J Forensic Sci, September 2008, Vol. 53, No. 5
doi: 10.1111/j.1556-4029.2008.00827.x
Available online at: www.blackwell-synergy.com

CASE REPORT

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Iatrogenic Tracheal Laceration Causing Asphyxia

ABSTRACT: Endotracheal intubation is a simple, rapid, and safe technique that is being used as a standard procedure for airway management. However, airway injury during endotracheal intubation could be a significant source of morbidity or even mortality for patients and a source of liability for physicians as well. We report an unusual case of fatal tracheal occlusion by intraluminal blood clot complicating endotracheal intubation. The patient, a 62-year-old woman, with renovascular hypertension and incipient renal failure was scheduled for renal autotransplantation. The surgery was uneventful but the postoperative course was complicated with a lethal airway obstruction. At autopsy a linear longitudinal tracheal laceration was identified with an intraluminal blood clot obstructing the tracheal lumen. Tracheal laceration as a cause of death is a rare and potentially fatal complication of endotracheal intubation with intratracheal bleeding, clot formation, tracheal occlusion, and subsequent asphyxia.

KEYWORDS: forensic science, asphyxia, tracheal occlusion, intratracheal intubation

Airway management is one of the most important aspects of anesthetic practice and emergency and critical care medicine with endotracheal intubation (ETI) being a standard procedure. However, occasional airway injury during ETI is responsible for acute and chronic complications thus contributing to morbidity or even mortality for patients and liability for physicians. Long-term complications such as inflammatory stenoses, tracheoesophageal fistulas, and tracheo-innominate artery fistulas are well documented in the literature (1). On the contrary, acute complications such as laceration of the membranous part of the tracheobronchial wall are rare and potentially devastating events (2). We report an unusual case of tracheal laceration following ETI with subsequent bleeding and clot formation leading to acute tracheal occlusion, asphyxia, and death of the patient.

Case Report

The patient, a 62-year-old woman, was hospitalized on multiple occasions at the Department of Nephrology for renovascular hypertension and incipient renal failure. Her previous medical history was unremarkable and she had no genetic disorder predisposing her to thrombosis. Dynamic renal scintigraphy revealed bilateral atherosclerotic stenosis of renal arteries with severe functional impairment of the left kidney. Transbrachial percutaneous transluminal angioplasty of the right renal artery had been previously performed but was not successful. The patient refused to undergo the procedure again and was scheduled for renal autotransplantation. Preoperative intubation and postoperative extubation were performed by an experienced anesthesiologist without any difficulties. The surgery was completely uneventful. After extubation the patient was eupnoic, fully conscious, and hemodynamically stabile. However, 3 h later she started coughing and complained of inability to expectorate. She received antiedematous therapy for suspected vocal cord irritation caused by prolonged intubation. However, as her complaints continued, the nasopharynx and trachea were aspirated using a thin aspiration catheter, and a thin coagulum was obtained. Her condition temporarily improved, but 2 h later she started to expectorate blood-tinged sputum. Breathing became stridorous, oxygen saturation started to decrease, and she became hypotensive and bradycardic. Emergency intubation was performed but subsequent ventilation was difficult due to airway resistance. Malposition of the tube was suspected, so the patient was reintubated without any consequence on the ventilation. Cyanosis progressed and the patient became asystolic and died in spite of resuscitation efforts.

At autopsy, a linear longitudinal laceration (4 cm in length) was identified on the right side of posterolateral tracheal wall in association with a large intraluminal red-gray blood clot obstructing the tracheal lumen and extending to the right main bronchus (Fig. 1). Bilateral hydrothorax (200 mL on each side), hypertrophy of the left ventricular wall, and acute pulmonary edema were identified. Histologically, the entire blood clot was composed of fibrin, platelets, and red blood cells. No morphological abnormality of the tracheal wall was identified on histological slides. The cause of death was classified as asphyxia secondary to acute airway obstruction by tracheal blood clot.

Disscusion

Tracheobronchial lacerations may be traumatic and iatrogenic in origin. These injuries are caused by completely different mechanisms leading to different morphological appearances and therapeutic options (3). In contrast to traumatic tracheobronchial injuries, which are high-velocity injuries leading to more or less complete horizontal transections of the trachea, iatrogenic lacerations are low

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Received 6 Oct. 2007; and in revised form 24 Jan. 2008; accepted 27 Jan. 2008.

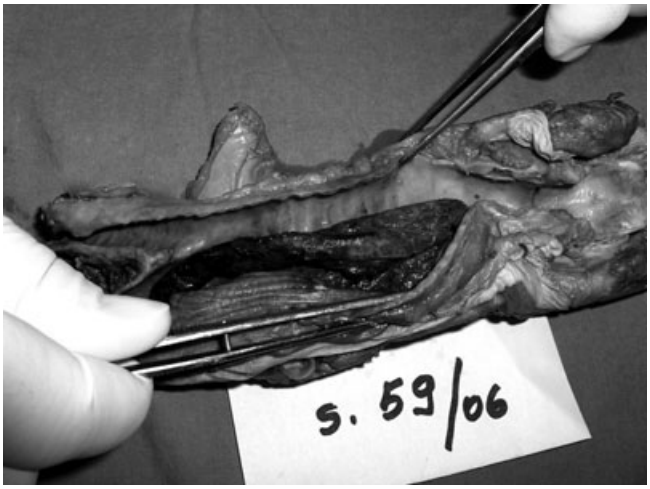

FIG. 1—*Section of the trachea showing linear tracheal laceration with intraluminal tracheobronchial thrombus.*

impact injuries (3). In more than 98% cases, iatrogenic tracheal lacerations are longitudinal tears affecting the membranous portion of the trachea or junction of membranous and cartilaginous part on the right side of the trachea (3–5). Iatrogenic tracheal lacerations are rare ETI complications occurring in about 1:10,000–20,000 intubations (3,5).

There are multiple mechanical, anatomic, and unidentified individual factors that lead to iatrogenic tracheal injury (6). The trachea has cartilaginous support, which protects it against injury, but the posterior tracheal membrane is unsupported by cartilaginous rings. The deleterious effect of high cuff pressures on tracheal mucosa is well known. If the cuff pressure exceeds the normal capillary perfusion pressure (normal range is 25–30 mmHg), the resulting cessation of capillary circulation may cause ischemic mucosal damage (7). Another possible cuff-related injury is simple mechanical disruption as soon as the diameter of the cuff exceeds the maximum diameter of the airway (7).

Physician errors (multiple attempts, inexperience, cuff overinflation, malposition of the tube, moving the tube with cuff inflated), inadequate equipment selection (inappropriate use of stylets, improper tube size), patient actions (abrupt movements, excessive coughing), and anatomic factors (weakened membranous part of tracheal wall owing to inflammatory disease, corticosteroid therapy and aging, chronic obstructive pulmonary disease, tracheomalacia) all contribute to this problem (4–6,8). The risk of tracheal laceration is greater in short females (2,3,6) as they have narrower airways increasing vulnerability to cuff overinflation (4). The risk is also greater in patients with tracheal distortion caused by neoplasm or large lymph nodes. Tracheal disruption has been reported to be more frequent if the airway management is difficult as a result of an emergency situation.

Recognizing injuries that can result from resuscitative ETI is important because these injuries may mimic injuries caused by neck compression such as that produced by manual strangulation (9). Such a distinction may be crucial in the final diagnosis and disposition of certain deaths (9). The clinical reports regarding intubation injury record mucosal lacerations, tracheal rupture, and cricoid cartilage fractures (2,7). However, more important to the forensic pathologist are the acute and relatively minor injuries of ETI because of their potential for misinterpretation (9). The study by Raven et al. shows that a large number of individuals intubated in the field sustained airway injury ranging from mucosal petechiae to confluent contusions distributed in various sites from the mouth to the carina (9).

None of these predisposing factors was noted in our case. In a case of an intubation without difficulties and predisposing factors to injury, it is difficult to determine the etiology of the laceration. Data from the literature indicate that most lesions are due to a hyperinflation of the cuff, which leads to a separation of the membranosa and a linear laceration of the posterior membranous wall (2,5). Considering the location of tracheal laceration in our case, we can assume that this mechanism of tracheal injury contributed to the formation of the injury.

Patients with a tracheobronchial laceration may be asymptomatic or have various degrees of respiratory distress and hemodynamic compromise. Two different clinical types may be recognized: one type shows a sudden development of marked mediastinal and subcutaneous emphysema, which is recognized usually without delay (3). The second type has no emphysema and is recognized usually with a delay up to a week (3). Subtle signs, such as coughing and chest pain, may be present and may be the only indication of a significant injury. Clinical symptoms observed in our case were subtle and did not raise any suspicion of a possible tracheobronchial laceration. Bradycardia and hypotension suggested a possible pulmonary embolism and difficult postoperative ventilation was explained only at autopsy.

Asphyxia caused by airway obstruction due to the presence of a tracheobronchial blood clot occurs in a variety of clinical settings and has been documented as a complication of different conditions including iatrogenic ones (10). Pathologic conditions include bronchiectasis, tuberculosis, mitral stenosis, pulmonary infarction, pulmonary arteriovenous malformation, sarcoidosis, bronchial carcinoma, and intrathoracic trauma. Iatrogenic mucosal damage from suction catheter manipulation, bronchoalveolar lavage, transbronchial biopsy, and tracheostomy placement could also lead to the formation of airway blood clots (10). The impact on respiratory function may be minimal or life-threatening. In the setting of substantial hemoptysis, this entity should readily be considered in the differential diagnosis (10). However, *c*. 30% of endobronchial blood clots occur without evidence of hemoptysis. Literature search revealed only two published cases of asphyxia caused by tracheal thrombotic occlusion after prolonged intubation of 10 and 12 days and both had no documented evidence of tracheal injury (11). According to our knowledge, this is the first case of acute asphyxia caused by tracheal laceration with subsequent intratracheal bleeding.

Conclusion

Tracheal laceration as a cause of death is a rare and potentially fatal complication of ETI with intratracheal bleeding, clot formation, tracheal occlusion, and subsequent asphyxia. Physicians performing an intubation should be aware of the consequences of a possible tracheal rupture so as to prevent potentially lethal events.

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