Louisiana

DEPARTMENT OF HEALTH AND HUMAN RESOURCES
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MONTHLY MORBIDITY REPORT

Reported Morbidity
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PUBLIC HEALTH STATISTICS and
DIVISION OF DISEASE CONTROL

Recommendation of the Immunization Practices Advisory Committee (ACIP)

Immune Globulins for Protection against Viral Hepatitis *

INTRODUCTION

The term "viral hepatitis" is commonly used for at least 3 clinically similar diseases that are etiologically and epidemiologically distinct [1]. Two of them, hepatitis A (formerly called "infectious hepatitis") and hepatitis B (formerly called "serum hepatitis"), have been recognized as separate entities since the early 1940s. The third, currently known as "non A/non B hepatitis," is probably caused by at least 2 different agents and, lacking a specific diagnostic test, remains a disease diagnosed by exclusion. It is an important cause of acute viral hepatitis in adults and is responsible for most of the post-transfusion hepatitis cases in the United States.

HEPATITIS SURVEILLANCE

Approximately 30,000 cases of hepatitis A, 16,000 cases of hepatitis B, and 8,000 cases of unspecified hepatitis are reported each year in the United States. Most patients are young adults.

IMMUNE GLOBULINS

Immune globulins used in medical practice are sterile solutions of antibodies (immunoglobulins) from human plasma. They are prepared by cold ethanol fractionation of large plasma pools and contain 10%-18% protein. In the United States, plasma is primarily obtained from professional donors. Only plasma shown to be free of hepatitis B surface antigen (HBsAg)* is used to prepare immune globulins.

Immune globulins (IG) (formerly called "immune serum globulin," ISG, or "gamma globulin") produced in the United States contains antibodies against the hepatitis A virus (anti-HAV) and the hepatitis B surface antigen (anti-HBs). Tests of lots of IG prepared since 1977 indicate that both types of antibody have uniformerly been present at stable titers.

Hepatitis B immune globulin (HBIG) is an immune globulin prepared from plasma containing extremely high titers of anti-HBs.

*Abbreviations are summarized in Table 2.

Neither IG nor HBIG when properly prepared transmits hepatitis A or B.

Serious adverse effects from immune globulins administered as recommended have been exceedingly rare. Globulins are prepared for intramuscular use and should not be given intravenously.

Immune globulins are not contraindicated for pregnant women if needed.

HEPATITIS A

Hepatitis A is caused by the hepatitis A virus (HAV), a 27-nm RNA (ribonucleic acid) agent that is a member of the picornavirus family. The illness caused by HAV characteristically has an abrupt onset with fever, malaise, anorexia, nausea, abdominal discomfort, and jaundice. Severity is related to age. In children, most infections are asymptomatic, and illness is not accompanied by jaundice. Fatality among hospitalized patients is quite low (about 0.1%).

Hepatitis A is primarily transmitted by person-to-person contact, generally through fecal contamination. Transmission is facilitated by poor sanitation and close personal contact, including sexual esposures. Common-source infections from contaminated food and water also occur.

The incubation period of hepatitis A is 15-50 days (average 28-30). HAV has consistently been demonstrated in stools of infected persons, with the highest concentrations of virus being excreted late in the incubation and early in the prodromal phase of illness. Virus excretion diminishes rapidly once jaundice appears. Greatest infectivity is during the 2-week period immediately before the onset of jaundice. Viremia is of short duration. A chronic carrier state with HAV in blood or feces has not been demonstrated. Although theoretically possible, transmission of HAV by blood transfusion or percutaneous routes appears to be extremely rare.

Specific tests are available to differentiate anti-HAV of the IgM class, which appears in the acute phase of illness, from anti-HAV of the IgG class, which appears in con-

* Reprint from MMWR: 30:34, 1981 pp. 423-428, 433-435.

TABLE 2. Hepatitis nomenclature

Abbreviation	Term	Comments
	Hepatitis A	
HAV	Hepatitis A virus	Etiologic agent of "infectious" hepatitis; probably an enterovirus; single serotype.
anti-HAV	Antibody to HAV	Detectable at onset of symptoms; lifetime persistence.
	Hepatitis B	
HBV	Hepatitis B virus	Etiologic agent of "serum" or "long-incubation" hepatitis; also known as Dane particle.
HBsAg	Hepatitis B surface antigen	Surface antigen(s) of HBV, detectable in large quantity in serum; several subtypes identified.
HBeAg	Hepatitis B e antigen	Soluble antigen; correlates with HBV replication; high titer HBV in serum, and infectivity of serum.
HBcAg	Hepatitis B core antigen	No commercial test available.
Anti-HBs	Antibody to HBsAg	Indicates past infection with and immunity to HBV, passive antibody from HBIG, or immune response from HBV vaccine.
Anti-HBe	Antibody to HBeAg	Presence in serum of HBsAg carrier suggests lower titer of HBV.
Anti-HBc	Antibody to HBcAg	Indicates past infection with HBV at some undefined time.
	Non A/non B hepatitis	
NANB	Non A/non B hepatitis	Diagnosis of exclusion; at least 2 candidate viruses; epidemiology parallels that of hepatitis B.
	Immune globulins	
IG	Immune globulin (previously ISG, immune serum globulin, or gamma globulin)	
HBIG	Hepatitis B immune globulin	

valescence (4-6 weeks after onset) and largely replaces IgM-class antibody. The diagnosis of acute hepatitis A is therefore confirmed by finding IgM-class anti-HAV as the predominant specific antibody in serum collected during the acute phase of disease. IgG-class anti-HAV, which replaces IgM-class antibody, remains detectable in serum for years and apparently confers life-long protection against reinfection.

Sero-epidemiologic studies show that hepatitis A is still a common infection in the United States. More than half the population over age 40 have serologic evidence of past infection.

IG AND HEPATITIS A

Numerous field studies conducted in the past 4 decades confirm that IG given before exposure or during the incubation period of hepatitis A is protective [2,4]. Its prophylactic value is greatest (80%-90%) when given early in the incubation period and declines thereafter[4]. In view of the need to give IG as soon as possible after exposure to HAV, and recognizing its intrinsic safety and the time required for – and cost of – antibody testing, routine serologic screening for anti-HAV before giving IG is not encouraged. Giving IG more than 2 weeks after exposure is not indicated.

RECOMMENDATIONS FOR IMMUNE GLOBULIN PROPHYLAXIS FOR HEPATITIS A.

Specific recommendations for IG prophylaxis for hepatitis A depend on the nature of the HAV exposure:

Post-Exposure Prophylaxis

Person-to-person contact:

Close personal contact. IG is recommended for all household and sexual (heterosexual or homosexual) contacts of persons with hepatitis A.

Day-care centers: Day-care centers with children in diapers can be important locales for HAV transmission[5,6]. If epidemiologic evidence shows that HAV transmission is occurring in a day-care center that cares for children in diapers, IG should be administered to staff, attendees,

and to all members of households whose diapered children attend. Careful handwashing after changing diapers is important.

Schools and preschools: Contact at school is usually not an important means of transmitting hepatitis A. Routine administration of IG is not indicated for pupils and teachers in contact with a patient. However, when epidemiologic study clearly shows the existence of a school- or classroom- centered outbreak, it is reasonable to give IG to those who have close personal contact with patients.

Institutions for custodial care: Living conditions in some institutions, such as prisons and facilities for the developmentally disabled, favor transmission of hepatitis A. When outbreaks occur, giving IG to residents and staff who have close contact with patients with hepatitis A may effectively reduce the spread of disease. Depending on the epidemiologic circumstances, prophylaxis can be limited in extent or can involve the entire institution.

Hospitals: Routine IG prophylaxis for hospital personnel is not indicated. Rather, sound hygienic practices should be emphasized. Intensive continuing staff education should point out the risk of exposure to hepatitis A and emphasize precautions regarding close contact with patients with hepatitis or with infective materials[7].

Offices and factories: Routine IG administration is not indicated under the usual office or factory conditions for persons exposed to a fellow worker with hepatitis A.

Common-source exposure:

IG might be effective in preventing foodborne or waterborne hepatitis A if exposure is recognized in time. However, IG is not recommended for persons exposed to a common-source of potential hepatitis infection once cases have begun to occur.

If a foodhandler is diagnosed as having hepatitis A, common-source transmission is possible. IG should be administered to other kitchen employees and may be considered for patrons if 1) the infected person is directly involved in handling foods that are not to be cooked or cooked foods before they are eaten, 2) the hygienic practices of the food-handler are deficient, and 3) consumers can be identified and treated within 2 weeks of exposure.

For postexposure IG prophylaxis, a single intramuscular dose of 0.02 ml/kg* is recommended.

Pre-Exposure Prophylaxis:

Travelers to foreign countries: The risk of hepatitis A for Americans traveling abroad appears to be small. It varies with living conditions, the prevalence of hepatitis A in the areas visited, and especially the length of stay [8,9]. As with any enteric infection, the best way to prevent hepatitis A is to avoid potentially contaminated water and food.

Travelers who follow the usual tourist routes may be at no greater risk of getting hepatitis A than they would be in the United States. IG is not recommended for them. However, travelers to high-risk areas outside ordinary tourist routes may be at increased risk. For such travelers, at risk for up to 2-3 months, a single IG dose of 0.02 ml/kg is recommended. For more prolonged travel, 0.06 ml/kg should be given every 5 months.

* Milliliters/kilogram of body weight.

HEPATITIS B

Hepatitis B is caused by the hepatitis B virus (HBV), a 42-nm, double-shelled DNA (deoxyribonucleic acid) virus. Several well defined antigen-antibody systems have been associated with HBV infection. HBsAg, formerly called "Australia antigen" or "hepatitis associated antigen," is an antigen found on the surface of the virus and on accompanying 22-nm spherical and tubular forms. HBsAg can be identified in serum 30-60 days after exposure to HBV and persists for variable periods. The various subtypes of HBsAg provide useful epidemiologic markers. Antibody against HBsAg, i.e., anti-HBs, develops after a typically resolved infection and is responsible for long-term immunity.

The frequency of chronically carrying HBsAg apparently relates both to age and immunologic competency. As many as 10% of HBV infections result in chronic carriage of HBsAg. The carrier state can be completely asymptomatic or, less commonly, associated with active liver disease. HBsAg carriers play an important role in the continuing transmission of hepatitis B, even though they show varying degrees of infectivity.

The hepatitis B e antigen (HBeAg) and antibody (anti-HBe) are distinct from HBsAg and anti-HBs. The potential infectivity of a carrier is higher if the HBeAg is present and lower if anti-HBe is present.

Routes of transmission of HBV include 1) direct percutaneous inoculation of infective serum or plasma by needle or transfusion of infective blood or blood products; 2) indirect percutaneous introduction of infective serum or plasma, such as through minute skin cuts or abrasions; 3) absorption of infective serum or plasma through mucosal surfaces, such as those of the mouth or eye; 4) absorption of other potentially infective secretions such as saliva or semen through mucosal surfaces, as might occur following sexual (heterosexual or homosexual) contact; and 5) transfer of infective serum or plasma via inanimate environmental surfaces or, possibly, vectors. Experimental data indicate that fecal transmission of HBV does not occur and that airborne spread is not epidemiologically important.

The onset of hepatitis B is generally insidious. Clinical symptoms and signs include various combinations of anorexia, malaise, nausea, vomiting, abdominal pain, and jaundice. Arthralgias and arthritis can also occur. Overall fatality rates for hospitalized patients generally do not exceed 1%. The incubation period of hepatitis B is long – 45-160 days (average 60-90). Cirrhosis and primary heptatocellular carcinoma are closely associated with chronic HBV infection.

IMMUNE GLOBULINS AND HEPATITIS B

IG and HBIG contain different amounts of anti-HBs. IG is prepared from plasma that is not preselected for anti-HBs content. Since 1977, all lots tested have contained anti-HBs at a titer of at least 1:100 by radioimmunoassay (RIA). HBIG, on the other hand, is prepared from plasma preselected for high titer anti-HBs. In the United States, HBIG has an anti-HBs titer of $>$ 1:100,000 by RIA. (Currently, the price of a dose of HBIG is more than 20 times that of IG.)

Recent studies have shown that immune globulins can prevent up to 75% of hepatitis B cases in certain settings[10 11]. What has been difficult to determine is the concentration of antibody that would be effective under various conditions of exposure, because studies differed in experimental design and in the immune globulins tested[12 19].

The studies generally indicated that: 1) HBIG is effective when given after percutaneous (needle stick) or mucous membrane exposure to blood containing HBsAg; 2) IG appears to have some effect in preventing clinical hepatitis; and 3) an immune globulin is most effective if given immediately after exposure.

It can be agreed further that HBIG is preferable to IG when there is bona fide percutaneous or mucous membrane exposure to blood known to contain HBsAg. However, because IG does contain anti-HBs, it remains an important alternative to HBIG whenever HBIG is unavailable, its cost is prohibitive, or a truly significant exposure to HBV may not have occurred.

Post-Exposure Prophylaxis:

Acute exposure to blood that might contain HBsAg: Percutaneous (needle stick) or mucous membrane exposure to blood that might contain HBsAg calls for a prompt decision about giving an immune globulin. In deciding whether to give a globulin and, if so, whether it should be IG or HBIG, one must recognize that the need is relative and depends on the kind of exposure. In the hospital, risk of clinical hepatitis B following exposure to blood known to contain HBsAg is approximately 1 in 20. If the blood is of unknown HBsAg status, the risk is 100 times lower, only about 1 in 2,000. This latter risk increases, however, in direct proportion to the likelihood that the blood is HBsAg-positive.

Recommendations on prophylaxis can, thus, be categorized as to 1) whether the source of blood is known or unknown and 2) whether the HBsAg status of the source blood is known or unknown. The following outline and summary table (Table 3) are based on these categories. Management of each exposure must be individualized in view of the number of contributing factors. Furthermore, it is important to emphasize that for greatest effectiveness,

TABLE 3. Summary of postexposure prophylaxis of acute exposures to HBV*

Exposure	HBsAg Testing	Recommended prophylaxis
HBsAg positive	. . . .	HBIG (0.06 ml/kg) immediately and 1 month later
HBsAg status unknown Source known:		
High Risk†	Yes‡	IG (0.06 ml/kg) immediately, and if –TEST POSITIVE– HBIG (0.06 ml/kg) immediately and 1 month later or if –TEST NEGATIVE– Nothing
Low Risk†	No	Nothing or IG (0.06 ml/kg)
HBsAg status unknown Source unknown	No	Nothing or IG (0.06 ml/kg)

* Important details are in the text.
† Characterized in text.
‡ If results can be known within 7 days of exposure.

globulin should be given promptly (its value beyond 7 days of exposure is unclear).

A. Source known, HBsAg status positive.

HBIG (0.06ml/kg) should be given immediately, ideally within 24 hours of exposure. A second identical dose should be given 1 month later. (If HBIG is not available, IG should be used in the same dose and schedule).

B. Source known, HBsAg status unknown.

Two decisions are involved here: whether to test for HBsAg and which immune globulin to give. Because these decisions relate both to the relative probability that the source will be HBsAg-positive and to the inherent delay in testing, the following operational guidelines are suggested:

1. High risk that the source is HBsAg-positive—such as associated with patients with acute, unconfirmed viral hepatitis; patients institutionalized with Down syndrome; patients on hemodialysis; persons of Asian origin; male homosexuals; users of illicit, intravenous drugs.

If HbsAg test results can be known within 7 days of the exposure, IG (0,06 ml/kg) should be given immediately, certainly within 24 hours. If test results are positive, HBIG (0.06 ml/kg) should be given at that time and again 1 month later.

If HBsAg test results cannot be known within 7 days of the exposure, the decision to use IG or HBIG must be based on the clinical and epidemiologic characteristics of exposure and the availablilty of globulin, remembering the importance of characterizing the source and giving globulin as soon after exposure as possible.

2. Low risk that the source is HBsAg-positive—such as associated with the average hospital patient.

Prophylaxis is optional; HBsAg testing is not recommended. If an immune globulin is to be used, IG (0.06 ml/kg) should be given promptly, certainly within 24 hours. No further action is necessary.

C. Source unknown, HBsAg status unknown.

Prophylaxis is optional. If an immune globulin is to be used, IG (0.06 ml/kg) should be given promptly, certainly within 24 hours. No further action is necessary.

Exposure of the newborn: Infants born to HBsAg-positive mothers (especially mothers who are HBeAg positive) are at risk of being infected with HBV and becoming chronic carriers. Recent studies have shown that the carrier state can be prevented in about 75% of such infections if newborns are given HBIG immediately after birth [20]. (IG was not included in the protocol.)

All infants born to HBsAg-positive mothers should be given HBIG, total dose 0.5 ml intramuscularly, as soon after birth as possible (no later than 24 hours). The same dose (0.5 ml) should be repeated 3 months and 6 months later.

Sexual contact with persons with hepatitis B: In only 1 study has there been any evaluation of the value of immune globulin for sexual contacts of patients with acute hepatitis B[21]. Although results suggest protection with HBIG, additional studies comparing IG, HBIG, and placebo group are needed before specific recommendations can be made.

Pre-Exposure Prophylaxis:

Staff and patients of hemodialysis units: Routine passive immunization against hepatitis B is not recommended for staff and patients of hemodialysis units. Instead, precautions such as serologic screening of patients and staff, segregation of carriers, and environmental hygiene should be encouraged. In the rare event that such measures fail to interrupt transmission, prophylaxis with an immune globulin may be considered. Because carefully controlled studies have failed to demonstrate an advantage of HBIG over IG in this setting, IG (0.05-0.07 ml/kg) every 4 months is recommended for patients and staff[22].

Staff and patients of institutions for custodial care of the developmentally disabled: HBV is commonly endemic in institutions for the developmentally disabled, but passive immunization is not routinely recommended for staff or clients unless it is shown that hepatitis B cannot be controlled by environmental measures alone. Then IG may be administered in the same dose and at the same intervals as for patients and staffs of hemodialysis units.

HEPATITIS NON A/NON B AND HEPATITIS – NONSPECIFIC

Without accurate tests for diagnosing nonA/nonB viral hepatitis, the value of prophylaxis with immune globulins cannot be determined. No specific recommendation can be made, but as with hepatitis that cannot be specifically diagnosed (hepatitis—nonspecific), it is reasonable to apply the recommendations for prophylaxis against hepatitis A.

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(References continued on page 6)

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SELECTED REPORTABLE DISEASES
(By Place of Residence)

STATE AND PARISH TOTALS REPORTED MORBIDITY OCTOBER, 1981	VACCINE PREVENTABLE DISEASES					ASEPTIC MENINGITIS	HEPATITIS A AND UNSPECIFIED	HEPATITIS B	LEGIONNAIRES DISEASE	MALARIA**	MENINGOCOCCAL INFECTIONS	SHIGELLOSIS	TUBERCULOSIS, PULMONARY	TYPHOID FEVER	OTHER SALMONELLOSIS	UNDERNUTRITION SEVERE	GONORRHEA	SYPHILIS, PRIMARY AND SECONDARY	RABIES IN ANIMALS (PARISH TOTALS CUMULATIVE, (1981)
	MEASLES	RUBELLA*	MUMPS	PERTUSSIS	TETANUS														
TOTAL TO DATE 19 80	12	12	66	34	5	73	751	256	5	42	83	208	413	2	163	7	19601	1169	14
TOTAL TO DATE 19 81	4	9	5	5	2	91	850	306	1	9	104	115	346	2	181	1	18867	1374	33
TOTAL THIS MONTH	0	0	0	0	0	9	145	44	0	3	2	33	28	0	34	0	1535	112	2
ACADIA								6				1	1		2		22		
ALLEN																	1		
ASCENSION													1				2		
ASSUMPTION																	3		
AVOYELLES																	3		1
BEAUREGARD																	8		
BIENVILLE																	2		3
BOSSIER						1						1					29		1
CADDO												21	3		9		213	19	1
CALCASIEU								1							1		112	8	
CALDWELL																			
CAMERON																	3	1	
CATAHOULA								1					1		3		5		
CLAIBORNE							1										10		
CONCORDIA								1									4		
DESOTO													1				2		
EAST BATON ROUGE							1				1	1					115	8	
EAST CARROLL																	4		
EAST FELICIANA																	2		1
EVANGELINE													1				3	1	1
FRANKLIN													1				4		
GRANT																	3		
IBERIA							5	1									2		
IBERVILLE							2										4		
JACKSON																		4	
JEFFERSON							61	8		1			1		1		95		
JEFFERSON DAVIS															1		8	1	
LAFAYETTE						1	4	5				4	1		3		30	1	
LAFOURCHE												2					11	1	
LASALLE																	1		1
LINCOLN															1		9	3	
LIVINGSTON													1				3		
MADISON							2										8		
MOREHOUSE							1				1						4		
NATCHITOCHES							4											3	7
ORLEANS						1	21	9		2		1	1		2		521	39	
OUACHITA							9						4				67	3	1
PLAQUEMINES							2										2	1	
POINTE COUPEE																	1		
RAPIDES								1					4				52		7
RED RIVER																		1	1
RICHLAND																	13		
SABINE																	1		1
ST. BERNARD							4										2		
ST. CHARLES													1				2	2	
ST. HELENA																			
ST. JAMES																	7		
ST. JOHN							1	1					1		1		4	2	
ST. LANDRY								1				1	1				12		
ST. MARTIN							1	2									4		
ST. MARY													1				8		
ST. TAMMANY								1									11	3	
TANGIPAHOA								2							3		22	4	
TENSAS																	1		
TERREBONNE						6	24	2				1	1		3		23	2	
UNION																	3	1	1
VERMILION								1					1		2		6		
VERNON																	7	1	5
WASHINGTON							1	1							1		4		
WEBSTER							1						1		1		12		1
WEST BATON ROUGE																	4	1	
WEST CARROLL																	2		
WEST FELICIANA																		1	
WINN																			
OUT OF STATE																	19	1	

* Includes Rubella, Congenital Syndrome.
** Acquired outside United States unless otherwise stated.

From January 1, 1981 - October 31, 1981 the following cases were also reported:
1-Leptospirosis; 10-Cholera; 1-Tularemia; 1-Psittacosis; 1-Brucellosis; 1-Actinomycosis; 2-Cryptococcosis; 2-Rocky Mountain Spotted Fever; 3-Reyes Syndrome.

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