

Chronic Hepatitis B and Hepatitis C in Asian Americans

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Both chronic hepatitis B and hepatitis C are prevalent among the 12 million Asians and Pacific Islanders living in the United States. Significant epidemiological and clinical differences exist between Asian Americans and the general U.S. population, most notably the higher rate of primary liver cancer and the differential response to various antiviral therapies. Perinatal and childhood transmission is common for hepatitis B virus. Transmission of hepatitis C virus probably also occurs early in life and results from nosocomial transmission and person-to-person spread. Asian patients with chronic hepatitis B commonly have hepatitis B e antigen-negative hepatitis B with either the precore or core-promoter mutant hepatitis B virus, which may require long-term antiviral therapy because of high rates of relapse following therapy. Asian patients with chronic hepatitis C may have a substantially higher risk of liver cancer but a better response to interferon-based therapy, both in terms of sustained virological response and reduced future incidence of liver cancer. Understanding these differences will lead to improved care for Asian Americans with viral hepatitis and better disease control for hepatitis B and hepatitis C for the entire U.S. population. This review briefly summarizes the major issues in the clinical care of patients with chronic viral hepatitis and focuses on pertinent epidemiological and clinical differences between Asian-American and Caucasian patients.

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Infection with the hepatitis B virus (HBV) and hepatitis C virus (HCV) is a major cause of morbidity and mortality in Asian patients worldwide. Common sequelae for both chronic hepatitis B and chronic hepatitis C include cirrhosis and hepatocellular carcinoma (HCC).^{1,2} Chronic hepatitis B has long been recognized as a major cause of morbidity and mortality in Asians. In contrast,

chronic hepatitis C in Asians is often underappreciated even though the prevalence of the disease in Asians is up to 3 times the overall U.S. prevalence.

According to the year 2000 census, there are approximately 11.9 million Asians and Pacific Islanders living in the United States.³ From 1990 to 2000 the Asian population grew at a faster rate than the total U.S. population did (48% vs 13%). Although Asians account for only 4.2% of the total population, over one half (51%) of the Asian population resides in only three states: California, New York, and Hawaii. The majority of the

is 8.0%–10.0% and of HCV infection is approximately 2.0%.^{4,5} The prevalence of HCV infection is especially high in Southeast Asia (5.6% in Thailand and 6.1% in Vietnam).⁶

In the United States, based on data derived from the second National Health and Nutrition Examination Survey (NHANES II), conducted between 1976 and 1980, the prevalences of past and/or present HBV infection is 3.2% for Caucasians and 13.7% for African Americans^{2,7}; the estimated total number of chronic HBV carriers is 1.25 million. The overall prevalence rate of HCV infection, derived from NHANES III,

of hepatitis B can also be illustrated by the high rate of infection in families who have adopted Asian children.⁹

The epidemiology of viral hepatitis in Asians differs significantly from that in the general U.S. population, not only in higher disease prevalences, but also in the mode and timing of acquisition of infection. Whereas many nonimmigrant U.S. patients acquire hepatitis B and C via use of injection drugs and high-risk sexual practices, most patients in Asia acquire hepatitis B either perinatally from infected mothers or from person-to-person contact during childhood. Similarly, they may acquire hepatitis C infection early in life from unsanitary medical practices or blood transfusions.^{9,10} Thus, Asians with hepatitis B and C usually acquire their infection at a young age; they are subsequently at much higher risk for long-term complications of these infections by the fourth or fifth decade of life—approximately 20 years earlier than other North American patients, who usually do not acquire their infections until the second or third decade of life.^{4,5}

It is estimated that 1.2 million, or 1 in 10, Asian Americans are chronically infected with hepatitis B virus.

remaining Asian population resides in just seven other states (Texas, New Jersey, Illinois, Washington, Florida, Virginia, and Massachusetts). Thus, Asians may represent a significant proportion of the patient population in the above-mentioned states.

Several aspects of the epidemiology, natural history, and responses to antiviral treatment of viral hepatitis in Asian American patients may differ substantially from those of non-Asian patients in the United States. Rather than give an exhaustive review of HBV or HCV infection in general, this article focuses on known and potential epidemiological and clinical differences between viral hepatitis in Asian Americans and the general U.S. population. Understanding these differences and their clinical implications may assist practitioners to care for Asian Americans with chronic viral hepatitis.

Epidemiology

According to the World Health Organization, in most of Asia the prevalence of chronic HBV infection

conducted between 1988 and 1994, is 1.8%⁸; the estimated total number of individuals with active HCV infections is 2.7 million. African Americans aged 30–49 years have the highest prevalence of HCV infection (6.1%), followed by Hispanics (3.4%), and Caucasians (2.9%), in the same age group.

The prevalences of chronic HBV and HCV infection in Asian Americans were not available from the above studies but can be expected to be significantly higher than those of Caucasians and closely resemble prevalence in their countries of origin.⁹ It is estimated that 1.2 million, or 1 in 10, Asian Americans are chronically infected with HBV. The highest prevalence (14.0%) was found in Vietnamese Americans and Laotian Americans. Of note, the rate of positive hepatitis B serological tests in families of patients with chronic hepatitis B is similar in Asians born in Asia and Asians born in the United States (44.0% vs 38.0%). The importance of familial transmission

Screening and Primary Prevention of Hepatitis B and C

The most effective and most important intervention to prevent HBV- or HCV-related end-stage liver disease and HCC is to prevent primary HBV and HCV infections. This includes the identification of infected patients and prevention of the spread of the disease. In Taiwan, universal vaccination against hepatitis B has proved successful in reducing the incidence of HCC, one of the most common causes of death among HBV-infected patients.^{11,12}

All high-risk patients should be encouraged to undergo serologic testing (Table 1),^{2,13,14} and infected patients should be offered appropriate therapy, follow-up, and counseling

Table 1
Screening for Hepatitis B Virus (HBV) and
Hepatitis C Virus (HCV) in High-Risk Patients

HBV	HCV
Persons born in hyperendemic areas	Anyone concerned with his/her HCV status
Men who have sex with men	Intravenous drug users
Injecting drug users	Persons receiving plasma-derived blood products prior to 1987
Dialysis patients	Persons receiving blood transfusion or solid organ transplantation prior to 1992
HIV-infected patients	Persons who have received chronic hemodialysis
Pregnant women	Persons with persistently elevated alanine aminotransferase
Family members of HBV-infected patients	
Household members of HBV-infected patients	
Sexual contacts with HBV-infected patients	

regarding lifestyle modifications and prevention of viral transmission. As blood transfusion and nosocomial spread are the major causes of HCV infection in Asia, Asian Americans born in HCV-endemic areas (eg, Southeast Asians) should probably also be offered testing for HCV infection.^{10,15}

In addition to safe medical and sexual practices, active immunization is available for HBV and should be recommended to all high-risk individuals (Table 2).¹⁶ Hepatitis B vaccines (Recombivax HB, Merck & Co., Inc., Whitehouse Station, NJ; or Engerix-B, GlaxoSmithKline Biologicals, Research Triangle Park, NC) can generally be given in three doses at 0, 1, and 6 months (doses vary depending on a patient's age and comorbidities, with higher doses in older, dialysis, and immunosuppressed patients).¹⁷ A combination HBV and hepatitis A virus (HAV) vaccine (Twinrix, GlaxoSmithKline Biologicals, Research Triangle Park, NC) is also available. Protective titers (≥ 10 mIU/mL HBV surface antibody

or anti-HBs) can be achieved in over 90% of healthy patients. Persons with continued exposure to HBV risk should, however, be tested for a

response to vaccination.²

Passive immunization with hepatitis B immunoglobulin (HBIG), a polyclonal hyperimmune antibody preparation, is indicated for infants born to hepatitis B surface antigen (HBsAg)-positive mothers (0.13 mL/kg within 12 hours of birth).¹³ A protection rate of 85%–95% is expected when given with an HBV vaccine.¹⁷ HBIG is also indicated after sexual, percutaneous, or mucous membrane exposure to HBV (0.05–0.07 mL/kg within 48 hours, or no longer than 7 days from exposures). Preventive vaccines against hepatitis C are not yet available.

The risks for HBV and HCV infection should be recognized, appropriate patients tested, and high-risk patients vaccinated against HBV. Current estimates of the hepatitis B vaccination rate among Asian-American children vary from 82% to less than 30%, according to reports from various geographic locations.^{18,19} More efforts should be made both by health care

Table 2
Recommendations for Vaccination for the Primary Prevention
of Hepatitis B Virus (HBV) Infection

High-risk individuals who should receive vaccination against HBV infection

- Household members of HBV-infected persons
- Sexual partners of HBV-infected persons
- Infants born to HBsAg-positive mothers
- All newborns
- Previously unvaccinated children and adolescents
- Health care workers
- Dialysis patients
- Recipients of clotting factor concentrates
- Persons institutionalized in correctional facilities or facilities for the developmentally disabled
- Travelers to endemic areas who stay for 6 months or longer

HBsAg, hepatitis B surface antigen.
Data from CDC.¹⁶

Table 3
Recommendations for the Treatment of Chronic Hepatitis B

ALT/Liver disease	HBeAg	HBV DNA	Treatment strategy
Normal ALT	-	-	No treatment required
Normal ALT	+	+	Observe and consider treatment when ALT is elevated
Elevated ALT*	-	+	Treatment with IFN- α or LAM or ADV
Elevated ALT*	+	+	Treatment with IFN- α or LAM or ADV
Cirrhosis	\pm	+	LAM or ADV (low-dose IFN may be used in case of compensated cirrhosis only, but this requires close monitoring for decompensation)

*Especially if greater than two times the upper normal limit.
ALT, alanine aminotransferase; HBeAg, hepatitis B e antigen; HBV, hepatitis B virus; IFN, interferon; LAM, lamivudine; ADV, adefovir dipivoxil.
Data from Nguyen MH, Wright TL,²⁰ Marcellin et al.,³⁰⁻³³ and Goodman et al.³⁴

providers and public health workers to improve prevention of viral hepatitis in high-risk populations such as Asian Americans.

Treatment of Chronic Hepatitis B and C

For chronically infected patients, the goal of antiviral therapy is to prevent progression to cirrhosis, liver failure, and HCC. Because complications may take many years to develop, most trials of treatment of patients with chronic HBV or HCV infection have used intermediate end points, such as inhibition of viral replication and improvement in liver histology, to evaluate efficacy. The treatment end points for chronic HBV infection are normalization of alanine aminotransferase (ALT) levels, loss of HBV DNA, loss of hepatitis B e antigen (HBeAg), with or without anti-HBe seroconversion, and improvement of liver histology.^{2,20} The treatment end points for chronic HCV are normalization of ALT levels; sustained virological response (SVR), defined as

loss of HCV RNA in serum using an assay with a sensitivity of at least 100 copies (50 IU) per mL at the end of treatment and 6 months afterwards; and improvement of liver histology.^{20,21}

Treatment options and expected therapeutic responses for the treatment of chronic HBV and HCV infection

Current estimates of the hepatitis B vaccination rate among Asian-American children vary from 82% to less than 30%, according to reports from various geographic locations.

have been reviewed elsewhere.^{2,13,17,20-29} The discussion below focuses on the salient aspects of therapy that require additional attention for Asian Americans.

Chronic Hepatitis B

Target treatment groups and available therapies for chronic HBV are summarized in Table 3 and Table 4.^{20,30-34} In one study, long-term treatment up to 60 weeks with adefovir dipivoxil was not associated with

resistance.³⁵ Adefovir was also effective in suppressing precore mutant HBV (HBeAg negative, HBV DNA > 10⁵ copies/mL) and was particularly useful in the treatment of lamivudine-resistant YMDD mutant HBV infection.³⁶⁻³⁹ Some studies suggest that peginterferon or interferon/lamivudine combination therapy may have added benefits, especially for patient subgroups with more difficult-to-treat disease,^{32,40,41} but more studies are needed in this area.

Compared to Western patients, Asian patients with elevated ALT levels and HBeAg-positive chronic hepatitis B show no significant differences in response to interferon- α (IFN- α) and lamivudine.² However, most Asian patients acquire their infection in the perinatal period, and these patients typically have low or normal ALT levels, which predict a lower response to IFN- α .¹³ High pretreatment HBV DNA levels (> 200 pg/mL) and long duration of infection are other factors commonly seen in Asian patients that are also predictors for poorer treatment response. These patients, therefore, are often not candidates for IFN therapy.

Furthermore, unlike the majority of Northern European and U.S. patients, many Asian and Southern European patients have HBeAg-negative chronic hepatitis B.¹³ Whereas both IFN- α and lamivudine are effective in suppressing HBV DNA levels in this precore mutant HBV infection, relapse rates are very high when treatment is discontinued (approximately 50% for IFN- α and 90% for lamivudine).² Therefore, long-term therapy with either lamivu-

Table 4
Rates of Response in Clinical Trials in the Treatment of
Hepatitis B e Antigen (HBeAg)-positive Chronic Hepatitis B

Response	IFN- α 12–24 weeks, % (placebo)	LAM 1 year, % (placebo)	LAM 2 years, % (placebo)	LAM 3 years, % (placebo)	LAM 4 years, % (placebo)	ADV 48 weeks, % (placebo)	ADV 72 weeks, % (placebo)
HBV DNA loss	37* (17) [§]	17–32* (6–11) [§]	50*	66 20 [†]	–	21 (0) [§]	21 [‡]
HBeAg seroconversion	33 (12) [§]	16–18 (4–6) [§]	27	27	47	12 (6) [§]	17
HBsAg loss	7.8 (1.8) [§]	< 1 (0) [§]	–	–	–	–	–
YMDD mutation	NA	14–32 (0) [§]	38	57	67	0	–
Biochemical improvement	28	72 (24) [§]	50	49	64	48 (16) [§]	21 [‡]
Histological improvement	–	49–56 (23–25) [§]	–	69	–	53 (25) [§]	–

*HBV DNA measured by hybridization assay (lower limit = 1.6 pg/mL).

[†]HBV DNA measured by PCR assay (lower limit = 1000 copies/mL).

[‡]Indicates response among nonresponders at 48 weeks.

[§]Corresponding values in placebo controls.

NA, not applicable; IFN- α , interferon- α ; LAM, lamivudine; ADV, adefovir dipivoxil; HBsAG, hepatitis B surface antigen; PCR, Polymerase chain reaction.

dine or adefovir is usually required for the majority of these patients.

Chronic Hepatitis C

Combination therapy with peginterferon and ribavirin is currently the standard of care for eligible patients with chronic hepatitis C. The strongest predictors for treatment response are viral genotype other than genotype 1, viral load < 2 million copies/mL, and absence of advanced fibrosis. The expected SVR is approximately 42% for genotype 1 and 75% for genotypes 2 and 3.^{22,23} The SVR is also associated with improved histology and a lower incidence of HCC on follow-up.

Race has been shown to influence both the histological progression and the response to interferon in patients with chronic hepatitis C.^{42–45} African Americans with chronic hepatitis C have a significantly slower histolog-

ical progression rate compared to the rates for Caucasians and Hispanics. At the same time, the response to IFN-based therapy is significantly poorer in African Americans than it is in Caucasians, based on the results of randomized controlled trials.^{45,46}

Only one of these trials, however, included Asian Americans, and in this trial only 10 patients out of a total of 472 study patients were Asian Americans.⁴⁵ Studies from Asia, especially those from Japan, generally report a higher response to IFN (up to 50%). In many of these studies, high doses of IFN were used (eg, 1 group was given 10 million international units 6 times a week for 2 weeks then 3 times a week for 12 additional weeks).^{47,48} Only a few small, randomized, controlled trials of IFN/ribavirin combination therapy have been carried out in Asia. Thus, data on treatment responses from large,

randomized, controlled trials using IFN/ribavirin combination therapy is lacking in Asian patients with chronic hepatitis C.

A recent nonrandomized study done in Australia of Southeast Asian patients with chronic hepatitis C reported a higher response to combination IFN/ribavirin therapy than the response of Caucasian patients.⁴⁹ In this study, all patients received 1000–1200 mg ribavirin, and all underwent an induction period of 4–8 weeks with daily high-dose IFN (5 million units subcutaneously). Among patients with genotype 1b, the SVR was 59% in Southeast Asians compared to 34% in Caucasians. In addition, many Southeast-Asian patients with genotype 7, 8, or 9 were initially mistyped as genotype 1b by a commonly used line probe assay (INNO-LiPA HCV II; Innogenetics, Ghent, Belgium).

Sequence analysis using the ABI PRISM® Dye Terminator Cycle Sequencing Ready Reaction Kit (Applied Biosystems, Foster City, CA) subsequently led to the identification of the correct genotype. Asian patients with genotype 7, 8, or 9 had a much higher response (79%) to the above combination therapy compared to those with genotype 1b (59%). According to the results of this study,

years).⁵¹ A greater reduction in risk may partly result from the higher HCC incidence and higher prevalences of the more responsive genotypes (ie, genotypes 2 and 3) in Asians.

IFN given after hepatic resection or local tumor ablation with ethanol or acetic acid may also significantly decrease the risk of recurrence, according to three randomized, controlled trials from Japan.⁵²⁻⁵⁴ The ben-

liver cancer reported to the California Cancer Registry for 1988–1992 was 49.5 per 100,000 among Vietnamese Americans compared to the overall California rate for 1988–1992 of 4.8 per 100,000, and the U.S. rate for 1985–1989 of 3.9 per 100,000.⁶⁰ This high incidence of liver cancer is probably the result of the high prevalence of hepatitis B as well as hepatitis C among Vietnamese Americans. HBV-infected patients, with or without cirrhosis, have a high risk for HCC. Compared to the relative risk for HCC among HBsAg-negative Asian patients, the relative risk among HBsAg-positive/HBeAg-negative patients is 9.6 and among HBsAg-positive/HBeAg-positive Asian patients is 60.2.⁶¹ Although the risk for HCC in HCV-infected patients without cirrhosis is minimal, the annual incidence of HCC in patients with cirrhosis is up to 8.0% in Asians (vs 3.0% in non-Asians).

The higher HCC prevalence rates in Asians may also result from other causes, such as environmental factors (aflatoxin in HBV cases),⁶² occult co-infection with hepatitis B (in HCV cases), long duration of infection, and potential race-related, genetic factors. The estimated risk for HCC was approximately tripled in Asian Americans compared to Caucasians with chronic hepatitis C and cirrhosis in a sample of over 400 patients in the San Francisco Bay area.⁶³ HBsAg-positive patients were excluded from this case-controlled study, and HCC risk estimates were adjusted for age, gender, and severity of liver disease.

Because they have such a high risk for HCC, Asian Americans with chronic viral hepatitis should undergo especially close surveillance for HCC. Although there are no randomized, controlled trials comparing HCC screening versus no screening in patients with chronic liver disease, several observational studies suggest

Although the risk for hepatocellular carcinoma (HCC) in HCV-infected patients without cirrhosis is minimal, the annual incidence of HCC in patients with cirrhosis is up to 8.0% in Asians (vs 3.0% in non-Asians).

Asians probably respond better to IFN-based therapy than Caucasians do, and accurate genotyping is essential in this patient group because of the high prevalence of novel genotypes and differential response rates in those with these unique genotypes.

Regarding the profile of treatment side effects, ribavirin-induced hemolytic anemia may be more common in Asians than it is in Western patients.⁵⁰ In a meta-analysis of 17 studies (N = 3520), the risk of ribavirin-induced hemolytic anemia was higher in the two Asian studies (n = 87; ribavirin 800–1200 mg daily) compared to the pooled risk for the non-Asian studies. A potential explanation for this observation may be the lower body-mass index in Asians. The number of Asian patients in this meta-analysis was, however, few and limits its conclusions.

Another notable difference in Asians compared to Western patients is the long-term effect of IFN therapy on the development of HCC. A 21.6% reduction in the risk of the incidence of HCC was seen in Asian patients treated with IFN-based therapy for chronic HCV compared to an only 10.0% reduction in non-Asian patients (approximate follow-up time was 4–5

years).⁵¹ A greater reduction in risk may partly result from the higher HCC incidence and higher prevalences of the more responsive genotypes (ie, genotypes 2 and 3) in Asians.

Treatment of End-Stage Liver Disease

The late results of chronic viral hepatitis are end-stage liver disease and HCC. Although a comprehensive review of the various complications of end-stage liver disease is beyond the scope of this article, the following issues warrant a separate discussion: measures of preventive care, chemoprophylaxis for latent tuberculosis, and outcomes of liver transplantation in Asian Americans.

Preventive Care with Screening for Hepatocellular Carcinoma

Aside from preventive care appropriate for age, additional preventive therapy for various known complications should be considered in patients with chronic liver disease, as outlined in Table 5.⁵⁵⁻⁵⁹ Among these, screening for HCC is probably one of the most important interventions. The annual, age-adjusted rate of the incidence of

Table 5
Preventive Care in Patients with Liver Disease*

Prevention of:	Indications	Specific prophylactic therapy
Hepatitis A superinfection	Chronic liver disease without evidence of hepatitis A immunity	Hepatitis A vaccine
Bleeding esophageal varices/gastric varices (EV/GV)	Large EV without a history of bleeding	Propranolol or nadolol
Bleeding EV/GV	History of bleeding varices	Esophageal band ligation + varices propranolol or nadolol TIPS for GV or refractory cases of bleeding from EV
Spontaneous bacterial peritonitis (SBP)	History of SBP Significant ascites with ascitic total protein < 1 g/L or active/recent gastrointestinal bleeding (GIB)	Ciprofloxacin 750 mg weekly or norfloxacin 400 mg bid for 7 days in case of GIB
Hepatocellular carcinoma	Cirrhosis of any cause Hepatitis B virus carriers or chronic hepatitis B patients aged 35 or older	Serum alpha-fetoprotein plus liver ultrasound every 6 months

*In addition to other preventive care appropriate for age.

TIPS, transjugular intrahepatic portosystemic shunt.

Data from Centers for Disease Control,⁵⁵ D'Amico et al.,⁵⁶ de Franchis and Primignani,⁵⁷ Rimola et al.,⁵⁸ and Nguyen and Keeffe.⁵⁹

that preclinical HCC cases detected via screening are more likely to be eligible for therapy and/or be associated with improved survival.^{59,64-68} Long-term survival can be expected if patients are eligible for curative treat-

Midwest, the rate of significant hepatotoxic side effects requiring discontinuation of isoniazid (INH) chemoprevention was 62% among HBeAg-positive patients compared to only 19% of those without HBeAg.

Long-term survival can be expected if patients are eligible for curative treatments such as hepatic resection or liver transplantation.

ments such as hepatic resection or liver transplantation.⁶⁹ Palliative treatments, such as transcatheter chemoembolization, have also been shown to improve survival at 2 and 3 years.⁷⁰⁻⁷³

Isoniazid Treatment in Patients with Hepatitis B Infection

The high prevalence of latent tuberculosis among Southeast Asian immigrants, including those with HBV infection (53%), merits attention.^{74,75} In a large cohort case study of Vietnamese immigrants in the

No significant differences were seen between HBsAg-positive and HBsAg-negative patients.⁷⁴ Thus, Southeast Asians with latent tuberculosis should be tested for HBV status, including HBeAg. Those with HBeAg positivity should be monitored closely for evidence of significant INH-induced hepatotoxicity.

Liver Transplantation in Asian Americans

The rate of posttransplant HBV infection is 20%–35% with HBIG and

less than 10% with HBIG and lamivudine.^{13,76} Anti-HBs may also develop spontaneously in patients receiving lamivudine prophylaxis (without HBIG) following liver transplantation for chronic HBV infection.⁷⁷ Asian race is not an independent predictor for poorer 2-year and 5-year survival in two single-center and one multi-center outcome studies of liver transplantation for chronic hepatitis B.⁷⁸⁻⁸⁰ However, 1-year mortality resulting from the recurrence of HBV may be higher in Asian-American patients.⁷⁸ Late referral and more advanced chronic liver disease at the time of transplantation may account for the higher early mortality in Asian Americans.

The recurrence of HCV posttransplantation is universal.¹³ Nonrandomized studies of IFN plus ribavirin or peginterferon plus or minus ribavirin suggest that SVR could be achieved in approximately 20% of treated patients.⁸¹ In addition, patients who

achieved viral eradication had no significant progression of fibrosis during therapy.⁸² A range of approximately 50%–72% of treated patients, however, required dose adjustments because of leukopenia and cytopenia.^{81,82} There are no known differential survival or HCV recurrence rates among Asians and non-Asians following liver transplantation for HCV.

One of the most recent and largest cohort studies of all liver transplantations performed in the United States between 1988 and 1996 (total, N = 14,282; Asian Americans, n = 416, or 2.9%) reported significantly lower overall 2-year and 5-year survival rates in African Americans (74% and 48%) and Asian Americans (69% and 37%), compared to Caucasians (83% and 58%).⁸³ These results are intriguing and conflict with results of other studies of liver transplantation for HBV in Asian Americans. The etiologies of liver disease leading to end-stage liver disease and transplantation were not looked at in this study. These observations warrant further examination. ■

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Main Points

- Chronic hepatitis B has long been recognized as a major cause of morbidity and mortality in Asians. Chronic hepatitis C in Asians, however, is often underappreciated, although the prevalence rate of the disease in Asians is up to 3 times the overall U.S. prevalence rate.
- Asians with hepatitis B and C usually acquire their infection at a young age—through perinatal and childhood transmission for hepatitis B, and nosocomial or person-to-person spread for hepatitis C; Asians are subsequently at much higher risk for long-term complications.
- Asian Americans have a higher rate of primary liver cancer and a differential response to various antiviral therapies than the general U.S. population.
- Asian patients with chronic hepatitis B commonly have hepatitis B e antigen-negative, with either the precore or core-promoter mutant hepatitis B virus; this may require long-term antiviral viral therapy because of high rates of relapse following therapy.
- Asian patients with chronic hepatitis C may have a substantially higher risk of liver cancer but a better sustained virological response to interferon-based therapy, and, subsequently, a reduced future incidence of liver cancer.

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