tients. It is likely that other factors present in these patients contributed to the development of the bradyarrhythmia. Mildly elevated potassium levels might have lowered the transmembrane action potential in the SA node and/or perinodal atrial tissues which would have potentiated the effects of procainamide. Also, clonidine, which one patient was receiving, and/or digitalis, may have had contributory effects. Clonidine and digitalis, even at the normal blood levels found in these patients, by their autonomic effects, can by themselves produce findings similar to those found in this report. That procainamide was a necessary factor was demonstrated by the observation that following cessation of procainamide, when procainamide levels were no longer elevated, normal sinus rhythm returned. Also, when one patient was rechallenged with clonidine for control of blood pressure at doses considerably higher than those administered at time of cardiotoxicity, and when both patients were given digitalis for congestive heart failure, neither patient developed recurrence of bradyarrhythmia. The possibility that these patients had latent sinus node dysfunction which became manifest after procainamide therapy must also be considered.

Another possible cause for the observed procainamide toxicity is elevation in N-acetyl procainamide, the metabolite of procainamide with similar antiarrhythmic actions. Since marked elevations of this substance have been reported in patients with renal failure who rapidly acetylate procainamide, it is possible that N-acetyl procainamide might have been a factor in one or both patients. Thus, in patients with chronic renal failure, procainamide can produce cardiac toxicity even when reduced doses are administered. Procainamide should therefore be avoided in these patients or used cautiously. Early signs of sinus node dysfunction should be looked for especially when other factors are present that might potentiate the effects of procainamide, since manifestation of "sick sinus syndrome" might be observed even before the QRS complex or the QT interval is appreciably altered.

REFERENCES

2 Helfant RH, Scherlag BJ, Damato AN: Use of diphenylhydantoin sodium to dissociate the effects of procainamide on automaticity and conduction in the normal and arrhythmic heart. Am J Cardiol 20:820-825, 1967

Esophageal Compression in Association with Silicosis and Mycobacterium Intracellulare*

Rosemary F. Rodgers, M.D., F.C.C.P.;**
Michael I. Appelbaum,t and Raul M. Heredia, M.D.;

An enameler with dysphagia was found to have extrinsic compression of the esophagus by enlarged mediastinal lymph nodes. Scalene lymph node biopsy revealed silicosis, and tissue cultures grew Mycobacterium intracellulare. We believe our patient is the first reported to have dysphagia due to silicotic adenopathy complicated by an atypical mycobacteriosis.

Dysphagia due to extrinsic compression of the esophagus by enlarged lymph nodes occurs infrequently in association with granulomatous diseases. Sarcoidosis and mediastinal granuloma have been reported to cause extrinsic esophageal compression.1-3 Silicosis has previously been shown to cause esophageal diverticulum, and when complicated by Mycobacterium tuberculosis, direct involvement of the esophageal wall.4-6

The relationship between silicosis and tuberculosis is well known. Also, mycobacteria such as M. kansasi and M. intracellulare often occur in association with pneumoconiosis.5-6 We report a patient with dysphagia due to extrinsic compression of the esophagus by silicot in mediastinal adenopathy complicated by M. intracellulare.

CASE REPORT

A 44-year-old black man was seen in consultation by the medical chest service for a two-month history of dysphagia for solid foods and cough productive of yellow sputum. He denied recent weight loss, dyspnea, wheeze, chest pain, hemoptysis, use of tobacco, and previous tuberculosis. For the past 15 years, he had worked at an enamel factory where he was exposed to silica dust. He had past history of hypertension and diabetes mellitus. Physical examination showed a well-developed, 100 kg man with temperature 37.2°C, pulse rate of 84 beats per minute, and blood pressure of 140/100 mm Hg. The only abnormal physical finding was a small lymph node palpable in the left axilla.

Laboratory investigation showed the following: hemoglobin level, 9.1 gm/100 ml; hematocrit reading, 31.7 percent; white blood cell count, 4,200/cu mm, with one metamyelocyte, nine bands, 38 segmented neutrophils, three eosinophils, one basophil, 44 lymphocytes, and four monocytes; erythrocyte sedimentation rate, 90 mm per hour; serum iron value, 20 μg/100 ml (60 to 150); iron binding capacity, 222 μg/100 ml (270 to 380); and fasting blood glucose value, 159 mg/100 ml. The chest roentgenogram showed bilateral pulmonary reticulonodular shadows, bilateral hilar adenopathy, and cardiomegaly (Fig 1).

From the Departments of Medicine and Pathology, Mount Sinai Hospital Medical Center and Rush Medical College, Chicago.
**Instructor in Medicine, Rush Medical College.
†Senior Medical Student, University of Illinois Medical School.
‡Assistant Professor of Pathology, Rush Medical College. Reprint requests: Dr. Rodgers, 701 South Arlington Hts Road, Arlington Hts, Illinois 60005

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An esophagram showed extrinsic compression of the middle third of the esophagus by adjacent mediastinal and subcarinal lymph nodes (Fig 2). Bone marrow examination revealed changes consistent with anemia of chronic disorders. Intermediate strength purified protein derivative (5 tuberculin units) was negative, but the patient was not anergic to intradermal tests with Candida albicans, mumps, and streptokinase-streptodornase. Pulmonary function tests showed mild obstructive changes and moderately impaired single breath diffusing capacity.

Right scalene lymph node biopsy was performed by the surgical service; two lymph nodes, black in color, were removed. Histologically these nodes showed hyalinized granuloma (Fig 3). Silica crystals were seen under polarized light. Subsequently, cultures of this tissue grew numerous colonies of M. intracellulare.

Our diagnoses in this patient were as follows: esophageal compression by mediastinal lymphadenopathy, silicosis, infection with M. intracellulare, anemia of chronic disorders, diabetes mellitus, and hypertension. The medical chest service recommended that the patient undergo a thoracotomy to remove the lymph nodes that compressed the esophagus. The patient elected to go home without further treatment. His symptoms reportedly have improved, but he has not returned for follow-up evaluation.

**DISCUSSION**

Our patient had dysphagia due to extrinsic compression of the esophagus by silicotic mediastinal lymphadenopathy complicated by infection with M. intracellularare. We assume the tissue obtained from scalene lymph node biopsy was also representative of the mediastinal lymph nodes. Although direct involvement of this patient's esophageal mucosa by silicosis or M. intracellularare cannot be ruled out because esophagoscopy was not done, the esophagram showed no evidence of mucosal abnormality, and adjacent, large lymph nodes could be well seen to compress his esophagus. There are two previous case reports in which esophageal involvement
occurred in association with probable pneumonoconiosis: Carasso et al\(^1\) reported a 48-year-old woman with a bronchoesophageal fistula due to silicotic mediastinal lymphadenopathy, and Frew\(^2\) reported a 51-year-old man with a chest roentgenogram suggestive of pneumonoconiosis and dysphagia due to a tuberculoma in the esophagus. Mediastinal lymph node enlargement due to other granulomatous diseases such as sarcoidosis and histoplasmosis have been shown to compress the esophagus.\(^1\)\(^2\)

Although this patient had no specific signs or symptoms of tuberculosis, tissue cultures of the cervical lymph nodes grew \textit{M. intracellulare}. The increased incidence of \textit{Mycobacterium tuberculosis} in silicotic patients is well known. Also atypical mycobacterial infections appear to be more frequent in patients with pneumonoconiosis.\(^3\)\(^4\) Blacks generally have a lower incidence of clinical atypical mycobacteriosis than whites, but our patient’s silicosis and diabetes mellitus may have increased his risk to infection.\(^5\) His anemia of chronic disorders may be related to the mycobacteriosis.\(^6\)

Although this patient was lost to follow up by the chest service, it is interesting to speculate what the most appropriate course of therapy for him would have been. We recommended that he undergo a thoracotomy to remove the mediastinal lymph nodes compressing the esophagus and perhaps reduce the burden of tuberculous tissue as well. We also would have administered chemotherapy with five to six antituberculosis drugs because \textit{M. intracellulare} is notoriously resistant to conventional therapy.\(^7\) Atypical mycobacterioses are even more difficult to treat in patients with pneumonoconioses.\(^8\)

This is the first reported case, to our knowledge, in which silicotic lymphadenopathy complicated by \textit{M. intracellulare} infection produced dysphagia by extrinsic compression of the esophagus. Silicosis should be considered among other granulomatous diseases such as mediastinal granuloma, tuberculosis, and sarcoidosis in the differential diagnosis of dysphagia.

REFERENCES


Invasive Aspergillosis Presenting as Pericarditis and Cardiac Tamponade

John M. Luce, M.D.; Richard C. Ostenson, M.D.; Steven C. Springmeyer, M.D.; and Leonard D. Hudson, M.D., F.C.C.P.

A 38-year-old leukemic patient developed pericarditis and cardiac tamponade due to \textit{Aspergillus niger} one month after undergoing bone marrow transplantation. She failed to improve even though amphotericin B and rifampin therapy had been initiated before infection was evident. Her unique case illustrates both the unusual presentations of invasive aspergillosis and the difficulty of diagnosing and treating this increasingly common disease.

Invasive aspergillosis is recognized with increased frequency among immunocompromised patients.\(^1\)\(^2\) However, recognition often is delayed by the unusual and nonspecific manifestations of this condition. We present the case of a bone marrow transplant recipient in whom pericarditis and cardiac tamponade were the first clinical indications of invasive aspergillosis.

**CASE REPORT**

The patient was a 38-year-old woman with acute myelomonocytic leukemia who was transferred to the Fred Hutchinson Cancer Research Center (FHCRC; UPN907) in second relapse. Previous chemotherapy had included cytarabine, 6-thioguanine, and daunomycin. Admission laboratory findings included pancytopenia with a peripheral neutrophil count of 150/cu mm. Chest roentgenogram and ECG were normal (Fig 1, left). The patient was placed in laminar air flow and was given prophylactic oral antibiotics. She was prepared for transplantation with 1 mg/kg body weight of nitrogen mustard followed by 1,200 rads of total body irradiation in six divided doses, as per FHCRC protocol.\(^4\)

During preparation, the patient was given intravenous carbencillin and gentamicin for fever. Intravenous amphotericin B, 25 mg/day, and rifampin, 300 mg/day, were added when blood cultures grew \textit{Candida tropicalis}. Two weeks after admission, the patient received a bone marrow transplant from her HLA-matched sibling, who also was the source of daily granulocyte infusions.

The patient’s posttransplantation course was complicated by an episode of pulmonary edema which was thought to be due to pre-existent anthracycline cardiac toxicity and fluid overload. Her respiratory status improved with diuretics, and her chest roentgenogram cleared but for a small right middle lobe (RML) infiltrate. She then developed toxic enteritis which was treated with intravenously administered corticosteroids.

Three weeks after admission, the patient noted anterior chest pain which radiated into her throat and was intensified in the supine position. The pain was felt to be due to Candida esophagitis after barium swallow disclosed esophageal...