Granulomatous Pneumonitis Following Exposure to the World Trade Center Collapse*

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We describe a 37-year-old male engineer who presented with cough and dyspnea 3 weeks after exposure to dust resulting from the collapse of the World Trade Center (WTC). Radiographs of the chest and high-resolution CT demonstrated diffuse mediastinal nodularity. Lung biopsy specimens confirmed the presence of diffuse, nonepithelioid granulomatous nodules. Scanning electron microscopy and energy-dispersive radiograph analysis revealed large quantities of silicates. Cellular immunologic studies showed normal response to beryllium, and results of Kveim testing were negative. We suspect that exposure to one or more materials resulting from the WTC catastrophe may be implicated in the development of granulomatous pulmonary disease. (CHEST 2003; 123:301–304)

Key words: dust inhalation; granulomatous pneumonitis; World Trade Center

Abbreviations: OSHA = Occupational Safety and Health Administration; WTC = World Trade Center

On September 11, 2001, the two towers of the World Trade Center (WTC) collapsed and sent > 1 million tons of steel, glass, cement, and other debris to earth in clouds of smoke. The result of an intentional attack by suicide hijackers who crashed two commercial jetliners with almost 100,000 L of jet fuel into the towers, the resulting explosions set fires at > 982.2°C, burning a vast number of materials, such as concrete, asbestos, plastic, computers, furniture, and carpeting, which created a cloud of dust and smoke that continued to smolder for weeks.

We describe a patient who presented with mild, non-specific respiratory complaints with radiographic and light microscopic findings initially suggestive of sarcoidosis. Lung biopsy specimens submitted to scanning electron microscopy and energy-dispersive radiograph analysis revealed large quantities of silicates and the presence of multiple, well-defined pulmonary granulomas. Cellular immunologic studies showed a normal response to beryllium, and results of Kveim testing were negative.

Granulomatous disease of the lung may occur in response to a variety of infectious agents and to the inhalation of both organic and inorganic substances. When a specific agent cannot be identified, sarcoidosis may be suspected, often on clinical, radiographic, and immunologic grounds. In most instances, a search for a causative agent that results in the granulomatous tissue reaction is unrewarding. We suspect that exposure to one or more materials resulting from the WTC catastrophe may be implicated in the development of granulomatous pulmonary disease.

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CASE REPORT

The patient is a 37-year-old, white male who worked as a project manager for a consulting engineering company. He worked at a building located one block from the WTC. On September 11th at 8:45 am, he left his building and was informed that a plane had struck WTC tower 1. He witnessed the second plane striking the second tower and saw the collapse of that tower. He returned to his building, closed his windows, and sealed them with wet towels because of the infiltration of smoke, dust, and particulate matter. At 1:15 pm, he attempted to leave for home via ferries transporting employees to New Jersey. He was ferried by tugboat to Hoboken, NJ, with 12 other employees. At Hoboken, he was met by Red Cross personnel, was quarantined by the fire department, and was washed down with fire hoses. He was evaluated by a medical physician and placed on a transit train to his home in Newark.

On September 14th, he returned to work at the disaster recovery site for 1 day, in an effort to retrieve files and equipment. On October 3rd, he returned to work, maintaining generators supplying power to lower Manhattan. On October 15th, he returned to 2 Financial Center as a power engineer. His job was to maintain and install power generators supplying electricity to lower Manhattan until Consolidated Edison installed temporary feeders from buildings in the subcenter at 7 World Trade Center. On October 16th, he complained of general body aches, sore throat, coughing, wheezing, and shortness of breath. A viral illness was diagnosed by his physician. Radiographs of the chest demonstrated diffused miliary nodules throughout all lung fields.

The patient had no history of asthma, allergies, or any family history of respiratory disorders. He never smoked cigarettes and denied having tuberculosis, night sweats, fever, chills, or weight loss previously. He had undergone routine radiographs of the chest, with normal findings, 6 months before. His family history included four sisters, two brothers, two children, and parents without any history of respiratory disorders. His medical history included local neck spasms, a previous knee injury, and chronic prostatitis. He took no medications and denied any hobbies involving birds, molds, farming, or construction work. He had no other toxic occupational exposures. His occupation was that of a power engineer and supervisor and involved the maintenance and installation of power generators. In the first few hours of the WTC disaster, he used no protective respiratory equipment and states he was exposed to large quantities of dust as he remained in the vicinity of the collapse. On his return to work in the first week of October, he remained on-site inspecting and installing power-generating equipment. He was issued high-efficiency mask respirators but used them intermittently.

Physical examination revealed a well-developed, well-nourished white man in no respiratory distress. Vital signs were normal, and respiratory rate was 12 breaths/min and unlabored. No evidence of uveitis, cervical or supravacular adenopathy, or adventitious breath sounds were found on physical examination. There was no enlargement of the liver, kidney, or spleen, and peripheral pulses were normal. Clubbing, cyanosis, and edema were absent, and there were no significant rashes. Pulmonary function studies revealed a vital capacity of 5.3 L (103% of predicted), with an FEV₁ of 4.3 L (108% of predicted). Total lung capacity measured 6.8 L (94% of predicted), and diffusion capacity of the lung for carbon monoxide measured 32.3 mL/min/mm Hg (100% of predicted). Radiographs of the chest, dating October 16, 2001, were compared to radiographs obtained in June 2000. Diffuse bilateral reticular nodular infiltrates were seen without zonal predominance, hilar adenopathy, or pleural effusion. There was no evidence of pleural thickening or bony abnormalities. Prior radiographic findings were entirely normal.

CT examination of the chest (Fig 1) confirmed the presence of innumerable tiny, discrete densities, widely distributed bilaterally, each measuring < 1 cm in diameter. No evidence of pleural fluid, hilar or mediastinal adenopathy, or cardiac enlargement was noted. Urinalysis results were unremarkable. Hemoglobin measured 15.1 g/dL, and WBC count was 5,900/mL with normal distribution. Total eosinophil count measured 2.6%, and erythrocyte sedimentation rate was 1 mm/h. Antinuclear antibody findings were negative. HIV testing was nonreactive, and the angiotensin-converting enzyme level was 76.9 U/L. Protein serum electrophoresis was normal. Total protein was 7.6 g/dL, alkaline phosphatase was 101 U/L, aspartate transaminase was 24 U/L, creatinine was 1.0 mg/dL, and glucose was 73 mg/dL.

Lung biopsy specimens obtained by video-assisted thoracotomy and interpreted by the Armed Forces Institute of Pathology demonstrated noncaseating granuloma distributed along bronchovascular bundles. No microorganisms were seen, and polarizing microscopy revealed no birefringent material. Scanning electron microscopy revealed particles. Energy-dispersive radiograph analysis revealed silica, silicates, and calcium oxalate. Beryllium-induced lymphocyte proliferation study results were negative. Cultures revealed no evidence of mycobacteria, fungi, or viruses. Serum precipitins to aspergillus and thermophilic actinomyces species were negative, and skin testing by Kveim test and aspergillus protein was nonreactive.

After exclusion of infectious agents, differential diagnosis included sarcoidosis or hypersensitivity pneumonitis due to inhalation of fibrous glass, silica, heavy metals, or unknown combinations of chemicals and inorganic dusts present during the collapse of the WTC towers. A 4-week course of oral prednisone, 60 mg/d, failed to resolve granulomatous changes on follow-up high-resolution CT scanning. Lung function continued to be normal, and clinical symptoms have completely abated. The absence of mediastinal adenopathy, extrapulmonary involvement, and negative Kveim test results reduced the likelihood of sarcoidosis.

DISCUSSION

Granulomatous pneumonitis is a clinical disorder due to the inhalation of particulate antigenic material and is characterized in its acute phase by constitutional symptoms and sarcoid-like granuloma in the walls of alveoli and airways. The chronic phase of the illness is characterized by irreversible and progressive diffuse pulmonary fibrosis. The acute phase can be clinically mild and subacute. The majority of affected individuals are nonatopic. A large group of antigens have been reported to cause granulomatous pneumonitis and are generally of organic and inorganic matter. Organic causes fall into two major groups, microbial spores and animal proteins. These illnesses usually occur in association with an occupation such as farming or a hobby such as bird fancying.

Granulomatous lesions can result from antigenic stimulation of the immune system. It is thought that sarcoidosis results from some unidentified antigen that activates T lymphocytes, resulting in the release of lymphokines that stimulate a cellular immune response that leads to the formation of granuloma. Despite generations of research, the search for that antigen has been fruitless. Indeed, some have queried the possibility that sarcoidosis is an environmental disorder due to the inhalation of many antigens capable of sensitizing its host. There may be a wide variety of substances in the environment that result in a granulomatous inflammatory response.
in the formation of granuloma, such as beryllium, cobalt, titanium, and glass fibers. Talc, a hydrated magnesium silicate, although usually associated with pleural fibrosis and calcified pleural plaque, can be found to cause nonnecrotizing granulomatous inflammation. Other elemental materials, such as rare earth elements characterized by low atomic numbers, have been implicated in the development of granulomatous type responses in the lung. These include lanthanum, scandium, and yttrium.

In many instances, the specific causal agent has only been identified in a small number of patients. Organic isocyanates such as toluene diisocyanate or diphenylmethane diisocyanate, beryllium, and other heavy metals, and glass fibers have all been reported to be associated with granulomatous pneumonitis. A number of heavy metals such as aluminum, cobalt, and copper have been implicated in causing granulomatous pneumonitis with sarcoid-like granuloma in the mining and metallurgical industry.

It is possible that heavy metals, organic compounds, copper, cadmium, titanium, lead, chromium, aluminum, and unknown dusts and gases were released into the air and could account for the development of granulomatous pneumonitis in our patient.

New York hospitals reported more patients with asthma and respiratory complaints in the weeks following the attack, but to our knowledge no incidence of granulomatous pneumonitis was recorded among firefighters. Reports issued by the US Occupational Safety and Health Administration (OSHA) found high levels of heavy metal in some samples, but no excesses of silica, carbon monoxide or volatile organic compounds in others. Air sampling recorded during and after the disaster and analyzed by the Association for Independent Analyses indicated high levels of fiberglass coated with formaldehyde. Asbestos levels reached as high as 3.3% in some dust samples and were higher than the 1% legally designated as hazardous. Concerns that dioxin and polychlorinated biphenyls were elevated because of the combustion of foam upholstery, plastic tiles, synthetic carpets, computers, and polychlorinated biphenyl-containing oils were not substantiated by air sampling levels. Zirconium was not found in any of the air samples according to the US Environmental Protection Agency.

The United States Geological Survey, using imaging spectroscopy mapping on September 18th and September 27th, reported the results of samples of dust and debris from 35 localities within a 1-mile radius of the WTC. Indoor locations were included. Using refractance spectroscopy, scanning electron microscopy, radiograph diffraction, chemical analyses, and chemical leach testing, they concluded that the dust released from the WTC collapse was largely composed of glass fibers, gypsum, concrete, paper, and other construction materials. Chrysotile asbestos in trace levels was found in two of three of the dust samples and air fall samples, but ambient air sampling had not revealed concentrations of asbestos at...
levels that pose a threat to public health. OSHA collected 40 samples from the disaster site and found no hazardous levels of silica. The highest level was less than half the OSHA limit. To our knowledge, inhalation of silica and asbestos have not been associated with granulomatous reactions in the lung. Fine particulate matter (up to 2.5 μm in diameter) measured by continuous monitors and consisting mostly of combustion fuels were <65 μg/m3, but rapidly fell to <40 μg/m3 after 3 days. No amphibole asbestos was detected. Chemical leach tests and infrared imaging spectroscopy demonstrated the presence of heavy metals and metalloids such as aluminum, chromium, antimony, and barium.

Because we were able to exclude common causes of granulomatous pneumonitis such as berylliosis, sarcoidosis, tuberculosis, extrinsic allergic alveolitis due to organic dusts, and other bacterial, fungal, and viral illnesses, we suspect that exposure to dust at the WTC accounted for his illness. His illness may have been due to the inhalation of heavy metals based on air-sampling studies in the first 48 h following the explosion. It is quite possible because of the many chemicals, both organic and inorganic, released into the air that a totally unknown etiologic factor produced these granulomatous reactions.

CONCLUSION

Physicians who care for patients who were exposed to the dust caused by the collapse of the WTC towers or who continue to work in its midst with or without respiratory protection should be alerted to the possibility of hypersensitivity pneumonitis as an aftermath of the exposure. This case report should promote additional research in determining what environmental agents trigger sarcoid-like reactions. The opportunity to identify additional cases may provide clues as to the cause of sarcoidosis in some individuals.

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Squamous Cell Lung Cancer Simulating an Acute Myocardial Infarction*

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Lung cancer involvement of the heart is silent, carries a poor prognosis, and is most commonly identified at autopsy. A patient with lung cancer presented with symptoms and ECG findings suggestive of an acute coronary syndrome. Persistent symptoms and normal creatine phosphokinase (CPK) levels led to use of MRI and radionuclide scintigraphy to diagnose neoplastic infiltration of the myocardium. Palliative care was established with significant symptom relief. Assessment for cardiac metastases should be considered in patients with advanced lung cancer presenting with chest pain, new ECG findings, and normal CPK levels.

(CHEST 2003; 123:304–306)

Key words: cardiac enzymes; cardiac metastases; lung cancer; MRI; myocardial infarction; NeoTect radionuclide scintigraphy; radionuclide imaging

Abbreviation: CPK = creatine phosphokinase

Neoplastic involvement of the heart occurs insidiously in patients with advanced lung cancer and is rarely recognized prior to autopsy. A patient with lung cancer presenting with signs and symptoms suggestive of an acute coronary syndrome was found to have cardiac metastases with the use of thoracic MRI and radionuclide scintigraphy.

CASE REPORT

A 54-year-old man with stage IIIA squamous cell lung cancer treated with radiation therapy arrived at the emergency department reporting dyspnea and dull precordial chest pain radiating to the shoulder. This new-onset chest pain was aggravated with exertion but not reproducible with coughing or movement. Physical examination was noted to be unremarkable. ECG demonstrated a prior inferior wall injury pattern and new pronounced ST-segment elevation in the precordial leads (Fig 1).

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