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CASE REPORT

Tracheal rupture after intubation due to cuff overinsufflation

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Abstract

A 59-year-old woman developed a tracheal rupture after intubation, which was most likely due to high cuff pressure. Several factors, mechanical and anatomical, may contribute to iatrogenic tracheal ruptures. The most common hypothesis is overinsufflation of the tracheal cuff. This can be prevented and doctors should be aware of the existing risk factors contributing to tracheal lesions.

Introduction

Tracheal rupture after intubation is a serious but rare complication. Several risk factors have been described in previous publications. In the following case, we describe these factors and how tracheal ruptures might be prevented.

Case

A 59-year-old woman, with no medical history or medication use, height 168 cm and weight 85 kg, was admitted to the neurology department because of a cerebrovascular accident (CVA) with right-sided paralysis. One week later, the admission was complicated by an ileus. She developed respiratory insufficiency most likely because of aspiration, for which she was admitted to the ICU for intubation and mechanical ventilation. Intubation was not anticipated to be difficult based on appearance; however, a Mallampati score was not obtained because of the urgency of the case. Intubation was uncomplicated using a tube size seven by an experienced anaesthesiologist-intensivist (Cormack-Lehane classification grade 1). A Parker flex-tip, high volume low pressure endotracheal tube was used. The stylet was removed at the moment the tube passed the vocal cords. Inspiratory pressures were initially high to obtain adequate oxygenation and ventilation. A recruitment manoeuvre was performed using up to 20 cmH_2O PEEP and a cuff pressure of >30 cmH_2O was needed for a maximum of 15 minutes because of air leakage. A chest X-ray performed immediately showed a deep tube position but still above the carina, and consolidation in the left basal lung field. The tube was withdrawn several centimetres. A CT chest and abdomen was then performed to determine the cause of the respiratory insufficiency, which showed diffuse dilatation of intestinal loops with liquid and air reaching from the proximal jejunum up to the rectum without evidence of obstruction. The left lower pulmonary lobe was completely atelectatic; there were no signs of aspiration. A significant pneumomediastinum with subcutaneous emphysema up to the neck was seen, as well as a left lateral wall tracheal lesion of about 3 cm above the carina, near the cuff (which was substantially inflated), was also present *(figure 1)*. A possibly iatrogenic tracheal lesion complication was reported. The pulmonologist was consulted who performed a bronchoscopy, which showed a large tracheal lesion along the pars membranacea (over a distance of 8-10 cm). A tear was seen at the junction of the cartilage, the greater part along the right side of the trachea, with no options for placing a tracheal stent

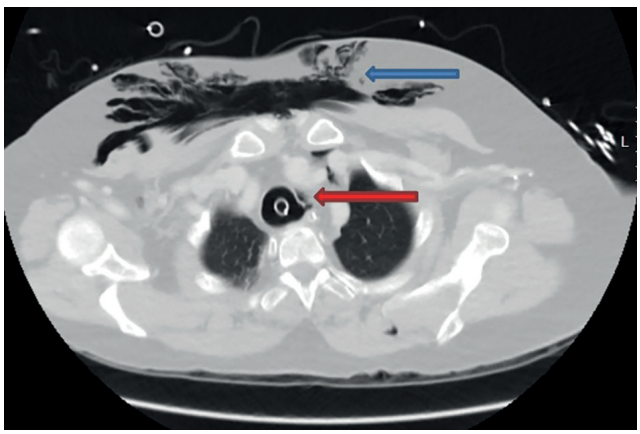

Figure 1: CT-chest of our patient, which showed subcutaneous emphysema (blue arrow) and a significant pneumomediastinum with a left lateral wall tracheal lesion of about three centimetres above the carina (red arrow), near the cuff which was substantially inflated.

(figure 2). After consulting the thoracic surgeon, it was decided that veno-venous extracorporeal membrane oxygenation (V-V-ECMO) was the only option to enable surgical closure of the defect. On day 3 of ICU admission, V-V-ECMO catheters were inserted (right jugular vein and right femoral vein) and the tracheal defect was closed using a xeno-pericardial patch. The tracheal tube was removed to allow optimal wound healing. No continuous infusion of heparin was started because of the pre-existing CVA. Daily tracheal suction was performed by the pulmonologist. Antibiotics were given for possible mediastinitis, following the findings on CT chest.

On day 6, substantial subcutaneous emphysema of the thorax and face developed. This finding was apparently due to dehiscence and failure of the pericardial patch to graft, most likely because of the pre-existing mediastinitis. At this point, no further treatment options were available. Thereby, the expectation of a poor outcome due to the CVA made the overall prognosis detrimental. After discontinuation of treatment, our patient passed away.

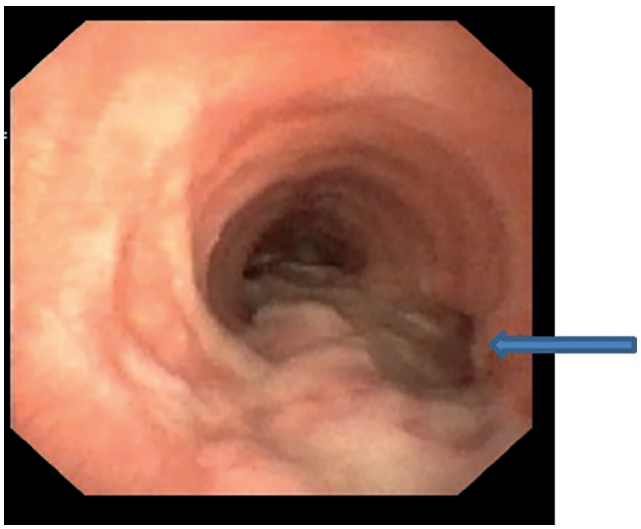

Figure 2: Bronchoscopy which shows a tear at the junction of the cartilage on both sides, the greater part along the right tracheal side (arrow).

Discussion

Iatrogenic tracheal rupture after intubation is a rare but serious complication and is estimated to occur in 0.05-0.37% of intubations.[1] Its complications include tension pneumothorax or anoxia (when most of the tidal volume is leaking through the tracheal tear). Mediastinitis and sepsis may occur at a later stage.[2,3] Risk factors may be divided into mechanical and anatomical. Mechanical risk factors include multiple forced intubation attempts, inexperience, overinflation of the cuff, incorrect position of the tip of the tube, inappropriate tube size, inflammation or advanced age. Anatomical risk factors include congenital abnormalities, pars membranacea weakness, female gender and short stature.[1,4,5] The most common probable cause for iatrogenic tracheal rupture after intubation is cuff overinsufflation and this is most commonly found in women (86.2%) who are on average over 50 years of age. Almost all ruptures described in the literature are found in the posterior wall of the trachea, usually longitudinally, at the junction of the cartilage, along the right side of the trachea.[2-4] The rupture usually corresponds to the length of the cuff, but extensive tearing can be explained by further dissection under positive pressure ventilation. It is difficult to explain these right posterolateral lacerations by intubating trauma, when the tip of the tracheal tube has been inserted anteriorly through the cord.[2] The posterior part of the trachea might have a higher tendency to rupture with cuff overinsufflation because it is weaker due to a lack of cartilaginous support.[6] Cuff pressures over 30 cmH_2O can already cause mucosal tissue damage because of reduced blood flow. Complete blockage of flow can occur with pressures exceeding 45 cmH_2O.[7] This may lead to tissue ischaemia and weakness of the tracheal wall. Ruptures will develop more easily in the case of high cuff pressures.

In our case, we performed an uncomplicated intubation. The cuff, however, may have been over inflated because of air leakage and secondary ventilatory difficulties. This serious complication can occur after only a short period of high cuff pressure. The use of high ventilator pressures and recruitment manoeuvres may have contributed to aggravation of the tracheal lesion. Therapeutic options when high pressures are needed due to ventilatory difficulties may be to check the cuff position (between vocal cords leading to air leakage?) and tube size (inefficient size related to trachea?), to deepen anaesthesia, and use neuromuscular blockade or bronchodilator therapy.

Our patient was of short stature, older than 50 years and female, therefore at risk for developing intubation or high cuff pressure related tracheal lesions. These factors may all have been contributing factors to the development of the tracheal rupture. Furthermore, as described in the literature, lesions caused by cuff overinflation are often located on the right side of posterolateral wall, which was also affected in our case.[2-4,6]

Overinsufflation of the cuff can be prevented by frequent measurement of the cuff pressure. We should be aware of cuff pressure values while ventilating patients on the ICU and keep in mind that short women of an older age are at risk of developing tracheal lesions. This could be because of the weaker pars membranacea and smaller diameter of the trachea compared with men.[4,8,9,10] Besides that, we should avoid manual regulation of the cuff without a pressure gauge because this will lead to the recommended pressure in less than 30% of cases.[8]

Disclosures

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