CASE REPORT

Extensive Tracheal Necrosis Associated with a Prestretched Tracheostomy Tube Cuff

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The use of cuffed tracheostomy tubes has exacted no small price in terms of damage to the patients' trachea.[1-6] Local ischemia and necrosis secondary to pressure from the cuff have been suggested as the principal causes of this damage.[7, 8] This belief has led to the introduction of cuffs designed to exert less pressure against the wall of the trachea while sealing it against air leaks during positive-pressure ventilation. Geffin and Pontopiddan [9] describe no "untoward effects" in more than two hundred patients on whom such plastic tubes with the cuffs prestretched under warm water with 20 to 30 ml of air for ten minutes, were used. The cuffs were allowed to cool while still inflated.

Following is a case report of a patient who developed mucosal ulceration, tracheal dilatation, and tracheoesophageal and tracheomediastinal fistulas at the site of a prestretched cuff prepared as recommended by Geffin and Pontopiddan.

REPORT OF A CASE

A 49-year-old woman was admitted with a history of weight loss, nausea, and vomiting. She was emaciated. She had a history of chronic alcoholism, chronic bronchitis, and malabsorption syndrome with secondary megaloblastic anemia. During treatment for the malabsorption syndrome and anemia, a lesion in the lower gum was noted, biopsied, and reported as infiltrating squamous cell carcinoma. A Commando procedure and tracheostomy were done using a Portex #42 tracheostomy tube with a built-in cuff prestretched as described above.

Postoperatively the patient was weak, with inadequate ventilatory function, and was unable to clear tracheobronchial secretions. Ventilation was assisted with a pressure-limited machine set at 30 cm H_2O airway pressure. She remained on the pressure-limited ventilator until the third postoperative day, when, as a result of intravenous therapy, she developed pulmonary edema. This was treated by intravenous furosamide, fluid restriction, and transfer to a volume-limited ventilator (Engström). Before the edema completely subsided, bilateral pneumonic infiltrates were seen on roentgenograms of the chest. These became more extensive with time. Bilateral pneumothorax subsequently developed and was treated by insertion of chest tubes and under water drainage. On the volume-limited ventilator on the seventh postoperative day the patient had peak airway pressures of 50–60 cm H_2O at $\dot{V}_E$ of 15 l/min. The diameter of the tracheostomy cuff as seen on roentgenograms at this time was 3 cm. Blood pressures were generally 80 to 90 systolic and 50 to 70 mm Hg diastolic postoperatively. On the twenty-second postoperative day a roentgenogram showed that the maximum diameter of the tracheostomy cuff was 5.2 cm. The tracheostomy cuff had been inflated only to the point of eliminating leakage during positive-pressure ventilation. The diameter of the tracheostomy tube as seen on roentgenograms was constant; therefore, measurements given above for the widths of the cuff are comparable.

On the 23rd postoperative day the patient's tracheostomy tube was changed as it had been every three to four days during her hospitalization. Later that day her abdomen became distended. The nasogastric tube, which had been in place intermittently since shortly after operation, was connected to suction, without improvement of the distention. It was then manipulated and finally replaced, but without effect. During the manipulation turbulence and gurgling could be felt and heard over the epigastrium, the sounds in phase with the ventilator. The tracheostomy tube was advanced as far as it would go, with some decrease of the gurgling. Early on the 27th postoperative day coffee-ground material was found in the tracheostomy tube, which was suctioned repeatedly. A Sengstaken-Blakemore tube was passed and the regurgitation stopped. Two hours later the patient was found cyanotic. The EKG tracing was flat, and the patient was pronounced dead.

Postmortem examination of the trachea revealed discoloration of tissue with mucosal ulceration, ex-

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posure of tracheal rings, and a fistula to the esophagus 2.6 cm below the lower limit of the stoma. This fistula measured 2 × 2 × 1 cm. Laterally adjacent to it was a tracheomediastinal fistula 3 × 2 × 1 cm. Both of these fistulas were at the level of the cuff. An anterior tracheomediastinal fistula 1 × 1 × 1 cm was present 5 cm below the lower limit of the stoma. It was probably secondary to pressure and trauma from the tip of the airway. Microscopic examination disclosed no neoplastic invasion of the affected areas.

Discussion

The virtues of low-pressure cuffs have been the subject of recent reports.[8, 10, 11] Their distinguishing characteristic is the low pressure they exert against the tracheal wall when inflated sufficiently to seal the airway during positive-pressure ventilation. They are easily reshaped to conform to the irregular interior surface of the trachea and have large contact areas. Changes in pressure within the cuff produce lateral wall pressures which are distributed over large areas of the trachea. By contrast, conventional high-pressure cuffs become nondeformable when small amounts of air are passed into them. Because they cannot as readily conform to the tracheal wall, they must be inflated to higher pressures until the trachea is stretched before the airway is sealed. Furthermore, they have small contact areas with the trachea and may inflate unevenly, exerting very high pressures on some parts of the tracheal wall.

Why should the patient presented here have suffered such disastrous trecheal necrosis despite the use of a prestretched, low-pressure cuff? It was pointed out by Adriani and Phillips[7] and later confirmed by Lumholt[10] that the lateral pressure exerted by a cuff against the tracheal wall must be equal to or greater than the peak airway pressure if the airway is to be sealed for positive-pressure ventilation. A low-pressure cuff will seal the trachea when inflated to a point such that the lateral pressure is close to the peak airway pressure. High-pressure cuffs, because of gaps left by their poor fit against the tracheal wall, must be inflated until they exert lateral pressures far higher than the peak airway pressure. In a patient such as ours increased airway impedance necessitated the use of high airway pressures. To effect a seal of the airway, the cuff had to be inflated to a point where high lateral pressures were exerted. This factor, combined with the low blood pressure, presence of a nasogastric tube, infection, and other suggested causes of tracheal necrosis and fistula formation, may have produced the unfortunate consequences which developed. Bain and Spoerel[4] first noted a causal relationship between high airway pressures, cuff inflation, and tracheal dilatation. It appears that the low-pressure modification in cuff design discussed here may not always protect patients from this chain of events. The advantages gained with the prestretched low-pressure cuff may be lost as higher airway pressures are needed.

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