Heart to HAART: Update on Cardiovascular Disease and HIV

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HIV and Cardiovascular Disease

- Antiretroviral therapy and CAD
- HIV and cardiovascular risk factors
- Atherosclerosis and HIV
- CABG/PTCA and HIV
- Mechanism of accelerated atherosclerosis in HIV patients
- Dilated cardiomyopathy and HIV
- Pulmonary hypertension and HIV
Take home messages

- Cardiovascular disease is becoming more increasingly common in patients with HIV disease
- Contributions from PI/HAART--only time will tell
- In the mean time, aggressively treat all risk factors
- Primary prevention for all patients
- Early referral for cardiovascular evaluation
Cases from HIV Cardiology Clinic at SFGH

- Subspeciality clinic focusing on cardiovascular disease and HIV infection
Patient Case 1 – Primary Prevention

- 44 y.o. male with HIV
  - CD4 280 and VL 50,000
  - Treated with Kaletra/ABC/TFV

- Cardiac Risk Factors
  - Blood pressure of 140/90
  - Cigarette smoker
  - HDL of 32, TG of 813
  - BMI 30

- Patient referred to Cardiology with dyspnea
Cardiac catheterization
Frequency of MI increased after introduction of PI in 1996 (test for trend, \( p=0.0125 \)).

After adjusting for age, sex, smoking, DM, hyperlipidemia, and HTN, the hazard ratio for PI users compared to non-users was 6.5 (95% CI 0.9-48).

*Holmberg SD et al Lancet 2002.*
VA Study of HIV-Infected Patients


Years of Exp/100 Pt-Yr

No. Events/100 Pt-Yr

Any ART
NRTI
PI
NNRTI

Death any cause

Admission for cvd

Data on Adverse Events of Anti-HIV Drugs (DAD Study)

23,000 pts (126 with MI)

Earlier detection of Atherosclerosis

- Measurement of carotid artery intima-media thickness (IMT) using ultrasound as a gauge for atherosclerosis.

- IMT measurements correlate with atherosclerotic risk, even after adjustment for risk factors.

- Carotid artery intima-media thickness (IMT) is an excellent predictor of MI and CVA.

  - O’Leary DH et al. Cardiovascular Health Study. NEJM 1999
All measurements performed by a single trained operator blinded to HIV and treatment status; reproducibility >0.9
HIV Pts have Accelerated Atherosclerotic Progression as assessed by IMT

- 148 HIV infected adults and 63 age and gender matched HIV negative controls studied
- At baseline, HIV patients had greater IMT compared to controls
- HIV infection was an independent predictor of higher IMT
- Traditional CAD risk factors (age, LDL-C, and HTN) and advanced immunodeficiency (nadir CD4 ≤200) were independently associated with IMT in HIV-infected individuals
- Patients with HIV had accelerated progression of IMT compared to non-infected controls

Hsue et al Circulation 2004
HOPS Cohort update

- > 8000 patients followed since 1993
- 84 cardiovascular events
  - Risk of CV events reduced with lipid lowering agents
  - No association with ARV class, duration of ARV, ARV switch, pre-HAART CD4 count, peak VL, peak LDL, peak TG

Lichtenstein K et al CROI Denver 2006
23,437 HIV patients followed for 4.5 yrs

After adjusting for CV risk factors (but not lipids), pts on PI had 16% increase in risk of MI per year vs. 5% per year in pts on NNRTI

Adjusting for lipids and other CV risk factors reduces RR for PI users to 10% and NNRTI users to 0%.

PIs and increased risk
  - Increased risk partly but not entirely due to dyslipidemia

Decreased risk from NNRTI?
HIV and MI:

- Database of 1.7 million pts tx’d at MGH and BWH since 1993
- Risk of MI almost doubled in all those with HIV (RR 1.89, P<0.0001), even after adjustment for traditional risk factors
- HIV-infected women had higher rates of MI than HIV-infected men (RR 2.98 compared to 1.4 for men)
- Cigarette smoking data available in 22% of HIV patients only

_Triant V et al J Clin Endo Metab 2007_
## ART Contribution to CVD Risk

<table>
<thead>
<tr>
<th>Study</th>
<th>N</th>
<th>Events</th>
<th>Outcomes</th>
</tr>
</thead>
<tbody>
<tr>
<td>VA(^1)</td>
<td>36,766</td>
<td>1,207 CHD</td>
<td>No increase risk of MI with HAART or PI</td>
</tr>
<tr>
<td>DAD(^2)</td>
<td>23,468</td>
<td>126 MI</td>
<td>Greater risk with ART and PI exposure</td>
</tr>
<tr>
<td>Kaiser(^3)</td>
<td>5430</td>
<td>86 MI</td>
<td>Greater risk of MI HIV+ vs HIV– No greater risk on PIs</td>
</tr>
<tr>
<td>Medi-Cal(^4)</td>
<td>28513</td>
<td>1360 CHD</td>
<td>Greater risk of CHD with ART in 18–33 year olds, but not older individuals</td>
</tr>
<tr>
<td>French(^5)</td>
<td>34,976</td>
<td>49 MI</td>
<td>Greater risk of MI on PI vs HIV–</td>
</tr>
<tr>
<td>Johns Hopkins(^6)</td>
<td>2671</td>
<td>43 CHD</td>
<td>Greater risk of CHD HIV+ vs HIV–</td>
</tr>
<tr>
<td>Frankfurt(^7)</td>
<td>4993</td>
<td>29 MI</td>
<td>Greater risk of MI after HAART introduction</td>
</tr>
<tr>
<td>HOPS(^8)</td>
<td>5672</td>
<td>21 MI</td>
<td>No increased risk of MI with specific antiretrovirals</td>
</tr>
<tr>
<td>SMART(^9)</td>
<td>5472</td>
<td>63 NF CV events</td>
<td>Increased risk of CV deaths with HAART discontinuation vs continuous treatment</td>
</tr>
</tbody>
</table>

Summary

- Taken together, these studies suggest an increased risk of coronary artery disease in HIV infected patients
  - Mechanism remains to be defined
  - HIV medication appears to increase risk in a duration-related manner
Atherosclerosis: how does HIV fit in?

- Multifactorial process
- Chronic disease state, with long term followup needed

HIV infection

HIV medication

Endothelial dysfunction

Chronic disease state, with long term followup needed

Altered immune response

Inflammation

Traditional risk factors
<table>
<thead>
<tr>
<th>Author</th>
<th>Year</th>
<th>Country</th>
<th>No. Pts</th>
<th>Framingham Risk</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hadigan C et al</td>
<td>2003</td>
<td>U.S.</td>
<td>91 +, fat red 30+, no fat 273 -</td>
<td>7.4% vs 5.3%, p=0.002 29% vs 13% HIV pts with risk ≥10%, p=0.001</td>
</tr>
<tr>
<td>Bergersen BM et al</td>
<td>2004</td>
<td>Oslo</td>
<td>219 +,ART 64 +,ART naïve 438 -</td>
<td>10year risk &gt;20% 11.9% ART pts 6.3% ART naive 5.3% controls</td>
</tr>
<tr>
<td>Smith CJ</td>
<td>2004</td>
<td>UK</td>
<td>394 +</td>
<td>High rate of non modifiable and modifiable risk factors</td>
</tr>
</tbody>
</table>
## Risk Factors in HIV Pts SFGH

<table>
<thead>
<tr>
<th></th>
<th>HIV+CAD N=68</th>
<th>CAD alone N=68</th>
<th>HIV alone N=148</th>
<th>HIV neg N=63</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Age yrs</strong></td>
<td>50±8</td>
<td>61±11</td>
<td>45±8</td>
<td>43±9</td>
</tr>
<tr>
<td><strong>Smoking</strong></td>
<td>68%</td>
<td>41%</td>
<td>55%</td>
<td>25%</td>
</tr>
<tr>
<td><strong>HTN</strong></td>
<td>53%</td>
<td>60%</td>
<td>23%</td>
<td>8%</td>
</tr>
<tr>
<td><strong>DM</strong></td>
<td>13%</td>
<td>41%</td>
<td>6%</td>
<td>2%</td>
</tr>
<tr>
<td><strong>LDL</strong></td>
<td>121±54</td>
<td>117±40</td>
<td>103±39</td>
<td>116±46</td>
</tr>
<tr>
<td><strong>HDL</strong></td>
<td>35±12</td>
<td>41±9</td>
<td>40±15</td>
<td>51±15</td>
</tr>
<tr>
<td><strong>TG</strong></td>
<td>262±278</td>
<td>218±185</td>
<td>228±186</td>
<td>118±82</td>
</tr>
<tr>
<td><strong>Hs-CRP</strong></td>
<td>1.6</td>
<td>0.8</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Lipid Levels and HIV

Riddler SA et al JAMA 2003
Protease inhibitors have been associated with marked increases in cholesterol and triglycerides.

Summary of studies that measured lipid changes in pts during treatment with protease inhibitors (RTV, IDV, NFV):

- Increase TC + 66%
- Increase TG + 80%
- Increase LDL + 37%

Management of PI-Associated Hyperlipidemia

Adult AIDS Clinical Trials Group – refers to updated NCEP/ATPIII guidelines

- All current PI inhibit the CYT P450.
- Statins + PI can result in rhabdomyolysis
- May not be effective in controlling high triglycerides, consider fibrates
- Difficult to reach goal
- Atazanavir
- Ezetimibe
Algorithm for Managing Elevated LDL-C in HIV Patients

Pt on PI

Avoid lovastatin/simvastatin
Atorvastatin at lower dose
(start at 10mg and titrate up
not to exceed 40 mg)
Pravastatin
Ezetimibe

Pt not on PI

Statin therapy per NCEP guidelines

If lipids remain high, consider changing PI to Atazanavir

High Triglycerides

LDL elevated
- Start statin
  - Triglycerides still ↑

LDL normal
- Start fibrate*
  - or niacin
  - or fish oil

Low HDL-C
- Exercise
- Smoking cessation
- Weight control
- Moderate alcohol
- Dietary intake
- Treatment with niacin, fibrates, or statins

*Caution is advised with combination of fibrate and statin
Fenofibrate preferred in combination with statin
If on RTV or NFV, efficacy of fibrate is reduced
Primary Prevention Issues

- Ecsa therapy
- Smoking cessation
- HTN control
- Lipid control
  - can be difficult even with statin therapy
  - Interaction of statins with ART
- How to address low HDL
- Role of alternative lipid lowering agents
- Exercise
- Weight loss
HIV and Atherosclerotic disease - Unknowns

- How to best predict cardiovascular risk in patients with HIV
  - Will Framingham be applicable
  - What is the role of HS-CRP
  - Utility of carotid IMT and coronary calcification
Case 2: HIV and PTCA

- 50 year old gentleman with HIV since 1998
- Never on HIV medication
- Only cardiac risk factor is family history
Cardiac Catheterization
Results
Patient represented to SFGH 4 months later with chest pain
Referred for cardiac catheterization
Clinical Features of HIV-infected Patients with Acute Coronary Syndromes

68 HIV infected patients admitted to San Francisco General Hospital with myocardial infarction or unstable angina from 1993-2003.

Clinical features and outcomes compared to uninfected patients

### PTCA Patients N=50

<table>
<thead>
<tr>
<th></th>
<th>HIV</th>
<th>HIV-Neg</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Refer for cath</td>
<td>56/68</td>
<td>61/68</td>
<td>0.216</td>
</tr>
<tr>
<td></td>
<td>(82%)</td>
<td>(90%)</td>
<td></td>
</tr>
<tr>
<td>Refer for PTCA</td>
<td>29/56</td>
<td>21/61</td>
<td>0.058</td>
</tr>
<tr>
<td></td>
<td>(52%)</td>
<td>(33%)</td>
<td></td>
</tr>
<tr>
<td>Restenosis</td>
<td>15/29</td>
<td>3/21</td>
<td>0.006</td>
</tr>
<tr>
<td></td>
<td>(52%)</td>
<td>(19%)</td>
<td></td>
</tr>
<tr>
<td>Receiving Stents</td>
<td>11/15</td>
<td>2/3</td>
<td>0.814</td>
</tr>
<tr>
<td></td>
<td>(73%)</td>
<td>(67%)</td>
<td></td>
</tr>
</tbody>
</table>
Higher Restenosis in HIV Patients

- HIV patients with acute coronary syndromes are more than a decade younger than HIV-negative patients, more likely to be male (50 vs 61) cigarette smokers and have a low HDL cholesterol.

- HIV patients are more likely to have single vessel disease and present with lower TIMI risk scores on admission.

- HIV patients referred for PTCA have significantly higher restenosis rates as compared to HIV-negative patients.

*Hsue et al Circulation 2004.*
Atherosclerotic Lesions: Features

Occlusive Thrombus

Plaque Rupture

Activation of macrophages, T cells, and mast cells

Hansson GK NEJM 2005
CMV Associated with Transplant Atherosclerosis

- CMV infection in cardiac transplant recipients was associated with more frequent rejection, graft atherosclerosis and death\(^1\)
- Ganciclovir treatment decreased development of CAD in transplant patients\(^2,3\)

\(^1\) Grattan MT et al JAMA 1989
\(^2\) Valantine HA et al Circulation 1999
\(^3\) Valantine HA et al Transplantation 2001
HIV-infected patients had higher carotid IMT compared to controls after controlling for traditional risk factors.

HIV patients had higher hsCRP, T cell activation, and CMV-associated T cell responses compared to HIV-negative patients.

CMV-specific T cell responses but not hsCRP and T cell activation were independently associated with higher carotid IMT (p=0.001).

MCP-1 Allele and IMT in HIV Pts

- 183 HIV patients
- MCP-1 -2518G more common in pts with atherosclerosis vs. those without (47.5% vs. 18.2%, P<0.001)
- After adjusting for risk factors, MCP-1-2518G allele was associated with atherosclerosis (OR 5.72, 95% CI 1.74 to 18.80, P=0.004)
- A subset of 40 pts had a mean rate of progression of 0.06mm/y more rapidly in pts with the MCP-1 allele (p=0.08)

Alonso-Villaverde C et al Circulation 2004
SMART study and Cardiovascular Disease

- Large randomized study of intermittent ART (DC arm) vs. continuous ART (VS arm).
- DC was discontinued early due to excessive number of events in intermittent arm.
- 48 cv events in DC arm as compared to 31 events in VS arm (HR 1.6, P<0.05).
- Importance of controlling HIV disease for risk of atherosclerosis?

El-Sadr W et al NEJM 2006
Endothelial Function Studies - Background

- Flow mediated vasodilatation of the brachial artery is a method to assess endothelial function
- Endothelial function in coronary and peripheral arteries are closely related \(^1\)
- FMD predicts long-term adverse CV events \(^2,3\)

\(^1\) Takase B Am J Cardiol 1998
\(^2\) Gokce N JACC 2003
\(^3\) Neunteufl T AJC 2000
Endothelial Function Studies and HIV

- Use of PIs associated with endothelial dysfunction

- Tx with pravastatin results in improvement in FMD in HIV patients

- A5152s to evaluate the effect of antiretroviral tx on endothelial function in treatment-naïve HIV pts

  - Pts assigned to:
    - PI sparing regimen
    - NNRTI sparing regimen
    - NRTI sparing regimen

  - All 3 ART regimens improved endothelial function in ART-naive HIV pts as early as 4 weeks after tx

1Stein JH Circulation 2001
2Hurlimann D Heart 2006
3Stein JH AHA 2005
ACTG 5152S Median Change in FMD Over Time

Baseline FMD = 3.6% (1.9 –– 5.5%)  

### Kruskal-Wallis Test

- Week 0: (p=0.82)
- Week 4: (p=0.61)
- Week 24: (p=0.78)

** = p≤0.01 compared to baseline, within group  
# = 0.01<p≤0.05 compared to baseline, within group
Case 3A: HIV and Acute MI

- 37 year old Latino gentleman, HIV positive since 1996, with CD4 nadir of 4, DM, hyperlipidemia, present with chest pain
Pt treated with TNK, however had continued chest pain
Referred for PTCA
PTCA unsuccessful, refer for CABG
Patient Case 3B:

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Value</th>
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<tbody>
<tr>
<td>Rate</td>
<td>67</td>
</tr>
<tr>
<td>PR</td>
<td>142</td>
</tr>
<tr>
<td>ORSD</td>
<td>71</td>
</tr>
<tr>
<td>QT</td>
<td>431</td>
</tr>
<tr>
<td>Qtc</td>
<td>455</td>
</tr>
<tr>
<td>P</td>
<td>45</td>
</tr>
<tr>
<td>QRS</td>
<td>45</td>
</tr>
<tr>
<td>T</td>
<td>69</td>
</tr>
</tbody>
</table>

- **Normal sinus rhythm, rate 67**
- **Borderline low voltage in frontal leads**: 6 frontal leads < .6 mV
- **Nonspecific Anterior T wave abnormalities**: T wave -.25 mV V2-V4
- **Cannot exclude ischemia**: T > -.20 mV
- **Widespread ST elevations**, consider Pericarditis: ST > .15 mV ANT/LAT/INF

**Preliminary-MD Must Review**
Cardiac catheterization
Cardiac Catheterization
Cardiac catheterization
Cardiac catheterization
Thrombotic complications in pts with HIV

- Prospective study of 100 medical clinics in U.S.\(^1\)
  - 42,935 HIV patients observed for 2.4 years
  - Incidence of thrombosis was 2.6/1000 PY
  - Associated with age, CMV, OI, hospitalization, megestrol, IDV

- Johns Hopkins HIV Clinical Cohort - identified 160 cases, incidence of 5.1/100 person yrs fu.\(^2\)
  - VTE associated with lower CD4 count, higher HIV VL, prior hospitalization, and elevated WBC.

\(^1\)Sullivan PS et al AIDS 2000
\(^2\)Ahonkhai A, Retrovirus 2007
Acute MI in HIV Patients

- 24 HIV pts with AMI, age 47 years

- 21/24 had cardiac cath
  - 16 (76%) had multivessel disease
  - 17 had PCI
  - 3 had cabg

- 14 pts with STEMI
  - 2 had thrombus, mild stenosis, no PCI
  - 12 had PCI

- 15 month followup
  - 20% had reinfarction
  - 45% had rehospitalization for recurrent coronary event
  - 30% pts had acute atherothrombotic events

Matetzky S et al Arch Intern Med 2003
D-dimers in the SMART study

- Virologic suppression vs. drug conservation
- Studied biomarkers using a nested case control design
- D-dimer levels increased in pts who stopped treatment compared to those who continued ART
- Higher baseline D-dimer levels were associated with 12.4 OR of death (highest quartile vs. lowest)
- Major CVD associated with adjusted OR of 1.12

Neaton J et al 4th IAS Sydney, Australia 2007
Cardiac surgery in Patients with HIV

- Retrospective review of 37 patients from 1994-2000\(^1\)
- Median age of 41 years, 34/37 male
- In the CABG group, 81 percent were free from recurrent angina, CHF, death, and repeat revascularization after 3 years.
- No opportunistic infections developed
- Long term followup?
- Heart transplantation\(^2\)

\(^1\) Trachiotis GD Ann Thor Surg 2003
\(^2\) Calabrese LH et al NEJM 2003.
hsCRP was an independent predictor of mortality in women with HIV infection\(^1\)

MAC Study – CRP associated with HIV disease progression independent of CD4 and HIV RNA levels\(^2\)

What is the relationship between CRP and cardiovascular risk in patients with HIV?

\(^1\) Feldman et al. JAIDS 2003
\(^2\) Lau et al. Arch Int Med 2006
Case 4:

- 42 year old gentleman with HIV diagnosed 6 months previously.
- CD4 count of 1149 cells/mm$^3$, VL of 28,607
- Not on HAART
- No alcohol or drug use
- Presents with SOB for 6 months
Echocardiogram
Echocardiogram
Incidence of Dilated Cardiomyopathy in HIV-Positive Patients

- 952 patients
- Echo dx of DCM made in 76 patients (8%): mean annual incidence of 15.9 per 1000 during 5 year followup
- Higher incidence in patients with CD4≤400 and in pts receiving AZT
- Patients with echo dx had endomyocardial biopsy
  - 63 (83%) myocarditis on histology
  - 36 (57%) pts with myocarditis had positive hybridization sequences for HIV nucleic acid sequences

Barbaro G et al. NEJM 1999
Cardiomyopathy and NRTI

- CM associated with severe mitochondrial damage in HIV-infected patient on AZT, 3TC, and Indinavir
- Pt admitted to hospital, with EM showing accumulation of myelin in mitochondria
- After medication stopped, patient treated for CHF
- Repeat echocardiogram showed improvement

Frerichs F et al. NEJM 2002.
Treatment of HIV-associated DCM

- Similar to non-ischemic cardiomyopathy
- In pediatric patients, IV immunoglobulin has been used
- Use of immunosuppressive regimens remains controversial
- Median survival to AIDS-related death is 101 days in pts with LV dysfunction vs 472 days in pts with normal heart and similar infection stage
- HIV-HEART: prospective study to define incidence and natural history of heart failure in HIV patients

Patient Case 5

- 44 y.o. male with HIV
  - CD4 345 and VL 83,000
  - Not on HAART

- Cardiac Risk Factors
  - HTN on medication
  - Active IV methamphetamine user

- Patient referred to Cardiology with dyspnea
  - Referred for echocardiography
Echocardiogram
Echocardiogram
### Prior Studies of HIV and Pulmonary HTN

<table>
<thead>
<tr>
<th></th>
<th>Himmelman</th>
<th>Speich</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Date</strong></td>
<td>1982-1988</td>
<td>1990</td>
</tr>
<tr>
<td><strong>#Cases</strong></td>
<td>6</td>
<td>6</td>
</tr>
<tr>
<td><strong>Comparison group</strong></td>
<td>1200 AIDS pts in hospital</td>
<td>1200 HIV pts followed in ID clinic</td>
</tr>
<tr>
<td><strong># Cases IVDU</strong></td>
<td>3/6</td>
<td>5/6</td>
</tr>
<tr>
<td><strong>Reported Prevalence</strong></td>
<td>6/1200 = 0.5%</td>
<td>6/1200 = 0.5%</td>
</tr>
</tbody>
</table>

1. Himmelman et al Am J Cardiol 1989
2. Speich et al Chest 1991
The role of HIV and HHV-8 in PAH

- 196 HIV patients and 52 uninfected controls
- After adjustment for IVDU and other factors, HIV patients had a 7.0 fold greater odds of having PASP> 30mm Hg
- Among all HIV patients – no evidence of association between HHV-8 and elevated PASP
  - Among those without risk factors for pulmonary htn, borderline relationship present

Hsue P et al presented at the Scientific Sessions AHA 2005
Pulm HTN related to HIV

- Retrospective study of 47 patients in the Swiss HIV Cohort with PAH related to HIV. Patients treated with ART had a decrease of 21mm Hg, P<0.005)\(^1\)

- 16 patients with HIV associated PAH treated with Bosentan (oral endothelin receptor antagonist) for 16 weeks. Patients had improved exercise tolerance and hemodynamic parameters\(^2\)
  - 2 pts had asym. LFT ≥ 3 ULN
  - 1/2 pts was Hep C coinfected

\(^1\) Zuber JP et al CIDS 2004
\(^2\) Sitbon O et al AJRCCM 2004
Patient Case 5

- 44 y.o. male with HIV
  - CD4 345 and VL 83,000
  - Not on HAART
- Cardiac Risk Factors
  - HTN on medication
  - Active IV methamphetamine user
- Patient referred to Cardiology with dyspnea
  - Referred for echocardiography
Pt referred for RHC, mean PAP of 60mm Hg was found, similar to echo findings.
No change after administration of vasodilator therapy
Pt started HAART
Pt treated with sildenafil with clinical improvement
Sildenafil for PAH in Patients with HIV

- in uninfected pts has been associated with increased functional class and hemodynamics in patients with symptomatic PAH\(^1\)

- Be aware of NAION\(^2\)
  - “blindness” associated with sildenafil
  - 86 cases from the National registry of drug-induced ocular effects, the FDA, and the WHO

- Sildenafil is metabolized by the CYP450 enzymes (CYP3A4 and CYP2C9) \(^3\)
  - RTV increases AUC for sildenafil by 11-fold
    - Use 25mg/48 hours if pt on RTV
  - SQV increased AUC for sildenafil 3.1 fold
    - Use 25mg start dose if pt on SQV

\(^1\) Galie N et al NEJM 2005
\(^2\) Fraunfelder FW et al Arch Ophthalmol 2006
\(^3\) Muirhead GJ et al Br J Pharmacol 2000
Pt Case 6:

- 50 year old gentleman with HIV, HTN, GERD, and anxiety presents with acid reflux after meal. No CP, SOB, N/V/D.
- EKG no acute changes
- Troponin 1.96 → 11.0
Cardiac Catheterization
Typical Anginal Symptoms in HIV patients?

- 42 year old gentleman with HIV, CD4 of 8 cells/mm$^3$, hospitalized with PCP
- Chest pain on day of discharge
Cardiac Catheterization
HIV and Cardiovascular Disease Update – Conclusions

- Cardiovascular disease is becoming increasingly common in pts with HIV disease
- Pathogenesis of cardiovascular disease in pts with HIV remains unclear
- Aggressively treat all risk factors
- Early referral to cardiology for workup for appropriate patients
Unmet Needs/Future Directions for Research in HIV-Related Cardiovascular Disease

- Mechanism behind increased cv risk in HIV patients
  - Increased inflammation
  - Role of thrombosis
  - Role of viral suppression

- Pts with HIV and CAD/CABG
  - Clinical presentation
  - Optimal treatment
  - Long term outcomes

- Predicting CAD in HIV patients

- HIV and DCM in the ART era
- HIV and PAH in the ART era
Acknowledgements

- Cardiology
  - David Waters
  - Ann Bolger
  - Husam Farah
  - Amanda Schnell

- SFGH Positive Health Program
  - Steven Deeks
  - Peter Hunt

- Department of Epidemiology and Biostatistics
  - Jeffrey Martin

- Gladstone Institute of Virology and Immunology/Core Immunology Laboratory
  - Mike McCune
  - Elizabeth Sinclair

- Centers for Disease Control and Prevention
  - Sheila Dollard

Grant Support: K23, R01, AHA, DDCF, UARP, ARI