spontaneous breathing as soon as possible[2] and low-pressure suction of the chest tubes after operation are critical.[2, 3] Some ventilatory support is necessary, however, when a patient continues to have hypoventilation after operation. Pressure-controlled ventilation rather than volume-controlled ventilation has been recommended for such patients because it can deliver high inspired gas flows in the presence of airway leaks.[3] Although NPV is generally less effective than PPV,[1] it places less stress on the airway. Even if a peak inspiratory pressure inside the cuirass respirator is highly negative, a fraction of the pressure is transmitted to pleural pressure in patients with chronic obstructive pulmonary disease during cuirass ventilation.[4] Marino and Pitchumoni[4] showed that esophageal pressure decreased from −5.4 cm H_2O during spontaneous breathing to −7.3 cm H_2O during inspiratory cycle of NPV while an inspiratory peak pressure of −40 cm H_2O inside the cuirass worked on the chest. In contrast, during PPV a high positive peak pressure is transmitted to the airway and lung. NPV may therefore be less likely than PPV to induce barotrauma in patients with fragile stapling lines of the lung. Indeed, the decrease in P_{CO_2} in our patient may have been rather slow compared with PPV; however, NPV successfully led him to stable spontaneous breathing without increasing air leakage. Although our patient died, we believe that NPV with the cuirass respirator could be an appropriate ventilatory support for the patient with hypercarbia after operation for emphysema when the lung is adequately inflated.

REFERENCES

1. Chihara K, Kawarasaki S, Hitomi S, Shimizu Y. A new cuirass respirator synchronizing with spontaneous respiration and its clinical application [Abstract]. Chest 1988;94:44S.
2. Cooper JD, Trulock EP, Triantafillou AN, et al. Bilateral pneumenectomy (volume reduction) for chronic obstructive pulmonary disease. J Thorac Cardiovasc Surg 1995;109:106-19.
3. Barker S, Clarke C, Trivedi N, Hyatt J, Fynes M, Roessler P. Anesthesia for thoracoscopic laser ablation of bullous emphysema. Anesthesiology 1993;78:44-50.
4. Marino WD, Pitchumoni CS. Reversal of negative pressure ventilation–induced lower esophageal sphincter dysfunction with metoclopramide. Am J Gastroenterol 1992;87:190-4.

MEDIASTINAL HISTOPLASMOSIS CAUSING MASSIVE HEMATEMESIS

Margarita T. Camacho, MD,[a] Morris Edelman, MB, BCh,[b] Alla Rozenblit, MD,[c] John C. McKitrick, PhD,[b] Kenneth Pinsker, MD,[d] and Stanley C. Fell, MD,[a] *Bronx, N.Y.*

The granulomatous lymphadenitis that occasionally follows pulmonary infection with *Histoplasma capsulatum* (HC) may lead to the development of mediastinal granuloma or may progress to diffuse mediastinal fibrosis. In some cases, the superior vena cava, tracheobronchial tree, pulmonary vasculature, or the esophagus is compressed or entrapped, with resultant compromise in function.[1] Recurrent massive hematemesis from an esophageal ulcer caused by HC mediastinal granuloma has not previously been reported.

A 27-year-old, previously healthy Ecuadorian man was admitted after an episode of hematemesis. Ten days before admission, he reported pleuritic chest pain, cough, and fever, for which antibiotics were prescribed. Physical examination revealed a hemodynamically stable man with a hematocrit of 40%. Results of chest radiography and routine laboratory studies were within normal limits, tuberculin test results were positive, and results of human immunodeficiency virus testing were subsequently reported as negative. Esophagogastroduodenoscopy revealed a 5 mm ulcer on the right anterolateral wall of the esophagus 30 cm from the incisors. The biopsy sample from the ulcer was described as granulation tissue with no growth on culture. The esophageal lumen was not narrowed, and the stomach and duodenum appeared unremarkable. Computed tomography of the chest demonstrated a 3 × 4 cm subcarinal mass inseparable from the midthoracic esophagus and deformity of the medial wall of the bronchus intermedius (Fig. 1). During the ensuing 10 days during which further diagnostic studies were performed, the patient had four episodes of hematemesis, necessitating transfusions of 8 units of blood; on one occasion, the patient's hematocrit fell to 20%. A barium esophogram was unremarkable. Flexible bronchoscopy revealed elevation and edema of the floor of the right

From the Departments of Cardiothoracic Surgery,[a] Pathology,[b] Radiology,[c] and Medicine,[d] Montefiore Medical Center, Bronx, N.Y.

Received for publication Sept. 12, 1995; accepted for publication Sept. 19, 1995.

J Thorac Cardiovasc Surg 1996;111:1283-6

0022-5223/96 $5.00 + 0 **12/54/69339**

The Journal of Thoracic and
Cardiovascular Surgery
June 1996

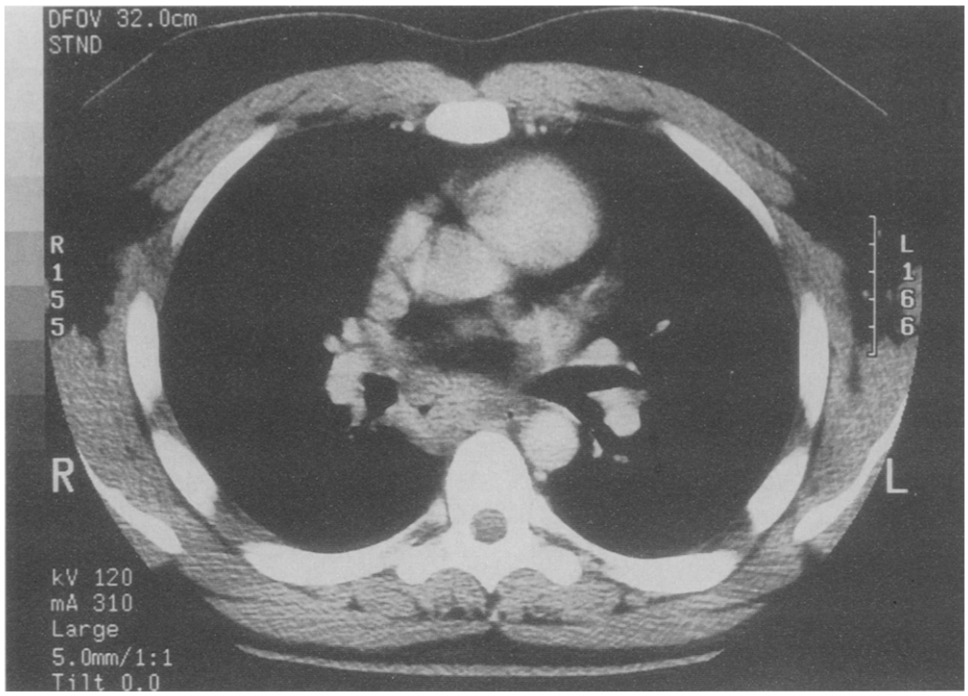

Fig. 1. Contrast-enhanced computed tomography demonstrates a 3 × 4 cm subcarinal mass, marked narrowing of esophageal lumen, and deformity of medial wall of bronchus intermedius.

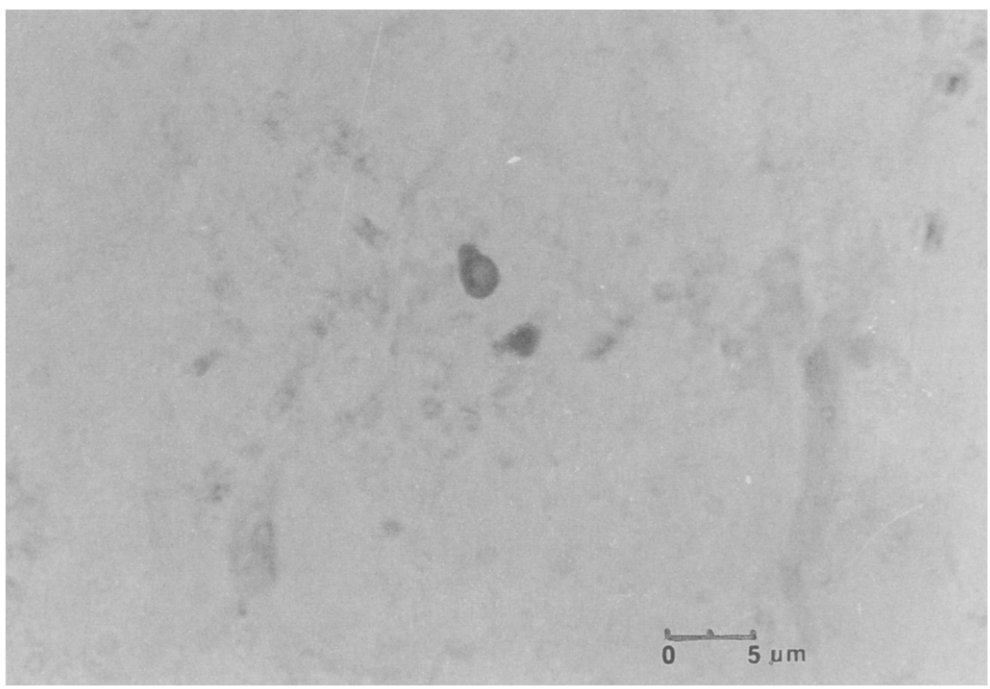

Fig. 2. Budding yeast-form HC (Gomori methenamine silver; original magnification ×1000).

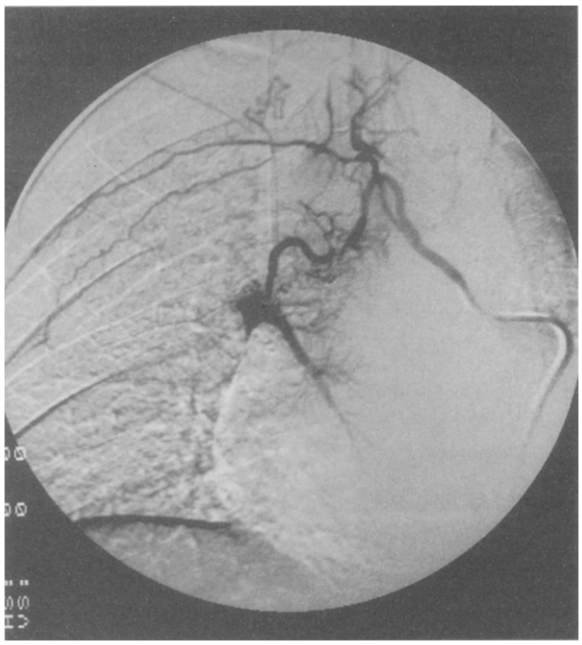

Fig. 3. Right bronchial arteriogram demonstrates a zone of hypervascularity corresponding to the subcarinal mass.

main bronchus. Bronchial brushings were nondiagnostic. Wang needle aspiration of the subcarinal mass yielded budding yeast forms characteristic of HC (Fig. 2). Treatment with 200 mg itraconazole twice daily was commenced. In an effort to occlude the upper esophageal arteries, bronchial arteriography was performed (Fig. 3). Two right bronchial arteries were demonstrated, one of which was successfully embolized. The other bronchial artery perfused a zone of hypervascularity corresponding with the mass noted on the computed tomographic scan. Hematemesis recurred after the incomplete bronchial artery embolization, and right thoracotomy was performed. A ligneous mass was noted posterior to the right main stem bronchus, extending inferiorly to the level of the lower lobe bronchus. The mass was densely adherent to the bronchus and the esophagus. Half the circumference of the esophagus was decompressed by partial excision of the nodal mass. Histopathologic study of this tissue showed acute and chronic inflammation as well as focal nonnecrotizing granulomata. Silver staining demonstrated rare budding yeast forms approximately 2 μm in diameter with the morphologic appearance of HC. Bacterial and fungal cultures remained sterile. Urine was tested twice for HC antigen, with negative results. Massive hematemesis recurred on the fifth postoperative day; 5 L blood was transfused before reoperation, at which time the esophagus was further separated from the nodal remnant, creating a mural defect at the site of ulceration. Brisk arterial bleeding from the node was controlled by suture ligatures, and the esophageal defect was repaired. The postoperative course was uneventful, and bleeding has not recurred in the year since discharge. Itraconizol therapy was continued for that year, with radiologic evidence of resolution of the mediastinal granuloma.

HC is a ubiquitous fungus present in soil and endemic in several areas of the United States, where as many as 80% of inhabitants demonstrate positive skin test reactions.[1] Infection with HC is usually acquired by inhalation and may produce no significant symptoms. The few thoracic complications that may develop usually follow asymptomatic infection in an immunocompetent host or opportunistic infection in an immunocompromised host. Accordingly, the disease course may vary from a relatively insignificant respiratory infection to a lethal, disseminated illness. It is often difficult to establish the diagnosis of thoracic infection with HC. Culture results may be negative; sensitivity of the urine antigen test is 25% to 39% in limited disease and 10% to 21% in chronic pulmonary disease.[2] Complement fixation test results are positive in only 39% of cases.[3] Right paratracheal, subcarinal, and tracheobronchial adenopathy, best demonstrated by computed tomographic scan, are commonly noted mediastinal manifestations. The development of HC mediastinal granuloma has been reported in 5% to 15% of cases, with symptoms developing in 60% of these patients.[1] Esophageal involvement by mediastinal HC is uncommon and has been reported in 5% to 13% of cases.[4] Esophageal involvement has been reported in cases of disseminated HC, in which oral or intestinal ulcerations also occur, however, and esophageal bleeding has been reported in acquired immunodeficiency syndrome and disseminated HC.[5] Mediastinal fibrosis does develop in 34% of patients who have had mediastinal granuloma. On occasion, the diagnosis has been established by mediastinoscopy when paratracheal adenopathy is present.[4]

The previously reported cases of esophageal involvement describe obstruction, bronchoesophageal fistula, traction diverticulum, and esophageal erosion and ulceration.[3, 4] Esophageal bleeding caused by an eroding mediastinal granuloma has not been previously described. The cause of the bleeding was a hypervascularized nodal mass that eroded into the esophagus. Transbronchial Wang needle aspiration demonstrated HC in this case. This technique may obviate the need for thoracotomy to establish the diagnosis when mediastinal granuloma caused by HC is suspected. Had the bronchial artery embolization been successful in occluding the bleeding source, it is quite likely that treatment with antifungal agents might have obviated the need for operation. A case of tracheal esophageal fistula caused by mediastinal HC granuloma and diagnosed by mediastinoscopy was successfully treated in this fashion.[4]

REFERENCES

1. Garrett HE, Roper CL. Surgical intervention in histoplasmosis. Ann Thorac Surg 1986;42:711-22.
2. Wheat LJ, Kohler RB, Tewari RP. Diagnosis of dissemi-

The Journal of Thoracic and Cardiovascular Surgery
June 1996

nated histoplasmosis by detection of *Histoplasm capsulatum* antigen in serum and urine specimens. N Engl J Med 1986;314:83-8.
3. Dines DE, Payne WS, Bernatz PE, Pairolero PC. Mediastinal granuloma and fibrosing mediastinitis. Chest 1979;75:320-4.
4. Coss KC, Wheat LJ, Conces DJ, Brashear RE, Hull MT. Esophageal fistula complicating mediastinal histoplasmosis. Am J Med 1987;83:343-6.
5. Forsmark CE, Wilcox CM, Darragh TM, et al. Disseminated histoplasmosis in AIDS: an unusual case of esophageal involvement and gastrointestinal bleeding. Gastrointest Endosc 1990;36:604-5.

UPPER AIRWAY OBSTRUCTION CAUSED BY LOW-GRADE TRACHEAL PAPILLARY ADENOCARCINOMA: AN UNUSUAL FLOW-VOLUME LOOP PATTERN

David E. Clarke, MD,[a] Ronald J. Green, MD,[a] James B. D. Mark, MD,[b] Robert C. Robbins, MD,[b] and Thomas A. Raffin, MD,[a] *Stanford, Calif.*

Tumors obstructing the upper airway often masquerade as asthma before definitive diagnosis.[1] As initially described by Miller and Hyatt,[2] flow-volume loops in patients with these obstructing lesions demonstrate characteristic patterns, depending on tumor location (extrathoracic or intrathoracic) and type (fixed or variable obstruction). We describe the case of a woman admitted for control of asthma who was found to have an unusual flow-volume loop pattern. Her flow-volume loop was different from previously described patterns of intrathoracic upper airway obstruction. Recognition of the different flow-volume loop patterns produced by upper airway obstruction is essential for early diagnosis and treatment of this potentially life-threatening problem.

Case report. A 60-year-old woman was brought to Stanford University Hospital with shortness of breath. She described paroxysms of dyspnea, often precipitated by coughing, that had occurred during the previous 4 to 5 months. In most instances, the shortness of breath lasted only a few minutes. The patient described the sensation as, "A cap went over my windpipe." She had sought medical advice from many different physicians, most of whom said she had asthma, and she had been using an albuterol inhaler intermittently for a 4-month period. Two weeks before admission, she was referred to a pulmonologist, who concurred with the diagnosis of asthma and prednisone was prescribed at a dosage of 60 mg per day. On the day of admission, she had a severe episode of shortness of breath and was brought to the emergency department by ambulance. She was a lifelong nonsmoker. She had drunk significant quantities of alcohol in her youth but none since.

From the Divisions of Pulmonary and Critical Care Medicine[a] and the Division of Thoracic Surgery,[b] Stanford University School of Medicine, Stanford, Calif.

Received for publication July 21, 1995; accepted for publication August 28, 1995.

J Thorac Cardiovasc Surg 1996;111:1286-8

0022-5223/96 $5.00 + 0 **12/54/68918**

At admission physical examination, the patient was breathing comfortably after a nebulized albuterol treatment. Results of head and neck examination were normal. Her breath sounds were clear. With a forced vital capacity maneuver, the patient could exhale for only a brief period before all air movement ceased. She had no difficulty, however, with quiet breathing. She had a grade II/VI systolic ejection murmur, and the remainder of her examination was unremarkable. An arterial blood gas analysis done on admission to the emergency department revealed a pH of 7.39, carbon dioxide tension of 49 mm Hg, and an oxygen tension of 98 mm Hg with the patient breathing 60% oxygen by face mask. One hour later, while she was breathing room air, her oxygen saturation was 98%. She had a leukocyte count of 20,100 cells/mm^3, with 82 polymorphonuclear leukocytes, 8 band cells, and 10 lymphocytes. Lactate dehydrogenase level was 792 IU/L (normal 313 to 618 IU/L); the remainder of her complete blood cell count and chemistry panel results were normal. A chest radiograph was normal.

The patient was admitted to the hospital and given nebulized albuterol and prednisone. Pulmonary function tests were performed. The flow-volume loop was remarkable, demonstrating an immediate cutoff of forced expiratory flow (Fig. 1, *A*). The expiratory loop was reminiscent of extremely severe emphysema, except that pulmonary function tests revealed a normal slow vital capacity. The differential diagnosis included upper airway obstruction and vocal cord dysfunction mimicking asthma. Results of otolaryngologic evaluation by fiberoptic laryngoscopy to the level of the vocal cords were normal. At fiberoptic bronchoscopy, a large mass was observed at the main carina. This mass appeared to be attached to the trachea by a stalk, and it prolapsed in and out of the left main stem bronchus during respiratory movements. The next day, because the mass nearly occluded the trachea, it was severed with a laser and removed by means of rigid bronchoscopy. The patient tolerated the procedure well, and her condition was markedly improved after she awakened from anesthesia. Pathologic examination of the mass revealed it to be a low-grade papillary adenocarcinoma with negative