CASE REPORT

Suffocation due to Thoracic Deformity Caused by Acromegaly

Toshihiko Yoshizawa¹, Masayuki Iwazaki², Kei Jitsuiki¹, Kouhei Ishikawa¹, Hiromichi Ohsaka¹ and Youichi Yanagawa¹

Abstract

A 61-year-old man with gigantism and acromegaly choked and fell into a coma. Immediate tracheal intubation resulted in a return of his consciousness. Enhanced computed tomography indicated that the trachea and left main bronchus were compressed by the thoracic spine and sternum. He required tracheotomy and positive end-expiratory pressure to maintain his pulmonary function. This is the first case of suffocation due to a thoracic deformity associated with acromegaly. Physicians should focus on clearing the tracheal airway using computed tomography to elucidate the anatomical relationship between the trachea and surrounding structures in acromegalic patients suffering from dyspnea.

Key words: suffocation, thoracic deformity, acromegaly

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Introduction

The causes of suffocation induced by airway obstruction include foreign objects, sputum, blood, vomiting material, allergic reaction, trauma, airway inflammation (including abscess), tumors, vascular issues, collapse of the airway wall (tracheomalacia or bronchomalacia), or chronic obstructive pulmonary disease (1). Suffocation may also be induced by a thoracic deformity due to compression of the trachea by the thoracic spine and sternum. Grillo et al. reported four adult patients who had tracheal compression caused by straight back syndrome, chest wall deformity, and anterior spinal displacement (2). In addition, Andrews et al. and Donnelly and Bisset reported abnormalities of the bony thorax causing tracheobronchial compression in children (3, 4). In Japan, we identified five cases of tracheal compression that were complicated with cerebral palsy, which could induce thoracic deformities (5-9).

In patients with acromegaly, the rate of death due to respiratory causes is three times higher than that in the general population and is most often caused by upper airway obstruction (10, 11). The upper airway obstruction in patients with acromegaly is generally considered to be caused by macroglossia and pharyngeal soft tissue hypertrophy and is rarely due to tracheal compression by thyroid swelling or the induction of bilateral paralysis of the vocal cord by nerve palsy due to soft tissue hypertrophy (10-15). In such cases, simple tracheotomy can achieve a favorable outcome. We herein report the first case of suffocation induced by an acromegalic thoracic deformity.

Case Report

A 61-year-old man admitted to the orthopedic ward for treatment of right artificial hip joint malfunction had complained of dyspnea, showed choking signs, had a low percutaneous oxygen saturation (SpO₂) (50%), and fell into a coma. Immediate tracheal intubation and back bulb ventilation resulted in a return of his consciousness. Sputum production was not confirmed by tracheal tube suction. On the same day, accidental self-extubation resulted in the reoccurrence of signs of upper airway obstruction, thus he underwent repeated tracheal intubation following deep sedation. At 36 years of age, the patient had undergone transcranial subtotal resection of a pituitary tumor following a diagnosis of acromegaly. The tube was removed and trachea intubation was maintained using the cough reflex. However, he had episodes of upper airway obstruction, such as choking, and required repeated intubation. In addition, the patient showed features of tracheomalacia and a thoracic deformity. Enhanced computed tomography revealed that the trachea and left main bronchus were compressed by the thoracic spine and sternum. The patient was diagnosed as suffering from acromegaly and was referred to our hospital for further treatment.

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Correspondence to Dr. Youichi Yanagawa, yyanaga@juntendo.ac.jp
of gigantism and acromegaly. After the operation, he developed pituitary dysfunction (requiring steroid and thyroxine replacement), postoperative epilepsy, steroid-induced diabetes mellitus and osteoporosis. He underwent right total hip replacement for coxarthrosis at 37 years of age. At 53 years of age, he became quadriplegic due to a cervical cord injury and required an apparatus for gate assistance. At 60 years of age, an abnormal mass in the right lung was confirmed, and this lesion was followed up on an outpatient basis. He did not have a history of an enlarged aorta, aortic tearing, dislocation of the lens of the eye or a family history of Marfan syndrome. Before this admission, the patient experienced dysphagia and normally consumed a liquid or soft food diet.

The findings of an arterial blood gas analysis before initial intubation (oxygen mask of 10 L/min) revealed a pH of 7.17, a pCO2 level of 74 mmHg, a pO2 level of 122 mmHg, a HCO3 level of 27 mEq/L and a lactate level of 1.0 mg/dL. Electrocardiogram showed right bundle branch block, and chest roentgen showed clear lung fields except for the abnormal lesion in the right lung. Enhanced computed tomography (CT) ruled out the existence of massive pulmonary embolism but indicated that the trachea and left main bronchus were compressed by the thoracic spine and sternum. After consultation with a respiratory physician and surgeon, we performed tracheotomy and inserted an Adjustfit tracheostomy tube (Fuji System Corporation, Tokyo, Japan) to maintain a long tracheal airway (Figure). He also required positive end-expiratory pressure to maintain his pulmonary function. When the patient attempted to breathe spontaneously through the tracheal tube, he showed retractions and frail chest-like movements with complaints of dyspnea in the supine position. He could occasionally breathe smoothly while in a sitting position. Permanent insertion of a tracheal stent was not recommended by respiratory physicians because this procedure could induce complete fatal obstruction of the tracheal airway. Accordingly, he was transferred to another hospital for thoracoplasty to achieve decompression of the trachea and left main bronchus. While waiting to undergo the operation, he died suddenly due to a lethal arrhythmia induced by pericarditis. An autopsy and pathological study failed to demonstrate any mechanical compression of the trachea.

Discussion

In the present case, a long tracheal tract and the main bronchus were compressed by hyperostosis and a thoracic spine and sternum; as such, simple tracheotomy was not effective. Scarpa et al. reported that arthropathy was the major cause of morbidity in cases of acromegaly compared with sex-, age-, and body mass index-matched healthy controls (16). In particular, a reduction in spinal mobility, alterations in the spinal profile and thoracic cage and features of diffuse idiopathic skeletal hyperostosis were significantly dominant in acromegalic patients. Accordingly, spinal deformity and the spine and sternum were found to compress the trachea in acromegalic patients.

The human body undergoes a variety of changes with age, including both morphological changes (e.g. increased thoracic kyphosis) and material changes (e.g. osteoporosis). Accordingly, the changes that occur with age in the present case may have resulted in the delayed onset of tracheal compression (17). However, in the present patient, resection of the ventral part of the thorax on autopsy, which was a decompressive procedure, failed to demonstrate compression of the trachea by the spine or sternum. This might be because the tracheal and bronchial cartilage is elastic; therefore, the decompression of the thoracic cage resulted in the restoration of the original shape of the lower airway. Grillo et al. also reported that the original shape of the trachea was restored after surgical procedures such as sternoplasty and sternal division (2). Accordingly, previous autopsies may have missed tracheal obstruction induced by thoracic deformity in acromegalic patients who died due to suffocation.

Physicians should focus on clearance of the tracheal airway using CT to determine the anatomical relationship between the trachea and surrounding structures in acromegalic patients suffering from dyspnea. In addition, as thoracoplasty is not simple to perform, an early diagnosis of acromegaly is mandatory to increase the chance of survival in such patients.

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