Amebic Pulmonary Suppuration*

J. P. Sethi, M.D.,** M. L. Gupta, M.D.† and R. M. Kasliwal, M.D., F.C.C.P.‡

Jaipur, India

Pleuropulmonary lesions next to the liver are the most common extra-intestinal manifestations of amebiasis. Since the original reports by Collin and Nixon¹ and Opie,² a good number of accounts based on clinical and necropsy data have appeared in the literature. In the majority of cases, pulmonary involvement is secondary to hepatic involvement. In approximately 75 per cent of the cases, the amebic liver abscess ruptures directly through the diaphragm giving rise to empyema, lung abscess, or a bronchohepatic fistula.³ In others, a contiguous spread or a transdiaphragmatic extension along the lymphatic channels may result in pulmonary involvement. It has also been described that hematogenous emboli can originate in the thrombosed hepatic veins and reaching the inferior vena cava, may lodge in the pulmonary capillaries giving rise to abscess formation.⁴ Girgis⁵ reported multiple abscesses in the lung, liver and bowels. E. histolytica were demonstrated in these cavities, as well as in a rider embolus in a pulmonary artery branch.

Cases of pulmonary amebiasis without hepatic involvement have also been described in the literature: the so called “primary pulmonary amebiasis.”⁶ Batson⁷ suggested that pulmonary involvement in such cases follows spread of infection along the vertebral system of veins which have anastomosis with other venous systems.

Amebic pulmonary suppuration generally involves the basal segment of the right lung, but involvement of the upper lobe⁸ and middle lobe⁹,¹⁰ have also been reported. Not infrequently abscess in the left lobe of the liver may rupture into the left lung or pleura producing lesion on the left side.

The clinical picture of amebic lung abscess may resemble that of any other pulmonary suppuration. It is characterized by fever, cough, pain in the chest, blood in sputum and physical signs of pneumonia or abscess. The lesion cannot be differentiated roentgenologically from pulmonary abscess due to other causes. Cavitation is common and may lead to the diagnosis of tuberculosis.

Entameba histolytica trophozoites have been demonstrated in the sputum and their presence is regarded to be diagnostic.⁵,¹⁰ Sullivan and Bailey⁴ found that forceful coughing may dislodge minute particles of tissue from the abscess wall which may help in detecting entameba in the sputum. However, a failure to find the parasite does not rule out the disease.⁴ Finding of Entameba histolytica in the stool is suggestive, but not diagnostic. Complement fixation test has been found to be positive in pulmonary amebiasis by Sullivan and Bailey,⁴ and Kasliwal et al.¹¹ and it may be regarded as a very useful diagnostic tool. By far the most important aid in the diagnosis of pulmonary amebiasis is the therapeutic response to emetine especially so when antibiotics and other therapeutic procedures have failed to produce any significant clinical improvement.¹⁰,¹⁰-¹⁴

Material and Methods

Nine cases of amebic pulmonary suppuration admitted as indoor patients in the S.M.S. Hospital have been analyzed in the present study. All the cases were evaluated by clinical history, physical examination, general laboratory and liver function tests, and repeated stool examination. Past history of diarrhea or dysentery was especially inquired in each case.

*From the Department of Medicine, Sawai Man Singh Medical College.
**Reader.
†Lecturer.
‡Professor and Head of the Department.
In addition to complete routine examination, sputum in each case was carefully examined in saline for *Entameba histolytica* trophozoites. Posteroanterior and lateral roentgenograms of the chest and fluoroscopic examination for the visualization of the position and amplitude of the movements of the right dome of the diaphragm were conducted in each case. Complement fixation test for amebiasis was performed in two cases by MicroKolmer technique.

**Observations**

Of the nine cases analyzed, eight were men. Seventy-eight per cent of patients were in the third decade. Past history of dysentery was given by five cases.

Table 1 shows the presenting symptoms. Fever was present in every case. In three, it was associated with chills and rigors. Pain in the chest was a common symptom. In most of the cases, it was associated with a dull, boring, at times stretching ache in the right hypochondrium. It was localized in the right inframammary, infraaxillary and infrascapular regions and in two cases was referred to the right shoulder. In one case, pain was substernal. Pain at times was severe and aggravated by coughing and walking. Cough in every case was associated with expectoration. Frank hemoptysis was encountered in four cases, though the history of blood-streaked sputum was present in all the cases. One case had a severe hemoptysis which proved fatal.

Table 2 shows the physical signs. Fever was observed in all the cases ranging between 100-104°F. Anemia was present in seven. Clubbing of the digits was conspicuously absent in all the cases. Tender hepatomegaly was encountered in eight in the present series. In all the cases, the lesion was situated in the right lung. Signs of consolidation were present in the right infrascapular region in eight cases and in right mammary region in one case. In two cases a pleural rub was also heard. In both of these cases, pulmonary suppuration was basal in location.

**Laboratory Findings**

Total leukocyte count varied between 8,800 to 22,400/mm³. In the differential picture, neutrophilia was encountered in all the cases and ranged between 78-88 per cent. Stool was positive for *Entameba histolytica* in three cases only and one showed E.Coli. Sputum was found to be anchovy sauce in two cases, mucopurulent in three and in three it had the appearance of dirty-grayish pus mixed with blood. In one case, it had a typically deep yellow appearance with high bilirubin content. *Staphylococcus aureus, N.catarrhalis, Streptococcus viridans* were grown on sputum culture. However, no Entameba could be demonstrated even on repeated examination.

**Serologic Findings**

Complement fixation test was done in two cases and was found to be positive in both.

**Radiologic Findings**

Immobility or diminished amplitude of movement, elevation and tenting of the right leaf of the diaphragm were the most common fluoroscopic findings encountered. Roentgenogram of the chest showed consolidation of the right basal segments in seven cases (Fig. 1A), right middle lobe in one case and superior segment of the right lower lobe in one case (Fig. 2A).

Penicillin or oxytetracycline followed by
etamine and chloroquine were administered in eight cases. Five recovered completely, two showed residual fibrosis and tenting of the diaphragm (Fig. 1B and 2B), and one died of hemoptysis and circulatory failure during the course of treatment. In one case with no hepatomegaly, sputum culture showed Staphylococcus aureus sensitive to all antibiotics. Penicillin and sulfadiazine were given in adequate dosage for six days with no improvement. This was followed by erythromycin 250 mg four hourly for four days with no improvement whatsoever. Then emetine hydrochloride, 65 mg.
I.M. daily with chloroquine diphosphate 250 mg b.i.d. were administered. On the third day of therapy, the temperature came to normal and then remained so for three weeks, subsequent to which a short bout of fever recurred for which emetine was restarted. Complete clinical and radiologic recovery was achieved after 35 days of treatment having been given two courses of emetine therapy during this period.

**Discussion**

Pulmonary amebic suppuration is not an infrequent occurrence, though primary pulmonary amebiasis is an uncommon entity which is difficult to diagnose. The majority of the cases, however, are secondary to hepatic involvement as encountered in all except one in the present series. The solitary case not associated with hepatomegaly may be classified as one of primary pulmonary amebiasis though secondary to intestinal affection. In this case, stool was found to be positive for *Entameba histolytica*, but the latter was not found in sputum. Adequate treatment with antibiotics failed to bring about improvement, while a therapeutic test with emetine ameliorated the symptoms. A complement fixation test done later was found to be positive in a significantly high titer.

All the cases in the present series had consolidation in the right lung. Out of nine cases, seven were basal in location, with only one in the middle lobe and one in the superior segment of the right lower lobe. Eight of these nine cases were associated with tender hepatomegaly, which would suggest that the presence of pulmonary suppuration, especially when right sided and basal points towards the presence of an amebic lesion. Symptoms and signs in general observed in this series of amebic pulmonary suppuration were found to resemble those of ordinary pyogenic abscess. Clubbing of the fingers, however, was conspicuously absent in all the cases in contrast to what we observe in pyogenic suppuration. Anchovy sauce pus in the sputum is not an invariable accompaniment, being seen only in two cases in the present series. In none of the cases in the present series did sputum examination reveal *Entameba histolytica*. This renders the diagnosis of amebic etiology difficult. In such cases, therapeutic response to emetine or chloroquine becomes a deciding factor in establishing the diagnosis.

Complement fixation test could be considered a satisfactory diagnostic aid especially when a high titer of the complement fixing antibodies is present. It was found to be positive in both the cases in the present series in whom it was done. However, the number is too meagre and further evaluation is needed for a statistical assessment of its significance. The difficulty encountered is usually in getting the amebic antigen. Finally, it may be emphasized that amebic etiology should always be considered in the diagnosis of pulmonary suppuration, especially in right sided lesions. Moreover, in abscesses not responding to the usual antibiotic treatment amebic etiology should be kept in mind constantly when amelioration of the symptoms with emetine therapy may substantiate the diagnosis. Complement fixation test for amebiasis should be done in all cases of lung abscesses of obscure etiology.

**Summary and Conclusions**

Clinical analysis of nine cases of amebic pulmonary suppuration is presented. Except one case of primary pulmonary amebiasis, all the rest were secondary to hepatic involvement. Seven of nine cases were right basal in localization, one case was in the right middle lobe and one in the superior segment of the right lower lobe. In the absence of case of demonstration of *Entameba histolytica* in the sputum, need for a suitable diagnostic tool for etiologic diagnosis is seriously felt. Complement fixation test could be considered a satisfactory diagnostic aid; further work in this line is desired. At present, therapeutic test with emetine continues to be the mainstay in the diagnosis of amebic pulmonary suppuration.
Resumen
El autor presenta nueve casos de supuración pulmonar de etiología amebiásica. Excepto un caso de supuración pulmonar primaria los demás casos fueron secundarios a abcesos hepáticos. Siete de los casos fueron de localización basal derecha, uno del lóbulo medio derecho, el otro del segmento superior del lóbulo inferior derecho. A falta de la demostración fácil de la presencia de ameba histolítica en el esputo, sería muy de desear un procedimiento apropiado para el diagnóstico etiológico. Las pruebas de fijación del complemento pueden ser consideradas como de utilidad y convendría proseguir la investigación en ese sentido. Al presente la prueba terapéutica con emetina continúa siendo el recurso principal para el diagnóstico etiológico de las supuraciones pulmonares amebiásicas.

Zusammenfassung

References
1 Collin and Nixon (1873) Quoted by Hakim, M. A. and Hogazi, A. M.: "Broncho-

For reprints, please write: Dr. M. L. Gupta, Sanghi Bhawan, B-201 Bapu Nagar, Jaipur, India.

Pericardial Effusion
The diagnosis of pericardial effusion by ultrasound promises to be a safe, accurate and convenient technique. It is based on the identification of an echo from the pericardium separate and distinct from the echo of the posterior heart wall. The quality of myocardial contraction is reflected in the pulsation of the posterior wall echo, providing evidence of any compromise secondary to the effusion (tamponade). The ultrasound and intravenous CO2 diagnoses of the presence of pericardial effusion are well correlated, but not the apparent volume of effusion. Possible explanations for this discrepancy are mentioned.

Proper positioning of the transducer and identification of a strong, definitely pulsatile echo from the posterior heart wall are essential for reliable interpretation and constitute the limitations of this technique.