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Immediate postoperative airway obstruction secondary to airway edema following tumor excision from the neck

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Abstract
A 46-year-old woman was scheduled for excision of a malignant peripheral nerve sheath tumor from the neck. The tumor had caused deviation of the trachea to the left and partial obstruction of the superior vena cava. Her upper airway at laryngoscopy after induction of anesthesia was normal. During tumor resection there were transient phases characterized by the complete disappearance of the peripheral oxygen saturation ($S_{p_{O_2}}$) and radial artery tracings. At the end of the operation, the trachea was extubated after ensuring adequate antagonization of neuromuscular blockade. However, immediately post-extubation, she showed signs of acute airway obstruction that necessitated reintubation of the trachea. Laryngoscopy revealed significant edema of the upper airway and vocal cords, requiring a smaller size tracheal tube. Many reports suggest the development of significant airway edema 24 h after such surgery. Our report highlights the fact that this can happen in the immediate postoperative period also. Some authors suggest that, in such surgery, extubation should routinely be done over pediatric tube exchangers. Routine leak testing and direct laryngoscopic/fiberoptic evaluation of the upper airway prior to extubation may also help. While our report reaffirms these points, it also stresses the importance of intraoperative monitoring for the compression of the great vessels, which may serve as a useful indicator of the early development of airway edema.

Key words Airway edema · Airway obstruction · Intubation · Neck surgery

Introduction

Multiple risk factors may predispose to the development of postoperative airway edema in a previously normal airway following extensive surgery in the neck. These factors may be related to the surgery or anesthesia, or they may be due to immune-mediated responses. Surgical factors such as the obstruction or destruction of major neck vessels and lymphatics or injury to the nerves supplying the airway are predominantly responsible for causing airway edema [1–5]. Anesthesia-related factors include the prolonged presence of an endotracheal tube and repeated attempts at intubation [6,7]. Often airway edema and obstruction develop as a subacute complication following radical neck surgery, maxillofacial surgery, or cervical spine surgery [8–11]. We report a case where, following the excision of a neck mass, a patient with a previously normal airway developed severe edema leading to obstruction that mandated reintubation of the trachea in the immediate postoperative period.

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Case report

A 46-year-old woman was scheduled for excision of a malignant peripheral nerve sheath tumor from the neck. She had no other comorbidities. She had distended veins in the upper limbs and on the chest, with mild edema of both the upper limbs. Although her trachea was significantly shifted to the left, she did not have any evidence of respiratory distress. Magnetic resonance imaging (MRI) of her neck and chest showed that the tumor measured approximately 6×4 cm and extended from just below the larynx to just beneath the clavicle on the right side and was causing partial obstruction of the superior vena cava (SVC), while resulting in significant deviation of the trachea to the left (Fig. 1). The MRI also showed intrathoracic extension and compression and deviation of the great vessels, but encroachment into the great vessels was not demonstrated (Figs. 2, 3). A 16-gauge intravenous (IV) cannula had been placed in her left upper limb, and the line showed free flow. Anesthesia was induced through the same line, with propofol and fentanyl, and neuromuscular

blockade was achieved with vecuronium after confirming the patient's ability to mask-ventilate. Post-induction another venous access was obtained in the left lower limb, and intraoperative fluid infusion was maintained through this access. The upper limb venous access was kept patent with a flow rate of approximately 25 ml·h^{-1}. Tracheal intubation was achieved with a single attempt by direct laryngoscopy with a 7.0-mm internal diameter (ID) polyvinylchloride (PVC) cuffed endotracheal tube. There was no evidence of airway edema or vocal cord edema on laryngoscopy. Post-intubation, an arterial line was secured in the right radial artery and invasive blood pressure monitoring commenced immediately. The patient was placed in a 20° head-up position. On five occasions during the surgery, it was noticed that the arterial trace had completely disappeared, along with the simultaneous absence of the peripheral oxygen saturation (S_{PO_2}) trace and the flow of IV fluid into the left upper limb. However, each instance was

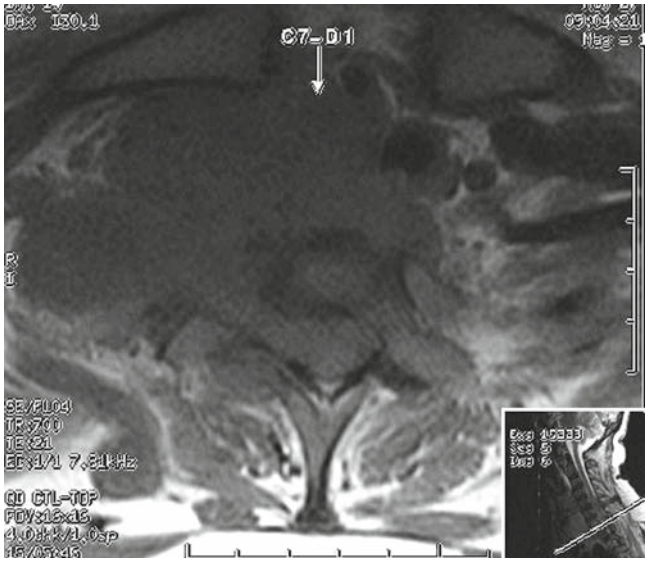

Fig. 1. Magnetic resonance imaging (MRI) of neck at C7-D1 level, showing significant deviation of the trachea to the left and compression of major neck vessels

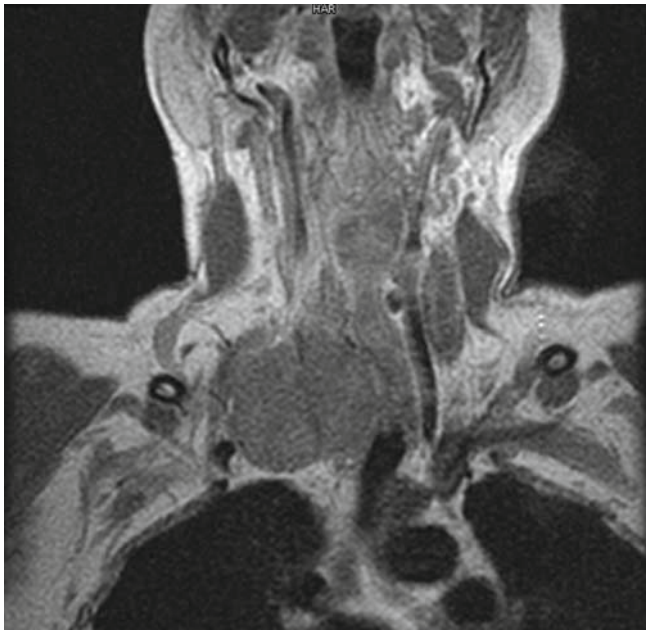

Fig. 2. MRI of neck; axial view showing deviation of the trachea to the left and the dimensions of the tumor

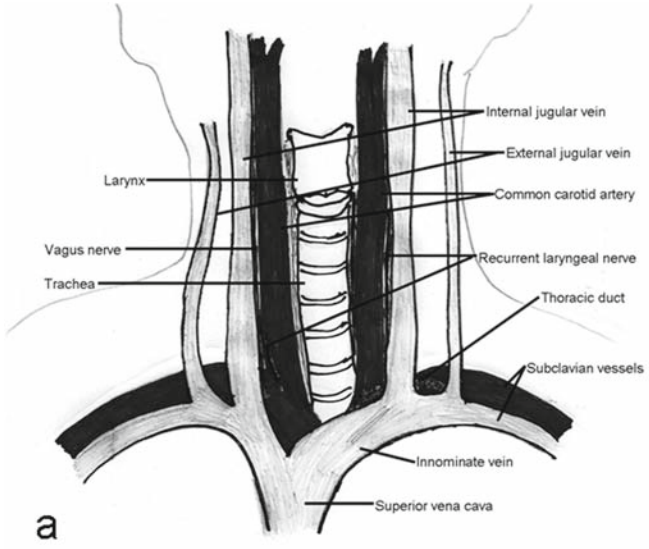

Fig. 3. **a** Schematic diagram representing normal relationship between larynx, vessels, nerves, and lymphatics in the neck and upper thorax. **b** Schematic diagram representing the changes in the anatomy of the neck structures as a result of tumor compression in our patient

brief and lasted less than 10 s, as the surgeon was notified immediately and pressure applied at the operating site was relieved; this resulted in the immediate restoration of the arterial trace, Sp_{O_2}, and the flow of fluid through the left upper limb. On three more occasions, it was noticed that the flow of IV fluid through the left upper limb was intermittently obstructed, although the Sp_{O_2} and arterial trace remained unaffected. The tumor was neither adherent to nor infiltrating the major vessels in the neck, but its complete removal was not possible, and a debulking procedure was performed. A total infusion of 2000 ml crystalloids was administered during this 3-h-long surgery, with a 300-ml blood loss. At the end of the procedure, the neuromuscular blockade was reversed. A cuff-leak test was not performed before tracheal extubation, as the surgeon felt that the tracheal wall was firm and not adherent to the tumor. At the end of the procedure it was observed that the patient had a moderate amount of conjunctival suffusion; however, there was no obvious edema of the eyelids or face. Extubation was performed once the patient was conscious, oriented, and generating tidal volumes of 8–10 $ml \cdot k^{-1}$.

Immediately following extubation the patient started struggling to breathe. Tracheal tug and nasal flaring developed, while an exhaled tidal volume of only 27–76 ml could be registered with a tightly fitting face mask. No improvement was observed despite instructing the patient to breathe slowly through the mouth. A triple maneuver with an appropriate-size nasopharyngeal airway resulted in partial relief to the patient, and the tidal volume improved to 140–165 ml. No further improvement occurred during the following 10 min. Therefore, we decided to reintubate the trachea. After inducing the patient with propofol, direct laryngoscopy was performed. This revealed significant edema of the vocal cords and supraglottic tissues, although vocal cord mobility was normal. Because of the significant edema of the supraglottic tissue, the patient was intubated with a smaller size (5.5-mm ID) endotracheal tube. Immediately following the intubation, as she started recovering from propofol and resumed breathing, it was noticeable that all the manifestations of respiratory distress had resolved. She was kept on humidified oxygen therapy through a T-piece and was nursed in the propped-up position. A course of steroids was given. Her trachea was extubated only 24 h after the reintubation, following a fiberoptic evaluation of the airway, which revealed the presence of minimal airway edema and normal vocal cords. The patient remained comfortable throughout her stay in the hospital. Following extubation, an indirect laryngoscopy was done, which confirmed normal movements of the vocal cords. She was discharged home after 7 days. The patient has kindly consented to the publication of the data and her images in the journal. We have obtained her written informed consent.

Discussion

Acute or subacute postoperative airway edema following surgery of the head and neck is a well-documented complication. This edema may occur as a result of hematoma formation at the surgical site, vocal cord dysfunction due to nerve injury, venous or lymphatic congestion due to surgical trauma, or compression during surgery and immune-mediated edema; it may also be caused by prolonged or repeated intubation attempts or the presence of an endotracheal tube [1–7]. Airway edema secondary to hematoma formation, vocal cord dysfunction, allergy, or prolonged intubation attempts, or due to the presence of an endotracheal tube is known to present immediately following extubation of the trachea [7–9]. As upper airway edema secondary to venous/lymphatic congestion following neck dissection develops slowly, it is known to present 24 h after the operative period [1]. Although there was preoperative obstruction of the SVC in our patient, this did not contribute to the airway edema or obstruction, as evidenced by a normal airway at initial intubation. This lack of contribution to the edema may have been because, as evidenced by MRI findings and reliable anesthetic induction through the upper limb IV line, the SVC obstruction in our patient was partial. In our patient, airway edema and obstruction were observed in the immediate postsurgical period. This could have been attributable to intraoperative compression of the great vessels, as observed by the disappearance of both Sp_{O_2} and the arterial blood pressure trace from the upper limb and the obstructed flow of fluids through the upper limb IV line. These brief episodes probably were indicative of complete or near-complete obstruction of the great vessels. However, it is possible that partial obstruction could have gone unnoticed during surgery. Figures 1, 2, and 3 show that the obstruction to venous drainage was brought about by other factors, as well as by the obstruction of the arterial flow in the neck. This may have resulted in a state of persistent impedance of venous drainage from the neck, coupled with persistently lower levels of perfusion. We believe this to be the most important underlying mechanism of the early development of upper airway edema in our patient. Injury to or compression of the nerves supplying the airway could have been an additional factor. However, our patient did not have any vocal cord dysfunction, as evidenced during postoperative evaluation.

Prior neck surgery has been documented as predisposing to the development of immediate postoperative upper airway edema in patients undergoing nonsimul-

taneous bilateral neck dissection [10]. As radical neck dissection may involve resection of the internal jugular vein, spinal accessory nerve, or sternocleidomastoid muscle, it has been known to result in airway edema postoperatively. Airway edema has also been observed in patients with previous neck surgery who are undergoing unrelated surgery [11]. This has been attributed to failure of the lymphatic drainage to tolerate a further insult after it has been compromised by previous neck surgery and prior irradiation of the surgical area. However, to the best of our knowledge, acute airway edema following the excision of a nonlaryngopharyngeal tumor that is not invading vascular structures in the neck has not been described in the literature.

Significant airway edema may result in severe respiratory distress that can rapidly become fatal if not identified and treated early. Hence, preventing the development of airway edema is crucial and may be achieved by simple measures such as limiting IV fluid, keeping the head elevated during surgery as well as during the postoperative period, and avoiding acute flexion of the neck intraoperatively to avoid kinking and obstruction of cervical drainage [11]. However, despite these measures, a patient can develop significant airway edema due to the risk factors described earlier in this article. Therefore, it has been advised that a cuff-leak test be performed and the upper airway and vocal cords be scrutinized under direct laryngoscopy at the end of the surgical procedure prior to taking a decision on extubation. Most studies suggest that, in the absence of a peritubular leak, the likelihood of postextubation airway obstruction is increased in the adult patient [12–14]. However, this test will not completely reduce the risk of airway compromise, as clinically significant edema may not present immediately. Also, the presence of paradoxical vocal cord adduction will not be visible while an endotracheal tube is in place. Hence, routine extubation over a pediatric tube exchanger has been advised in such cases [8]. The use of a pediatric airway exchange catheter helps not only in oxygenating the patient in a scenario of significant post-extubation airway obstruction but also aids in railroading the endotracheal tube into the trachea under direct laryngoscopy even when the glottis is poorly visualized. Thus, the use of a pediatric airway exchange catheter can minimize airway trauma due to repeated intubation attempts and aid in quick airway control in an emergency situation of can't ventilate and can't visualize the glottis (can't intubate).

Based on our experience, we would suggest that intraoperative monitoring for possible obstruction of the great vessels during surgery in the neck or upper thorax is essential. This may help prevent prolonged unintentional compression of the great vessels by the surgeon. If repeated compression is observed, this could also serve as an early warning sign heralding the possibility of significant airway edema and thus help the anesthesiologist to decide regarding the method and timing of extubation.

We conclude that surgery around the great vessels in the neck is associated with the possibility of the development of immediate postoperative airway edema; we consider that intraoperative monitoring for possible obstruction of the great vessels could be a useful tool both for the surgeon, to minimize unintended compression of the great vessels, and for the anesthesiologist, to anticipate the possibility of significant airway edema.

References

1. Yoshihiro H, Yukihiro M, Takehiro K, Tetsuya T, Satoshi K. A case of postoperative laryngopharyngeal edema following bilateral neck dissection. Practica Otologica (Kyoto). 2002;95:957–61.
2. Roy S. Acute postoperative neck hematoma. Am J Emerg Med. 1999;17:308–9.
3. Penberthy A, Roberts N. Recurrent acute upper airway obstruction after anterior cervical fusion. Anaesth Intensive Care. 1998; 26:305–7.
4. Krnacik M, Heggeness M. Severe angioedema causing airway obstruction after anterior cervical surgery. Spine. 1997;22: 2188–90.
5. Bexton M, Radford R. An unusual cause of respiratory obstruction after thyroidectomy. Anaesthesia. 1982;37:596.
6. Tanaka A, Isono S, Ishikawa T, Sato J, Nishino T. Laryngeal resistance before and after minor surgery: endotracheal tube versus Laryngeal Mask Airway. Anesthesiology. 2003;99:252–8.
7. Burkle CM, Walsh MT, Harrison BA, Curry TB, Rose SH. Airway management after failure to intubate by direct laryngoscopy: outcomes in a large teaching hospital. Can J Anaesth. 2005;52:634–40.
8. Dosemeci L, Yilmaz M, Yegin A, Cengiz M, Ramazanoglu A. The routine use of pediatric airway exchange catheter after extubation of adult patients who have undergone maxillofacial or major neck surgery: a clinical observational study. Critical Care. 2004;8:R385–90.
9. Loevner LA. Anatomic and functional lesions resulting in partial or complete upper airway obstruction. Semin Roentgenol. 2001; 36:12–20.
10. Haraguchi HH, Hentona H, Ishikawa N, Sugimoto T, Tsunoda A, Tatsumi A, Komatsuzaki A. Three cases of postoperative laryngopharyngeal edema following nonsimultaneous bilateral radical neck dissection (in Japanese). Nippon Jibiinkoka Gakkai Kaiho. 1995;98:1903–8.
11. Burkle CM, Walsh MT, Pryor SG, Kasperbauer JL. Severe postextubation laryngeal obstruction: the role of prior neck dissection and radiation. Anesth Analg. 2006;102:322–5.
12. Kriner EJ, Shafazand S, Colice GL. The endotracheal tube cuff-leak test as a predictor for postextubation stridor. Respir Care. 2005;50:1632–8.
13. Miller RL, Cole RP. Association between reduced cuff leak volume and postextubation stridor. Chest. 1996;110:1035–40.
14. Fisher MM, Raper RF. The "cuff-leak" test for extubation. Anaesthesia. 1992;47:10–2.