WEEKLY EPIDEMIOLOGICAL REPORT

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WER SRI LANKA 2008

Meningococcal meningitis - Part I

During the last few years few isolated clinically suspected meningococcal meningitis cases were reported mainly from some Paediatrics Units in Sri Lanka.

In this series of articles we hope to discuss the epidemiology, clinical picture, diagnosis, treatment and prevention of meningococcal meningitis.

Meningitis is an infection of the meninges, the thin lining that surrounds the brain and the spinal cord. Several different bacteria can cause meningitis out of that Neisseria meningitidis being one of the most important because of its potential to cause epidemics. Meningococcal disease was first described in 1805 when an outbreak swept through Geneva, Switzerland. The causative agent, Neisseria meningitidis (the meningococcus), was identified in 1887.

Twelve subtypes or serogroups of N. meningitidis have been identified and four (N. meningitidis. A, B, C and W135) are recognized to cause epidemics. The pathogenicity, immunogenicity, and epidemic capabilities differ according to the serogroup. Thus the identification of the serogroup responsible for a sporadic case is crucial for epidemic containment.

Meningococcal disease is caused by the gram-negative bacterium *Neisseria meningitidis*, also known as meningococcus. Infection occurs both endemically and epidemically, in developed and developing countries. The impact of the disease persists due to the lack of effective control measures necessary to significantly decrease the number of asymptomatic carriers. For every case of meningococcal disease there are hundreds of persons in normal conditions with upper respiratory tract colonization. Humans are the only reservoir in nature.

Why a particular individual colonized by the microorganism develops infection while others who are equally colonized develop immunity to infection is not known. There are two main forms of clinical manifestation of the disease meningococcal meningitis, which has a good prognosis if it is adequately treated and meningococcemia or *Meningococcal septicemia*, which is less frequent and highly lethal even when treated. It is characterized by positive blood cultures and an exaggerated systemic inflammatory response, associated with endotoxemia. Cases of simultaneous meningitis and bacteremia are generally considered as cases of meningitis. *Meningococcal septicemia* is considered a medical emergency and can result in death rapidly.

Epidemiology of Meningococcal Disease

Meningococcal meningitis occurs sporadically in small clusters throughout the world with seasonal variations and accounts for a variable proportion of endemic bacterial meningitis. In temperate regions the number of cases increases in winter and spring. Serogroups B and C together account for a large majority of cases in Europe and the Americas. Several local outbreaks due to N. meningitidis serogroup C have been re-

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Major African epidemics are associated with N. meningitidis serogroups A and C and serogroup A is usually the cause of meningococcal disease in Asia. Outside Africa, only Mongolia reported a large epidemic in the recent years (1994-95). There is increasing evidence of serogroup W135 being associated with outbreaks of considerable size. In 2000 and 2001 several hundred pilgrims attending the Hajj in Saudia Arabia were infected with N. meningitidis W135. Then in 2002, W135 emerged in Burkina Faso, striking 13,000 people and killing 1,500.

In Delhi the outbreaks have occurred at regular intervals in 1935, 1966-67 and in1985-86. In 1966, 616 cases were recorded with peaks - May and December. In 1985, 1731 cases with 569 deaths and in 1986, 6133 cases of meningitis with 799 deaths were documented. Overall case fatality rate was 13%. In Delhi during the past three years (2002-2004) the number of cases, have been 971 with 118 deaths. It is obvious that the majority of the cases are adolescents and young adults. Most of the cases were from crowded inner city areas.

Transmission mechanisms

Transmission results from person-to-person contact or from inhalation of respiratory droplets containing meningo-cocci. It does not survive in the environment or in animals, is vulnerable to temperature changes and desiccation. Coughing and sneezing contribute to the transmission mechanism . The colonization rate is greater than 50% during periods associated with an increase in viral infections and upper respiratory tract infections.

The carrier rate is low during childhood and high in adolescents and young adults. Transmission is relatively slow in open populations and is greater in isolated populations and is aggravated by smoking or respiratory tract infections.

Health workers may contract the disease only when directly exposed to the patient's secretions. It has been determined that the risk of the sibling of a child for being infected is 2 to 3% and the attack rate for persons living in the same household is 2 to 4 per 1000 subjects. In outbreaks, colonization may be subsequent or simultaneous by the same or different serogroups.

When cases occur in schools, a student's risk of becoming infected ranges between 0.04% and 2.5%, with a higher risk in middle schools than in elementary schools. The variation in the attack rate is representative of the variation in the established control measures, but it also depends on factors related to the bacterium, the environment, and the host . The distance between student's seats has proved a risk factor for colonization. Similar situations are found in prisons. Outbreaks due to type C serogroup have been identified in these institutions and associated with 40% overpopulation.

Microbiological characteristics and pathogenesis

N. meningitidis is a gram negative, aerobic, immobile, non-sporulated bacterium; it is usually encapsulated and has pilli. It is classified in serogroups according to the immune reactivity of its capsular poly-saccharide, which is the basis of the polysaccharide vaccines currently available. Serogroup B contains a polysaccharide of low immunogenicity, probably due to its polysialic acid content. This acid is also present in human fetal neurons. Meningococcus can change from serogroup B to C or vice versa. The pathogenic process of *N. meningitidis* begins when the bacterium adheres to the surface of the microvilli of the cylindrical non-ciliated epithelium of the nasopharynx, where it reproduces.

Most subjects colonized by *N. meningitidis* remain asymptomatic. However, a lower percentage of meningococci enters the mucosa and the circulatory system, causing systemic disease. An increase in the incidence of meningococcal disease in a given population reflects the introduction, transmission, and acquisition of a virulent strain of a clonal group previously inexistent in a susceptible population. These bacterial strains produce a protective capsule that aids in evading the host's immune response, particularly the activation of complement-mediated and antibody-dependent lysis. Individuals with a deficiency of complement mediated antibody-dependent bactericidal system are susceptible to meningococcal infection. Predisposed individuals include people who have been splenectomized, or with functional asplenia, properdine deficiency, or deficiency of the terminal component of the complement's cascade. However, although these predisposed individuals have an increased risk of meningococcal disease, they represent only a small proportion of total cases . It is thought that AIDS patients may also have an increased risk for infection, although not as high as compared with other encapsulated organisms. Other genetic characteristics have been associated with an increased risk of the disease, including polymorphisms in the genes for lectin, associated to mannose, and in the genes for tumor necrosis factor alpha.

Active or passive exposure to cigarette smoke, viral infections of upper respiratory tract, damage of the respi-

Table 1: Vaccine-preventable Diseases & AFP

16th - 22nd August 2008 (34th Week)

Disease	No. of Cases by Province									Number of cases during current week in 2008	Number of cases during same week in 2007	Total number of cases to date in 2008	Total number of cases to date in 2007	Difference between the number of cases to date between 2008 & 2007
	W	C	S	N	E	NW	NC	U	Sab					
Acute Flaccid Paralysis	01 GM=1	01 NE=1	00	00	00	00	00	00	01 RP=1	03	00	67	59	+13.6%
Diphtheria	00	00	00	00	00	00	00	00	00	00	00	00	00	00.0%
Measles	00	00	00	00	00	00	00	00	00	00	00	85	50	+70.0%
Tetanus	00	00	00	00	00	00	00	00	01 RP=1	01	01	26	24	+8.3%
Whooping Cough	00	00	00	00	00	01 PU=1	00	00	00	01	01	30	31	-3.2%
Tuberculosis	124	49	14	11	11	00	38	10	11	265	106	5998	6624	-9.5`%

Table 2: Newly Introduced Notifiable Diseases

16th - 22nd August 2008 (34th Week)

Disease	No. of Cases by Province									Number of cases during current week in 2008	Number of cases during same week in 2007	Total number of cases to date in 2008	Total number of cases to date in 2007	Difference between the number of cases to date between 2008 & 2007
	W	C	S	N	E	NW	NC	U	Sab					
Chicken-pox	18	11	18	00	19	10	02	06	11	95	33	3640	2263	+60.8%
Meningitis	04 GM=1 KL=2 CB=1	03 ML=3	01 HB=1	00	00	02 KR=2	01 AP=1	01 BD =1	01 KG=1	13	23	919	345	+166.3%
Mumps	13	04	12	18	07	05	05	03	05	72	41	1914	1492	+28.4%

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.
DPDHS 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.

Table 3: Laboratory Surveillance of Dengue Fever

16th - 22nd August 2008 (34th Week)

Samples	Number tested		Number positive *		Serotypes									
					D_1		D_2		D_3		D_4		Negative	
	GT	AH	GT	AH	GT	AH	GT	AH	GT	AH	GT	AH	GT	AH
Number for current week	04	02	00	00	00	00	00	00	00	00	00	00	00	00
Total number to date in 2008	124	130	09	22	00	00	06	08	01	08	00	00	02	00

Sources: Genetech Molecular Diagnostics & School of Gene Technology, Colombo [GT] and Genetic Laboratory Asiri Surgical Hospital [AH]
* Not all positives are subjected to serotyping.
NA= Not Available.

Data Sources:
Weekly Return of Communicable Diseases: Diphtheria, Measles, Tetanus, Whooping Cough, Human Rabies, Dengue Haemorrhagic Fever, Japanese Encephalitis, Chickenpox, Meningitis, Mumps.
Special Surveillance: Acute Flaccid Paralysis.
National Control Program for Tuberculosis and Chest Diseases: Tuberculosis.

Table 4: Selected notifiable diseases reported by Medical Officers of Health

16th - 22nd August 2008 (34th Week)

DPDHS Division	Dengue Fever / DHF*		Dysentery		Encephal-itis		Enteric Fever		Food Poisoning		Leptos-pirosis		Typhus Fever		Viral Hepatitis		Human-Rabies		Re-turns Re-ceive
	A	B	A	B	A	B	A	B	A	B	A	B	A	B	A	B	A	B	%
Colombo	24	1210	16	153	02	11	03	74	01	76	15	304	00	02	03	89	00	00	**92**
Gampaha	15	737	02	141	01	16	01	39	00	95	39	335	01	06	01	101	00	04	**79**
Kalutara	06	353	08	238	00	10	01	45	00	18	08	343	00	02	01	33	00	01	**92**
Kandy	07	188	03	221	00	05	01	44	00	53	05	323	02	79	01	96	00	01	**72**
Matale	04	86	03	156	00	02	01	36	00	04	02	613	00	01	00	23	00	00	**67**
Nuwara Eliya	01	22	03	184	00	02	01	198	06	164	00	39	00	35	02	89	00	01	**77**
Galle	01	84	04	136	00	12	00	13	00	43	05	246	00	12	00	06	00	03	**88**
Hambantota	03	73	00	68	00	05	00	07	00	11	02	74	05	71	01	13	00	00	**91**
Matara	08	210	01	138	01	11	01	29	00	06	16	257	07	154	01	12	00	01	**88**
Jaffna	00	52	01	102	00	02	01	223	00	11	00	00	00	150	00	32	00	00	**50**
Kilinochchi	00	00	00	26	00	00	00	01	00	04	00	02	00	00	00	01	00	00	**00**
Mannar	00	25	00	15	00	06	00	145	00	00	00	00	00	01	00	13	00	00	**75**
Vavuniya	00	11	01	46	00	02	02	08	00	14	00	05	00	01	00	05	00	00	**75**
Mullaitivu	00	00	00	09	00	00	00	13	00	13	00	00	00	01	00	09	00	00	**00**
Batticaloa	00	85	02	92	00	04	00	20	00	20	00	05	00	01	01	83	00	05	**82**
Ampara	00	28	03	224	00	00	00	07	00	01	02	20	00	00	00	08	00	00	**29**
Trincomalee	00	176	02	72	00	00	00	13	00	12	01	29	00	16	00	12	00	00	**90**
Kurunegala	07	266	01	171	00	14	03	47	00	14	69	331	01	22	01	54	00	04	**89**
Puttalam	00	271	02	63	00	08	00	137	00	26	03	35	00	33	01	28	00	03	**78**
Anuradhapur	02	111	00	64	00	09	00	10	00	06	00	223	00	10	00	12	00	02	**53**
Polonnaruwa	00	59	05	94	00	01	00	21	05	12	00	55	00	01	00	18	00	00	**100**
Badulla	03	69	11	349	00	05	04	105	80	93	06	39	02	100	06	108	00	01	**100**
Monaragala	00	49	01	281	00	03	03	32	00	116	00	87	02	78	00	38	00	00	**73**
Ratnapura	05	218	08	244	01	27	00	42	00	51	00	128	00	75	01	46	00	00	**69**
Kegalle	03	321	03	234	00	24	01	52	00	04	15	256	02	55	02	435	00	01	**82**
Kalmunai	00	33	08	209	00	02	00	09	01	16	00	00	00	02	00	21	00	00	**77**
SRI LANKA	**89**	**4732**	**88**	**3730**	**05**	**181**	**23**	**1370**	**93**	**883**	**188**	**3749**	**22**	**908**	**22**	**1385**	**00**	**27**	**76**

Source: Weekly Returns of Communicable Diseases (WRCD).

***Dengue Fever / DHF** refers to Dengue Fever / Dengue Haemorrhagic Fever.

****Timely** refers to returns received on or before 30 August, 2008 Total number of reporting units =238. Number of reporting units data provided for the current week: 233

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