WEEKLY EPIDEMIOLOGICAL REPORT

A publication of the Epidemiology Unit
Ministry of Health

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

Vol. 40 No.26 **22nd – 28th June 2013**

SRI LANKA - 2013 WER

Measles (Part II)

This is the second in a series of two articles on Measles

Gastro-intestinal Complications

Measles probably infects the intestinal tracts of most persons with measles. A gastric biopsy obtained the day before rash onset from a 44-year-old man revealed characteristic giant cells that were positive for measles by immunologic staining. Several cases of appendicitis have developed before and during measles rash, and characteristic giant cells typical for measles have been found in appendix tissue

Diarrhoea

Measles-associated diarrhoea typically begins just before the onset of the rash, suggesting that measles virus is responsible for most of the diarrhoea episodes but that secondary bacterial or viral infections may contribute to the severity and duration of illness (Stools of children with measles-associated diarrhoea usually have the same bacteria as those of children with diarrhoea which are not associated with measles)

High rates of gastrointestinal complications such as mouth sores, decreased food intake, protracted diarrhoea, weight loss , Noma (cancrum oris-a progressive oral lesion that destroys orofacial tissue) and precipitation of severe protein calorie malnutrition has been noted after measles. In young adults, measles is associated with hepatitis, hypocalcemia and elevation of creatinine phosphokinase levels.

Neurological Complications

Febrile seizures

Febrile seizures associated with measles are usually benign and not associated with residual damage. Most children with uncomplicated measles have changes visible on electroencephalography, but these changes are most likely due to fever and other metabolic changes. Post-infectious encephalomyelitis (PIE) occurs in 13 per 1000 infected persons, usually 3–10 days after onset of rash. Higher rates of PIE due to measles occur in adolescents and adults than in school-aged children. PIE usually begins with the abrupt onset of fever, seizures, altered mental status and multifocal neurological signs. Although measles virus was found in cerebrovascular endothelial cells in a person who died during the first few days of rash, the virus usually is not found in the central nervous systems of persons with PIE. PIE appears to be caused by an abnormal immune response that affects myelin basic protein.

As many as 25% of people with PIE due to measles die, and ~33% of survivors have lifelong neurological sequelae, including severe retardation, motor impairment, blindness and sometimes hemiparesis .

Subacute sclerosing panencephalitis (SSPE)

SSPE is caused by persistence of measles virus in central nervous system tissue for several years, followed by a slowly progressive infection and demyelination affecting multiple areas of the brain. The initial SSPE symptoms, usually decreased school performance and behavioural disorders, are often misdiagnosed as psychiatric problems. Subsequently, myoclonic seizures develop and a characteristic burst-suppression pattern may be seen on electroencephalography. Measles antibody is present in the cerebrospinal fluid. The disease slowly progresses until affected persons are in a vegetative state. Wild-type measles viruses, but not measles vaccine viruses, have been found in brain tissue. Factors responsible for persistence of measles virus in these persons are not known, nor is it known whether measles virus persists in otherwise normal hosts. Geographic clustering of SSPE oc-

Contents **Page**

1. *Leading Article – Measles (Part II)*	1
2. *Surveillance of vaccine preventable diseases & AFP (15th– 21st June 2013)*	3
3. *Summary of newly introduced notifiable diseases (15th– 21st June 2013)*	3
4. *Summary of selected notifiable diseases reported (15th– 21st June 2013)*	4

curs in several countries, and there is an increased incidence in children residing in rural areas. In 2 studies, children with SSPE had more close exposure to birds than did control subjects. These data suggest that as-yet-undefined environmental factors, most likely another infectious agent, contribute to this disease.

Measles encephalitis in immune-compromised patients
A progressive central nervous system measles virus infection, termed "measles inclusion body encephalitis," occurs in immune-compromised persons with disorders such as human immunodeficiency virus (HIV) infection or leukemia. Onset is usually 5 weeks to 6 months after acute measles. The illness begins with mental-status changes and seizures in the absence of fever; >80% of deaths occur within weeks.

Ocular Complications
Conjunctivitis occurs in most persons with measles, and inflammation of the cornea (keratitis) is common. In well-nourished persons, these lesions usually heal without residual damage. However, secondary bacterial (e.g., Pseudomonas or Staphylococcus) or viral infections (e.g., HSV or adenovirus) can lead to permanent scarring and blindness. Vitamin A deficiency predisposes to more severe keratitis, corneal scarring and blindness. Measles associated with vitamin A deficiency is one of the most common causes of acquired blindness in children in developing countries. Blindness can also result from cortical damage from measles encephalitis.

Other associations
Measles has been hypothesized to cause or contribute to multiple sclerosis, but available evidence is weak and inconclusive. Studies from different laboratories have had conflicting evidence for persistence of measles virus nucleocapsid in affected tissue from patients with otosclerosis, Paget's disease and inflammatory bowel disease

Factors affecting measles associated mortality and morbidity rates

Sex-Recent surveillance data show equal rates of complications for men and women. Pregnant women have an increased risk of complications, including death, following measles

Age-Complication rates, including mortality, from measles are highest in children <5 years and adults. Most infants are protected during the first months of life via maternally derived antibodies. However, when immunity is lacking, measles can be severe. Adults more commonly have encephalitis, hepatitis, hypocalcemia or pancreatitis after measles. The increased severity of measles in adults most likely reflects the decline in cell-mediated immunity that begins in adulthood. Some researchers found that young infants and adults have more severe and a longer duration of lymphopenia after measles than do children

Crowding-Several studies show that children who develop measles after within-household exposure have higher case-fatality rates than do children who are exposed to measles outside the household. This phenomenon is most likely secondary to a higher inoculum from more intensive and prolonged exposure compared with more casual exposures outside the home.

Immunosuppression-Children with defects in macrophage function only (e.g., chronic granulomatous disease) do not have increased rates of complications from measles. Suppression of lymphocyte function, resulting from congenital defects in T lymphocyte function, bone marrow transplantation and chemotherapy for cancer or immunosuppressive doses of steroids is associated with increased severity of measles. In a review of 40 measles cases in children with malignancies, 58% of children had pneumonitis, 20% had encephalitis, and 8% had both [99]. Only 60% of the case-patients had typical measles rash [99]. The fatality rate was 55% overall [99]. In some immunosuppressed patients with measles, multiple organ systems are affected 39, 40, 181–183]. Measles has developed after bone marrow transplantation even when both donor and recipient have histories of measles vaccination [104]. Patients with B cell immune deficiency syndromes without T cell abnormalities do not appear to have increased rates of complications associated with measles.

Children born to HIV-infected women become susceptible to measles at an earlier age than do children born to HIV-negative women because the former transmit reduced amounts of antibodies to their infants.
Malnutrition-Malnourished children have impairments in multiple aspects of the immune system, prolonged excretion of measles virus and higher measles case-fatality rates. Measles contributes to the development of malnutrition because of protein-losing enteropathy, increased metabolic demands and decreased food intake. Children who have measles early in life have significantly lower mean weights for age than do children of the same age who do not develop measles.

Vitamin A deficiency-Children with clinical or subclinical vitamin A deficiency in many developing countries have increased case-fatality rates. Measles and other illnesses are associated with reductions in serum retinol concentrations and may induce overt vitamin A deficiency. Hospitalized US measles patients frequently have deficiencies in vitamin A; these children are more likely to have pneumonia or diarrhoea after measles. In countries with high measles mortality, treatment with vitamin A once daily for 2 days (200,000 IU for children ⩾12 months of age or 100,000 IU for infants <12 months) is associated with an ~50% reduction in mortality. The World Health Organization recommends vitamin A therapy for all children with measles

Note-Increased numbers of Measles cases were identified since January 2013. Majority (60%) of the confirmed measles cases were below the age of 1 year, with the highest proportion of cases between 6 -12months. Supplementary Immunization Activity (SIA) with measles vaccination will be started in this age group (6-12 months) as an outbreak control activity with a National Immunization Day on the 5th July, 2013.

Source-The Clinical Significance of Measles, A Review, available from *http://jid.oxfordjournals.org/content/189/Supplement_1/S4.full*

Compiled by Dr. Madhava Gunasekera of the Epidemiology Unit

Table 4: Selected notifiable diseases reported by Medical Officers of Health 15th – 21st May 2013 (25th Week)

RDHS	Dengue Fever		Dysentery		Encephaliti		E Fever		F Poisoning		Leptospiros		T Fever		V Hepatitis		H Rabies		Chickenpox		Meningitis		Leishmaniasis		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	41	4085	3	96	0	13	3	67	1	21	2	125	0	5	0	40	0	0	1	244	0	29	0	0	**62**	**38**
Gampaha	38	1748	7	78	0	11	0	23	0	22	4	192	0	11	3	114	0	0	5	95	2	55	0	5	**73**	**27**
Kalutara	32	852	3	78	0	14	1	45	0	12	5	223	0	1	0	12	0	0	6	158	1	39	0	0	**85**	**15**
Kandy	54	931	5	71	0	6	0	12	0	7	1	41	1	74	2	57	0	0	2	83	1	6	0	2	**91**	**9**
Matale	7	232	2	48	0	2	0	8	0	0	0	40	0	2	2	25	0	0	0	29	0	16	1	3	**77**	**23**
NuwaraEliya	2	123	3	88	0	2	1	6	0	3	0	18	0	42	2	13	0	0	0	47	0	3	0	0	**92**	**8**
Galle	30	437	3	44	1	11	0	2	0	74	5	124	0	25	0	6	0	1	5	152	2	27	0	0	**79**	**21**
Hambantota	4	168	0	23	0	2	0	7	0	11	1	134	0	38	0	64	0	0	0	62	0	14	9	142	**83**	**17**
Matara	12	271	5	41	0	9	1	15	0	27	1	107	1	39	1	112	0	2	2	171	1	35	2	52	**100**	**0**
Jaffna	8	468	4	113	0	5	7	253	0	76	0	6	4	318	0	10	0	0	2	113	3	34	0	0	**92**	**8**
Kilinochchi	0	27	0	13	0	0	0	6	0	2	0	9	0	15	0	0	0	0	0	2	0	7	0	5	**0**	**100**
Mannar	0	55	2	27	0	1	0	52	0	11	0	11	0	16	1	2	0	0	0	11	0	4	0	1	**80**	**20**
Vavuniya	2	47	0	25	0	10	1	7	0	8	0	46	0	2	0	1	0	2	0	18	1	21	0	4	**100**	**0**
Mullaitivu	0	82	0	6	0	1	0	6	1	5	1	24	0	5	0	0	0	2	0	3	0	3	0	8	**60**	**40**
Batticaloa	15	396	5	147	0	3	0	0	1	14	0	23	0	2	0	8	0	0	1	21	0	2	0	0	**79**	**21**
Ampara	1	72	1	45	0	0	0	4	0	2	0	17	0	0	0	2	0	0	1	49	0	7	0	1	**43**	**57**
Trincomalee	1	149	0	37	0	3	0	4	0	1	0	49	1	7	0	3	0	1	0	25	0	2	0	14	**67**	**33**
Kurunegala	30	1937	1	100	0	25	0	26	0	8	1	180	1	18	0	31	0	1	6	215	4	75	1	26	**81**	**19**
Puttalam	18	588	3	32	0	4	0	11	0	35	0	16	0	10	0	1	0	0	1	49	4	15	0	3	**62**	**38**
Anuradhapura	3	335	1	50	0	12	0	3	0	4	0	266	0	15	0	13	0	0	2	93	6	64	2	192	**63**	**37**
Polonnaruwa	4	204	0	39	0	1	1	12	32	53	6	135	0	2	0	19	0	1	1	86	0	10	3	78	**100**	**0**
Badulla	11	239	5	85	0	3	0	10	0	7	4	24	3	45	5	30	0	0	5	76	6	29	1	4	**82**	**18**
Monaragala	3	129	1	56	0	3	1	12	0	18	0	175	0	26	3	49	0	1	0	33	0	10	0	6	**82**	**18**
Ratnapura	35	1108	4	226	0	80	1	30	0	16	3	219	0	19	5	148	0	1	3	91	2	46	0	8	**89**	**11**
Kegalle	22	626	8	48	0	10	0	10	0	4	6	107	1	53	5	137	0	0	9	194	1	61	0	0	**100**	**0**
Kalmune	3	468	1	80	0	1	0	3	0	66	0	4	0	2	0	4	0	0	1	54	0	6	0	1	**62**	**38**
SRI LANKA	**376**	**15777**	**67**	**1696**	**01**	**232**	**17**	**634**	**35**	**507**	**40**	**2315**	**12**	**792**	**29**	**901**	**00**	**12**	**53**	**2174**	**34**	**620**	**19**	**555**	**79**	**21**

Source: Weekly Returns of Communicable Diseases (WRCD).

***T=**Timeliness refers to returns received on or before 21st June , 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

15th – 21st May 2013 (25th 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*	02	00	00	00	00	00	00	00	00	02	02	42	42	0 %
Diphtheria	00	00	00	00	00	00	00	00	00	-	-	-	-	-
Mumps	06	07	01	07	01	03	01	01	01	28	03	795	2037	- 60.9 %
Measles	18	11	32	01	00	01	02	01	07	73	00	790	23	+ 3334.7 %
Rubella	01	00	00	00	00	00	00	00	00	00	-	13	-	-
CRS**	00	00	00	00	00	00	00	00	00	00	-	06	-	-
Tetanus	00	00	00	00	00	00	00	00	00	00	00	10	05	+ 100.0 %
Neonatal Tetanus	00	00	00	00	00	00	00	00	00	00	-	00	-	-
Japanese Encephalitis	00	00	01	00	00	00	00	00	00	01	-	232	-	-
Whooping Cough	00	00	01	00	00	00	01	00	01	03	00	41	34	+ 20.6 %
Tuberculosis	04	04	00	03	05	01	07	00	25	49	155	3909	4223	+ 07.4 %

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

To prevent dengue, remove mosquito breeding places in and around your home, workplace or school once a week.

PRINTING OF THIS PUBLICATION IS FUNDED BY THE WORLD HEALTH ORGANIZATION (WHO).

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