# PREVALENCE OF VULVOVAGINITIS IN WOMEN WITH HUMAN PAPILLOMA VIRUS INFECTION

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|---|---|
| ***BV*** | *Bacterial Vaginosis* |
| ***CIN*** | *Cervical Intraepithelial Neoplasia* |
| ***HPV ٤*** | *Quadrivalent HPV vaccine* |
| ***HPV*** | *Human Papilloma Virus* |
| ***PAP*** | *Papanicolaou* |
| ***PCR*** | *Polymerase Chain Reaction* |
| ***SIL*** | *Sequamous Intraepithelial Lision* |
| ***STD*** | *Sexually Transmitted Diseases* |
| ***STI*** | *Sexually Transmitted Infections* |
| ***TV*** | *Tricomonas Vaginalis* |
| ***VVC*** | *Vulvovaginal Canidiasis* |


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# INTRODUCTION

Infectious processes in the vagina caused by potentially pathogenic microorganisms are very common and are the result of growing profusion of infectious or saprophytic agents ***(Edwards, ٢٠٠٤)***.

Evaluation of the vaginal secretions under the microscope may assist in arriving at the correct diagnosis, but it is better to obtain confirmation by culturing, since non-infectious inflammatory processes may present similar symptomatology ***(Edwards, ٢٠٠٤; Di Bartolomeo et al., ٢٠٠٢).***

Bacterial vaginosis is the most prevalent cause of abnormal vaginal discharge, affecting women of reproductive age ***(Morris et al., ٢٠٠١).***

Some risk factors have been associated with bacterial vaginosis, including cigarette smoking, use of intrauterine devices, frequent vaginal douches, multiple sexual partners, early age at first intercourse ***(Cherpes et al., ٢٠٠٨; Fethers et al., ٢٠٠٨)***.

Lesions induced by the human papillomavirus are usually associated with vaginal infections. Among the various microbiological studies regarding genital infections, few in the literature have related the presence of koilocytosis to infections by other microorganisms ***(Alves et al., ٢٠٠٤)***.

# AIM OF THE WORK

To assess the prevalence of vulvovaginitis caused by Trichomonas vaginalis (TV), Candida species , and bacterial vaginosis (BV) in women with and without human papilloma virus (HPV) infection.

# HUMAN PAPILLOMAVIRUS (HPV)

Human papilloma virus are small nonenveloped double stranded DNA virus in the family papovaviridae the circular genome is ٨ kilobases in length ***(Graziottin et al., ٢٠٠٩)***.

HPV have a common gene organization: an early region encoding non-structural genes, the late region for structural genes, and a regulatory region (long control region: LCR). The functions of each viral protein are summarized as. E١ and E٢ are cooperatively involved in the initiation of viral DNA replication. E٢ also functions as a transcriptional transactivator. E٦ and E٧ modulate the cell cycle control and contribute to viral genome maintenance. They also contribute to cancer development though E٤ and E٥ are speculated to modulate the productive phase of the HPV life cycle, their biological roles remain unclear). Both L١ and L٢ are capsid proteins ***(Wilson, ٢٠٠٧)***.

**Types of HPV**

More than ١٢٠ types of HPV have been identified ***(de Villers et al., ٢٠٠٤)*** and one-third of them target mucosal membranes, mucosa-tropic HPVs can be classified into two types based on their association with malignant

carcinomas: high risk types (١٦. ١٨. ٣١. ٣٣. ٣٩. ٤٥. ٥٢. ٥٨ and ٦٩) and no-or low risk types (٦. ١١. ٤٢. ٤٤. ٥٣ … ect) ***(Reich et al., ٢٠٠٨)***.

**Life cycle**

The target of a HPV infection is the stratified epithelium. In the normal stratified epithelium, the cell attached to the basal membrane (basal cell) is the only cell that has the potential to proliferate. The basal cell divides into a new basal cell and a daughter cell that is detached from the basal membrane, and the daughter cell launches its differentiation process .The daughter cells exit from the cell cycle and change their gene expression pattern, proceeding to terminal differentiation, then peel off from the epithelium ***(Kines et al., ٢٠٠٩)***.

The life cycle of HPV is tightly regulated by the differentiation program of the host cell .The duration of incubation for HPV disease is poorly known, for cutaneous warts, experimental inoculation leads to the development of lesions within ٣ to ٤ months on average, but it can occur as early as ٦ weeks or as late as ٢ years (***Schiffman et al., ٢٠٠٣).***

**Immunity**

HPV suppresses its replication to a “maintenance level” or “latent infection mode” in the basal cells, and maintains the DNA synthesis potential of the infected cells detached from the basal membrane to maintain viral genome replication. In terminally differentiated cells, a tremendous level of genome amplification and late gene expression takes place. After completion of virion assembly, the virions are released externally with the cornified cells. One of the reasons for this unique life cycle of HPV is escape from the immune- surveillance system ***(Bodily and Laimins, ٢٠١١)***.

## Epidemiology and prevelance of HPV

The estimated prevalence of anogenital tract HPV infections in the United States is ٢٠ million, with an annual incidence of ٥,٥ million. It has been estimated that ٧٥ to ٨٠٪ of sexually active adults will acquire a genital tract HPV infection before the age of ٥٠ ***(Workowski et al., ٢٠٠٦)***.

## Mode of transmition

Several groups of studies have clearly shown that HPV is predominantly and largely transmitted through sexual intercourse ***(Kjaer et al., ٢٠٠١)***.

HPVs can also be transmitted in other ways especially from mother to child ***(Burchel et al., ٢٠٠٦).***

The evidence for the non-sexual transmission of HPVs has been reviewed by several authors concluding that: ١) Genital HPV infections, including genital warts, may occur in sexually native populations such as virgins, infants, and children; ٢) There is some evidence of horizontal transmission of low-risk HPVs; ٣) Vertical and perinatal transmission of HPVs from mother to child, does exist, although rates are small and vary widely; ٤) High-risk genital HPVs have been detected in non-genital mucosa, such as that in the mouth, oropharynx and conjunctiva, and they have been associated with a fraction of cancers of the oral cavity and oropharynx ***(Castellsague et al., ٢٠٠٧).***

There are the possible non-`sexual horizontal transmission of HPVs, particularly of the low-risk HPV types. Such as contaminated fomites (clothing, sheets, towels, objects, and instruments) but its impact in passing and inducing active infections is most likely small if any ***(IARC, ٢٠٠٥).***

**Risk factor**

The main factors associated with HPV infection are sex, age, race, sociodemographic characteristics, prior sexually transmitted infections(STI ), parity, contraceptive methods and smoking ***(IARC, ٢٠٠٧).***

The key determinants among women are the number of sexual partners, the age at which sexual intercourse was initiated ***(Castellsague et al., ٢٠٠٦)***.

**Clinical features**

Studies have shown that subclinical infections are the most common manifestation of HPV infection. The vast majority of HPV infections regress spontaneously within ٢ years in immunocompetent host ***(Parkin and Bray, ٢٠٠٦).***

**Cervical HPV infection**

They are recognized as papillary epithelial proliferations, the lesion may be single or multiple, scattered or confluent, usually detected on the cervical transformation zone ***(Reich et al., ٢٠٠٨).***

**Anogenital manifestation**

The most common clinically apparent manifestation of genital HPV infection is genital warts **(condyloma acuminata)** ***(Insinga et al., ٢٠٠٣).***