Life-threatening upper airway obstruction associated with prior radiotherapy

Dear Editor,

A 40-year-old female was admitted to our hospital with the complaint of increased cough with mucopurulent expectoration, breathlessness, and fever for two days. Two years earlier, she had received radiation therapy for squamous cell carcinoma of left tonsil. General examination revealed temperature: 100°F, pulse rate: 110/minute, blood pressure: 110/70 mm Hg, respiratory rate: 26/minute, and oxygen saturation on room air was 80%. Respiratory system examination revealed crepitation in right basal region on auscultation. Laboratory investigation revealed hemoglobin: 10 gm/dl, total leukocyte count of 12,100/cm^3 with neutrophil predominance (72%). Other biochemical and serological parameters were within normal limits. Chest radiograph revealed consolidation in right lower lobe. She was managed with oxygen inhalation, broad-spectrum antibiotics, and other supportive care. Next day, she complained of increasing breathlessness associated with tachypnea and falling oxygen saturation despite oxygen supplementation. ABG showed type-I respiratory failure. Chest evaluation did not reveal any significant finding. Evaluation of laryngopharynx revealed minor changes to the airway consistent with prior radiation. She was intubated with an endotracheal tube of 7.0 mm inner diameter and placed on mechanical ventilation. Thirty-six hours after intubation and mechanical ventilation, she showed improvement in oxygenation and was maintaining oxygen saturation at minimal FiO^2 and PEEP. Therefore, she was placed on T-piece. While on T-piece, patient was comfortable and maintained oxygen saturation. Cuff leak test was also done, which was positive. Therefore, she was extubated after one hour of successful T-piece trial. One hour post-extubation, patient developed stridor and oxygen saturation dropped to 90%. She was treated with both hydrocortisone and epinephrine nebulization with some improvement in stridor. After two hours, patient developed severe respiratory distress (respiratory rate: 32/minute, use of accessory muscle, oxygen saturation: 92%). She was taken for re-intubation, but laryngoscopy showed significant laryngeal edema. During laryngoscopy, she began to desaturate to oxygen saturation of 62% and, therefore, emergency intubation was done with an endotracheal tube of 4.5 mm inner diameter followed by surgical tracheostomy. Twelve hours after tracheostomy, she was weaned off from the mechanical ventilator and maintained oxygen saturation at room air. On 3rd day, bronchoscopic evaluation showed significant edema of the epiglottis and aryepiglottic folds. Therefore, she was changed to 6.0 mm metal tracheostomy tube and discharged with her tracheostomy tube in place with tracheostomy care instructions. Follow-up evaluation after one month revealed near-resolution of her laryngeal edema.

Laryngeal edema is a common cause of airway obstruction in patients admitted to intensive care unit after extubation, but only some of them develop clinical symptoms.[1] As per literature, 1-4% of all extubated patients requiring re-intubation are due to laryngeal edema.[1] Patients with head and neck cancers are often treated with radiotherapy, and chronic laryngeal edema is one of the known complications of radiotherapy.[2] Besides edema, a neuronal imbalance of the abductor and adductors of the vocal cords also plays a role in airflow reduction.[3] In our patient, many factors could have contributed despite apparently normal initial airway. Radiation therapy alters tissue morphology resulting in diminished flexibility of native tissues.[4] In larynx, this diminished tolerance to brief insult (endotracheal intubation) produces edema and swelling, thereby placing such patient at increased risk for post-extubation airway compromise.

Dyspnea and stridor immediately post-extubation are obvious signs of laryngeal edema. There are other mechanisms, which are responsible for delayed onset of symptoms. Significant increase in laryngeal resistance to breathing as a result of marked laryngeal swelling after intubation[5] leads to generation of powerful negative airway pressure, which activates the vocal cord adductor and the development of stridor in these patients.[3] This suggests that both passive glottis narrowing due to edema and active narrowing during inspiration are the contributing factors (as in our patient). The presence of airway resistance by either cause may not be apparent clinically till airflow increases up to that level resulting in turbulence of airflow[6] (this may be the reason of positive cuff leak in our patient).

We suggest that close observation and emergency airway measures should be considered before extubation in patients with a prior history of radiotherapy to head and neck regions.

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