ketoconazole, our experience, added to the conclusion of Wheat et al., does not fully support this initial optimism. Our four patients either had a relapse after more than one year of treatment (two cases) or were judged to be primary treatment failures (two cases). All of our patients were thought to have been compliant with their schedules for medication. None was receiving antacids or cimetidine or had undergone a gastrectomy, all of which are known to impair absorption of ketoconazole from the gastrointestinal tract. In addition, none of the patients was receiving glucocorticoids or had other congenital or acquired immunodeficiencies. While in vitro data have previously demonstrated that *H. capsulatum* is not only inhibited but killed by relatively small concentrations of ketoconazole, in vivo susceptibility or resistance to ketoconazole does not necessarily predict clinical response. Testing, therefore, remains problematic and was not pursued by our laboratories.

In view of this experience, we concur that once the diagnosis of chronic cavitary histoplasmosis has been established, oral treatment should be instituted with ketoconazole at 400 mg/day; however, careful follow-up evaluations, including chest roentgenograms and cultures of sputum when indicated, should be obtained at appropriate intervals during the course of therapy and especially at the end of treatment.

In the case of primary treatment failure, systemic therapy with amphotericin B is likely to be required, although suppression of recurrent disease might be obtained by extended treatment with ketoconazole. Preliminary prospective data using itraconazole have shown clinical effectiveness against histoplasmosis; additional data may show that this drug could supplant ketoconazole in treating chronic cavitary histoplasmosis, therefore obviating our concerns.

**REFERENCES**


**Nd-YAG Laser-Induced Endobronchial Burn**

**Management and Long-Term Follow-Up**

Steve Krautz, M.D.; Atul C. Mehta, M.D., F.C.C.P.; Herbert P. Wiedemann, M.D., F.C.C.P.; Glenn DeBoer, M.D.; Kenneth D. Schoepf, C.R.N.A.; and Marian Z. Tomaszewski, M.D.

Endobronchial fires are a rare complication of Nd-YAG laser photoresection. Short-term morbidity is secondary to sloughing mucosa and mucous plugging. Aggressive pulmonary hygiene, including frequent bronchoscopies and possibly a tracheostomy, may be required. The major long-term complication is obstruction of the airways from granulation tissue. Long-term follow-up is required to evaluate and treat clinically significant granulation tissue in the airways.

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Fires can produce pulmonary injury through different mechanisms, the most common being smoke inhalation; however, direct thermal injury to the endobronchial tree rarely occurs because of the ability of the upper airways to dissipate heat. The advent of laser therapy for airway lesions introduces a situation in which a direct thermal injury can more readily occur. A small number of case reports have described endobronchial ignition of combustible materials, such as the ET tube and the FOB, by both the Nd-YAG and carbon dioxide lasers. Even though much has been written about the prevention of such a complication and its immediate management, the current literature does not provide information on long-term management. We experienced an endobronchial fire during an Nd-YAG laser photoresection. This case not only illustrates our experience with the immediate and short-term management, but also describes our observations of the long-term sequelae.

**CASE REPORT**

A 65-year-old white man with a permanent tracheal stoma from a prior thyroidectomy and neck dissection for Hürthle cell carcinoma developed hemoptysis and dysphonia. An exophytic lesion from metastatic carcinoma produced a 90 percent subglottic tracheal obstruction, and laser photoresection was considered. Under general anesthesia, ventilation was delivered through a PVC ET tube wrapped with metal tape to the level of the cuff and inserted through the tracheostomy site. The lesion was approached transnasally with the FOB. The procedure was performed using contact as well as noncontact laser tips and biopsy forceps. A total of 6,600 joules was delivered with 417 pulses of 10 to 40 W and 0.4-second duration over two hours. Oxygenation was maintained with an FIO2 of 40 percent. Suddenly, black smoke began to arise from the ET tube while using a contact tip at 30 W and 0.4-second pulse. An endobronchial fire was suspected, and the FOB and ET tube were removed immediately. The tip of the ET tube was charred, and a perforation was noted immediately proximal to the balloon and distal to the metal tape wrapping. Ventilation was reestablished by...

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Laser-induced Endobronchial Burn (Krautz et al)
intubation with a new PVC ET tube. An examination of the airways revealed charred mucosa in the distal trachea and both main-stem bronchi, with extension to the RUL orifice and bronchus intermedius (Fig 1).

Our greatest concern during the period immediately after the burn was the development of airways obstruction. This can result from several mechanisms, including sloughing of the charred mucosa, mucosal edema, reactive bronchospasm, and loss of mucociliary clearance. In the recovery room, we administered humidified supplemental oxygen and initiated hydration with intravenous physiologic saline solution to maintain a euolemic state. Physiologic measures included acetylcysteine and metaproterenol aerosols, followed by chest physical therapy and postural drainage every four hours, intravenous aminophylline, methylprednisolone (1 mg/kg every six hours), and cefotaxime (1 g every six hours). The patient’s clinical course was smooth until the third postoperative day, when dyspnea and refractory hypoxia developed. Emergent flexible bronchoscopy was performed to remove several large mucous plugs occluding both main-stem bronchi. Grossly, these plugs were comprised of charred mucosa and tenacious mucoid secretions (Fig 2). Portions of the bronchial mucosa were still charred, with mucosal sloughing. On the seventh and ninth postoperative days, deterioration in the pulmonary status recurred, necessitating flexible bronchoscopy to clear airway plugs and secretions. The mucosa on the ninth postoperative day was still inflamed, but most of the charred mucosa had sloughed. The patient was discharged on the tenth postoperative day in a relatively asymptomatic status on a three-week course of prednisone and oral antibiotics.

A bronchoscopy six weeks after the injury showed regenerating mucosa at the carina and small polypoid lesions of granulation tissue at the orifice of the RUL. We were concerned that significant narrowing would develop in the endobronchial tree, especially at the RUL orifice. Bronchoscopic examinations were repeated at the fourth and seventh month following the incident. The RUL orifice was becoming moderately stenosed from granulation tissue; however, its three subsegments were patent and could be visualized without much difficulty. A bronchoscopy at one year showed the RUL stenosis to be stable and clinically insignificant (Fig 3).

**DISCUSSION**

This case emphasizes several major points regarding laser-induced endobronchial burns. Prevention is still of utmost importance. The smallest possible FIO\textsubscript{2} should be used to maintain adequate oxygenation. The risk of laser surgery may outweigh the benefits if an FIO\textsubscript{2} of greater than 50 percent is used. If possible, pulse energy should be less than 40 W for a noncontact tip and less than 20 W for a
contact tip. Heat should be allowed to dissipate with adequate time spacing between repeated pulses. Maximum possible distance should be maintained between the ET tube and the surgical field. Otherwise, the tube should be meticulously wrapped with tape. A red rubber tube may be less likely to ignite as compared to a PVC tube; however, this purported advantage is not well documented. Alternatively, a flexible metal ET tube (Norton's tube) can be used in selected cases. Of course, combustible anesthetic agents should not be used during laser photoressection.

If a fire should occur, the immediate intervention is removal of the combustible endobronchial materials including the ET, FOB, laser fiber, and suction catheter. This will minimize the thermal injury and help prevent smoke inhalation. The airways should be examined for the presence of remaining foreign materials and to assess the extent of the injury. A chest roentgenogram should be obtained to rule out the possibility of pneumothorax or pneumomediastinum. If significant smoke inhalation or extensive thermal injury involving peripheral airways occurs, then mechanical ventilation, including positive end-expiratory pressure, might be required.

Obstruction of the airways after a burn can produce significant morbidity. Flexible bronchoscopy can be important to aid in clearing mucous plugs. Our patient had a preexisting tracheostomy which facilitated removal of large mucous plugs. The decision to employ a tracheostomy for the management of airway complications needs to be individualized, based on the patient's clinical course. Aggressive pulmonary hygiene should be instituted with humidification, hydration, aerosols with mucolytic and B2-adrenergic agonist agents, and chest physical therapy. Steroids and empiric antibiotics were also instituted in our patient for prophylactic treatment of bronchospasm and infection, respectively. Clinical judgment needs to be exercised because empiric steroids and antibiotics are not recommended for injuries due to smoke inhalation. Although we cannot prove that our patient benefited from the steroids and antibiotics, these drugs did not produce any apparent adverse effects. The use of steroids, as well as of antibiotics under similar circumstances, has been recommended by others.

Little emphasis has been given to the long-term follow-up of laser-induced burns of the tracheobronchial tree. We believe that long-term care is required because endobronchial granulation tissue can produce airway compromise. Regular follow-up is necessary to assess the airways for obstruction using clinical and radiographic information. Pulmonary function testing and ventilation-perfusion lung scanning could be of great value in detecting significant upper airway narrowing. If there is a suspicion of a developing airway problem, then flexible bronchoscopy can be used as a diagnostic and therapeutic tool.

**References**


**Failure of Corticosteroid Therapy to Prevent Induction of Ventricular Tachycardia in Sarcoidosis**

Bernard Belhassen, M.D.; Amos Pines, M.D.; and Shlomo Laniado, M.D.

Programmed ventricular stimulation was performed in a patient with sarcoidosis who exhibited an episode of sustained ventricular tachycardia. Sustained ventricular tachyarrhythmias requiring cardioversion for termination were induced by double right ventricular apical extrastimuli during control, and treatment with disopyramide, quinidine, and fluocortolone. In contrast, only four repetitive ventricular complexes were induced during combined therapy with quinidine, mexiletine and amiodarone. While receiving the latter regimen, the patient has been asymptomatic during 28 months of follow-up.

* (Chest 1989; 95:918-20)

Ventricular tachyarrhythmias and sudden death are common in patients with cardiac sarcoidosis. In isolated case reports, a beneficial effect of corticosteroid therapy on ventricular ectopic activity or ventricular tachycardia was observed. It has been suggested that this beneficial effect could be due to the healing of cardiac sarcoid granulomas by corticosteroids. In the present report, we describe a patient with sarcoidosis and sustained ventricular tachycardia in whom the effects of corticosteroid treatment and multiple antiarrhythmic drugs were tested by programmed ventricular stimulation. To the best of our knowledge, this is the first report evaluating the effects of corticosteroid therapy on ventricular tachycardia inducible by programmed ventricular stimulation in a patient with sarcoidosis.

**Case Report**

A 44-year-old white man was referred from another hospital for electrophysiologic study after he experienced a syncopal episode of sustained ventricular tachycardia that required cardioversion for termination. Two years earlier, the patient was diagnosed as suffering from sarcoidosis based on the presence of bilateral hilar adenopathy, histologic confirmation by fiberoptic bronchoscopy, and positive Kveim test. During the year prior to the present hospitalization, symptomatic ventricular arrhythmia consisting of multiple ventric-