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**Literature search results**

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**Search details**
The urological complications of HIV.

**Resources searched**
NHS Evidence – Guidance and Specialist Library, TRIP, Cochrane Library, Medline, Embase

**Database search terms**: HIV infections, renal, bladder, urinary tract, prostate, genitourinary

**Google search string**: hiv infection and urinary tract or bladder or renal or prostate (complications).

**Summary**
General items on HIV in the Evidence based reviews section, HIV and Urology in the published research section.

**Guidelines**

- HIV for non-HIV specialists - diagnosing the undiagnosed. Medical Foundation for Aids and Sexual Health (Medfash) 2008

- Guidelines for kidney transplantation in patients with HIV disease
  BHIV 2005

**Evidence-based reviews**
Antimotility agents for chronic diarrhoea in people with HIV/AIDS
2009.

Deworming helminth co-infected individuals for delaying HIV disease progression
2009.

Population-based interventions for reducing sexually transmitted infections, including HIV infection
2009.

Behavioral interventions to reduce risk for sexual transmission of HIV among men who have sex with men
2008.

Published research
From TRIP:

Brachial amyotrophic diplegia in a patient with human immunodeficiency virus infection: widening the spectrum of motor neuron diseases occurring with the human immunodeficiency virus.

Although amyotrophic lateral sclerosis and progressive spinal muscular atrophy have been recognized to occur in association with human immunodeficiency virus infection, to our knowledge, brachial amyotrophic diplegia, a form of segmental motor neuron disease, has not been previously reported. Brachial amyotrophic diplegia results in severe lower motor neuron weakness and atrophy of the upper extremities in the absence of bulbar or lower extremity involvement, pyramidal features, bowel and bladder incontinence, and sensory loss. We describe a human immunodeficiency virus-seropositive man without severe immunosuppression or prior AIDS-defining illnesses who had brachial amyotrophic diplegia. This disorder may represent one end of a spectrum of motor neuron diseases occurring with this retrovirus infection.

From Medline:

1. Characteristics of patients with HIV and biopsy-proven acute interstitial nephritis.

Author(s): Parkhie SM, Fine DM, Lucas GM, Atta MG

Citation: Clinical Journal of The American Society of Nephrology: CJASN, May 2010, vol./is. 5/5(798-804), 1555-9041;1555-905X (2010 May)

Publication Date: May 2010

Abstract: BACKGROUND AND OBJECTIVES: The objective of this study was to describe the characteristics of patients with HIV infection and biopsy-proven acute interstitial nephritis (AIN). DESIGN, SETTING, PARTICIPANTS, & MEASUREMENTS: Pathology reports were reviewed
for patients who had HIV infection and underwent renal biopsy at Johns Hopkins Hospital from January 1, 1995, through January 1, 2008. Patients who received a diagnosis of AIN without evidence of HIV-associated nephropathy were identified, and their clinical course was reviewed up to 18 months after biopsy. RESULTS: Of 262 biopsies, 29 (11%) patients who had AIN without evidence of HIV-associated nephropathy were identified. The mean age at the time of biopsy was 47.5 years (range 28 to 71 years), 17 (59%) were men, and 23 (79%) were black. The majority (62%) of patients were on antiretroviral therapy, 59% were current or former intravenous drug users, and 62% had hepatitis C co-infection. Drugs were identified as the cause of AIN in the majority (72%) of cases. Nonsteroidal anti-inflammatory drugs were most commonly implicated, followed by sulfamethoxazole/trimethoprim. Antiretroviral therapy was identified as the cause in only three cases. None of the patients presented with the classic triad of fever, rash, and pyuria, and only seven (24%) patients presented with <1 g/d proteinuria. CONCLUSIONS: In our series, AIN was prevalent (11%) and was often drug induced. AIN should not be excluded from the differential diagnosis on the basis of absence of the classic clinical triad of fever, rash, and pyuria.

Source: MEDLINE

2. Long-term evolution and determinants of renal function in HIV-infected patients who began receiving combination antiretroviral therapy in 1997-1999, ANRS CO8 APROCO-COPIOLTE.

Author(s): Leport C, Bouteloup V, Rossert J, Garre M, Iordache L, Dellamonica P, Herson S, Raffi F, Chene G

Citation: Clinical Infectious Diseases, December 2009, vol./is. 49/12(1950-4), 1058-4838;1537-6591 (2009 Dec 15)

Publication Date: December 2009

Abstract: Among 1121 patients (90% Caucasian) infected by the human immunodeficiency virus (HIV), the glomerular filtration rate increased (+0.72 mL/min/1.73 m(2)/month) from treatment initiation to month 16 (the rate increase was lower among men and those with low body mass index, AIDS, or receipt of indinavir), then remained stable up to 7 years. Kidney function should be monitored in patients previously exposed to indinavir.

Source: MEDLINE


Author(s): anonymous

Citation: Treatment Update, February 2009, vol./is. 21/2(5-6), 1181-7186;1181-7186 (2009 Feb)

Publication Date: February 2009

Source: MEDLINE

Author(s): Barraclough K, Er L, Ng F, Harris M, Montaner J, Levin A

Citation: Nephron, 2009, vol./is. 111/1(c39-48), 1660-2110;1660-2110 (2009)

Publication Date: 2009

Abstract: BACKGROUND: Glomerular filtration rate (GFR) estimation equations have never been validated in the HIV population. This pilot study aimed to compare all currently available methods of kidney function assessment with nuclear GFR in HIV-infected adults. METHODS: Patients underwent GFR measurement with (99m)Tc-diethylenetriaminepentaacetic acid (Tc-99m Pentetate), and measured values were compared with results of creatinine-based estimation equations [abbreviated 4-variable Modification of Diet in Renal Disease (MDRD) formula and Cockcroft-Gault (CG) formulae], 24-hour urine creatinine clearance and estimated cystatin C GFR. RESULTS: Twenty-seven HIV-infected adults were studied. Most were male and Caucasian, with a mean age of 52 years. Median CD4 was 290 cells/mm(3), 70% of patients had HIV RNA <50 copies/ml and all were receiving highly active antiretroviral therapy (median 5 drugs). Median Tc-99m Pentetate-GFR was 91 ml/min/1.73 m(2). Despite greater bias and similar accuracy, the MDRD formula was more precise than the CG formula, regardless of whether CG estimations were corrected for ideal body weight or body surface area. Relative accuracy within 30% of nuclear GFR was greater for the MDRD formula than for all other methods. The performance of 24-hour urine creatinine clearance was similar to that of the MDRD formula for patients with GFR <90 ml/min/1.73 m(2), although it performed less well at higher GFR. The performance of cystatin C GFR was inferior to that of all the creatinine-based methods. CONCLUSIONS: While no method of kidney function estimation performed highly, both 24-hour urine creatinine clearance and the MDRD formula performed with a level of precision and accuracy sufficient for clinical decision making. Our findings support the preferential use of the MDRD formula in the treated HIV population and suggest that there are no HIV-specific factors that limit equation applicability. Larger validation studies are needed to confirm our findings and allow generalization to the HIV population at large. Copyright 2008 S. Karger AG, Basel.

Source: MEDLINE

5. Antiretroviral nephrotoxicities.

Author(s): Atta MG, Deray G, Lucas GM

Citation: Seminars in Nephrology, November 2008, vol./is. 28/6(563-75), 0270-9295;0270-9295 (2008 Nov)

Publication Date: November 2008

Abstract: With the introduction of combination antiretroviral therapy, there
have been substantial declines in both morbidity and mortality associated with human immunodeficiency virus (HIV)-1 infection. However, data increasingly indicate that HIV-1-infected individuals are faced with accelerated rates of chronic diseases that afflict the general population such as diabetes mellitus, hypertension, and dyslipidemia, as well as cardiovascular, liver, and kidney diseases. Furthermore, this population is exposed to a variety of adverse effects from long-term use of antiretroviral medications, which may cause clinically important renal toxicities. However, it often is challenging to distinguish antiretroviral-related renal toxicity from either direct effects of HIV-1 on the kidney or from a multitude of non-HIV-related kidney diseases. A timely and coordinated effort by the HIV primary provider and a nephrologist is likely to facilitate the evaluation of HIV-1-infected patients with new kidney problems.

Source: MEDLINE

6. Immune complex renal disease and human immunodeficiency virus infection.

Author(s): Cohen SD, Kimmel PL

Citation: Seminars in Nephrology, November 2008, vol./is. 28/6(535-44), 0270-9295;0270-9295 (2008 Nov)

Publication Date: November 2008

Abstract: Immune complex glomerulonephritis is a common diagnosis in renal biopsy series of human immunodeficiency virus (HIV)-infected patients. There are a variety of glomerulonephritides associated with HIV infection, including IgA nephropathy, membranoproliferative glomerulonephritis, membranous nephropathy, lupus-like glomerulonephritis, immunotactoid glomerulopathy, and fibrillary glomerulonephritis. In addition, HIV-related proteins may be implicated in circulating immune complexes directly related to a response to the infection. In some cases, the relationship of the HIV infection to the glomerulonephritis is unclear. HIV infection is associated with the development of polyclonal hypergammaglobulinemia, which can promote the development of circulating immune complexes. It is not clear if HIV-associated glomerulonephritis is caused by the passive trapping of these circulating immune complexes or the in situ deposition of antibodies binding to HIV viral antigens. Some renal lesions that are seen in the setting of HIV infection more likely may be related to the presence of a co-infection such as hepatitis C virus infection. The optimal therapy for immune complex glomerulonephritis in the setting of HIV infection is unknown. Because of the underlying immunosuppressed state of many HIV-infected patients, caution with traditional cytotoxic therapies is advised. The role of antiretroviral therapy in modifying the course of these renal lesions is unclear.

Source: MEDLINE

7. Imaging and histopathologic features of HIV-related renal disease.
Author(s): Symeonidou C, Standish R, Sahdev A, Katz RD, Morlese J, Malhotra A

Citation: Radiographics, September 2008, vol./is. 28/5(1339-54), 0271-5333;1527-1323 (2008 Sep-Oct)

Publication Date: September 2008

Abstract: Despite extraordinary recent advances in the management of human immunodeficiency virus (HIV) infection and acquired immunodeficiency syndrome, patients infected with HIV are still susceptible to a variety of complications that stem either from immunodeficiency or from side effects of antiretroviral regimens. Diagnosis is often challenging, since every organ in the body can be affected by HIV, and the kidneys have been increasingly shown to be involved by a variety of disease processes. Opportunistic infections including those caused by atypical organisms, malignancies such as lymphoma and Kaposi sarcoma, and disease processes specific to HIV infection such as HIV-associated nephropathy have all been shown to affect the kidneys. In this era of highly active antiretroviral therapy (HAART), renal disease arising secondary to antiretroviral medication has been added to the list. Furthermore, the introduction of HAART has increased survival of HIV-infected patients; consequently, the frequency of HIV-associated and incidental renal disease is expected to rise in this population. Because mortality and morbidity rates are affected by the early recognition of renal disease in HIV-infected patients, it is paramount that the radiologist be familiar with the imaging features that can be encountered in such cases. (c) RSNA, 2008.

Source: MEDLINE

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Available in fulltext at The Radiological Society of North America; Note: Username: ulhlibrary Password: library

Available in fulltext at Highwire Press

Available in fulltext at Lincoln County Hospital Professional Library; Note: Username: lincoln/Password: library


Author(s): Crisi GM

Citation: AIDS Reader, August 2008, vol./is. 18/8(405-6), 1053-0894;1053-0894 (2008 Aug)

Publication Date: August 2008

Source: MEDLINE
9. HIV and the kidney.
Author(s): Post FA, Hendry BM
Citation: British Journal of Hospital Medicine, March 2008, vol./is. 69/3(137-40), 1750-8460;1750-8460 (2008 Mar)
Publication Date: March 2008
Abstract: Kidney disease is an important complication of HIV infection. Antiretroviral therapy has dramatically improved the life expectancy of HIV-infected patients with end-stage renal disease. Renal replacement therapy, including kidney transplantation, should be offered to HIV-positive patients.
Source: MEDLINE

Author(s): Fine DM, Perazella MA, Lucas GM, Atta MG
Citation: American Journal of Kidney Diseases, March 2008, vol./is. 51/3(504-14), 0272-6386;1523-6838 (2008 Mar)
Publication Date: March 2008
Source: MEDLINE

11. Anaplastic large cell lymphoma in a human immunodeficiency virus-positive patient with cytologic findings in bladder wash: a case report.
Author(s): Proca DM, De Renne L, Marsh WL Jr, Keyhani-Rofagha S
Citation: Acta Cytologica, January 2008, vol./is. 52/1(83-6), 0001-5547;0001-5547 (2008 Jan-Feb)
Publication Date: January 2008
Abstract: BACKGROUND: Anaplastic large cell lymphoma (ALCL) (Ki-1/CD-30 positive) is an uncommon lymphoproliferative disorder that may be of T cell or null cell type. ALCL has been reported in fine needle aspirations of lymph nodes and pleural or peritoneal fluid cytology. In human immunodeficiency virus (HIV)-positive patients, ALCL appears to be more common and run a more aggressive course. CASE: A 39-year-old black man, seropositive for HIV, presented with acute renal failure secondary to bilateral ureteral obstruction by a pelvic mass involving the urinary bladder. Bladder wash cytology and subsequent biopsy of the mass were diagnostic of ALCL. The ALCL was CD30+ and null cell type, with negative CD2, CD3, CD4, CD5, CD7, CD8, CD20, CD45, CD79a, ALK-1, granzyme B, cytokeratin (AE1/AE3), placental alkaline phosphatase (PLAP) and S-100. The patient expired 9 months after the diagnosis, despite aggressive therapy. CONCLUSION: This is a rare occurrence of ALCL (CD 30 positive, null cell type) in the urinary bladder in an HIV+ patient. Presumptive diagnosis was made by bladder wash cytology and
subsequently confirmed by biopsy. Urinary cytologic examination is a useful diagnostic tool. In HIV+/immunosuppressed patients with urinary symptoms and an obstructive mass, ALCL should be considered in the differential diagnosis.

Source: MEDLINE

regoHuman prostate supports more efficient replication of HIV-1 R5 than X4 strains ex vivo.

Author(s): Le Tortorec A, Satie AP, Denis H, Rioux-Leclercq N, Havard L, Ruffault A, Jegou B, Dejucq-Rainsford N

Citation: Retrovirology, 2008, vol./is. 5/(119), 1742-4690;1742-4690 (2008)

Publication Date: 2008

Abstract: BACKGROUND: In order to determine whether human prostate can be productively infected by HIV-1 strains with different tropism, and thus represent a potential source of HIV in semen, an organotypic culture of prostate from men undergoing prostatic adenomectomy for benign prostate hypertrophy (BPH) was developed. The presence of potential HIV target cells in prostate tissues was investigated using immunohistochemistry. The infection of prostate explants following exposures with HIV-1 R5, R5X4 and X4 strains was analyzed through the measure of RT activity in culture supernatants, the quantification of HIV DNA in the explants and the detection of HIV RNA+ cells in situ. RESULTS: The overall prostate characteristics were retained for 21/2 weeks in culture. Numerous potential HIV-1 target cells were detected in the prostate stroma. Whilst HIV-1 R5SF162 strain consistently productively infected prostatic T lymphocytes and macrophages, the prototypic X4IIIB strain and a primary R5X4 strain showed less efficient replication in this organ. CONCLUSION: The BPH prostate is a site of HIV-1 R5 replication that could contribute virus to semen. A limited spreading of HIV-1 X4 and R5X4 in this organ could participate to the preferential sexual transmission of HIV-1 R5 strains.

Source: MEDLINE

Full Text:

Available in fulltext at BioMedCentral

Available in fulltext at National Library of Medicine

regoCystatin C level as a marker of kidney function in human immunodeficiency virus infection: the FRAM study.

Author(s): Odden MC, Scherzer R, Bacchetti P, Szczek LA, Sidney S, Grunfeld C, Shlipak MG

Citation: Archives of Internal Medicine, November 2007, vol./is. 167/20(2213-9), 0003-9926;0003-9926 (2007 Nov 12)
**Publication Date:** November 2007

**Abstract:** BACKGROUND: Although studies have reported a high prevalence of end-stage renal disease in human immunodeficiency virus (HIV)-infected individuals, little is known about moderate impairments in kidney function. Cystatin C measurement may be more sensitive than creatinine for detecting impaired kidney function in persons with HIV. METHODS: We evaluated kidney function in the Fat Redistribution and Metabolic Change in HIV Infection (FRAM) cohort, a representative sample of 1008 HIV-infected persons and 290 controls from the Coronary Artery Risk Development in Young Adults (CARDIA) study in the United States. RESULTS: Cystatin C level was elevated in HIV-infected individuals; the mean +/- SD cystatin C level was 0.92 +/- 0.22 mg/L in those infected with HIV and 0.76 +/- 0.15 mg/L in controls (P < .001). In contrast, both mean creatinine levels and estimated glomerular filtration rates appeared similar in HIV-infected individuals and controls (0.87 +/- 0.21 vs 0.85 +/- 0.19 mg/dL [to convert to micromoles per liter, multiply by 88.4] [P = .35] and 110 +/- 26 vs 106 +/- 23 mL/min/1.73 m(2) [P = .06], respectively). Persons with HIV infection were more likely to have a cystatin C level greater than 1.0 mg/L (OR, 9.8; 95% confidence interval, 4.4-22.0 [P <.001]), a threshold demonstrated to be associated with increased risk for death and cardiovascular and kidney disease. Among participants with HIV, potentially modifiable risk factors for kidney disease, hypertension, and low high-density lipoprotein concentration were associated with a higher cystatin C level, as were lower CD4 lymphocyte count and coinfection with hepatitis C virus (all P <.001). CONCLUSIONS: Individuals infected with HIV had substantially worse kidney function when measured by cystatin C level compared with HIV-negative controls, whereas mean creatinine levels and estimated glomerular filtration rates were similar. Cystatin C measurement could be a useful clinical tool to identify HIV-infected persons at increased risk for kidney and cardiovascular disease.

**Source:** MEDLINE

**Full Text:**

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Available in selected fulltext at [Highwire Press](http://www.highwire.org)

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14. **Cocaine use and hypertensive renal changes in HIV-infected individuals.**

**Author(s):** Fine DM, Garg N, Haas M, Rahman MH, Lucas GM, Scheel PJ, Atta MG

**Citation:** Clinical Journal of The American Society of Nephrology: CJASN, November 2007, vol./is. 2/6(1125-30), 1555-905X (2007 Nov)

**Publication Date:** November 2007

**Abstract:** BACKGROUND AND OBJECTIVES: Cocaine causes kidney damage, but data linking cocaine use to chronic kidney disease in HIV
patients is not described. This study was conducted to evaluate the possible association of cocaine use and histopathologic findings on biopsy in this population. DESIGN, SETTING, PARTICIPANTS, & MEASUREMENTS: Kidney biopsies that were performed in HIV-infected patients during the course of 11 yr were reviewed. Demographic and clinical data were collected. Hypertensive changes were defined on the basis of the Banff 97 classification. Criteria of both arterial intimal fibrosis and thickening and hyaline arteriolar sclerosis were used and graded as absent (0), mild (1), moderate (2), and severe (3). Hypertensive renal changes were considered present when the combined pathology score was > or = 2. To minimize confounding, those with hypertension or diabetes were excluded.

RESULTS: Of the 193 HIV patients who underwent kidney biopsy, 53 had no history of hypertension or diabetes with HIV infection. Of those, 29 (55%) had hypertensive renal changes on kidney biopsy. Cocaine use was present in 16 (55%) of 29 with hypertensive renal changes compared with six (25%) of 24 without hypertensive renal changes (odds ratio [OR] 3.7; 95% confidence interval [CI] 1.2 to 11.7). In the adjusted analyses, only age (/yr; OR 1.08; 95% CI 1.00 to 1.16) and cocaine use (OR 3.55; 95% CI 1.04 to 12.14) were significantly associated with hypertensive renal changes on renal biopsy. CONCLUSIONS: Cocaine use is associated with hypertensive renal changes in HIV-infected patients in the absence of hypertension and diabetes.

Source: MEDLINE

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Available in fulltext at Highwire Press

15. Genetic susceptibility, HIV infection, and the kidney.

Author(s): Kiryluk K, Martino J, Gharavi AG

Citation: Clinical Journal of The American Society of Nephrology: CJASN, July 2007, vol./is. 2 Suppl 1/(S25-35), 1555-905X (2007 Jul)

Publication Date: July 2007

Abstract: In recent years, the sequencing of mammalian and microbial genomes has provided the opportunity to study how genetic variation in the host and pathogen influence the course of infectious disease. In the case of HIV-1 infection, such studies have led to identification of key viral proteins that determine pathogenicity, immune evasion, or drug resistance. In addition, candidate gene association studies have uncovered a large number of host genetic variants that influence the outcome of infection and some organ-specific complications. HIV-associated nephropathy (HIVAN) is a pathologically distinct complication of HIV infection. Interindividual variability in incidence, skewed ethnic distribution, and familial aggregation of HIVAN with other forms of ESRD have suggested genetic susceptibility as a major contributing factor. This article reviews the host genetic factors that influence the course of HIV-1 infection and discusses murine models that have increased the understanding of HIVAN pathogenesis and demonstrated the role of genetic background on determination of disease.
16. HIV-1 and HIV-Associated Nephropathy 25 Years Later.

Author(s): Wyatt CM, Klotman PE

Citation: Clinical Journal of The American Society of Nephrology: CJASN, July 2007, vol./is. 2 Suppl 1/(S20-4), 1555-905X (2007 Jul)

Publication Date: July 2007

Abstract: Twenty-five years after the first published description of AIDS, HIV-associated nephropathy (HIVAN) remains an important cause of kidney disease in HIV-infected patients. The pathogenesis of HIVAN involves direct HIV infection of the kidney, with both viral and host genetic factors playing an important role. The widespread use of antiretroviral therapy has influenced the epidemiology of HIV-related kidney disease, and the nephrology community should support efforts to improve access to therapy and limit HIV transmission in susceptible minority populations. This article reviews the history of HIV and HIVAN, focusing on advances in the understanding of pathogenesis, epidemiology, and treatment.

Source: MEDLINE

Full Text:
Available in fulltext at Highwire Press

17. Renal safety of tenofovir disoproxil fumarate in HIV-1 treatment-experienced patients with adverse events related to prior NRTI use: data from a prospective, observational, multicenter study.


Citation: Journal of Acquired Immune Deficiency Syndromes: JAIDS, July 2006, vol./is. 42/3(385-7), 1525-4135;1525-4135 (2006 Jul)

Publication Date: July 2006

Source: MEDLINE

18. Nephrotoxicity in a child with perinatal HIV on tenofovir, didanosine and lopinavir/ritonavir.

Author(s): Hussain S, Khayat A, Tolaymat A, Rathore MH

Citation: Pediatric Nephrology, July 2006, vol./is. 21/7(1034-6), 0931-041X;0931-041X (2006 Jul)
Publication Date: July 2006

Abstract: Tenofovir-related tubule damage characterized by Fanconi syndrome, renal insufficiency and nephrogenic diabetes insipidus has been reported in the adult HIV-infected population. To our knowledge there has been no reported case of such complications in the pediatric population. We report the case of a 12-year-old perinatally HIV-infected African-American girl who developed nephrogenic diabetes insipidus, renal insufficiency and Fanconi-like syndrome while taking tenofovir (Viread) in combination with lopinavir-ritonavir (Kaletra) and didanosine (Videx).

Source: MEDLINE


Author(s): Boyd MA, Siangphoe U, Ruxruntham K, Reiss P, Mahanontharit A, Lange JM, Phanuphak P, Cooper DA, Burger DM

Citation: Journal of Antimicrobial Chemotherapy, June 2006, vol./is. 57/6(1161-7), 0305-7453;0305-7453 (2006 Jun)

Publication Date: June 2006

Abstract: OBJECTIVES: Indinavir is associated with nephrotoxicity. Therapeutic drug monitoring of indinavir improves clinical outcome, but there is little data regarding therapeutic drug monitoring for patients with established indinavir-associated renal impairment. We prospectively studied the use of therapeutic drug monitoring in patients with virological success but established nephrotoxicity on an indinavir-containing regimen. METHODS: We measured indinavir C(trough)/C(2h), serum creatinine, pyuria, blood pressure (BP), weight and HIV RNA. The major endpoint of interest was the number of patients achieving a normal creatinine level 20 weeks following final indinavir dose adjustment. Primary analysis was by intention to treat (ITT). RESULTS: A total of 35 patients were enrolled; mean (SD) age 40.3 (5.8) years; mean (SD) BMI 21.5 (2.8) kg/m(2). At baseline 6/35 (17%) had a serum creatinine concentration within normal limits, but were offered enrolment because of previous nephrotoxicity (nephrolithiasis and/or abnormal serum creatinine), and a screening pharmacokinetic profile associated with increased nephrotoxicity risk. By ITT analysis 11/35 (31%) had normal creatinine at study end (P = 0.18). Of the 29 patients with abnormal creatinine at baseline, 7/29 (24.1%) had normal creatinine at study end (P = 0.016). Patients had a median (IQR) indinavir per dose adjustment over the study of 400 (400-800) mg. We observed improvements in estimated creatinine clearance, pyuria, resting BP and indinavir pharmacokinetic profile. HIV RNA control was maintained with continued immune recovery despite lower indinavir doses. CONCLUSIONS: Patients experiencing nephrotoxicity on an indinavir-containing regimen were safely maintained on indinavir by means of therapeutic drug monitoring. Parameters of renal function improved but did not return to baseline values, at least in the short-term.

Source: MEDLINE
20. Lower genitourinary tract sources of seminal HIV.

**Author(s):** Coombs RW, Lockhart D, Ross SO, Deutsch L, Dragavon J, Diem K, Hooton TM, Collier AC, Corey L, Krieger JN

**Citation:** Journal of Acquired Immune Deficiency Syndromes: JAIDS, April 2006, vol./is. 41/4(430-8), 1525-4135 (2006 Apr 1)

**Publication Date:** April 2006

**Abstract:** OBJECTIVE: To investigate genital tract sources of HIV-1, we conducted extensive genitourinary sampling of 23 seropositive men without urethritis who shed HIV in their seminal plasma. DESIGN: Semen was collected, then samples were obtained for HIV RNA in blood plasma, urethral fluid, pre-prostate massage fluid/urine (PMF/U) and post-PMF/U, and expressed prostatic secretions. Systematic transrectal ultrasound-guided prostate biopsies obtained from multiple prostate areas were evaluated for HIV RNA and DNA. RESULTS: Seminal HIV RNA levels correlated with HIV RNA levels in urethral fluid and post-PMF/U and with prostate biopsies HIV DNA, but not with expressed prostatic secretions HIV RNA. However, only the HIV RNA level in post-PMF/U independently predicted that in semen (2.77-fold change in semen for each 10-fold change in post-PMF/U; 95% confidence interval, 1.0-7.7) accounting for one third of the seminal HIV RNA level variation, irrespective of adjustment for antiretroviral therapy. CONCLUSIONS: These data indicate that distal genitourinary sources other than the prostate appear to be the major source of seminal HIV in men without clinical urethritis or prostatitis. Because the HIV RNA level in blood plasma is not reliable as an independent clinical predictor of virus levels in seminal plasma, these findings also extend the concept that the male genital tract is a distinct virological compartment from blood.

**Source:** MEDLINE

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**Citation:** Antiviral Therapy, 2006, vol./is. 11/1(79-86), 1359-6535;1359-6535 (2006)

**Publication Date:** 2006

**Abstract:** BACKGROUND: Tenofovir (TDF) exposure has been associated
with renal dysfunction. Mitochondrial nephrotoxicity was investigated as an underlying mechanism. Given the interaction between TDF and didanosine (ddl), their concurrent use was also investigated. **DESIGN:** Relative kidney biopsy mitochondrial DNA (mtDNA) to nuclear DNA ratios were measured retrospectively. HIV+ individuals on TDF within 6 months preceding the biopsy (HIV+/TDF+, n=21) were compared to HIV+ individuals who never received TDF (HIV+/TDF-, n=10) and to HIV uninfected controls (HIV-, n=22). Twelve of the HIV+/TDF+ individuals received concurrent ddl, 10 of those once at unadjusted ddl dosage. Tubular mitochondria morphology was also examined by electron microscopy. Statistical analyses were done on log-transformed mtDNA/nDNA, using non-parametric tests. **RESULTS:** Kidney mtDNA levels were different among the three groups (P=0.046). mtDNA ratios were lower in HIV+/TDF+ subjects (7.5 [2.0-12.1]) than in HIV- ones (14.3 [6.0-16.5], P=0.014), but not lower than HIV+/TDF-controls (6.4 [2.8-11.9], P=0.82). Among HIV+ subjects, there was a difference between TDF-, TDF+/ddl- and TDF+/ddl+ (P=0.005), with concurrent TDF/ddl use associated with lower mtDNA (2.1 [1.9-5.5], n=12) than TDF+/ddl- (13.8 [7.5-16.4], n=9, P=0.003). No TDF-/ddl+ biopsies were available. In regression analyses, only HIV infection (P=0.03), and TDF/ddl use (P=0.003) were associated with lower mtDNA. At the ultrastructural level, abnormal tubular mitochondria was more prevalent in HIV+/TDF+ biopsies than HIV+/TDF- and HIV- ones together (P<0.001) but not more so in TDF+/ddl+ biopsies than TDF+/ddl- ones (P=0.67). **CONCLUSIONS:** Renal dysfunction in this population may be mediated through mitochondrial nephrotoxicity that involves more than one drug and/or pathogenesis. Kidney mtDNA depletion was associated with HIV infection and concurrent TDF/ddl therapy but not TDF use alone, while kidney ultrastructural mitochondrial abnormalities were seen with TDF use. The interaction between TDF and ddl may be relevant in the kidney where both drugs are cleared. The clinical relevance of our findings needs to be evaluated given the current recommendation for reduced doses of ddl when used in conjunction with TDF.

**Source:** MEDLINE

22. **NF-kappaB regulates Fas-mediated apoptosis in HIV-associated nephropathy.**

**Author(s):** Ross MJ, Martinka S, D'Agati VD, Bruggeman LA

**Citation:** Journal of the American Society of Nephrology, August 2005, vol./is. 16/8(2403-11), 1046-6673;1046-6673 (2005 Aug)

**Publication Date:** August 2005

**Abstract:** Renal parenchymal injury in HIV-associated nephropathy (HIVAN) is characterized by epithelial proliferation, dedifferentiation, and apoptosis along the entire length of the nephron. Although apoptotic cell death in HIVAN has been well documented, the mechanism for HIV-induced apoptosis is poorly understood. Whether the epithelial apoptosis in HIVAN is mediated by NF-kappaB-activated Fas ligand expression was investigated here. In human HIVAN and HIV-1 transgenic mouse kidney specimens, the expression of Fas receptor and ligand proteins were markedly upregulated on epithelium in diseased glomerular and
tubulointerstitial compartments when compared with normal. Podocyte cell lines that were derived from HIV-1 transgenic mice showed a similar upregulation of Fas receptor expression and de novo expression of Fas ligand by semiquantitative reverse transcription-PCR and Western blotting. In cultured podocytes, cross-linking of the Fas receptor to mimic ligand binding induced caspase 8 activity and apoptosis in both normal and HIVAN podocytes. Because constitutive NF-kappaB activity has been demonstrated in HIVAN epithelia, evidence for transcriptional control of the Fas ligand expression by NF-kappaB was sought. With the use of cultured podocytes, expression of a Fas ligand promoter reporter plasmid was higher in HIVAN podocytes, indicating increased transcriptional activity. In addition, chromatin immunoprecipitation assays were performed to demonstrate that p65-containing (RelA) complexes bound the Fas ligand promoter and that suppression of activated NF-kappaB with a peptide inhibitor could reduce the expression of Fas ligand mRNA in HIVAN podocytes. These results suggest that NF-kappaB may regulate Fas-mediated apoptosis in HIVAN by controlling the expression of Fas ligand in renal epithelium.

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23. The prostate as a reservoir for HIV-1.

Author(s): Smith DM, Kingery JD, Wong JK, Ignacio CC, Richman DD, Little SJ

Citation: AIDS, July 2004, vol./is. 18/11(1600-2), 0269-9370;0269-9370 (2004 Jul 23)

Publication Date: July 2004

Abstract: As the prostate can harbor bacterial and fungal pathogens, it was investigated as a reservoir for HIV. Nine men chronically infected with HIV participated in a crossover trial in which weekly semen samples were collected with and without previous prostate massage (PM). Six of the nine participants had undetectable seminal plasma (SP) HIV RNA in all samples collected with and without previous PM, but had detectable SP HIV RNA (> 25 copies/ml) in one to three samples collected after PM.

Source: MEDLINE

24. Human immunodeficiency virus infection and kidney transplantation in the era of highly active antiretroviral therapy and modern immunosuppression.

Author(s): Abbott KC, Swanson SJ, Agodoa LY, Kimmel PL

Citation: Journal of the American Society of Nephrology, June 2004, vol./is. 15/6(1633-9), 1046-6673;1046-6673 (2004 Jun)
**Publication Date:** June 2004

**Abstract:** Before the era of highly active antiretroviral therapy, kidney transplant recipients infected with HIV had increased risk of death compared with HIV-uninfected recipients. More recent single-center reports have indicated improved results, but this has not been assessed in a national population. Therefore, a retrospective cohort study of US adult deceased donor kidney transplant recipients from January 1, 1996, to May 31, 2001 was conducted; patients were followed until October 31, 2001. A total of 27,851 patients had valid recipient HIV serology. Cox regression analysis was used to model adjusted hazard ratios for mortality and graft loss, respectively, adjusted for other factors, including comorbid conditions from Centers for Medicare and Medicaid Studies Form 2728. Factors independently associated with HIV infection were also assessed by logistic regression analysis. Only 12.8% of HIV-infected recipients were black, compared with 27.6% in the entire study cohort. HIV-infected kidney transplant recipients were significantly less likely to be black in logistic regression analysis (adjusted OR, 0.29; 95% CI, 0.08 to 0.99; P = 0.049), which was the only factor independently associated with HIV infection. It was found that HIV-infected recipients had improved survival compared with HIV-uninfected recipients, although this was not statistically significant in adjusted analysis (adjusted HR, 0.36; 95% CI, 0.05 to 2.53; P = 0.31). Kidney transplantation in HIV-infected patients is plausible and ongoing, but HIV-infected candidates who underwent kidney transplantation in the United States during the course of the study were demographically unrepresentative of HIV-infected candidates generally.

**Source:** MEDLINE

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**25. Kidney damage reported in some tenofovir users.**

**Author(s):** anonymous

**Citation:** Treatment Update, February 2003, vol./is. 15/2(6-7), 1181-7186;1181-7186 (2003 Feb-Mar)

**Publication Date:** February 2003

**Source:** MEDLINE

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**26. Suppression of HIV-1 expression by inhibitors of cyclin-dependent kinases promotes differentiation of infected podocytes.**

**Author(s):** Nelson PJ, Gelman IH, Klotman PE

**Citation:** Journal of the American Society of Nephrology, December 2001, vol./is. 12/12(2827-31), 1046-6673;1046-6673 (2001 Dec)

**Publication Date:** December 2001
Abstract: The glomerular lesions of HIV-associated nephropathy (HIVAN) are associated with the expression of HIV-1 in podocytes. Infected podocytes proliferate and lose several differentiation markers in vivo and in vitro, which suggests that HIV-1 gene expression induces these changes. Flavopiridol and roscovitine, newly identified inhibitors of cyclin-dependent kinase-9, markedly decrease HIV-1 promoter activity in cell lines of various lineages. In this study, the inhibitors were used to determine whether suppression of HIV-1 transcription in infected podocytes correlated with an inhibition of proliferation and a return to the differentiated phenotype. Dose-response analysis showed that both flavopiridol and roscovitine reversibly suppressed HIV-1 transcription in podocytes in vitro at an IC(50) of 25 nM and 3 microM, respectively. Despite equivalent suppression of HIV-1 transcription, roscovitine was a more effective inhibitor of podocyte proliferation than flavopiridol. Suppression of HIV-1 transcription by flavopiridol or roscovitine was marked by re-expression of the podocyte differentiation markers, synaptopodin and podocalyxin. These results suggest that inhibition of HIV-1 transcription decreases podocyte proliferation and permits the reexpression of differentiation markers. Thus, suppression of HIV-1 transcription by selective cyclin-dependent kinase-9 inhibitors may be a useful therapeutic strategy for the treatment of HIVAN.

Source: MEDLINE

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27. Nephropathy and establishment of a renal reservoir of HIV type 1 during primary infection.

Author(s): Winston JA, Bruggeman LA, Ross MD, Jacobson J, Ross L, D'Agati VD, Klotman PE, Klotman ME

Citation: New England Journal of Medicine, June 2001, vol./is. 344/26(1979-84), 0028-4793;0028-4793 (2001 Jun 28)

Publication Date: June 2001

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Available in print at Pilgrim Hospital Staff Library

**Author(s):** Kilmarx PH, Mock PA, Levine WC

**Citation:** Sexually Transmitted Diseases, June 2001, vol./is. 28/6(347-8), 0148-5717;0148-5717 (2001 Jun)

**Publication Date:** June 2001

**Source:** MEDLINE

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Available in print at Pilgrim Hospital Staff Library

29. Nephrotic syndrome due to thrombotic microangiopathy (TMA) as the first manifestation of human immunodeficiency virus infection: recovery before antiretroviral therapy without specific treatment against TMA.

**Author(s):** Sacristan Lista F, Saavedra Alonso AJ, Oliver Morales J, Vazquez Martul E

**Citation:** Clinical Nephrology, May 2001, vol./is. 55/5(404-7), 0301-0430;0301-0430 (2001 May)

**Publication Date:** May 2001

**Abstract:** BACKGROUND: Among the possible renal complications that can develop a human immunodeficiency virus- (HIV) infected patient, thrombotic microangiopathy (TMA) is one of them. This is a type of vascular lesion more common in HIV patients than in normal population, and sometimes it can be the first manifestation of the HIV infection. METHODS: We present a patient with TMA in whom the subsequent investigation to find the cause of TMA revealed HIV infection and Giardia Lamblia in stool. RESULTS: Before antiretroviral therapy was started the patient began to show recovery of the hemolytic anemia, recovery of the nephrotic syndrome and partial remission of the proteinuria, so that he did not receive specific therapy for TMA. CONCLUSIONS: HIV infection should be suspected in patients presenting with TMA, and a HIV test should be routinely performed as part of the initial clinical evaluation of TMA. If the patients have not developed acquired immunodeficiency syndrome, the prognosis of TMA is equal to non-infected ones.

**Source:** MEDLINE

30. HIV: much at stake with kidneys?

**Author(s):** Moore JP, Doms RW
31. Renal epithelium is a previously unrecognized site of HIV-1 infection.

Author(s): Bruggeman LA, Ross MD, Tanji N, Cara A, Dikman S, Gordon RE, Burns GC, D'Agati VD, Winston JA, Klotman ME, Klotman PE

Citation: Journal of the American Society of Nephrology, November 2000, vol./is. 11/11(2079-87), 1046-6673;1046-6673 (2000 Nov)

Publication Date: November 2000

Abstract: The striking emergence of an epidemic of HIV-related renal disease in patients with end-stage renal disease provided the rationale for the exploration of whether HIV-1 directly infects renal parenchymal cells. Renal glomerular and tubular epithelial cells contain HIV-1 mRNA and DNA, indicating infection by HIV-1. In addition, circularized viral DNA, a marker of recent nuclear import of full-length, reverse-transcribed RNA, was detected in the biopsies, suggesting active replication in renal tissue. Infiltrating infected leukocytes harbored more viral mRNA than renal epithelium. Identification of this novel reservoir suggests that effectively targeting the kidney with antiretrovirals may be critical for patients who are seropositive with renal disease. Thus, renal epithelium constitutes a unique and previously unrecognized cell target for HIV-1 infection.

Source: MEDLINE

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Author(s): Nzerue C, Drayton J, Oster R, Hewan-Lowe K

Citation: American Journal of the Medical Sciences, November 2000, vol./is. 320/5(299-303), 0002-9629;0002-9629 (2000 Nov)

Publication Date: November 2000

Abstract: BACKGROUND: There has been a resurgence of tuberculosis (TB) in the United States, largely because of the HIV epidemic. The impact of this epidemic on the incidence and clinical presentation of genitourinary TB is largely unknown. We describe the clinical findings and outcomes of
genitourinary TB in patients infected with HIV and compare them with those in patients not infected with HIV. METHODS: We retrospectively studied the case records of 16 patients infected with HIV and genitourinary TB and compared them with those of 8 patients without HIV infection diagnosed with genitourinary TB between January 1, 1991, and December 31, 1997, at a large, urban, inner-city, tertiary hospital. Data abstracted from records include demographics, symptoms, signs, laboratory and radiologic findings, and in-hospital mortality. RESULTS: Of 1282 patients with tuberculosis, 24 patients had positive urine cultures for Mycobacterium tuberculosis. HIV infection was present in 16 patients (75%). Patients infected with HIV were younger (mean age, 39.1 +/- 6.2 versus 53.9 +/- 17.2, P = 0.047) but did not differ significantly in clinical presentation from patients who did not have HIV infection. The combined mortality rate was 16.7%. Advanced age was the strongest predictor of poor outcome (P = 0.03). CONCLUSIONS: HIV infection was present in 66.7% of patients with genitourinary TB seen an inner-city hospital. Increasing age was associated with poor survival. No significant differences in clinical presentation nor in-hospital mortality were observed between those with HIV infection and those without HIV infection.

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