HPV-RELATED DISEASE IN HIV PATIENTS

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F006 - AIDS AND STDs: HOT TOPICS
DISCLOSURE OF RELEVANT RELATIONSHIPS WITH INDUSTRY

Carrie Kovarik, MD
F006 - AIDS and STDs: Hot Topics

I do not have any relevant relationships with industry.
HPV IN HIV PATIENTS

- General verruca
- Condyloma/carcinoma
- Periungual warts/carcinoma
- Oral warts/condyloma/carcinoma
- Plantar warts
- Diffuse flat warts
- Laryngeal papillomas/carcinoma
- Skin cancers?
Table 1. Recommended and Alternative Regimens for Treatment of External Anogenital Warts

<table>
<thead>
<tr>
<th>Recommended Patient-Applied Regimen</th>
<th>Dosing</th>
</tr>
</thead>
<tbody>
<tr>
<td>Imiquimod 5% cream</td>
<td>Topically every night at bedtime for 3 times/wk up to 16 wk</td>
</tr>
<tr>
<td>Imiquimod 3.75% cream</td>
<td>Topically every night at bedtime up to 16 wk</td>
</tr>
<tr>
<td>Podophlox 0.5% solution or gel</td>
<td>Topically twice daily x 3 d followed by 4 d off for up to 4 cycles</td>
</tr>
<tr>
<td>Sinecatechins 15% ointment</td>
<td>Topically 3 times daily, for up to 16 wk</td>
</tr>
<tr>
<td>Bichloroacetic acid 80%–90%</td>
<td>Applied once every 1–2 wk</td>
</tr>
<tr>
<td>Cryotherapy</td>
<td>Applied once every 1–2 wk</td>
</tr>
<tr>
<td>Surgical removal</td>
<td></td>
</tr>
<tr>
<td>Trichloroacetic acid 80%–90%</td>
<td>Applied once every 1–2 wk</td>
</tr>
</tbody>
</table>

Source: CDC, MMWR Recomm Rep 2015; 64(No. RR-3):1–137.
Human papillomavirus–related genital disease in the immunocompromised host

Part I

Rachel H. Gormley, MD, and Carrie L. Kovarik, MD

Philadelphia, Pennsylvania

CAPSULE SUMMARY

- Human papillomavirus causes both benign condyloma acuminata and anogenital malignancies.
- Immunosuppressed patients, including patients with HIV infection and organ transplant recipients, are at increased risk for developing these conditions.
- Patients with HIV infection and organ transplant recipients require heightened screening and aggressive treatment.
**PERSISTENCE OF HPV RELATED SKIN DISEASE**

- Immune status has a significant impact on HPV disease course & response to treatment
- **Increased rates** of HPV infection, with increased duration/persistence in HIV pts
- In addition to **increasing susceptibility** to new HPV infection, HIV ↑ reactivation of latent virus and hastens the course of established HPV infections
- Reduced cytotoxic T-lymphocyte reactivity to HPV oncoproteins E6 and E7 leads to impaired ability to clear HPV

PERSISTENCE OF HPV RELATED SKIN DISEASE

- Beneficial effect of HAART may be less pronounced when \textit{HPV-related disease has progressed} with HIV, because:
  - HPV-specific immunity has been irreversibly damaged, or
  - HPV related changes have persisted long enough the sufficient genetic changes have accumulated by the time of HAART initiation.

• Cancers not previously associated with HIV/AIDS are increasing in incidence.
• These *non–AIDS-defining cancers* (NADCs) include cancers of the lung, liver, kidney, anus, head and neck, skin, Hodgkin’s lymphoma, & others.
• In populations benefiting from ART, some NADCs appear to have a *higher relative incidence* compared with the same cancer rates seen in the general population, even after controlling for known cancer risk factors.
Non–AIDS-Defining Cancer Mortality: Emerging Patterns in the Late HAART Era

Antonella Zucchetto, PhD,* Saverio Virdone, ScD,* Martina Taborelli, ScD,* Enrico Grande, ScD,† Laura Camoni, PsyD,‡ Marilena Pappagallo, ScD,† Vincenza Regine, ScD,‡ Francesco Gripp, ScD,‡ Jerry Polesel, ScD,* Luigino Dal Maso, PhD,* Barbara Suligoi, MD,‡ Luisa Frova, PhD,† and Diego Serraino, MD*

J Acquir Immune Defic Syndr • Volume 73, Number 2, October 1, 2016

• Non–AIDS-defining cancers (not KS, NHL, cervix) are the leading non–AIDS-related causes of death in the late HAART era, among both people with HIV and people with AIDS (PWA).

• Significant excess mortality emerged for cancers associated with viruses, for which HIV-infected individuals are likely to lose the immune control of infections, and for cancers associated with unhealthy behaviors, such as tobacco smoking.
The Rising Challenge of Non–AIDS-Defining Cancers in HIV-Infected Patients

John F. Deeks,¹ Angelique Tjen-A-Looi,² Michelle A. Rudak,³ Catherine Okulier,³ Mary Young,² Richard F. Little,⁵ and Bruce J. Desnick⁴

Table 1. Standard Incidence Ratios of Selected Non–AIDS-Defining Cancers [2, 5–8]

<table>
<thead>
<tr>
<th>Non–AIDS-Defining Cancer</th>
<th>Cancer Risk (Standardized Incidence Ratio)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anal</td>
<td>33.4–42.9</td>
</tr>
<tr>
<td>Hodgkin’s Lymphoma</td>
<td>14.7–31.7</td>
</tr>
<tr>
<td>Liver</td>
<td>7.0–7.7</td>
</tr>
<tr>
<td>Skin</td>
<td></td>
</tr>
<tr>
<td>Squamous cell carcinoma/Basal cell</td>
<td>3.2</td>
</tr>
<tr>
<td>Melanoma</td>
<td>1.1–2.6</td>
</tr>
<tr>
<td>Head and Neck</td>
<td>1.0–4.1</td>
</tr>
<tr>
<td>Lung</td>
<td>2.2–6.6</td>
</tr>
<tr>
<td>Leukemia</td>
<td>2.2–2.5</td>
</tr>
<tr>
<td>Renal</td>
<td>1.8–2.2</td>
</tr>
<tr>
<td>Cancer Site/Type (ICD-10 Codes)*</td>
<td>Total (14,180 Person-Years)</td>
</tr>
<tr>
<td>----------------------------------</td>
<td>-----------------------------</td>
</tr>
<tr>
<td></td>
<td>Obs./Exp.</td>
</tr>
<tr>
<td>AIDS-defining</td>
<td>282/0.7</td>
</tr>
<tr>
<td>Kaposi sarcoma (C46)</td>
<td>63/0.1</td>
</tr>
<tr>
<td>Cervix uteri (C53)</td>
<td>6/0.1</td>
</tr>
<tr>
<td>Non-Hodgkin lymphoma (C82–88, C96)</td>
<td>221/0.6</td>
</tr>
<tr>
<td>Non-AIDS-defining†‡</td>
<td>127/17.3</td>
</tr>
<tr>
<td>Head and neck (C00–14, C30–32)</td>
<td>9/1.1</td>
</tr>
<tr>
<td>Stomach (C16)</td>
<td>4/1.2</td>
</tr>
<tr>
<td>Colon-rectum (C18–20)</td>
<td>10/1.8</td>
</tr>
<tr>
<td>Anus (C21)</td>
<td>5/&lt;0.1</td>
</tr>
<tr>
<td>Liver and bile ducts (C22)</td>
<td>17/1.3</td>
</tr>
<tr>
<td>Bronchus and lung (C34)</td>
<td>38/4.7</td>
</tr>
<tr>
<td>Skin melanoma (C43)</td>
<td>4/0.4</td>
</tr>
<tr>
<td>Uterus, not otherwise specified (C55)</td>
<td>4/&lt;0.1</td>
</tr>
<tr>
<td>Hodgkin lymphoma (C81)</td>
<td>12/&lt;0.1</td>
</tr>
<tr>
<td>Leukemia (C91-95)</td>
<td>5/0.7</td>
</tr>
</tbody>
</table>
Younger ages at diagnosis were observed for PLWH compared with general population for lung, anal, oral cavity/pharynx, kidney cancers and myeloma.
Table 2. Comparison of Median Ages at Diagnosis Between HIV-Infected People in the North American AIDS Cohort Collaboration on Research and Design (NA-ACCORD) and the General Population, After Weighting the General Population to the Age Structure of NA-ACCORD

<table>
<thead>
<tr>
<th>Cancer</th>
<th>Observed in HIV-Infected Individuals</th>
<th>Observed in General Population</th>
<th>General Population After Weighting*</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Cases</td>
<td>Median Age, y</td>
<td>Median Age, y</td>
<td>Median Age, y</td>
</tr>
<tr>
<td>Lung</td>
<td>644</td>
<td>54</td>
<td>68</td>
<td>58</td>
</tr>
<tr>
<td>Prostate</td>
<td>504</td>
<td>58</td>
<td>66</td>
<td>59</td>
</tr>
<tr>
<td>Anus</td>
<td>291</td>
<td>47</td>
<td>57</td>
<td>51</td>
</tr>
<tr>
<td>Liver</td>
<td>226</td>
<td>54</td>
<td>62</td>
<td>54</td>
</tr>
<tr>
<td>Oral cavity and pharynx</td>
<td>173</td>
<td>51</td>
<td>60</td>
<td>53</td>
</tr>
<tr>
<td>Hodgkin lymphoma</td>
<td>171</td>
<td>44</td>
<td>39</td>
<td>45</td>
</tr>
<tr>
<td>Colon</td>
<td>111</td>
<td>55</td>
<td>67</td>
<td>56</td>
</tr>
<tr>
<td>Kidney</td>
<td>109</td>
<td>52</td>
<td>62</td>
<td>54</td>
</tr>
<tr>
<td>Larynx</td>
<td>86</td>
<td>53</td>
<td>63</td>
<td>56</td>
</tr>
<tr>
<td>Melanoma (whites only)</td>
<td>77</td>
<td>49</td>
<td>56</td>
<td>51</td>
</tr>
<tr>
<td>Breast</td>
<td>56</td>
<td>48</td>
<td>58</td>
<td>47</td>
</tr>
<tr>
<td>Pancreas</td>
<td>55</td>
<td>53.5</td>
<td>67</td>
<td>57</td>
</tr>
<tr>
<td>Myeloma</td>
<td>49</td>
<td>52</td>
<td>66</td>
<td>56</td>
</tr>
</tbody>
</table>
HPV RELATED MALIGNANCY IN HIV
Spectrum and progression of disease from condyloma to aggressive anogenital squamous cell carcinoma in 3 HIV-positive patients

Olaf Rodriguez, BS, and Carrie L. Kovarik, MD
Phialadelphia, Pennsylvania
21yo HIV positive female on HAART (CD4 183) with genital warts not responding to cryotherapy
45 yo HIV + female with biopsy proven SCCIS on the left inner thigh, needing scouting biopsies to rule out invasive SCC
Perianal Bowen Disease in a Child with Human Immunodeficiency Virus

Kathleen A. Carroll, M.D.,* Jeffrey Pierce, M.D.,† and Carrie L. Kovarik, M.D.‡

Squamous Cell Carcinoma in situ
41 yo female HIV + (CD4 952) with large verrucous macerated plaque

Biopsy showed SCCIS
38 yo female, HIV+, history of cervical cancer s/p hysterectomy and 2 year history of “ulcers” on the perineum
Invasive Squamous Cell Carcinoma
59 yo HIV + CD4 450, on HAART, growing over the last 2-3 years since circumcision
Invasive Squamous Cell Carcinoma
Clearance of HPV infection, including infection with oncogenic types, was slower in the glans/coronal sulcus of the penis of uncircumcised men than circumcised men.
53yo HIV(+) with CD4 count 600's & a few month history of friable nodular exophytic penile plaque with surrounding verrucous change

Invasive Squamous Cell Carcinoma
Squamous Cell Carcinoma

Penile Cancer

Vulvar Cancer

Courtesy of Dr. Justin Finch
Anal cancer is rare in general population (1–2 cases/100,000 PY), but burden is much higher among MSM
- Anal cancer incidence HIV-neg MSM = 5 cases/100,000 PY
- Anal cancer incidence HIV-pos MSM = 45.9 cases/100,000 PY

Anal HPV is nearly ubiquitous in HIV+ MSM (93%), with
- **High-risk HPV prevalence = 73.5% for HIV-pos MSM**
- High-risk HPV prevalence = 37.2% for HIV-neg MSM

> ½ of HIV+ MSM have **abnormal cytology** (57%), and 29% have HSIL

Incidence of HSIL among HIV+ MSM ranges from 8.5-15.4% PY

*HSIL=high grade squamous intraepithelial lesion*
Distribution of HPV Genotype in Anal Condyloma (CA): Higher Prevalence of HR-HPV in MSM with HIV

- HR-HPV was detected in 45.2% of CA and HSIL in 15.3%.
- **Prevalence of HR-HPV:**
  - HIV+ MSM (70.4%)
  - HIV-negative MSM (33.3%, \( P=0.0311 \))
  - HIV-negative MSW (18.8%, \( P=0.0016 \)).
- Logistic regression showed **HIV+ as primary risk factor for HR-HPV** infection in CA.
- **HSIL was detected in 25% of CA from HIV+ MSM** (25.9%), compared to none in HIV-negative MSW (0.0%, \( P=0.0346 \)).
- **Surveillance** is a necessity for the HIV+ MSM population!
Higher Prevalence of HR HPV in MSM with HIV Infection

Figure 1. Distribution of HPV genotypes in CA patients. (A) Distribution of HPV genotypes in overall CA patients or CA patients with or without HIV infection.
• **745 HIV+ women** were screened with anal cytology.
• 15-39% had abnormal anal cytology
• 208 underwent HRA following abnormal anal cytology: **HSIL found** in 18-26% of anal biopsies
• **Cigarette smoking** > doubled HSIL risk.
• Neither meeting criteria for screening nor history of receptive anal sex was significantly associated with HSIL.

### Table 3. Anal Cytology Results Preceding High-Resolution Anoscopy Evaluation Compared to High-Resolution Anoscopy Results

<table>
<thead>
<tr>
<th>Cytology</th>
<th>Benign</th>
<th>LSIL/Condyloma</th>
<th>HSIL/Cancer</th>
<th>PValue</th>
</tr>
</thead>
<tbody>
<tr>
<td>Whole cohort</td>
<td>&lt;.001</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ASCUS</td>
<td>70 (92)</td>
<td>48 (58)</td>
<td>31 (63)</td>
<td></td>
</tr>
<tr>
<td>ASC-H</td>
<td>0 (0)</td>
<td>4 (5)</td>
<td>1 (2)</td>
<td></td>
</tr>
<tr>
<td>LSIL</td>
<td>6 (8)</td>
<td>29 (35)</td>
<td>7 (14)</td>
<td></td>
</tr>
<tr>
<td>HSIL</td>
<td>0 (0)</td>
<td>2 (2)</td>
<td>10 (21)</td>
<td></td>
</tr>
</tbody>
</table>

Abbreviations: ASC-H, atypical squamous cells, cannot rule out HSIL; ASCUS, atypical cells of undetermined significance; HSIL, high-grade squamous intraepithelial lesion; LSIL, low-grade squamous intraepithelial lesion.
High-grade Dysplasia in Anogenital Warts of HIV-Positive Men

Alexander Kreuter, MD; Christos Siorokos, MD; Frank Oellig, MD; Steffi Silling, PhD; Herbert Pfister, PhD; Ulrike Wieland, MD

Key Points

**Question**  Do anogenital lesions of HIV-positive men that clinically appear as benign warts contain areas of dysplasia, and if so, what are the virological characteristics of those lesions?

**Findings**  In this case series, a high proportion of anogenital warts contained areas of high-grade and low-grade dysplasia or even invasive cancer. Some of these lesions contained only low-risk-HPV types. Dysplasia was absent in all lesions of immunocompetent control patients.
High-grade Dysplasia in Anogenital Warts of HIV-Positive Men

Box. Recommendations for the Diagnosis and Treatment of Anogenital Warts in HIV-Positive Men

**Clinical Examination**
1. Clinical inspection of the entire anogenital area
2. Documentation of size and location of anogenital warts (AGWs) and search for clinical signs of dysplasia (eg, punctuation, mosaicism, neovascularization/abnormal vessels)
3. Perform (or send patient to) high-resolution anoscopy to exclude anal intraepithelial neoplasia and/or anal carcinoma
4. Obtain biopsy specimens of representative lesions

**Histopathological Examination**
1. Perform routine histopathological examination (standard fixation with hematoxylin-eosin staining)
2. In case of histopathological signs for dysplasia, add immunohistochemical staining for Ki67 and p16
3. Optional: add HPV genotyping in case of signs of dysplasia in histopathology

**Treatment**
1. Perform ablative treatment of AGWs (eg, electrocautery, infrared coagulation, surgical excision)
2. Consider additional treatment with imiquimod 5% cream

\(^a\) Anal AGWs represent a risk factor for AIN and anal carcinoma
Digital squamous cell carcinoma and association with diverse high-risk human papillomavirus types

Rachel H. Gormley, BS,a Caroline M. Groff, MD, PhD,a Christopher J. Miller, MD,a and Carrie L. Kovarik, MD,a,b Philadelphia, Pennsylvania

- Digital squamous cell carcinoma (SCC) is rare and often mimics benign conditions, making it a diagnostic challenge.
- Digital SCC is often associated with high-risk, oncogenic human papillomavirus subtypes.
- Although the majority of previously published reports have implicated human papillomavirus-16, a variety of high-risk oncogenic subtypes, including human papillomavirus-16, -33, -51, and -73, may be associated with digital SCC.
- Digital SCCs often recur and aggressive treatment is recommended.

Case report

Human papillomavirus type 73 associated with multiple cutaneous squamous cell carcinomas in an immunosuppressed patient

Camille E. Introcaso¹, MD, Peter L. Rady², MD, PhD, Qin He², MD, Stephen K. Tyring³, MD, PhD, MBA, and Carrie L. Kovarik³, MD
ORAL HPV IN HIV PATIENTS

Treatment of oral condylomata acuminata in a HIV-1 patient with bleomycin

Leonor Girão, Isabel Franca, Helena Macedo, Carmo Ornelas, Maria Nunes, Carlos Araújo, Kamal Mansinho

J EADV (2000) 14, 313—333
Spectrum and progression of disease from condyloma to aggressive anogenital squamous cell carcinoma in 3 HIV-positive patients

Olaf Rodriguez, BS, and Carrie L. Kovarik, MD

Philadelphia, Pennsylvania

Fig 3. HPV-related oral leukoplakia with biopsy-proven invasive squamous cell carcinoma. This is the clinical appearance before Mohs surgery (A) and after clearance of the tumor (B).
Oral Human Papillomavirus in Men Having Sex with Men: Risk-Factors and Sampling

Tim R. H. Read1,2, Jane S. Hocking3, Lenka A. Vodstrcil1,2, Sepehr N. Tabrizi4, Michael J. McCullough5, Andrew E. Grulich6, Suzanne M. Garland4, Catriona S. Bradshaw1,7, Marcus Y. Chen1,2, Christopher K. Fairley1,2

Table 1. Prevalence of oral HPV types, by HIV status, in 500 men who have sex with men.

<table>
<thead>
<tr>
<th>HPV type</th>
<th>HIV negative N = 251 n (% 95% CI)</th>
<th>HIV positive N = 249 n (% 95% CI)</th>
<th>Prevalence ratioa</th>
<th>Overall n (% 95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Any HPV type</td>
<td>17 (7, 4–11)</td>
<td>48 (19, 15–25)</td>
<td>2.8</td>
<td>65 (13, 10–16)</td>
</tr>
<tr>
<td>HPV 16</td>
<td>2 (0.8, 0.1–3)</td>
<td>11 (4, 2–6)</td>
<td>5.5</td>
<td>13 (3, 1–4)</td>
</tr>
<tr>
<td>High risk HPV typesa</td>
<td>5 (2, 0.6–5)</td>
<td>20 (8, 5–12)</td>
<td>4.0</td>
<td>25 (5, 3–7)</td>
</tr>
<tr>
<td>HPV* types 6 or 11 or 16 or 18</td>
<td>5 (2, 0.6–5)</td>
<td>16 (6, 4–10)</td>
<td>3.2</td>
<td>21 (4, 3–6)</td>
</tr>
<tr>
<td>More than 1 type of HPV</td>
<td>3 (0.6, 0.2–3)</td>
<td>18 (7, 4–11)</td>
<td>6.0</td>
<td>21 (4, 3–6)</td>
</tr>
</tbody>
</table>

CI: confidence interval.
a: Ratio of prevalence in HIV-positive to HIV-negative MSM.
a: One or more of types 16, 18, 31, 33, 39, 45, 51, 52, 58, 59, 68 which are considered oncogenic in the cervix.
a: Included in the quadrivalent vaccine.

doi:10.1371/journal.pone.0049324.t001
Intralesional cidofovir for the treatment of a plantar wart

Elizabeth Moore, BA, and Carrie Kovarik, MD
Philadelphia, Pennsylvania

**Fig 1.** Plantar wart before treatment with intralesional cidofovir: 4 × 5 cm hyperkeratotic plaque on plantar aspect of right foot (A), and after 4 treatments with intralesional cidofovir (B).
First evidence of an association between HPV and non-melanoma skin cancer comes from patients with EDV.

EDV is a rare heritable disease characterized by cutaneous warts that display not only a high rate of progression to SCC on sun-exposed sites, but also a strong predisposition to infection by β-HPVs, for which HPV 5 and 8 predominate.

Two EV genes (EVER1 and EVER2) identified.

Variant genotype in this study was related to SCC risk [adjusted OR for homozygous variant versus homozygous wild type for the EVER2 polymorphism 1.7, 95% CI 1.1–2.7].
EDV-LIKE FLAT WARTS
Acquired Epidermodysplasia Verruciformis Syndrome in HIV-Infected Pediatric Patients: Prospective Treatment Trial With Topical Glycolic Acid and Human Papillomavirus Genotype Characterization

Rachael L. Moore, MD
Virginie de Schaetzen, MD
Marissa Joseph, MD
Ivy Ann Lee, MD
Yoette Miller-Montheope, MD
B. Ryan Phelps, MD, MPH
Shimane S. Lehalale, RN
Sarah J. Ratcliffe, PhD
Harrison Nguyen
Qin He, MD
Peter Rady, MD, PhD
Stephen Tyring, MD, PhD, MBA
Carrie L. Kovarik, MD
• Majority of HPV associated **cancers are caused by HPV 16 or 18** (65% females, 63.3% males)

• **10% are attributable** to 5 additional types HPV 31, 33, 45, 52, 58 (14% females, 4% males)

• **Advisory Committee on Immunization Practices recommends:**
  – Routine HPV vaccination for girls at age 11 or 12 (9v, 4v or 2v) & boys (9v, 4v)
  – Also recommend vaccination if not vaccinated previously:
    • For girls from age 13-26 (9v, 4v or 2v)
    • For boys from age 13-21 (9v, 4v)
    • For men through age 26 if MSM or immunocompromised (HIV) (9v, 4v)
Human Papillomavirus and Genital Warts: A Review of the Evidence for the 2015 Centers for Disease Control and Prevention Sexually Transmitted Diseases Treatment Guidelines

Ina U. Park,¹² Camille Introcasa,² and Eileen F. Dunne²*
Clinical Infectious Diseases® 2015;61(S8):S849–55

Table 3. Human Papillomavirus Vaccine Recommendations From the Advisory Committee on Immunization Practices

<table>
<thead>
<tr>
<th>Population</th>
<th>Age Group, y</th>
<th>Recommendation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Females</td>
<td>11–12 (may start at 9)</td>
<td>Routine vaccination with either 2vHPV, 4vHPV, or 9vHPV</td>
</tr>
<tr>
<td></td>
<td>13–26</td>
<td>Routine vaccination with either 2vHPV, 4vHPV, or 9vHPV³</td>
</tr>
<tr>
<td>Males</td>
<td>11–12 (may start at 9)</td>
<td>Routine vaccination: 4vHPV or 9vHPV</td>
</tr>
<tr>
<td></td>
<td>13–21</td>
<td>Routine vaccination: 4vHPV or 9vHPV</td>
</tr>
<tr>
<td></td>
<td>22–26</td>
<td>4vHPV or 9vHPV may be administered</td>
</tr>
<tr>
<td>MSM and HIV⁺</td>
<td>22–26</td>
<td>Routine vaccination: 4vHPV or 9vHPV</td>
</tr>
</tbody>
</table>
The Human Papillomavirus Vaccine: Current Perspective and Future Role in Prevention and Treatment of Anal Intraepithelial Neoplasia and Anal Cancer

Felix A. Mensah, Mudresh R. Mehta, James S. Lewis Jr., A. Craig Lockhart

Implications for Practice: The incidences of human papillomavirus (HPV)-related anal cancer and its precursor lesion, anal intraepithelial neoplasia, are on the rise in the U.S. and globally. Based on recent studies, the HPV vaccine is approved for prevention of the infection and development of HPV-related anal cancer. In addition, several small studies have shown that the vaccine may be useful as adjuvant therapy for anal cancer. There is a need for public health strategies aimed at education of both patients and practitioners to improve the use of the vaccine for prevention of HPV-related anal cancer. The development of a therapeutic vaccine is a work in progress.
QUESTIONS?