Anal Cancer Prevention in a High-risk Population

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ABSTRACT

Anal cancer is an uncommon disease that is occurring more frequently each year in the United States, disproportionately affecting patients with human immunodeficiency virus and men who have sex with men. The natural history of anal cancer is thought to mimic that of cervical cancer, with human papillomavirus infection progressing to anal dysplasia and invasion of cancer in susceptible individuals. Screening for anal cancer is an easy, cost-effective intervention in high-risk individuals, but, with few clinicians specializing in the diagnosis and treatment of anal dysplasia, some planning is necessary. Patients with abnormal cytology or physical examination should be referred for specialist care.

Keywords: anal cancer, anal dysplasia, cancer screening, human papillomavirus

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INTRODUCTION

Human papillomavirus (HPV) is the most common sexually transmitted infection, causing the majority of anal cancers.[1] Anal cancer shares a similar etiology and histology with cervical cancer and is thought to mimic its natural progression. Although the incidence of anal cancer continues to rise in the United States,[2] there are no national guidelines regarding screening or treatment of cancerous or precancerous lesions in the anus.

Nurse practitioners (NPs) are well aware of the potential for cervical cancer in persistent HPV infection; however, despite their similarities, NPs tend to have less awareness and understanding of HPV-related cancers of the anus.[3] In this article we provide a basic understanding of HPV-related diseases of the anus and their prevention and treatment, with a focus on gay men and the role of the primary-care clinician in their management.

REVIEW OF PERTINENT ANATOMY

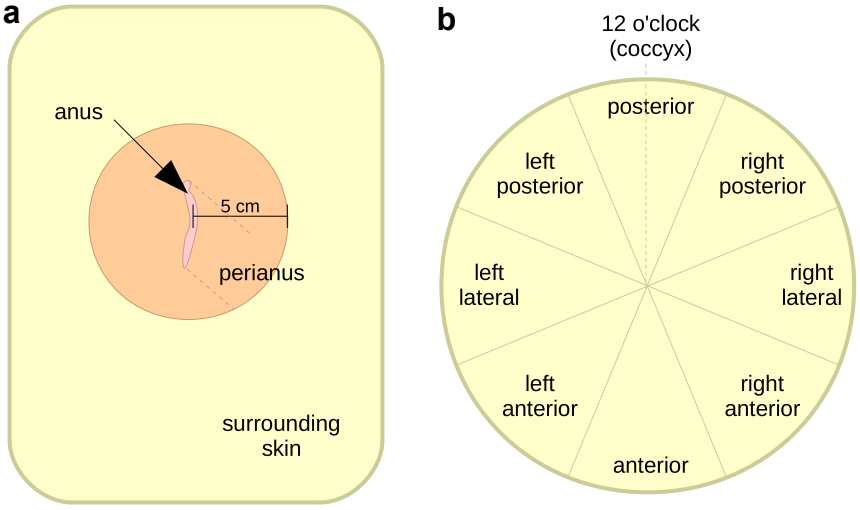
The anus is the most distal portion of the gastrointestinal tract, consisting of the last 3 to 5 cm of the large intestine. It is surrounded by an internal and external sphincter that forms the anal opening (or anal verge). The distal anal mucosa is made up of squamous epithelial cells and the more proximal rectum is made of columnar epithelium. The junction of the two is called the anal transition zone. There is a similar transition zone between the cervix and vagina, and these analogous sites are at highest risk for development of squamous cell carcinoma (SCC) due to HPV infection. HPV does not lead to cancers in the columnar epithelium of the colon and rectum, so these cancers are not discussed here.

To improve communication with other providers, a few conventions are recommended. Clinicians should describe the location of lesions according to both position on a clock face and with the octet system (eg, right anterolateral, left lateral, posterior). Unlike the cervix, the coccyx is labeled as "12 o'clock" when describing the anus. Anal lesions may start either inside or outside the verge and are at least partially visible by gently spreading the buttocks apart, whereas perianal lesions lie totally outside and up to 5 cm beyond the anal verge (Figure 1).[4]

VIROLOGY

HPV is a nonenveloped, double-stranded DNA virus with more than 100 known genotypes, grouped by similarity of their genotypes and carcinogenicity. Viruses from each group tend to infect either cutaneous surfaces or mucous membranes. There are about 13 so-called high-risk HPV types (HR-HPV)

Figure 1. Descriptors for anatomic sites of the anus and perianus. (a) Location of the anus, perianus, and surrounding skin for describing lesions. (b) Location descriptors for anal and perianal lesions, including the clock and octant system. The coccyx is labeled as posterior or 12 o'clock.



associated with the development of cervical and anogenital cancers and another 27 that produce anogenital warts without cancerous potential.

EPIDEMIOLOGY

HPV is spread by direct contact with HPV virions, typically during sexual intercourse. However, receptive anal sex is not required for infection of the anus. Friction creates microscopic tears in the epithelial surface, which allow HPV virions to reach the basement membrane of the skin or mucous membranes. Here, they bind to receptors on the cell surface, enter the nucleus, and, in the case of HR-HPV, integrate within host DNA. HPV replicates and sheds as epithelial cells mature and reach the surface. Contact with another susceptible cellular surface continues the cycle.

Unlike low-risk HPV infections, HR-HPV types express two viral proteins (E6 and E7) that inhibit natural cell death (apoptosis), allowing for unrestricted growth and ongoing HPV replication. Chronic infection and inflammation in the absence of apoptosis allows for persistent DNA damage. Without a healthy response to cellular DNA damage, persistent HR-HPV infections and the lack of cellular control portend the development of SCC.

Human papillomavirus is spread efficiently by sexual contact. In fact, anal HPV infection is found in one third of young human immunodeficiency virus (HIV)–negative men who have sex with men (MSM) with a history of only one receptive anal sex partner[5] and in up to 93% of all MSM.[6]

Abnormal anal cells are found in about 57% of HIV-positive MSM, with low-grade cytology in 28% and high-grade cytology in 7%.[6] (The remainder are abnormal cells of uncertain significance.) Regardless of the grade of abnormal cytology, high-grade disease is common when histologic examination of directed biopsies is performed. Low-grade lesions were found in 29% of HIV-positive MSM and 24% had high-grade lesions, with 8% and 15%, respectively, in HIV-negative cohorts.[6] These high-grade lesions are thought to be the precursor to SCC of the anus.

Anal cancer itself is an uncommon disease with only 7,210 new cases expected worldwide in 2014.[7] Women in the general population are more likely to develop anal cancer than men,[7] with HIV-positive women being up to 6.8 times more likely to have an anal cancer diagnosis than HIV-negative women.[8] Incidence in HIV-positive MSM, however, is about ninefold higher than in their HIV-negative counterparts.[6] The incidence of anal cancer in HIV-positive MSM is even higher than the rates of cervical cancer seen in women prior to the onset of widespread cervical screening.[8]

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