occurred in association with probable pneumoconiosis: Carasso et al\(^1\) reported a 48-year-old woman with a bronchoesophageal fistula due to silicotic mediastinal lymphadenopathy, and Frew\(^1\) reported a 51-year-old man with a chest roentgenogram suggestive of pneumoconiosis 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 pneumoconioses.\(^5\)-\(^6\) Blacks generally have a lower incidence of clinical atypical mycobacterioses than whites, but our patient's silicosis and diabetes mellitus may have increased his risk to infection.\(^6\) His anemia of chronic disorders may be related to the mycobacteriosis.\(^7\)

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.\(^8\) Atypical mycobacterioses are even more difficult to treat in patients with pneumoconioses.\(^6\)

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.

\section*{REFERENCES}

Invasive Aspergillosis Presenting as Pericarditis and Cardiac Tamponade*

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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.\(^1\)-\(^3\) 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.

\section*{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, \textit{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 carbenicillin 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 esopha-
FIGURE 1. Chest roentgenograms taken on admission (1A, left), at the time pericarditis became evident (1B, center), and shortly before death (1C, right). Note progressive increase in RML infiltrate and cardiac silhouette caused by invasive aspergillosis.

gual ulcerations. The pain worsened over the next two days, and the patient developed a rapid and irregular pulse. A pericardial friction rub then was noted, and chest roentgenogram revealed that the RML infiltrate had increased and now was associated with an enlarged cardiac silhouette (Fig. 1, right). An ECG showed low voltage and atrial fibrillation, and echocardiogram demonstrated a pericardial effusion of approximately 50 ml.

The patient was taken to surgery, where the right side of the chest was entered through a parasternal incision. The RML was found to be firm and pale, the pericardium was covered with a shaggy exudate, and the pericardial sac contained 50 ml of serosanguinous fluid. A pericardial window was created, and the pericardium and RML were biopsied.

On microscopic examination, the RML tissue was hemorrhagic and infarcted. Branching, septate hyphae were seen on the pleural surface; sheets of the same hyphae were found adjacent to and invading the pericardium. Cultures of the pericardium and pericardial fluid subsequently grew *Aspergillus niger*.

Amphotericin B was continued to a total dose of 750 mg, and the patient’s neutrophil count rose to 700/cu mm, indicating increased graft function. The patient was stable for two days until her cardiac silhouette enlarged even more on chest roentgenogram, and she developed a 30-mm paradoxical pulse (Fig 1, center). That night, the patient required endotracheal intubation and mechanical ventilation for worsening cardiopulmonary function. Pericardiectomy was planned, but she developed massive hemoptysis and died in ventricular fibrillation the following day.

At autopsy, the patient’s pericardium, lungs, pleurae, heart, kidneys intestines and thyroid gland were found to be involved with Aspergillus. The fungus had invaded the aorta and the left main and left anterior descending coronary arteries, as well as the proximal right pulmonary arterial tree.

**DISCUSSION**

Invasive aspergillosis is second only to candidiasis of the fatal mycoes seen in cancer patients. Although it occurs most commonly in persons with neoplasms, aspergillosis also has been reported in immunosuppressed persons with sarcoidosis and collagen vascular diseases and in individuals undergoing renal, cardiac, and bone marrow transplantation. Factors predisposing to aspergillosis include granulocytopenia, broad spectrum antibiotic therapy, and the administration of corticosteroids. All of these factors were present in this patient.

Aspergillus characteristically enters the body through the respiratory tract. In the lungs, tissue invasion usually is manifest by bronchopneumonia or by a distinctive pattern of hemorrhagic infarction. Such infarction, which was seen in this patient, occurs secondary to vascular invasion by mycelial elements with thrombosis and occlusion of the pulmonary vessels. The fungus then may spread from the lungs by direct invasion, or more commonly, by hematologic dissemination.

The organs most often involved in disseminated aspergillosis are the lungs, intestine, brain, kidneys, liver, esophagus, and heart, in that order. Pericardial involvement is unusual, occurring in only three of 93 patients in a series from the Memorial Sloan-Kettering Cancer Center and four of 98 patients from the National Cancer Institute (NCI). None of the NCI patients had signs or symptoms suggestive of pericarditis or pericardial tamponade. In 1962, Fraumeni and Fear described a lymphoma patient with distended neck veins and pulsus paradoxus, but that is the only reported case in which Aspergillus caused an obvious pericarditis.

Even when the diagnosis of aspergillosis is made, treatment often is unsuccessful. Therapy includes the administration of amphotericin B, flucytosine, and aerosolized nystatin, either alone or in combination. Rifampin and amphotericin B also have been used together.

According to one recent report, the clinical outcome of cancer patients with aspergillosis correlates best not with the total dose of amphotericin but with the recovery of circulating neutrophils. Outcome also has been reported to improve with early diagnosis and treatment. However, this patient already was receiving
amphotericin B and rifampin when her aspergillosis became apparent, and she had achieved partial bone marrow engraftment. Her case underscores the difficulty of diagnosing and treating invasive aspergillosis in immunocompromised hosts as well as the unusual manifestations of this disease.

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Spontaneous Return of Patency in a Completely Occluded Coronary Artery*

Neale D. Smith, M.D., and Henry DeMots, M.D.

We observed a 56-year-old man in whom an occluded right coronary artery was observed to be widely patent on a subsequent angiogram 40 months later. This “regression,” which occurred without a change in his risk factors, shows that manipulation of risk factors can be proven to be a cause of regression only in controlled studies.

Serial coronary arteriography has shown that the expected course of coronary atherosclerosis is a steady and relentless progression of disease. The rate of progression of an individual lesion is highly unpredictable but there appears to be a relationship between the number and severity of risk factors such as cigarette smoking, hypertension, and hyperlipoproteinemia and the rate of progression of coronary artery disease.

Regression of atherosclerosis in animals and in the peripheral vessels in man has been shown in response to manipulation of the atherogenic stimuli. Because it is generally presumed that coronary artery disease always progresses, or that if regression is possible it occurs only after vigorous therapy of atherogenic stimuli, we are reporting a patient with a prior inferior myocardial infarction in whom angiographic “improvement” of a right coronary artery occlusion was demonstrated. The improvement occurred without treatment of known risk factors for coronary artery disease.

CASE REPORT

The patient is a 56-year-old man who presented to another hospital with acute chest pain, diaphoresis, and syncope in December 1974. The ECG showed evidence of an acute inferior myocardial infarction and complete heart block. He was treated with intravenous isoproterenol, atropine, and oral prednisone. Eight days after myocardial infarction, he had a right femoral artery embolus which was uneventfully removed.

After discharge from the hospital, he noted persistent chest pain and dyspnea on exertion. He was admitted for re-evaluation in April 1975. He was 177.8 cm (70 inches) tall and weighed 72.3 kg (160 pounds). The arterial blood pressure was 115/80 mm Hg. The fasting blood sugar level was 90 mg/100 ml. A plasma cholesterol determination was not made.

Coronary arteriography was performed using the Judkins femoral percutaneous technique. Cineangiograms were made of the right and left coronary arteries in the right anterior oblique and left anterior oblique projections. Large film serial roentgenograms using a rapid film changer programmed for ten exposures in three seconds were made of each coronary artery in the lateral, right anterior oblique, and left anterior oblique projections. Secobarbital, 100 mg intramuscularly, was given prior to the procedure. Nitroglycerin 0.4 mg sublingual, was given prior to introduction of the right coronary artery catheter. The procedure was accomplished without complication; there was no angina during the procedure.

The examination revealed a complete occlusion in the midportion of the right coronary artery, complete occlusion of the left anterior descending coronary artery, and irregularity without significant obstruction in the proximal left circumflex coronary artery (Fig 1). There was complete morpologic consistency of all films. The left ventriculogram demonstrated inferior and apical akinesis and an ejection fraction of 0.15. Coronary bypass surgery was not recommended because of the poor ventricular function and because distal coronary arteries suitable for bypass grafting were not identified.

He was treated with progressively larger doses of propranolol until the dose of that medication reached 320 mg per day. Digoxin, 0.25 mg, was given daily and a thiazide diuretic was given for a brief period. No other drug therapy was used.

When the patient was first evaluated at our hospital in April 1978, he continued to complain of severe angina and dyspnea on exertion. In the interim, he had continued to smoke 18 packs of cigarettes per day as he had done for the previous 30 years. He had not changed his diet. His disability was such that he engaged in minimal physical activity.

He weighed 168 pounds. His arterial blood pressure was 130/85 mm Hg. He had bibasilar rales, an S3, and pedal