

contents: Viral Hepatitis

Part I: Recognizing Hepatitis A Dengue Surveillance Typhoid Fever Alert Viral Isolates for October 1983

Results of PDN Survey

BUREAU OF EPIDEMIOLOGY 1100 West 49th Street, Austin, Texas 78756 (512-458-7207)

VIRAL HEPATITIS
PART I: RECOGNIZING HEPATITIS A

During the latter half of 1981, a six-part series on viral hepatitis was published in Texas Morbidity This Week (now Texas Preventable Disease News). Due to numerous requests for reprints, the series has been updated and will be published in PDN throughout the following weeks.

Inflammation of the liver (the literal meaning of the word hepatitis) can result from a wide variety of pathologic conditions. Not all types of hepatitis are communicable. There are, for example, instances where liver injury is due to adverse reactions to certain drugs such as analgesics (aspirin, acetaminophen), antibiotics (tetracycline), and antineoplastic agents (6-mercaptopurine). However, most of the hepatitis seen in any given community is caused by infectious agents. These can be bacterial species or viruses from the Herpes family, but the majority of hepatitis cases are due to infection with either hepatitis A virus (HAV) or hepatitis B virus (HBV). Recently, a third classification of hepatitis viruses, "non-A/non-B," has been set forth to document cases where a diagnosis has ruled out other viral etiological agents.

A specific diagnosis of viral hepatitis is important information for the physician, patient, and public health official. For the physician and his or her patient, the type of hepatitis involved has clearly different implications, especially regarding prognosis. For example, hepatitis A is an acute viral illness with a relatively short period of infectivity, and recovery is usually complete. Hepatitis B, however, is generally characterized by a longer period of clinical symptoms, and prognosis is initially difficult to assess. The hepatitis B patient may recover completely. However, 5-10% of those with hepatitis B infections go on to become carriers, a state which may progress to chronic hepatitis over time. The public health professional must know the specific viral etiology of a hepatitis case in order to recommend proper control measures to protect the public. Epidemiologic factors such as modes of transmission and incubation are different for hepatitis A and hepatitis B. An example of this is that patients with hepatitis A may commonly cause foodborne or person-to-person spread of the disease, while those patients with hepatitis B rarely present a problem in restaurant or school settings.

It is, therefore, a good idea to be aware of the distinctive aspects of the two major types of viral hepatitis. In this issue information on hepatitis A is presented; a discussion of hepatitis B will follow in Part II of this series.

Clinical Picture of Hepatitis A

Hepatitis A is generally an acute and self-limited disease. Formerly known as "infectious hepatitis" or "short incubation hepatitis," it is a disease found primarily in children and young adults. In many individuals, especially young children, a subclinical infection with hepatitis A is common. Clinical symptoms,

when they do occur, include abrupt onset of fever, malaise, headache, anorexia (loss of appetite), myalgia, nausea, and abdominal discomfort. Jaundice may or may not develop. If it does, it usually follows within a few days. Mortality due to HAV is quite low (<0.5 percent), with the elderly or debilitated patient at high risk.

Initially, a diagnosis of hepatitis A is based on the characteristic epidemiology of the disease. Briefly, the incubation period is dose-related, ranging from 15 to 50 days, with the average period being 25 to 30 days. Hepatitis A is spread predominantly by the fecal-oral route and is easily transmitted from person-to-person when good personal hygiene is not practiced. Susceptibility is general, and the disease is distributed worldwide. For these reasons, hepatitis A occurs primarily in epidemics or outbreaks in which a source can be identified, eg, in restaurants (where an infectious foodhandler can unknowingly contaminate the food or where shellfish harvested from waters contaminated by hepatitis A virus are prepared) or day care centers, especially those caring for children in diapers (where the virus spreads rapidly among children and staff). Day care centers present the greatest problems mainly because most of the children will experience subclinical infections, and although the children appear healthy, they are nonetheless infectious and can serve as point sources of infection.

Another characteristic of hepatitis A infections is the short period of infectivity compared to that for hepatitis B. HAV is excreted in large quantities in the feces as early as two weeks prior to onset of symptoms and for one week thereafter. The presence of virus in the bloodstream or in other body fluids is transient and does not contribute significantly to the transmission of disease.

The definitive diagnosis of hepatitis A depends solely on specific serological assays for anti-HAV immunoglobulins. As with many viral diseases, infection with HAV results in prolonged immunity that is protective against subsequent reinfection. It is important, therefore, to be able to ascertain the nature of the antibodies present in a patient's serum. Figure 1 shows a typical course of an HAV infection and depicts the period of infectivity, the time of clinical illness, and the resulting antibody responses.

There are currently two radioimmunoassay (RIA) tests commercially available for the detection of anti-HAV immunoglobulins. One test is specific for IgM antibodies; anti-HAV IgM is synthesized early in infection and can be detected during illness and sometimes for up to two to three months following recovery. The other test available is an RIA test for anti-HAV IgG (immunoglobulin G). IgG is synthesized in increasing amounts as the hepatitis A infection progresses toward recovery and, thus, is the sole indicator of past infection. Both the IgM and IgG results should be considered in determining if and when a hepatitis A infection has occurred (Table 1).

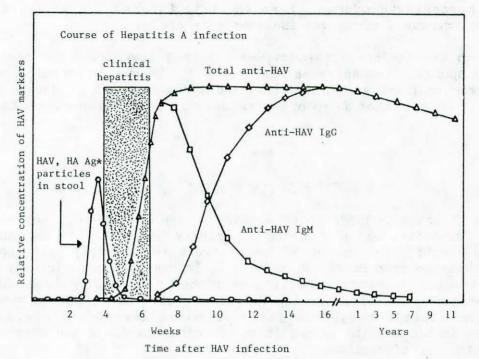
The diagnosis of hepatitis in any patient has certain public health implications. Further investigation to determine its etiology enables health professionals to apply appropriate preventive measures for controlling its spread.

Health professionals are reminded that hepatitis (A, B, or unspecified), is a reportable disease.

For further information on hepatitis, contact the Bureau of Epidemiology, Texas Department of Health at (512)458-7328 or STS 824-9328. For toll-free reporting, call 1-800-252-8239.

This report was prepared by Lynne Sehulster, Ph.D., Staff Epidemiologist/Microbiologist, Bureau of Epidemiology, Texas Department of Health.

Figure 1.
Hepatitis A Viral Markers in the Blood and Stool During HAV Infection



*HA Ag: Hepatitis A Antigen

Table 1.
Interpretation of RIA of IgM and IgG

Test Results	Interpretation
IgM+, IgG <u>+</u>	Current infection, serum drawn very early in infection before IgG levels rise
IgM+, IgG+	Current infection
IgM-, IgG+	Past infection
IgM-, IgG-	It is extremely unlikely that this patient has hepatitis A

DENGUE SURVEILLANCE

In the October 1983 Dengue Surveillance Summary distributed by the Centers for Disease Control, there are reports of dengue activity in Central America. Increased numbers of cases were reported in late June and early July in the state of Puebla in Mexico. In subsequent weeks, there were reports of outbreaks of dengue-like illness in widespread areas from the Pacific Coast, Veracruz, and the Yucatan. Three dengue 1 viruses were isolated from patients in Puebla in late July, but the majority of other cases were confirmed serologically and the precise serotype not specified.

Dengue-like illness was also reported from El Salvador in late June and early July. Cases continued to be reported through August with the majority reported since June. Dengue 4 virus has been isolated and serologically confirmed from most of the cases. Cases have been reported from all areas of El Salvador with the majority from the eastern region bordering Honduras. Less specific information is available from Honduras; however, dengue 1 virus was isolated from one case.

Dengue activity in the Caribbean islands has remained low throughout the year. In Puerto Rico only sporadic transmission has occurred. Dengue 4 transmission occurred throughout the year in Trinidad and Jamaica, and sporadic cases of dengue 2 occurred in both islands. Information from other islands in the region suggests low or no activity.

TYPHOID FEVER ALERT

Between November 7 and 21, 1983, three confirmed and four suspect cases of typhoid fever have been identified among students at Trinity University in San Antonio. An investigation to identify the source of the outbreak is currently being conducted by the San Antonio Metropolitan Health District. It is possible some Trinity University students will develop clinical infection over the next week or two. Since these students went home for the Thanksgiving Holiday, health officials throughout Texas should be on the alert for typhoid fever among college-age individuals. The organism isolated in San Antonio is sensitive to both ampicillin and chloramphenicol, the drugs of choice for treatment.

* * *

VIRAL ISOLATES FOR OCTOBER 1983

Adenovirus	Bell(1), Bexar(2), Willacy (2)
Cytomegalovirus	Bell(1), Coryell(1), Galveston (4), Lubbock(1)
Coxsackie (A16)	Bexar(1)
Coxsackie (B02)	Bexar(1)
Echo (06)	El Paso(1)
Echo (09)	Lubbock(1)
Echo (24)	Bexar(1)
Echo (25)	Bexar(1)
Echo (27)	Bexar(1)
Polio (3)	Bexar(1)
Chlamydia Trach.	Bexar(5), Travis(2)

RESULTS OF PDN SURVEY

We would like to take this opportunity to thank everyone who returned the PDN readership survey recently. Our mailing system has been revised in conjunction with this survey. Should any subscribers experience problems such as wrong addresses or missed issues due to the change in the mailing system please contact the Bureau of Epidemiology at 1100 West 49th Street, Austin, Texas 78756 or call 512/458-7207 or STS 824-9207. To assist this office in complying with requests for cancellation of a subscription or change of address, please furnish your old address. Other changes in the newsletter will be implemented in the future as a result of suggestions from the readership survey. Thank you for your ideas.

WEEK NO: 46 ENDING: NOVEMBER 19. 1983

												¥	EEK	NO: 46	END ING:	: N	OVEMBER 19	, 1983	,
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WEEK NO: 46 ENDING: NOVEMBER 19, 1983

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OTHER COUNTIES:	NO	COMMU	NICABL	E DISEAS	ES:	5		OTHER DISEAS	ES ONLY:	a	·NOT	REPORTIN	G: 13 ·	
PUBLIC HEALTH REGION 7	7 TYLI	ER, TX		PĤONE:	214	/595-3	585	POPULAT	ION =	866,694				
COUNTIES		•		•										
ANDERSON BOWIE CASS CHEROKEE GREGG HARRISON HENDERSON MARION			***				* * * * * * * * * *	# # # # #		* * * * *	1 30 4 1 10	2	* * * * * * * * * * * * *	
MORRIS PANOLA RAINS SMITH UPSHUR WOOD CASES THIS WEEK CUMULATIVE 1983	27		9 ******	35	22	73	~ * * * * * * * * * * * * * * * * * * *	***		* * * * * * * * * * * * * * * * * * *	1 2 1 16 5 1 74 2,679	2 2 2 2 2 3	* 12 * 39 * 80 * 4,525	70
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WILLACY CASES THIS WEEK CUMULATIVE 1983 OTHER COUNTIES:	59 NO		10		2 209 DISEAS	3 109 SES:	561	* * *	1 0T			23 ONL Y:	1 : 2	* *	1 62 2,159 NOT	4 202 REPORTI	* * *	206 19 ₊ 868	191
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WEEK NO: 46 ENDING: NOVEMBER 19, 1983 .

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MIDLAND		*				*	*		*	14	1	* 25	
REEVES		*				*	*		*	• •	-	* 15	
WINKLER		*				*	*		*			* 7	
CASES THIS WEEK		*				*	*		*	29	5		
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OTHER REPORTING SOURCE	ΕS												
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V.A. HOSPITALS		**	1	1		τ ★	*		*	36		* 27 ₁	
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CASES THIS WEEK		**	1	1		*	* *		≁	36		* * 271	
CUMULATIVE 1983	6	2 *	23	26	27	*	1 *		*	2,263	121		

OTHER REPORTABLE DISEASES	REPORTED 1982	THIS WEEK 1983		ATIVE
	1762	1703	1982	1983
ACOUTTED THOUSE DESIGNED CANDONE (AIDC)		5		59
ACQUIRED IMMUNE DEFICIENCY SYNDROME (AIDS) AMEBIASIS	,,	3	4.4.0	-
ANTHRAX	4 0	3	440 0	35.3 D
BOTULISM	ů ů	Ö	0	2
BRUCELLOSIS	Õ	3	24	67
CHICKENPOX	57	101	10087	14056
CHOLERA	3	o	0	0
DIPHTHERIA	٥	, o	1	0
ENCEPHALITIS, ST. LOUIS	3	0	18	1
ENCEPHALITIS, WESTERN EQUINE	0	1	4	1
ENCEPHALITIS, VENEZUELAN EQUINE	a	0	٥	D
ENCEPHALITIS, ALL OTHER	3	3	138	101
LEPROSY (HANSENS DISEASE)	8	3	27	32
LEPTOSPIROSIS	0	0	13	0
MALARIA	0	<u>o</u>	Ō	0
MALARIA ACQUIRED OUTSIDE USA	2	3	48	44
MUMPS	3	6	204	202
PERTUSSIS	4	٥	71	87
PLAGUE	۵	0	1	0
POLIOMYELITIS, PARALYTIC	õ	0	<u>o</u>	a
PSITTACOSIS	٥	σ	7	5
Q FEVER	0	0	1	O
RABIES IN MAN	3	a	٥	0
RELAPSING FEVER	a	0	3	0
RHEUMATIC FEVER RUBELLA CONGENITAL SYNDROME	1 0	0	9 0	12
ROBELLA CONGENITAL SINDROME	U	0	u	0
SALMONELLOSIS	53	46	2133	2123
SHIGELLOSIS	43	55	1952	1707
STREP THROAT & SCARLET FEVER	828	636 1	41880	33094 17
REYE SYNDROME TETANUS	0	ũ	,	
ILIANUS	a	ບ	. 6	6
TRICHINOSIS	0	a	2	1
TULAREMI A	1	1	10	9
TYPHOID FEVER	1	1	27	46
TYPHUS, EPIDEMIC YELLOW FEVER	0	0 0	0 0	0 0
ILECON ILTER	u	J	U	Ü

15