ISSN: 0975-833X

Available online at http://www.journalcra.com

International Journal of Current Research
Vol. 9, Issue, 03, pp.48535-48537, March, 2017

INTERNATIONAL JOURNAL OF CURRENT RESEARCH

CASE STUDY

HUMAN PAPILLOMA VIRUS AND RELATED DISEASES IN HUMAN BEINGS: A COMPREHENSIVE REVIEW

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ARTICLE INFO

Article History:
Received 19th December, 2016
Received in revised form
02nd January, 2017
Accepted 04th February, 2017
Published online 31st March, 2017

Key words:
Oncogenic, Virulence,
Papilloma virus, Warts.

ABSTRACT

Human Papillomavirus (HPV) is one of the most common causes of sexually transmitted disease in both men and women worldwide. HPV is associated with a variety of clinical conditions that range from innocuous lesions to cancer. Genital HPV types are divided into high and low-risk types, according to the oncogenic potential. Strong evidence for a causal etiology with HPV has been stated by the International Agency for Research on Cancer for cancers of the cervix uteri, penis, vulva, vagina, anus and oropharynx (including base of the tongue and tonsils). The present review is a detailed discussion about the human papilloma virus and its causative infections and diseases in human beings.

Citation: Dr. Asmita Jain, Dr. Anindya Bhalla and Dr. Sankalp Verma, 2017. "Human papilloma virus and related diseases in human beings: A comprehensive review", *International Journal of Current Research*, 9, (03), 48535-48537.

INTRODUCTION

Human Papillomaviruses (HPV) are small, double stranded DNA viruses that belong to family Papillomaviridae. (Bonnez and Reichman, 2000) Papillomaviruses were first identified, cloned and sequenced from cervical tumor specimens and were subsequently established as important causative agents for development of cervical cancer, the discovery which was honored by conferring Nobel Prize of Physiology and Medicine for the year 2008 to its inventor Harald zur Hausen. (Bonnez and Reichman, 2000; Cates, 1996) Subsequent research demonstrated infection of papillomaviruses in cutaneous and mucosal tissues of the oral cavity, upper gastrointestinal tract, anogenital tract and skin of hands and feet. Infection with human papillomavirus (HPV) is recognized as one of the major causes of infection-related cancer worldwide, as well as the causal factor in other diseases. (Muñoz *et al*., 2003) Of the estimated 12.7million new cancers occurring in 2008 worldwide, 4.8% were attributable to HPV infection, with substantially higher incidence and mortality rates seen in developing versus developed countries. (Muñoz *et al*., 2003; Franco *et al*., 2001)

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Human Papilloma Virus (HPV)

Viral components and physical properties

Papillomaviruses are small, non-enveloped, icosahedral DNA viruses that have a diameter of 52–55 nm. The viral particles consist of a single double-stranded DNA molecule of about 8000 base-pairs (bp) that is bound to cellular histones and contained in a protein capsid composed of 72 pentameric capsomers. The capsid contains two structural proteins — late (L)1 and L2 (70 kDa) — which are both virally encoded. Virus-like particles (VLPs) can be produced by the expression of L1, alone or in combination with L2, in mammalian or non-mammalian expression systems. (Anderson, 2002)

Pathogenesis

HPV infection occurs at the basal epithelium. Although the incidence of infection is high, most infections resolve spontaneously. A small proportion of infected persons become persistently infected; persistent infection is the most important risk factor for the development of cervical cancer. The most common clinically significant manifestation of persistent genital HPV infection is cervical intraepithelial neoplasia, or CIN. Within a few years of infection, low-grade CIN—called

CIN 1—may develop, which may spontaneously resolve and the infection clear. (Cates, 1996; Franco *et al*., 2001; Anderson, 2002) Persistent HPV infection, however, may progress directly to higher-grade CIN, called CIN2 or CIN3. High-grade abnormalities are at risk of progression to cancer and so are considered cancer precursors. Some high-grade abnormalities spontaneously regress. If left undetected and untreated, years or decades later CIN2 or 3 can progress to cervical cancer. (Jin *et al*., 1999) Infection with one type of HPV does not prevent infection with another type. Of persons infected with mucosal HPV, 5% to 30% are infected with multiple types of the virus. (Muñoz *et al*., 2003; Anderson, 2002; Jin *et al*., 1999)

1996; Anderson, 2002; Walboomers *et al*., 1999; Franco, 1995) The link between genital HPV infections and cervical cancer was first demonstrated in the early 1980s by Harold zur Hausen, a German virologist. (Bonnez and Reichman, 2000; Cates, 1996) Since then, the link between high-risk HPV types and cervical squamous cell carcinoma has become well known. In 1996, the World Health Association recognized HPV as an important cause of cervical cancer. HPV has been implicated in 99.7% of cervical squamous cell carcinoma cases worldwide (Walboomers *et al*., 1999). The magnitude of the association between HPV and cervical carcinoma is higher than that for the association between smoking and lung cancer (Franco, 1995).

Table 1. The clinical manifestations of HPV in humans

Clinical manifestations	HPV
Plantar warts	1,2,4,63
Common warts	2,1,7,4,26,27,29,41,57,65,77,3,10,28
Flat warts	3,10,26,27,28,38,41,49,75,76
Other cutaneous lesions (epidermoid cysts, laryngeal carcinoma)	6,11,16,30,33, 36, 37, 38, 41, 48, 60, 72, 73
Epidermodysplasia verruciformis	2, 3, 10, 5, 8, 9, 12, 14, 15, 17, 19, 20, 21, 22, 23,24, 25, 36, 37, 38, 47, 50.
Recurrent respiratory papillomatosis	6, 11
Focal epithelial hyperplasia de Heck	13,22
Conjunctival papillomas/carcinomas	6,11,16
Genital warts (condyloma acuminatum)	6, 11, 30, 42, 43, 45, 51, 54, 55, 70
Low-risk cervical intraepithelial neoplasia	6, 11, 16, 18, 31, 33, 42, 43, 44, 45, 51, 52, 74
High-risk cervical intraepithelial neoplasia	16, 18, 6, 11, 31, 34, 33, 35, 39, 42, 44, 45, 51,52, 56, 58, 66
Cervical carcinoma	16, 18, 31, 45, 33, 35, 39. 51, 52, 56, 58, 66, 68,70
Other genital carcinomas (vagina, vulva, penis and anus)	16, 18, 31, 45, 33, 35, 39. 51, 52, 56, 58, 66, 68,70

Transmission of HPV

HPV is transmitted by direct contact, usually sexual, with an infected person. Transmission occurs most frequently with sexual intercourse but can occur following nonpenetrative sexual activity. Studies of newly acquired HPV infection demonstrate that infection occurs soon after onset of sexual activity. In a prospective study of college women, the cumulative incidence of infection was 40% by 24 months after first sexual intercourse. HPV 16 accounted for 10.4% of infections. (Muñoz *et al*., 2003; Walboomers *et al*., 1999) Genital HPV infection also may be transmitted by nonsexual routes, but this appears to be uncommon. Nonsexual routes of genital HPV transmission include transmission from a woman to a newborn infant at the time of birth.

Temporal Pattern

There is no known seasonal variation in HPV infection.

Communicability

HPV is presumably communicable during the acute infection and during persistent infection. This issue is difficult to study because of the inability to culture the virus. Communicability can be presumed to be high because of the large number of new infections estimated to occur each year.

Clinical Features

Most HPV infections are asymptomatic and result in no clinical disease. Clinical manifestations of HPV infection include anogenital warts, recurrent respiratory papillomatosis, cervical cancer precursors (cervical intraepithelial neoplasia), and cancers, including cervical, anal, vaginal, vulvar, penile, and oropharyngeal cancer. Table 1 summarises the clinical manifestations of HPV. (Bonnez and Reichman, 2000; Cates,

Adenocarcinomas of the cervix are also related to HPV (especially HPV-18), but the correlation is less pronounced and is age dependent. In women younger than 40 years, HPV was present in 89% of adenocarcinomas, whereas in women aged 60 years and older, HPV was observed in only 43%. (Franco, 1995; Andersson *et al*., 2001)

Management of HPV related infections and diseases

Prevention

HPV transmission can be reduced but not eliminated with the use of physical barriers such as condoms. Recent studies demonstrated a significant reduction in HPV infection among young women after initiation of sexual activity when their partners used condoms consistently and correctly.

Human Papilloma Vaccine

The HPV vaccines are non-infectious subunit vaccines. The antigen for the vaccines is the L1 major capsid protein of HPV, produced by using recombinant DNA technology. HPV vaccines are highly immunogenic. More than 99% of recipients develop an antibody response to HPV types included in the respective vaccines 1 month after completing the three-dose series. (Andersson *et al*., 2001) HPV vaccines are each administered in a 3-dose series. The second dose should be administered 1 to 2 months after the first dose and the third dose 6 months after the first dose. Vaccination also is recommended for females aged 13 through 26 years and for males aged 13 through 21 years, who have not been previously vaccinated or who have not completed the 3-dose series. If females or males reach age 27 years before the vaccination series is complete, the second and/or third doses of vaccine can be administered after age 26 to complete the vaccination series. (Elfgren *et al*., 2000; Chua and Hjerpe, 1996)

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