8. Wijesundera P de S: Acute volvulus of the stomach. Ceylon Med J 19:33-36, Mar-Jun, 1974
9. Cole BC, Dickinson SJ: Acute volvulus of the stomach in infants and children. Surgery 70:707-717, Nov. 1971
10. Buchanon J: Volvulus of the stomach. Br J Surg 99:99-112, 1930
11. Nadkarni SV, Srinivasan M, Gowda HDC, et al: Acute volvulus of the stomach. J Indian Med Assoc 52:76-77, Jan 1969
12. Creedon PJ, Burman JF: Volvulus of the stomach. Am J Surg 110:964-966, Dec 1965
13. Chakrabarty PB, Panda K: Volvulus of the stomach. J Indian Med Assoc 63:131-132, Aug 1974
14. Metcalfe-Gibson C: A case of haemorrhage from volvulus of the gastric fundus. Br J Surg 62:224-225, Mar 1975
15. Borchardt M: Zur Pathologie und Therapie des Magenvolvulus. Arch Klin Chir 74:243-260, 1904
16. Campbell JB, Rappaport LN, Skerker LB: Acute mesenteroaxial volvulus of the stomach. Radiology 103:153-156, Apr 1972
17. Fink DW: Gastric volvulus: The angiographic appearance. Am J Roentgenol Radium Ther Nucl Med 115:268-270, Jun 1972
18. Winckler K, Schuster R: Acuter partieller Magenvolvulus mit spontaner Ruckbildung. Verh Dtsch Ges Inn Med 85:556-557, 1974
19. Edmondson HT, Veazey PR: Volvulus of the stomach. J Med Assoc Ga 63:339-341, Aug 1974
20. Maingot R: Abdominal Operations, Vol 1, 6th Ed. New York, Appleton-Century-Crofts, 1974
21. Tanner NC: Chronic and recurrent volvulus of the stomach with late result of colonic displacement. Am J Surg 115:505-515, Apr 1968
22. Myerson, DA, Myerson PJ, Lawson JP: Antral infracolic volvulus of the stomach. J Canad Assoc Radiol 26:128-131, Jun 1975
23. Raffin S: Gastric volvulus, chap 48, *In* Sleisenger MH, Fordtran JS: Gastrointestinal Disease. Philadelphia, WB Saunders Co, 1973, pp 612-615

Refer to: Schiffner RO, Sharma OP: Acute pulmonary cavitation in sarcoidosis. West J Med 127:346-349, Oct 1977

Acute Pulmonary Cavitation in Sarcoidosis

ROBERT O. SCHIFFNER, MD
Redwood City, CA

OM P. SHARMA, MD
Los Angeles

SARCOIDOSIS IS A relatively common multisystemic disorder whose cardinal findings are most frequently pulmonary and consist of bilateral hilar adenopathy or diffuse pulmonary fibrosis, or both. The purpose of this report is to present a rare manifestation of sarcoidosis: pulmonary cavitation.

Report of a Case

A 31-year-old black woman presented to the hospital with cough, sharp pain on the left side of the chest and dyspnea. A chronic cough had been present for a year, producing small to moderate amounts of whitish to yellowish sputum. She had lost eight pounds in the four months before admission. The patient said that there had been no fever, chills, night sweats, skin rash, arthralgias, or cardiac or renal problems. One year before admission she had been admitted to another hospital for delivery of her second child. At that time, an abnormality was noted on a chest x-ray film which was felt to be consistent with pulmonary tuberculosis. Sputum mycobacterial cultures were negative. The patient was given isonicotinic acid hydrazide, ethambutol and streptomycin. After two months she was lost to follow-up. She was followed at another clinic for hypertension and took alphamethyldopa and hydrochlorothiazide until five months before the present admission.

On physicial examination at admission the patient appeared chronically ill and was in mild respiratory distress. Temperature was 102.8°F (39.3°C) orally; pulse, 160 per minute; respirations, 44 per minute and labored; blood pressure, 160/100 mm of mercury. Breath sounds in the left side of the chest were decreased and moderate tenderness in the abdominal right lower quadrant was noted. Results of the examination were otherwise unremarkable.

The leukocyte count was 11,000, with a mild left shift of the differential count. Analysis of urine showed no abnormalities. The serum glutamic pyruvic transaminase value was 50 (normal, 5 to 40 units) and the serum glutamic oxaloacetic transaminase value was 70 (normal, 5 to 40 units). The rest of the liver function studies, electrolytes and renal function studies showed no abnormalities. Gram stain of the sputum showed moderate numbers of polymorphonuclear leukocytes and Gram-positive cocci. A few Gram-negative rods were also noted. Sputum cultures showed normal aerobic flora. Fungal and mycobacterial cultures were negative. A transtracheal aspirate study showed the presence of anaerobic streptococcus, diphtheroids and bacteroides melaninogenicus. Tuberculin, histoplasmin and coccidioidin skin tests were negative. However, there was a 5 mm induration for mumps skin test. The serum immunoelectrophoresis showed slight elevation of IgA.

An x-ray film of the chest (Figure 1) showed a left pneumothorax. A chest tube was placed. During the ensuing two-month hospital stay, the patient had a stormy course, marked by fever and continued pulmonary cavitation (Figure 2).

From the Pulmonary Disease Service, University of Southern California School of Medicine, Los Angeles County-University of Southern California Medical Center, Los Angeles.
Submitted October 26, 1976.
Reprint requests to: Robert O. Schiffner, MD, Redwood Medical Clinic, 2900 Whipple Ave., Redwood City, CA 94062.

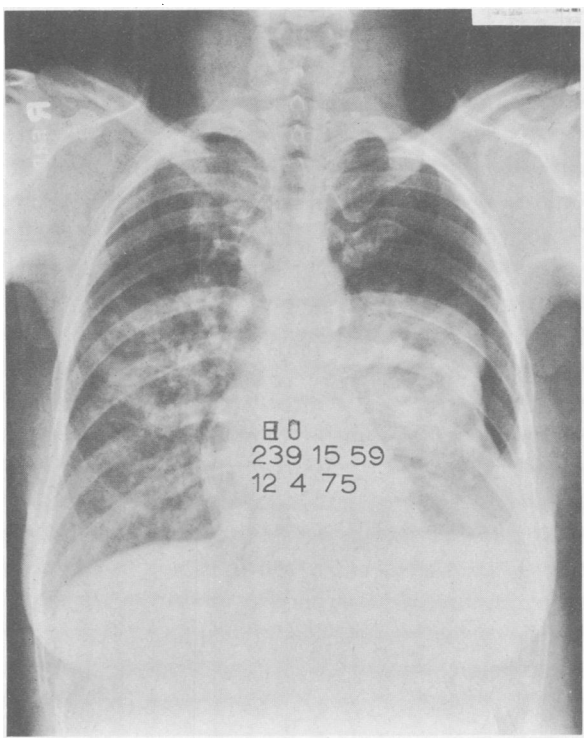

Figure 1.—Posteroanterior chest roentgenogram showing diffuse reticular, linear and nodular infiltration in both lungs with pneumothorax on the left. Note the rarifaction in the center of the collapsed lung.

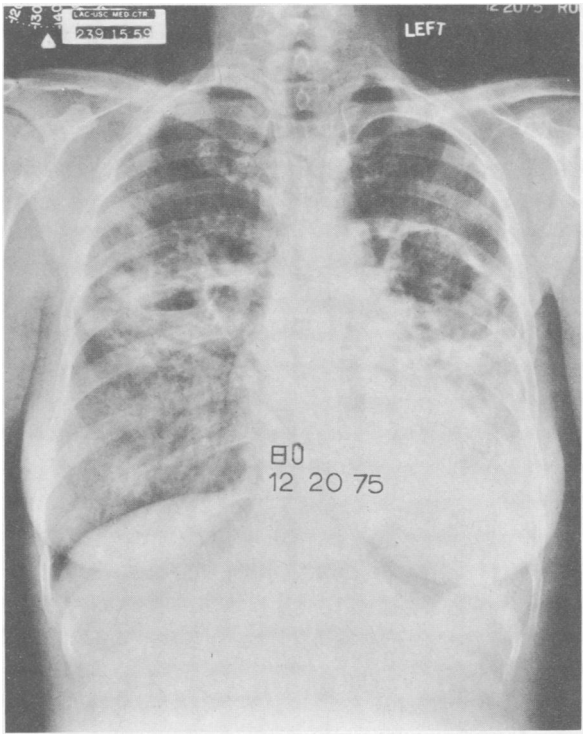

Figure 2.—Posteroanterior chest roentgenogram showing diffuse interstitial infiltration with multiple cavities in both lungs, two with air-fluid levels.

High doses of penicillin G, gentamycin, methacillin and chloramphenicol in varying combinations were given. Two bronchoscopic examinations, one with transbronchial forceps biopsy, were not rewarding. A bone marrow biopsy and aspirate did not identify an etiologic agent. A percutaneous trephine lung biopsy was carried out and showed only interstitial fibrosis and a histiocytic reaction in the alveolar walls. The patient died after a two-month stay in the hospital.

At autopsy there were cavities in both upper lobes, measuring 4 by 8 cm. The cavities had thick, densely fibrotic, bland walls (Figure 3). There was no pus in these cavities. In surrounding areas there was bronchopnuemonia. Anterior mediastinal lymph nodes were enlarged. There was no pleural fluid in either hemithorax. Nothing remarkable was noted on examination of the heart. Examination of the abdomen showed many large mesenteric and periaortic nodes. Cultures of the autopsy material did not grow any bacteria or fungi.

Microscopic examination showed many non-caseating granulomas with giant cells in the lungs (Figure 4), anterior mediastinal nodes, liver (Figure 5) and abdominal lymph nodes. Most of the granulomas were densely hyalinized, indicating long-standing advanced disease. Special stains for acid-fast bacilli and fungi were negative. Examination of the specimens under polarized light did not show birefringent particles. These features were consistent with a diagnosis of sarcoidosis.

Comment

The lungs are affected in more than 90 percent of the patients with sarcoidosis. Common pulmonary manifestations include bilateral hilar node enlargement with or without interstitial infiltration and fibrosis. Bullae formation may occur in some patients with extensive fibrosis. Uncommon pulmonary manifestations are pleural effusion, nodular shadows, calcification of the hilar nodes, pneumothorax, unilateral hilar adenopathy and localized lung infiltrate.[1] Pulmonary cavitation is rare in sarcoidosis. A major pulmonary radiology text does not include sarcoidosis in an exhaustive differential diagnosis of lung cavities.[2]

There have been a few reports of pulmonary cavitation in sarcoidosis.[3-5,8-11] Some of the reports leave open to question whether or not

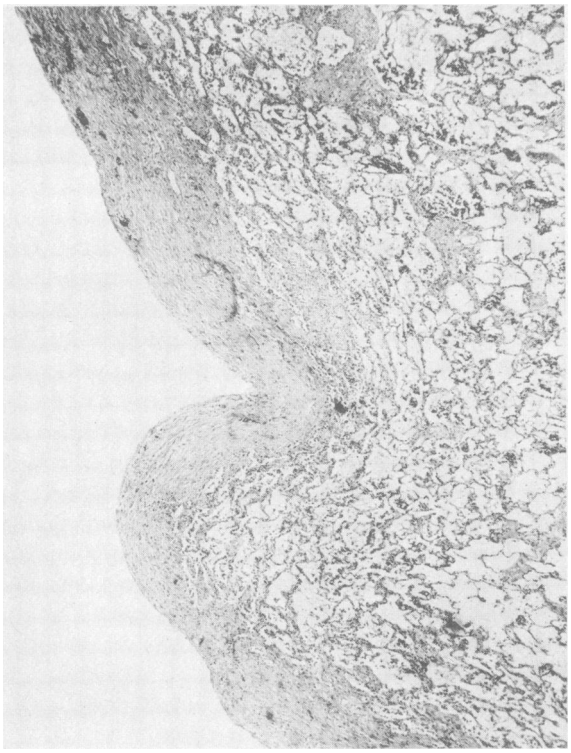

Figure 3.—Microscopic section of a cavity wall showing a thick, fibrotic wall with little inflammatory reaction (H & E × 75).

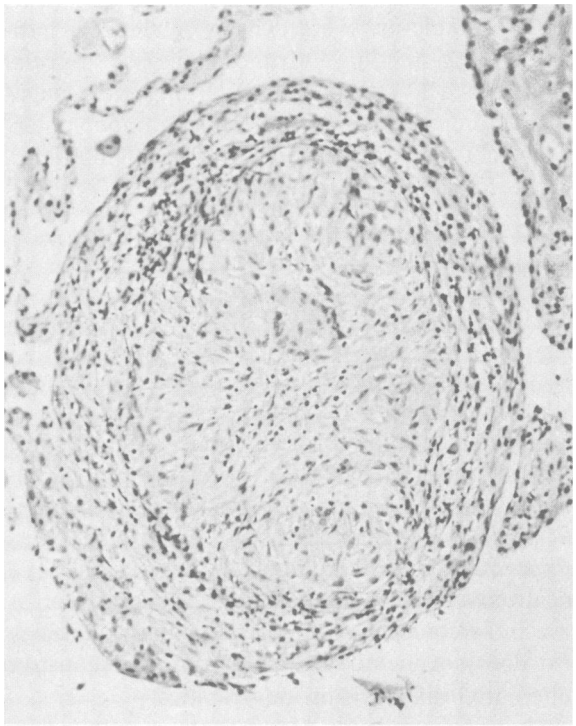

Figure 4.—Microscopic section of the lung showing noncaseating granuloma with giant cell. Surrounded by relatively normal alveolar wall (H & E × 200).

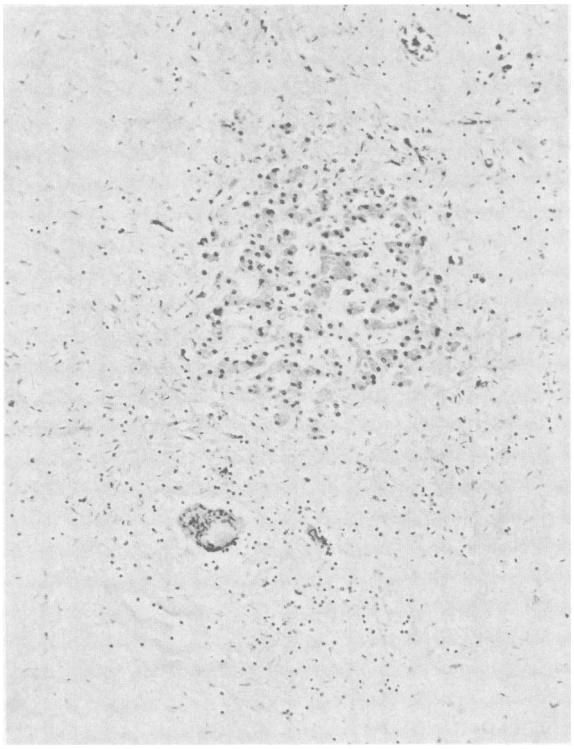

Figure 5.—Microscopic section of the liver showing relatively normal liver surrounded by hyalinized granulomatous tissue with giant cell (H & E × 120).

sarcoidosis was the primary cause of the cavitation. Hamilton and co-workers[5] emphasize that other causative agents must be excluded, such as fungi, mycobacteria or pyogens. Initially in our patient a pyogenic abscess was the likely diagnosis. However, failure to isolate an aerobic organism associated with lung abscess, failure to halt the progress of the illness with antibiotics active against anaerobes and aerobes, and the autopsy findings of thick-walled, bland, fibrous cavities seemed to exclude the diagnosis of bacterial abscess. Tuberculosis, fungal diseases and parasitic diseases were also excluded. There was no clinical or pathologic evidence to support Wegener granulomatosis or rheumatoid lung disease. Absence of a relevant occupational and clinical history ruled out the possibility of pneumoconioses, cystic fibrosis, bronchiectasis or traumatic cavities.

A review of the literature has found nine other cases of pulmonary sarcoidosis and cavity formation. This brings the total number of patients with cavity sarcoidosis to ten (Table 1). Seven of the nine patients in whom the clinical data were known were between 20 and 40 years in age. There were five women and four men. In half

of the cases there were emphysematous bullae or subpleural blebs and true cavities were not present. Therefore in only five patients, including the one reported here, were true pulmonary cavitations present. Our patient is the only case reported with true cavities in both lungs. Only the left lung was involved in three others and the fifth patient had right upper lobe involvement. The clinical manifestations included spontaneous pneumothorax in two patients, whereas, hemoptysis occurred in one patient and dyspnea in another; one patient with a cavity was asymptomatic. Although no consistent clinical features of cavitary pulmonary sarcoidosis emerged from the study, we feel that sarcoidosis should be considered in a patient with cavitary lung disease. Whether the institution of aggressive corticosteroid therapy would have changed the cause of the disease in our patient remains uncertain. The cause of cavity formation in sarcoidosis also remains unknown. Scadding[7] states that thick-walled cavities may form in one of three ways: (1) emphysematous bullae whose walls are thickened by secondary infection, (2) intercurrent pyogenic lung abscess and (3) necrosis at the centers of dense hyaline fibrosis formed by the coalescence of hyalinizing granulomas. It is our feeling that only the last category represents true sarcoidosis cavities.

Summary

A case of progressive cavitary lung disease is reported. The patient was found to have sarcoidosis. Other causes for cavitation were excluded. Sarcoidosis must be considered in a patient with cavitary lung disease, particularly in patients in those groups commonly affected by sarcoidosis: women, blacks and patients between 20 and 40 years old.

REFERENCES

1. Sharma OP: Sarcoidosis: A Clinical Approach. Springfield, Ill, Charles C Thomas, 1975, pp 30-64
2. Fraser RG, Pare JAP: Diagnosis of Disease of the Chest—An Integrated Study Based on the Abnormal Roentgenogram. Philadelphia, WB Saunders, 1970, pp 421-433
3. Bistrong HW, Tenney RD, Sheffer AL: Asymptomatic cavitary sarcoidosis. JAMA 213:1030-1032, 1970
4. Harden KA, Barthakur A: Cavitary lesions in sarcoidosis. Dis Chest 35:607-614, 1959
5. Hamilton R, Petty TL, Haiby G: Cavitary sarcoidosis of the lung. Arch Intern Med 116:428-430, 1965
6. Freundlich IM, Libshitz HI, Glassman LM, et al: Sarcoidosis: Typical and atypical manifestations and complications. Clin Radiol 21:376-383, 1970
7. Scadding JG: Sarcoidosis. London, Eyre and Spottiswoods, 1967, pp 115-135
8. Tice F, Sweany HC: A fatal case of Besnier-Boeck-Schaumann's disease with autopsy findings. Ann Intern Med 15:597-609, 1941
9. Hogan GF: Sarcoidosis: Report of five cases with one autopsy. Am Rev Tuberc 54:166-178, 1948
10. Ustvedt HJ: Autopsy findings in sarcoid. Tubercle 29:107-111, 1948
11. King DS: Sarcoid disease as revealed in the chest roentgenogram. Am J Roentgen 45:501-512, 1941

TABLE 1—*Cases of Pulmonary Sarcoidosis and Cavity Formation*

Author	*Age*	*Sex*	*Race*	*Location*	*Size of Cavities*	*Course*	*Hilar Adenopathy*
Hamilton[5] ...	30	M	black	LUL cavity*	3 cm	Hemoptysis, stable course after surgical operation	yes
Harden[4]	31	F	black	Bilateral* midlung zone bullae, thick-walled cavity Left lung	4 cm	Cor pulmonale, died	no
	30	F	black	Bilateral upper lobe blebs, thick-walled cavity RUL*	4-5 cm	Spontaneous pneumothorax, underwent pleurectomy, thereafter chronically dyspneic on exertion	no
	41	M	black	Bilateral lower lobe and upper lobe emphysematous bullae	>6 cm	Underwent resection of LLL bullae, chronically dyspneic on exertion	no
Tice[8]	48	F	NA	RUL blebs	2 cm	Shortness of breath, cor pulmonale, died	yes
Hogan[9]	36	M	black	Bilateral apical bullae	>6 cm	Dyspneic, died	yes
Ustvedt[10] ...	39	F	white	Bilateral large apical bullae	>6 cm	Sudden severe hemoptysis, died	no
King[11]	NA	NA	NA	Bilateral apical bullae	>6 cm	Pleuritic chest pain, died	no
Bistrong[3] ...	20	M	white	LUL cavity*	<3 cm	Asymptomatic	no
Schiffner	31	F	black	Bilateral* upper lobe cavities	8 cm	Spontaneous pneumothorax, febrile course, respiratory failure, died	no

NA = information not available LUL = left upper lobe RUL = right upper lobe LLL = left lower lobe

*In these patients true cavities seem present.