Long-term Survival after Endobronchial Fire during Treatment of Severe Malignant Airway Obstruction with the Nd:YAG Laser

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We present a case of Nd:YAG laser treatment of nearly total airway obstruction by malignant tumor in which an endobronchial fire occurred. The patient survived without complications of the fire and was followed-up until death over 22 months after the fire. The events leading to the fire are presented and recommendations provided to prevent similar occurrences. (Chest 1988; 94:1066-88)

Laser phototherpay (LPT) utilizing the neodymium-yttrium-aluminum-garnet (Nd:YAG) laser has been shown to be an effective tool in the therapy of malignant endobronchial obstruction especially when combined with radiation therapy. While LPT is generally safe, there are potential complications which can increase morbidity and mortality. We present a case of endobronchial fire during LPT and report long-term follow-up after successful outcome with no residual damage resulting from the occurrence.

CASE REPORT

A 65-year-old woman with squamous cell carcinoma (left upper lobe resection in June, 1985) was admitted to another hospital in November, 1985 with dyspnea and chest pain. Physical examination revealed pulse rate of 88/min; respirations, 24/min; blood pressure, 164/94 mm Hg; inspiratory and expirary rhonchi and diffuse wheezes. The chest x-ray film showed elevation of the left hemidiaphragm with well expanded left lower lobe. Arterial blood gas determinations showed: pH, 7.37; Pao2, 49; Pco2, 46 (room air). Over nine days of therapy with bronchodilators and antibiotics, she developed stridor with Pco2 increasing to 55. She was transferred to our hospital.

Examination upon arrival showed a normal upper airway, labored respirations at 30/min, pulse rate of 126/min, and blood pressure, 126/90 mm Hg without pulsus paradoxus; arterial blood gas levels: Fio2, 0.45; pH, 7.58; Pao2, 56; Pco2, 47. Lung auscultation revealed coarse rhonchi and wheezes diffusely. Chest x-ray film findings were unchanged from above. She received intensive bronchodilator therapy; however, over seven hours her Pco2 increased and Pao2 declined. She was intubated and mechanically ventilated.

Fiberoptic bronchoscopic examination was performed 20 hours after admission (findings are given in Table 1, item 1). Thirty-six hours after admission, bradycardia, hypotension, and cardiac dysrhythmia developed. A pulmonary artery oximetry catheter showed: CVP, 4.0; PAP, 18/11; PCWP, 5.0; CQ, 4.3; SvO2, 75 percent. Her hemodynamic status remained unstable. Emergency LPT was done under general anesthesia using enflurane and Fentanyl 44 hours after admission (Fig 1A). The 9.0 mm endotracheal tube (ETT) cuff was distended with water. An Olympus BF 2TR flexible fiberoptic bronchoscope (FOB) and Nd:YAG laser

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Table 1—Nd:YAG Laser Treatment and Bronchoscopic Findings

<table>
<thead>
<tr>
<th>No.</th>
<th>Date</th>
<th>Pulses</th>
<th>Duration (s)</th>
<th>Energy (watts)</th>
<th>Power (joules)</th>
<th>FIO2</th>
<th>O2 Sat (oximeter)</th>
<th>Bronchoscopic Findings*</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>12-4-85</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>0.5</td>
<td></td>
<td>2 cm mass at level of carina with distortion. (RMB†) 5 mm (opening diameter); (LMB†) less than 1 mm</td>
</tr>
<tr>
<td>2</td>
<td>12-5-85</td>
<td>201</td>
<td>0.7</td>
<td>30-52</td>
<td>4763</td>
<td>0.5</td>
<td>93-97</td>
<td>Widespread carbonization of distal trachea and carina from endobronchial fire; RMB 5 mm, LMB 2 mm</td>
</tr>
<tr>
<td>3</td>
<td>12-5-85</td>
<td>65</td>
<td>0.7</td>
<td>15-22</td>
<td>768</td>
<td>0.5-1.0</td>
<td>90-93</td>
<td>Diffuse inflammation of the distal trachea and carina. RMB 3mm, LMB 0mm</td>
</tr>
<tr>
<td>4</td>
<td>12-7-85</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>0.5</td>
<td></td>
<td>2 cm mass at carina with distortion. Diffuse inflammation of the distal trachea and carina. RMB 3mm, LMB 0mm</td>
</tr>
<tr>
<td>5</td>
<td>12-10-85</td>
<td>371</td>
<td>0.7</td>
<td>15-35</td>
<td>7757</td>
<td>0.4</td>
<td>88-95</td>
<td>Widespread carbonization of distal trachea and carina from endobronchial fire; RMB 5 mm, LMB 2 mm</td>
</tr>
<tr>
<td>6</td>
<td>12-11-85</td>
<td>676</td>
<td>0.7</td>
<td>25-54</td>
<td>20060</td>
<td>0.4-0.6</td>
<td>90-96</td>
<td>Diffuse erythema of distal trachea and both mainstem bronchi. Slightly widened carina. RMB 5mm, LMB 6mm</td>
</tr>
<tr>
<td>7</td>
<td>12-12-85</td>
<td>452</td>
<td>0.7</td>
<td>25-50</td>
<td>12920</td>
<td>0.4</td>
<td>93-99</td>
<td>Posterior tracheal mass located 2 cm proximal to carina with less than 10% obstruction of tracheal lumen. No evidence of tumor in either mainstem. Rounded mass in the left lower lobe bronchus just distal to superior segment take-off with 90 + % obstruction of the lumen. RMB 5mm, LMB 6mm</td>
</tr>
<tr>
<td>8</td>
<td>1-15-86</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>0.35</td>
<td>93-99</td>
<td>Diffuse carbonization of distal trachea and carina from endobronchial fire; RMB 5 mm, LMB 2 mm</td>
</tr>
<tr>
<td>9</td>
<td>2-6-87</td>
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<td></td>
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<td></td>
<td></td>
<td></td>
<td>Diffuse carbonization of distal trachea and carina from endobronchial fire; RMB 5 mm, LMB 2 mm</td>
</tr>
</tbody>
</table>

*Measurements pertain to the end of each procedure.
†RMB and LMB = right main bronchus and left main bronchus, respectively.

System (Molecron 8000) were used. Both arterial oxygen saturation (Sao2) (Biox II ear oximeter) and mixed venous oxygen saturation (Svo2) were monitored. Her hemodynamic status became unstable and she required FIO2 0.5 to maintain Sao2 above 90 percent and Svo2 above 60 percent. Table 1, item 2 gives the laser settings and bronchoscopic findings. During LPT directed to the left main bronchus, the Teflon-coated laser fiber (TCF) tip was maintained 1 cm distal to the bronchoscope and 1 cm from the treatment site. After 201 pulses, the TCF was damaged and failed to function properly. Another TCF was not available. Since insufficient improvement in left main bronchus patency had been achieved, LPT was continued using an 0.6 mm quartz monofilament fiber without Teflon sheath (NTCF) (designed for intravascular procedures and lacking an air-feeding jacket). After 38 pulses were delivered uneventfully over 5-10 minutes, she became hypotensive and the Sao2 dropped below 90 percent. She was stabilized over 15 minutes. Administration of enflurane was stopped and the FIO2 increased to 1.0. The laser operator, unaware of the FIO2 of 1.0, resumed LPT. After 27 pulses over 4-5 min, microrinfection with flash fire and explosive sound occurred. The endobronchial portions of the FOB, NTFC, ETT, and anesthesia circuit caught fire immediately, and were withdrawn within seconds, disconnected from the anesthesia machine, and the gases were turned off. The vigorously burning ETT and anesthesia circuit were extinguished by stepping on them. The distal FOB insertion tube melted. The patient was initially mask ventilated, then reintubated. Her airways were inspected (Table 1, item 3). She was given methylprednisolone 40 mg IV, racemic epinephrine 0.3 ml by aerosol, and continued on mechanical ventilator in the ICU. Arterial blood gas levels 45 min later were: pH, 7.41; Po2, 63; Pco2, 48 (FIO2 0.5). A chest film showed total opacification of the left hemithorax. Emergency radiation therapy was begun. Her Karnofsky score was 10 percent.

Her clinical status remained critical and required FIO2 0.4 to 0.8 to maintain her Po2 above 60 mm Hg. Significant worsening of the airway narrowing had occurred by 48 hours after the fire. She required several therapeutic bronchoscopic procedures for alleviation of mucous plugging and three additional LPT sessions (Table 1, items 4-7) to establish full patency of both main bronchi. The chest film slowly showed improvement as her condition stabilized. She was weaned from ventilatory support, extubated on the 19th hospital day and discharged home 23 days after admission. Room air arterial blood gas levels at discharge were: pH, 7.45; Po2, 72; Pco2, 37.

By six weeks after the fire, she has received 4,500 rads of radiation therapy, her chest film has returned to baseline, and her Karnofsky score improved to 90 percent. The bronchoscopic appearance is summarized in Table 1, item 8 (Fig 1B).

Pulmonary function testing ten months after the fire showed: FVC, 2.25; FEV1, 1.50; Dco, 10.0; and KCO, 3.13 (respectively 79, 72, 47, and 67 percent of predicted).

She had a recurrence of tumor in February, 1987 unnamable to LPT (Table 1, item 9), and received additional radiation therapy to the mediastinum. Transient improvement was seen. She lived at home with her family, maintaining a Karnofsky score of 50-60 percent until September, 1987. She died with respiratory failure nearly 23 months after the endobronchial fire.

**Discussion**

Laser phototherapy has been shown to be effective with acceptable risks in the management of malignant and benign obstructing airway lesions. The concomitant use of radiation therapy after LPT for malignant lesions improves the quality of life and survival, which may exceed one year. At initial treatment, our patient had a Karnofsky score of 10 percent, indicating an extremely poor prognosis with a high likelihood
of early mortality. As a result of the endobronchial fire, there was initial worsening of the airway obstruction and her hospital stay was lengthened by a few days. Combined LPT and radiation therapy enabled her to survive nearly 23 months with improvement of her Karnofsky score and no long-term morbidity due to the fire.

Major complications occurring during LPT include bronchial perforation, hemorrhage, esophageal fistula, hypoxemia, anesthetic complications, endobronchial fire, and death. Careful case selection and meticulous technique can reduce these complications.

Patients with severe malignant airway obstruction frequently have significant underlying cardiac, pulmonary, or other diseases which can cause instability, significantly complicate their management, and require rapid therapeutic intervention. In these patients, intensive monitoring is necessary during LPT. Continuous communication among members of the operating team during LPT regarding changes in condition or therapy is imperative, since serious consequences can occur when these elements fail to operate effectively.

Several points deserve discussion concerning potential fire during LPT. First, several authors have suggested using a rigid metal bronchoscope (RB) during LPT since it contains no flammable materials. However, endobronchial fire is rarely seen during LPT utilizing the FOB. Some problems associated with the rigid bronchoscope include lack of familiarity in technique, potential damage to upper airway, limited access to lower airways, inability to use local anesthesia, difficulty in using inhalation agents, and frequently, prolonged recovery time after narcotic anesthesia. Second, TCF's employ high airflow to assist in local cooling of treated tissue, but NTCFs lack this feature. Thus, higher local tissue temperatures may occur during treatment with NTCFs and could increase the likelihood of fire. Third, laser fires may occur at any oxygen concentration. This risk increases as the oxygen concentration increases and becomes very high above FlO2 0.5. Not infrequently, brief self-limited flash fires occur after tissue has carbonized. Fortunately, extensive fire rarely results.

We reviewed the laser record in our case for possible causes of the fire. No differences in pulse frequency or total energy delivered were found by 10-second or 2-minute treatment intervals. We found a maximum of five pulses delivered per any 10-second interval, and only three pulses were delivered at the time of the fire. Thus, we concluded the most likely cause of the fire was the increased FlO2.

When complications occur, quick, decisive action is imperative. Our case illustrates that rapid removal of the burning materials can minimize trauma and morbidity. It should also be noted that the potential exists for larger disaster when vigorously burning materials are directly connected to anesthesia equipment and volatile gases.

We agree with the previously reported recommendations for LPT and suggest the following additional points:

1. There must be adequate pre- and intraoperative discussion between the laser therapist and anesthesia team to review procedures and safety precautions, with emphasis on the risk factors for fire. All changes in therapy must be reported immediately to all members of the team.

2. During LPT, the FlO2 should not exceed 0.5 and preferably remain less than 0.4. An oximetric device should be employed to continuously monitor oxygen saturation. If high FlO2 levels are necessary, use of the rigid bronchoscope may be desirable.

3. At least two functioning laser fibers should be available at any time, and the use of nonsheathed catheters should be discouraged.

4. If an endobronchial fire occurs, the FOB, laser fiber, and ETT should be removed from the patient as quickly as possible. The anesthesia circuit should be separated immediately from the anesthesia machine, and all gas flow turned off to reduce the chance for the fire to spread. The patient should be ventilated by mask with room air until the fire has been extinguished.

5. A fire extinguisher should be available in the room during LPT cases using FOB.

REFERENCES


Osler's Nodes on the Dorsum of the Foot*

Chatrchai Watanakunakorn, M.D., F.C.C.P.

Osler's nodes developed on the dorsum of the left foot of a patient with enterococcal endocarditis and on the dorsum of the right foot of another patient with Staphylococcus aureus endocarditis. The lesions were erythematous, raised, and very painful, but they resolved completely. There was no evidence of other embolic phenomena at the time the Osler's node appeared. (Chest 1988; 94:1088-90)

Osler's nodes are described as small, painful, nodular lesions usually found in the pads of fingers or toes and occasionally in the thenar eminence. I recently saw Osler's