WEEKLY EPIDEMIOLOGICAL REPORT

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Japanese Encephalitis (Part I)

This is the first in a series of two articles on Japanese Encephalitis.

Japanese encephalitis (JE) is numerically one of the most important causes of viral encephalitis worldwide, with an estimated 50,000 cases and 15,000 deaths annually. About one third of patients die and half of the survivors have severe neuropshychiatric sequelae. Most of China, Southeast Asia, and the Indian subcontinent are affected by the virus.

Historical perspective

Epidemics of encephalitis were described in Japan from the 1870s onwards. Major epidemics were reported about every 10 years, with more than 6, 000 cases reported in the 1924 epidemic. The term type B encephalitis was originally used to distinguish these summer epidemics from von Economo's encephalitis lethargica (sleeping sickness, known as type A), but the B has since been dropped. The prototype Nakayama strain of Japanese encephalitis virus was isolated from the brain of a fatal case in 1935. The virus was later classed as a member of the genus Flavivirus (family *Flaviviridae*) named after the prototype yellow fever virus (Latin; yellow=flavi). Although of no taxonomic significance, the ecological term arbovirus is often used to describe the fact that JE virus is insect (arthropod) borne.

Epidemiology

Neurotropic Flaviviruses-A Global Perspective

JE virus transmission occurs across eastern and southern Asia and the Pacific rim. However, related neurotropic flaviviruses are found across the globe; they share many virological, epidemiological and clinical features. Molecular virological studies suggest that all flaviviruses derived from a common ancestor some 10–20,000 years ago and are rapidly evolving to fill ecological niches. Examples of mosquito borne neurotropic flaviviruses include Murray Valley encephalitis virus in Australia, and St Louis encephalitis virus in North America.

In northern Europe and northern Asia, flaviviruses have evolved to use ticks as vectors because they are more abundant than mosquitoes in cooler climates.

Geographical Distribution

In the past 50 years the geographical area affected by JE virus has expanded. Differences in diagnostic capabilities and in reporting of encephalitis make it impossible to plot this expansion precisely. However, the timing of the first reported cases or new epidemics in each area gives an impression of the relentless spread of Japanese encephalitis. There are currently 10–20,000 cases a year, although in the early 1970s it was over 80,000 cases annually. In the far eastern Russian states, JE first occurred in 1938. In 1949, large epidemics were reported from South Korea for the first time. Epidemics in northern Vietnam followed in 1965 and in northern Thailand in 1969. Japanese encephalitis was recognised in southern India from 1955, but was confined to the south until the 1970s. Since then, large outbreaks (2,000–7,000 cases a year) have been reported from eastern and north-eastern states. The fact that adults and children were equally affected in these Indian states strongly supports the idea that the virus was introduced here for the first time. The late 1970s also saw the first cases in Burma and Bangladesh, and large epidemics in south-western Nepal. In 1985, Sri Lanka experienced its first epidemic with 410 cases and 75 deaths. JE virus continues to spread west with cases occurring in Pakistan and epidemics in the Kathmandu valley of Nepal.

Charting the progression of the disease southeast across Asia and the Pacific rim is harder because sporadic cases in endemic areas do not command the same attention as the massive epidemics that occur in temperate climates. The disease has occurred on the western Pacific islands with outbreaks in Guam in 1947 and Saipan in 1990. In Malaysia the disease is endemic; the virus was first isolated in the 1960s and about 100 cases are recorded annually. The epidemiology has recently been complicated by a superimposed epidemic of Nipah virus encephalitis. JE is endemic in Indonesia and 1,000–2,500 cases of encephalitis are reported annually, although in most the aetiological agent is not confirmed. Further

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east, Japanese encephalitis occurs sporadically in the Philippines and New Guinea. The first cases occurred in the Australian Torres Straits islands in 1995 and it was reported for the first time north of Cairns on the Australian mainland in 1998.

The reasons for the spread of JE are incompletely understood, but probably include changing agricultural practices, such as increasing irrigation (which allows mosquito breeding) and animal husbandry (which provides host animals). In Indonesia, the lower prevalence of antibody to Japanese encephalitis virus in Borneo than neighbouring Bali has been attributed to the lack of pigs in this predominantly Moslem culture. In developed countries such as Japan, Taiwan, and South Korea the number of cases has fallen, probably due to a combination of mass vaccination of children, spraying of pesticides, changing pig rearing practices, separation of housing from farming, better housing with air conditioning and less availability of mosquito breeding pools. However, in Korea the widespread use of vaccine in children has been associated with a higher incidence of Japanese encephalitis in those over 15 years.

Enzootic Cycle

JE virus is transmitted naturally between wild and domestic birds and pigs by Culex mosquitoes—the most important for human infection being *Culex tritaeniorrhynchus* which breeds in pools of stagnant water (such as rice paddy fields).In addition, Culicine mosquito species such asCx. gelidus, Cx.vishnui, Cx.pseudovishnui and Cx fuscocephala can also transmit the virus.

Although many animals can be infected with the JE virus, only those which develop high viraemias are important in the natural cycle. As well as maintaining and amplifying JE virus in the environment, birds may also be responsible for the spread to new geographical areas. Pigs are the most important natural host for transmission to humans, because they are often kept close to humans, have prolonged and high viraemias and produce many offsprings—thus providing a continuous supply of previously uninfected new hosts. The virus does not typically cause encephalitis in these natural hosts (pigs) although abortions occur in pregnant sows.

Epidemiology of Human Disease

Humans become infected with JE virus coincidentally when living or travelling in close proximity to the enzootic cycle of the virus. Although most cases occur in rural areas, JE virus is also found on the edge of cities. Epidemiological studies have shown that after the monsoon rains mosquitoes breed prolifically and as their numbers grow, so does their carriage of JE virus and the infection rate of pigs. Human infection soon follows. In sentinel studies, previously unexposed pigs placed in endemic areas were infected with the virus within weeks.

Although the virus has occasionally been isolated from human peripheral blood, viraemia is usually brief and titres are low; thus humans are considered dead end hosts from which transmission does not normally occur. Cross sectional serological surveys have shown that in rural Asia, most of the population is infected with JE virus during childhood or early adulthood. About 10% of the susceptible population is infected each year. However, most infections of humans are asymptomatic or result in a non-specific flu-like illness; estimates of the ratio of symptomatic to asymptomatic infection vary between 1 to 25 and 1 to 1,000.

JE is mostly a disease of children and young adults. In northern Thailand the incidence has been estimated to be up to 40 per 100,000 for ages 5 to 25, declining to almost zero for those over 35. The incidence is lower among young children (<3 years old) than in older children, possibly reflecting behavioural factors—for example, playing outside after dusk. When epidemics first occur in new locations, adults are also affected.

Broadly speaking, two epidemiological patterns of JE are recognised. In northern areas (northern Vietnam, northern Thailand, Korea, Japan, Taiwan, China, Nepal, and northern India) huge epidemics occur during the summer months, whereas in southern areas (southern Vietnam, southern Thailand, Indonesia, Malaysia, Philippines, Sri Lanka and southern India) JE tends to be endemic and cases occur sporadically throughout the year with a peak after the start of the rainy season.

Virology

In common with all flaviviruses, Japanese encephalitis virus has a small (50 nm) lipoprotein envelope surrounding a nucleocapsid comprising of core protein and 11 kb single stranded RNA (3800 kD). At least five genotypes of Japanese encephalitis virus occur in Asia, which relate roughly to the geographical area of isolation. The complete nucleotide sequence has been published, and includes 5′ and 3′ untranslated regions and a single open reading frame encoding genes for three structural proteins (capsid protein (C); precursor to the membrane M protein (PrM); and envelope protein (E)) and seven non-structural proteins. The search for genetic determinants of virulence in animal models of flavivirus encephalitis has focused on the E protein.

Pathogenesis

Only about 1 in 25 to 1 in 1,000 humans infected with JE virus develop clinical features of infection. These may range from a mild flu-like illness to a fatal meningoencephalomyelitis. The factors determining which of all the humans infected develop disease are unknown, but could include viral factors such as route of entry, titre and neurovirulence of the inoculum, and host factors such as age, genetic make up, general health, and pre-existing immunity.

After the bite of an infected mosquito, the virus is thought to amplify peripherally, causing a transient viraemia before invading the CNS. Based on data from mice and macaque monkeys, the site of peripheral amplification is thought to be dermal tissue and then lymph nodes. The means by which JE virus crosses the blood-brain barrier is unknown. However, immunohistochemical staining of human postmortem material has shown diffuse infection throughout the brain, indicating a haematogenous route of entry. Although experimental evidence suggest that replication within endothelial cells may be an important means of crossing the blood-brain barrier in some flaviviruses, for JE virus passive transfer across the endothelial cells seems a more likely mechanism. Other factors which compromise the integrity of the blood-brain barrier have also been implicated as risk factors for neuroinvasion. In several studies, a disproportionate number of fatal cases had neurocysticercosis at necropsy. It has also been suggested that head trauma (for example, due to a road traffic accident) during the transient viraemia could facilitate viral entry into the CNS.

Electron microscopic studies of the brains of infected mice show that the virus replicates in the rough endoplasmic reticulum and golgi apparatus. There is hypertrophy of the endoplasmic reticulum and degeneration into cyctic structures causing extensive dysfunction

Source

Japanese encephalitis, available from http://jnnp.bmj.com/content/68/4/405.full

Compiled by Dr. Madhava Gunasekera of the Epidemiology Unit

to be continued

Table 1: Vaccine-preventable Diseases & AFP

19th – 25th January 2013 (04th 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					
Acute Flaccid Paralysis	01	00	00	01	01	00	01	00	00	04	02	07	08	- 12.5 %
Diphtheria	00	00	00	00	00	00	00	00	00	-	-	-	-	-
Measles	05	00	00	00	00	00	00	00	00	05	01	18	03	+ 500.0%
Tetanus	00	00	00	00	00	00	00	00	00	00	01	02	02	%
Whooping Cough	00	00	01	00	00	00	00	00	00	01	02	06	05	+ 20.0 %
Tuberculosis	44	00	15	08	02	06	09	12	18	114	157	606	726	- 16.6 %

Table 2: Newly Introduced Notifiable Disease

19th – 25th January 2013 (04th 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					
Chickenpox	28	05	14	12	03	06	09	02	11	90	57	301	205	+ 46.8 %
Meningitis	03 GM=1 CB=2	00	06 MT=2 GL=4	02 JF=1 VU=1	00	00	01 AP=1	01 BD=1	04 RP=2 KG=2	17	06	86	41	+ 109.7 %
Mumps	03	01	06	00	04	00	02	00	02	18	57	117	240	+ 25.5 %
Leishmaniasis	00	00	09 HB=4 MT=5	00	00	01 KG=1	21 AP=4 PO=13	00	02 RP=2	29	27	88	44	+ 100.0%

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.

Data Sources:

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

Special Surveillance: Acute Flaccid Paralysis.

Leishmaniasis is notifiable only after the General Circular No: 02/102/2008 issued on 23 September 2008. .

Dengue Prevention and Control Health Messages

Reduce, Reuse or Recycle the plastic and polythene collected in your home and help to minimize dengue mosquito breeding.

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

19th – 25th January 2013 (04th Week)

DPDHS Division	Dengue Fever / DHF*		Dysentery		Encephalitis		Enteric Fever		Food Poisoning		Leptospirosis		Typhus Fever		Viral Hepatitis		Human Rabies		Returns Received
	A	B	A	B	A	B	A	B	A	B	A	B	A	B	A	B	A	B	%
Colombo	162	685	6	15	1	2	3	12	1	4	3	13	0	1	2	6	0	0	**77**
Gampaha	73	431	4	11	1	4	1	7	0	0	5	10	0	3	4	22	0	0	**73**
Kalutara	32	141	5	18	0	3	0	6	0	0	6	31	0	1	0	1	0	0	**69**
Kandy	13	34	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	**35**
Matale	5	43	3	15	0	0	0	0	0	0	1	1	1	1	0	7	0	0	**50**
Nuwara	4	15	1	5	0	0	0	1	0	1	0	0	1	8	0	0	0	0	**54**
Galle	4	47	1	4	0	2	0	0	0	1	2	10	2	5	2	2	0	0	**79**
Hambantota	3	28	1	6	0	0	0	1	0	0	1	9	3	8	2	15	0	0	**58**
Matara	16	48	1	2	1	2	0	1	0	2	0	8	1	2	2	43	1	1	**94**
Jaffna	16	95	2	17	0	1	12	53	0	0	0	0	16	72	2	3	0	0	**92**
Kilinochchi	1	4	0	2	0	0	0	2	0	1	0	1	0	0	0	0	0	0	**50**
Mannar	3	23	1	5	1	1	2	7	11	11	2	4	0	0	0	0	0	0	**100**
Vavuniya	0	11	3	9	1	5	1	2	1	3	0	5	0	0	0	0	0	0	**75**
Mullaitivu	0	11	1	1	0	0	0	1	0	0	1	2	0	2	0	0	0	0	**60**
Batticaloa	15	46	3	14	0	1	0	0	0	0	1	2	0	0	0	2	0	0	**79**
Ampara	5	13	0	13	0	0	0	1	0	0	1	3	0	0	0	0	0	0	**43**
Trincomalee	5	22	0	6	0	0	0	0	0	0	0	11	0	1	0	0	0	0	**67**
Kurunegala	130	666	3	23	1	1	1	7	0	0	0	10	2	5	0	2	0	0	**73**
Puttalam	22	123	1	8	0	1	0	2	0	1	0	2	0	0	0	0	0	0	**58**
Anuradhapu	6	68	0	4	0	4	0	0	0	0	2	10	0	3	0	2	0	0	**47**
Polonnaruw	21	34	9	17	0	0	1	3	0	0	24	38	0	0	1	2	0	0	**86**
Badulla	9	35	4	14	0	0	1	3	0	0	2	3	0	3	0	5	0	0	**71**
Monaragala	3	27	0	8	0	0	0	1	0	0	5	10	0	2	0	3	0	0	**45**
Ratnapura	26	109	7	36	9	29	0	5	0	2	2	12	0	1	0	27	0	1	**61**
Kegalle	28	133	0	4	0	2	0	2	0	2	1	8	4	8	2	20	0	0	**91**
Kalmune	5	77	0	5	0	1	0	0	0	2	0	1	0	0	0	1	0	0	**31**
SRI LANKA	**607**	**2969**	**56**	**263**	**15**	**59**	**22**	**117**	**13**	**30**	**59**	**204**	**30**	**126**	**17**	**163**	**01**	**02**	**65**

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 25th January, 2013 Total number of reporting units 336. Number of reporting units data provided for the current week: 218
A = Cases reported during the current week. **B** = Cumulative cases for the year.

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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.**

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