Thoracic Complications of Amebic Abscess of the Liver

Report of 501 Cases

Carlos Ibarra-Pérez, M.D.

During an 18-year period, 501 cases of thoracic complications of amebic abscess of the liver were studied; 175 had inflammatory reactions of thoracic structures (165 with pleural effusions and pneumonitis, ten with pericarditis) and 326 ruptured through the diaphragm (175 into the airways, 106 into the pleural cavity, 5 into the pericardium, 39 into the airways and pleura, and 1 into the pleura and pericardium). The thoracic complication was preceded by a picture suggesting an acute inflammatory process or a chronic wasting disease. Depending on type, the complication itself was signaled by increase or change in character of right upper abdominal or lower thoracic pain, dyspnea, or overt respiratory insufficiency, hemoptysis, and expectoration of necrotic material, sepsis, tamponade, and shock. Chest roentgenograms showed small to massive pleural effusions, basal pneumonitis, and cardiomegaly; serology, liver scans, and induced pneumoperitoneum were diagnostic. Treatment included metronidazole and emetine, drainage of pleural or pericardial contents or promotion of bronchial drainage, and meticulous care of associated respiratory, circulatory, and systemic derangements. Mortality for cases with rupture was 11.4 percent, due mainly to sepsis, shock, respiratory insufficiency, and tamponade. The rest of the patients were discharged in cured or improved condition.

Amebic abscess of the liver (AAL) near or involving the diaphragmatic surface of the organ can induce inflammatory reactions of the diaphragm, the pleura, the pericardium, or the lungs. The abscess can also rupture through necrosis of the diaphragm and empty its contents into the pleural and pericardial cavities or into the airways; occasionally, the pleural cavity and the airways, or the pleural and pericardial cavities may be soiled simultaneously.

We report our experience in the diagnosis and management of the thoracic complications of AAL at a concentration unit for cardiac and pulmonary diseases, the Hospital de Cardiología y Neumología, Centro Médico Nacional, Instituto Mexicano del Seguro Social in México City during an 18-year period.

CLINICAL MATERIAL

Five hundred one cases of AAL with thoracic complications were studied from May 1961 to September 1979, representing all cases with thoracic complications seen at our hospital during this period (Table 1). Ages ranged from 4 to 84 years; 67 percent were male; 175 had inflammatory reactions of neighboring structures, 326 had rupture of AAL into the chest contents. Four percent of the complications were left-sided. Most of the cases were preceded by a clinical picture, diagnostic or at least very suggestive, of AAL. Its clinical course ran from three days to eight months before the onset of the thoracic complication.

Pleural and lung inflammatory involvement was suspected by pleural pain irradiated to the tip of the scapula, the shoulder, or the lower back. Cough was usually dry or productive of mucous sputum, and dyspnea was mild to moderate. Chest x-ray films showed small- to medium-sized effusions and sometimes associated patchy densities due to pneumonitis-atelectasis (Fig 1). Roentgenograms taken in the upright position after induced pneumoperitoneum with 500 to 750 ml of air showed adhesions between the upper surface of the liver and the diaphragm (Fig 2). If tapped, the fluid was clear, without amebas.

Pericarditis was more common with central or left-sided lesions; the clinical, ECG, and echocardiographic signs of

Table 1—Thoracic Complications of Amebic Abscess of the Liver, May 1961 to September 1979, 501 Cases

<table>
<thead>
<tr>
<th>Complication</th>
<th>No. of Cases</th>
</tr>
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<tbody>
<tr>
<td>Inflammation of contiguous structures</td>
<td></td>
</tr>
<tr>
<td>Pleura, lung</td>
<td>165</td>
</tr>
<tr>
<td>Pericardium</td>
<td>10</td>
</tr>
<tr>
<td>Rupture into contiguous structures</td>
<td></td>
</tr>
<tr>
<td>Airways</td>
<td>175</td>
</tr>
<tr>
<td>Pleural cavity</td>
<td>106</td>
</tr>
<tr>
<td>Pericardial cavity</td>
<td>5</td>
</tr>
<tr>
<td>Simultaneously into</td>
<td></td>
</tr>
<tr>
<td>Airways and pleural cavity</td>
<td>39</td>
</tr>
<tr>
<td>Pleural and pericardial cavities</td>
<td>1</td>
</tr>
</tbody>
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*From the Teaching and Investigation Department and the Division of Surgery, Hospital de Cardiología y Neumología, Centro Médico Nacional, Instituto Mexicano del Seguro Social, México City, México.
showed inflammatory titis and seen Rupture small in common of the lung field with mediastinal shift. Pleural fluid was purulent and showed amebas in less than 10 percent of cases.

Rupture into the pericardium was not necessarily immediately fatal. The patient could have had previous signs of pericarditis and aggravated his already critical condition by a clinical picture simulating heart failure, or had a cardiac tamponade. Both conditions could appear de novo and either could have led to death. Chest roentgenograms were similar to those of inflammatory reactions. The diagnosis of simultaneous rupture of AAL into the airways and pleural cavity was made when patients with bronchial drainage also had an empyema, with or without a spontaneous air-fluid level.

**General Principles of Treatment**

Dehydroemetine and metronidazole were the mainstay of drug therapy, both being used for ten days either singly or, preferably, in combination during the latter years. Chloroquine was given as an alternate drug or after a full course of dehydroemetine or metronidazole or both (Table 2).

Supportive treatment to correct malnutrition, hypovolemia, anemia, acid-base abnormalities, respiratory insufficiency, sepsis, and shock was mandatory. Many of these patients were seen in the intensive care unit.

Inflammatory complications usually responded to treatment of the responsible abscess. Large effusions were drained at the time of the diagnostic tap. In cases of pericarditis drainage of the pericardium and of the offending abscess was done.

Patients with rupture of AAL into the airways usually improved after this natural drainage, especially if promoted by postural drainage, physiotherapy, and endoscopy when necessary. Persistent bronchohepatic fistulae were better managed with second courses of drugs, singly or in combination. Resection of residual pulmonary lesions was indicated on an individual basis.

Amebic empyema was treated by immediate closed thoracostomy with large bore tubes (at least a size 30 French) and strong suction to induce prompt total expansion of the lung. Repeated pleural taps or use of small tubes usually resulted in chronic empyema requiring open thoracostomy or decortication or both.

For rupture into the pleural cavity, a diagnostic and therapeutic tap was followed immediately by drainage through the subxyphoid or intercostal routes, and digital curetting of the abscess through the diaphragmatic rent. The largest bore tube was connected to suction.

**Results**

Our 18-year cumulative mortality is a little more than 8 percent; for cases with inflammatory reactions, it has been 2.3 percent; for those rupturing...
FIGURE 2. Chest roentgenograms in cases of AAL with inflammatory thoracic complications. Posteroanterior view (a). Mild to moderate pleural effusion, elevation of diaphragm and slight mediastinal shift. Posteroanterior (b) of same patient, several days after drug therapy, after induction of diagnostic pneumoperitoneum. The effusion is now minimal; notice hepato-diaphragmatic adhesion; serology and liver scans were positive.

FIGURE 3. Chest roentgenogram in case of AAL with thoracic complication. Posteroanterior view of patient with "hourglass" abscess involving liver and right lower lobe; notice two air-fluid levels (arrows).

into the chest, the average is 11.4 percent, ranging from 5.2 percent for the least malignant, rupture into the airways, to 60 percent for those into the pericardium (Table 3).

Causes of death included sepsis, respiratory insufficiency, shock, massive aspiration of liver contents, tamponade, and pulmonary edema; in a few cases more than one cause was responsible for the patient's death. The rest of the patients were discharged as cured or improved.

### Table 2—Medical Treatment of Amebiasis by Amebicidal Drugs

<table>
<thead>
<tr>
<th>Drug</th>
<th>Dosage/Duration/Route</th>
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<tbody>
<tr>
<td>Dehydroemetine</td>
<td>1 mg/kg/day/10 days, parenteral</td>
</tr>
<tr>
<td>Metronidazole</td>
<td>2.4 g/day/10 days, oral</td>
</tr>
<tr>
<td>Chloroquine</td>
<td>500-750 mg/day/15-21 days, oral</td>
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### Table 3—Thoracic Complications of Amebic Abscess of the Liver, May 1961 to September 1979 (Mortality in 501 Cases)

<table>
<thead>
<tr>
<th>Complication</th>
<th>Incidence (%)</th>
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<tbody>
<tr>
<td>Inflammatory reactions*</td>
<td>4/167 (2.3)</td>
</tr>
<tr>
<td>Rupture into contiguous structures</td>
<td></td>
</tr>
<tr>
<td>Airways†</td>
<td>9/173 (5.2)</td>
</tr>
<tr>
<td>Pleural cavity</td>
<td>15/106 (14.2)</td>
</tr>
<tr>
<td>Pericardial cavity</td>
<td>3/5 (60.0)</td>
</tr>
<tr>
<td>Airways-pleural cavity</td>
<td>9/39 (23.0)</td>
</tr>
<tr>
<td>Pleural and pericardial cavities</td>
<td>1/1 (100.0)</td>
</tr>
<tr>
<td>Total in cases with rupture</td>
<td>37/324 (11.4)</td>
</tr>
<tr>
<td>Overall mortality</td>
<td>41/491 (8.3)</td>
</tr>
</tbody>
</table>

*Eight lost to follow-up
†2 lost to follow-up

### COMMENTS

In previous reports, considerations of pathogenesis, diagnosis, and treatment of several of the types of thoracic complications of AAL were made; others seem pertinent at this time.

In this era of mass movement of populations in and out of areas with poor sanitary conditions, more cases of AAL and its thoracic complications are being seen in countries where they were previously
uncommon, both in aliens and natives, in children and adults.

As classified by others, some of these complications are pooled together: pleural effusion and empyema, or are mentioned in descriptive terms at best: “consolidation,” “pneumonitis,” etc. The classification used here, although imperfect, is useful and sound. It signals a pathogenic mechanism and has therapeutic implications.

The diagnosis of AAL during the uncomplicated stage can be elusive. The clinical picture, usually clear-cut, may mimic other acute inflammatory or chronic wasting diseases of the gall bladder, kidney, pancreas, adrenal glands, pleura, lungs, or even of the same liver. Something similar occurs with the diagnosis of the thoracic complications, especially when left-sided. Several of our cases went undiagnosed until the post-mortem study. In this respect, induction of pneumoperitoneum is to be encouraged in cases with unusually high diaphragm, pleural effusions, and basal pneumonitis. If positive, serology for invasive amebiasis and scintillation and ultrasonic and computerized tomographic scans should leave no diagnostic doubt.

The symptoms of the thoracic complication usually were easily identifiable from those of the uncomplicated AAL; however, sometimes they seemed to mix, without apparent clue as to the onset of thoracic disease; still, in others, the complication seemed to appear without preceding disease.

The specific diagnosis of the inflammatory complications also depends on criteria other than the sensitivity of the procedures. For example, patients without effusion in the chest roentgenograms taken in the upright position, but with a pleural rub, do have a thoracic complication, but they are not included in this review. It remains to be seen how many of them show an effusion when studied by lateral decubitus roentgenograms or ultrasound scans of the thorax. In cases of uncomplicated AAL, the pain can be similar to that of cases with inflammatory complications, as there are intercostal nerve irrigations in both circumstances. Also, some cases of AAL may show pronounced elevation of the right dome of the diaphragm, simulating a large effusion.

In cases of rupture into the airways, both patient and physician may consider the products of bronchial drainage to be dark blood, but on closer inspection they prove to be chocolate-colored necrotic debris. Rupture into the airways may produce a lung abscess; a lung abscess also may be produced by progression of liver and diaphragmatic necrosis, resulting in an hourglass abscess. We have been unable to differentiate one from the other, and both types are classified together in the group draining into the airways (which they do). We have not seen hematogenous amebic abscess of the lung.

In cases with diaphragmatic elevation and basal pneumonitis, confusion between inflammatory pneumonitis and rupture of AAL into the airways may arise (Fig 4). Inspection of the sputum should

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**Figure 4. Chest roentgenograms in cases of AAL with thoracic complications. Posteroanterior films of two patients with small pleural effusions and basal pneumonitis. Patient in (a) has inflammatory reaction. Patient in (b) has rupture into right lower lobe.**
resolve the question.

The most important aspect of treatment of these complications is their prevention, achieved by a high index of suspicion, prompt diagnosis of uncomplicated AAL, adequate medical treatment, and drainage by needle aspiration in cases of impending rupture. Such cases include large abscesses producing persistent or increased sepsis and pain, nonresolving pleural effusions, or in central or left-sided lesions. If ominous signs persist despite orthodox medical therapy and needle aspiration, the abscess should be drained surgically.

Usually, good treatment is followed by immediate improvement; however, this is no guarantee against the presence of complications by inflammation or rupture, and clinicians and surgeons must be aware of this ever-present danger, particularly in cases of persistent sepsis and pain. If necessary, needle drainage of the liver can be done without the aid of ultrasound techniques at the point of maximal tenderness. In our experience, this maneuver has proved consistently successful. At the Hospital General, Centro Médico Nacional, the need for needle drainage of the liver has decreased from 17 percent to less than 2 percent of cases since the advent of metronidazole.19,20

Amebic “pus” from the liver, airways, pleural or pericardial cavities, frequently is chocolate-colored or like anchovy sauce, but color and consistency are variable. The absence of amebas in these products is no evidence against the diagnosis. In such cases, delaying drug treatment or drainage of the pleural or pericardial contents also can be fatal.

The mortality we noted is different from that cited by others,10,11,21 possibly owing to socioeconomic and nutritional differences in the affected population, invasiveness of the ameba, immunologic condition of the patient, arrival of cases at different stages of disease, dissimilar diagnostic perception by clinicians, percentage of necropsies performed, etc.

It is important to stress that this series of thoracic complications of AAL involves a highly heterogeneous biologic material, subjected to many variables influencing the results—amebicidal drugs, better management of fluids, electrolytes, blood and its components, hyperalimentation, and intensive care facilities, to mention just the obvious. Therefore, an analysis directed toward detecting the influence of a single factor is probably unwarranted and may even be misleading. In another hospital of our medical center for cases of uncomplicated acute AAL, emetine plus metronidazole 96 percent;19 however, for our cases, we know that drug treatment without proper drainage, or drainage without drugs, may prove to be regrettable; drugs and drainage without resolution of associated respiratory, cardiovascular, or nutritional problems may not be adequate for many patients.

Conclusions

Three sequential questions should be raised in every case of pathology of the lower chest, right or left, or of the pericardium: (1) Is there an AAL present? (2) Is there a thoracic complication of AAL? (3) If present, is the complication inflammatory or secondary to rupture of AAL into the chest? If these questions are raised and answered satisfactorily, earlier diagnosis and prompt therapy should improve the results of this series. Treatment must include the following: (1) amebicidal drugs, (2) needle drainage of the liver in cases with premonitory signs of rupture, central or left-sided lesions, or nonresolving pleural effusions, (3) in cases of ruptured AAL, drainage of pleural or pericardial contents, or promotion of bronchial drainage, and (4) meticulous care of associated respiratory, circulatory, and systemic derangements.

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European Academy of Allergology and Clinical Immunology

The annual meeting of the European Academy of Allergology and Clinical Immunology will be held in Clermont-Ferrand, France, September 24-26, 1981. For information, contact Prof Claude Molina, Hospital Sabourin, 63108 Clermont-Ferrand, France.

Topics in Pulmonary Disease

The 8th Annual Seminar, Topics in Pulmonary Disease, will be held at Colby College, Waterville, Maine, August 23-26, 1981. Direct inquiries to: R. H. Kany, Director, Special Programs, Colby College, Waterville, Maine 04901.