WEEKLY EPIDEMIOLOGICAL REPORT

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231, de Saram Place, Colombo 01000, Sri Lanka
Tele: + 94 11 2695112, Fax: +94 11 2696583, E mail: epidunit@sltnet.lk
Epidemiologist: +94 11 2681548, E mail: chepid@sltnet.lk
Web: http://www.epid.gov.lk

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Hepatitis A

Hepatitis A is an acute, usually self-limiting disease of the liver caused by hepatitis A virus (HAV). The incidence of hepatitis A is closely related to socio-economic development and sero-epidemiological studies show that prevalence of anti-HAV antibodies in the general population varies from 15% to close to 100% in different parts of the world.

An estimated 1.5 million clinical cases of hepatitis A occur each year. In young children, HAV infection is usually asymptomatic whereas symptomatic disease occurs more commonly among adults. Infection with HAV induces lifelong immunity. In areas of low endemicity, hepatitis A usually occurs as single cases among persons in high-risk groups or as outbreaks involving a small number of persons. In highly endemic areas, most persons are asymptomatically infected with HAV during childhood and clinical hepatitis A is uncommon. In countries of low and intermediate endemicity, adult disease is seen more often and hepatitis A may represent a substantial medical and economic burden.

Transmission occurs primarily through the faeco-oral route and is closely associated with poor sanitary conditions. The most common modes of transmission include close personal contact with an infected person and ingestion of contaminated food and water. The virus is shed in the faeces of persons with both asymptomatic and symptomatic infection. Under favourable conditions HAV may survive in the environment for months. Bloodborne transmission of HAV occurs, but is much less common.

The average incubation period is 28 days, but may vary from 15-50 days. Approximately 10-12 days after infection, the virus can be detected in blood and faeces. In general, a person is most infectious from 14-21 days before the onset of symptoms, through 1 week after the onset of symptoms.

Antibodies against HAV develop in response to infection and sero-prevalence can be used as a marker of viral transmission in a community. The lowest seroprevalence is found in the Nordic countries (about 15%). In other parts of Europe and Australia, Japan and in the United States, 40%-70% of the adult population has demonstrable antibodies to HAV. Practically all adults living in developing areas of the world have serological evidence of past infection.

The risk of developing symptomatic illness following HAV infection is directly correlated to age. In children aged < 6 years, HAV infection is usually asymptomatic, with only 10% developing jaundice. Among older children and adults, infection usually causes clinical disease, with jaundice occurring in more than 70% of cases. Therefore, highly HAV-endemic regions are characterized by asymptomatic childhood infection, with only the occasional occurrence of clinical hepatitis A.

For practical purposes, the world can be divided into areas of low, intermediate and high endemicity, although there may be regional differences in endemicity within a country. In areas of low endemicity, the disease occurs mainly in adolescents and adults in high-risk groups (e.g. homosexual men, injection-drug users), and in certain subpopulations (e.g. closed religious communities). Some of these groups may also experience periodic outbreaks of hepatitis A. In areas of low endemicity, occasional food and waterborne outbreaks of hepatitis A occur.

In areas of intermediate endemicity, transmission occurs primarily from person to person in the general community, often with periodic outbreaks. In these countries many individuals escape early childhood infection, but are exposed later in life when clinical hepatitis occurs more frequently. In these areas, most cases occur in late childhood and early adulthood.

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In areas of high endemicity, where the lifetime risk of infection is greater than 90%, most infections occur in early childhood and are asymptomatic. Thus, clinically apparent hepatitis A is rarely seen in these countries. Countries in transition from developing to developed economies will gradually move from high to intermediate endemicity and hepatitis A is likely to become a more serious problem in these areas.

Although hepatitis A is mostly self-limiting and rarely fatal, the disease may represent a substantial economic burden, particularly in countries with low and intermediate incidence rates

The pathogen and the disease

HAV is a member of the Picornaviridae family that includes both the enteroviruses and rhinoviruses of humans. Being the only species member, it constitutes its own genus named hepatovirus. HAV is a non-enveloped (naked) virus of 27-28nm diameter without morphological features differentiating it from other picornaviruses. Four structural proteins encapsulate the RNA genome. Neutralization sites for anti-HAV antibodies are mainly contained in 2 of these proteins. Although 6 genotypes of HAV have been identified, there appears to be no variation detectable by serology in these neutralization sites. The virus is relatively stable at low pH and moderate temperatures, but is inactivated by high temperature (almost instantly at 85 0C), and by formalin or chlorine.

The clinical course of acute hepatitis A is indistinguishable from other types of acute viral hepatitis. Symptoms typically include fever, malaise, anorexia, nausea and abdominal discomfort, followed by dark urine and jaundice. The severity of disease and mortality increases in older age groups. The convalescence following hepatitis A may be slow and is characterized by fatigue, nausea and lack of appetite. Complications of hepatitis A include relapsing hepatitis, cholestatic hepatitis and fulminant hepatitis. Fulminant hepatitis occurs in approximately 0.01% of clinical infections and is characterized by rapid deterioration in liver function and a very high fatality rate. Chronic infection with HAV does not occur. No specific antiviral therapy is currently available.

The aetiological diagnosis is made by the demonstration of IgM antibodies to HAV (IgM anti-HAV) in serum. Detection of the virus or viral antigens in the stool is of limited value for routine diagnosis.

Protective immune response

Protective antibodies develop in response to infection and persist for life. The protective role of anti-HAV antibodies have been demonstrated by the protection against hepatitis A resulting from passive immunization with serum immunoglobulin. The effect of mucosal immunity on HAV infection is not known.

Justification for vaccine control

Although usually a self-limiting disease without serious sequelae and with a low case-fatality rate, human suffering may be considerable. In addition, direct and indirect medical costs including the infection control measures involved may impose a considerable economic burden on society. In countries where clinical hepatitis A is an important health problem, immunization is likely to be a cost-effective public health tool to control the disease.

Hepatitis A vaccines-Techniques for growing HAV in cell culture have made it possible to generate sufficient amounts of virus for vaccine production. Several inactivated or live attenuated vaccines against hepatitis A have been developed, but only 4 inactivated hepatitis A vaccines are currently available internationally. All 4 vaccines are similar in terms of efficacy and side-effect profile. The vaccines are given parenterally, as a 2-dose series, 6-18 months apart. No vaccine is licensed for children aged < 1 year.

Three vaccines are manufactured from cell culture adapted HAV propagated in human fibroblasts. Following purification from cell lysates, the HAV preparation is formalin-inactivated and adsorbed to an aluminium hydroxide adjuvant. The fourth vaccine is manufactured from HAV purified from infected human diploid cell cultures and inactivated with formalin. This preparation is adsorbed to biodegradable 150 nm phospholipid vesicles spiked with influenza haemagglutinin and neuramidase.

A combination vaccine containing inactivated hepatitis A and recombinant hepatitis B vaccines has been licensed since 1996 for use in children aged 1 year or older in several countries. The combination vaccine is given as a 3-dose series, using a 0, 1, 6 month schedule.

Hepatitis A vaccines are all highly immunogenic. Nearly 100% of adults will develop protective levels of antibody within 1 month after a single dose of vaccine. Similar results are obtained with children and adolescents in both developing and developed countries. The protective efficacy of the vaccine against clinical disease was determined in 2 large trials. Among almost 40 000 Thai children aged 1-16 years the protective efficacy was 94% (95% confidence intervals: 82%-99%) following 2 doses of vaccine given 1 month apart. Among approximately 1 000 children aged 2-16 years, living in a highly endemic community in the United States, the efficacy of 1 dose of vaccine was 100% (95% confidence intervals: 87%-100%). Although 1 dose of vaccine provides at least short-term protection, the manufacturers currently recommend 2 doses to ensure long-term protection. In studies evaluating the duration of protection of 2 or more doses of hepatitis A vaccine, 99%-100% of vaccinated individuals had levels of antibody indicative of protection 5-8 years after vaccination. Kinetic models of antibody decay indicate that the duration of protection is likely to be at least 20 years and possibly lifelong.

Post-marketing surveillance studies are needed to monitor vaccine-induced long-term protection and to determine the need for booster doses of vaccine. This is especially true in areas of low endemicity where natural boosting does not occur.

Contraindications to hepatitis A vaccination include a known allergy to any of the vaccine components. Hepatitis A vaccine may be administered with all other vaccines included in the Expanded Programme on Immunization and with vaccines commonly given for travel. Concurrent administration of immune serum globulin does not appear to influence significantly the formation of protective antibodies.

Source-Hepatitis A vaccine,available from http://www.who.i n t/ immunization/wer7505Hepatitis%20A_Feb00_position_ paper.pdf

Compiled by Dr. Madhava Gunasekera of the Epidemiology Unit

Table 4: Selected notifiable diseases reported by Medical Officers of Health 02th – 08th October (45th Week)

RDHS	Dengue Fe-		Dysentery		Encephalit		E Fever		F Poisoning		Leptospirosi		T Fever		V Hepatitis		H Rabies		Chickenpox		Meningitis		Leishmani-		WRCD %	
	A	B	A	B	A	B	A	B	A	B	A	B	A	B	A	B	A	B	A	B	A	B	A	B	T*	C**
Colombo	177	8696	10	197	0	17	6	149	0	56	0	189	0	8	1	79	0	1	8	417	1	63	0	0	**77**	**23**
Gampaha	62	3264	6	196	0	21	0	48	0	39	12	398	1	21	4	177	0	0	1	161	5	92	0	5	**80**	**20**
Kalutara	23	1587	6	171	0	20	1	78	1	26	11	368	1	7	0	24	0	0	2	251	0	69	0	0	**77**	**23**
Kandy	25	1602	3	154	0	11	1	27	2	12	1	70	2	99	1	114	0	0	6	135	0	16	1	5	**83**	**17**
Matale	10	428	5	96	0	4	1	25	0	10	3	64	0	4	1	51	0	0	0	45	0	35	1	12	**77**	**23**
NuwaraEliya	2	231	2	152	0	2	1	16	0	217	1	30	1	61	2	24	0	0	3	129	0	12	0	0	**77**	**23**
Galle	13	778	5	117	0	19	0	7	0	89	6	203	2	64	0	15	0	2	6	303	0	46	1	2	**84**	**16**
Hambantota	4	299	3	58	0	3	0	15	0	34	2	163	0	63	2	89	0	0	1	97	1	51	0	309	**75**	**25**
Matara	6	428	5	82	0	13	0	28	0	29	5	148	3	86	2	142	0	2	3	247	3	81	3	91	**100**	**0**
Jaffna	11	649	12	395	0	10	4	312	12	113	0	9	5	344	0	17	0	1	0	141	1	56	0	0	**83**	**17**
Kilinochchi	0	60	1	40	0	0	0	15	0	5	0	9	0	16	0	0	0	2	0	2	0	7	0	11	**50**	**50**
Mannar	0	68	0	74	0	3	0	65	0	36	0	15	0	20	0	2	0	0	0	12	0	5	0	4	**40**	**60**
Vavuniya	0	68	0	58	0	13	1	13	0	20	0	50	0	3	0	3	0	2	0	22	0	34	1	12	**50**	**50**
Mullaitivu	0	117	1	24	0	2	0	10	0	43	0	38	0	7	0	1	0	2	0	8	0	6	0	15	**60**	**40**
Batticaloa	2	517	20	312	0	5	0	10	0	73	0	33	0	2	0	14	0	3	1	45	0	8	0	0	**86**	**14**
Ampara	7	193	3	179	0	1	0	5	0	12	1	37	0	1	0	9	0	0	1	88	0	18	0	3	**71**	**29**
Trincomalee	1	187	1	64	0	3	0	6	0	3	1	60	0	15	0	3	0	1	0	40	0	4	1	29	**58**	**42**
Kurunegala	13	2575	10	193	1	42	1	39	0	26	14	314	0	45	2	59	0	1	3	336	3	99	0	55	**85**	**15**
Puttalam	10	835	0	76	0	7	0	16	0	36	0	43	0	14	0	7	0	1	1	82	0	35	0	10	**69**	**31**
Anuradhapur	4	486	1	101	0	17	0	3	1	67	0	308	0	24	0	27	0	2	3	167	0	94	2	388	**68**	**32**
Polonnaruwa	11	432	3	83	0	2	0	14	1	65	1	162	0	3	1	32	0	2	1	125	1	20	1	160	**86**	**14**
Badulla	6	474	7	202	0	5	3	21	0	11	1	57	2	85	0	46	0	0	1	126	1	69	0	7	**71**	**29**
Monaragala	4	242	0	113	0	4	0	24	0	35	1	198	1	60	6	173	0	1	0	51	1	25	0	10	**64**	**36**
Ratnapura	7	1615	8	366	0	83	0	40	0	18	8	350	1	72	13	514	0	1	2	179	1	78	0	13	**61**	**39**
Kegalle	16	1063	1	126	0	17	1	30	0	11	3	239	1	74	2	222	0	0	5	313	0	103	0	2	**64**	**36**
Kalmune	0	494	2	147	0	2	0	3	1	122	0	10	0	2	0	5	0	0	6	93	0	11	0	1	**46**	**54**
SRI LANKA	**414**	**27388**	**115**	**3776**	**1**	**326**	**20**	**1019**	**18**	**1208**	**71**	**3565**	**20**	**1200**	**37**	**1849**	**0**	**24**	**54**	**3615**	**18**	**1137**	**11**	**1144**	**74**	**26**

Source: Weekly Returns of Communicable Diseases (WRCD).

***T=**Timeliness refers to returns received on or before 08th November , 2013 Total number of reporting units 339. Number of reporting units data provided for the current week:266 C** Completeness

A = Cases reported during the current week. **B** = Cumulative cases for the year.**H Rabies***= Human Rabies, **E Fever***=Enteric Fever, **F Poison*** =Food Poisoning, **T Fever***=Typhus Fever, **V Hepatitis***=Viral Hepatitis

Table 1: Vaccine-Preventable Diseases & AFP 02th November– 08th November 2013 (45th Week)

Disease	No. of Cases by Province									Number of cases during current week in 2013	Number of cases during same week in 2012	Total number of cases to date in 2013	Total number of cases to date in 2012	Difference between the number of cases to date in 2013 & 2012
	W	C	S	N	E	NW	NC	U	Sab					
AFP*	01	00	00	01	01	00	00	01	00	04	01	90	68	+ 32.03 %
Diphtheria	00	00	00	00	00	00	00	00	00	-	-	-	-	-
Mumps	00	05	01	01	02	01	01	00	00	11	21	1347	4021	- 66.5 %
Measles	20	00	07	01	02	04	03	00	31	68	01	3561	59	+ 5935.6 %
Rubella	00	00	00	00	00	00	00	00	00	00	-	27	-	-
CRS**	00	00	00	00	00	00	00	00	00	00	-	06	-	-
Tetanus	01	00	00	00	00	00	00	00	00	01	01	22	12	+ 83.3 %
Neonatal Tetanus	00	00	00	00	00	00	00	00	00	00	-	00	-	-
Japanese Encephalitis	00	00	00	00	00	00	00	00	00	00	-	66	-	-
Whooping Cough	00	00	02	00	00	00	00	00	00	02	01	77	92	- 16.3 %
Tuberculosis	03	00	00	02	12	00	00	18	37	72	226	7088	7711	- 8.1 %

Key to Table 1 & 2

Provinces: **W**: Western, **C**: Central, **S**: Southern, **N**: North, **E**: East, **NC**: North Central, **NW**: North Western, **U**: Uva, **Sab**: Sabaragamuwa.

RDHS Divisions: **CB**: Colombo, **GM**: Gampaha, **KL**: Kalutara, **KD**: Kandy, **ML**: Matale, **NE**: Nuwara Eliya, **GL**: Galle, **HB**: Hambantota, **MT**: Matara, **JF**: Jaffna, **KN**: Killinochchi, **MN**: Mannar, **VA**: Vavuniya, **MU**: Mullaitivu, **BT**: Batticaloa, **AM**: Ampara, **TR**: Trincomalee, **KM**: Kalmunai, **KR**: Kurunegala, **PU**: Puttalam, **AP**: Anuradhapura, **PO**: Polonnaruwa, **BD**: Badulla, **MO**: Moneragala, **RP**: Ratnapura, **KG**: Kegalle.

Data Sources:

Weekly Return of Communicable Diseases: Diphtheria, Measles, Tetanus, Neonatal Tetanus, Whooping Cough, Chickenpox, Meningitis, Mumps., Rubella, CRS,

Special Surveillance: AFP* (Acute Flaccid Paralysis), Japanese Encephalitis

CRS =**Congenital Rubella Syndrome

AFP and all clinically confirmed Vaccine Preventable Diseases except Tuberculosis and Mumps should be investigated by the MOH

Dengue Prevention and Control Health Messages

Thoroughly clean the water collecting tanks bird baths, vases and other utensils once a week to prevent dengue mosquito breeding.

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Comments and contributions for publication in the WER Sri Lanka are welcome. However, the editor reserves the right to accept or reject items for publication. All correspondence should be mailed to The Editor, WER Sri Lanka, Epidemiological Unit, P.O. Box 1567, Colombo or sent by E-mail to **chepid@sltnet.lk. Prior approval should be obtained from the Epidemiology Unit before publishing data in this publication**

ON STATE SERVICE

Dr. P. PALIHAWADANA
CHIEF EPIDEMIOLOGIST
EPIDEMIOLOGY UNIT
231, DE SARAM PLACE
COLOMBO 10