

The Diagnosis and Treatment of Pyogenic Liver Abscesses

BRENT W. MIEDEMA, M.D., PETER DINEEN, M.D., F.A.C.S.

Pyogenic liver abscesses in 106 adult patients at The New York Hospital were reviewed to define optimum treatment. Mortality in the surgically treated patients was 26% (17/65), while those treated nonsurgically had a fatality rate of 95% (39/41). Multiple abscesses treated surgically had a surprisingly low mortality of 29% (5/17). Modern noninvasive tests are highly sensitive in diagnosing liver lesions greater than 2 cm. Difficulty remains in identifying small hepatic abscesses and differentiating large abscesses from tumor. Most liver abscesses have an identifiable source outside the liver. The most common source (31%) was cholangitis secondary to extrahepatic biliary obstruction. Multiple abscesses, mixed organisms, hyperbilirubinemia, and abscess complications are all associated with a significantly increased mortality. However, the lethality of the primary disease process was the most important factor determining survival. Most patients who have the underlying pathogenesis of the abscess controlled will survive surgical treatment. Transperitoneal surgical drainage and antibiotics remain the mainstay of treatment. Percutaneous drainage is recommended for high risk patients only.

BEFORE THE TURN of the century, survival from a pyogenic liver abscess was rare.^{1,2} In 1934, Rothenberg and Linder reported a cure rate of 58% in the individual with a single pyogenic liver abscess.³ Improvement was due to advanced operative techniques and a better understanding of the pathophysiology of the pyogenic liver abscess. However, individuals with multiple liver abscesses still carried a 95–100% mortality, and overall mortality was a disappointing 77% as reported by Ochsner, DeBakey, and Murray in 1938.⁴ With the advent of antibiotics, improved survival was possible even with multiple hepatic abscesses.⁵ By 1968, most unselected series were reporting a mortality rate of 50–70% with liver abscesses.^{6–10}

Over the past 15 years, however, marked improvements in diagnosis, therapy and surgical management have not improved mortality.^{11–14} This poor survival has been attributed to neoplastic disease, multiple hepatic abscesses, and an older patient population. Current theories on how

From the Department of Surgery, The New York Hospital-Cornell Medical Center, New York, New York

to improve survival include earlier diagnosis, the use of percutaneous drainage under radiographic control, and more aggressive drainage. This review was undertaken to define the optimum management of the pyogenic hepatic abscess and to assess new trends in treatment.

Methods

All adults with a pyogenic liver abscess at The New York Hospital from 1945–1982 were reviewed. Hepatic abscesses were documented by autopsy, surgery, or needle aspiration. All abscesses had frank pus, a positive culture, or organisms identified microscopically. Hepatic abscesses diagnosed clinically or radiographically were excluded along with 11 pediatric patients.

Pathogenesis, signs and symptoms, laboratory data, diagnostic tests, treatment, pathology, bacteriology, complications, and outcome were analyzed. The pathogenesis was considered to be extrahepatic biliary disease if obstruction of the common bile duct was present or if cholangitis was documented concurrently with the liver abscess. The portal vein was implicated as the route of bacterial spread in all intra-abdominal infections within the portal system but remote from the liver abscess. The source of the hepatic abscess was considered to be general septicemia with bacterial entry *via* the hepatic artery, if the primary infection arose outside the portal system. No source of infection could be positively identified in the cryptogenic abscess.

The length of illness was defined as time from the first symptom attributable to the liver abscess to the time of definitive treatment. The delay in diagnosis was from the first visit to a physician to the time of definitive treatment. Bacterial data was compiled from the initial culture result only. The abscess was considered to be microscopic if it was less than 2 cm in greatest dimension. Mortality was defined as death within 30 days of treatment or before discharge from the hospital. Statistical analysis of the laboratory data was done by Student's t-test, while all other associations were studied by chi square tests. Data was

Presented at the 104th Annual Meeting of the American Surgical Association, Toronto, Canada, April 25–27, 1984.

Reprint requests: Dr. Peter Dineen, Department of Surgery, The New York Hospital-Cornell Medical Center, 525 East 68 Street, New York, NY 10021.

Submitted for publication: April 30, 1984.

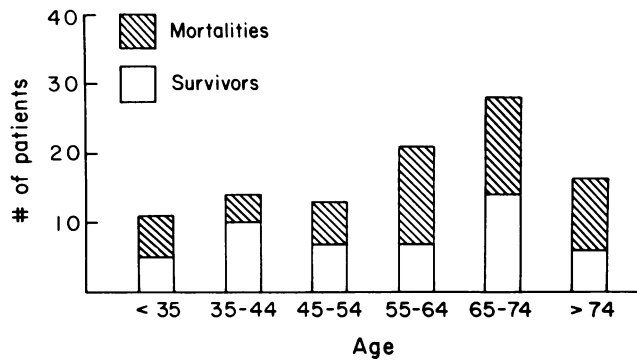


FIG. 1. Age distribution and mortality.

analyzed in three time periods (1945–1957, 1958–1970, and 1971–1982).

Results

Incidence. Pyogenic liver abscesses were identified in 106 adults and 11 children not included in this study. During this study, 1,186,286 patients were admitted to The New York Hospital for an incidence of 0.01%. Autopsies were done on 19,340 patients and 62 pyogenic liver abscesses were identified for an autopsy incidence of 0.32%. These incidences did not change significantly over time.

Age and Sex

The average age was 59 and ranged from 13 to 88. The average age increased from 55 to 60 over the study period. The age distribution and mortality rate are presented in Figure 1. Although those in the older age group had a higher mortality, this is not statistically significant ($p > 0.10$). More males (68) than females (38) developed liver abscesses ($p > 0.01$).

Pathogenesis

The pathogenesis of hepatic abscesses with the frequency and mortality rate can be seen in Table 1. Extrahepatic biliary tract disease was the most common source and carried a significantly worse prognosis ($p < 0.001$).

Cryptogenic liver abscesses were seen in 14 patients and all but one had a likely primary cause for the liver abscess. The pathogenesis of seven was probably portal seeding, three patients probably had a generalized septicemia, and three patients had extensive hepatic necrosis documented histologically. The cause of the hepatic abscess in the remaining patient was totally unknown. Eleven of the 14 cryptogenic abscesses have been seen since 1970, which is a significant ($p < 0.005$) increase in the recent time period.

TABLE 1. Mortality by Therapy and Pathogenesis

Source	Surgical		Medical		Total	
	No. Patients	Mortality (%)	No. Patients	Mortality (%)	No. Patients	Mortality (%)
Biliary	17	59	16	100	33	79
Portal drainage	15	7	2	100	17	18
Contiguous	12	8	5	100	17	35
Tumor	0	0	3	100	3	100
Trauma	1	0	1	0	2	0
Infected cyst	7	14	0	0	7	14
Cryptogenic	12	17	2	50	14	29
Sepsis	1	100	11	100	12	100
Vasculitis	0	0	1	100	1	100
Total	65	26	41	95	106	53

Clinical

The length of illness and the delay in diagnosis varied from 0 to 260 weeks. The average length of illness (6.4 weeks) and the average delay in diagnosis (4.3 weeks) have decreased only slightly over time (Fig. 2). The liver abscess was an indolent process in which 73% had the illness longer than 2 weeks, and 48% had the diagnosis delayed more than 2 weeks.

The symptoms and signs in this patient population are listed in Table 2. Abdominal tenderness and hepatomegaly were the most helpful signs in suggesting a liver abscess. Pulmonary changes were present in 35% of the patients, but the changes lateralized to the side of the pathology in only nine per cent. Twenty-three of the 28 clinically jaundiced patients had extrahepatic biliary disease.

Associated Diseases

Diabetes was present in eight, severe cardiac disease in 13, severe chronic obstructive pulmonary disease in

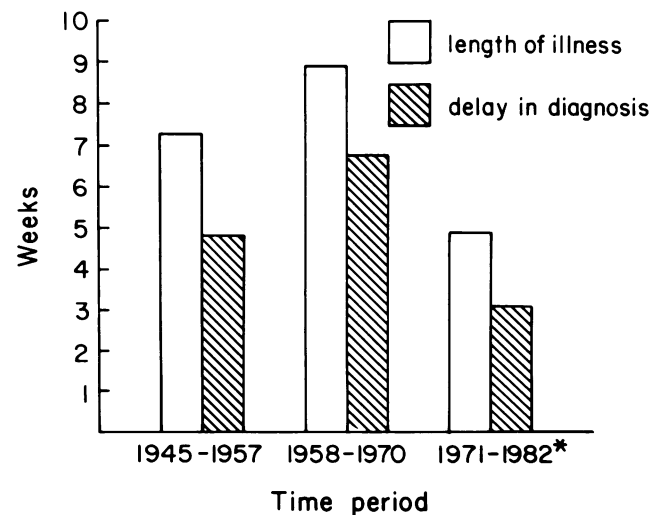


FIG. 2. Length of illness and delay in diagnosis. *Excludes one patient with undiagnosed illness for 260 weeks.

TABLE 2. *Symptoms and Signs*

Symptoms	%	Signs	%
Anorexia	97	Abdominal tenderness	71
Malaise	97	Right upper quadrant	65
Fever	93	Epigastric	15
Weight loss	76	Diffuse	8
Chills	44	Right flank	4
Vomiting	24	Other	8
Chest pain	16	Hepatomegaly	52
Cough	14	Pulmonary changes	35
Diarrhea	14	Jaundice	26
Night sweats	7	Splenomegaly	8

seven, and aplastic anemia in three patients. Thirty-three of the patients had a current or previously treated malignancy, of which 31 died.

Laboratory Data

The white blood cell count was elevated in most patients with an average of 16.2 k/ml. Hyperbilirubinemia is highly correlated with a decreased survival rate ($p < 0.001$). Hemoglobin and globulin are significantly lower in non-survivors ($p < 0.05$), while serum glutamic oxaloacetic transaminase (SGOT) is significantly higher in nonsurvivors ($p < 0.05$). The 5' nucleotidase and alkaline phosphatase were the most sensitive liver function tests, being abnormal in 88% and 73% of patients, respectively.

Diagnosis

The liver scintiscan (Tc^{88} sulfur colloid) was abnormal in 81% of patients tested, but had a 95% sensitivity for

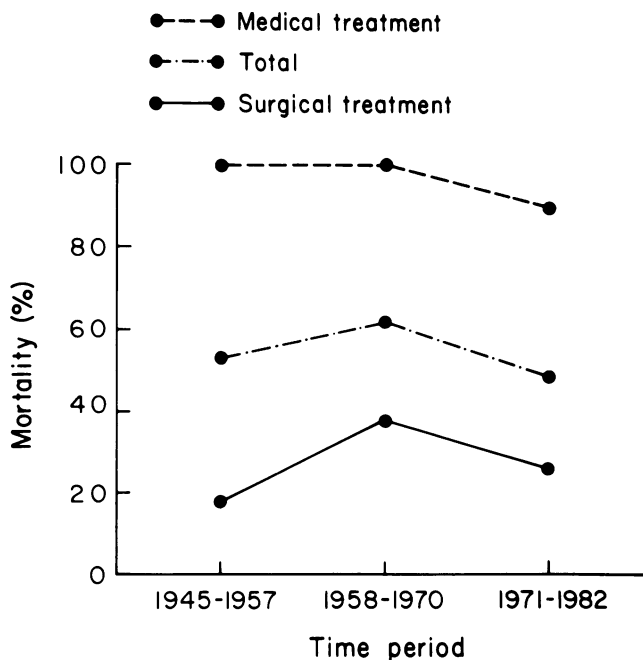


FIG. 3. Mortality rate in different time periods.

Thirty of 106 patients would have qualified for percutaneous drainage.*

Three died for a 10% mortality of these:

- 1) One fatality may have been preventable.
- 2) One patient needed surgery (cholelithiasis).
- 3) One death was not preventable (sclerosing cholangitis).

Thus: Percutaneous drainage would have had a minimal impact on mortality in this series.

*Contraindications to percutaneous drainage:

- Continuous source of bacterial seeding
- Another indication for surgery
- Vital structures near the drainage route
- Greater than two abscesses
- Ascites

FIG. 4. Percutaneous drainage of the hepatic abscess.

macroscopic abscesses of the liver. The hepatic sonogram had an 81% overall sensitivity and 85% for macroscopic hepatic abscesses. The computed transaxial tomography (CTT) and celiac arteriogram were 100% sensitive in the small number of patients studied.

Treatment and Mortality

The overall mortality rate of 53% has not changed significantly over time (Fig. 3). The mortality by therapy in different pathogenic groups is listed in Table 1. The 26% surgical mortality includes ten patients with extra-hepatic biliary disease. Excluding patients with biliary pathology, the surgical mortality was 15%. Patients with a solitary abscess treated surgically had a 25% mortality (12 of 48), while those with multiple hepatic abscesses treated surgically had a surprisingly low mortality rate of 29% (5 of 17).

Of the surgically treated patients, 55 had hepatotomy and drainage of the abscess with an accompanying liver biopsy. Other forms of surgical therapy included four major nonanatomic resections, one hepatic lobectomy, two needle aspirations, and three terminally ill patients had exploration only. Drains were placed in 60 patients, but the type of drain did not appear to affect the outcome.

Percutaneous drainage of the hepatic abscess would have been indicated in 30 patients in this series who were treated surgically (Fig. 4). Of the 48 single abscesses, 26 could have undergone percutaneous drainage by current criteria, but only four of 17 multiple hepatic abscesses could have been treated percutaneously. In this series percutaneous drainage was attempted in three patients and was successful in two. The failure was in a patient with a multiloculated liver abscess that required surgical drainage for definitive treatment. Only two patients treated nonsurgically survived, and both had percutaneous drainage of their hepatic abscesses. Thus, the medical therapy of patients without any form of drainage had a mortality of 100%. It should be pointed out that many

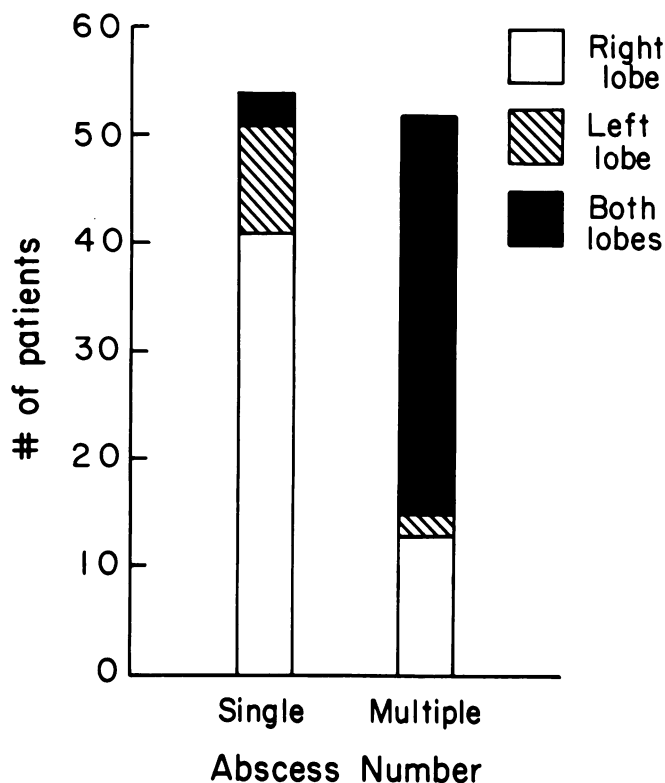


FIG. 5. Abscess location.

of these patients were elderly, moribund, or had hepatic abscesses discovered at autopsy.

Antibiotics were used in 95 patients. Broad spectrum antibiotics were usually used for 7 to 10 days. The antibiotic spectrum was narrowed when final culture results were available. Antibiotics were used an average of 4.1 weeks in those patients who survived.

Pathology

Patients with a single hepatic abscess had a 30% mortality. The abscess number and location is shown in Figure 5. The size of the single abscess was recorded in 47 patients and ranged in size from 30 to 4000 ml with an average of 446 ml. It is interesting that the average size of a single abscess increased from 228 cc from 1945-1957 to 597

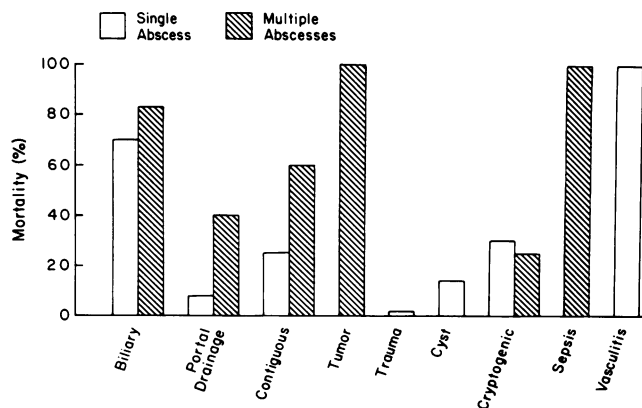


FIG. 6. Mortality with single and multiple abscesses.

cc from 1971-1983. Patients with multiple hepatic abscesses had a 72% mortality. This is a significantly worse survival ($p < 0.001$) than for patients with a single abscess. The mortality of multiple and single hepatic abscesses in different pathogenic groups is illustrated in Figure 6.

Bacteriology

Positive culture results were obtained in 71 patients and six cultures had no growth identified. A single organism was present in 32 (45%), while 39 (55%) of the cultures had two or more organisms. No culture was obtained in 29 patients; the majority of these had their hepatic abscess discovered at autopsy. Anaerobes were recovered in 16 patients and 50% of these patients survived. Gastrointestinal organisms were present in ten of the 11 cryptogenic abscesses where cultures were obtained. Patients whose hepatic abscess had a single organism had a 31% mortality rate, while patients with mixed organisms had a significantly ($p < 0.05$) worse mortality rate (59%). The culture results in the 77 patients who had cultures obtained are shown in Table 3.

Complications

The 60 patients with one or more complications following either medical or surgical therapy had a 73% mortality rate, which is significantly higher ($p < 0.001$) than

TABLE 3. Pyogenic Liver Abscess Organisms

Organism	Per Cent of Patients	Organism	Per Cent of Patients
<i>Escherichia coli</i>	36	<i>Bacteroides species</i>	8
<i>Klebsiella-enterobacter</i>	27	Sterile	8
<i>Enterococcus</i>	22	<i>Clostridia perfringens</i>	5
<i>Proteus species</i>	14	<i>Microaerophilic streptococcus</i>	5
<i>Streptococcus unclassified</i>	14	<i>Diphtheroids</i>	3
<i>Streptococcus viridans</i>	14	<i>Aeromonas hydrophilia</i>	1
<i>Pseudomonas aeruginosa</i>	10	<i>Citrobacter freundii</i>	1
<i>Staphylococcus aureus</i>	10	<i>Lactobacillus</i>	1
<i>Fusobacterium nucleatum</i>	9	<i>Providencia stuartii</i>	1
<i>Staphylococcus epidermidis</i>	9	<i>Serratia species</i>	1

TABLE 4. Major Complications and Their Frequency

Complication	Frequency
	(%)
Septicemia	33
Intra-abdominal abscess	9
Subphrenic	4
Subhepatic	2
Lesser sac	1
Pelvic	1
Mesenteric loop	1
Recurrent liver abscess	7
Liver failure	4
Renal failure	4
Massive UGI bleed	3
Myocardial infarction	2
Prolonged biliary drainage	2
Free peritonitis	1
Liver abscess hemorrhage	1
Other	13

the 25% fatality rate of those without a major complication. Septicemia was the most common complication and carried an 89% mortality rate (Table 4). Seven patients had recurrent liver abscesses and four subsequently died of this complication. Six of the liver complications recurred while the patient was on appropriate antibiotics.

Death Analysis

Death occurred in 56 patients with pyogenic liver abscesses and was probably inevitable for 44 of these patients even with optimal management. Twenty patients had advanced carcinoma, three had unreconstructable extrahepatic biliary disease, and four were elderly patients whom the primary physician elected to treat nonaggressively. The hepatic abscess was documented at autopsy in 17 of these 44 deaths.

The remaining 12 deaths may have been preventable. Three of these were in patients with peritonitis from a gastrointestinal perforation and died from inanition after a long hospital course. Modern total parenteral nutrition may have prevented those deaths. One patient died of an unrepaired biliary stricture, one of bleeding gastritis, and another of pneumonia. One patient early in the series had a liver abscess drained but the choledocholithiasis causing the abscess was not surgically corrected until 3 weeks later. Draining the common bile duct initially may have salvaged this individual. Three high risk patients died after prolonged hospital courses. One patient died from an acute episode while convalescing (no autopsy was done) and another patient died of progressive liver failure following a hepatic lobectomy.

Discussion

This series of 106 patients with pyogenic liver abscesses is the largest detailed analysis of this problem in the United States. Balasegaram has reported a series of 125 patients

from Malaysia where there is a higher incidence of hepatic abscesses.¹⁵ Although not universally fatal as it once was, pyogenic hepatic abscesses still carry a 50% fatality in most series.¹¹⁻¹⁴ The majority of liver abscesses have an underlying source that must be controlled before successful treatment of the abscess is possible. Most patients with hepatic abscesses can be cured with aggressive surgical and antibiotic therapy if the origin of the abscess is removed. Open surgical drainage has been the traditional treatment, but now percutaneous drainage is available because of newer radiologic techniques.

Survival has not improved significantly over the 38 years encompassed in this study (Fig. 3). Historically, liver abscesses developed in young, otherwise healthy, patients with an intra-abdominal infection. Ochsner reported a peak incidence in the fourth decade while the average age was 59 in this series.⁴ The sharp increase in the age of patients with hepatic abscesses probably is due to the aggressive treatment of acute appendicitis and other intra-abdominal infections of the young which prevents the abscesses from occurring. In Ochsner's series, 45% of abscesses were from suppuration in the portal vein bed (34% from appendicitis) while tumor and biliary causes were relegated to the miscellaneous category. In contrast, 31% of the patients in the current series had a malignancy. The recent change in pathogenesis to include more malignancies and complex biliary disease has resulted in an older age group and a greater proportion of poor risk patients with hepatic abscesses. Advances in the diagnosis and treatment of liver abscesses are being made. However, the severe underlying diseases in many patients with hepatic abscesses continue to limit successful treatment of this disease in many instances.

The incidence of pyogenic abscesses has remained constant in this century. The clinical incidence of 0.01% in this series is similar to the 0.008% reported by Ochsner in 1938.⁴ Recently, Rubin, Schwartz, and Malt reported an incidence of 0.016% at the Massachusetts General Hospital¹⁴ and Pitt and Zuidema reported a 0.013% incidence at the Johns Hopkins Hospital.¹¹ In this study, significantly more males were affected without an apparent reason from the data. Most studies show a male majority,^{4,13} but recent reports suggest a trend to an equal sex incidence.^{11,12} This study contradicts that view.

The source of the liver abscess greatly affects the subsequent mortality. Biliary tract disease, generalized sepsis, and metastatic tumor are associated with 73% of all deaths while they make up only 45% of the patient population. The poor survival in these groups is almost surely due to the multiplicity of the abscesses, and to the large proportion of patients who had an uncontrolled primary disease process. Extrahepatic biliary disease was the largest etiologic group in this series. The disappointingly high mortality (79%) has been seen by others as well.¹¹ Un-

fortunately, most of these deaths are not preventable with our present day therapeutic tools. Most had advanced carcinoma, or nonreconstructable biliary tract disease, and the hepatic abscess reflected an inability to deal adequately with the primary disease. A more aggressive approach to the diagnosis and treatment of biliary disease in general will salvage a small proportion of these patients.

The incidence of bacterial seeding of the liver from the portal circulation has decreased in recent years. This suggests that liver abscesses are prevented by the modern aggressive surgical approach to intra-abdominal infections such as acute appendicitis. The frequency of hepatic seeding through the portal circulation has decreased from 45% in 1938⁴ to 18% in the present series. More dramatic has been a change in the primary site of portal vein seeding. Appendicitis accounted for 77% of all infections in the portal bed before 1983,⁴ while since 1945 this has dropped to 12%. In this series all cases of acute appendicitis leading to a hepatic abscess occurred before 1957. Currently most causes of portal vein seeding leading to a liver abscess are from acute diverticulitis or from a post-operative intra-abdominal infection.

Regional enteritis leading to a liver abscess *via* the portal vein is of special interest. There have been only five reported cases of regional enteritis associated with a hepatic abscess,¹⁶⁻¹⁹ and two additional cases are added from this report. Both patients had a long history of regional enteritis with multiple explorations and bowel resections. The chronic infections present in many patients with regional enteritis would appear to place them at special risk for developing a hepatic abscess. One can only speculate on possible immune or mechanical factors that are protecting the patients with regional enteritis from developing a liver abscess.

With the large number of cryptogenic abscesses in this and other reports, a better understanding of the underlying pathogenesis is needed. The incidence has been reported from 4%¹⁴ to nearly 60%,^{4,20} but in recent series a fairly consistent 20% incidence is reported.¹¹⁻¹⁴ The incidence in this series of 13% is lower than most recent reports but correlates well with the 11% incidence reported by Sherman and Robbins in an autopsy study at the Mallory Institute of Pathology.²¹

Many investigators have tried to find a unifying pathogenesis for all cryptogenic hepatic abscesses but multiple causes appear to play a role.²² Our data is consistent with the observation of Beaver that most abscesses are secondary to an infection within the region of the portal drainage.²³ This may be from a healed focus or from a persistent underlying disease that was not identified in the diagnostic evaluation. The culture results also add validity to this thesis, since 10/11 cultures grew gastrointestinal flora. Three of our patients did have hepatic necrosis which corresponds to the theory of Lee and Block.²⁴

Rothenberg and Linder felt that a hematologic spread *via* the hepatic artery was the explanation of cryptogenic abscesses.³ This mechanism was apparent in three of the patients in this series.

Modern noninvasive diagnostic tests are very sensitive in diagnosing macroscopic abscesses of the liver. The liver scan was the most helpful screening test, but the sonogram gave more information when biliary tract disease was present. All diagnostic modalities were unreliable in identifying microscopic liver abscesses and only the celiac arteriogram allowed radiologists to differentiate multiloculated single abscesses from a liver tumor. Improvements in diagnostic techniques are needed to help define macroscopic hepatic lesions and to identify microscopic lesions. Recent limited experience with nuclear magnetic resonance suggests this technique will be of great value.

Surgical treatment continues to give the best chance of survival in patients with a pyogenic hepatic abscess. The surgical mortality of 26% is a marked improvement over the 69% surgical mortality that Ochsner, DeBakey, and Murray reported from 1928-1937.⁴ Especially gratifying results were seen in the surgical treatment of multiple hepatic abscesses where 12 of 17 patients survived. Historically, multiple hepatic abscesses have a 95-100% fatal outcome⁴ and recent series report a mortality from 77-88%.^{11,14,22} The high survival in this subgroup is due to aggressive surgical drainage, treatment of the underlying disease, and intravenous antibiotic treatment. The first cure in a patient with multiple hepatic abscesses attributed in part to antibiotics was reported by Ottenberg and Berck in 1938 using sulfonilamide.²⁵ In 1954, Glenn and Johnson reported three survivors from multiple pyogenic liver abscesses and collected 13 others from the literature.⁵ Today, most patients with multiple pyogenic abscesses can be salvaged if the underlying disease state is controlled.

Surgical drainage should generally be done transperitoneally to allow for abdominal exploration and to thoroughly explore the liver for multiple hepatic abscesses. It is difficult to evaluate what type of drain is best from the data. Dependent drainage with multiple drains consisting of large tube drains and soft rubber drains are recommended. The tube drains allow for diagnostic contrast studies and irrigation treatment after surgery. In this series the addition of a suction or sump drain did not enhance abscess cavity evacuation. In Figure 7, the principles of management of patients with liver abscesses are summarized.

Medical treatment alone without drainage was universally fatal in this series. The poor survival rate has been documented by other reports.^{4,6,11} Despite this, some authors continue to encourage the use of antibiotics alone to treat the pyogenic liver abscess.²⁶ This approach is risky and all hepatic abscesses should be drained to provide optimum treatment.

- Liberal use of noninvasive diagnostic tests
- Definition of abscess pathogenesis before surgery
- Celiac arteriography for suspected hepatic tumors
- Broad spectrum antibiotics before surgery
- Transperitoneal surgical exploration and correction of underlying disease
- Intraoperative atraumatic abscess location using thin needles
- Abscess sample for culture and gram stain with attention to anaerobic culture technique
- Rule out multiple abscesses
- Tube and soft rubber drains to exit in a direct and dependent manner
- Be alert for intra-abdominal infectious complications after surgery
- Attempt percutaneous transhepatic drainage in high risk patients

FIG. 7. Principles of hepatic abscess management.

Recently, percutaneous catheter drainage of the pyogenic hepatic abscess has been suggested as the treatment of choice.²⁷ A collected series of 65 patients with abscesses of the liver have been treated in this way with a 1.5% mortality.²⁸ However, contraindications to percutaneous drainage (Fig. 4) leave a highly selected patient population and should not be compared with a series describing a total experience. Surgical drainage has the advantage of allowing for a search for the source of the infection, a better chance to identify multiple hepatic abscesses, a more immediate and thorough evacuation of pus, and decreases the risk of uncontrolled peritoneal spillage. Percutaneous drainage of liver abscesses does hold promise for definitive therapy or to delay surgery in high risk patients that may not tolerate general anesthesia. However, a prospective randomized trial between patients drained surgically or percutaneously is needed to evaluate treatment differences in cost and morbidity.

References

1. Bryant JH. Suppurative pylephlebitis. *Guy's Hospital Reports* 1900; 54:77-112.
2. Gerster AG. On septic thrombosis of the roots of the portal vein in appendicitis, together with some remarks on "peritoneal sepsis." *Med Rec* 1903; 63:1005-1015.
3. Rothenberg RE, Linder W. The single pyogenic liver abscess. A study of 24 cases. *Surg Gynecol Obstet* 1934; 59:31-40.

DISCUSSION

DR. SEYMOUR I. SCHWARTZ (Rochester, New York): I appreciated the invitation to discuss this paper and the opportunity of reviewing this excellent manuscript.

The experience at our own institution agrees completely with that reported by Drs. Miedema and Dineen in regard to incidence, prevalent etiologic factors, bacteriology, and results. There are three specific issues that I would like to address.

The first relates to a subset of patients who present with fever of unknown origin, and I would suggest that hepatic abscess should be considered in this group. Of 21 such patients who came to laparotomy in our institution, three proved to have hepatic abscess. This was in the era prior to CT scanning and ultrasonography. One of the patients had

4. Ochsner A, DeBakey M, Murray S. Pyogenic abscess of the liver. II. An analysis of 47 cases with review of the literature. *Am J Surg* 1938; 40:292-319.
5. Johnson G Jr, Glenn F. Multiple liver abscesses following biliary tract surgery. *Ann Surg* 1954; 140:227-233.
6. Odgen WW, Hunter PR, Rives JB. Liver abscess. *Postgrad Med* 1961; 30:11-19.
7. Joseph WL, Kahn AM, Longmire WP. Pyogenic liver abscess. Changing patterns in approach. *Am J Surg* 1968; 115:63-68.
8. Block MA, Schuman BM, Eyley WR, et al. Surgery of liver abscesses. *Arch Surg* 1964; 88:602-610.
9. Butler TJ, McCarthy CF. Pyogenic liver abscess. *Gut* 1969; 10:389-399.
10. Cronin K. Pyogenic abscess of the liver. *Gut* 1961; 2:53-59.
11. Pitt HA, Zuidema GD. Factors influencing mortality in the treatment of pyogenic hepatic abscess. *Surg Gynec Obst* 1975; 140:228-234.
12. Lazarchick J, de Souza e Silva NA, Nichols DR, Washington JA II. Pyogenic liver abscess. *Mayo Clin Proc* 1973; 4:349-355.
13. Altmeier WA, Schowergert CD, Whiteley DH. Abscesses of the liver: surgical considerations. *Arch Surg* 1970; 101:258-266.
14. Rubin RH, Swartz MN, Malt R. Hepatic abscess: changes in clinical, bacteriologic and therapeutic aspects. *Am J Med* 1974; 57:601-610.
15. Balasegaram M. Management of hepatic abscess. *Curr Probl Surg* 1981; 18(5):282-340.
16. Snavelly JR. Clinicopathologic conference: diarrhea and abdominal tenderness. *Bull Tulane University Med Fac* 1946; 6:22-25.
17. Taylor FW. Regional enteritis complicated by pylephlebitis and multiple liver abscesses. *Am J Med* 1949; 7:838-840.
18. Lerman B, Garlock JH, Janowitz HD. Suppurative pylephlebitis with multiple liver abscesses complicating regional ileitis: review of literature 1940-1960. *Ann Surg* 1962; 155:441-448.
19. Sparberg M, Gottschalk A, Kirsner JB. Liver abscess complicating regional enteritis: report of two cases. *Gastroenterology* 1965; 49:548-551.
20. Bourne WA. The diagnosis of pyogenic liver abscess. *Lancet* 1954; 2:1093-1094.
21. Sherman JD, Robbins SL. Changing trends in the casuistics of hepatic abscess. *Am J Med* 1960; 28:943-950.
22. Brodine WN, Schwartz SI. Pyogenic hepatic abscess. *NY State J Med* 1973; 73:1657-1661.
23. Beaver DC. Granulomatous abscess of the liver of pyogenic origin. *Am J Path* 1931; 7:259-276.
24. Lee JF, Block GE. The changing clinical pattern of hepatic abscesses. *Arch Surg* 1972; 104:465-470.
25. Ottenberg R, Berck M. Sulfanilamide therapy for suppurative pylephlebitis and liver abscesses. *JAMA*; 111:1374-1375.
26. Maher JA Jr, Reynolds TB, Yellin AE. Successful medical treatment of pyogenic liver abscess. *Gastroenterology* 1979; 77:618-622.
27. Karlson KB, Martin EC, Frankuchen EI, et al. Percutaneous abscess drainage. *Surg Gynec Obst* 1982; 154:44-48.
28. Sheinfeld AM, Steiner AE, Rivkin LB, et al. Transcutaneous drainage of abscesses of the liver guided by computed tomography scan. *Surg Gynec Obstet* 1982; 155:662-666.

no previous operation and no primary source could be defined. In this patient, *Bacteroides* was cultured.

The two other patients had *Escherichia coli* as the etiologic bacterial agent, and they had undergone biliary surgery 2 and 4 years prior to this febrile episode.

The second point focuses on a point of disagreement in reference to the therapeutic use of interventional radiology. (Slide) This is a CT scan of a patient with a large central hepatic abscess. (Slide) This was drained by pigtail catheter percutaneously; the patient was discharged in 10 days.

At our institution we have managed 11 such patients with percutaneous drainage, now under ultrasonographic control. Two have required surgical intervention; the remaining nine have been discharged with a minimal hospital stay. Two of the patients had multiple abscesses, and in one patient four abscesses were drained by this approach.

I would, therefore, ask the authors, at this point in time, if they had