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Hepatic Steatosis, Measured by Transient Elastography with Controlled Attenuation Parameter, is Highly Prevalent in Japanese HIV-Infected Patients

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Abstract

Objective: HIV-infected patients are at an increased risk of developing Hepatic Steatosis (HS), which may progress to nonalcoholic steatohepatitis. The aim of this study was to evaluate HS in Japanese HIV-infected patients using a novel technique called Controlled Attenuation Parameter (CAP).

Methods: Sixty HIV-infected outpatients were included in this study and underwent transient elastography with CAP. Significant Hepatic Steatosis (SHS) was defined using the previously established CAP cut-off of 232dB/m. The relationships between SHS and blood chemistry, HBV/HCV co-infection and antiretroviral drugs were analyzed in HIV-infected patients.

Results: The mean CAP level was 228dB/m. SHS was detected in 25 patients (42.3%). In the univariate analysis, the percentage of patients with SHS increased with the levels of triglyceride, fasting plasma glucose, HbA1c and BMI. SHS was observed less frequently among those who had been treated with oral tenofovir for 2 years or longer. Neither Hepatitis B surface Antigen (HBsAg) -positive rates, the duration for which patients were positive for HBsAg, HCV antibody-positive rates, nor the duration for which patients were positive for HCV RNA were associated with SHS. In a multivariate analysis, the only factor associated with SHS was BMI (adjusted odds ratio 1.727, 95% CI: 1.095 - 2.724; P = 0.019).

Conclusion: The prevalence of SHS was markedly higher as 42.3% among HIV-infected patients and higher BMI in Japanese individuals was associated with SHS.

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Introduction

Hepatic steatosis (HS) is one of the most common causes of liver disease in human immunodeficiency virus (HIV)-infected patients as well as in the general population[1,2]. HS is associated with an increased risk of liver inflammation and fibrosis, "so called" Nonalcoholic Steatohepatitis (NASH), a recognized cause of Liver Cirrhosis (LC) and Hepatocellular Carcinoma (HCC). HS in HIV-infected patients may progress to NASH, LC and HCC. The prevalence of HS and its risk factors in HIV-infected patients currently remain unclear[3]. HS rates in HIV-infected patients have been estimated based on the findings of previously conducted liver biopsy studies, which mostly involved HIV/HCV-co-infected patients. Liver biopsy is an invasive technique and sampling errors are common[4-6]. A new non-invasive assessment of HS through the measurement of controlled attenuation parameter (CAP) using the

transient elastography (M probe) (FibroScan®; Echosens, Paris, France) has been developed. This novel technique estimates the presence of Significant Hepatic Steatosis (SHS) based on more than 10% of hepatocytes being detected in a liver biopsy sample[7]. Therefore, the aim of the present study was to evaluate SHS in Japanese HIV-infected patients using CAP[8].

Materials and Methods

Between January 2014 and May 2015, all HIV-infected outpatients in the Department of Infectious Diseases and Immunology, Clinical Research Center, National Hospital Organization Nagoya Medical Center, Nagoya, were included in this study. They provided written informed consent to participate in this study and underwent transient elastography. CAP measurements were performed by an experienced practitioner at the Department of Gastroenterology, National Hospital Organization Nagoya Medical Center, Nagoya, using FibroScan®. CAP is an estimate of total ultrasonic attenuation and expressed in dB/m. In a previous study, examinations with ten successful shots, an interquartile range (IQR) for liver stiffness of less than 30% of the median value, and a success rate of at least 60%, were considered as reliable[9] and included in this study.

Sasso M, et al.[8] previously reported the CAP cut-off that defines SHS by presenting at least 10% hepatocytes to be 238dB/m. But, Masaki K, et al.[10] identified the CAP cut-off for defining SHS in Japanese patients as 232dB/m. Therefore, we herein selected 232dB/m as the CAP cut-off.

This study was designed in accordance with the Helsinki declaration and approved by the Ethics Committee of the National Hospital Organization Nagoya Medical Center.

Statistical Analysis

The primary outcome was the presence of SHS, defined as a CAP value equal to or greater than 232dB/m. The relationships between the following factors and SHS were analyzed: age, sex, race, rate of HIV infection, alcohol intake, tobacco consumption, CD4+ cell count, HIV RNA viral load, use of Antiretroviral Therapy (ART), AST, ALT, γGTP, platelet count, triglycerides, total cholesterol, LDL cholesterol, HDL cholesterol, fasting plasma glucose, HbA1c, Hepatitis B surface antigen (HBsAg), HBeAg, HBeAb, HBV DNA, the HBV genotype, HCV Ab, HCV RNA, the HCV genotype, BMI, waist circumference and Liver Stiffness Measurement (LSM). Continuous variables were expressed as a median (minimum-maximum) or mean (standard deviation, SD). Categorical variables were expressed as numbers (percentage). Factors associated with the primary outcome in logistic regression analysis with a P-value less than 0.05 were considered to be significant. Statistical analyses were performed using the SPSS 22 statistical software package (SPSS, Chicago, Illinois, USA).

Results

Patient Characteristics

The characteristics of the 60 patients who participated in this study are presented in (Table 1). All patients successfully underwent elastography. It was possible to measure CAP in 59 patients (98%) using M probe. Thus, data collected from these patients were analyzed statistically. No patient had a self-reported alcohol intake of more than 40g/day. Five patients (8%) tested negative for HCV Ab, HBsAg, HBc Ab and HBsAb. The number of HBsAg-negative and HBc and/or HBsAb-positive (indicating a history of HBV infection) patients was 13 (22%). Among the 28 (46%) HBsAg-positive patients, 14 (50%), 10 (36%), and 12 (43%) were also HBeAg-, HBeAb-, and HBV DNA-positive, respectively. The median duration for which patients were positive for HBsAg was 46 (12-85) months. Eighteen patients (64%) had HBV genotype A, two (8%) genotype B, and four (14%) genotype C. Fourteen patients (23%) tested positive for HCVAb. Five of them were negative for HCV RNA because they had previously received pegylated interferon with Ribavirin therapy and achieved a Sustained Virological Response (SVR). The median duration for which patients were positive for HCV RNA was 72 (2-80) months, while the median duration after SVR was 36 (24-84) months. Six patients (43%) had HCV genotype 1, three (31%) genotype 2, four (29%) genotype 3, and one (7%) genotype 6.

Table 1: Patient Characteristics of this study (n=60)

Characteristics		
Male, n (%)	58	(97)
Age[a], years	42	(31-73)
Race, n (%)		
Japanese	57	(95)
Chinese	2	(3)
Korean	1	(2)
Route of HIV infection		
Sextual transmission	51	86
Blood transfysion	5	7
Intravenous drug use	5	7
Daily alcohol intake, n (%)		
0	31	(52)
1-39 g/day	29	(48)
≧40 g/day	0	(0)
Tobacco consumption, n (%)	15	(25)
CD4+ cell counts[b] (cells/μl)	487	(258)
HIV RNA, n (%)		
Undetectable	33	(55)
<20 copies/ml	14	(23)
≧20 copies/ml	13	(22)
under AART, n (%)	58	(97)
AST[a] (IU/L)	24	(12-237)
ALT[a] (IU/L)	27	(7-308)
γGTP[a] (IU/L)	37	(9-246)
Platelet[b] (10^4/μl)	21	(7.7)
Triglycerides[a] (mg/dl)	127	(38-641)
Total cholesterol[b] (mg/dl)	152	(79-233)
LDL cholesterol[a] (mg/dl)	85	(42-155)
HDL cholesterol[a] (mg/dl)	39	(10-75)
Fasting plasma glucose[a] (mg/dl)	101	(76-211)
Hb A1c[b] (%)	5.3	(0.6)
HCV Ab HBsAg HBcAb HBsAb, n (%)		

(-) (-) (-) (-)	5	(8)
(-) (-) (+) and/or (+)	13	(22)
(-) (+)	28	(47)
(+) (-) (+) and/or (+)	12	(20)
(+) (-) (-) (-)	2	(3)
positive for HBsAg, n (%)	28	(46)
positive for HBeAg, n (%)	14	(50)
positive for HBeAb, n (%)	10	(36)
positive for HBV DNA, n (%)	12	(43)
Duration positive for HBsAg[a] (months)	46	(12-85)
HBV genotype, n (%)		
A	18	(64)
B	2	(8)
C	4	(14)
unknown	4	(14)
positive for HCV Ab, n (%)	14	(23)
HCV RNA positive	9	
HCV RNA negative[c]	5	
Duration positive for HCV RNA[a] (months)	72	(2-80)
Duration after SVR[a] (months)	36	(24-84)
HCV genotype, n (%)		
1	6	(43)
2	3	(21)
3	4	(29)
6	1	(7)
BMI[b] (kg/m^2)	23	(3.9)
BMI<25, n (%)	43	(72)
BMI≧25, n (%)	17	(28)
Waist circumference[b] (cm)	83	(10)
Liver stiffness		
measured by M probe : XL probe	59 : 1	
Liver stiffness[a] (Kpa)	4.7	(2.6-22.8)
CAP		
measured by M probe	59	(98)
CAP[b] (dB/m)	228	(59)
CAP≧232, n (%)	25	(42.3)

AART, active antiretroviral therapy; AST, aspartate aminotasferase; ALT, alanine aminotasferase;
γGTP, gamma-glutamyl transferase; LDL, low-density lipoprotein; HDL, high -density lipoprotein;
HBV, heptitis B virus, HCV, hepatitis C virus; HBsAg: HB surface antigen, HBsAb: HB surface antibody
HBcAb: HB core antibody, HBeAg: HB e antigen, HBeAb: HB e antibody
HCV Ab: HCV antibody CAP, SVR: sustaind virological response, controlled attnuation parameter
[a]Median (minimum-maximum)
[b]Mean (SD)
[c]Pegylated interferon with ribavirin was carried out and achieved SVR.

The mean BMI was 23 (3.9). Obesity, defined as BMI more than 25, was observed in 17 patients (28%). The mean waist circumference was 83 (10) cm.

Fifty-eight patients (97%) were treated with ART, with 33 (55%) showing undetectable HIV RNA levels. The distribution of antiretroviral drugs administered to patients is summarized in [Tables 2-3].

Table 2: Antiretroviral drugs ever administered to this study patients and significant hepatic steatosis (CAP≧232 dB/m)

Drug	n	CAB≧232 dB/m, n (%)	p
tenofovir			0.187
yes	47	13 (28)	
no	12	7 (58)	
emtricitabine			0.308
yes	43	12 (28)	
no	16	8 (50)	
lamivudine			0.545
yes	21	10 (48)	
no	38	10 (6)	
abacavir			0.434
yes	18	9 (50)	
no	41	11 (29)	
sanilvudine			0.122
yes	9	6 (67)	
no	50	14 (28)	
didovudine			0.596
yes	9	3 (33)	
no	50	17 (34)	
didanosine			0.691
yes	6	3 (50)	
no	53	17 (32)	
reltegravir			0.767
yes	27	12 (44)	
no	32	8 (22)	
dolutegravir			0.182
yes	12	3 (25)	
no	47	17 (36)	
elvitergravir			0.396
yes	7	3 (43)	
no	52	17 (33)	
lopinavir			0.655
yes	11	4 (36)	
no	48	16 (33)	
atazanavir			0.199
yes	9	2 (22)	
no	50	18 (36)	
darunavir			0.830
yes	16	3 (19)	
no	43	17 (40)	
ritonavir			0.993
yes	27	7 (26)	

no	32	13 (41)	
nelfinavir			0.312
yes	5	1 (20)	
no	54	19 (35)	
fosamprenavir			0.638
yes	6	0	
no	53	20 (38)	
saquinavir			-
yes	1	0	
no	58	20 (34)	
indinanir			-
yes	1	0	
no	58	20 (34)	
nevirapine			0.999
yes	3	3 (100)	
no	56	17 (30)	
rilpivirine			0.911
yes	5	2 (40)	
no	54	18 (33)	
efavirenz			0.891
yes	9	4 (44)	
no	50	16 (32)	
etravirine			0.312
yes	5	1 (20)	
no	54	19 (35)	
maraviroc			0.999
yes	3	0	
no	56	20 (36)	

Table 3: Antiretroviral drugs exposure (drug exposure<2 years vs ≧ 2 years) and significant hepatic steatosis (CAP≧232 dB/m)

antiretroviral drugs		n (%)	odds ratio (95%CI)	p
tenofovir	none	12 (20)		
	drug exposure<2 years	14 (24)	0.035 (0.101-2.185)	0.469
	≧ 2 years	33 (56)	0.210 (0.054-0.810)	0.023
emtricitabine	none	16 (27)		
	drug exposure<2 years	14 (23)	0.565 (0.122-3.603)	0.462
	≧ 2 years	29 (50)	0.358 (0.100-1.283)	0.115
lamivudine	none	38 (65)		
	drug exposure<2 years	2 (3)	2.800 (0.160-49.103)	0.481
	≧ 2 years	19 (32)	2.240 (0.690-7.29)	0.179
abacavir	none	41 (70)		
	drug exposure<2 years	5 (8)	1.818 (0.267-12.376)	0.541
	≧ 2 years	13 (22)	3.182 (.875-11.569)	0.079
sanilvudine	none	50 (85)		
	drug exposure<2 years	3 (5)	7.714 (0.739-80.554)	0.088
	≧ 2 years	6 (10)	3.857 (0.581-25.0601)	0.162
didovudine	none	50 (859		
	drug exposure<2 years	3 (5)	1.031 (0.87-12.236)	0.981
	≧ 2 years	6 (10)	1.031 (0.171-6.235)	0.973
reltegravir	none	32 (54)		
	drug exposure<2 years	10 (17)	3.472 (0.760-15.896)	0.108
	≧ 2 years	17 (29)	1.667 (0.470-5.916)	0.429
dolutegravir	none	47 (80)		
	drug exposure<2 years	12 (20)	0.684 (0.160-2.923)	0.608
	≧ 2 years	0		
elvitergravir	none	52 (88)		
	drug exposure<2 years	7 (14)	1.500 (0.301-7.467)	0.621
	≧ 2 years	0		
lopinavir	none	48 (82)		
	drug exposure<2 years	2 (3)	-	0.999
	≧ 2 years	9 (15)	1.600 (0.377-6.789)	0.524
atazanavir	none	50 (85)		
	drug exposure<2 years	1 (2)	-	1.000
	≧ 2 years	8 (14)	0.733 (0.129-4.168)	0.733
darunavir	none	43 (73)		
	drug exposure<2 years	3 (5)	-	0.999
	≧ 2 years	13 (22)	0.476 (0.115-1.9839	0.308
ritonavir	none	32 (54)		
	drug exposure<2 years	1 (2)	-	
	≧ 2 years	26 (44)	0.567 (0.186-1.725)	0.567
nevirapine	none	56 (95)		
	drug exposure<2 years	2 (3)	-	1.000
	≧ 2 years	1 (2)	-	0.999
rilpivirine	none	54 (92)		
	drug exposure<2 years	3 (5)	1.000 (0.085-11.778)	1.000
	≧ 2 years	2 (3)	2.000 (0.118-33.856)	0.631
efavirenz	none	50 (85)		
	drug exposure<2 years	2 (3)	2.125 (0.125-36.182)	0.602
	≧ 2 years	7 (12)		0.571

95%CI: 95% confidential interval

Prevalence of and factors associated with SHS

The mean CAP in the 59 HIV-infected patients examined in this study was 228 (59) dB/m. SHS, with CAP of at least 232dB/m, was observed in 25 patients (42.3%). Neither

age, sex, the CD4+ cell count, HIV RNA viral load, AST, ALT, γGTP, nor the platelet count were associated with SHS. Higher concentrations of triglycerides (odds 1.012, 95%CI: 1.003-1.021; P=0.006), fasting plasma glucose (odds 1.048, 95%CI: 1.008-1.190; P=0.018), HbA1c (odds 2.821, 95%CI: 1.002-7.945; P=0.050), BMI (odds ratio 1.709, 95%CI: 1.280-2.382; P=0.0001), and waist circumference (odds ratio 1.303, 95%CI: 1.125-1.510; P=0.001) were associated with SHS. HBsAg-positive rates, the duration for which patients were positive for HBsAg, HCV Ab-positive rates, and the duration for which patients were positive for HCV RNA were not associated with SHS. LSM was also not associated with SHS. Relationships with SHS identified by a univariate analysis are summarized in [Table 4].

Table 4: Associations with significant hepatic steatosis (CAP≧232 dB/m)

Varialle	odds ratio (95%CI)	p
Age, years	0.98 (0.941-1.029)	0.478
CD4+ cell counts[a] (cells/μl)	1.002 (1.000-1.004)	0.115
HIV RNA	0.750 (0.06-2.134)	0.590
AST (IU/L)	1.012 (0.994-1.030)	0.184
ALT (IU/L)	1.009 (0.997-1.022)	0.151
γGTP (IU/L)	1.008 (0.997-1.019)	0.153
Platelet (10^4/μl)	0.951 (0.887-1.020)	0.159
Triglycerides (mg/dl)	1.012 (1.003-1.021)	0.006
Total cholesterol (mg/dl)	0.998 (0.982-1.015)	0.814
LDL cholesterol (mg/dl)	0.994 (0.974-1.013)	0.524
HDL cholesterol (mg/dl)	0.963 (0.917-1.011)	0.126
Fasting plasma glucose (mg/dl)	1.048 (1.008-1.190)	0.018
Hb A1c (%)	2.821 (1.002-7.945)	0.050
positive for HBs antigen, n (%)	1.038 (0.369-2.920)	0.943
Duration positive for HBsAg (months)	0.995 (0.977-1.013)	0.574
Duration positive for HBV DNA (months)	0.995 (0.974-1.017)	0.656
positive for HCV antibody, n (%)	1.500 (0.449-5.008)	0.150
Duration positive for HCV RNA (months)	1.010 (0.989-1.032)	0.342
Duration after SVR (months)	1.035 (0.981-1.093)	0.205
BMI (kg/m^2)	1.709 (1.280-2.282)	0.0001
Waist circumference (cm)	1.303 (1.125-1.510)	0.0001
Liver stiffness (Kpa)	1.074 (0.960-1.201)	0.213

95%CI: 95% confidential interval

The relationship between the antiretroviral drugs administered to our patients and SHS is shown in [Table-2]. The administration or not of antiretroviral drugs was not associated with SHS. The relationship between antiretroviral drug exposure for less than two years and equal to or greater than two years and SHS is shown in [Table-3]. The incidence of SHS decreased only when administering oral Tenofovir for 2 years or longer (odds ratio 0.210, 95%CI: 0.054-0.810; P=0.023).

A multivariate analysis revealed that the only factor associated with SHS was BMI [Table-5]. Waist circumference and HbA1c were excluded, because these were considered to present same aspects with BMI and fasting plasma glucose. BMI was the only predictor of SHS (per unit increase, adjusted odds ratio 1.727, 95%CI: 1.095-2.724; P=0.019)

Table 5: Association of significant hepatic steatosis with the factors in multivariate analysis (CAP≧232 bB/m).

Variable		Adjusted odds ratio (95%CI)	Multivariate p
Age	per year	0.927 (0.849-1.013)	0.094
BMI	per unit increase	1.727 (1.095-2.724)	0.019
Undetectable HIV RNA	yes vs no	0.159 (0.020-1.260)	0.082
Triglycerides	per unit increase	1.014 (0.999-1.028)	0.070
Fasting plasma glucose	per unit increase	1.018 (0.976-1.063)	0.405
Undetectable HBsAg	yes vs no	1.289 (0.107-15.582)	0.842
Undetectable HCV Ab	yes vs no	7.109 (0.549-92.002)	0.133
tenofovir	exposure≧2years vs the others	0.269 (0.043-1.666)	0.161

95%CI: 95% confidential interval

Discussion

SHS, estimated to contain more than 10% of hepatocytes in liver biopsy samples and CAP of at least 232dB/m, was detected in 25 patients (42.3%) in the present study. The prevalence of HS in the general population in Western countries was previously reported to be approximately 20-30%; 30% in USA and 25% in Italy. In Japan, the prevalence of HS has been increasing and was reported to be 29.7% between 2009 and 2010[2]. CAP measurements revealed that the prevalence of SHS was higher among HIV-infected patients than in the general population[11]. Macias J, et al. found that the prevalence of SHS among HIV-infected patients in Spain, as measured by CAP, was 40%, which is consistent with the results of the present study. A survey to estimate HS using transient elastography with CAP was recently performed on various hepatic disorders[12-15]. However, these surveys are rarely conducted on HIV-infected patients[16]. Previous studies demonstrated that it was possible to accurately detect SHS using a CAP cut-off of 238dB/m in Western countries and 232dB/m in Japan. The prevalence of HS and its risk factors in HIV-infected patients have not yet been clarified[8,10,15]. The previously reported prevalence of HS in HIV-infected patients was mostly derived from biopsy studies involving HIV/HCV-co-infected patients. Liver biopsy is an invasive technique and sampling errors are common[4-6]. The prevalence of HS in HIV/HCV-co-infected patients are known to be approximately 50%[17] however, few studies have examined HS in HIV-infected patients without HCV. In some cohort studies of HIV-infected patients, the prevalence of HS diagnosed using abdominal Ultrasound (US) was 31-53%[6,18].The prevalence of HS in HIV-infected patients without hepatitis and diagnosed using abdominal CT was found to be 37%[19]. US is not sufficiently sensitive to detect HS and the rate obtained may be an underestimation or overestimation of the actual value. CAP may be simultaneously measured with LSM and more precisely

estimates the rate of HS than the previously identified CAP cut-off of 232dB/m.

HS is the one symptom of metabolic syndrome. Patients in the present study also showed a close association with metabolic syndrome complicated by SHS[17-19]. In a univariate analysis, metabolic factors including triglycerides, fasting plasma glucose, HbA1c, BMI and waist circumference were associated with SHS. In a multivariate analysis, the only factor associated with SHS was BMI. Increases in the BMI of HIV-infected patients may have reflected the presence of obesity or metabolic syndrome involving SHS among such patients.

In the present study, the only factor associated with SHS was BMI. Obesity has been identified as one of the most important risk factors for HS in the general population. HS occurs due to the excessive accumulation of triglycerides in hepatocytes[2,11]. Another mechanism responsible for HS is the impaired oxidation of fatty acids due to mitochondrial toxicity associated with Nucleoside analogue Reverse Transcriptase Inhibitors (NRTI) and Efavirenz. But some studies reported that no relationship existed between HS and antiretroviral drugs[5,20-22]. In the present study, while univariate analysis suggested that oral Tenofovir administration for 2 years or longer prevented SHS, multivariate analysis did not show a relationship[4,18,19]. On the other hand, while univariate analysis suggested that lower percentage of patients with a BMI of 25 or greater when treated with oral Lopinavir was observed (the results not shown in Table 2,3), multivariate analysis did not show a relationship. Thus, SHS and BMI were not more frequently among the patients who had been treated with the oral antiretroviral drugs in the present study.

BMI is known to increase in HIV-infected patients following the initiation of ART. Gracia A, et al. reported that the amounts of limb and visceral fat were higher in HIV-infected patients within the first 96 weeks of ART. As HS is a type of visceral fat, it may be appropriate to consider the redistribution of fat related to HIV infection which increases its volume[23].

In the present study, 28 (46%) patients tested positive for HBsAg and 14 (23%) were positive for HCV Ab. Although 42 (70%) patients had a hepatitis virus co-infection, HBsAg-positive rates, the duration for which patients were positive for HBsAg, HCV Ab-positive rates and the duration for which patients were positive for HCV RNA were not associated with SHS.

In conclusion, the prevalence of HS was higher as 42.3% among HIV-infected patients. The prevalence of HS needs to be determined more precisely because it progresses to NASH, LC and HCC. Increases in BMI and obesity of HIV-infected patients are the most important factors associated with HS. Clinicians need to recommend changes to diet and lifestyle-related habits in order to lower BMI, thereby reducing the risk of HS progressing to NASH.

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Conflict of Interest: The authors declare no conflict of interest

References

1. Ristig, M., Drechsler, H., Powderly, W.G. Hepatic steatosis and HIV infection. (2005) AIDS Patient Care STDS 19(6): 356-365.
2. Bellentani, S., Scaglioni, F., Marino, M., et al. Epidemiology of non-alcoholic fatty liver disease. (2010) Dig Dis 28(1): 155-161.
3. Ascha, M.S., Hanouneh, I.A., Lopez, R., et al. The incidence and risk factors of hepatocellular carcinoma in patients with nonalcoholic steatohepatitis. (2010) Hepatology 51(6): 1972-1978.
4. Woreta, T.A., Sutcliffe, C.G., Mehta, S.H., et al. Incidence and risk factors for steatosis progression in adults coinfected with HIV and hepatitis C virus. (2011) Gastroenterology 140(3): 809-817.
5. Macias, J., Berenguer, J., Japon, M.A., et al. Hepatic steatosis and steatohepatitis in human immunodeficiency virus/hepatitis coinfected patients. (2012) Hepatology 56(4):1261-1270.
6. Valentina, L.V., Maurizio, S., Lyndia, G., et al. Prospective evaluation of hepatic steatosis in HIV-infected patients with or without hepatitis C virus co-infection. (2012) Int J Infect Dis 16(5): e397-e402.
7. Sasso, M., Beaugrand, M., de Ledinghen, V., et al. Controlled attenuation parameter (CAP): a novel VCTE guided ultrasonic attention measurement for the evaluation of hepatic steatosis: preliminary study and validation in cohort patients with chronic liver disease from various causes. (2010) Ultrasound Med Biol 36(11): 1825-1835.
8. Sasso, M., Miette, V., Sandrin, L., et al. The controlled attenuation parameter (CAP): A novel tool for the non-invasive evaluation of steatosis using Fibroscan. (2012) Clin Res Hepatol Gastroenterol 36(1): 13-20.
9. Castera, L., Forns, X., Alberti, A. Noninvasive evaluation of liver fibrosis using transient elastography. (2008) J Hepatol 48(5): 835-847.
10. Masaki, K., Takaki, S., Hyogo, H., et al. Utility of controlled attenuation parameter measurement for assessing liver steatosis in Japanese patients with chronic liver disease. (2013) Hepatol Res 43(11): 1182-1189.
11. Eguchi, Y., Hyogo, H., Ono, M., et al. Prevalence and associated metabolic factors of nonalcoholic fatty liver disease in the general population from 2009 to 2010 in Japan: a multicenter large retrospective study. (2012) Gastroenterol 47(5): 586-595.
12. Sasso, M., Tengher-Barna, I., Ziol, M., et al. Novel controlled attenuation parameter for noninvasive assessment of steatosis used Fibroscan(®): validation in chronic hepatitis C. (2012) J Viral Hepat 19(4): 244-253.
13. Myers, R.P., Pollett, A., Kirsch, R., et al. Controlled attenuation parameter (CAP): a noninvasive method for the detection of hepatic steatosis based on transient elastography. (2012) Liver Int 32(6): 902-910.
14. Chon, Y.E., Jung, K.S., Kim S.U., et al. Controlled attenuation parameter (CAP) for detection of hepatic steatosis in patients with chronic liver diseases: a prospective study of a native Korean population. (2014) Liver Int 34(1): 102-109.
15. De Ledinghen, V., Vergniol, J., Foucher, J., et al. Noninvasive diagnosis of liver steatosis using controlled attenuation parameter (CAP) and transient elastography. (2012) Liver Int 32(6): 911-918.
16. Macias, J., Gonzalez, J., Tural, C., et al. Prevalence and factors associated with liver steatosis as measured by transient elastography with controlled attenuation parameter in HIV-infected patients. (2014) AIDS 28(9): 1279-1287.
17. Machado, M.V., Oliveria, A.G., Cortez-Pinto, H. Hepatic steatosis in patients coinfected human immunodeficiency virus/hepatitis C virus: a meta-analysis of the risk factors. (2010) Hepatology 52(1): 71-78.
18. Curum-Cianflone, N., Dailay, A., Collins, G., et al. Nonalcoholic fatty liver disease among HIV-infected persons. (2009) J Acquire Immune Defic Syndr 50(5): 464-473.
19. Guaraldi, G., Squilace, N., Stentarelli, C., et al. Nonalcoholic fatty liver disease among HIV-infected patients referred a metabolic clinic: prevalence, characteristics, and predictors. (2008) Clin Infect Dis 47(2): 250-257.
20. Maagaard, A., Kvale, D. Mitochondrial toxicity in HIV-infected pa-

tients both off and on antiretroviral treatment: a continuum or distinct underlying mechanism. (2009) J Antimicrob Chemother 64(5): 901-909.

21. Venhoff, N., Setzer, B., Melkaoui, k., et al. Mitochondrial toxicity of tenofovir, emtricitabine and abacavir alone and in combination with additional nucleoside reserve transcriptase inhibitors. (2007) Antivir Ther 12(7): 1075-1085.

22. Blas-Garcia, A., Apostolova, N., Ballesteros, D., et al. Inhibition of mitochondrial function by efavirenz increases lipid content in hepatic cells. (2010) Hepatology 52(1): 115-125.

23. McComsey, G.A., Kitch, D., Sax, P.E., et al. Peripheral and central fat changes in subjects randomized to abacavir-lamivudine or tenofovir-emtricitabine with atazanavir or efavirenz: ACTG study A522s. (2011) Clin Infect Dis 53(2): 185-196.

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