

WEEKLY EPIDEMIOLOGICAL REPORT A publication of the Epidemiology Unit Ministry of Health, Nutrition & Indigenous Medicine 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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10th- 16th August 2019

Human Papillomavirus (HPV) and Cervical Cancer Part I

This is the first in a series of two articles on Human papillomavirus (HPV)

Human papillomavirus (HPV) is the most common viral infection in the genital tract. Most sexually active women and men will be infected at some stage of their life while repeated infection also may occur. Skin to skin genital contact or sexual transmission is identified as modes of transmission. Both men and women are at the risk of acquiring infection shortly after becoming sexually active.

Historical Information

HPV was discovered in 1956 by a group of scientists. In 1984 Harald Zur Hausen discovered, cloned, and attributed cervical cancer to HPV 16 and 18. Through his research, he contributed to the production of the vaccines. As certain strain causes papilloma or viral warts thereby attributing virus was named as "HPV".

Natural History of HPV

HPV is a small DNA virus belonging to "Family Papillomaviridae". It consists of a genome of approximately 8000 base pairs. HPV targets the basal cells in the stratified squamous epithelium and the metaplastic cells at the squamocolumnar junction of the cervix. HPV may also infect the glandular epithelium of the endocervix thus resulting in glandular neoplasms as adenocarcinoma in situ or invasive adenocarcinoma. E6

and E 7 are the high-risk HPV types of two primary oncogenes. These two genes expressed early in the HPV life cycle is being indicated by the use of "E" designation. The products of the two genes amend the host-cell metabolism to support neoplastic development. E6 binds to host cell protein p53 which demeans it. Thereby it prevents the apoptosis of the infected host epithelial cells. As telomerase is also activated it further augments oncogenic changes. E7 protein too has a similar effect on cell metabolism which binds to retinoblastoma protein thus inhibiting its function. This disrupts the cell cycle. E6 and E7 proteins may cause chromosomal destabilization. It also inhibits cyclin-dependent kinase inhibitors and host interferons. Expression of HPV E6 and E7 is highly correlated with the type of cervical lesion. In low-grade lesions, E6 and E7 are expressed at low levels in the basal cells while higher levels in the upper layers of the epithelium. High-grade lesions E6 and E7 are expressed at high levels throughout the epithelium. HPV is in an episomal form in low-grade lesions while in higher grade lesions and cancer, the HPV DNA is more likely to be integrated into the host cell chromosome. Integration of HPV DNA into the host DNA boosts the cellular proliferation and the chance of malignancy.

Before cervical cancer occurs, an area on the cervix would have been abnormal for ten to fifteen years. The abnormal area is referred to as a precursor of cervical cancer or "precancer." Precancerous lesions are indicated as cervical intraepithelial neoplasia (CIN) and grade it as

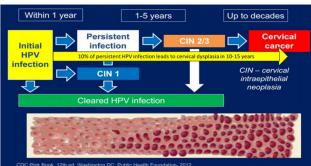
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mild, moderate, or severe (CIN 1, 2, or 3). Screening tests such as the Pap smear and visual inspection with acetic acid were designed to detect CIN.



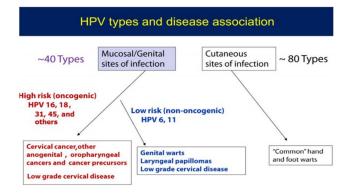


Types of HPV

There are more than 100 types of HPV. Most of them do not cause problems. HPV infections generally resolve within a few months of acquisition without any intervention while around 90% of the time it clears within 2 years.

Around 60 to 80 HPV types cause warts on the skin surface such as the hands or feet. Other 40 HPV types are contracted during sexual contact. The virus transmits to <u>mucous</u> membranes, mainly in moist layers around the mouth, throat, anus and genitals. All of these HPV viruses do not cause serious health implications. HPV 16 and 18 are high risk as they cause about 70% of cervical cancers. Other high-risk HPV viruses include 31, 33, 45, 52, 58, and a few others.

HPV strains 6 and 11 are low risk as 90% of <u>genital warts</u> are caused by them. These viral wart growths appear as bumps or as cauliflower shapes



Symptoms

Frequently there are no symptoms of HPV infection once the virus stays on mucus membrane. The body immune system clears the infection on its own while people never know they were infected.

Genital warts



These appear as flat lesions, small cauliflower-like bumps or tiny stem like protrusions. In women it appear frequently on the vulva but may occur near the anus, on the cervix or in the vagina.

In men it appears on the penis and scrotum or around the anus. Genital warts rarely cause discomfort or pain. There may be itch or tender and may hamper sexual life.

Cervical cancer

The high-risk types of HPV persist after acquiring on the surfaces or mucous membranes. It causes changes in the cells of the cervix which could lead to <u>cancer</u>. Thus Cervical cancer is the most common HPV related disease and almost 99% of cervical cancers can be attributed to any genotype of HPV.

There is a risk for women that HPV infection may become chronic. Several years with persistent HR-HPV develop as pre-cancerous lesions. The proportion of precancerous lesions can progress into invasive cervical cancer. In women with normal immune systems, the development of cervical cancer will take 15-20 years while in women with the weak-ened immune system it will take in 5-10 years.

HR-HPV can cause other cancers.

The genotypes causing cervical cancer may also contribute to cancers of the genitals as vulva, vagina or penis, anus, oral and upper respiratory tract

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Table 1: Selected notifiable diseases reported by Medical Officers of Health 03rd - 09th Aug 2019 (32nd Week)

Leishmania- WRCD sis	*0 *1	4 47	8 133 51 98	0 3 62 100	1 35 63 100	2 156 56 100	0 0 26 100	0 3 60 100	3 555 73 100	13 374 59 100	0 0 24 93	2 11 49 100	0 1 56 100	0 1 55 100	0 4 31 87	0 0 51 100	0 4 58 100	0 1 30 100	11 538 59 100	0 8 61 100	10 361 42 100	3 196 61 100	0 12 63 100	0 22 60 97	1 111 45 100	2 29 67 100	0 0 63 100	
	B	35	15	75	46	4	29	36	29	14 1	15	7	1	6	9	22	7	7	72 1	40	65 1	14	143	112	118	39	16	076 E7
Meningitis	٩	-	7 0	2 1	9 1	0	8	0 2	0 2	0 2	1 0	7 0	0 0	4	8 0	6 1	8	9 1	7 2	5 1	0 2	2	0 7	2 0	2 4	4	0	000
Chickenpox	-	5 324	3 297	8 472	4 189	0 63	2 88	0 310	3 230	5 210	2 221	1	0	1 64	m	2 196	6 208	4 189	5 447	2 115	6 400	1 235	8 220	0 212	7 262	9 334	4 173	
	٩			-	2	2	0	0 10	-	0	0	0	0	0	0	-	0	, 1	2	0	2	2	0	0	4	0	0	
Human Rabies	AB	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	¢
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Typhus Fever	A	0	0	0	m	0		0	m	2	1 2	0	0	0	0	0	0	0	2	0	0	0	m	0	1	1	0	0 7 7
		144	67	358	51	39	35	283	86	256	23	18	1	49	20	41	32	10	118	29	95	60	146	189	622	155	25	100
Leptospirosis	A	9	7	10	4	1	0	9	7	6	0	0	0	0	0	0	-	0	2	0	-	1	4	0	25	4	0	6
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Dengue Fever	8	8052		3865	2328	368	146	4101	1044	1916	2098	119	78	207	108	1025	164	914	1255	544	416	231	563	333	1852	1060	569	01000
Dengue	A	391	372	234	134	15	ω	253	94	130	23	1	0	2	1	15	8	12	63	30	1 26	9	20	0	95	65	11	1000
RDHS Division		Colombo	Gampaha	Kalutara	Kandy	Matale	NuwaraEliya	Galle	Hambantota	Matara	Jaffna	Kilinochchi	Mannar	Vavuniya	Mullaitivu	Batticaloa	Ampara	Trincomalee	Kurunegala	Puttalam	Anuradhapura	Polonnaruwa	Badulla	Monaragala	Ratnapura	Kegalle	Kalmune	SDTI ANKA

Source: Weekly Returns of Communicable Diseases (WRCD). •1=Timeliness refers to returns received on or before 09th August , 2019 Total number of reporting units 353 Number of reporting units data provided for the current week: 331 C**-Completeness A = Cases reported during the current week. B = Cumulative cases for the year.

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Table 2: Vaccine-Preventable Diseases & AFP

10th- 16th August 2019

03rd - 09th Aug 2019 (32nd Week)

Disease	No. of	Cases b	y Province	Ð					Number of cases during current	Number of cases during same	Total num- ber of cases to	Total number of cases to date in	Difference between the number of		
	W	С	S	Ν	E	NW	NC	U	Sab	week in 2019	week in 2018	date in 2019	2018	cases to date in 2019 & 2018	
AFP*	00	00	00	00	00	00	00	00	00	00	00	47	38	23.6 %	
Diphtheria	00	00	00	00	00	00	00	00	00	00	00	00	00	0 %	
Mumps	00	02	00	00	01	00	00	00	00	03	11	222	224	- 0.8 %	
Measles	00	03	00	00	01	01	01	00	02	08	00	223	81	175.3 %	
Rubella	00	00	00	00	00	00	00	00	00	00	00	00	04	0 %	
CRS**	00	00	00	00	00	00	00	00	00	00	00	00	00	0 %	
Tetanus	00	00	00	00	00	00	00	00	00	00	00	13	15	- 13.3 %	
Neonatal Tetanus	00	00	00	00	00	00	00	00	00	00	00	00	00	0 %	
Japanese En- cephalitis	00	00	00	00	00	00	00	00	00	00	01	09	19	- 52.6 %	
Whooping Cough	00	00	00	00	00	00	00	00	00	00	01	36	35	2.8 %	
Tuberculosis	45	04	18	07	17	20	11	13	03	138	144	5255	5175	1.5 %	

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

NA = Not Available

Dengue Prevention and Control Health Messages Look for plants such as bamboo, bohemia, rampe and banana in your surroundings and maintain them free of water collection.

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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

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