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HIV infection and kidney disease: literature review

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ABSTRACT: Combination antiretroviral-therapy (cART) has improved HIV-positive patients mortality and life expectancy. On the other hand, the prevalence of comorbidities, such as cardiovascular disease, hepatic and renal disease has increased. Kidney disease is one of the main causes of morbidity and mortality, especially if accompanied by older age, diabetes, hypertension, black race and hepatitis C coinfection. Renal function needs to be monitored in order to identify kidney disease during early stages. Even if certain cART regimens have been associated with kidney disease, new antiretroviral drugs with a better toxicity profile are now available. Further studies on these new regimens will bring us information on their role in the management of renal disease among HIV-infected patients.

Key words: ARF, CKD, HAART, HIV, HIVAN, Kidney.

INTRODUCTION

Combination antiretroviral therapy cART cannot eradicate HIV infection, although it has drastically diminished HIV-related mortality and morbidities[1]. Non-AIDS-related diseases, such as malignancies, cardiovascular disease, bone and renal disease, have emerged as the leading cause of morbidity and mortality among HIV-positive patients[2-54].

Kidney disease is the most frequent comorbidity[55] and cause of end-stage renal disease (ESRD). Up to 30% of HIV-infected patients have abnormal renal function and it has been correlated with enhanced progression to AIDS and death[56-59]. Race is an critical risk factor, in fact, black individuals have a 11-fold increased risk for chronic kidney disease (CKD)[60]. Furthermore, the two most common risk factors for CKD in the general population, such as diabetes and hypertension, are over-represented among HIV-positive individuals. A family history of renal disease, age and Hepatitis C virus coinfection are also considered risk factors. Of note, virological and immunological parameters, affect the development of kidney disease, in fact higher baseline HIV plasma viral load (>4000 copies/mL) and lower baseline CD4+ T-cell count (<200 cells/μL) have been correlated with reduced renal function in HIV-positive subjects[56].

We report an update of the literature about risk factors, screening methods, and management of HIV-related renal disease.

Renal Disease in the setting of HIV infection

HIV-associated nephropathy (HIVAN) is a collapsing form of focal glomerulosclerosis with tubulointerstitial damage. HIVAN represents the most common cause of CKD in HIV-infected individuals. In fact, it has been found in up to 60% of renal biopsies of HIV-positive patients with CKD. CKD severity depends on the renal function and estimated glomerular filtration rate (eGFR). The pathogenesis of CKD in HIV-infected patients is multifactorial, and the presence of other risk factors for CKD, such as diabetes and hypertension, is as important as in the general population[61].

Of note, HIVAN is the third leading cause of ESRD in black people aged 20-64[62-64]. Since most patients present with proteinuria and reduced renal function, renal biopsy may be useful to confirm the diagnosis of HIVAN[65].

Kidney disease in patients with HIV infection is not just HIVAN. As a matter of fact, acute renal failure (ARF) is more common in HIV-infected subjects than in HIV-negative ones. ARF is a characterized by a sudden reduction in GFR over days to weeks with an increase in serum creatinine level to values > 1.5 mg/dl (or > 1.3 times the laboratory upper limit of normal), which returns to baseline values within 3 months. Advanced HIV disease and HCV coinfection are risk factors for ARF[66-67].

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Laboratory markers and diagnostic tools to evaluate kidney dysfunction

Elevated urinary protein excretion indicates tubular damage and is evaluated qualitatively with the urine dipstick or quantitatively by spot urine protein/creatinine ratio. Urinary albumin is used to estimate glomerular damage, which is a risk factor for cardiovascular disease[68]. Moreover, microalbuminuria has been reported to correlate with Framingham risk score and to represent an independent predictor for cardiovascular disease in subjects with a Framingham risk score <20%.

Among HIV-infected persons, the presence of proteinuria has been linked to an increased risk of CKD, ESRD, new AIDS-defining illness and mortality. Of importance, HIV infection itself is a strong independent risk factor for the presence of microalbuminuria. It has been demonstrated that there is an association between microalbuminuria and the stage of HIV infection. HIV RNA level, CD4 count, African-American race are also important determinants of overt proteinuria. Decreased CD4 count, high viral load and African-American race have all been associated with higher albumin to creatinine ratio (ACR)[69].

The Cockcroft-Gault and Modification of Diet in Renal Disease (MDRD) equations are creatinine-based estimates of GFR. Both are based on several parameters, such as serum creatinine level, age, sex, race and anthropomeric data[70,71], but they have some limitations as they do not adjust for creatinine tubular reabsorption. A more accurate equation is the Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI), even though the differences found between eGFR calculated using the Cockcroft-Gault formula standardized for BSA and the CKD-EPI formula were modest in the EuroSIDA study[72].

Other biomarkers have been studied and used to estimate kidney function. Cystatin C, a cysteine protease inhibitor freely filtered by the glomerulus, reabsorbed and then catabolized by renal tubules, seems to be a good marker of moderate kidney dysfunction and a stronger predictor of death[73-77]. Of importance, age, gender, race and an inflammatory state may bias the results of cystatin C[78]. Neutrophil gelatin-associated lipocalin (NGAL), produced by neutrophils and epithelial cells, represents an early, sensitive marker of acute kidney injury. Serum NGAL levels are lower in HIV-infected patients and increase to normal levels after virological response to HAART[79].

All HIV-positive patients should be assessed for existing kidney disease at the time of HIV diagnosis with a screening urinalysis; if there is no evidence of proteinuria at initial evaluation, patients should undergo annual screening for renal disease, including risk assessment, eGFR and urine dipstick analysis[65]. More frequent monitoring is recommended in the presence of CKD risk factors or treatment with nephrotoxic drugs. If there is significant proteinuria, haematuria, decreasing eGFR or eGFR <60 ml/min, current guidelines suggest to perform renal ultrasound, discontinue or adjust drug dosages where appropriate and refer to a nephrologist, for further evaluation and potentially biopsy[65].

As for the role of renal ultrasound, the diagnostic value of renal echogenicity is useful in ruling out HIV-associated nephropathy when a grade 0 or I echogenicity is found or establishing the diagnosis of HIV-associated nephropathy in the presence of grade III echogenicity. The limitation of using echogenicity alone is that renal echogenicity is not a useful measure among 50% of HIV patients with renal disease who have grade II renal echogenicity[80].

When renal biopsy cannot be performed, combining CD4 T-cell count >200 with the absence of nephrotic range proteinuria can be useful in excluding HIV-associated nephropathy with a pretest probability <50%. In any case, the combination of nephrotic range proteinuria and CD4 count <200 has not a strong diagnostic value in detecting HIV-associated nephropathy. Other factors such the rate of increase in serum creatinine and renal echogenicity should be taken into account to establish pretest probability. Recently, Jotwani et al. found that among HIV-infected African American women the presence of 2 APOL1 risk alleles correlates with albuminuria but not with other markers of kidney damage, including IL-18, KIM-1, NGAL and A1M. This population had a faster decline of kidney function and a higher incidence of CKD (10% annual eGFR decline compared with women having 0/1 risk allele)[81].

Treatment of Renal Disease in HIV-Infected Patients

Since HIV infection, either directly or indirectly, is implicated in the development of HIVAN, antiretroviral therapy is an important tool for the treatment of HIV-related renal disease. cART has a critical effect on both preservation and improvement of kidney function in HIV-infected subjects[84-91]. The beneficial effects have been validated by renal biopsy, demonstrating an overt improvement of histo-pathological features[92,93]. On the contrary, the long-term use of HAART has been related with increased risk of progression to ERSD[94]. However, the development of CKD may also be affected by the prolonged survival and increased prevalence of hypertension and diabetes among cART-treated patients[95].

It is paramount to manage diabetes and hypertension promptly and properly. Blood pressure should be monitored regularly and antihypertensive treatment started accordingly. Angiotensin-converting enzyme inhibitors or angiotensin-receptor blockers are the first-choice drugs, because they improve renal hemodynamics, reduce urinary protein excretion and slow the progression to ESRD[97,98]. Glycemic control is also mandatory. Diabetic patients have to maintain glycated hemoglobin level <7%, preprandial plasma glucose level of 90-130 mg/dl and peak postprandial plasma glucose level <180 mg/dl (data from The American Diabetes Association).

In the PREVEND TI study it was observed that fosinipril, but not pravastatin, had an effect on microalbuminuria. On the other hand, pravastatin had a significant beneficial effect on Framingham score after a four-year follow up[99].

Antiretroviral Therapy and kidney function

Renal disease has been mostly correlated with the use of indinavir and TDF, even though isolated reports of nephrotoxicity have been reported with all antiretroviral drugs.

Patients treated with zidovudine, didanosine or integrase inhibitors can develop ARF and the possibility of rhabdomyolysis with pigment-related kidney injury has also to be considered[100-101].

Indinavir has been reported to cause nephrolithiasis and chronic interstitial nephritis[102-103]. Renal adverse effects may occur even after discontinuation of indinavir therapy and may be prevented by adequate hydration. Several investigations have reported crystalluria and nephrolithiasis with atazanavir/ritonavir (ATV/RTV), with an incidence of 7.3-23.7 per 1000 person-year. In some cases, pure ATV stones have been identified, while other reports were associated chronic interstitial nephritis. One study suggested that CKD may be a risk factor for ATV-associated nephrolithiasis. ATV/RTV has also been associated with CDK progression and eGRF reduction[104].

The typical presentation of TDF-associated kidney toxicity is proximal tubulopathy, likely related to the effect on mitochondrial DNA polymerase γ and decreased mitochondrial DNA replication leading to renal function impairment[105-107]. TDF use is associated with proteinuria, mostly of tubular origin[106]. Labarga et al[107] also reported that TDF exposure was linked to an increased risk of kidney tubular abnormalities over time, in the absence of impaired glomerular function. Furthermore, another study reported that each year of TDF was associated with a 33% increased risk of CKD. Since serum creatinine is a late marker of kidney dysfunction, urinary phosphate wasting reflective of proximal tubular dysfunction may be a more sensitive marker for TDF-induced kidney injury. After TDF interruption, it may take several months to normalize serum phosphate and observe eGFR improvement. Of note, after discontinuation of TDF, eGFR did not return to baseline levels in none of the patients after a mean follow-up of 23 months[108].

Gallant et al[109] compared the changes in kidney function in patients taking nucleoside reverse-transcriptase inhibitors with those on TDF. TDF was correlated with a greater eGFR decrease; other risk factors were lower renal function at baseline, low BMI, older age, diabetes, a lower CD4+ T-cell count, underlying CKD and the presence of genetic polymorphisms in the genes encoding MRP2, MRP4 and MRP7[104]. Furthermore, a retrospective analysis in HIV-infected patients with baseline CKD stage 0 or 1, started on either TDF or abacavir from 1998 to 2008, showed that progression to CKD 2 occurred in 48.8% *vs.* 23.7% of patients on TDF or abacavir, respectively; progression to CKD 3 was reported in 5.8% of the TDF group *vs.* 0% of the abacavir group[110].

Gervasoni et al[111] reported that women with low body weight, but not men, had the highest risk of being overexposed to TDF plasma concentration, increasing their risk to develop renal and/or bone disorders. Especially, women with a body weight <50 kg had significantly higher plasma tenofovir concentrations than those weighting >50 kg.

In a large pre-exposure prophylaxis (PrEP) randomized, -o-controlled trial, with median follow-up of 18 months and maximum follow-up of 36 months, daily oral TDF-based PrEP resulted in a small but non-progressive decline in eGFR that was not accompanied by a substantial increase in the risk of clinically relevant eGFR decline. PrEp effects were reversible after drug treatment discontinuation[112].

TDF use has also been associated with Fanconi syndrome (FS), a proximal tubule disease characterized by proteinuria, hypophosphatemia, euglycemic glycosuria, hypouricemia, hypokaliemia and metabolic acidosis. In a prospective, controlled study, Gupta et al. have shown that previous or current use of LPV/r, but not other PIs, and lower eGFR at TDF initiation were significantly and independently associated with the development of FS. Elevated TDF levels may lead to worsening of renal function and may be related with PI use or decreased initial renal function. Moreover, the majority of the proximal tubulopathy markers returned to normal levels within 8 weeks after TDF interruption, although proteinuria persisted in several patients. Lower level dipstick proteinuria at time of TDF discontinuation was also a favorable indicator for renal function recovery[113]. Renal tubular disease/FS has also been reported in patients who received ATV/RTV with TDF/FTC[104]. In a meta-analysis, TDF-containing regimens were correlated with higher risk of acute renal damage and a significant decline of kidney function[114]. Urinary β_2-microglobulin and α1-microglobulin have been shown to be potential screening tools for TDF-induced kidney tubulopathy, especially in early detection of TDF nephrotoxicity[115].

Ritonavir-boosted protease inhibitors (PIs/r) have been associated with an increased risk of TDF toxicity, probably due to the fact that PIs/r compete with TDF for the same renal transporters, reducing its secretion and potentiating its nephrotoxicity. In fact, patients receiving TDF and PI/r had a greater median decline in GFR than those taking TDF and a NNRTI at 6 months (p=0.01), with trends at 12 (p=0.08) and 24 months (p=0.08). There was no difference in median GFR decline between patients receiving NRTI and PI/r compared with those taking NRTI and NNRTI[116]. Another observational longitudinal cohort study found that renal dysfunction was more frequent if TDF was associated with PI/r than NNRTI (9.44% vs. 5.01%, p=0.003)[117]. In the D:A:D cohort of 22,603 patients on HAART with normal baseline renal function, the decline in eGFR was associated with the use of TDF, ritonavir-boosted atazanavir and ritonavir-boosted lopinavir[118].

A number of clinical trials have shown initial reduction in eGRF of 10-15% in patients in whom TDF was initiated with RTV or COBI-boosted protease inhibitors or integrase inhibitors[104]. COBI inhibits the tubular secretion of creatinine, leading to a slight increase (<10%) in serum creatinine levels and an eGFR reduction. These changes are reversible upon drug withdrawal[119].

Tenofovir alafenamide fumarate (TAF) is a novel prodrug and is used at a lower dosage compared with TDF, with lower serum levels of the parent drug. The renal toxicity, which is observed in some patients under TDF therapy,

is less evident in patients treated by TAF. In fact, in contrast to TDF, TAF does not interact with OAT1 or OAT3 organic anion transporters, producing only minimal OAT-mediate cytotoxicity, *in vitro*. These data confirm a better renal safety profile of TAF compared with TDF[119]. In two randomized clinical trials, treatment with a co-formulated tablet of EVG/COBI/FTC/TAF provided non-inferior virological suppression to an already approved and guidelines-recommended tablet of EVG/COBI/FTC/TDF. Of importance, compared to TDF, TAF showed significantly more favorable effects on renal and bone parameters[120].

Rilpivirine, a NNRTI with a virologic efficacy comparable to EFV, has a high protein binding capacity and is excreted only minimally by the kidney. In two large trials, rilpivirine use was also associated with an increase in serum creatinine (approximately 0.1 mg/dl) in patients with normal renal function. Of importance, this increase was stable over time and other laboratory parameters of kidney dysfunction remained in the normal range. eGFR formula based on serum cystatin C did not show any reduction during rilpivirine therapy, even if cystatin C levels may be altered by the virological and immunological status of the patient[119].

Dolutegravir, a novel integrase inhibitor with a renal elimination <1%, induces a serum creatinine increase and moderate reduction (10-15%) of eGFR. These laboratory changes appeared within a week from treatment onset, plateau and then revert towards the baseline values, during dolutegravir treatment[119]. However, it has been demonstrated that dolutegravir does not affect actual eGFR measured by iohexol clearance. In fact, other markers of tubular damage (serum cystatin C, along with urinary B2-microglobulin, N-acetyl-beta-D-glucosaminidase and retinol-binding protein) were not significantly correlated with dolutegravir pharmacokinetic parameters[119].

It is possible to distinguish HIVAN from cART nephrotoxicity evaluating historical information and physical examination, laboratory tests or renal ultrasonography. Signs of uncontrolled HIV infection in association to uncontrolled blood pressure and poor diabetic control are responsible for HIVAN, while proximal tubulopathy with hypophosphatemia or either a partial or full-blown FS suggest cART-associated nephrotoxicity, especially if TDF is part of the regimen. Adequate CD4 T-cell count and undetectable HIV viral load suggest that HIVAN is unlikely the cause of kidney disease. High-grade proteinuria, evaluated by either spot protein/creatinine measurement or 24h urine collection, can be indicative of HIVAN rather than cART nephrotoxicity. Low-grade or tubular proteinuria suggest cART-related kidney injury and dipstick positive glucosuria in the setting of normal serum glucose concentration supports FS and TDF-related injury. The presence of hyaline/proteinaceous casts and scattered RTE cells on urine microscopy are typically present in patients with HIVAN. As for renal ultrasonography, very large and intensely echogenic kidneys with high-grade proteinuria and bland urine sediment are usually indicative of HIVAN[121].

As for the management of HIV-positive patients treated with potentially nephrotoxic drugs, especially in association with the novel drugs that impact on creatinine excretion, there is a need for frequent monitoring, on a monthly basis for the first three months and quarterly thereafter. In the presence of a rise in creatinine concentrations of less than 0.3-0.4 mg/dl during the first 2-8 weeks of therapy, treatment may be maintained. A greater or progressive increase in creatinine should alert for the presence of nephrotoxicity. Patients that experience a decrease of eGFR >25% or show *de novo* occurrence of signs of tubular dysfunction need a nephrology consultation[119].

CONCLUSIONS

Renal disease is an increasing cause of morbidity and mortality in HIV-positive patients.

Careful evaluation of renal function is paramount to identify early stages of kidney disease. Clinically management includes monitoring of serum creatinine and urinary albumin or protein and treatment of comorbidities, including hypertension and diabetes.

Conflict of Interests:
The Authors declare that they have no conflict of interests.

REFERENCES

1. Palella FJ, Baker RK, Moorman AC, Chmiel JS, Wood KC, Brooks JT, Holmberg SD; HIV Outpatient Study Investigators. Mortality in the highly active antiretroviral therapy era: changing causes of death and disease in the HIV outpatient study. J Acquir Immune Defic Syndr 2006; 43: 27-34.
2. Pinzone MR, Castronuovo D, Di Gregorio A, Celesia BM, Gussio M, Borderi M, Maggi P, Santoro CR, Madeddu G, Cacopardo B, Nunnari G. Heel quantitative ultrasound in HIV-infected patients: a cross-sectional study. Infection 2015 Sep [Epub ahead of print].
3. Pinzone MR, Moreno S, Cacopardo B, Nunnari G. Is there enough evidence to use bisphosphonates in HIV-infected patients? A systematic review and meta-analysis. AIDS Rev 2014; 16: 213-222.
4. Scarpino M, Pinzone MR, Di Rosa M, Madeddu G, Focà E, Martellotta F, Schioppa O, Ceccarelli G, Celesia BM, d'Ettorre G, Vullo V, Berretta S, Cacopardo B, Nunnari G. Kidney disease in HIV-infected patients. Eur Rev Med Pharmacol Sci 2013; 17: 2660-2667.
5. Pinzone MR, Nunnari G. Prevalence of comorbidities in a cohort of women living with HIV. Infect Dis Trop Med 2015; 1(3): e165.
6. Martellotta F, Berretta M, Vaccher E, Schioppa O, Zanet E, Tirelli U. AIDS-related Kaposi's sarcoma: state of the art and therapeutic strategies. Curr HIV Res 2009; 7: 634-638.
7. Simonelli C, Tedeschi R, Gloghini A, Talamini R, Bortolin MT, Berretta M, Spina M, Morassut S, Vaccher E, De Paoli P, Carbone A, Tirelli U. Plasma HHV-8 viral load in HHV-8-related lymphoproliferative disorders associated with HIV infection. J Med Virol 2009; 81: 888-896.
8. Berretta M, Zanet E, Di Benedetto F, Simonelli C, Bearz A, Morra A, Bonanno S. Berretta S, Tirelli U. Unusual presentation of metastatic hepatocellular carcinoma in an HIV/HCV coinfected patient: case report and review of the literature. Tumori 2008; 94: 589-591.
9. Cenderello G, Tittle V, Pasa A, Dentone C, Artioli S, Barbour A, Setti M, Giacomini M, Fraccaro P, Viscoli C, Cassola G, Nelson M, Di Biagio A. The impact of liver disease: a leading cause of hospital admissions in people living with HIV. Infect Dis Trop Med 2015; 1(3): e167.

10. Di Benedetto f, Di Sandro S, De Ruvo N, Berretta M, Masetti M, Montalti R, Ballarin R, Cocchi S, Potenza L, Luppi M, Gerunda GE. Kaposi's sarcoma after liver transplantation. J Cancer Res Clin Oncol 2008; 134: 653-658.
11. Castronuovo D, Pinzone MR, Moreno S, Cacopardo B, Nunnari G. HIV infection and bone disease: a review of the literature. Infect Dis Trop Med 2015; 1(2): e116.
12. Berretta M, Martellotta F, Simonelli C, Di Benedetto F, De Ruvo N, Drigo A, Bearz A, Spina M, Zanet E, Berretta s, Tirelli U. Cetuximab/targeted chemotherapy in an HIV-positive patient with metastatic colorectal cancer in the HAART era: a case report. J Chemother 2007; 19: 343-346.
13. Di Benedetto F, De Ruvo N, Berretta M, Masetti M, Montalti R, Di Sandro S, Quintini C, Codeluppi M, Tirelli U, Gerunda GE. Dont' deny liver transplantation to HIV patients with hepatocellular carcinoma in the highly active antiretroviral therapy era. J Clin Oncol 2006; 24: e26-27.
14. Berretta M, Tirelli U. Colorectal cancer screening in HIV-infected patients 50 years of age and older: missed opportunities for prevention. Am J Gastroenterol 2006; 101: 907.
15. Berretta M, Di Benedetto F, Simonelli C, Bearz A, Berretta S, Maugeri D, Tirelli U. Multidisciplinary approach in a HIV/HCV positive patient with liver metastases by colorectal cancer in the HAART era. Ann Oncol 2006; 17: 1333-1334
16. Nasti G. Martellotta F, Berretta M, Mena M, Fasan M, Di Perri G, Talamini R, Pagano G, Montroni M, Cinelli R, Vaccher E, D'Arminio Monforte A, Tirelli U; GICAT; ICONA. Impact of Highly active antiretroviral therapy on the presenting features and outcome of patients with acquired immunodeficiency syndrome-related Kaposi sarcoma. Cancer 2003; 98: 2440-2446;
17. Berretta M, Cinelli R, Martellotta F, Spina M, Vaccher E, Tirelli U. Therapeutic approaches to AIDS-related malignancies. Oncogene 2003; 22: 6646-6659.
18. Spina M, Berretta M, Tirelli U. Hodgkin's disease in Hiv. Hematol Oncol Clin North Am 2003; 17: 843-858.
19. Nunnari G, Xu Y, Acheampong Ea, Fang J, Daniel R, Zhang C, Zhang H, Mukhtar M, Pomerantz RJ. Exogenous IL-7 induces Fas-mediated human neuronal apoptosis: potential effects during human immunodeficiency virus type 1 infection. J Neurovirol 2005; 11: 319-328.
20. Nunnari G, Coco C, Pinzone MR, Pavone P, Berretta M, Di Rosa M, Schnell M, Calabrese G, Cacopardo B. The role of micronutrients in the diet of HIV-1-infected individuals. Front Biosci (Elite Ed) 2012; 4: 2442-2456.
21. Zanet E, Berretta M, Di Benedetto F, Talamini R, Ballarin R, Nunnari G, Berretta S, Ridolfo A, Lleshi A, Zanghì A, Cappellani A, Tirelli U. Pancreatic Cancer in HIV-positive patients: a clinical case-control study. Pancreas 2012; 41: 1331-1335.
22. Berretta M, Garlassi E, Cacopardo B, Cappellani A, Guaraldi G, Cocchi S, De Paoli P, Lleshi A, Izzi I, Torresin A, Di Gangi P, Pietrangelo A, Ferrari M, Bearz A, Berretta S, Nasti G, Di Benedetto F, Balestreri L, Tirelli U, Ventura P. Hepatocellular carcinoma in HIV-infected patients: check early, Treat Hard. Oncologist 2011; 16: 1258-1269.
23. Berretta M, Lleshi A, Cappellani A, Bearz A, Spina M, Talamini R, Cacopardo B, Nunnari G, Montesarchio V, Izzi I, Lanzafame M, Nasti G, Basile F, Berretta S, Fisichella R, Schiantarelli C, Garlassi E, Ridolfo A, Guella L, Tirelli U. Oxaliplatin based chemotherapy and concomitant highly active antiretroviral therapy in the treatment of 24 patients with colorectal cancer and HIV infection. Curr HIV Res 2010; 8: 218-222.
24. Berretta M, Cappellani A, Di Benedetto F, Lleshi A, Talamini R, Canzonieri V, Zanet E, Bearz A, Nasti G, Lacchin T, Berretta S, Fisichella R, Balestreri L, Torresin A, Izzi I, Ortolani P, Tirelli U. Clinical Presentation and Outcome of Colorectal Cancer in HIV-positive patients: a clinical case-control study. Onkologie 2009; 32: 319-324.
25. Berretta M, Zanet E, Basile F, Ridolfo Al, Di Benedetto F, Bearz A, Berretta S, Nasti G, Tirelli U. HIV-positive patients with liver metastases from colorectal cancer deserve the same therapeutic approach as the general population. Onkologie 2010; 33: 203-204.
26. Zanet E, Berretta M, Martellotta F, Cacopardo B, Fisichella R, Tavio M, Berretta S, Tirelli U. Anal cancer: focus on HIV-positive patients in the HAART era. Curr HIV Res 2011; 9: 70-81.
27. Nunnari G, Xu Y, Acheampong Ea, Fang J, Daniel R, Zhang C, Zhang H, Mukhtar M, Pomerantz RJ. Exogenous IL-7 induces Fas-mediated human neuronal apoptosis: potential effects during human immunodeficiency virus type 1 infection. J Neurovirol 2005; 11: 319-328.
28. Nunnari G, Pomerantz RJ. IL-7 as a potential therapy for HIV-1-infected individuals. Expert Opin Biol Ther 2005; 5: 1421-1426.
29. Nunnari G, Berretta M, Pinzone MR, Di Rosa M, Cappellani A, Berretta S, Tirelli U, Malaguarnera M, Schnell JM, Cacopardo B. Hepatocellular carcinoma in HIV positive patients. Eur Rev Med Pharmacol Sci 2012; 16: 1257-1270.
30. Martellotta F, Berretta M, Cacopardo B, Fisichella R, Schioppa O, Zanghì A, Spartà D, Cappellani A, Talamini R, Izzi I, Ridolfo A, Torresin A, Fiorica F, Tirelli U. Clinical presentation and outcome of squamous cell carcinoma of the anus in HIV-infected patients in the HAART-era: a GICAT experience. Eur Rev Med Pharmacol Sci 2012; 16: 1283-1291.
31. Berretta M, Di Benedetto F, Dal Maso L, Cacopardo B, Nasti G, Facchini G, Bearz A, Spina M, Garlassi E, De Re V, Fiorica F, Lleshi A, Tirelli U. Sorafenib for the treatment of unresectable hepatocellular carcinoma in HIV-positive patients. Anticancer Drugs 2013; 24: 212-218.
32. Di Rosa M, Malaguarnera G, De Gregorio C, Palumbo M, Nunnari G, Malaguarnera L. Immuno-modulatory effects of vitamin D3 in human monocyte and macrophages. Cell Immunol 2012; 280: 36-43.
33. Di Rosa M, Malaguarnera L, Nicolosi A, Sanfilippo C, Mazzarino C, Pavone P, Berretta M, Cosentino S, Cacopardo B, Pinzone MR, Nunnari G. Vitamin D3: an ever green molecule. Front Biosci (Schol Ed) 2013; 5: 247-260.
34. Pinzone MR, Fiorica F, Di Rosa M, Malaguarnera G, Malaguarnera L, Cacopardo B, Zanghì G, Nunnari G. Non-AIDS-defining cancers among HIV-infected people. Eur Rev Med Pharmacol Sci 2012; 16: 1377-1388.
35. Bearz A, Vaccher E, Talamini R, Berretta M, Tirelli U. Comment on "Lung cancer in the Swiss HIV Cohort Study: role of smoking, immunodeficiency and pulmonary infection". Br J Cancer 2012; 106: 1899-1900.
36. Nunnari G, Smith JA, Daniel R. HIV-1 Tat and AIDS-associated cancer: targeting the cellular anti-cancer barrier. J Exp Clin Cancer Res 2008; 27: 3.
37. Pinzone MR, Celesia BM, Di Rosa M, Cacopardo B, Nunnari G. Microbial translocation in chronic liver diseases. Int J Microbiol 2012; 2012: 694629.
38. Pinzone MR, Di Rosa M, Malaguarnera M, Madeddu G, Focà E, Ceccarelli G, D'ettorre G, Vullo V, Fisichella R, Cacopardo B, Nunnari G. Vitamin D deficiency in HIV infection: an underestimated and undertreated epidemic. Eur Rev Med Pharmacol Sci 2013; 17: 1218-1232.
39. Pinzone MR, Di Rosa M, Cacopardo B, Nunnari G. HIV RNA Suppression and immune restoration: can we do better? Clin Develop Immunol 2012; 515962.
40. Nunnari G, Otero M, Dornadula G, Vanella M, Zhang H, Frank I, Pomerantz RJ. Residual HIV-1 disease in seminal cells of HIV-1-infected men on suppressive HAART: latency without on-going cellular infections. AIDS 2002; 16: 39-45.
41. Nunnari G, Leto D, Sullivan J, Xu Y, Mehlman Ke, Kulkosky J, Pomerantz RJ. Seminal reservoirs during an HIV type 1 eradication trial. AIDS Res Hum Retroviruses 2005; 21: 768-775.

42. Nunnari G, Sullivan J, Xu Y, Nyirjesy P, Kulkosky J, Cavert W, Frank I, Pomerantz RJ. HIV type 1 cervicovaginal reservoirs in the era of HAART. AIDS Res Hum Retroviruses 2005; 21: 714-718.
43. Nunnari G, Gussio M, Pinzone MR, Martellotta F, Cosentino S, Cacopardo B, Celesia BM. Cryptococcal meningitis in an HIV-1-infected person: relapses or IRIS? Case report and review of the literature. Eur Rev Med Pharmacol Sci 2013; 17: 1555-1559.
44. Pomerantz RJ, Nunnari G. HIV and GB virus C--can two viruses be better than one? N Engl J Med 2004; 350: 963-965.
45. Dornadula G, Nunnari G, Vanella M, Roman J, Babinchak T, De Simone J, Stern J, Braffman M, Zhang H, Pomerantz RJ. Human immunodeficiency virus type 1-infected persons with residual disease and virus reservoirs on suppressive highly active antiretroviral therapy can be stratified into relevant virologic and immunologic subgroups. J Infect Dis 2001; 183: 1682-1687.
46. Otero M, Nunnari G, Leto D, Sullivan J, Wang FX, Frank I, Xu Y, Patel C, Dornadula G, Kulkosky J, Pomerantz RJ. Peripheral blood dendritic cells are not a major reservoir for HIV type 1 in infected individuals on virally suppressive HAART. AIDS Res Hum Retroviruses 2003; 19: 1097-1103.
47. Nunnari G, Argyris E, Fang J, Mehlman KE, Pomerantz RJ, Daniel R. Inhibition of HIV-1 replication by caffeine and caffeine-related methylxanthines. Virology 2005; 335: 177-184.
48. Smith JA, Nunnari G, Preuss M, Pomerantz RJ, Daniel R. Pentoxifylline suppresses transduction by HIV-1-based vectors. Intervirology 2007; 50: 377-386.
49. Pinzone MR, Cacopardo B, Condorelli F, Di Rosa M, Nunnari G. Sirtuin-1 and HIV-1: an overview. Curr Drug Targets 2013; 14: 648-652.
50. Wang FX, Xu Y, Sullivan J, Souder E, Argyris EG, Acheampong EA, Fisher J, Sierra M, Thomson MM, Najera R, Frank I, Kulkosky J, Pomerantz RJ, Nunnari G. IL-7 is a potent and proviral strain-specific inducer of latent HIV-1 cellular reservoirs of infected individuals on virally suppressive HAART. J Clin Invest 2005; 115: 128-137.
51. Pinzone MR, Di Rosa M, Celesia BM, Condorelli F, Malaguarnera M, Madeddu G, Martellotta F, Castronuovo D, Gussio M, Coco C, Palermo F, Cosentino S, Cacopardo B, Nunnari G. LPS and HIV gp120 modulate monocyte/macrophage CYP27B1 and CYP24A1 expression leading to vitamin D consumption and hypovitaminosis D in HIV-infected individuals. Eur Rev Med Pharmacol Sci 2013; 17: 1938-1950.
52. Celesia BM, Castronuovo D, Pinzone MR, Bellissimo F, Mughini MT, Lupo G, Scarpino MR, Gussio M, Palermo F, Cosentino S, Cacopardo B, Nunnari G. Late presentation of HIV infection: predictors of delayed diagnosis and survival in eastern Sicily. Eur Rev Med Pharmacol Sci 2013; 17: 2218-2224.
53. Castronuovo D, Cacopardo B, Pinzone MR, Di Rosa M, Martellotta F, Schioppa O, Moreno S, Nunnari G. Bone disease in the setting of HIV infection: update and review of the literature. Eur Rev Med Pharmacol Sci 2013; 17: 2413-2419.
54. La Ferla L, Pinzone MR, Nunnari G, Martellotta F, Lleshi A, Tirelli U, De Paoli P, Berretta M, Cacopardo B. Kaposi' s sarcoma in HIV-positive patients: the state of art in the HAART-era. Eur Rev Med Pharmacol Sci 2013; 17: 2354-2365.
55. Adih WK, Selix RM, Hu X. Trends in Diseases Reported on US Death Certificates that mentioned HIV infection, 1996-2006. J Int Assoc Physicians AIDS Care (Chic III) 2011; 10: 5-11.
56. Szczech LA, Gange SJ, van der Horst C, Bartlett JA, Young M, Cohen MH, Anastos K, Klassen PS, Svetkey LP. Predictors of proteinuria and renal failure among women with HIV infection. Kidney Int 2002; 61: 195-202.
57. Gupta SK, Mamlin BW, Johnson CS, Dollins MD, Topf JM, Dube MP. Prevalence of proteinuria and the development of chronic kidney disease in HIV-infected patients. Clin Nephrol 2004; 61: 1-6.
58. Gardner LI, Holmberg SD, Williamson JM, Szczech LA, Carpenter CC, Rompalo AM, Schuman P, Klein RS; HIV Epidemiology Research Study Group. Development of proteinuria or elevated serum creatinine and mortality in HIV infected women. J Acquir Immune Defic Syndr 2003; 32: 203-209.
59. Szczech LA, Hoover DR, Feldman JG, Cohen MH, Gange SJ, Goozé L, Rubin NR, Young MA, Cai X, Shi Q, Gao W, Anastos K. Association between renal disease and outcomes among HIV-infected women receiving or not receiving antiretroviral therapy. Clin Infect Dis 2004; 39: 1199-1206.
60. Stehman-Breen CO, Gillen D, Steffes M, Jacobs DR Jr, Lewis CE, Kiefe CI, Siscovick D. Racial differences in early-onset renal disease among young adults: the coronary artery risk development in young adults (CARDIA) study. J Am Soc Nephrol 2003; 14: 2352-2357.
61. Berliner AR, Fine DM, Lucas GM, Rahman MH, Racusen LC, Scheel PJ, Atta MG. Observations on a cohort of HIV-infected patients undergoing native renal biopsy. Am J Nephrol 2008; 28: 478-486.
62. UUSRDS: Annual Data Report. Bethesda, MD, National Institute of Health, NIDDK, 1997.
63. Winston JA, Burns GC, Klotman PE. The human immunodeficiency virus epidemic and HIV-associated nephropathy. Semin Nephrol 1998; 18: 373-377.
64. Winston JA, Klotman ME, Klotman PE. HIV-associated nephropathy is a late, not early, manifestation of HIV-1 infection. Kidney Int 1999; 55: 1036-1040.
65. European Aids Clinical Society. Guidelines: prevention and management of non-infectious comorbidities in HIV. Available at http: //www.europeanaidsclinicalsociety. org/images/stories/EACSPdf/EacsGuidelines-v6.12edition.pdf. Accessed May 2013.
66. Franceschini N, Napravnik S, Eron J, Szczech LA, Finn WF. Incidence and etiology of acute renal failure among ambulatory HIV-infected patients. Kidney Int 2005; 67: 1526-1531.
67. Wyatt CM, Arons RR, Klotman PE, Klotman ME. Acute renal failure in hospitalized patients with HIV: risk factors and impact on in-hospital mortality. AIDS 2006; 20: 561-565.
68. Choi AI, Li Y, Deeks SG, Grunfeld C, Volberding PA, Shlipak MG. Association between kidney function and albuminuria with cardiovascular events in HIV-infected persons. Circulation 2010; 121: 651-658.
69. Szczech LA, Menezes P, Quinlivan EB, van der Horst C, Bartlett JA, Svetkey LP. Microalbuminuria predicts overt proteinuria among patients with HIV-infection. HIV Med 2010; 11: 419-426.
70. K/DOQI clinical practice guidelines for chronic kidney disease: evaluation, classification and stratification. Am J Kidney Dis 2002; 39: S1-266.
71. Stevens LA, Coresh J, Greene T, Levey AS. Assessing kidney function-measured and estimated glomerular filtration rate. N Engl J Med 2006; 354: 2473-2483.
72. Mocroft A, Ryom L, Reiss P, Furrer H, D'Arminio Monforte A, Gatell J, de Wit S, Beniowski M, Lundgren JD, Kirk O. A comparison of estimated glomerular filtration rates using Cockroft-Gault and the Chronic Kidney Disease Epidemiology Collaboration estimating eqations in HIV infection. HIV Medicine 2014; 15: 144-152.
73. O' Riordan SE, Webb MC, Stowe JH, Simpson DE, Kandarpa M, Coakley AJ, Newman DJ, Saunders JA, Lamb EJ. Cystatin C improves the detection of mild renal dysfunction in older patients. Ann Clin Biochem 2003; 40: 648-655.
74. Christensson AG, Grubb A, Nilsson JA, Norrgren K, Sterner G, Sundkvist G. Serum cystatin C advantageous compared with serum creatinine in the detection of mild but no severe diabetic nephropathy. J Intern Med 2004; 256: 510-518.
75. Shlipak MG, Katz R, Sarnak MJ, Fried LF, Newman AB, Stehman-Breen C, Seliger SL, Kestenbaum B, Psaty B, Tracy RP, Siscovick DS. Cystatin C and prognosis of cardiovascular and kidney outcomes in elderly persons without chronic kidney disease. Ann Intern Med 2006; 145: 237-246.

76. Shlipak MG, Sarnak MJ, Katz R, Fried LF, Seliger SL, Newman AB, Siscovick DS, Stehman-Breen C. Cystatin C and the risk of death and cardiovascular events among elderly persons. N Engl J Med 2005; 352: 2049-2060.
77. Larsson A, Helmersson J, Hansson LO, Basu S. Increased serum cystatin C is associated with increased mortality in elderly men. Scand J Clin Lab Invest 2005; 65: 301-305.
78. Knight EL, Verhave JC, Spiegelman D, Hillege HL, de Zeeuw D, Curhan GC, de Jong PE. Factors influencing serum cystatin C levels other than renal function and the impact on renal function measurement. Kidney Int 2004; 65: 1416-1421.
79. Landro L, Damas JK, Flo TH, Heggelund L, Ueland T, Tjønnfjord GE, Espevik T, Aukrust P, Frøland SS. Decreased serum lipocalin-2 levels inhuman immunodeficiency virus-infected patients: increase during highly active anti-retroviral therapy. Clin Exp Immunol 2008; 152: 57-63.
80. Atta MG, Choi MJ, Longenecker JC, Haymart M, Wu J, Nagajothi N, Racusen LC, Scheel PJ, Brancati FL, Fine DM. Nephrotic range proteinuria and CD4 count as non invasive indicators if HIV-associated nephropathy. Am J Med 2005; 118: 1288.e21-1288.e26.
81. Jotwani V, Shlipak MG, Scherzer R, Parekh RS, Kao WH, Bennett M, Cohen MH, Nowicki M, Shamra A, Young M, Tien PC, Parikh CR. APOL1 genotype and glomerular and tubular kidney injury in women with HIV. Am J Kidney Dis 2015; 65: 889-898.
82. Ravani P, Tripepi G, Malberti F, Testa S, Mallamaci F, Zoccali C. Asymmetrical dimethylarginine predicts progression to dialysis and death in patients with chronic kidney disease: a competing risks modeling approach. J Am Soc Nephrol 2005; 16: 2449-2455.
83. Fliser D, Kronenberg F, Kielstein JT, Morath C, Bode-Böger SM, Haller H, Ritz E. Asymmetric dimetholarginine and progression of chronic kidney disease: the mild to moderate kidney disease study. J Am Soc Nephrol 2005; 16: 2456-2461.
84. Kalayjian RC, Franceschini N, Gupta SK, Szczech LA, Mupere E, Bosch RJ, Smurzynski M, Albert JM. Suppression of HIV-1 replication by antiretroviral therapy improves renal function in persons with low CD4 cell counts and chronic kidney disease. AIDS 2008; 22: 481-487.
85. Krawczyk CS, Holmberg SD, Moorman AC, Gardner LI, McGwin G Jr, HIV Outpatient Study Group. Factors associated with renal failure in HIV-infected ambulatory patients. AIDS 2004; 18: 2171-2178.
86. Babut-Gay ML, Echard M, Kleinknecht D, Meyrier A. Zidovudine and nephropathy with human immunodeficiency virus infection. Ann Intern Med 1989; 111: 856-857.
87. Ifudu O, Rao TK, Tan CC, Fleischman H, Chirgwin K, Friedman EA. Zidovudine is beneficial in human immunodeficiency virus associated nephropathy. Am J Nephrol 1995; 15: 217-221.
88. Kirchner JT. Resolution of renal failure after initiation of HAART: three cases and a discussion of the literature. AIDS Read 2002; 12: 103-105, 110-112.
89. Szczech LA, Edwards LJ, Sanders LL, van der Horst C, Bartlett JA, Heald AE, Svetkey LP. Protease inhibitors are associated with a slowed progression of HIV-related renal diseases. Clin Nephrol 2002; 57: 336-341.
90. Strategies for Management of Antiretroviral Therapy (SMART) Study Group. CD4+ count-guided interruption of antiretroviral treatment. N Engl J Med 2006; 355: 2283-2296.
91. Longenecker CT, Scherzer R, Bacchetti P, Lewis CE, Grunfeld C, Shlipak MG. HIV viremia and changes in kidney function. AIDS 2009; 23: 1089-1096.
92. Winston JA, Bruggeman LA, Ross MD, Jacobson J, Ross L, D'Agati VD, Klotman PE, Klotman ME. Nephropathy and establishment of a renal reservoir of HIV type 1 during primary infection. N Engl J Med 2001; 344: 1979-1984.
93. Atta MG, Gallant JE, Rahman MH, Nagajothi N, Racusen LC, Scheel PJ, Fine DM. Antiretroviral therapy in the treatment of HIV-associated nephropathy. Nephrol Dial Transplant 2006; 21: 2809-2813.
94. Ross MJ, Klotman PE, Winston JA. HIV-associated nephropathy: case study and review of the literature. AIDS Patient Care STDS 2000; 14: 637-645.
95. Szczech LA. Tackling the unknowns in HIV-related kidney diseases. N Engl J Med 2010; 363: 2058-2059.
96. Chobanian AV, Bakris GL, Black HR, National Heart, Lung, and Blood Institute Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure; National High Blood Pressure Education Program Coordinating Committee. The seventh report of the Joint National Committee on prevention, detection, evaluation, and treatment of high blood pressure: the JNC 7 report. JAMA 2003; 289: 2560-2572.
97. Molitch ME, DeFronzo RA, Franz MJ, Keane WF, Mogensen CE, Parving HH, Steffes MW; American Diabetes Association. Nephropathy in diabetes. Diabetes Care 2004; 27(Suppl 1): S79-83.
98. Bakris GL, Williams M, Dworkin L, Elliott WJ, Epstein M, Toto R, Tuttle K, Douglas J, Hsueh W, Sowers J. Preserving renal function in adults with hypertension and diabetes: a consensus approach. National Kidney Foundation Hypertension and Diabetes Executive Committees Working Group. Am J Kidney Dis 2000; 36: 646-661.
99. Wasselbergs F, Hillege HL, van Gilst WH. Framingham score and microalbuminuria: combined future targets for primary prevention? Kidney Int 2004; 66: 111-114.
100. Joshi MK, Liu HH. Acute rhabdomyolysis and renal failure in HIV-infected patients: risk factors, presentation, and pathophysiology. AIDS Patient Care and STDs 2000; 14: 541-548.
101. Dori L, Buonomini AR, Viscione M, Sarmati L, Andreoni M. A case of rhabdomiolysis associated with raltegravir use. AIDS 2010; 24: 473-475.
102. Tashima KT, Horowitz JD, Rosen S. Indinavir nephropathy. N Engl J Med 1997; 336: 138-139.
103. Dieleman JP, Sturkenboom MC, Jambroes M, Gyssens IC, Weverling GJ, ten Veen JH, Schrey G, Reiss P, Stricker BH; Athena Study Group. Risk factors for urological symptoms in a cohort of users of the HIV protease inhibitor indinavir sulfate: the ATHENA cohort. Arch Intern Med 2002; 162: 1493-1501.
104. Yombi JC, Pozniak A, Boffito M, Jones R, Khoo S, Levy J, Post FA. Antiretrovirals and the kidney in current clinical practice: renal phamacokinetics, alterations of renal function and renal toxicity. AIDS 2014; 28: 621-632.
105. Birkus G, Hitchcock MJM, Cihlar T. Assessment of mitochondrial toxicity in human cells treated with tenofovir: comparison with other nucleoside reverse transcriptase inhibitors. Antimicrob Agents Chemother 2002; 46: 716-723.
106. Mauss S, Berger F, Schmutz G. Antiretroviral therapy with tenofovir is associated with mild renal dysfunction. AIDS 2005; 19: 93-95.
107. Labarga P, Barreiro P, Martin-Carbonero L, Rodriguez-Novoa S, Solera C, Medrano J, Rivas P, Albalater M, Blanco F, Moreno V, Vispo E, Soriano V. Kidney tubular abnormalities in the absence of impaired glomerular function in HIV patients treated with tenofovir. AIDS 2009; 23: 689-696.
108. Waheed S, Attia D, Estrella MM, Zafar Y, Atta MG, Lucas GM, Fine DM. Proximal tubular dysfunction and kidney injury associated with tenofovir in HIV patients: a case series. CKJ 2015; 8: 420-425.
109. Gallant JE, Parish MA, Keruly JC, Moore RD. Changes in renal function associated with tenofovir disoproxil fumarate treatment, compared with nucleoside reverse-transcriptase inhibitor treatment. Clin Infect Dis 2005; 40: 1194-1198.
110. Monteagudo-chu MO, Chang MH, Fung HB, Bräu N. Renal toxicity of long-term therapy with tenofovir in HIV-infected patients. J Pharm Pract 2012; 25: 552-559.
111. Gervasoni C, Meraviglia P, Landonio S, Baldelli S, Fucile S, Castagnoli L, Clementi E, Riva A, Galli M, Rizzardini G, Cattaneo D. Low body weight in females is a risk factor for increate Tenofovir exposure and drug-related adverse events. PLoS One 2013; 8: e80242.

112. Mugwanya KK, Wyatt C, Celum C, Donnell D, Mugo NR, Tappero J, Kiarie J, Ronald A, Baeten JM. Changes in glomerular kidney function among HIV-1-uninfected men and women receiving Emtricitabine-Tenofovir disoproxil Fumarate preexposure prophylaxis. JAMA Intern Med 2015; 175: 246-254.
113. Gupta SK, Anderson AM, Ebrahimi A, Fralich T, Graham H, Scharen-Guivel V, Flaherty JF, Fortin C, Kalayjian RC, Rachils A, Wyatt CM. Fanconi syndrome accompanied by renal function decline with tenofovir disoproxil fumarate: a prospective, case-control study of predictors and resolution in HIV-infected patients. PLoS One 2014; 9: e92717.
114. Cooper RD, Wiebe N, Smith N, Keiser P, Naicker S, Tonelli M. Systematic review and meta-analysis: renal safety of tenofovir disoproxil fumarate in HIV-infected patients. Clin Infect Dis 2010; 51: 496-505.
115. Nishijima T, Shimbo T, Komatsu H, Takano M, Tanuma J, Tsukada K, Teruya K, Gatanaga H, Kikuchi Y, Oka S. Urinary beta-2 microglobulin and alpha-1 microglobulin are useful screening markers for tenofovir-induced kidney tubulopathy in patients with HIV-1 infection: a diagnostic accuracy study. J Infect Chemother 2013; 19: 850-857.
116. Gallant JE, Moore RD. Renal function with use of a tenofovir containing initial antiretroviral regimen. AIDS 2009; 24: 619-620.
117. Patel KK, Patel AK, Ranjan RR, Patel AR, Patel JK. Tenofovir-associated renal dysfunction in clinical practice: an observational cohort from Western India. Indian J Sex Transm Dis 2010; 31: 30-34.
118. Ryom L, Mocroft A, Kirk O, Worm SW, Kamara DA, Reiss P, Ross M, Fux CA, Morlat P, Moranne O, Smith C, Lundgren JD; D:A:D Study Group. Association Between Antiretroviral Exposure and Renal Impairment Among HIV-Positive Persons With Normal Baseline Renal Function: the D:A:D Study. J Infect Dis 2013; 207: 1359-1369.
119. Maggi P, Montinaro V, Mussini C, Di Biagio A, Bellagamba R, Bonfanti P, Calza L, Cherubini C, Corsi P, Gargiulo M, Montella F, Rusconi S. Novel antiretroviral drugs and renal function monitoring of HIV patients. AIDS 2014; 16: 144-151.
120. Sax PE, Wohl D, Yin MY, Post F, Dejesus E, Saag M, Pozniak A, Thompson M, Podzamczer D, Molina JM, Oka S, Koenig E, Trottier B, Andrade-Villanueva J, Crofoot G, Custodio JM, Plummer A, Zhong L, Cao H, Martin H, Callebaut C, Cheng AK, Fordyce MW, McCallister S; GS-US-292-0104/0111 Study Team. Tenofovir alafenamide versus tenofovir disoproxil fumarate, coformulated with elvitegravir, cobicistat, and emtricitabine for initial treatment of HIV-1 infection: two randomized, double-blind, phase 3, non-inferiority trials. Lancet 2015; 385: 2606-2615.
121. Kumar N, Perazella MA. Differentiating HIV-associated nephropathy from antiretroviral drug-induced nephropathy: a clinical challenge. Curr HIV/AIDS Rep 2014; 11: 202-211.