was observed, with no progression compared to admission and no obvious cerebral edema.

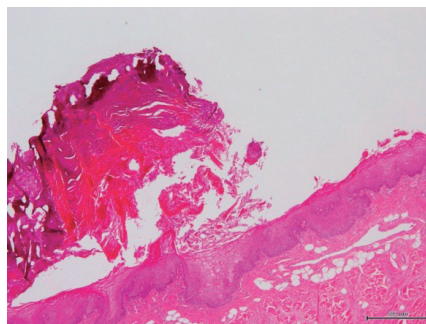


Fig. 3. Materials, mainly keratin, were adhered to the tongue surface, and exfoliated conditions were observed around the lingual root (HE $\times 40$).

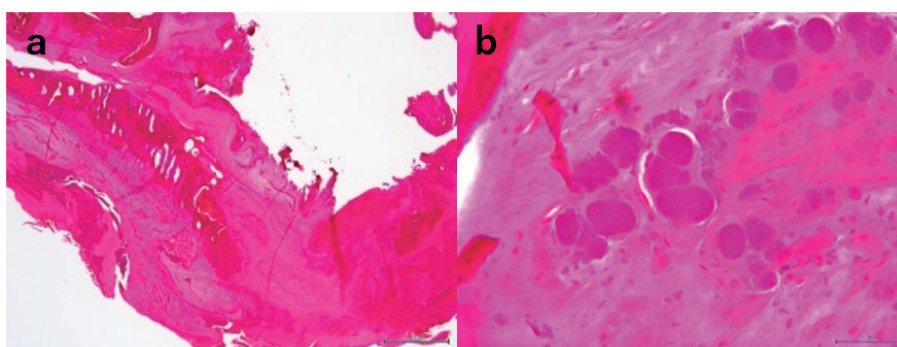


Fig. 4. Materials from the larynx and the substance collected in the hospital revealed bacterial masses or inflammatory cells scattered inside, mainly including keratin (a: HE $\times 20$, b: HE $\times 400$).

plaque was adhered to almost the entire anterior tongue, and a large vestige of exfoliation was observed near the lingual root. The substances incarcerated in the larynx were not detected in the respiratory tract.

A right thalamic hemorrhage was observed, partially extending into the thalamic ventricle, which showed no progression compared to the findings at hospital admission. The cerebrospinal fluid appeared bloody; however, cerebral edema and brain herniation were not particularly pronounced (Fig. 2). No petechiae were observed on the serous membrane of each organ, including the epicardium, or on the mucous membrane of the larynx and renal pelvis. No injuries or lesions that might have caused

death were observed in any of the organs.

Hematoxylin and eosin staining of the tongue revealed that substances such as tongue coating, which mainly included keratin, were adhered to the tongue surface, and exfoliated conditions were observed around the lingual root (Fig. 3). The observation of solids from the pharynx to larynx and the substances collected in the hospital revealed that bacterial masses or inflammatory cells were scattered inside, mainly including keratin. These substances were recognized as tongue coating (Fig. 4). Weak aspiration pneumonia was observed partially in the alveolus of the lung but was not assumed to be a cause of death. No specific lesions were observed microscopically in the other organs.

DISCUSSION

Although unexpected death is highly possible among inpatients, conducting a thorough investigation into the cause of death remains crucial. This is because of the difficulty in distinguishing between natural causes (e.g., pathological conditions or sudden circulatory failure) and accidental ones (e.g., suffocation by aspiration). In particular, the risk of accidental suffocation by aspiration is well known to be high in older adults and children, necessitating sufficient care^{10, 11)}. However, in the field of forensic medicine, when a foreign body is incarcerated in the throat, sudden death may not be caused by asphyxiation but by the vasovagal reflex.

Considering the microscopic findings and the condition of the tongue surface, the solids incarcerated in the patient's pharynx and entrance of the larynx were identified as tongue coating and the mass found in the larynx during tracheal intubation. Immediately before death, no deterioration in respiratory status was observed, and postmortem findings revealed no worsening of the thalamic hemorrhage or cerebral edema. Therefore, respiratory arrest was not considered to have been caused by these factors. Asphyxiation was suspected to be the cause of death, as no lesions that could be the potential cause of death were observed in any organ. However, no congestion was observed on the face or other organs, and no obvious petechiae were found. As no typical signs of asphyxiation were observed, the cause of death was concluded to be sudden neurogenic cardiac arrest due to the vasovagal reflex, a bolus mechanism triggered by incarceration in the larynx. This pattern of death is well recognized in the field of forensic medicine and is commonly referred to as "Bolustod". The exfoliation of the highly formed tongue coating directly caused death.

In the field of forensic pathology, diagnosing neurogenic cardiac arrest caused by vasovagal reflex is difficult. The most common cause is the physical

stimulation of the carotid sinus, such as hanging or strangulation; it may also be caused by mental conditions such as fear or anxiety and massive injury or pain¹²⁻¹⁵⁾. As in this case, direct stimulation by the mucous membranes of the pharynx and larynx could be a common trigger. According to *Forensic Pathology* by Saukko and Knight, death due to the vasovagal reflex is possible when foreign substances are incarcerated in the pharynx and larynx without symptoms such as respiratory distress or any typical signs of asphyxia¹⁶⁾. Accordingly, the present case is assumed to correspond to this state.

Based on our research, death due to the incarceration of tongue coating in the larynx has not yet been reported. Pneumonia is one of the most common causes of death in older inpatients and those living in care facilities. The general assumption is that the accumulation of tongue coating should be prevented to avoid it. Thus, pneumonia was regarded as a cause of death related to tongue coating. However, it has been found to be the direct cause of death in cases of high accumulation of tongue coating, as in the present case. In this case, the patient died due to the vasovagal reflex, which is believed to have progressed very rapidly to cardiac arrest. Therefore, successful intervention would only have been possible with immediate discovery. The underlying reason for the unusually pronounced formation of tongue coating in this patient remains unclear; however, it has been documented that there is considerable interindividual variability in the degree of tongue coating formation^{1, 3, 4)}, and the present case exhibited an extremely excessive accumulation of tongue coating despite typical oral care. Patients who tend to develop tongue coating require more meticulous oral care than routine care to avoid sudden and acute unexpected death.

DECLARATION OF INTERESTS

None.

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