Pulmonary Amebiasis of the Upper Lobe
Report of Two Cases
SAYYED E. WARRAKI, M.D.*
Cairo, Egypt

IN TROPICAL AND SUBTROPICAL COUNTRIES, pleuropulmonary amebiasis is occasionally diagnosed. The usual findings in the chest are either in the form of basal pneumonitis or abscess in the right lower lobe, or much less commonly in the middle lobe. This results from direct extension of infection from amebic hepatitis or liver abscess. In the roentgenogram, there is no zone of healthy lung tissue between the lung lesion and the diaphragm. Such cases represent basal pulmonary amebiasis.

Amebic lung infection located in the upper or lower lobes with no direct proximity to the liver has been reported by several authors and is decidedly a rare type. Bunting1 described consolidated nodules in the lungs in which Endamoeba histolytica was found in section. Manson-Bahr2 reported three cases with no involvement of the liver. Harrington3 reported a solitary lung abscess in the right upper lobe, while two necropsies were described by Girgis4 with multiple abscesses in the lungs, liver and bowels; Endamoeba histolytica was demonstrated in abscess cavities, as well as in a rider embolus in a pulmonary artery branch of one of the two cases. Amebic pulmonary lesions of the right upper lobe, apex of the lower lobe, as well as disseminated focal foci were described by Zaki.5

In this communication, two cases of non-basal amebic lung infection are described, one with a lesion in the right upper lobe, the other in the lingula of the left upper lobe. The two cases have been diagnosed during the last ten years, which in an endemic country should point to the rarity of the condition.

*Assistant Professor of Chest Diseases, Ain-Shams University Hospitals, Cairo, Egypt.

CASE REPORTS

CASE 1

F. E. F., a man, age 29, presented with a chief complaint of cough and expectoration of about 30-60 ml. of purulent sputum together with sudden intermittent fever and chills for the last two weeks. The patient had not complained of any important antecedent chest disease. He stated that an x-ray shadow had been found in his chest and he had been told that he had "pneumonia." Oxytetracycline (Terramycin) and chloramphenicol were given for five days, with no response from either.

Physical examination revealed a well built man with pronounced pallor and toxemia. Oral temperature was 101°F. Chest examination showed slight impairment on percussion and faint breath sounds below the right clavicle. The liver was tender and enlarged (three fingers). A chest roentgenogram on February 26, 1956 (Fig. 1) showed a homogeneous triangular opacity in the right upper zone periipherally with suspicion of breakdown, and some mottling medial to the main opacity. The leukocyte count was 14,200 per cmm., polymorphs were 74 per cent and eosinophils 3 per cent. Sputum examination showed ordinary microbial flora which were sensitive to oxytetracycline (Terramycin), chloramphenicol and to various other antibiotics; concentrated smears for tubercle bacilli were negative. The sputum was also negative for Endamoeba histolytica, fungi and spirochetes. Cysts of Endamoeba histolytica were demonstrated in the stools. Urinalysis was negative. Bronchoscopy was done on February 28, 1956 and revealed no abnormal finding.

Considering the lack of response to previously administered antibiotics in full doses, the enlarged tender liver and the presence of the parasite in the stools, a second sputum examination was done; Endamoeba histolytica could be demonstrated after warming the slide. Emetine hydrochloride, one grain daily intramuscularly, was started on March 2, 1955 for ten days. A pronounced and rapid response followed the third injection and an x-ray examination of the chest on March 9, 1956 (Fig. 2) showed complete resolution of the shadow. The case was followed for two years with no evidence of relapse.
CASE 2

M. K. A., man, aged 47, was first seen on November 12, 1961, with the chief complaint of cough and hemoptysis for the last two days, together with chills, fever and chest pain in the left side. He had no history of any important antecedent chest disease. In 1954, he was operated upon for a mass in the descending colon which was resected and proved to be carcinoma. Prior to the operation, he had complained of distension, constipation, melena and occasional dysenteric symptoms.

Physical examination revealed a well built, middle aged man with toxic look and pallor. Oral temperature was 102° F. Chest examination showed only few rales in the left mammary region. No abdominal masses were felt and the liver was not enlarged. Examination of other systems was negative.

A chest x-ray film on November 12, 1961 (Fig. 3) showed a homogeneous opacity in the left paracardiac zone which is circumscribed, with a suspicious breakdown in its lower and outer part. The leukocyte count was 9,700 per cmm., the differential count showed polymorphs of 80 per cent and eosinophils were 5 per cent. Sputum examinations were negative for tubercle bacilli, including concentration method. Other bacterial flora mainly Hemophilus influenzae and Neisseria were found; Endamoeba histolytica was not reported. Examination of stools and urine was negative for parasites.

A diagnosis of a malignant metastatic lesion was provisionally made and bronchoscopy was performed on November 14, 1961. No abnormal finding was reported. Sigmamycin administration was started on the same day. Sigmoidoscopy on November 17, 1961 revealed evidence of an old amebic infection with no sign of malignancy. No response to the previously administered sigmamycin was noted.

Because of the acute clinical picture with fever and toxemia, the sigmoidoscopic report and the lack of response to sigmamycin, one grain emetine injections intramuscularly daily for ten days were started on November 17, 1961, a rapid clinical improvement followed and a chest roentgenogram on November 26, 1961 (Fig. 4) showed a clear left lung. The response was lasting since the case was followed up for the next eight months without evidence of recurrence.

DISCUSSION

Non-basal pulmonary amebiasis with or without liver involvement is rarely diagnosed even where amebiasis is endemic.
PULMONARY AMEBIASIS

Abdel-Shaffi (1952) reported that of 1000 necropsies in Egypt, 40 had amebiasis, of these, 16 had lung abscesses in various locations in both lungs. This high prevalence in necropsy material strongly indicated that non-basal lung amebiasis is much more common than is generally believed.

Various routes of infection have been suggested. Petzetakis' observed cases in which infection was confined to the bronchi causing "amebic bronchitis" with a viscous, glary sputum which was occasionally blood-tined. He suggested that primary infection of the lungs and bronchi may result from inhalation of dust containing amebic cysts. Abdel-Hakeem and Higazi* reported that among 28 cases of basal pulmonary amebiasis, two had "amebic bronchitis" in addition to their lung lesions. Warraki* stated that in the absence of lung lesions, lesions; however, there seems to be no available evidence that upper lobe lesions can result from infection carried from the liver via the lymphatics while sparing healthy intermediate lung tissue. Direct embolism to the lungs by the systemic circulation through the middle and inferior hemorrhoidal veins draining the rectum and anal canal is the most accepted route. The liver is not involved in about 14.3 per cent and thus such cases are considered as primary pulmonary amebiasis." Blood-borne emboli can also originate from thrombosed hepatic veins from which site they are carried by the blood to the right side of the heart and the lungs.*

In pulmonary amebiasis of the upper lobes as in basal lesions, history of dysentry may be missing. Clinical symptoms of intermittent fever, pallor and pronounced anorexia are similarly found. History of previous dysentry was negative in case 1; in case 2, history of colonic obstruction together with melena was obtained. Chest pain and hemoptysis occurred only in case 2, while purulent sputum was coughed up in both cases. The bitter taste of the sputum (attributed to bile content) and the chocolate or anchovy-sauce color (attributed to liver pus) are characteristics of basal amebic infection and were not recorded in either of the cases herein reported.

Chest roentgenograms of both cases showed homogeneous shadows with suspicion of breakdown. In upper lobe lesions of the right lung, Zaky* described a tell-tale increase in linear markings due to infiltrated lymphatics along the main inferior bronchus and leading to the hilum. This was not observed in Case 1. Slight limitation of diaphragmatic mobility was observed on fluoroscopy only in the first case, in which the liver was palpated, but the level of the diaphragm was not abnormally high.

In endemic areas, the diagnosis of non-basal pulmonary amebiasis should be suspected if there is history of dysentry or if the liver is tender and palpable. Inter-

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*Figures 3 and 4 show chest roentgenograms.
specific involvement of the bronchi by amebic infection is doubtful. Lymphatic spread across the diaphragm is considered to be the route of infection in basal amebic mitten fever with pallor and toxemia are constant features and the leukocyte count is usually high with increase in polymorphonuclears and eosinophilia. The lack of resolution of an x-ray shadow in the lungs in response to broad-spectrum antibiotics, when the microbial flora in the sputum may not be possible. Sigmoidoscopy bronchoscopic examination is negative, should arouse suspicion. Demonstration of Endamoeba histolytica in the stools or the sputum may not be possible. Sigmoidoscopy may support the diagnosis if evidence of amebic infection is existing. Indirect proof of the etiology is obtained by the therapeutic test using emetine, the dramatic and lasting response both clinically and radiographically is diagnostic. The response of pulmonary amebic lesions to emetine is similar to that of amebic hepatitis, i.e., striking and more rapid and lasting than in the case of the bowels.

References


For reprints, please write Dr. Warraki at 11 Midan El Falaki, Cairo, Egypt.

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