1. (Amy) What is the role of viral titers and CD4 counts in the management of HIV?

   The CD4 count refers to the number of T-helper lymphocytes with CD4 cell surface markers. It is used to assess immune status and susceptibility to opportunistic infections (OI), to assess the need for HAART and OI prophylaxis, and to define AIDS (CD4 <200). Normal adult CD4 count reference ranges vary by laboratory but are generally within 500 -1500/mm³. CD4% is less variable than absolute CD4 count but is less useful for predicting risk of OI. CD4 counts have seasonal and diurnal variation (lowest at 12:30 PM, highest at 8:30 PM) and are also affected by surgery, viral infections, and tuberculosis. Counts decrease with some medications, such as corticosteroids (especially with acute use), IFN, and chemotherapy. Acute changes also arise due to redistribution of CD4 in lymphatics, spleen and bone marrow. Sex, race, psychologic stress, & physical stress have minimal effects on CD4.

   CD4 counts decline abruptly after acute infection and often return to normal range. CD4 counts generally decline with increasing viral load. HAART causes a biphasic increase in CD4 counts. In approximately 10% of cases, HAART results in viral load suppression without an increase in CD4 counts. CD4 counts are helpful in determining when to start antiretroviral treatment and medications to prevent OIs. Usually, counts are checked more frequently in patients on HAART (q2-4 months) than those who are not currently being treated (q3-6 months)

   Viral load is a measurement of the amount of HIV in the blood. Primary HIV infection is associated with a markedly elevated VL. A VL above 100,000 copies/mL is "high". A VL less than 50 copies/mL is "undetectable". The VL trend is more significant than the individual measure, which can fluctuate greatly. VL and CD4 counts are useful in monitoring disease progression and response to HAART.

2. (Amy) What drug classes and medications currently comprise the preferred treatment regimen for HIV and AIDS? Is there a role for drug holiday?

   Highly active antiretroviral therapy (HAART) against HIV is a combination of drugs with the goal of preventing viral replication and delaying the onset of AIDS. A common initial regimen is two NRTIs and a NNRTI or protease inhibitor. Common side effects of HAART include bone marrow suppression, lipid abnormalities, renal toxicity, transaminitis, rash, and GI intolerance. Possible serious toxicities include hepatic necrosis, Stevens-Johnson syndrome/TEN, severe lactic acidosis, hypersensitivity reactions, pancreatitis, and peripheral neuropathy. In general, whenever a particular agent needs to be interrupted or discontinued, all agents should be stopped simultaneously to avoid the risk of resistance during monotherapy. Indications to change medications include development of new neurologic symptoms, clinical disease progression, worsening immunologic function, increasing viral load, or a lack of decrease in viral load.

   **Nucleoside reverse transcriptase inhibitors (NRTI):** interrupt viral replication; Examples: zidovudine (AZT), zalcitabine (Hivid), stavudine (Zerit), lamivudine (Epivir), emtricitabine

   **Non-nucleoside reverse transcriptase inhibitors (NNRTI):** work in combination with other drugs to interrupt viral replication; Examples: delavirdine (Rescriptor), nevirapine (Viramune)

   **Protease inhibitors:** interrupt viral replication at a later step in its life cycle; Examples: ritonavir (Norvir), lopinavir/ritonavir combination (Kaletra), saquinavir (Invirase), indinavir sulphate (Crixivan), amprenavir (Agenerase), and nelfinavir (Viracept)

   **Fusion inhibitors:** newest drug class, blocks HIV from entering lymphocytes; Example: enfuvirtide (Fuzeon)

   Anti-retroviral medications are associated with significant side effects, which can reduce compliance to treatment regimens. Structured treatment interruption (STI) has been proposed by some researchers. STI involves cycling patients on and off of HAART in order to suppress viral replication while also decreasing toxicity and improving patient compliance. Many theories exist for the potential efficacy of STI. In primary infection, STI may stimulate immune response to HIV antigens. In chronic infection, STI may decrease drug toxicity. In drug resistance, it may allow the re-emergence of drug-sensitive virus. Initial trials of STIs have not been encouraging; their use is experimental and not a standard part of the HIV treatment regimen.

3. (Deya) Classify and define the causes of cervical adenopathy in HIV patients.

   **DDx of LAD in HIV patients**

   I. Infectious
      - Mycobacterial lymphadenitis: tuberculosis* and atypical organisms
      - Pneumocystis lymphadenitis*
      - Pneumocystis thyroiditis*
      - Viral lymphadenitis: cytomegalovirus, Epstein-Barr virus
      - *Toxoplasma* lymphadenitis
      - Bacterial lymphadenitis or abscess secondary to oropharyngeal infection
      - Cat-scratch disease

   II. Neoplastic
      - Lymphoma- presents as a growing mass and constitutional symptoms (fever, night sweats, wt loss).
      i. Non-Hodgkin's (AIDS-defining illness)- risk rises in conjunction with duration of HIV. Majority of pts have CD4+ < 200. HAART has lowered the incidence of NHL in HIV pts.
ii. Hodgkin's disease- most common type of non-AIDS-defining tumor. Risk of HL 10-fold and course is more aggressive in HIV+ patients. Histology more frequently c/w aggressive mixed cellularity and lymphodepleted subtypes.

- Metastatic Kaposi's sarcoma (AIDS-defining illness)- mesenchymal cell tumor involving blood and lymphatic vessels.

Generally accepted that HHV-8 plays a causative role in the development of KS. KS occurs in the H&N in as many as 63% of cases. Cutaneous dx presents as multicentric purple or red macular lesions that are neither tender nor blanching; frequently coalesce to violaceous, nodular lesions. Mucosal KS more likely to be symptomatic with pain, ulceration, and bleeding. HAART has resulted in a clear decline in the incidence of AIDS-KS.

- Metastatic carcinoma
- Metastatic melanoma
- Salivary gland tumors
- Thyroid tumors

III. Idiopathic (Significantly increased in HIV)

- Persistent generalized lymphadenopathy- common early Sx of HIV and a common cause of cervical LAD. Defined as LAD w/o identifiable infectious or neoplastic etiology, involves two or more extracranial sites for at least 3 months, in a patient at risk for or confirmed to be HIV infected. It is the most common cause of cervical LAD in HIV patients (present in 12-45% of HIV pts). 3 histological subtypes: follicular hyperplasia, follicular involution, lymphoid depletion (often see progression in that order as HIV infection approaches end stage AIDS).

- Lymphoepithelial cysts of the parotid gland

Work-up of cervical LAD includes thorough H&P and FNA. LAD greater than 2 cm, unilateral, painful, deep, or asymmetric is suspicious for pathology, specifically granulomatous disease or lymphoma. Tender LAD is more likely to be secondary to bacterial infections, including TB, whereas nontender enlarging neck nodes may result from malignancy. Thorough PE should include search for potential primary sites of infection or malignancy. Lymphoma or mycobacterial infection are more likely to be present when the CD4+ count is less than 100 cells/mL or a history of AIDS is present, whereas PGL is more likely when the CD4+ count is greater than 500 cells/mL.

4. (Deya)What is the differential diagnosis for otorrhea in HIV patients?

**HIV related diseases of outer & middle ear**

I. OE- risk of OE and malignant OE is increased in patients with AIDS, with *P. aeruginosa* being the primary causative agent of both disease entities. Susceptibility to *P. aeruginosa* is heightened by impaired humoral immunity, neutrophil function, and complement activation. OE may progress to necrotization and malignant OE if not managed aggressively and in a timely fashion. Tx: frequent cleaning of EAC and otic drops with adequate pseudomonal coverage, with duration dependent on response. PO abx, particularly fluoroquinolones, should be considered for severely immunocompromised or when auricular perichondritis is present.

a. Temporal bone osteomyelitis- should be suspected when otalgia, swelling, and otorrhea persist despite therapy or when there is onset of facial nerve paralysis. Often see granulation at bony cartilaginous junction of EAC with diagnosis confirmed by bone scan. EAC debris should be cultured and stained for bacteria, fungi, acid-fast bacilli, and *Pneumocystis* sp. Management of malignant OE consists of 6 weeks of broad-spectrum intravenous antibiotics with adequate coverage against *P. aeruginosa*. Serial bone scans help monitor the response to therapy.

II. OM- ET obstruction caused by adenoidal hypertrophy or sinonasal disease is prevalent in HIV. Frequency of recurrent AOM increases as immune function deteriorates. Bacteriology generally that same as healthy individuals except in severely immunocompromised pts in whom *S. aureus* causes OM at a significantly higher rate.

III. *P. carinii* otomastoiditis and OE- p/w unilateral otalgia, otorrhea, hearing loss, and a polypoid mass on otoscopy. CT T-bone reveals bony sclerosis without erosions and opacification of the middle ear and mastoid air spaces. Persistent aural polyp w/ persistent symptoms despite otic drops and abx should undergo biopsy for histopathology and special stains (silver stain). Tx: Bactrim Serial bone scans help monitor the response to therapy.

IV. Invasive aspergillosis of the external and middle ear- *A. fumigatus* infection of the EAC causes chonic OE in immunocompetent HIV pts and but in severely immunocompromised patients, invasive aspergillus infection of the temporal bone can cause malignant OE and otomastoiditis with skull base and intracranial extension. Risk factors: low CD4+ count. AIDS diagnosis, neutropenia, corticosteroid therapy, anti-neoplastic therapy, and prolonged antibiotic therapy. Sx: otalgia, otorrhea, and hearing loss. Otoscopy reveals white debris in EAC resembling cholesteatoma; facial nerve weakness indicates bone invasion, which can often be confirmed by the presence of bony erosion on temporal bone CT. May cause destruction of ossicles, erosion of the facial canal with nerve invasion, and destruction of dural plates with possible extension to the dural sinuses and other intracranial structures. Tx: prompt surgical (debridement) and drug therapy (high-dose amphotericin B).

5. (Deya)What clinical factors distinguish HIV from AIDS?

All patients with AIDS are HIV +, but not all HIV+ patients have AIDS. When HIV infection becomes advanced, it is referred to as AIDS. AIDS is defined as CD4 count below 200/mL OR history of one of the 26 opportunistic infections designated as "AIDS-defining illnesses." Such illnesses include (among others) things like pneumocystis carinii pneumonia, toxoplasmosis, TB, wasting syndrome, meningitis, fungal infections, salmonella septicemia, histoplasmosis, CMV and certain malignancies such as invasive cervical cancer, lymphoma and kaposi sarcoma.
6. (Dara) Describe the pathophysiology of parotid enlargement in HIV patients? What is the role of parotidectomy in this population?

HIV+ patients have a higher incidence of both benign and malignant lymphoproliferative disorders of the parotid. The majority of parotid gland enlargement in HIV+ patients is the result of benign cystic lymphoproliferative process known as "benign lymphoepithelial" (BLC). This type of lesion is associated with ductal metaplasia of the parotid and typically occurs in a setting of relative immune competence. The mechanism proposed is that proliferation of CD8 lymphocytes causes obstruction of lymphatic channels followed by squamous metaplasia of ducts and cyst formation. These lesions are almost always associated with progressive generalized lymphadenopathy. BLC produces persistent, non-tender parotid enlargement.

For minimally symptomatic patients without significant cosmetic deformity, observation alone is the best option. Low dose radiation results in a reduction in size of more than 50% (though improvement generally lasts less than 10 months). Some patients can be treated with repeated needle aspirations, although the need for repeated treatment is suboptimal. Needle aspiration combined with doxycycline solution has been used, injecting 1-2 cc though an angiocath after aspiration. Most patients are left with fibrotic masses after treatment and long term results of this treatment are not well-known. HIV-associated BLC typically does not require parotidectomy. Parotidectomy may be considered in cases if BLC that undergo rapid size change, are disfiguring, or have significant pressure symptoms. Of note, early in the course of HIV medicine, parotidectomy was frequently used for these lesions, but it was seen that very rarely is malignancy associated with these lesions. Approximately 1% can have malignancy associated. It is important to notice that if the mass in an HIV parotid lesion is a solid mass, the incidence of malignancy jumps to 40%.

7. (Dara) What Head and Neck malignancies are HIV/AIDS patients susceptible to and briefly describe modalities of treatment for each?

Non-Hodgkin's Lymphoma- AIDS-related lymphoma occurs in 3-10% of HIV-positive patients. The risk of developing NHL steadily rises with duration of infection and the associated immune suppression. The majority of patients who develop NHL have CD4+ counts less than 200, though higher CD4+ counts do not rule out the diagnosis (Burkitt's lymphoma, in particular, has been noted to occur in patients with relatively high CD4+ counts). HAART has a relatively small impact on NHL incidence, and incidence of NHL remains more than 50% of what it was before the introduction of HAART. The prognosis of NHL in HIV-positive patients is significantly worse than that of the general population and tends to be more rapidly progressing. 95% of HIV+ NHL are of B-cell origin, with high-grade (60%) and medium-grade (33%) making up the majority. HIV-related NHL is treated with multiagent chemotherapy. The therapy must balance the need to treat the neoplasm with the risk of further immune suppression. Concurrent use of HAART with chemo tends to increase survival; this may be related to a decreased incidence of opportunistic infections. Radiotherapy has a role in patients with localized disease or symptomatic lesions. Patients with NHL WITHOUT HIV are treated with high-dose chemo as well as stem cell support. This approach is not used in HIV+ patients for fear of increasing opportunistic infection and toxicity.

Kaposi's Sarcoma- KS is a mesenchymal tumor involving blood and lymphatic vessels. There is general agreement that infection with HHV-8 virus plays causative role in the development of KS. Overall risk of KS in patients with AIDS is estimated to be 20,000 times that of the general population, tough Incidence of KS has dropped more than 2/3 since the introduction of HAART. in HIV + patients, the disease is more aggressive and less responsive to therapy. KS is associated with a shortened life expectancy, though most patients die from opportunistic infection of lymphoma, not KS per se. Specific indications for treatment of KS include cosmetically disfiguring lesions, symptomatic oral or visceral lesions, or pain or edema associated with lymphadenopathy or extensive cutaneous disease. Local therapies include alitretinoin topical gel, local radiation, intralesional chemo injection, cryotherapy, laser treatment, and surgical excision. Systemic chemotherapy is reserved for visceral disease and use of systemic therapy in HIV+ patients carries a high risk of opportunistic infection. Other therapies are targeted at HHV infection and the cytokines and growth factors associated with KS proliferation and include antivirals, thalidomide, IC-862 and retinoids. INF-alpha has also been used, but doses are limited by side effects.

Hodgkin's Lymphoma- Risk of HL is approximately 10-fold higher in HIV+ patients and is associated with a more aggressive clinical course with non-contiguous spread and liver and bone marrow involvement. The histology of HIV-associated HL is different from that of the general population. HIV+ patients tend to develop the aggressive mixed cellularity and lymphodepleted subtypes. 80-100% association with EBV has been reported for HIV+ patients with HL. Overall survival and response to treatment are much lower for HL in HIV+ patients. Treatment consists of chemotherapy and antiretroviral therapy. Concurrent treatment with HAART improves response to chemotherapy, increases disease-free survival interval and overall survival.

Cutaneous Neoplasms- both basal cell carcinoma and melanoma may prove to be more common in the HIV+ population. Both tumors occur more frequently in the setting of immunocompromise. Infiltrative BCC is significantly more common in HIV+ patients. Because of the locally aggressive nature of BCC in this population, Mohs surgery is the treatment of choice. Melanoma also has more aggressive features in the setting of HIV infection. Depth of invasion has an inverse relationship to CD4 count.

Squamous Cell Carcinoma- tends to have a more virulent course in HIV-infected patients (patients present at an earlier age and with more advanced disease), though there is no clear increase in the prevalence.

8. (Kathy) Discuss the bugs that are important in sinusitis in HIV/AIDS patients.

Nearly 70% of these pts develop acute sinusitis, and 58% develop either recurrent acute or chronic sinusitis. Higher incidence is due to impaired systemic and local immunity, decreased mucociliary clearance, and increased atopy. The most commonly cultured
Nov 15: Head & Neck Manifestations of AIDS (updated 09/06)

organisms are Strep pneumoniae, Strep viridians, coagulase-negative Staph, S. aureus, and H. Influenzae. These causes of acute sinusitis most likely resolve with standard medical management for pts with CD4+ counts (>200/uL). Unusual organisms cultured from HIV+ pts include Cryptococcus neoformans, Legionella pneumophila, Acanthamoeba, Mycobacterium kansasii, and CMV. CMV may act as a primary cause of erosive sinusitis or as a cofactor in the pathogenesis of bacterial or fungal sinusitis via local effects on mucosa and systemic effect of neutrophil dysfunction. CMV sinusitis has been reported as the presenting symptoms for HIV infection. Pseudomonas aeruginosa sinusitis occur in HIV+ pts with CD4+ counts (<50/uL) and may be associated with Pseudomonas bacteremia and orbital complication in up to 50% of infections. Fungi such as Aspergillus and mucormycosis are particularly important organisms as they can be particularly invasive in face of depressed humoral immunity and neutrophil dysfunction.

9. (Kathy) What is the differential diagnosis of SNHL in HIV patients?

**Peripheral** auditory pathology caused by drug-induced ototoxicity, otosyphilis, and by idiopathic processes may produce SNHL at any stage of HIV infection. Progressive central auditory dysfunction is a common cause of SNHL in HIV pts is caused by the direct effects of HIV and the secondary effects of immunosuppression, which results in early demyelination of the central auditory tract. Multiple cranial nerve abnormalities, including deafness, may also result from central demyelination associated with PML (progressive multifocal leukoencephalopathy of the brainstem). Opportunistic diseases including CNS toxoplasmosis, TB meningitis, cryptococcal meningitis, and NHL may also cause SNHL in a patient with advanced HIV-AIDS.

10. (Josh) Describe common oral lesions in HIV patients?

11. (Josh) What is the most common oral lesion in AIDS patients? Discuss the treatment.

<table>
<thead>
<tr>
<th>Lesion</th>
<th>Description</th>
<th>Diagnosis</th>
<th>Treatment</th>
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</thead>
<tbody>
<tr>
<td><strong>Malignancies</strong></td>
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<tr>
<td>NHL</td>
<td>Persistent sore, enlarging mass or loose teeth affecting the gingiva and palate</td>
<td>biopsy</td>
<td>Multiagent chemotherapy</td>
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<tr>
<td>Kaposi’s Sarcoma</td>
<td>Multicentric purple or red macular lesions of oral mucosa, usually hard palate and gingiva, can coalesce and progress to violaceous nodular lesions, can have pain, ulceration bleeding, assoc with lowe CD4 than cutaneous dz</td>
<td>Biopsy</td>
<td>Treatment is palliative, no cure exists, alitritinoin gel, local XRT, intralesional chemo, cryotherapy, laser therapy, excision</td>
</tr>
<tr>
<td>SCCa</td>
<td>Oral cavity, oropharynx</td>
<td>biopsy</td>
<td>Depends on staging</td>
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<tr>
<td><strong>Infectious</strong></td>
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<tr>
<td>*Oral Candidiasis</td>
<td>Most common oral manifestation of HIV, occurs in 70-90% of symptomatic HIV pts, occurs when CD4&lt;150, four forms 1. pseudomembranous: smooth white or cottage cheese like plaques on any mucosal surface, erythematous bleeding base when wiped off 2. Atrophic: zones of hyperemia and tenderness on dorsum of tongue or hard palate 3. hyperplastic: involves buccal mucosa with raised white plaques that cannot be scraped off 4. angular chelitis: tender erythematous fissures and ulcers at the oral commisure</td>
<td>Resolution with empiric anticanidal therapy, biopsy with KOH, Gm stain or PAS of scraping</td>
<td>Nystatin 200,000 to 400,000 units 5x/day Clotrimazole 10mg 5x/day Ketoconozole 200mg/d Fluconozole 50mg/d Use combination when severely immunocompromised</td>
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<tr>
<td>Oral Hairy leukoplakia</td>
<td>White lesion with corrugated abd shaggy surface. Affects 17-25% of AIDS pts, local opportunistic infection with EBV, CD4 &lt; 150, prognosticates disease progression</td>
<td>Biopsy if lesion persists after short course of antifungal therapy DDx: leukoplakia, CIS, hypertrophic candidiasis, lichen planus.</td>
<td>Further management rarely needed after dx, typically no malignant transformation, poss benefit of acyclovir, AZT, sulfa drugs</td>
</tr>
<tr>
<td>Herpes simplex stomatitis</td>
<td>Overall incidence 5%, 9-29% in late stage HIV, CD4&lt;100, labial lesions most common, intraoral lesions usually affect keratinized mucosa, esp hard palate and gingiva. Small round ulcers without erythematous halo, produce discomfort with mastication and swallowing</td>
<td>Viral culture from lesion, HSV cells in scraping from tongue base using Tzanck smear</td>
<td>Topical acyclovir 5x/d Combined with systemic for severely immunocompromised, 200mg 5x/gay</td>
</tr>
</tbody>
</table>
### Gingivitis and periodontitis

Gingivitis presents as a red line at free gingival margin, gums bleed with minimal trauma; periodontitis results from exterior to peridontium and resorption of alveolar bone; acute necrotizing ulcerative gingivitis (ANUG) if above untreated for 4 weeks, necrosis of gingiva and bone, with deep jaw pain, bleeding, halitosis, loose teeth

<table>
<thead>
<tr>
<th>History and microbiology</th>
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<tbody>
<tr>
<td>DDx: lymphoma, SCC, KS, bacillary angiomatosis, fungal or mycobacterial infxn</td>
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### Apthous ulcers

Incidence 14% with HIV, CD4 <200

Three types:
1. Herpetiform: smaller than 0.2mm in diameter, self limited
2. Minor: well circumscribed painful ulcers < 6mm in diameter with erythematous halo, may coalesce to form larger lesions lasting about 2 weeks
3. Major (Sutton’s dz): larger than 6mm, painful, persist for weeks, threaten nutritional intake

<table>
<thead>
<tr>
<th>Rule out malignancy (lymphoma or SCCa), biopsy edge of ulcer for path</th>
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### Treatment

- Plaque removal, peridex rinses, debridement of necrotic tissue, systemic anti-anerobic therapy
- Symtomatic relief, monitor nutritional status
- Topical Triamcinolone or flucinonide 6x/d
- Tetracycline mouth wash, peridex mouth wash
- Prudent use if systemic corticosteroids

### Additional Information

12. Is allergic rhinitis more common in HIV patients?

Yes. According to Dr. Stewart's article (Porter, Am J Rhinol., 1999), 80% of HIV patients self-reported symptoms of allergic rhinitis. Also, increased atopy has been noted in up to 87% of HIV-positive patients. This may be due to polyclonal B-cell activation and increased Ig production (including IgE) in these patients. Severity of symptoms has not been found to be linked to CD4 count.

13. Discuss rhinosinusitis incidence and treatment in HIV patients? Does a low CD4 counts correlate with treatment failure?

Sinus infections develop in 68% of HIV patients

Up to 95% of randomly chose AIDS patients have MRI findings consistent with sinus disease

Management consists of medical therapy – broad-spectrum abx, nasal steroids, antihistamines, decongestants, mucolytics

If disease persists, surgical therapy

This study looked at 3 surgical groups: (1)radical surgery – disease that extended beyond sinuses; (2) minimally invasive ESS – limited to sinuses; (3) standard ESS

- Patients undergoing standard ESS enjoyed a satisfactory success rate of 75%
- According the numbers of this study CD4 count did not seem to correlate with patient improvement rate or treatment failure
- A low CD4 counts (<100) does not serve as a contraindication for definitive surgery

14. Discuss parotid masses in HIV patients. Is FNA a useful diagnostic tool in this populations?

Diagnost Cyotpathol 1999;21:260. DID NOT GIVE ME ANSWER


The article describes perspectives on the molecular pathogenesis of virus-induced Ca in HIV/AIDS.

In brief, most of the cancers in HIV/AIDS have a viral etiology. They include virus such as EBV, HPV, HHV8, HIV, HBV/HCV. The reason is several fold: 1) relatively young age of HIV patients and low incidence of non-viral Ca in younger patients; 2) HIV patients are at higher risk for other viral infections; 3) viruses have evolved to evade immune responses; 4) HIV infection alters immune response and predisposes to viral Ca; 5) HIV allows proviral intergration and cellular disregulation.

-HPV: HPV 16/18: cervical, penile, anal Ca; HPV5/8: epidermal dysplastic lesions, carcinomas; HPV infects basal epithelial cells and persist as an episome in the latently infected cell. Most HPV infected immunocompent patients do not progress to Ca because immune mechanisms are able to clear body. HIV+ patients have higher latent levels of HPV.

-EBV: become oncogenic due to integration/overexpression, genes important for oncogenesis are normally expressed in latent infection. Spread by saliva and replicates in OP epithelium. HIV+ allow further chromosomal changes that advance the malignant phenotype (disregulation of c-myc)

Associated with Burkitt's, T-cell lymphoma, HD, anaplastic NP Ca, leiomyosarcoma

-HHV8=KSHV: express CD54, herpes family; Cause persistent lytic poxvirus infections and hypertrophic cutaneous lesions

-HIV: can cause Ca on own (p24+, proviral DNA+)

-Further molecular biology in article.