WEEKLY EPIDEMIOLOGICAL REPORT
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Blindness (Part 2)

4. Childhood blindness

Childhood blindness refers to a group of diseases and conditions occurring in childhood or early adolescence which if left untreated, result in blindness or severe visual impairment that are likely to be untreatable later in life. The major causes of blindness in children vary widely from region to region, being largely determined by socioeconomic development, and the availability of primary health care and eye care services. In high income countries, lesions of the optic nerve and higher visual pathways predominate as the cause of blindness, while corneal scarring from measles, vitamin A deficiency, the use of harmful traditional eye remedies, ophthalmia neonatorum, and rubella cataract are the major causes in low-income countries. Retinopathy of prematurity is an important cause in middle-income countries. Other significant causes in all countries are congenital abnormalities, such as cataract, glaucoma, and hereditary retinal dystrophies

According to Gilbert and Foster, the prevalence of blindness in children varies according to socioeconomic development and under-5 mortality rates. In low-income countries with high under-5 mortality rates, the prevalence may be as high as 1.5 per 1000 children, while in high-income countries with low under-5 mortality rates, the prevalence is around 0.3 per 1000 children. Using this correlation to estimate the prevalence of blindness in children, the number of blind children in the world is approximately 1.4 million. Approximately three-quarters of the world's blind children live in the poorest regions of Africa and Asia.

Prevention and treatment of childhood blindness is disease specific. For Vitamin A deficiency, at a cost of only 5 US cents a dose, vitamin A supplements reduce child mortality by up to 34% in areas where Vitamin A deficiency is a public health problem. As vitamin A deficiency manifests often during an outbreak of measles, properly planned and implemented national vaccination programmes against measles has reduced the prevalence of eye complications. In middle income countries, retinopathy of prematurity (ROP) is among the leading causes of blindness, the incidence of which can be reduced through availability and affordability of screening and curative services. Early treatment of cataract and glaucoma can be beneficial, while low vision devices are helpful in children with residual vision.

5. Refractive errors and low vision

Refractive errors include myopia (short-sightedness), and hyperopia (long-sightedness) with or without astigmatism (when the eye can sharply image a straight line lying only in one meridian).

For low vision, the following two definitions are in use:

- Low vision is visual acuity less than 6/18 and equal to or better than 3/60 in the better eye with best correction.
- A person with low vision is one who has impairment of visual functioning even after treatment and/or standard refractive correction, and has a visual acuity of less than 6/18 to light perception, or a visual field less than 10 degrees from the point of fixation, but who uses, or is potentially able to use, vision for the planning and/or execution of a task for which vision is essential.

Recent studies have confirmed the existence of a large burden of uncorrected refractive errors, although the interventions required are significantly cost effective and have an important impact on economic development and quality of life. Severe refractive errors have been estimated to account for about 5 million blind people. According to the most recent data available to WHO, there are an estimated 124 million people in the world with low vision. About a fourth of these would benefit from low vision services.

Refractive errors can be rectified with appropriate optical correction while people with low vision may be helped with low vision devices.

6. Diabetic retinopathy

Diabetic retinopathy is composed of a characteris-

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tic group of lesions found in the retina of individuals having had diabetes mellitus for several years. The abnormalities that characterize diabetic retinopathy occur in predictable progression with minor variations in the order of their appearance. Diabetic retinopathy is considered to be the result of vascular changes in the retinal circulation. In the early stages vascular occlusion and dilations occur. It progresses into a proliferative retinopathy with the growth of new blood vessels. Macular oedema can significantly decrease visual acuity.

There are important differences over the past few decades in diagnosis, medical care, socioeconomic factors and other risk factors that influence the prevalence and geographic distribution of diabetes and retinopathy as well. It is estimated that in 2002 diabetic retinopathy accounted for about 5% of world blindness, representing almost 5 million blind. As the incidence of diabetes gradually increases, there is the possibility that more individuals will suffer from eye complications which, if not properly managed, may lead to permanent eye damage.

Risk factors for diabetic retinopathy include duration of diabetes, level of glycemia, presence of high blood pressure, dependence on insulin, pregnancy, levels of selected serum lipids, nutritional and genetic factors. Medical interventions can decrease some of the risk to vision caused by diabetic retinopathy. The control of glycemia decreases the risk of the incidence and the progression of the retinopathy. If sight threatening retinopathy is present, timely laser photocoagulation of the retina decreases the risk of a subsequent severe visual lesion.

7. Glaucoma

Glaucoma can be regarded as a group of diseases that have as a common end point a characteristic optic neuropathy which is determined by both structural change and functional deficit. The medical understanding of the nature of glaucoma has changed profoundly in the past few years and a precise comprehensive definition and diagnostic criteria are yet to be finalized. There are several types of glaucoma, however, the two most common are primary open angle glaucoma (POAG), having a slow and insidious onset, and angle closure glaucoma (ACG), which is less common and tends to be more acute.

The number of persons estimated to be blind as a result of primary glaucoma is 4.5 million, accounting for slightly more than twelve per cent of all global blindness. Risk factors are those limited to the onset of disease and those associated with progressive worsening in already established disease. The primary risk factors that are linked to the individual and the onset of the disease are age and genetic predisposition. The incidence of POAG rises with age and its progression is more frequent in people of African origin. ACG is the common form of glaucoma in people of Asian origin.

There is little known about primary prevention of glaucoma, however, there are effective methods of medical and surgical treatment if the disease is diagnosed in its early stage. Through appropriate treatment, sight may be maintained; otherwise the progression of the condition leads eventually to severe restriction of the visual field and irreversible blindness.

8. Age related Macular Degeneration (AMD)

Age-related Macular Degeneration (AMD) is a condition affecting people over the age of 50 and involves the loss of the person's central field of vision. It occurs when the macular (or central) retina develops degenerative lesions. It is thought that cause is due to circulatory insufficiency leading to reduction in the blood flow to the macular area. Several forms of AMD exist.

Globally, AMD ranks third as a cause of visual impairment with a blindness prevalence of 8.7%. It is the primary cause of visual impairment in industrialized countries. The main risk factor is ageing. Other risk factors may include the use of tobacco, genetic tendencies, the degree of pigmentation (with light coloured eyes being at higher risk), arterial hypertension, the ultraviolet rays, and consumption of a non-balanced diet.

At present, there is neither prevention nor a cure. Palliative treatments which seem to be able to retard the progress include the use of lasers, dynamic phototherapy and sometimes surgery. Rehabilitative training of those with impaired vision includes the availability of bright lighting in the living and work spaces and the use of special aids for viewing and computer use.

9. Corneal opacities

Corneal visual impairment encompasses a wide variety of infectious and inflammatory eye diseases that cause scarring of the cornea, the clear membrane that covers the outside of the eye. Significant scarring ultimately leads to functional vision loss.

The 4th cause of blindness globally (5.1%), corneal blindness is one of the major causes of visual deficiency after cataract, glaucoma and age related macular degeneration (AMD). Trachoma is responsible for nearly 4.9 million blind, mainly as a result of corneal scarring and vascularization. Ocular trauma and corneal ulcerations are significant causes of corneal blindness. They are often underreported but they are estimated at 1.5 to 2.0 million new cases of unilateral blindness every year. Among the causes of childhood blindness (approximately 1.5 million cases in the world and 5 million children with visual impairment) appear xerophthalmia (350,000 cases per year), new-born conjunctivitis, and rarer ocular infections like herpes and keratoconjunctivitis.

Even though the control of onchocerciasis and leprosy are public health success stories, these diseases are still significant causes of blindness, affecting approximately 250,000 individuals each. Traditional eye medicines have also been implicated as a major risk factor in the current epidemic of corneal ulceration in developing countries.

Corneal visual impairment is encompasses a wide variety of infectious and inflammatory eye diseases that cause corneal scarring, which ultimately leads to functional vision loss.
Public health prevention programmes are the most cost-effective means of decreasing the global burden of corneal blindness Indeed, the only currently available curative treatment is the surgery, by graft of cornea.

10. Genetic eye diseases

Genetic eye diseases include a large number of ocular pathologies which have in common the transmission from parents to children by their genetic inheritance. All do not cause visual impairment.
Knowledge about genetic eye diseases has increased dramatically during the last twenty years. Although there are no global statistics which let us know the extent of the burden of visual impairment from genetic causes, it does seem that genetic eye pathology represents a significant percentage of the causes of blindness in industrialized countries.

The only current means of prevention of genetic eye pathology is genetic counseling. Treatment of genetic eye disorders is largely experimental, with the exception of surgeries on the cornea, lens and vitreous, which are well documented in certain cases. The best hopes for treatment, however, lie in the use of gene therapy, growth promotion therapies for degenerative diseases, and possibly the grafting of retinal cells.

Source : World Health Organization

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Table 1: Vaccine-preventable Diseases & AFP

10th - 16th July 2010(28th Week)

Disease	No. of Cases by Province									Number of cases during current week in 2010	Number of cases during same week in 2009	Total number of cases to date in 2010	Total number of cases to date in 2009	Difference between the number of cases to date in 2010 & 2009
	W	C	S	N	E	NW	NC	U	Sab					
Acute Flaccid Paralysis	01	00	00	00	00	00	00	00	00	01	00	48	43	+ 11.6 %
Diphtheria	00	00	00	00	00	00	00	00	00	00	00	00	00	-
Measles	00	00	00	01	00	00	00	00	00	01	02	56	65	- 13.8 %
Tetanus	00	00	00	00	00	00	00	00	00	00	00	14	15	- 06.7 %
Whooping Cough	00	01	00	00	00	00	00	01	00	02	01	16	30	- 46.7 %
Tuberculosis	20	12	23	06	11	22	22	00	16	132	187	4719	5386	- 12.4 %

Table 2: Newly Introduced Notifiable Disease

10th - 16th July 2010(28th Week)

Disease	No. of Cases by Province									Number of cases during current week in 2010	Number of cases during same week in 2009	Total number of cases to date in 2010	Total number of cases to date in 2009	Difference between the number of cases to date in 2010 & 2009
	W	C	S	N	E	NW	NC	U	Sab					
Chickenpox	12	02	10	00	02	03	03	01	03	36	179	1999	10751	- 81.4 %
Meningitis	01 GM=1	00	04 GL=1 HB=2 MT=1	00	03 TR=1 AP=2	05 KN=5	03 PO=2 AP=1	04 BD=4	05 KG=2 RP=3	25	21	1029	570	+ 80.6 %
Mumps	02	01	05	02	01	03	03	00	03	20	25	569	1042	- 45.4 %
Leishmaniasis	00	00	00	00	00	00	01 AP=1	00	00	01	05	166	461	- 64.0 %

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

Check the roof gutters regularly for water collection where dengue mosquitoes could breed.

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

10th - 16th July 2010(28th 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	257	3546	6	181	0	14	0	39	1	29	6	362	0	6	0	35	0	1	**77**
Gampaha	93	2650	11	91	2	17	1	28	1	11	2	234	0	8	0	56	0	4	**60**
Kalutara	35	1127	4	139	0	11	1	14	0	73	0	201	0	1	2	19	0	1	**75**
Kandy	38	1000	7	207	0	1	2	19	0	3	3	60	4	95	4	39	0	1	**91**
Matale	13	444	3	219	0	3	0	21	0	67	0	66	0	4	1	30	0	0	**67**
Nuwara	6	112	10	246	0	0	1	83	0	84	0	17	1	47	1	27	0	0	**100**
Galle	29	663	4	153	0	3	0	3	0	12	3	55	2	8	0	7	0	3	**79**
Hambantot	24	480	1	45	0	4	0	1	0	10	0	62	0	53	1	6	0	0	**73**
Matara	39	315	4	122	0	5	0	5	0	43	2	193	5	87	0	15	0	0	**100**
Jaffna	32	2436	9	171	0	3	9	389	1	7	0	1	0	108	0	43	0	2	**75**
Kilinochchi	0	5	0	4	0	0	1	4	0	1	0	0	0	0	0	0	0	0	**75**
Mannar	16	208	2	31	0	0	0	33	0	10	0	0	0	0	0	14	0	0	**60**
Vavuniya	2	521	0	26	0	2	0	37	0	8	0	2	0	1	0	10	0	1	**75**
Mullaitivu	0	2	0	1	0	0	0	1	0	0	0	0	0	0	0	0	0	0	**20**
Batticaloa	9	1104	7	99	0	3	0	16	0	28	0	10	0	2	0	4	0	2	**79**
Ampara	5	89	6	54	0	1	0	6	0	6	0	30	0	0	0	10	0	0	**57**
Trincomale	11	833	0	103	0	11	0	3	0	9	0	17	1	11	0	13	0	1	**60**
Kurunegala	82	900	13	189	1	15	2	23	0	9	6	218	2	33	3	73	0	3	**95**
Puttalam	26	751	10	78	0	6	0	40	0	124	0	57	0	0	0	20	0	1	**100**
Anuradhap	10	821	2	42	1	4	0	8	0	37	2	56	0	22	2	33	0	3	**79**
Polonnaru	18	313	4	53	0	1	1	5	0	7	2	49	0	1	0	33	0	0	**86**
Badulla	47	576	4	124	0	1	3	63	3	16	1	46	7	60	4	77	0	0	**80**
Monaragala	58	499	1	121	0	1	0	29	0	4	0	27	5	46	0	59	00	2	**73**
Ratnapura	63	1683	1	329	0	4	0	10	0	22	4	262	0	40	0	63	0	2	**61**
Kegalle	23	594	2	90	1	10	3	36	0	19	8	151	2	12	0	58	0	0	**91**
Kalmunai	0	487	4	150	0	1	0	5	0	2	0	0	0	0	1	10	0	1	**69**
SRI LANKA	**936**	**22159**	**115**	**3068**	**05**	**121**	**24**	**921**	**06**	**641**	**39**	**2176**	**29**	**645**	**19**	**754**	**00**	**28**	**78**

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 16th July, 2010 Total number of reporting units =311. Number of reporting units data provided for the current week: 249
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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