Short reports

Tracheobronchial amyloidosis: treatment by carbon dioxide laser photoresection

RAPHAEL BREUER, GEORGE T SIMPSON, ALAN RUBINOW, MARTHA SKINNER, ALAN S COHEN

From the Pulmonary and Arthritis Centers; the Thorndike Memorial Laboratory, Boston City Hospital; and the Departments of Medicine and Otolaryngology, Boston City Hospital and University Hospital, Boston University School of Medicine, Boston, Massachusetts, USA

Amyloidosis localised to the lower respiratory tract occurs in three forms: diffuse interstitial deposits, single or multiple pulmonary nodules, and most commonly tracheobronchial deposits.¹ Cough, dyspnoea, wheezing, haemoptysis, and recurrent pneumonia are the common presenting symptoms of patients with localised tracheobronchial amyloidosis.¹⁻³ Localised bronchial deposits are usually found in the large lobar or segmental airways and they project into the lumen as rounded, smooth, greyish white sessile tumours. Intermittent bronchoscopic resection is the usual mode of treatment for obstructing endobronchial amyloidosis,¹⁻⁵ although there have been isolated reports of treatment by pneumonectomy⁶ and radiotherapy.³ Laser photoresection vaporises or coagulates living tissue to a certain preselected depth. We here report two patients with tracheobronchial amyloidosis successfully treated by carbon dioxide laser photoresection.

Case reports

PATIENT 1

A 40 year old woman presented in March 1982 with shortness of breath and "persistent asthmatic attacks" unresponsive to theophylline and sympathomimetic bronchodilators. On examination she was in mild respiratory distress, breathing at a rate of 23 per minute. Diffuse inspiratory and expiratory wheezes and a prolonged expiratory phase were heard over both lungs. A chest radiograph showed streaky left lower lobe atelectasis and left hilar prominence. Pulmonary function tests showed a forced vital capacity (FVC) of 2.2 l (64% predicted), a one second forced expiratory volume of 1.63 l (61% predicted and 74% of FVC), normal static lung volumes, and normal single breath carbon monoxide diffusing capacity. Arterial blood gas measurements during the breathing of room air showed a pH of 7.42, an oxygen tension (Pao₂) of 85 mm Hg, and a carbon dioxide tension (Paco₂) of 39 mm Hg. Flexible bronchoscopy showed widespread yellow lesions in the trachea and along both mainstem bronchi. Almost complete obstruction of the left mainstem bronchus and considerably less narrowing in the right mainstem bronchus were observed. A biopsy specimen from one of these lesions was stained with Congo red and showed the typical apple green birefringence of amyloid when viewed by polarised microscopy. The results of urine analysis and the

serum chemistry were normal. An abdominal fat aspirate showed no evidence of systemic amyloid deposition.

Under general anaesthesia a 20-50 W carbon dioxide laser was used to perform ablation through a Wolfe 7 × 400 mm rigid ventilation bronchoscope. Safety precautions included use of moist cotton eyepads secured with tape, wet cotton towels covering the skin of the face, saline inflated bronchoscopic cuffs, muscular paralysis to prevent inadvertent movement by the patient, and eye glasses or protective goggles for all operating room personnel. The first procedure, performed in May 1982, ablated the obstructing amyloid at the distal trachea and the orifice of the left mainstem bronchus. No bleeding occurred perioperatively. One day later the patient developed respiratory distress and stridor with subglottic edema that were relieved by temporary tracheostomy. Subsequently the patient felt less short of breath during rest and exercise, with considerably less wheezing. Follow up chest radiography showed clearing of the left lower lobe atelectasis and repeat spirometric tests showed the FVC to be 3.39 l (98% of predicted) and the FEV₁ 2.07 l (73% of predicted).

Two additional carbon dioxide laser procedures were performed at intervals of three and five months for ablation of left lower lobe and right mainstem lesions. These were performed after complaints of worsening exercise tolerance and increased wheeze without appreciable changes in the chest radiograph. After both treatments exercise tolerance and wheezing were improved. None of the procedures was complicated by haemorrhage, but they did necessitate vigorous drainage of mucoid secretions.

PATIENT 2

A 47 year old man presented in May 1980 with a four month history of increasing dyspnoea. He had a history of heavy smoking and a few episodes of mild haemoptysis. Two years earlier he had had right upper lobe pneumonia, which had resolved. Physical examination showed nothing abnormal except for expiratory wheezes localised to the posterior right lung fields. A chest radiograph was normal. Spirometric tests showed a moderate obstructive defect, FVC being 4.01 l (81% of predicted) and FEV₁ 2.05 l (56% of predicted and 51% of FVC). The flow-volume loop was characteristic of upper airway obstruction. Flexible bronchoscopy showed an endotracheal nodule 1 cm below the vocal cords, causing approximately 50% narrowing. Multiple distal tracheal nodules were also seen. The right mainstem bronchus was indented and narrowed by an extrinsic compression and an exophytic anterior nodule. The right upper lobe bronchus was narrowed with a pale yellow low nodule at its orifice. A biopsy specimen

Address for reprint requests: Dr Raphael Breuer, Pulmonary Unit, Kiryat Hadassah, PO Box 12000, Jerusalem, Israel.

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obtained from the tracheal nodules was diagnostic of amyloid, showing green birefringence on polarisation microscopy after being stained with Congo red. The results of routine blood tests and liver and spleen scans were normal; an abdominal fat pad aspirate was negative for amyloid.

In September 1980, carbon dioxide laser ablation was performed on the subglottic area under general anaesthesia, the techniques described above being used. There were no postoperative complications and the estimated blood loss was 150 ml. In April 1982 the patient returned because of recurrent pneumonia and a right lung infiltrate, which had been present on the chest radiograph for the previous four months. Diagnostic bronchoscopy showed no recurrence of the subglottic amyloid. Carbon dioxide laser ablation was performed on the lower tracheal deposits. The estimated blood loss was 150 ml. After the procedure a chest radiograph showed clearing of the right lung infiltrate and the patient reported improvement in exercise tolerance. Five months later he complained of increasing shortness of breath, and a third carbon dioxide laser ablation was performed on amyloid nodules on the left side of the main carina and in the right mainstem bronchus; no recurrence of the previously ablated nodules was noted. Follow up evaluation over 18 months showed no change in symptoms or chest radiograph.

Discussion

Two patients with symptomatic localised tracheobronchial amyloid deposits were treated by repeated carbon dioxide laser ablations. Both the patients had three treatments, covering one and a half and three years respectively. The six excisions relieved symptoms and improved radiographic abnormalities and pulmonary function. The procedures were tolerated well, and there was no postoperative bleeding. Excessive mucoid secretions requiring aggressive treatment followed all procedures. Temporary tracheostomy was necessary on one occasion to treat subglottic oedema, which occurred one day after operation.

Repeated procedures were necessary for ablation of

amyloid deposits that were producing symptoms in our patients but we found no recurrence of amyloid in previously treated areas. Dumon *et al*⁷ and Toty *et al*⁸ made brief reference to ablation of airway amyloid deposits by means of 4 Nd-YAG lasers but they did not report their localisation or give follow up data. To judge by our initial experience in the treatment of tracheobronchial amyloidosis by carbon dioxide laser photoresection, further attempts to treat this rare and disabling disease by this means would appear to be justified.

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