Articles

Amebic Liver Abscess: Epidemiology, Clinical Features, and Outcome

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Amebic liver abscess (ALA) is a serious, but readily treatable form of hepatic infection. In order to understand the clinical features of this condition in the United States, we reviewed the medical histories of 56 patients with ALA at two large San Francisco Hospitals from 1979 to 1994. Patients were divided into the following groups based on the presumed manner in which they had acquired ALA: those born or raised in the United States, with a history of travel to an endemic area (Tr-ALA); those from an endemic area, but living in the United States for less than one year (En-ALA); and those neither from nor having traveled to an endemic area (N-ALA). We found distinct clinical patterns in patients from different epidemiological groups. Patients with Tr-ALA were a decade older than those from endemic regions, were more likely to be male, and tended to have an insidious onset. Furthermore, compared to patients with En-ALA, those with Tr-ALA were more likely to have hepatomegaly ($P < 0.0001$) and large abscesses (ALA >10 cm; $P < 0.01$). One third of the patients studied had no associated travel history or endemic origin as risk factors. Of these, 63% had a condition consistent with severe immunosuppression, such as infection with the human immunodeficiency virus (HIV), malnourishment with severe hypoalbuminemia, or chronic infection. In patients with N-ALA, the presence of a presumed immunosuppressed state increased significantly, as compared to patients with endemic or travel risk factors for ALA. During the last five years of the study, one third of all patients diagnosed with ALA were HIV positive (including 2 with a new diagnosis of AIDS), many of whom were discovered to be HIV-infected only after presentation with ALA. We conclude that travel to and origin in an endemic area are important risk factors for the development of ALA, and patients in these different epidemiological groups appear to have distinct clinical features. Further, in the absence of recognized risk factors, the development of ALA may suggest an immunocompromised host.

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Infection with the parasite *Entamoeba histolytica* is common worldwide. Most patients infected with *Entamoeba histolytica* have only colonic symptoms and signs. Although less common than colonic amebiasis, extra-intestinal amebiasis is often more consequential. The most frequent form of extra-intestinal amebiasis is amebic liver abscess (ALA), which may develop serious complications if not rapidly detected and properly managed. With appropriate management, however, patients with ALA have an excellent prognosis.[1]

Although *Entamoeba histolytica* infection is most common in underdeveloped countries, immigration and the modernization of transport have contributed to an increased incidence and awareness of ALA in developed countries, such as the United States. Therefore, the aim of this study was to investigate the clinical features of patients with ALA in the United States. We report on a large cohort of patients with ALA and address both the epidemiologic profiles and clinical characteristics of patients with ALA in this setting.

Methods

Amebic liver abscess was identified in 56 patients between January 1, 1979, and December 31, 1994, at two hospitals affiliated with the University of California, San Francisco, Moffit-Long Hospital and San Francisco General. Each ALA was confirmed by radiological or surgical demonstration of an intrahepatic abscess cavity and by positive amebic serology. A presumptive diagnosis of ALA was made in two patients; amebic serology was not performed, but each had amebic stool cysts and a rapid clinical response to antiamebic treatment. In one patient, a histopathologic diagnosis of disseminated amebiasis

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TABLE 1.— *Epidemiological Features in 56 Patients with ALA* *

Features†	Endemic (En-ALA)	Travel (Tr-ALA)	No risk factors (N-ALA)
Number	27	13	16
Age, y†	28 (19–70)	40 (23–67)	36 (1–52)
Female:Male	5:22	0:13	2:14
Born in USA (%)	0 (0%)	3 (23)	13 (81)
Symptoms PTA, d‡	9 (1–120)	28 (2-180)	14 (3–60)
Symptoms > 1 mo (%)	8 (30%)	7 (54%)	2 (13%)
Hospitalization, d	7 (3–19)	8 (3–18)	8 (3–38)
Defervescence, d	2 (1–9)	3 (1–7)	3 (1–9)
Ethanol (%)§	11 (41)	9 (69)	4 (25)
HIV positive (%)	0 (0)	0 (0)	6 (38)#
Malnutrition (%)‖	2 (7)	3 (23)	2 (13)
Tuberculosis (%)	3 (11)	2 (15)	3 (19)
Immunosuppressed (%)¶	5 (19)	4 (31)	10 (63)**
Immunosuppressed and/or ethanol (%)	15 (56)	9 (69)	12 (75)

PTA = prior to admission

*Patients with En-ALA were born and had lived the majority of their lives in an endemic region and had been in the United States for less than one year. Patients with Tr-ALA were born in the USA or had lived the great majority of their lives in the USA and had traveled on one occasion to an endemic region. Patients with N-ALA were neither from nor had traveled to an endemic area.

†Numbers of patients in each group are shown, numbers in parentheses represent the percent of the total number in each specific subgroup (i.e. En-ALA, Tr-ALA and N-ALA), except where a range is shown.

‡Symptoms were any of those listed in Table 2.

§Heavy ethanol consumption was defined as > 150 grams ethanol/day.

‖Has evidence of wasting with severe hypoalbuminemia (<2.6 grams/dl) not due to any other cause (i.e. cardiac, liver, or renal failure).

¶An immunosuppressed condition was considered to be present in patients with HIV infection, infection with *Mycobacterium tuberculosis*, or with malnutrition and hypoalbuminemia (albumin <2.6 g/dL) not due to other causes.

#$P < 0.01$ compared to either En-ALA or Tr-ALA.

**$P < 0.001$ versus En-ALA group.

was made at autopsy. Specimens for parasitology were collected from fresh stools or drainage from interventional radiology or surgery. Patients with non-amebic etiologies for liver abscesses were excluded. Substantial alcohol consumption was considered to be present when ingested ethanol was >150 grams/d. A state of clinical immunosuppression was defined by the presence of either of the two following conditions: severe clinical malnutrition with muscle wasting and an albumin level of less than 2.6 gram/dl, demonstrated by physical and laboratory examination and having no other potential cause; or a serious, chronic, underlying infection, such as tuberculosis or HIV infection. Recurrence was defined as an ALA appearing subsequent to initial clinical or radiographic resolution.

We defined the following epidemiologic profiles: Endemic ALA (En-ALA) occurred in patients born and living the majority of their lives in an endemic region, who had been in the United States for less than one year. In this study, 16 patients came from Mexico, 7 from Asia, and 4 from South America. ALA occurring after travel (Tr-ALA) occurred in patients born or having lived the majority of their lives in the United States, who had a reported history of travel to a country where amebiasis is endemic. In this study, eight had travelled to Mexico, four to Asia, and one to South America. The final epidemiologic group was considered to be those neither from nor having traveled to an endemic area (N-ALA). Acute and chronic ALA were defined by the presence of symptoms for less than or more than 14 days, respectively. Statistical analyses were performed with either Fisher's exact test or the Student's *t* test. This study was approved by the Committee on Human Research of the University of California, San Francisco and was performed in accordance with the guidelines set forth in the Declaration of Helsinki[2] for ethical conduct in the study of human subjects.

Results

Epidemiology and Etiology

In this series, the incidence of ALA was 8.5 patients/100,000 hospital admissions. Of those diagnosed, 75% were young men born outside the United States, and only 18% of patients were older than 50. We segregated ALAs by etiology (Table 1). The median time to presentation after travel was 20 weeks, with a range of 2 to 60 weeks. One third of all patients with ALA had associated medical conditions. Eight had tuberculosis, three had syphilis, three had hepatitis C, three had peptic ulcer disease, one had diabetes mellitus, and one had had a posttraumatic splenectomy. In addition, 43% of the patients studied had a history of heavy alcohol consumption.

Patients with travel-related ALA were all men, many with histories of significant alcohol consumption. Tr-ALA patients were older than patients with En-ALA, and Tr-ALA presented subacutely in comparison with all other ALAs (Table 1). ALA was more prevalent in

TABLE 2.—*Symptoms and Signs at Presentation in 56 Patients with ALA*

Finding	*Number*	*%*
Symptoms		
Abdominal pain*	47	84
Fever	45	80
Chills	41	73
Nausea, vomiting and/or anorexia†	36	64
Sweats	32	57
Pleurisy, shortness of breath, and/or cough††	23	41
Diarrhea§	16	29
Weight loss‖	16	29
Signs		
Fever¶	47	84
Abdominal tenderness	45	80
Hepatomegaly (>14 cm)	14	25
Respiratory findings#	14	25
Guarding or rebound	11	20
Jaundice	2	4

*Pain was located in the following areas: right sided (31), epigastric (7), diffuse (4) or not stated (5). Pain was referred to the following areas: back or flank (9), shoulder (7) and chest wall (2).

†There were 23, 22, and 18 patients with nausea, anorexia and vomiting, respectively.

††There were 13, 8 and 8 patients with pleurisy, shortness of breath and cough, respectively.

§Bloody diarrhea was recorded in 5 patients.

‖Median weight loss was 20 lb (range 5-40 1b). There was a statistically significant difference for weight loss between ALA with an acute and chronic presentation (11% versus 60%, respectively) with $P < 0.0004$ (Fisher's exact test).

¶There were 31 high grade (>38.5°C) and 16 low grade (37.5<T<38.4°C) temperatures. Three initial temperature readings were not available. All patients afebrile on admission became febrile during admission.

#Respiratory findings included decreased breath sounds, dullness to percussion or added sounds.

men than women in all groups. In the N-ALA group, there were six homosexual male patients, three of whom also used intravenous drugs. Three of these patients were known to be HIV-infected prior to admission, and three were diagnosed after their clinical presentation with ALA. Of note, the five HIV-infected patients who presented between 1990 and 1994 accounted for one third of all ALA cases during that time period. Other conditions associated with immunosuppression included tuberculosis, severe malnutrition with severe hypoalbuminemia (<2.6 g/dl) in the absence of other causes, splenectomy and diabetes (Table 1). The presence of HIV was significantly greater in patients with N-ALA than in other ALA groups (versus En-ALA $P < 0.001$; versus Tr-ALA $P < 0.02$). Similarly, the presence of an immunosuppressed state in patients with N-ALA was also significant (versus En-ALA and Tr-ALA patients, $P < 0.01$).

Clinical Features

Symptoms were generally nonspecific (Table 2), but a large proportion of patients presented with fever and pain in the right upper quadrant; both symptoms were present in 72% of patients. Physical signs reflected symptoms (Table 2). All afebrile patients exhibited an elevation in temperature during admission. Notably, 83% of all fevers were high grade. Defervescence occurred rapidly in most patients (median 2.5 days, range 1–9 days), except HIV-infected patients, whose fevers lasted three times longer than other patients'. Of all patients with ALA, 39% had anorexia, and 29% had weight loss. Only five patients reported bloody diarrhea. Jaundice was extremely rare.

The median duration of symptoms prior to presentation was relatively short, although 30% of patients had symptoms for at least 30 days. The majority of patients with En-ALA (63%) and N-ALA (82%) had a short duration of symptoms prior to admission (<14 days), and 90% were discharged within 2 weeks of admission. By contrast, Tr-ALA patients had an indolent course with more than 50% having symptoms lasting over 1 month prior to presentation (Table 1). The presentation of patients with En-ALA was notable for the presence of fever and abdominal tenderness (93%). Nausea (48%), vomiting (44%), and pleurisy (30%) were most frequently observed in patients with En-ALA. Of N-ALA patients, 88% had a significant history of fever and chills, and 93% had high-grade temperatures during admission. Only two thirds, however, reported a history of abdominal pain. On admission, Tr-ALA patients were least likely to be febrile or exhibit abdominal tenderness, but were most likely to display constitutional symptoms (80% sweats, 31% myalgias, 23% headaches), hepatomegaly (69%) and respiratory findings (40%). Hepatomegaly was found four and ten times more often in patients with a travel history than in patients with N-ALA or En-ALA, respectively. Moreover, constitutional symptoms (sweats, myalgias, headaches, $P < .05$) and hepatomegaly were more common in Tr-ALA than En-ALA (69% versus 7%, $P < 0.0001$), although abdominal tenderness was less frequent (62% versus 93%, $P < .02$).

Laboratory Findings

The white blood cell count (WBC) was elevated in 70% of patients ($15 \pm 1 \times 10^9$/liter), and 10% had WBC $> 20 \times 10^9$/liter). Serum liver tests were generally unremarkable and were as follows (mean + SEM): albumin 3.4 + 0.2 grams/dl (normal >3.3); total bilirubin 1.0 + 0.1 mg/dl (normal <1.2); alanine transaminase (ALT) 52+7 U/liter (normal <40); aspartate transaminase (AST) 44 + 8 U/liter (normal <50); alkaline phosphatase (SAP) 159 + 12 U/liter (normal <115). No patient had a bilirubin level >3.2 mg/dl, though 20% of patients had levels >1.2 mg/dl. Two fifths of patients had hypoalbuminemia with none having levels <1.9 grams/dl. One third had an underlying anemia (12.8 ± 0.3 grams/dl), but severe anemia was uncommon.

Imaging

Chest and abdominal plain films were nonspecific (Table 3). The sensitivities of computed tomography (CT) and ultrasonography (US) in detecting ALA were 100% and 85%, respectively (Table 3). The five ALAs missed by US were confirmed by two repeat USs, one CT, one

TABLE 3.—*Initial Radiographic Imaging in 56 Patients with ALA**

Features	Number	%
Abnormal chest x-ray†	21	44
Abnormal abdominal x-ray††	17	52
Abnormal abdominal US§	47	90
<5.0 cm	14	25
5–10 cm	25	45
>10 cm	13	23
Abnormal abdominal CT scan	23	100

US = ultrasound and CT = computed tomography

*Available images included 48 chest X-rays, 33 plain abdominal films, 52 ultrasounds, 23 CT scans.

†There were 13 patients with a pleural effusion, 13 with elevated right hemidiaphragm, and 12 with atelectasis. Some studies had more than one abnormal finding.

††There were 9 patients with a sentinel loop, 6 with air fluid levels, five with hepatomegaly, and 1 with free air under the diaphragm. Some studies had more than one abnormal finding.

§If multiple ALAs were present, then the largest was measured.

TABLE 4.—*Location and Number of Amebic Abscesses*

Feature	Number	%
Location of ALA		
Right lobe	44	79
Left lobe	5	9
Both lobes (left, right, caudate)	5	9
Caudate	2	4
Number of ALA		
Single	43	77
Multiple	13	23

ALAs were identified by ultrasound, computed tomography or autopsy.

surgery, and one autopsy. Amebic liver abcessess were most often single, located on the right posterior or anterior segment, and <10 cm in size (Table 4). Amebic liver abscessess on the left side were distributed equally between the lateral and medial segments. Of patients with Tr-ALA, 55% had ALAs >10 cm in size. This group accounted for the greatest proportion of multiple ALAs (40%) and ALAs involving the left or both lobes (40%). Patients with Tr-ALA or a chronic presentation were more likely to have an ALA >10 cm (Tr-ALA, 55% versus En-ALA, 22%, $P < .01$; chronic presentation, 47% versus acute presentation, 12%, $P < 0.01$) (Table 4). All six ALAs in HIV-infected patients were right-sided; five were located in the posterior segment.

Microbiology

Amebic serology was performed using the following tests: indirect hemagglutination (IHA) in 37 patients, enzyme-linked immunoabsorbent assay (ELISA) in 15 patients, counterimmunoelectrophoresis (CIE) in 14 patients, latex fixation (LF) in 8 patients, immunodiffusion (ID) in two patients, and complement-fixation (CF) in 1 patient. Of the 77 tests, 5 were initially negative, including 2 IHAs, 2 CIEs, and 1 LF. In 13 out of 43 patients (30%), cultures of stool specimens revealed cysts or trophozoites, either singly or in combination (50%, 37% and 23% in Tr-ALA, En-ALA and N-ALA patients, respectively). Trophozoites were identified in 4% of abscess aspirates. Abscess cultures from two patients grew one contaminant and an *Escherichia coli*. Blood cultures from three patients grew two contaminants and an *Escherichia coli*.

Treatment

All patients received antibiotic treatment: 75% received a combination of metronidazole and diiodoquinol, and 25% received metronidazole alone. Almost half the patients underwent invasive diagnostic or therapeutic procedures, of which percutaneous needle aspiration was the most commonly performed. Of 20 patients who underwent aspiration, 1 developed acute respiratory distress (ARDS). Patients in the N-ALA group were most likely to undergo an invasive procedure (>60%). Defervescence took longer in patients who underwent invasive procedures than in those treated with antibiotics alone (Table 5).

Morbidity and Mortality

Complications included contiguous extension or rupture in 6 (11%) of patients, ARDS after an interventional radiological procedure in 1 (2%), recurrence in 2 (4%) and superinfection in 1 patient (2%). This latter patient, the only patient who died in this series, had *Escherichia coli* septicemia and multisystem organ failure with positive blood and abscess cultures. The diagnosis was not suspected prior to death and only made at autopsy. Patients with Tr-ALA (15%) and N-ALA (13%) were twice as likely as En-ALA (7%) patients to have a contiguous extension or rupture, but the latter had the only recorded death (4%). The recurrence rate was 0% for patients with N-ALA, 4% for patients with En-ALA, and 7% for patients with Tr-ALA. There was no consistent relationship between clinical improvement and a changed radiological appearance.

Discussion

We have identified distinct clinical features in patients with ALA who have different epidemiological profiles. Differences were found in groups of patients developing ALA after travel to an endemic area (Tr-ALA) and those with ALA after living in an endemic area (En-ALA). The former were older and more likely to be male and had a more insidious onset of illness, more marked hepatomegaly, larger ALAs, and a greater proportion of multiple ALAs, including those involving the left or both lobes. These differences in clinical presentation, found in our subgroups, are consistent with differences reported previously between subgroups of patients with either acute or chronic clinical

TABLE 5.—*Specific Treatment in 56 Patients with ALA**

Treatment	*Number (%)*	*Metronidazole (days)†*	*Time to defervesce (days)*
Antibiotics alone	31 (55)	11 (10–28)	2 (1–6)
Simple aspiration	12 (21)	13 (10–18)	4 (1–8)
Percutaneous drainage	8 (14)	15 (10–18)	4 (1–9)
Surgical drainage	5 (9)	17 (10–20)	3 (1–5)

*Median number of days are given with range in parentheses.
†Metronidazole (oral and/or intravenous) was the tissue active antibiotic used in all cases. It was used in combination with an antiluminicidal agent (most commonly a three-week course of Diidohydroxyquin) in 75% of ALA cases. The other 25% were treated with metronidazole alone.

presentations of ALA.[3] Hepatomegaly has also been found to be prevalent in patients who presented subacutely.[3] The longer duration of illness in patients with Tr-ALA may result in greater hepatomegaly and larger abscesses. Despite divergent epidemiological, clinical, and radiological features in patients with ALA in different settings, overall rates of complication, recurrence and mortality were low.

It has been previously proposed that those patients acquiring ALA as endemic residents are more likely to exhibit a chronic than an acute presentation.[3] According to this view, previous exposure and presumed immunization through infection modulate the immunological response, leading to a subacute illness in those with recurrent exposure to *Entamoeba histolytica*. Those with first-time exposure to *Entamoeba histolytica*, on the other hand, are thought to develop an "acute" clinical illness. Our findings do not support this postulate. We found that En-ALA patients had a more acute presentation with symptoms lasting for less than 14 days, while patients with Tr-ALA were most likely to have a chronic presentation, with symptoms lasting for over 1 month). Our patients and those reported earlier did, however, share notably similar clinical features in both "acute" and "chronic" presentations.[3] Indeed, patients with chronic presentation were older and more likely to have weight loss, respiratory findings, hepatomegaly and a positive initial US than patients with an acute presentation, although only differences for weight loss and ALA size were statistically significant. Our data suggest that regardless of epidemiological background, patients with ALA tend to exhibit acute or chronic clinical presentations. Additionally, our findings raise the possibility that "acute" ALA in the En-ALA group may represent a low-level, rapidly evident, and, perhaps, rapidly clearing infection, whereas "chronic" ALA in the Tr-ALA group may represent severe, persistent infection. One alternative postulate to explain the differences in presentations between the different epidemiological groups reported here and previously[3] (that is between the En-ALA and Tr-ALA groups), is that our patients and those reported previously could have been infected with different strains of *Entamoeba histolytica,* which could, in turn, be associated with divergent presentations. This postulate is consistent with a previously proposed hypothesis.[4]

Amebic liver abcess is a relatively uncommon complication of colonic amebiasis. Although the pathogenesis of ALA is not known, it is possible that an altered immune response to *Entamoeba histolytica* could contribute to the development of ALA. Alcohol consumption in particular is an important potential immunosuppressive factor, and it has been reported in up to 80% of patients with En-ALA.[5] Alcohol may make the liver more susceptible to *Entamoeba histolytica* infection by directly impairing Kupffer cell function in the liver or by hindering the cellular and humoral immunologic response to *Entamoeba histolytica*. In this series, over 40% of patients had a history of heavy alcohol consumption, and consumption was highest in patients with Tr-ALA (69%).

Experimental studies have shown impaired immunity to be an important factor in the pathogenesis of ALA.[6–8] Immunosuppression after thymectomy or splenectomy in animals results in an increased incidence of ALA.[6,7] Stimulation of the immune system with bacillus Calmette-GuErin (BCG), however, reduces the risk of developing ALA.[8] Furthermore, iatrogenic exacerbation of ALA in patients unsuspected of harboring ALA has been documented after the administration of corticosteroids.[9,10] In our series, of the 56 patients reported, 16 (29%) had not been placed at risk for ALA by either endemic origin or travel history (N-ALA). Furthermore, 10 of those 16 patients with N-ALA (63%) had an associated condition compatible with severe immunosuppression. These patients had significant medical conditions, such as HIV infection (38%), malnourishment with severe hypoalbuminemia (25%), chronic infections, namely tuberculosis and syphilis (25%), heavy alcohol use (25%), hepatitis (13%), or had undergone splenectomy (6%).

Together, our data and the experimental data raise the possibility that an immunocompromised state could be a risk factor for the development of ALA. It is also possible, however, that invasive amebiasis itself leads to immunosuppression.

Whether HIV infection is a risk factor for development of ALA is unknown. We identified six HIV-infected patients with ALA, two of whom had AIDS by standard criteria. All of the HIV-infected patients were born in the United States, and none had a history of

travel to an endemic area. All six were homosexual. Homosexual men are known to have an increased incidence of colonic amebiasis due to increased fecal-oral contact. During the 1970s, the incidence of amebiasis increased by 8000% in homosexual men in San Francisco[11] over a 10-year period, and 40% of patients with amebiasis in a New York hospital were homosexual.[12] Thus, a homosexual lifestyle appears to facilitate the transmission of *Entamoeba histolytica*. Whether HIV infection in conjunction with the enhanced transmission of *Entamoeba histolytica* increases the risk of ALA remains an open question requiring further study.

The relationship between HIV/AIDS and ALA may be important. In the last five years of our series, one third of all patients with ALA were HIV-infected, and it is notable that three of the six HIV-infected patients were found to be HIV-positive after the ALA was diagnosed (including 2 patients who were found to have AIDS by CD4 criteria). It has been proposed that there could be an increase in extra-intestinal amebiasis, including ALA, in HIV-infected patients due to the emergence of less virulent strains.[13] Although our data suggest that this may be possible, a population-based, epidemiological study comparing an HIV-infected population to a population with travel-related risk and to a population without risk is required. Furthermore, such a study could explore possible differences between strains of *Entamoeba*, such as *Entamoeba histolytica*, the "pathogenic" species, and *Entamoeba dispar*, the "nonpathogenic" species. This study could also investigate the conversion of nonpathogenic strains, presumable prevalent in many homosexual populations,[14] into pathogenic strains, which has been postulated to occur *in vitro*.[15] In any case, the presence of a space-occupying hepatic lesion in an HIV-infected patient should indicate the possible presence of ALA, and the patient should be managed accordingly.

In conclusion, travel to and origin in an endemic area are important risk factors for the development of ALA, and patients in each of these groups appear to have distinct clinical features. In addition, a subset comprising one third of patients in this series had no travel to or origin in an endemic area, but appeared to be susceptible to development of ALA. In this group, the majority had associated immunosuppressive conditions, of which HIV infection was the most common. These data raise the possibility that impaired host immunity may be an important factor in the pathogenesis of ALA. Moreover, from a clinical management perspective, the finding of an ALA in a patient without travel or endemic risk factors should suggest the possibility of a compromised immune system.

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REFERENCES

1. Nordestgaard AG, Stapleford L, Worthen N, Bongard FS, Klein SR. Contemporary management of amebic liver abscess. Am Surg 1992; 58:315–320

2. World Medical Association Declaration of Helsinki. Recommendations guiding physicians in biomedical research involving human subjects. JAMA 1997; 277:925–926

3. Katzenstein D, Rickerson V, Braude A. New concepts of amebic liver abscess derived from hepatic imaging, serodiagnosis, and hepatic enzymes in 67 consecutive cases from San Diego. Medicine 1982; 61:237–246

4. Reed SL. New concepts regarding the pathogenesis of amebiasis. Clin Infect Dis 1995; 21(Suppl 2):5182–5185

5. Gupta RK. Amebic liver abscess: a report of 100 cases. Int Surg 1984; 69:261–264

6. Ghadirian E, Meerovitch E. Effect of immunosuppression on the size and metastasis of amoebic liver abscesses in hamsters. Parasite Immunol 1981; 3:329–338

7. Ghadirian E, Meerovitch E. Effect of splenectomy on the size of amoebic liver abscesses and metastatic foci in hamsters. Infect Immun 1981; 31:571–573

8. Ghadirian E, Meerovitch E. Macrophage requirement for host defense against experimental hepatic amebiasis in the hamster. Parasite Immunol 1982; 4:219–225

9. Stuiver PC, Goud TJ. Corticosteroids and liver amoebiasis. Br Med J 1978; 2:394–395

10. Kanani SR, Knight R. Relapsing amoebic colitis of 12 year's standing exacerbated by corticosteroids. Br Med J 1969; 2:613-614

11. Pearce RB. Intestinal protozoal infections in AIDS. Lancet 1983; 2:51

12. Schmerin MJ, Gelston A, Jones TC. Amebiasis. An increasing problem among homosexuals in New York City. JAMA 1977; 238:1386–1387

13. Thompson JE Jr, Freischlag J, Thomas DS. Amebic liver abscess in a homosexual man. Sex Transm Dis 1983; 10:153–155

14. Weinke T, Friedrich-Janicke B, Hopp P, Janitschke K. Prevalence and clinical importance of *Entamoeba histolytica* in two high-risk groups: travelers returning from the tropics and male homosexuals. J Infect Dis 1990; 161:1029–1031

15. Mirelman D, Bracha R, Chayen A, Aust-Kettis A, Diamond LS. *Entamoeba histolytica*: effect of growth conditions and bacterial associates on isoenzyme patterns and virulence. Exp Parasitol 1986; 62:142–148