

Human Papillomavirus Disease

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Recommendations for Cervical Cancer Screening for People With HIV

Figure 1. Screening Algorithm for Cervical Cancer in People With HIV Aged 21 to 29 Years

Figure 2. Screening Algorithm for Cervical Cancer in People With HIV Aged 30 Years and Older

Recommendations for Anal Cancer Screening for People With HIV

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Epidemiology

At least 12 human papillomavirus (HPV) types are considered oncogenic, including HPV16, 18, 31, 33, 35, 39, 45, 51, 52, 56, 58, and 59.¹⁻³ HPV68 is considered “probably oncogenic,” and several other HPV types are considered “possibly oncogenic.” HPV16 alone accounts for approximately 53% to 73% of cervical cancers in the general population and HPV18 for another 12% to 21%.⁴ The other oncogenic HPV types each account for under 5% of cervical cancers.⁴ Anal cancer and a subset of tumors of the vulva, vagina, penis, and oropharyngeal carcinoma (OPC) are also associated with HPV, and HPV16 and 18 are the most commonly detected in noncervical HPV-positive tumors.^{2,5-14}

While anal cancer and OPC occur in both women and men with HIV, these two tumors disproportionately affect males with HIV, as well as African Americans.¹⁵⁻¹⁸ Data also suggest that the distribution of oncogenic HPV types detected in cervical and anal cancers among people with HIV may differ from those in the general population.^{19,20}

HPV infection is the major risk factor for development of cervical cancer,^{5,21} the fourth most common cancer in women worldwide.²² Nearly all cervical cancers contain oncogenic HPV DNA sequences.²³⁻²⁵ While HPV is a common sexually transmitted cervical infection, most of these infections resolve spontaneously.²⁶⁻³⁰ Cervical tumorigenesis occurs mostly, if not exclusively, in the presence of persistent oncogenic HPV infection.^{1,5,31} Women with HIV have high incidence and persistence of HPV relative to women without HIV, as well as high rates of cervical intraepithelial neoplasia (CIN), cervical precancer (CIN 3), and invasive cancer.³²⁻⁴⁰ Rates of cervical cancer in women with HIV were elevated significantly compared with the general population—3 to 4 times overall (95% confidence interval [CI], 3.13–3.70).⁴¹ Most of these relative risks increase with decreasing CD4 T lymphocyte (CD4) cell counts, and cervical cancer is itself associated with advanced HIV.⁴²⁻⁵⁴ The percentage with adenocarcinoma histology compared with squamous cell carcinoma is lower in women with HIV than in the general population. Several studies found decreased incident detection, persistence, and progression of HPV and CIN with effective

antiretroviral therapy (ART) use,^{55,56} including one study that distinguished between adherent versus nonadherent or effective versus ineffective ART use (based on HIV RNA level).

In a report from the HIV/AIDS Cancer Match Study (2002–2016)—which included a population of 164,084 women with HIV—552 cases of invasive cervical cancer (ICC) occurred in 1.16 million person-years of follow-up (rate = 47.7 per 100,000). By age group, the highest incidence rates occurred in the 40- to 44- and 35- to 39-year-old age groups (rate = 66.1 and 64.5 per 100,000, respectively). No cases of ICC were identified in the under 25-year-old age group during 69,900 person-years of follow-up (standardized incidence ratio [SIR] = 0; 95% CI, 0,7.1).⁴¹

People with HIV have an increased incidence of anogenital tumors (vulva, vagina, penis) and OPC relative to the general population.^{23,57-60} Low CD4 counts in people with HIV have been associated with increased risk of anal cancer,⁶¹⁻⁶³ as well as high-grade anal intraepithelial neoplasia (AIN; the likely anal cancer precursor lesion),⁶⁴⁻⁶⁶ anal and genital warts, and vulvar intraepithelial neoplasia (VIN) and vaginal intraepithelial neoplasia (VAIN).⁶⁷⁻⁶⁹ Registry-based data indicate a downward trend in anal cancer incidence relative to the general population (i.e., a reduction in SIR from approximately SIR ~40 in 1996 to SIR ~20 in 2012; $P = 0.0001$),⁵⁹ as well as a possible ($P = 0.09$) decrease in cervical cancer from SIR ~5 in 1996 to SIR ~3 in 2012, and a nonsignificant decrease in OPC.^{70,71} Other HPV-related tumors are less common, and less is known about trends in their incidence.

The elevated risk of HPV-associated cancers in people with HIV continues into older age (>50 years of age).³⁹ Registry-based data show that the 5-year risk (cumulative incidence) of anal cancer was 0.65% and 0.33% in men aged 45 to 59 years with HIV who have sex with men with and without AIDS, respectively, whereas the results were 0.10% and 0.04% for men with HIV who do not have sex with men, and 0.20% and 0.08% for women with HIV.⁷⁰ Similar results were obtained in a recent meta-analysis of available studies.⁷² The ANCHOR study estimated the cumulative 4-year progression from high-grade squamous intraepithelial lesion (HSIL) to anal cancer was 1.8%.⁷³

Anogenital warts have very low carcinogenic potential but are an important HPV-associated disease in people with HIV. These lesions are common, and more likely to be persistent in people with HIV than in the general population. Approximately 80% to 90% of anogenital warts are caused by non-oncogenic HPV types 6 or 11.⁷⁴ HPV types 6 and 11 also have been associated with conjunctival, nasal, oral, and laryngeal warts. In the United States, prior to the introduction of HPV vaccination, the incidence of anogenital warts was 60.2 per 10,000 women (aged 20–24 years) and 53.8 per 10,000 men (aged 20–24 years),⁷⁵⁻⁷⁷ but with several-fold greater rates in people with HIV.⁶⁷ Low-grade vulvar lesions and genital warts were both found to decrease with ART.⁶⁷

Clinical Manifestations

The principal clinical manifestations of mucosal HPV infection are genital, anal, and oral warts; CIN; VIN; VAIN; AIN; anogenital squamous cell cancers; and cervical adenocarcinomas. A subset of oropharyngeal cancers is also caused by HPV.⁷⁸

Oral, genital (condyloma acuminata), and anal warts are usually flat, papular, or pedunculated growths on the mucosa or epithelium. The lesions may measure a few millimeters to 1 to 2 centimeters in diameter. Most warts are asymptomatic, but warts can be associated with itching or discomfort. In cases associated with more severe immunosuppression, marked enlargement may cause dyspareunia or dyschezia. Lesions of any size may cause cosmetic concerns.

Low-grade squamous intraepithelial lesions (LSIL) and HSIL in the cervix, vagina, vulva, and anal canal are often asymptomatic but may manifest with bleeding or itching. Related cancers also may be asymptomatic or may manifest with bleeding, pain, odor, or a visible/palpable mass. External lesions may be visible or palpable. Similarly, squamous cell cancers at these sites also can be asymptomatic or may manifest with bleeding, pain, or a visible/palpable mass.⁷⁹

Preventing HPV Infection

Recommendations for Preventing HPV Infection
<ul style="list-style-type: none"> • HPV vaccine is recommended for routine vaccination at age 11 or 12 years. <ul style="list-style-type: none"> ○ Administer three doses of 9-valent HPV vaccine (Gardasil 9) at 0, 1 to 2, and 6 months (AIII). Ideally, the series should have been initiated at age 11 or 12 years but may be started as early as age 9 years. The two-dose series is not recommended in people with HIV. • For all people with HIV aged 13 to 26 years who were not vaccinated previously: <ul style="list-style-type: none"> ○ Administer three doses of 9-valent HPV vaccine (Gardasil 9) at 0, 1 to 2, and 6 months (AIII). The two-dose series is not recommended in people with HIV. • For people with HIV aged 27 to 45 years who were not adequately vaccinated previously: <ul style="list-style-type: none"> ○ HPV vaccine is not routinely recommended; instead, shared clinical decision-making regarding HPV vaccination is recommended for people who may be at risk for a new HPV infection (AIII). • For people who were adequately vaccinated with bivalent or quadrivalent HPV vaccine: <ul style="list-style-type: none"> ○ Some experts would consider additional vaccination with 9-valent HPV vaccine, but data are lacking to define the efficacy and cost-effectiveness of this approach (CIII). • HPV vaccination is not recommended during pregnancy (CIII).

HPV Vaccine

HPV vaccination prevents HPV infection and is ideally administered before sexual exposure to HPV. Although HPV vaccine is most effective in people with few or no sex partners prior to vaccination, HPV vaccination in people with multiple lifetime sex partners can still prevent HPV infection from subtypes they have not been exposed to yet. Three U.S. Food and Drug Administration (FDA)–approved HPV vaccines are licensed: bivalent, quadrivalent, and 9-valent. Currently, only the 9-valent vaccine (9vHPV, protective against HPV types 6, 11, 16, 18, 31, 33, 45, 52, and 58) is available in the United States.^{14,80} This vaccine has an FDA indication for prevention of cervical, vaginal, vulvar, and anal cancer; genital warts, and oropharyngeal and other head and neck cancers⁸¹ based on randomized clinical trial (RCT) data; however, these studies were not conducted in people with HIV.^{82–88} These RCTs evaluated several endpoints accepted by FDA and established the safety of the vaccine in children as young as 9 years of age and young people aged 16 to 26, as well as older women (aged 27–45 years).^{89,90} Although no clinical trials have been conducted to demonstrate HPV vaccine efficacy in prevention of oropharyngeal cancers, some evidence exists that the prevalence of oral HPV infections from types contained in the vaccines are reduced with vaccination.^{91,92} Protection against more subtypes might be more useful in people with HIV because there is more diversity of oncogenic subtypes of HPV.^{14,80}

Routine HPV vaccination with the 9-valent vaccine should be initiated at age 11 or 12 years but may be started as early as age 9 years.^{93,94} Although the Centers for Disease Control and

Prevention (CDC) Advisory Committee on Immunization Practices recommends a two-dose series,⁹⁵ the Panel recommends that people with HIV receive a three-dose series (0, 1–2, and 6 months) because their immune response to vaccination might be attenuated **(AIII)**. Because HPV vaccination is safe and immunogenic and has the potential benefit of preventing HPV-associated disease and cancer, catch-up HPV vaccination is recommended for people with HIV aged 13 to 26 years **(AIII)**. Although routine vaccination beyond age 26 is not recommended, shared clinical decision-making regarding HPV vaccination is recommended for adults aged 27 to 45 years who are not adequately vaccinated and are at risk for a new HPV infection **(AIII)**.⁹⁴ Considerations to help guide [shared clinical decision-making are available on the CDC website](#).

For patients who have completed a vaccination series with the recombinant bivalent or quadrivalent vaccine, some experts would give an additional full series (three doses) of vaccination with the 9-valent vaccine **(CIII)**, however no data exist to define who might benefit or how cost-effective this approach might be.

Several studies have established the safety and immunogenicity of HPV vaccines in a broad range of people with HIV.^{95–97} Some studies have demonstrated lower antibody levels in people with HIV than in those who do not have HIV; however, the clinical significance of this observation is unknown.^{98–100} Studies have shown that HPV vaccination induces an anamnestic response in children and adults with HIV.^{83,96,101} Immune responses appear stronger among those with higher CD4 counts and suppressed HIV viral loads.^{97,102}

Although HPV vaccine clinical trials in people with HIV reported appropriate immunogenicity and safety,^{95–104} few, if any, RCTs have utilized clinical endpoints, such as CIN 3 or incident persistent infection with vaccine HPV types. There is also a paucity of prospective epidemiologic studies using these endpoints.¹⁰⁵ One randomized, double-blind clinical trial evaluated the efficacy of the quadrivalent HPV vaccine (4vHPV) in a population of people with HIV who were older than 27 years with high rates of prior and current HPV infection. The trial did not show efficacy for prevention of new anal HPV infections or improvement in anal HSIL outcomes.¹⁰⁶ Anal cancer endpoints, including anal HSIL and anogenital wart incidence, were studied in another RCT of 4vHPV that involved 129 men who have sex with men (MSM) and who were on ART with a mean age of 38.8 years and who had history of AIDS.¹⁰⁷ Although the vaccine and placebo arms did not differ by HSIL or genital wart incidence, vaccine HPV types were less common in the vaccine arm, and in secondary analyses the investigators found that those with the longest time since immunization had significantly reduced risk of HSIL. A one-arm study of 260 MSM with HIV, aged 18 to 26 years, who received 4vHPV and were followed with high-resolution anoscopy at 7, 12, and 24 months found that no participants who were naive at baseline for one or more 4vHPV types developed LSIL or HSIL related to those HPV.¹⁰⁸ Conversely, a Phase 3 4vHPV RCT involving older males and females with HIV (aged ≥27 years) ended early due to an insufficient vaccine effect to meet stopping rules.¹⁰⁶ This trial did, however, suggest efficacy for short-term prevention of oral HPV infection, which decreased significantly from 88% to 32% after 6 months. A prospective observational cohort study of female youth who received 4vHPV showed unexpectedly high rates of abnormal cervical cytology, occurring in 33 of 56 youth who acquired HIV perinatally and only 1 of 7 of youth who were exposed but uninfected and yielding incidence rates of 100 person-years of 15 (10.9–29.6) and 2.9 (0.4–22.3), respectively. The majority of the diagnoses were LSIL or less, and the genotypes associated with these abnormal cytology results were unknown.¹⁰³

People with HIV who have been vaccinated should continue routine cervical cancer screening because the vaccine does not prevent all HPV types that may be precursors to cervical cancer, and

because the vaccine may be less effective in people with HIV (especially those with low CD4 counts) than in people without HIV.

Condom Use

The use of male latex condoms is strongly recommended for preventing transmission or acquisition of HPV infection, as well as for preventing HIV and other sexually transmitted infections (STIs) **(AII)**.¹⁰⁹⁻¹¹¹ Latex condoms provide a sufficient barrier to prevent passage of particles the size of HPV. Consistent and proper use of latex male condoms has been associated with 70% lower incidence of oncogenic HPV infection among women.¹¹¹ Similarly, cross-sectional data suggested that among heterosexual men with no steady sex partner, consistent condom use was associated with 50% lower odds of HPV infection of the penis.¹¹² A meta-analysis found that condom use was associated with reduced risk of genital warts and, in women, with lower rates of CIN.¹⁰⁹ An RCT of condom use in heterosexual couples found significantly more frequent clearance of CIN and HPV among women randomized to condom use and of penile lesions among their male partners.^{113,114} Male condoms have benefits in reducing risk of transmission of nearly all STIs (including HIV infection) during heterosexual intercourse and same-sex intercourse between men. In circumstances when a male condom cannot be used properly, a female condom (e.g., an FC1 or FC2 Female Condom) should be considered for heterosexual vaginal intercourse **(AII)** and for heterosexual or male same-sex anal intercourse **(BIII)**. Data on FC1 and FC2 Female Condoms suggest that the devices are protective against STIs.¹¹⁰

Male Circumcision

There is evidence that male circumcision reduces rates of oncogenic HPV infection of the penis, based on data from RCTs and observational studies.¹¹⁵⁻¹¹⁸ Observational studies in the general population also suggest that circumcision is associated with lower risk of penile cancer and of cervical cancer in sexual partners. Relevant data in men with HIV, however, are limited; findings to date suggest that the effects of circumcision against HPV infection (while protective) may be less in people with HIV than in those without. Furthermore, no clinical trials have assessed whether circumcision of men who have HIV reduces the risk of genital or anal HPV-related cancer or precancer (such as AIN) or oncogenic HPV infection of the anal or oral mucosa for them or their sexual partners. Evidence is insufficient to recommend adult male circumcision solely to reduce the risk of oncogenic HPV infection in men with HIV or their sex partners.

Preventing Disease

Cervical Cancer Screening Recommendations

Figure 1. Screening Algorithm for Cervical Cancer in People With HIV Aged 21 to 29 Years

Figure 2. Screening Algorithm for Cervical Cancer in People With HIV Aged 30 Years and Older

The same cytology and colposcopic techniques with biopsy are used to detect CIN among people with and without HIV (see section on Preventing Disease). At the time of cytology screening, the genitalia and anal canal should be inspected carefully for visual signs of warts or invasive cancer.

Available HPV tests can detect up to 14 oncogenic HPV types in clinical specimens and are sensitive for the detection of cervical cancer precursors. Some commercially available HPV tests will specify whether the oncogenic HPV includes genotypes HPV16 or HPV16/18. The available tests for oncogenic HPV have been incorporated into the screening algorithms. HPV testing is always for oncogenic HPV types only; there is no role for non-oncogenic HPV testing.

Observational epidemiologic studies in people with HIV have been instrumental in the decisions to adopt several cervical cancer screening guidelines that had been validated in large clinical trials in the general population. This included studies that supported the incorporation of cervical HPV testing for determining referral to colposcopy versus retesting in 1 year or during routine follow-up. For example, despite the very high prevalence of HPV in women with HIV, normal cytology with negative HPV co-testing had a strong negative predictive value, with low 3- to 5-year incidence of cervical intraepithelial neoplasia grade 2 (CIN 2+) regardless of CD4 count.^{119,120} Conversely, the risk of precancer was high in women with HIV who tested positive for oncogenic HPV despite normal cervical cytology results and several-fold greater still if HPV16 was specifically detected.¹²¹ Additional studies showed that oncogenic HPV testing had high sensitivity and negative predictive value in the triage of borderline cervical cytology results (i.e., atypical squamous cells of uncertain significance [ASC-US]).^{122,123}

Possible cervical cytology results include the following:

- Normal (negative for intraepithelial lesion or malignancy)
- LSIL or CIN 1 (cervical intraepithelial neoplasia grade 1)
- HSIL or CIN 2, 3
- ASC-US
- ASC-H (atypical squamous cells, cannot rule out a high-grade lesion)
- AGC (atypical glandular cells)

For people with HIV, cervical cancer screening and treatment of precancer are, in and of themselves, a major burden. Positive HPV screening tests are several-fold more common in women with HIV than in the general population, and as many as 16% of women with HIV have abnormal cervical cytology with ASC-US or worse at each clinical visit.¹²⁴ This often leads to repeated colposcopy and biopsy, although most of these colposcopies and biopsies in people with HIV find LSIL rather than clinically relevant disease (e.g., HSIL, cancer). A study of “primary oncogenic HPV screening”—which uses HPV testing as the initial screening method and, if positive, often reflex-triage (e.g., HPV16/18-genotyping, cervical cytology)—found that this approach reduced unnecessary colposcopies by almost half relative to currently recommended HPV/cervical cytology co-testing for women with HIV.¹²⁵ However, these findings require confirmation. There is also a significant need for technical advancement to improve the positive predictive value of the screening tests—especially as many women with HIV exceed the age for routinely recommended HPV vaccination.¹²⁵

People With HIV Aged 21 to 29 Years

Cervical cytology is the primary mode for cervical cancer screening for women with HIV under 30 years of age. People aged 21 to 29 years with HIV should have cervical cytology at the time of initial diagnosis with HIV **(AII)**. See [Figure 1. Screening Algorithm for Cervical Cancer in People With HIV Aged 21 to 29 Years](#) for detailed recommendations. The absolute incidence of ICC is

exceedingly low among women with HIV under 25 years; therefore, cervical cancer screening is recommended to start at age 21. The rationale for beginning screening at age 21 is to provide a 3- to 5-year window prior to age 25, when the risk of ICC in women with HIV exceeds that of the general population.⁴¹ Co-testing (cervical cytology and HPV test) and reflex high-risk HPV (hr-HPV) testing (HPV testing in the presence of abnormal cytology results) is routinely recommended for people without HIV and might be considered for people aged 25 to 29 years with HIV; however, there is a relatively high prevalence of transient HPV before age 30 years, which may lead to unnecessary colposcopy.¹²⁶ If cytology reveals ASC-US and reflex hr-HPV testing is performed, repeat cytology should be evaluated in 6 to 12 months **(AII)**. If repeat cytology shows ASC-US and reflex hr-HPV is positive, individuals should be referred for colposcopy **(CIII)**.

The [American Society for Colposcopy and Cervical Pathology \(ASCCP\)](#) and the [American College of Obstetrics and Gynecology \(ACOG\)](#) recommend screening for cervical cancer using cytology alone for women aged 21 to 29 years. The [American Cancer Society \(ACS\)](#) now recommends initiating cervical cancer screening at age 25 with primary HPV screening (hr-HPV testing alone) every 5 years in the general population. The FDA recently approved self-testing for HPV screening in clinical settings.¹²⁷ There are ongoing studies to evaluate the use self-testing for HPV screening in people with HIV. The [U.S. Preventive Services Task Force \(USPSTF\)](#) is reviewing its current recommendations and will issue an update soon regarding the use of primary HPV screening for cervical cancer.

People With HIV Aged 30 Years and Older

Cervical cancer screening in people with HIV should continue throughout their lifetime (and not, as in the general population, end at 65 years of age) **(BIII)**. Either cytology only or cytology and HPV co-testing is acceptable for screening **(BIII)**. See [Figure 2. Screening Algorithm for Cervical Cancer in People With HIV Aged 30 Years and Older](#) for detailed recommendations. Current guidelines from both the [ACS](#) and the [USPSTF](#) allow use of HPV co-testing with cytology. A negative HPV test predicts prolonged low risk of cancer. Cytology/HPV co-testing can allow a prolonged cervical cancer screening interval in women with HIV who are older than 29 years and have normal cervical cytology with concurrent negative HPV testing.

For people aged more than 65 years, it is recommended to continue cervical cancer screening because people with HIV are at higher risk for cervical cancer **(BIII)**. However, clinicians should consider other factors, such as the life expectancy of the patient and the risk for developing cervical cancer at this age.¹²⁸

Overview of Cervical Cancer Screening Guidelines					
	<21 Years	21–24 Years	25–29 Years	≥30 Years	Comments
NIH OAR Adult and Adolescent OI Guidelines (specific to people with HIV)	No screening recommended	Cytology Only <ul style="list-style-type: none"> • Cytology yearly <ul style="list-style-type: none"> ◦ If normal cytology on 3 consecutive annual tests, adjust to every 3 years 	Cytology Only <ul style="list-style-type: none"> • Cytology yearly <ul style="list-style-type: none"> ◦ If normal cytology on 3 consecutive annual tests, adjust to every 3 years 	Co-testing^a <ul style="list-style-type: none"> • Co-testing yearly <ul style="list-style-type: none"> ◦ If normal cytology and hr-HPV negative on 3 consecutive years, adjust to every 3 years. Cytology Only <ul style="list-style-type: none"> • Cytology yearly • If normal cytology on 3 consecutive years, adjust to every 3 years 	
USPSTF (no HIV-specific guidance)	No screening recommended	<ul style="list-style-type: none"> • Cytology every 3 years 	<ul style="list-style-type: none"> • Cytology every 3 years 	Cytology Only <ul style="list-style-type: none"> • Every 3 years hr-HPV Testing Only <ul style="list-style-type: none"> • Every 5 years Co-testing^a <ul style="list-style-type: none"> • Every 5 years 	Not specific to people with HIV
ASCCP					Same as USPSTF
ACOG					Same as USPSTF
ACS (no HIV-specific guidance)	No screening recommended	No screening recommended	Preferred <ul style="list-style-type: none"> • Primary HPV test^b every 5 years Acceptable <ul style="list-style-type: none"> • Co-testing every 5 years • Cytology alone every 3 years 	Preferred <ul style="list-style-type: none"> • Primary HPV test^b every 5 years Acceptable <ul style="list-style-type: none"> • Co-testing every 5 years • Cytology alone every 3 years 	Updated July 2020 Not specific to people with HIV

Overview of Cervical Cancer Screening Guidelines					
WHO (HIV-specific guidance)	No screening recommended	No screening recommended	Preferred <ul style="list-style-type: none"> • Primary HPV test^b (provider-obtained or self-collection) every 3–5 years 	Preferred <ul style="list-style-type: none"> • Primary HPV test^b (provider-obtained or self-collection every 3–5 years) 	Updated July 2021

^a Co-testing refers to combined cytology and high-risk HPV (hr-HPV) testing.

^b Primary HPV testing is hr-HPV testing alone.

Key: ACOG = American College of Obstetricians and Gynecologists; ACS = American Cancer Society; ASCCP = American Society for Colposcopy and Cervical Pathology; HPV = human papillomavirus; hr-HPV = high-risk HPV; NIH OAR = National Institutes of Health Office of AIDS Research; OI = opportunistic infection; USPSTF = United States Preventive Services Task Force; WHO = World Health Organization

Preventing Vaginal and Vulvar Cancer

VAIN and VIN are recognized through visual inspection, including colposcopy and biopsy as needed. Most patients are asymptomatic, however. Abnormalities are usually detected after colposcopic examination and biopsy in response to abnormal cervical cytology. Following hysterectomy for benign disease, routine screening for vaginal cancer is not recommended for people with HIV (**AIII**). However, people with a history of high-grade CIN, adenocarcinoma *in situ*, or ICC are at increased risk and should be followed with annual vaginal cuff cervical cytology (**BIII**). For patients not known to have had a hysterectomy for a benign indication, continued screening is recommended since studies have shown that CIN is the most common indication for hysterectomy for people with HIV (**CIII**). Although vaginal cervical cytology results are often abnormal in women with HIV and more common than in women without HIV, VAIN 2+ and vaginal cancers are infrequent.¹²⁹ Another study in women with HIV with previous hysterectomy and no previous abnormal cervical cytology results, showed that among those with vaginal biopsies, 29% had VAIN 2 or VAIN 3.¹³⁰ However, this retrospective study was limited due to sample size. For patients with abnormal vaginal cuff cervical cytology results with no visible vaginal colposcopic abnormalities, the use of Lugol's iodine to stain the vagina is recommended (**AIII**). Vaginal colposcopy also is indicated in the presence of concomitant cervical and vulvar lesions. Classification of VAIN (i.e., VAIN 1, VAIN 2, and VAIN 3) parallels that of the cervix.

No screening procedure is available for vulvar cancer. However, biopsy or referral is indicated when inspection/palpation identifies lesions suspicious for VIN or cancer.

Screening for Anal Cancer

Figure 3. Screening Algorithm for Anal Cancer in Asymptomatic People With HIV

Figure 4. Assessment of Anal Cytology and HPV Results in People With HIV

Based on the high incidence of anal cancer in people with HIV, the high prevalence of anal HSIL in people with HIV, the high progression rate of anal HSIL to anal cancer in the absence of treatment, and efficacy in treating anal HSIL to reduce progression to anal cancer, screening for anal HSIL (**AII**)^{73,131} and treatment of anal HSIL (**AI**) are recommended for people with HIV based on age.⁷³ See [Figure 3. Screening Algorithm for Anal Cancer in Asymptomatic People With HIV](#) and [Figure 4. Assessment of Anal Cytology and HPV Results in People With HIV](#) for detailed recommendations.

People with HIV, regardless of history of anal intercourse, should undergo annual assessment of anal symptoms (e.g., unexplained itching, anal bleeding, or pain; presence of perianal lesions). MSM and transgender women below the age of 35 and others below the age of 45 with anal symptoms should undergo digital anorectal examination (DARE) and standard anoscopy (**AIII**). See [International Anal Neoplasia Society Guidelines for the Practice of Digital Anal Rectal Examination](#) and [Performing a Digital Anal Rectal Examination](#) on how to perform a proper DARE.

MSM and transgender women aged 35 and above and all others with HIV aged 45 and above with symptoms or abnormal examinations should be referred to high-resolution anoscopy (HRA) if available (**BIII**). HRA identifies anal HSIL and (following biopsy for histopathologic confirmation) enables treatment of anal HSIL to prevent progression to anal cancer. If HRA is not available, patients should undergo standard anoscopy (**BIII**) and be referred for biopsy of identified lesions to determine level of histologic changes and to rule out invasive cancer. Standard anoscopy involves

visualization of the anal canal and perianal region through an anoscope without application of 5% acetic acid or Lugol's iodine to identify lesions. HRA requires specialized training and is performed with 5% acetic acid and Lugol's iodine to identify lesions under magnification typically provided by a colposcope. HRA allows flat lesions typical of HSIL or cancer to be identified with greater precision than standard anoscopy.

When to start screening for anal HSIL in asymptomatic individuals specifically should be based on the overall risk for anal cancer. The risk for anal cancer in people with HIV appears to differ based on age, sex at birth, and HIV exposure group, as evidenced by national estimates from the AIDS/Cancer Match Study, which links HIV/AIDS registries with data from the National Cancer Institute's Surveillance Epidemiology End Results (SEER), and by findings from a comprehensive meta-analysis of anal cancer screening and treatment studies (see [figure on anal cancer incidence](#) from this meta-analysis).^{70,72}

Based on their incidence of anal cancer, and until definitive screening guidelines are available, experts in the field recommend that screening in asymptomatic people with HIV begin at different ages depending on sex and HIV risk group. Initiating screening for anal precancer and cancer is recommended at age 35 for MSM and transgender women who have HIV **(AII)**. Screening for anal cancer should be initiated in all other persons with HIV at age 45 years **(AII)**. MSM and transgender women aged 35 years and older, and other people with HIV aged 45 years and older, should continue to be assessed annually for anal symptoms and undergo DARE regardless of symptoms **(BIII)**.

Older age, longer known duration of immune suppression and HIV infection, history of AIDS, smoking, positive HPV16 or 18 status, and higher grade of cytologic abnormality are associated with increased risk of anal cancer.^{72,132-135} People with HIV who meet any of these criteria should be screened and referred for HRA as soon as feasible **(BIII)**.

Screening can be performed using anal cytology alone or with hr-HPV co-testing. Screening individuals with anal cytology to identify those who need HRA with the goal of diagnosing and treating anal HSIL should be performed only when HRA and HRA-based treatment are available. There currently are no FDA-cleared anal HPV tests, but testing is available in many clinical laboratories. It is strongly recommended to use only clinical laboratories that have undergone CLIA certification to conduct anal HPV tests. If cytology will be obtained for screening, defer DARE until after swabbing anal canal to decrease potential for lubricant interfering with cytology results. Until further data on screening algorithms are available, the recommended screening approaches shown in [Figure 3. Screening Algorithm for Anal Cancer in Asymptomatic People With HIV](#) can be considered based on testing availability.

The International Anal Neoplasia Society (IANS) recently published [recommendations for anal cancer screening](#) including but not specific for people with HIV. We concur with the recommendation to screen for anal cancer for MSM and transgender women aged more than 35 years with HIV and all other people aged 45 years or above with HIV. We agree with the use of anal cytology or anal cytology with hr-HPV co-testing as screening modalities. In contrast to the IANS guidelines, we do not recommend HPV screening without cytology at this time due to insufficient supporting evidence in people with HIV **(BIII)**. The prevalence of anal hr-HPV infection is very high among persons with HIV, and the specificity and positive predictive value for anal HSIL are expected to be low. Although screening for HPV16 or 18 specifically may improve the specificity and positive predictive value, anal cancer is associated with a broader spectrum of hr-HPV types in

people with HIV than in people without HIV; therefore, there may still be insufficient sensitivity for anal HSIL in people with HIV.¹⁹

Overview of Anal Cancer Screening Guidelines in People With HIV		
	NIH OAR Adult and Adolescent OI Guidelines	IANS Guidelines
Primary anal HPV testing alone without cytology as screening option	No	Yes
High-priority patients if HRA availability limited (no priority order specified in either guideline)	<ul style="list-style-type: none"> • Higher grade of cytologic abnormality • HPV16 on HPV testing • Smokers • >60 years of age • Longer known duration of HIV • History of AIDS 	<ul style="list-style-type: none"> • Higher grade of cytologic abnormality • HPV16 on HPV testing

Key: HPV = human papillomavirus; HRA = high-resolution anoscopy; IANS = International Anal Neoplasia Society; NIH OAR = National Institutes of Health Office of AIDS Research; OI = opportunistic infection

If HSIL is identified on biopsy, treatment of the lesion should be performed to reduce the incidence of anal cancer among people with HIV **(AI)**. Further details are presented in the section “Treating AIN and Anal Cancer.”

Preventing Oropharyngeal Cancer

Although HPV DNA detection might be useful in identifying individuals at high risk of oropharyngeal cancer, no adequate methods currently exist to determine the site of HPV-associated oropharyngeal precancer or cancer to target biopsy or treatment, despite ongoing efforts. It also should be noted that rates of non-HPV-associated oral cancer also are increased in people with HIV,¹⁵ and potentially malignant oral disorders can be diagnosed and followed by biopsy in some cases; the effectiveness of this approach has not been tested in RCTs.¹³⁶

Diagnosis

Warts/Condyloma

Diagnosis of genital and oral warts is made by visual inspection and can be confirmed by biopsy. However, biopsy is needed only if the diagnosis is uncertain, the lesions do not respond to standard therapy, or the warts are pigmented, indurated, fixed, bleeding, or ulcerated. No data support the use of HPV testing for screening, diagnosis, or management of visible genital/oral warts or oral HPV disease in people with HIV.¹³⁷

Cervical Neoplasia

The same cytology and colposcopic techniques with biopsy are used to detect CIN among patients without HIV and people with HIV (see section on Preventing Disease). At the time of cytology

screening, the genitalia and anal canal should be inspected carefully for visual signs of warts, mucosal abnormalities that may indicate intraepithelial neoplasia, or invasive cancer.

Anal and Vulvar/Vaginal Neoplasia

AIN, VAIN, and VIN are recognized through visual inspection, including high-resolution anoscopy, colposcopy, and biopsy as needed. A digital examination of the anal canal to feel for masses should be performed as part of routine evaluation.¹³⁸

Treating Disease

Cancer-specific survival following treatment of anal cancer and OPC was reported to be similar in people with HIV and the general population, whereas cervical cancer survival following treatment was reported to be lower in women with HIV.^{139,140} Another study found that although response to initial therapy for ICC (e.g., radiation treatment) was similar in women with HIV compared with others, HIV was associated with higher risk of relapse (hazard ratio [HR] 3.6; 1.86–6.98) and higher cervical cancer mortality.¹⁴¹ Data from the AIDS Malignancy Consortium showed that women with HIV on ART with locally advanced cervical cancer in sub-Saharan Africa can complete routine cisplatin and radiation therapy. Furthermore, 1-year progression-free overall survival rates observed among women with high-risk advanced tumors were similar to reported studies of women without HIV with generally smaller tumors.¹⁴²

Treating Genital and Oral Warts

Patient-Applied Treatments Options

For Uncomplicated External Warts That Can Be Easily Identified by Patients

- Topical imiquimod (5% cream) at bedtime 3 nonconsecutive nights a week, for up to 16 weeks (BII). Each treatment should be washed with soap and water 6 to 10 hours after application.
- Topical podofilox (0.5% solution or gel) twice a day for 3 days, followed by 4 days of no therapy. Can be repeated, as necessary, up to four times (BIII).
- Topical sinecatechins (15% ointment) three times a day for up to 16 weeks until warts are cleared completely and not visible (BIII).
- Topical cidofovir 1% daily for 5 days per week for 8 weeks (CIII). Not commercially available but may be compounded in pharmacies with required equipment.

Provider-Applied Treatment Options

For Complex or Multicentric Lesions, or Lesions Inaccessible to Patient, or Due to Patient or Provider Preference

- Cryotherapy (liquid nitrogen or cryoprobe) applied until each lesion is thoroughly frozen, with treatment repeated every 1 to 2 weeks for up to 4 weeks until lesions are no longer visible (BIII). Some specialists recommend allowing the lesion to thaw and freezing a second time in each session (BIII).
- TCA and BCA (80% to 90%) applied to warts only and allowed to dry until a white frosting develops. The treatment can be repeated weekly for up to 6 weeks, until lesions are no longer visible (BIII).
- Intralesional cidofovir (15 mg/mL solution) injected directly into the wart (maximum 1 mL per session). May be repeated every 4 weeks for total of three to four treatments (CIII).
- Surgical treatments (e.g., tangential scissor excision, tangential shave excision, curettage, electrosurgery, electrocautery, infrared coagulation) can be used for external genital and anal warts (BIII). Laser surgery is an option but is usually more expensive (CIII).

Note: Many treatments for anogenital warts cannot be used in the oral mucosa. Surgery is the most common treatment for oral warts that interfere with function or for aesthetic reasons.

Considerations in Pregnancy

- Topical treatments such as BCA and TCA, as well as ablative therapies (i.e., laser, cryotherapy, and excision) can be used during pregnancy (AIII).
- Obstetrical management should not change for people with genital warts unless extensive condylomata might impede vaginal delivery or cause extensive bleeding (AIII).
- Cervical and anal cancer screening is recommended during pregnancy.
- Endocervical curettage is contraindicated during pregnancy (AIII).

Key: BCA = bichloroacetic acid, TCA = trichloroacetic acid

Treating Genital and Oral Warts

People with HIV may have larger or more numerous warts, may not respond as well to therapy for genital warts as individuals who are immunocompetent, and may have more frequent recurrences after treatment. Genital warts are not life-threatening and may regress without therapy, even in people with HIV and especially in those whose immunity is relatively preserved. Treatments are available for genital warts, but none are effective or preferred uniformly. Lacking RCTs specific to people with HIV, guidelines for the treatment of STIs in people without HIV should be followed. More than one treatment option may be required for refractory or recurrent lesions in people with HIV. Histologic diagnosis should be obtained for refractory lesions to confirm the absence of high-

grade disease. Intra-anal, vaginal, urethral, or cervical warts should be treated and managed by a specialist.

Patient-applied treatments are recommended generally for uncomplicated external warts that can be identified easily and treated by the patient. Imiquimod (5% cream) is a topical cytokine inducer that should be applied at bedtime on 3 nonconsecutive nights per week, for up to 16 weeks, until lesions are no longer visible. The treatment area should be washed with soap and water 6 to 10 hours after the application **(BII)**. Podofilox 0.5% solution or gel should be applied to visible anogenital warts twice a day for 3 days, followed by 4 days of no therapy. This cycle can be repeated, as necessary, up to four times **(BIII)**. Another option is sinecatechins (15% ointment), a topical botanical product that contains active catechins from green tea and should be applied three times daily for up to 16 weeks, until warts are cleared completely and not visible **(BIII)**.¹⁴³ No clinical trials of this latter treatment option have been conducted in people with HIV. Topical application of cidofovir or intralesional cidofovir has reported activity against genital warts **(CIII)**. Topical formulation is not commercially available but may be compounded in pharmacies with required equipment.¹⁴⁴⁻¹⁴⁶

Provider-applied treatments—such as cryotherapy, trichloroacetic acid (TCA), bichloroacetic acid (BCA), and surgery—typically are recommended for complex or multicentric lesions, lesions inaccessible to patient-applied therapy, or because of patient or provider preference.

Cryotherapy (liquid nitrogen or cryoprobe) destroys lesions by thermal-induced cytolysis and should be applied until each lesion is thoroughly frozen, with treatment repeated every 1 to 2 weeks for up to 4 weeks, until lesions are no longer visible **(BIII)**. Some specialists recommend allowing the lesion to thaw and freezing a second time in each session **(BIII)**.

TCA and BCA (80% to 90%) both act as caustic agents to destroy wart tissue and should be applied to warts only and allowed to dry until a white frosting develops. If an excess amount of acid is applied, the treated area should be powdered with talc, sodium bicarbonate, or liquid soap to remove unreacted acid. The treatment can be repeated weekly for up to 6 weeks, until lesions are no longer visible **(BIII)**.

Surgical treatments (e.g., tangential scissor excision, tangential shave excision, curettage, electrosurgery, electrocautery, infrared coagulation) can be used for external genital and anal warts **(BIII)**. Laser surgery is an option but is usually more expensive **(CIII)**.

Intralesional interferon has been used for the treatment of genital warts, but because of cost, difficulty of administration, and potential for systemic adverse effects—such as fever, fatigue, myalgias, and leukopenia—it is not recommended for first-line treatment **(CIII)**.

Podophyllin resin may be an alternative provider-applied treatment, with strict adherence to recommendations on application. It has inconsistent potency in topical preparations and can have toxicity that may limit routine use in clinical practice.

No consensus on optimal treatments of oral warts exists. Treatments for anogenital warts cannot be used in the oral mucosa. Given the lack of RCTs, surgical removal is the most common treatment for oral warts that interfere with function or need to be removed for aesthetic reasons.

These recommendations align with the [CDC STI Treatment Guidelines](#).

Treating CIN and Cervical Cancer

People with HIV with CIN should be managed by a clinician with experience in colposcopy and treatment of cervical cancer precursors. In general, CIN in people with HIV should be managed according to [ASCCP guidelines](#).

People with satisfactory colposcopy (transformation is fully visualized) and biopsy-confirmed high-grade CIN (CIN 2/3) can be treated with either ablation (e.g., cryotherapy, laser vaporization, electrocautery, diathermy, cold coagulation) or excisional methods (e.g., loop electrosurgical excision procedure, laser conization, cold knife conization), whereas people with unsatisfactory colposcopy should be treated only with excisional methods **(AII)**. In patients with recurrent high-grade CIN, diagnostic excisional methods are recommended **(AII)**. Hysterectomy is acceptable for treatment of recurrent or persistent biopsy-confirmed high-grade CIN **(BII)**; if invasive disease is suspected, the patient should be managed in consultation with a gynecologic oncologist. The ASCCP guidelines for adolescents and young women aged 21 to 24 years should continue to be followed. In these patients, progression of lesions is more common, and so is recurrence. Therefore, close observation, as outlined in the guidelines, should be considered for management of CIN 1; CIN 2; CIN 2,3 not otherwise specified (when pathology is HSIL but does not specify if CIN 2 or 3); and histologic HSIL in adolescents and adults with HIV who are younger than 25 years **(BIII)**. If concern for loss to follow-up, excisional methods of treatment for CIN 2; CIN 2,3; and HSIL may be preferred **(BIII)**.

Management of ICC may follow [National Comprehensive Cancer Network \(NCCN\) guidelines](#). Although complication and failure rates may be higher in people with HIV, standard treatment appears safe and efficacious.¹⁴²

Treating VIN, Vulvar Cancer, VAIN, and Vaginal Cancer

Low-grade VIN/VAIN (VIN/VAIN1) can be observed or managed the same as vulvovaginal warts. Treatment of high-grade VIN/VAIN should be individualized in consultation with a specialist and is dependent upon the patient's medical condition and the location and extent of the disease. Various treatment modalities are available for VIN, including local excision, laser vaporization, ablation, and topical therapies (e.g., imiquimod or cidofovir¹³⁵ therapy). Treatment options for VAIN include topical 5-fluorouracil (5-FU), laser vaporization with CO₂ laser, and excisional procedures.¹⁴⁷⁻¹⁴⁹

Management of vulvar and vaginal cancer must be individualized in consultation with a specialist, following NCCN guidelines.

Treating AIN and Anal Cancer

The ANCHOR study was not designed to compare different treatment modalities for efficacy. However, almost all participants were treated with office-based ablation of HSIL, most often hyfrecation. The rate of treatment-associated serious adverse events was very low. Office-based hyfrecation is therefore a reasonable first-line approach to treatment of anal HSIL **(AI)**.⁷³ Those with anal cancer should be referred to Oncology for appropriate treatment.

Treating HPV-Associated Disease at Other Sites, Including the Penis and the Oropharynx

Penile and some oropharyngeal cancers are associated with HPV infection. Treatment options do not differ for men and women with and without HIV. Data suggest a more favorable prognosis for HPV-associated oropharyngeal cancers than for non-HPV-associated oropharyngeal cancers.¹⁵⁰⁻¹⁵² Surgery, chemotherapy, and radiation are treatment modalities used for oropharyngeal cancers.

Special Considerations Regarding Antiretroviral Therapy Initiation

Given the strong evidence that early ART initiation is clinically beneficial in reducing risk of AIDS and opportunistic infections (OIs), there is no reason to consider HPV-related oral, anal, or genital disease when deciding whether or when to initiate ART.

Monitoring Response to Therapy and Adverse Events (Including IRIS)

Monitoring by physical examination is required during and after treatment of genital warts to detect toxicity, persistence, or recurrence, all of which are common with each of the treatments. Because recurrences of CIN and cervical cancer after conventional therapy are more common with HIV, these individuals should be followed after treatment with frequent cytologic screening and colposcopic examination (see Preventing Disease and Treating Disease sections). Treatment of CIN with ablative and excisional modalities can be associated with several adverse events, such as pain and discomfort, intraoperative hemorrhage, postoperative hemorrhage, infection, and cervical stenosis. Individualized treatment of adverse events is required.

Each of the treatment modalities for AIN described above is associated with adverse events, primarily pain, bleeding, ulceration, and, in rare cases, development of abscesses, fissures, or fistulas. Patients can be monitored for adverse events using the methods previously described.

Treatment for anal cancer with combination radiation and chemotherapy is associated with a high rate of morbidity, even when the treatment is successful. The most important complication is radiation-associated proctitis.

During IRIS, HPV may manifest as a paradoxical increase in warts after introduction of ART or by inflammation of existing warts.^{153,154} A few studies also have shown the development of oral warts while starting ART.¹⁵⁵⁻¹⁵⁸

Managing Treatment Failure

For persistent or recurrent genital warts, retreatment with any of the modalities previously described should be considered (**AIII**). Biopsy should be considered to exclude VIN. Genital warts often require more than one course of treatment.

Recurrent cytologic and histologic abnormalities after therapy for CIN should be managed according to [ASCCP guidelines](#).

No consensus on the treatment of biopsy-proven recurrent VIN exists, and surgical excision can be considered.

Preventing Recurrence

Monitoring after therapy for cervical disease should follow [ASCCP guidelines](#). In one study of women with HIV treated for high-grade CIN, low-dose intravaginal 5-FU (2 g twice weekly for 6 months) reduced the short-term risk of recurrence. Clinical experience with this therapy, however, is too limited to provide a recommendation for its use, and no follow-up study to confirm these observations has been reported. No guidelines exist regarding frequency of monitoring after therapy for VIN, but twice-yearly vulvar inspection appears reasonable for people who have been treated for VIN. People who have been treated for high-grade VAIN should be managed like those with CIN 2, that is, with cytology at 6 and 12 months after therapy, and annually thereafter.

No indication exists for secondary prophylaxis (chronic maintenance therapy) with any of the conventional modalities to prevent recurrence of genital warts, CIN, or AIN.

Special Considerations During Pregnancy

Pregnant women with HIV who have genital warts or anogenital HPV-related neoplasia are best managed by an interdisciplinary team of specialists, such as an obstetrician or gynecologist and an infectious disease provider. Pregnancy may be associated with an increased frequency and rate of growth of genital warts. Podofilox should not be used during pregnancy (**BIII**). At present, the evidence is insufficient to recommend imiquimod use during pregnancy. No anomalies have been observed with the use of imiquimod in animals during pregnancy. Several case series describe the use of imiquimod during pregnancy, also without any significant adverse effects.^{159,160}

Other topical treatments—such as BCA and TCA—and ablative therapies (i.e., laser, cryotherapy, and excision) can be used during pregnancy (**AIII**).

Transmission of genital HPV6 and 11 from vaginal secretions at delivery is the presumed mechanism of juvenile-onset recurrent respiratory papillomatosis in children. This condition is rare but is seen more frequently among children born to women who have genital warts at delivery. Cesarean delivery is not known to prevent this condition in infants and children.¹⁶¹ No change in obstetrical management is indicated for people with genital warts unless extensive condylomata are present that might impede vaginal delivery or cause extensive bleeding (**AIII**).

Cervical and anal cancer screening is recommended during pregnancy. Cytobrush sampling can be done during pregnancy. Pregnant women with abnormal cervical cytology results should undergo colposcopy and cervical biopsy of lesions suspicious for high-grade disease or cancer (**BIII**). Increased bleeding may occur with cervical biopsy during pregnancy. Endocervical curettage is contraindicated during pregnancy (**AIII**).

During pregnancy, ASC-US or LSIL can be managed the same as for all people with HIV, although deferral of colposcopy until at least 6 weeks postpartum is acceptable (**CIII**). Treatment of CIN is not recommended during pregnancy unless invasive disease is suspected (**AIII**). Patients with suspected cervical cancer should be referred to a gynecologic oncologist for definitive diagnosis, treatment, and development of a delivery plan. Vaginal delivery is not recommended for people with ICC (**AIII**). For people with CIN and without suspicion of invasive disease, re-evaluation with co-testing and colposcopy is recommended after 6 weeks postpartum (**AIII**). People with CIN can deliver vaginally.

Pregnancy testing is not needed before vaccination. HPV vaccination is not recommended during pregnancy **(CIII)**, as there are limited data for its use in pregnancy; however, no intervention is needed if inadvertently given.¹⁶² In a combined analysis of five RCTs of the HPV6/11/16/18 vaccine, administration of the vaccine to women who became pregnant during the course of the trial did not appear to negatively affect pregnancy outcomes.¹⁶³ Additionally, in a population-based study in Denmark, no increased risk of spontaneous abortion, stillbirth, or infant mortality was observed in more than 5,200 pregnancies exposed to at least one dose of the quadrivalent HPV vaccine. Also in Denmark, an analysis of the Medical Birth Register and National Patient Register found that among 1,665 exposed pregnancies, quadrivalent HPV vaccination was not associated with a significantly increased risk of adverse pregnancy outcomes, including major birth defect, preterm birth, or low birth weight.¹⁶⁴ Data on the use of the 9-valent vaccine during pregnancy are more limited, but to date are also reassuring.¹⁶⁵⁻¹⁶⁹

The effects of treatment of AIN on pregnancy are unknown. Most experts recommend deferral of diagnosis and treatment of AIN until after delivery unless a strong clinical suspicion of anal cancer exists.

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