CHEST Postgraduate Education Corner

CONTEMPORARY REVIEWS IN CRITICAL CARE MEDICINE

Critical Management Decisions in Patients With Acute Liver Failure*

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Few admissions to the ICU present a greater clinical challenge than the patient with acute liver failure (ALF), the syndrome of abrupt loss of liver function in a previously unaffected individual. Although advances in the intensive care management of patients with ALF have improved survival, the prognosis of ALF remains poor, with a 33% mortality rate and a 25% liver transplant rate in the United States. ALF adversely affects nearly every organ system, with most deaths occurring from sepsis and subsequent multiorgan system failure, and cerebral edema, resulting in intracranial hypertension (ICH) and brainstem herniation. Unfortunately, the optimal management of ALF remains poorly defined, and practices are often based on local experience and case reports rather than on randomized, controlled clinical trials. The paramount question in any patient presenting with ALF remains defining an etiology, since specific antidotes can save lives and spare the liver. This article will consider recent advances in the assignment of an etiology, the administration of etiology-specific treatment to abate the liver injury, and the management of complications (*eg*, infection, cerebral edema, and the bleeding diathesis) in patients with ALF. New data on the administration of *N*-acetylcysteine to patients with non-acetaminophen ALF, the treatment of ICH, and assessment of the need for liver transplantation will also be presented.

(CHEST 2008; 134:1092–1102)

Key words: hepatitis; liver; liver failure

Abbreviations: ALF = acute liver failure; APACHE = acute physiology and chronic health evaluation; APAP = acetaminophen; CPP = cerebral perfusion pressure; FFP = fresh-frozen plasma; HTS = hypertonic saline; ICH = intracranial hypertension; ICP = intracranial pressure; INR = international normalized ratio; MELD = model for end-stage liver disease; MOSF = multiorgan system failure; NAC = *N*-acetylcysteine; OLT = orthotopic liver transplantation; PT = prothrombin time; rFVIIa = activated recombinant factor VIIa; SIRS = systemic inflammatory response syndrome

Acute liver failure (ALF) may be defined as the abrupt loss of liver function, characterized by hepatic encephalopathy and coagulopathy, within 26 weeks of the onset of symptoms (classically jaundice) in a patient without previous liver disease.[1] Many authorities[2] further subdivide ALF into hyperacute liver failure (jaundice-to-encephalopathy interval, ≤ 7 days), acute liver failure (jaundice-to-encephalopathy interval, 8 to 28 days), and subacute liver failure (jaundice-to-encephalopathy interval, > 28 days), since the tempo of its clinical evolution has important implications about etiology and outcome (Table 1). In general, patients with hyperacute liver failure have a more favorable rate of spontaneous survival (*ie*, survival without orthotopic liver transplantation [OLT]), are more likely to have ALF as a result of acetaminophen (APAP) overdose or acute hepatitis A, and are more likely to have cerebral edema develop.[2] In contrast, those patients with subacute liver failure have a dismal rate of spontaneous survival, are more likely to have liver injury due to idiosyncratic drug reactions or indeterminate etiol-

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The author has reported to the ACCP that no significant conflicts of interest exist with any companies/organizations whose products or services may be discussed in this article.

Manuscript received April 23, 2008; revision accepted July 18, 2008.

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DOI: 10.1378/chest.08-1071

Table 1—*Characteristics of ALF According to the Tempo of Evolution (Jaundice-to-Encephalopathy Interval)

Liver Failure Subcategory	Jaundice-to-Encephalopathy Interval	Common Etiologies	Spontaneous Survival, %	Clinical Presentation
Hyperacute	0–7 d	APAP, Hep A, ischemia ("shock liver")	80–90	Cerebral edema most common
Acute	8–28 d	Hep B, drugs	50–60	Cerebral edema less common
Subacute	5–26 wk	Drugs, indeterminate	15–20	Ascites, peripheral edema, renal failure

*Hep A = acute hepatitis A; Hep B = acute hepatitis B; drugs = idiosyncratic drug reactions; indeterminate = no etiology identifiable; spontaneous survival = survival without liver transplantation. It should be noted that these observations are generalizations based upon large population studies and do not apply to individual patients.[2–4,77]

ogy, and more frequently exhibit symptoms and signs of chronic liver failure, such as ascites and azotemia (Table 1).[2,3]

An attempt at defining the "optimal" management of ALF must begin with the following disclaimer: the management of ALF has largely defied systematic study. As alluded to above, ALF is a syndrome, not a disease, and may be precipitated by many insults to the liver (Fig 1), resulting in very different clinical courses and complications. Thus, studying a specific treatment in a homogeneous population of patients with ALF has been very difficult. Furthermore, the syndrome is rare, with an estimated incidence of 2,000 cases per year in the United States[4]; indeed, few of even the largest liver transplant centers care for > 10 cases a year. The need for multicenter trials spawned the founding of the US Acute Liver Failure Study Group in 1998, which was composed of 23 centers in an ongoing effort to study all aspects of ALF.[4] Finally, ALF generally has a poor prognosis without OLT (spontaneous survival rate, < 50%), the application of which interrupts its natural history, rendering the efficacy of a therapeutic maneuver difficult to interpret.

The current synopsis will highlight important practical developments in the management of patients with ALF including the accurate identification of etiology, the administration of agents to treat the liver injury, and the management of the three major complications of ALF (*ie*, infection, cerebral edema, and the bleeding diathesis). Specific details of all aspects of the management of patients with ALF are also available in a recent ALF Study Group consensus report.[5] The reader is also referred to two recent state-of-the-art treatises on artificial and bioartificial liver support devices,[6,7] which will not be discussed

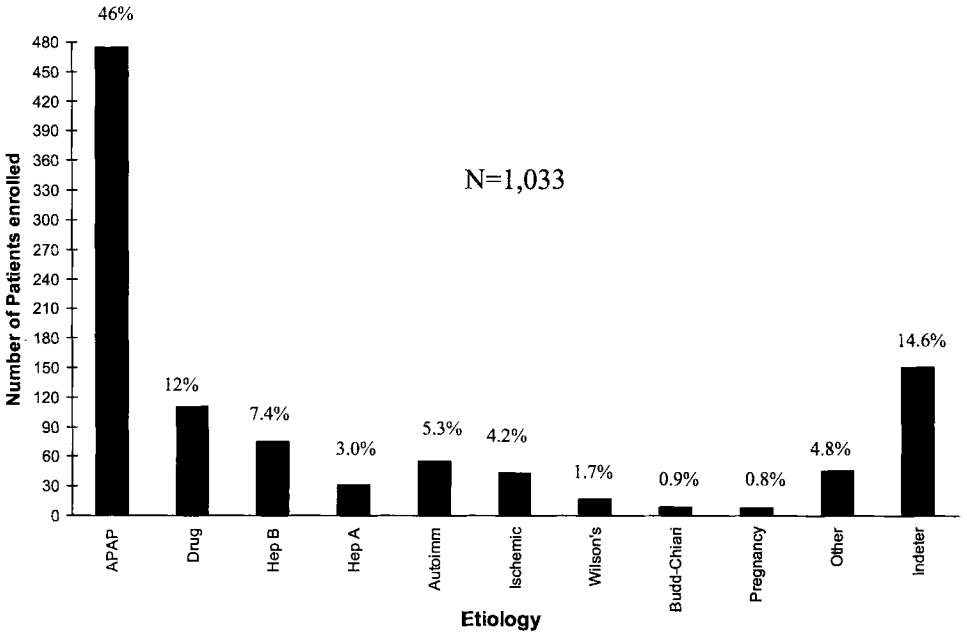

FIGURE 1. Etiologies of acute liver failure in the United States: data from the Acute Liver Failure Study Group Registry, from 1998 to 2007. Percentages of the total number of patients enrolled are shown above each etiology (unpublished data courtesy of W.M. Lee, Principal Investigator, the Acute Liver Failure Study Group). See Table 1 footnote for expansion of abbreviations.

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