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CASE REPORT

Fatal bilateral pneumothorax and generalized emphysema following contraindicated speaking-valve application

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Abstract

We report a case of a contraindicated attachment of a speaking valve to a tracheal tube with an inflated cuff, which rapidly resulted in the patient's death. The attached one-way valve allowed unrestrained inspiration through the tracheal tube but prevented physiological expiration. The increased pulmonary pressure resulted in alveolar rupture and replaced expiration with a steady release of air into the peribronchial sheaths and the mediastinum, resulting in what is commonly known as the Macklin effect. From the mediastinum, air inflated both pleural cavities, the peritoneum, and the subcutaneous tissue of the entire body. No gas was found in the blood vessels, the brain, the bones, or in the inner organs. The entire air volume was estimated by radiological segmentation to be more than 25 l. This implies continuous inspiration, while expiration turned into an aberrant pulmonary decompression by whole-body gas-enclosure. Death ultimately resulted from asphyxia following bilateral (tension) pneumothorax.

Keywords Tension pneumothorax · Speaking valve · Tracheotomy · Tracheostomy · Subcutaneous emphysema · Macklin effect

Introduction

Tracheostomy and insertion of a tracheal tube allows consistent mechanical ventilation in patients with upper airway obstruction, copious secretions, and weaning failure [1]. The insertion of a tracheal tube prevents the patient from speaking, as air passes below the larynx and not by the vocal chords (Fig. 1a). Speaking valves, first introduced by Passy and Muir in 1986 [2], are connected to the end piece of the tracheostomy tube. Speaking-valves act as a one-way valve, allowing inspiration through the tube but closing on expiration, thus directing air upwards past the vocal chords to enable phonation (Fig. 1b). Whenever a cuffed tracheal tube is used, the application of a speaking valve demands prior cuff-deflation. If the tube remains cuffed, no air can escape the lungs and the trachea (Fig.1c). This scenario is mentioned as an explicit contraindication in speaking valve manuals [3], warning that "the patient will be unable to breathe if the cuff is not completely deflated". It is likely that a contraindicated misuse such as the cuff on a tracheal tube remaining inflated has occurred since the introduction of speaking valves, but no description of such a case is available in the current literature. In this paper we illustrate a case that ended in the patient's demise. The use of post-mortem computed tomography highlights the complex pathophysiology of this grave misapplication.

Case report

A 58-year-old man suffered from a non-identified inflammatory disease of the central nervous system. Despite multiple examinations (magnetic resonance imaging, blood samples, brain biopsy), the cause of the rapid progressive disease remained unclear. The patient suffered from fatigue with mood swings, disturbance of memory and speech, dysphagia, and spastic paralysis of the legs and the right side of the body.

Due to the increasing severity of neurological symptoms, the man lived in a special care home. To limit recurrent aspiration pneumonia, a tracheostomy was performed by cricothyrotomy (Fig. 1) about 9 months before the incident that led to his death. The patient regularly used a speaking valve several times a day for periods of 20 min. On the day of his death, a nurse attached the speaking-valve to the tracheal tube

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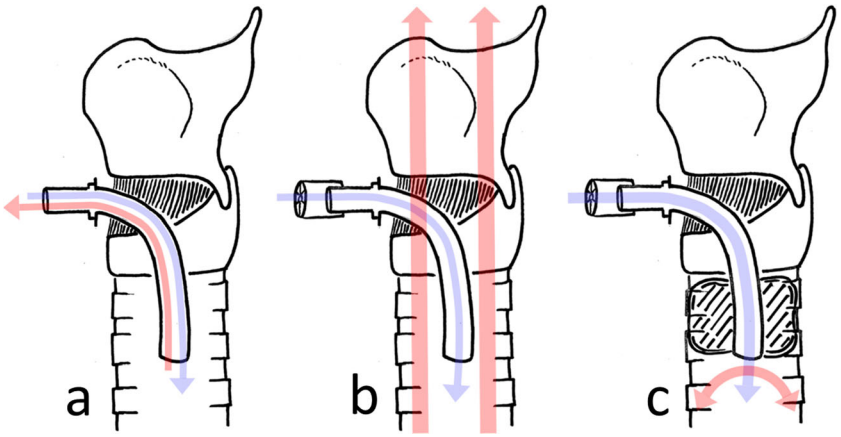

Fig. 1 Illustration of a tracheal tube and usage of a speaking-valve. **a** baseline state of a tracheal tube, inserted by cricothyrotomy as in the described case. The tracheal tube allows breathing, and may be cuffed, or uncuffed, depending on the indication. **b** correct application of a speaking-valve. The one-way valve only allows inspiration through the tube. In expiration, air is forwarded upwards to enable phonation. If present, the cuff must be deflated. **c** contraindicated use of a speaking-valve as in the reported case. While the one-way valve only allows inspiration, air can neither escape through the tube nor through the larynx because of the inflated cuff

without deflating the cuff. She had remained in the patient's room for about 30 more seconds. The patient did not attempt to speak or remove the valve by himself, which he was reported to have done previously, according to the care personnel. Nursing staff reentered the room 90 min later to find the patient dead and visibly swollen.

The body was brought to our institution roughly 5 h postmortem and was placed in a cooling cell. Whole-body postmortem computed tomography (PMCT) was conducted 18 h postmortem on a 128-slice scanner (Somatom Definition Flash, Siemens Medical Solutions, Forchheim, Germany) with a slice thickness of 1 mm, 0 .6mm increment and 120 kV tube voltage. PMCT revealed a correctly placed tracheal tube with an inflated cuff (8 mm tracheal tube, Mallinckrodt, Staines-upon Thames, England) and an attached speaking valve (Shiley Phonate Speaking Valve, Covidien/Medtronic, Dublin, Ireland) (Fig. 2). Pneumoperitoneum and bilateral pneumothoraces were detected. The mediastinum exhibited a significant shift to the left. The lung tissue appeared congested and compressed. Pronounced generalized emphysema in the mediastinum and in whole-body subcutaneous and muscle tissue was noted. No gas collections were present in the inner organs, vessels, brain, and osseous tissue. In order to exclude gas formation due to putrefaction, a radiological alteration index of 8/100 was computed [4]. A rough segmentation performed with dedicated software (syngo.via; Siemens Healthineers, Erlangen, Germany) estimated the total gas volume to be >25 l (excluding pleural cavities and intestines), but an exact measurement was impossible due to diffuse and often minimal gas collections, and the true volume is likely to have been much higher. The subsequent autopsy confirmed the PMCT-findings and supplied no divergent information. The cause of death was asphyxia. Infection or colonization with gas-forming bacteria could be excluded by microbiological sampling from the peripheral blood, liver, and spleen.

Discussion

Assessing the chronological order of the proposed events that led to the patient's death, the pathophysiology becomes evident. The attachment of a speaking valve immediately prevented all expiration of air. As air could not escape the trachea, pressure increased in the alveoli, which then ruptured at their base and released air into the peribronchial sheaths, a process called the "Macklin effect" [5]. When pressure gradients prevail, this process may be continuous, a process that is sometimes referred to as "milking" air from the ruptured alveoli [6]. Once in the perivascular sheath, air may travel freely in to the mediastinum, the retroperitoneum, the peritoneum, and the subcutis, and the peritoneum [6]. It also may lead to pneumothorax by a pressure-related rupture of the parietal pleura, resulting in (tension-)pneumothorax, as observed in this case.

Postmortem computed tomography allowed the total volume of gas in the deceased to be roughly estimated at more than 25 l, excluding gas in both the small and large intestine and the pleural cavities. As the normal breathing volume is about 0.5 l [7], the high amount of intracorporal gas implies that pulmonary decompression by "alveolar milking" was an ongoing process. Furthermore, as the patient was not mechanically ventilated, we must assume that he continued to "breathe" by normal inspiration through the tracheal tube and passive deviant expiration – by "whole-body pulmonary decompression". Death ultimately resulted from asphyxia, following bilateral pneumothorax and, therefore, a compression of functional pulmonary tissue and insufficient gas exchange.

While Byard et al. described lethal complications of tracheostomy as seen in autopsy [8], no cases connected to the application of speaking valves have been reported before.

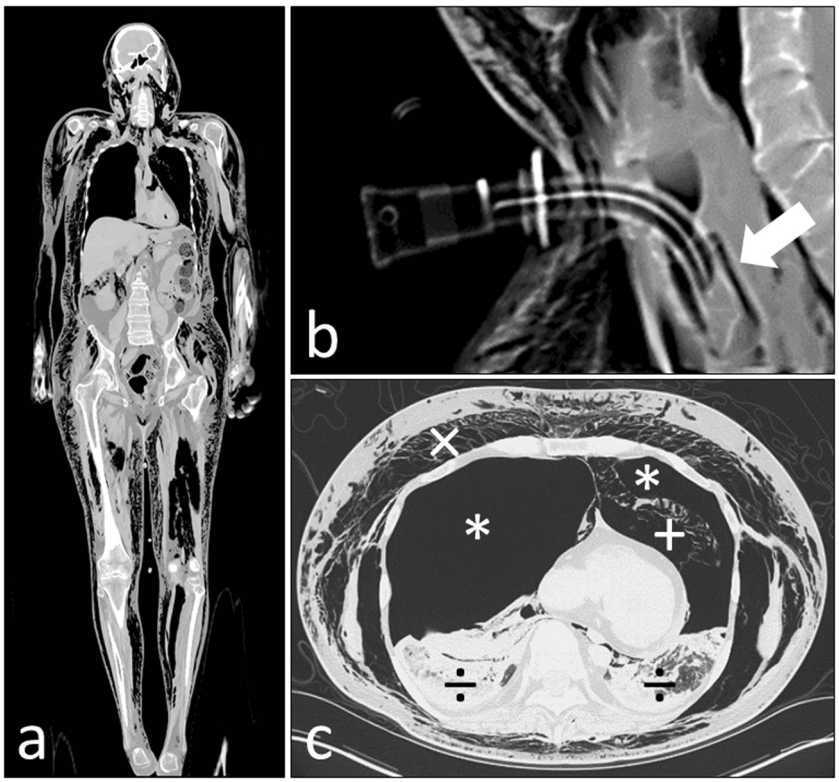

Fig. 2 Overview of computed tomography findings. **a** coronal whole-body plane (slice thickness: 15 mm, window center: −200 HU, width: 900 HU), generalized subcutaneous emphysema, no gas collections in the brain and abdominal organs. **b** sagittal plane of the neck (slice thickness: 15 mm, window center: −250, width HU: 1700 HU): inserted tracheal tube with attached speaking valve, the cuff remained inflated postmortem (white arrow). **c** transverse plane of the chest (slice thickness: 0.6 mm, window center: −600 HU, width: 1200 HU): * bilateral pneumothoraces, + pneumomediastinum, **x** subcutaneous emphysema, ÷ congested and compressed lungs

The formation of mediastinal and subcutaneous emphysema, often in conjunction with pneumothorax, has however been reported many times in the clinical literature. The common denominator in these cases is an increased intrapulmonary pressure, either by airway obstruction and forced expiration, increased pressure in mechanical ventilation (barotrauma), or simple valsalvoid states such as coughing or labor [9–11]. The observed fulminant emphysema in this case, in comparison to other states of airway obstruction, is coherent when considering the decisive difference: the unhindered inspiration enabled by a one-way valve.

The proposed pathophysiology is dependent on the claim that the intracorporal gas was not formed by anything other than inspiration. Gas-forming bacteria are known to present similar PMCT images as were observed in this case. The negative microbiology for gas forming bacteria excludes this confounder with a high probability. In addition, generalized gas collections are a very common phenomena in PMCT with increasing postmortem interval as part of the stages of putrefaction. With a low postmortem interval of 18 h (13 h cooled) and an RA index of 8/100, we can exclude gas formation due to putrefaction.

Conclusion

This case report illustrates the consequence of a severe misapplication of a speaking valve while leaving the cuff of a tracheal tube inflated. The suggested pathophysiology involves a highly aberrant expiration by whole-body pulmonary decompression. It may serve as a reference for future guidelines of speaking valves, and as an explicit call for the careful handling of speaking-valves in the tracheotomized patient.

Key points

1. The application of a speaking valve in a tracheotomized patient is contraindicated while the tube-cuff is inflated.
2. In the reported case, a severe misapplication of a speaking-valve resulted in the patient's demise.
3. Increased pulmonary pressure leads to alveolar rupture and the release of air into the mediastinum, the so-called Macklin effect.
4. The speaking-valve prevented physiological expiration, which led to pulmonary decompression to the whole body subcutaneous tissue via the Macklin effect.
5. Death occurred due to asphyxia in bilateral tension pneumothorax, resulting from high mediastinal air-pressure.

Compliance with ethical standards This article does not contain any studies with human participants or animals performed by any of the authors. Ethical approval was obtained by the Ethics Committee of the Canton of Zurich, Nr. KEK ZH-Nr. 15-0686.

Conflict of interest None.

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