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Human Papilloma Virus-Related Cervical Dysplasia

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Introduction

Among sexually active men and women, the human papilloma virus (HPV) is one of the most common sexually transmitted infections. With more than 200 different genotypes of HPV known, there are more than 500,000 cases each year in the United States alone (Lowy, 2016). The virus is associated with nearly all cervical cancers, anal cancers, vaginal cancers, penile cancers, and head and neck cancers (Jorge & Wright, 2016). In women, the virus is capable of causing slow cellular changes on the cervix, and this dysplasia is graded based on severity (Jorge & Wright, 2016).

The statistics related to the prevalence and transmission rate of this virus are staggering, and primary healthcare providers must have a solid knowledge base of the pathophysiology behind the disease process to better serve their patient population. This project will examine the pathophysiology associated with the different stages of cervical dysplasia, and to review the nursing implications in managing this disease population.

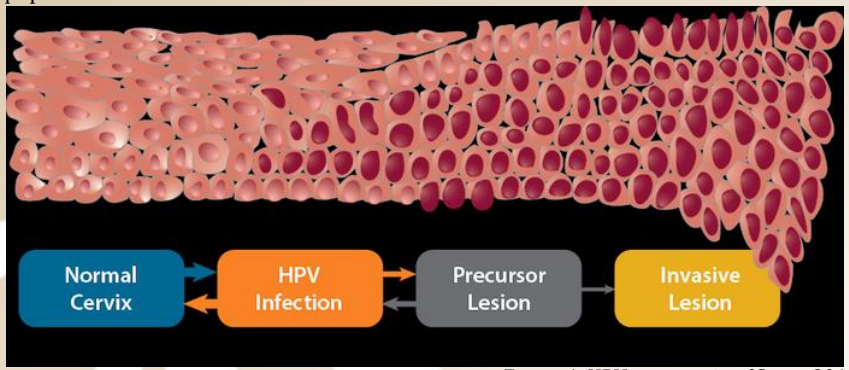

Figure 1. HPV progression (Crow, 2012)

Transmission, Signs & Symptoms

- The virus is spread by direct skin-to-skin contact of the genitals and other skin surfaces. Since the virus requires a tissue to tissue interaction, HPV cannot be spread by touching inanimate surfaces, such as a toilet seat (Smith & Travis, 2011).
- Engaging in high-risk sexual behavior increases the risk of HPV transmission. High-risk behaviors include inconsistent condom use, multiple sex partners, having a new partner, or having sex while under the influence of drugs/alcohol (Goyle, Mattocks, & Sadler, 2012).
- Roughly 6 million new cases of HPV are reported yearly worldwide in both men and women, with approximately 20 million men and women already infected in the United States (Smith & Travis, 2011).
- Human papillomavirus has a high infectivity rate with up to a 60% chance of transmission per sexual contact (Jorge & Wright, 2016).
- The patient will not usually experience symptoms with cervical changes from the high-risk HPV strains, which is why routine Pap smears are important for discovering and managing these precancerous lesions (Smith & Travis, 2011).

Underlying Pathophysiology

- There are more than 100 types of HPV (Smith & Travis, 2011).
- HPV infects the basal layer of the epithelium, and in these lesions, the viral HPV DNA is integrated into the human genome. These oncogenes can manipulate healthy cells by inducing chromosomal abnormalities and blocking apoptosis (Jorge & Wright, 2016).
- Most HPV infections are transient, but cytology changes may persist. The patient will generally not experience symptoms of the infection (Smith & Travis, 2011).
- High-risk HPV proteins E6 and E7 are responsible for protein degradation, causing chromosomal instability and cell proliferation (Orlando et al., 2013).
- E6 and E7 act as mutagens and promote cell division and delayed cell differentiation, leading to hyperproliferative lesions (Orlando et al., 2013).
- Early HPV proteins can increase the number of epidermal growth factor receptors, allowing HPV-infected cells to ignore antigrowth signals (Orlando et al., 2013).

Significance of Pathophysiology

- HPV types 16 & 18 cause 70% of cervical cancers (Smith & Travis, 2011).
- Persistent infections have the highest risk for developing high-grade precancerous lesions or cervical cancer, and co-infection with more than 1 HPV serotype increases this risk(Smith & Travis, 2011).
- HPV infection cofactor classification (Charlton et al., 2014):
  - **Environmental/exogenous factors** (smoking, diet, oral contraceptives)
  - **Host factors** (endogenous hormones, genetics, immune response)
  - **Viral factors** (HPV type, viral load, viral integration)
- HPV infection is slow progressing. 15% of high=grade lesions progress to invasive cervical cancer in 5-10 years (Jorge & Wright, 2016).

Nursing Considerations

- Vaccination with the HPV Quadrivalent Recombinant vaccine is standard of care for all females aged 9-26, but is contraindicated in pregnancy (Smith & Travis, 2011).
- The counseling approach of younger patients and their parents may be different than that of a sexually active, more mature population (Sussman et al., 2015).
- HPV types 16 & 18 are responsible for most cervical lesions & cancers (Moore et al., 2011).
- HPV DNA testing with cervical cytology is recommended (Nelson et al., 2104).
- Treatment of persistent HPV: colposcopies, cold knife conization, and loop electrosurgical excision procedures (LEEP) (Charlton et al., 2014).
- Excisional procedures cause cervical collagen remodeling, and this may lead to an increased risk of preterm birth (Miller et al., 2015).
- Smoking, number of sexual partners, and use of oral contraceptives are risk factors (Charlton et al., 2014).

Conclusion

Human papilloma virus is a common virus spread among sexually active men and women. The implications for women are HPV infections that may cause dysplastic changes of the cervix over time. Nursing care should be geared toward prevention, with vaccination and Pap smears among the two best practice modalities. Close monitoring with use of the most current algorithms should guide the management of HPV lesion surveillance.

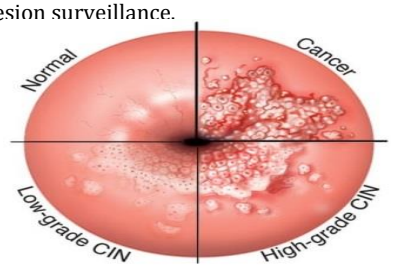

Figure 2. Correlation of pap test with cross-section of cervix (mckinley.illinois.edu, 2016).

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