

Factors Associated with Abdominal Obesity among HIV-infected Adults on Antiretroviral Therapy in Malaysia

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Abstract

Abdominal or central obesity is a common morphological alteration among HIV-infected subjects on antiretroviral treatment. There is concern that this condition places the subjects at risk of cardiovascular disease. This is a cross-sectional study of 334, HIV-infected adult subjects on antiretroviral therapy at a public hospital in Malaysia. It was aimed at determining the association between nutritional factors and abdominal obesity among PLHIV receiving antiretroviral treatment. Abdominal obesity was prevalent in 36.5% of the respondents. Respondents with abdominal obesity were significantly ($p < 0.05$) older in age, had significantly higher blood triglycerides, fasting plasma glucose, lower HDL-cholesterol, higher BMI at the start of medication and also at the time of the study, bigger waist circumference, higher waist hip ratio, body fat mass, systolic and diastolic blood pressure. They also had lower mean CD4 cell count at start of medication and body lean mass than those without abdominal obesity. After adjusting for the covariates, a significantly higher risk of abdominal obesity was observed in those who were older (adjusted OR=1.053, CI=1.012-1.095), had higher fasting plasma glucose (adjusted OR=1.189, CI=1.014-1.394), higher BMI at the time of study (adjusted OR=1.426, CI=1.215-1.674). Being Malay was a protective factor (adjusted OR=0.264, CI=0.102-0.685) for abdominal obesity. These results suggest that care of the HIV-infected population must include intervention to address abdominal obesity in order to provide better quality of life.

Keywords: Abdominal obesity, HIV/AIDS, ARV treatment, Nutrition, Dietary intake, Waist-Hip ratio, Malaysia

1. Introduction

Acquired Immune Deficiency Syndrome (AIDS or Aids) is a rapidly growing global problem that is accompanied by high morbidity and mortality. According to the latest report (UNAIDS, 2008) the number of people living with the human immunodeficiency virus (HIV) in the world totaled 33 million people as of December 2007 of whom 5 million were in Asia. In Malaysia there were more than 80 000 [52 000-120 000]

people living with HIV (PLHIV) as of the end of 2008 (UNAIDS, 2008). Antiretroviral (ARV) drugs are the only medication available to inhibit viral replication and reduce morbidity and mortality due to AIDS in the absence of vaccination (WHO, 2000a). The strategy of free access to antiretroviral (ARV) treatments adopted by Malaysia has notably increased the survival rate as a result of a reduction in the incidence of opportunistic infections and also HIV/AIDS complications (Malaysia UNGASS Report, 2008).

On the other hand, ART (Antiretroviral Therapy) can cause a variety of side effects, some of them adversely affecting the nutritional status of PLHIV (WHO, 2007). Changes in body composition and metabolic complications such as dyslipidemia, insulin resistance (Miller *et al.*, 2003; Richter *et al.*, 2005; Hansen *et al.*, 2009) and increased rate of cardiovascular disease (CVD) (Depairon *et al.*, 2001; Baum *et al.*, 2006; Sankatsing *et al.*, 2009) are the most commonly quoted adverse nutritional effects on patients receiving highly active antiretroviral therapy (HAART). Related to this, abdominal obesity as a typically morphological abnormality has been observed among both HIV-positive men and women (Galli *et al.*, 2003; Jaime *et al.*, 2006) in many countries where HAART has been available for a long time. Two recent studies showed that 45.7% (n= 223) of Brazilian HIV+ (Jaime *et al.*, 2006) and 30.7% (n=471) of American HIV infected subjects (Mondy *et al.*, 2007) had central obesity.

It is believed that protease inhibitors (PIs) are the main cause of morphologic changes in the form of fat accumulation (Carr, 2000; Martínez *et al.*, 2001, Saves *et al.*, 2002). Besides, other factors may contribute to fat redistribution including HIV infection related factors like severity and length of HIV infection (Lichtenstein *et al.*, 2001), hypertriglyceridemia and insulin resistance (Martínez *et al.*, 1999; Rodríguez-Guardado *et al.*, 2001) and dietary intake (Hendricks *et al.*, 2003; Jaime *et al.*, 2006).

Abdominal obesity is defined as the localized accumulation of adipose tissue in abdomen irrespective of proportion to total body fat (NHI, 1998). In general, the presence of excess body fat around certain parts of the body, especially around the abdominal area is considered a risk factor for certain diabetes, metabolic syndrome and cardiovascular disease (Kannel *et al.*, 1991, Denke *et al.*, 1993; Insel *et al.*, 2007).

Presently waist-hip circumference ratio (WHR) is not so often used for evaluating central adiposity in the general population and in its place waist circumference is the most widespread anthropometric measurement used. However waist hip ratio (WHR) is useful in detecting possible signs of excess fat deposition (lipodystrophy) in those infected with the HIV (Florindo *et al.*, 2004; Dolan *et al.*, 2005; Jaime *et al.*, 2006).

The present study is aimed at estimating the prevalence of abdominal obesity and comparing the significantly associated factors between those with had abdominal obesity with those who did not. The study was also designed to predict those factors that have the potential to contribute to the development of abdominal obesity in PLHIV receiving HAART. This is the first study on abdominal obesity among PLHIV in Malaysia.

2. Materials and Methods

2.1 Study design and sampling method

Study subject involved a total of 340 adult HIV diagnosed patients aged 20 years or older undergoing treatment with at least one ARV drug, proportionally sampled from all PLHIV who were initially investigated between February and September, 2008, at Hospital Sungai Buloh, Malaysia. The missing data related to anthropometric measurements were the result of some conditions during clinical examinations including pregnancy (4 cases), inability to measure height and weight (2 cases) due to inappropriate position of subjects (sitting or lying down and not able to stand up).

Finally, the waist and hip measurements of 334 of studied population were measured for the calculation of waist-hip ratio (WHR). Patients were recruited when they were at the hospital for their normal follow-up. Patients who were undergoing treatment with b-blockers, diuretics, steroids, oral contraceptive pills, lipid-lowering and hyperglycemic agents, corticoids, anabolic steroids or growth hormones before and during ART as well as those who had active opportunistic diseases (infections or tumors) in the six months prior to the study as well as non Malaysians were also excluded. The process of sampling was applied on respondents' records. Before selecting respondents' records, the investigator used a sampling fraction in each stratum that was proportional to that of the total population. The investigator determined the total HIV respondents receiving antiretroviral medication till February 2008 at the infectious diseases clinic at Hospital Sungai Buloh, and then calculated the percentage in each ethnic group and gender. In order that the sample was well represented from the standpoint of gender and ethnicity a two-stage proportional stratified sampling method was applied with the Chinese making up 64.7% (216 patients), and the Malay and Indians each making up 24.9% (83patients) and 10.4% (35 patients) respectively. Meanwhile, 79.9% (267 patients) were male and 20.1% (67 patients) females.

2.2 Data collection

The data collection was conducted in 29 weeks between February and September, 2008, at Hospital Sungai Buloh, Malaysia. A bi-lingual structured questionnaire (in Malay and English) was developed to obtain the information about socioeconomic status (gender, ethnicity, age, employment and educational level, monthly household income), medical history and health status (duration of HIV infection, current supplementation, history of drug abused, CD4 cell count and HIV viral load at start of medication and at time of the study) ARV regimen (line, length of time on ARV and exposure to AZT, d4T, PI). Informed written consents were obtained from the respondents by two trained assistants prior to taking measurements (all done by the principal investigator to reduce error) and conduct of interview.

Weight (kg) was taken using the Tanita weighing scale that was calibrated weekly and height (m) measures using a SECA body meter. Two weight and height measurements were taken and the average recorded. Body mass index (BMI, kg/m²) was calculated according to the World Health Organization (WHO) criteria for classification (WHO, 2000b).

Body composition including total fat and total fat-free mass (lean mass) were measured using bioelectric impedance analyzer (BIA) BODYSTAT[®]1500. In accordance to the instructions in its manual, respondents were told the following; (a) not to eat or drink four hours before the test, (b) avoid heavy exercise 12 hours before testing, (c) to not drink alcohol within 24 hours of the test, (d) empty bladder completely prior to testing and (e) to avoid taking diuretics prior to testing unless instructed by the physician. Because all respondents had also to fast overnight (12 to 14 hours) for blood to be taken for various tests and therefore it was the best opportunity for the researcher to carry out the BIA at the same time.

Both waist and hip circumferences were measured using the SECA[®] (SECA, Germany) non stretchable tape to the nearest 0.1 centimeter. Waist circumferences were obtained by measuring the distance around the smallest area below the rib cage and above umbilicus (belly button). Hip circumference measurements were taken at the point yielding the maximum circumference over the buttocks with the tape in a horizontal plane, touching but not compressing the skin. The measurements were carried out twice, and then the average of the two readings was recorded as the final reading. All measurements were taken by the researched herself.

Abdominal obesity was defined as waist-to-hip ratio greater than 0.90 for men and 0.85 for women (WHO, 1998). Generally male waist circumference equal to or more than 102 cm and female waist circumference equal to or more than 88 cm were considered unhealthy (WHO, 1998).

Blood pressure was measured using digital blood pressure machine model (General Electric, DINAMAP ProCare 120). Blood pressures of respondents were measured in a sitting position on the right upper arm after a rest of a few minutes in the infectious disease clinic.

Biochemical assessment in adults included laboratory measurements of fasting lipid profile [Total Cholesterol (TC), LDL- Cholesterol (LDL-C) and HDL-Cholesterol (HDL-C)], fasting plasma glucose (FPG), CD4 cell count and HIV RNA load. All biochemical parameters were obtained from the computerized medical records of patients. The CD4 cell counts were categorized according to the standard of CENTRE FOR DISEASE CONTROL AND PREVENTION (CDC, 1993). Tests for HIV-RNA viral load was used to determine HIV RNA load during ARV medication and then classified using WHO (2006) references into undetectable level (value below 50 copies/mL) and detectable level (as equal or greater than 50 copies/mL).

The 24-hour dietary recall method of data collection required individuals to remember the specific foods and amount of food (macronutrient, micronutrient and total energy) they consumed in the past twenty-four hours. Detailed descriptions of all foods and beverages consumed including cooking methods were taken into account. Information such as ingredients of the cooked food and amount of raw food used in cooking were also obtained. Quantities of food consumed were estimated using household measures and were later entered into the data sheet. The twenty-four hour recalls were carried out over two days with one on a weekday and one on the weekend. Data obtained were analyzed based on the Malaysian food database using Nutritionist Pro software (First Data Bank, 2005). Malaysian food composition tables (Tee *et al.*, 1997) were applied to measure the quantity of some food. If cooked dishes were not included in the database of Nutritionist Pro[™] Software, Malaysian Food Composition database (Tee *et al.*, 1997) were used to identify recipe for each dish and then the quantitative information was entered to Nutritionist Pro software. The average energy and nutrient content of the two days 24-hour dietary recall were used for statistical analysis. The dietary variables studied included consumption of energy (in kcal), macronutrients (in g and % energy), sodium and potassium.

2.3 Data analysis

Statistical analysis was performed using SPSS statistical software (version 16.0). The association between abdominal obesity and demographic, clinical and anthropometric variables was assessed using the Chi-square test (categorical variables) and Independent Sample t-test (continuous variables) and Fisher's Exact test.

A multiple logistic regression model was used to predict the risk or protective factors for abdominal obesity adjusting for potential confounders, such as gender, age, ethnicity, years of education, ever abused drug (yes/no), line of ARV regimen, monthly household income (in Ringgit Malaysia- RM), length of time on ARV (Months), BMI (kg/m²) at start of medication and at the time of the study, CD4 cell count at the time of study and triglyceride, fasting plasma glucose (mmol/L), body fat mass (%), energy intake (kcal), protein intake (g), carbohydrate intake (g), fat intake (g), sodium (mg), potassium (mg), % energy from protein, % energy from carbohydrate and % energy from total fat. The estimates were presented as odds ratio (OR) with a 95% confidence interval (95% CI). Guidelines provided had stipulated that total carbohydrate intake should contribute 55-70%, total fat 20-30%, and protein 10-15% of total daily energy intake for the Malaysian adult population (NCCFN, 2005). Total energy requirements were increased by 10 percent (WHO, 2003) over the level of energy intake recommended for healthy non- HIV-infected persons of the same age (adults), sex, and physical activity level for asymptomatic patients in stage 1 AIDS (WHO, 2006).

2.4 Ethical issue and Consent Form

Ethical considerations and approvals to conduct the study were obtained from the following list of individuals/committees: (1) Medical Research Ethics Committee of the Faculty of Medicine and Health Science, University PUTRA Malaysia, (2) The director, Hospital Sungai Buloh, (3) The director, Hospital Selayang, (4) Clinical Research Center (CRC) of the Ministry of Health Malaysia, (5) National Institute of Health (NIH) Malaysia and (6) Ministry of Health Malaysia.

All participants were fully informed about the purpose of the study. Their anonymity was maintained by asking participants not to write their names on the questionnaire. Confidentiality was maintained by not identifying any responses and by reporting the information in a summary form. In addition, their participation in the study was on a voluntary basis, they can withdraw from the study at any time without affecting their treatment. All the respondents filled and signed a consent form before they were interviewed.

3. Results

3.1 Prevalence of abdominal obesity

Prevalence of abdominal obesity according to waist circumference and waist hip ratio is presented in Table 1. The overall prevalence of abdominal obesity based on waist hip ratio was 36.5%. The comparison of percentage distribution by characteristics revealed that a significantly higher percentage of Indians and Chinese, respondents who had no history of drug abuse, those currently exposure to a protease inhibitor (PI) agent, those respondents that were overweight/obese at start of medication or at the time of study had abdominal obesity (Table 2).

The comparison with the background of respondents as shown in Table 2 revealed that the respondents with abdominal obesity had significantly higher ($p < 0.05$) mean age, blood triglycerides, fasting plasma glucose, HDL-cholesterol, BMI at the start of medication and at the time of the study, waist circumference, waist hip ratio, body fat mass, systolic and diastolic blood pressure and also lower mean CD4 cell count at start of medication and body lean mass than those without abdominal obesity.

The comparison of mean dietary intakes for patients with and without abdominal obesity is presented in Table 3. There were no significant differences in average dietary intake of various nutrients between two groups.

As shown in Table 4, the results of the final logistic regression models for selected variables found that before adjusting for covariates, age (unadjusted OR=1.050, CI=1.023-1.078), current exposure to PI (unadjusted OR=2.237, CI=1.212-4.129), higher triglycerides (unadjusted OR=1.192, CI=1.032-1.378), higher fasting plasma glucose (unadjusted OR=1.292, CI=1.119-1.491), higher BMI at start of medication (unadjusted OR=1.207, CI=1.128-1.291), higher BMI at the time of study (unadjusted OR=1.319, CI=1.214-1.434), higher BMI at the time of observation (unadjusted OR=1.044, CI=1.012-1.078) and higher body fat mass (unadjusted OR=1.044, CI=1.012-1.078) significantly increased the risk of abdominal obesity. In contrast being Malay protected (unadjusted OR=0.471, CI=0.266-0.834) them against abdominal obesity. After adjusting for the covariates, a significant higher risk of abdominal obesity were observed in those who were older (adjusted OR=1.053, CI=1.012-1.095), those who had higher fasting plasma glucose (adjusted OR=1.189, CI=1.014-1.394), those with higher BMI at the time of observation (adjusted OR=1.426, CI=1.215-1.674) and being Malay was a protective factor (adjusted OR=0.264, CI=0.102-0.685) for abdominal obesity.

4. Discussion

This study revealed that WHR is a more accurate anthropometric predictor of abdominal obesity than WC confirming studies by Florindo *et al.* (2004) and Jaime *et al.* (2006). Florinda and colleagues (2004) using computerized tomography of the abdomen (CTA) found that visceral fat had better association with the measurement of WHR ($r=0.74$; $p=0.009$) than with the measurement of WC ($r=0.60$; $p=0.050$) among men ($n=10$) and women ($n=5$) in Brazil. The main reason for this is the fact that PLHIV on medication experience morphological alterations including reduction in hip circumference due to lipoatrophy along with increase in waist circumference as a result of subcutaneous and/or visceral fat accumulation

Similarly, a high incidence of centralized distribution of adiposity has been observed among PLHIV under treatment with ARV regimen, (Schwenk *et al.*, 2001; Dolan *et al.*, 2005; Jaime *et al.*, 2006; Sutinen & Yki-Järvinen, 2007). Some previous studies (Miller *et al.*, 1998; Carr, 2000; WHO, 2006) had attributed the increased risk of abdominal obesity mainly to the prescription of PIs.

Morphological abnormalities observed in subjects receiving ARV has been found to differ by gender as well as race/ethnicity. Some study of positive HIV infected subjects under ARV medication showed that abdominal fat and mean WHR/WC were higher in HIV positive men (Jacobson *et al.* 2005; Shah *et al.*, 2005) or among HIV infected women (Jamie *et al.* 2006). In this study abdominal obesity was more prevalent among the women but the difference was not significant. Fasting hyperinsulinemia (Hadigan *et al.*, 1999) in human immunodeficiency virus-infected women with the increased abdominal fat may be the possible explanations for tendency to progress an android body formation characterized by increased trunk fat. The finding of this research revealed that abdominal obesity was less prevalent among Malay and Chinese. Also, A number of researches revealed that Caucasian HIV positive women (Bausserman *et al.*, 2004), African-American women and White men (Shen *et al.*, 2006) had higher WC. Similarly, Kee *et al.* (2008) reported that among 32,900 Malaysian subjects, the prevalence of abdominal obesity was higher among the Indian (28.2%) than Malays (18.6%) and Chinese (14.1%). It may be a reason for higher incidence of abdominal obesity among studied HIV subjects that was influenced by race/ethnicity differences in general population (Kee *et al.*, 2008).

This study revealed that PLHIV on ARV experienced an increase in central adiposity with age confirming the findings of Martinez *et al.* (2001), Jaime *et al.* (2006). WHO (2006) also made mention of this in their guidelines on ARV for PLHIV. PLHIV as a relatively small portion of general population may experience the similar consequences of aging including deficiencies in growth hormone, dehydroepiandrosterone, testosterone and, decline in resting metabolism that may be accompanied with weight gain (Racette *et al.*, 2003). Another factor that was associated with prevalence of abdominal obesity was CD4 cell count at the start of medication confirming the study by Mallon *et al.* (2003) and WHO (2006).

Higher LDL-cholesterol (Mallon *et al.*, 2003), triglycerides (Kosmiski *et al.*, 2001; Mallon *et al.*, 2003; Guimarães *et al.*, 2007), and lower HDL cholesterol (Kosmiski *et al.*, 2001; Fessel *et al.*, 2002; Mallon *et al.*, 2003; Scherzer *et al.*, 2008), FPG (Meininger *et al.*, 2002; Guimarães *et al.*, 2007) in blood were observed in patients with more abdominal adiposity. Similarly, Meininger *et al.* (2002) demonstrated that increasing in WHR (CI = 18.6-136.1; $p = 0.011$) is associated with incidence of fasting hyperinsulinemia (as predictor of elevated plasma glucose).

Joy *et al.* (2008) using linear regression model in the USA found that the relationship between BMI and abdominal fat accumulation was linear ($p < 0.0001$). In the other words, increase in BMI was accompanied with the increase in abdominal obesity. In terms of abdominal obesity, Jacobson *et al.* (2005) using multivariate analysis in USA found that greater body fat mass (relative risk (RR) = 3.1, CI = 1.4-6.7; $p < 0.005$) was associated with the risk of fat deposition in abdomen and the respondents with higher percentage of body fat had more central fat deposition (waist-to-hip ratio of > 0.95 cm for men and of > 0.85 cm for women).

In addition, it was noted that increased WC, WHR and abdominal obesity occurred more frequently in individuals with hypertension, expressed by increased systolic and/or diastolic blood pressure (Sattler *et al.*, 2001; Jung *et al.*, 2004; Guimarães *et al.*, 2007; Nyamdorj *et al.*, 2008).

Contrary to some studies (Hendricks *et al.*, 2003; Jaime *et al.*, 2006), there were no significant difference in dietary intake between HIV infected subjects with and without abdominal obesity in this study. It seems that dietary factors did not play a key role in occurrence of this morphological abnormality.

One limitation of this study is that it is based on a cross-sectional design and the availability of longitudinal follow-up data can further explain and confirm the association of abdominal obesity with other factors. Another limitation was the fact that the result of this study only indicates abdominal obesity and nutritional factors of

PLHIV receiving antiretroviral medication at Hospital Sungai Buloh and therefore it cannot be generalized to all PLHIV in Malaysia. In spite of the advantages of using bioelectrical impedance analysis (BIA), it cannot measure regional body composition and thus was not used to measure abnormal body-fat deposition and this is a BIA measurement limitation. Consequently, the more advanced and precious medical instruments such as DEXA (Dual-Energy X-Ray Absorptiometry), MRI (Magnetic Resonance Imaging) or CT scan (Computed Tomography) are recommended. Since this study focused on fat deposition as abdominal obesity, these findings cannot be applicable to regional fat loss or fat atrophy.

The investigation on the incidence of morphological changes as fat atrophy and fat accumulation should be further studied to determine the prevalence of lipodystrophy. The estimated dietary intakes in this study were based on 24-hour recall and despite using trained interviewers inaccurate estimates and underreporting of food intake recall by respondents with little or no education was another limitation. Dietary data collected by 24-hour recall may be less accurate than food record data due to the inability of memory recall by older respondents as a result of their mental cognitive impairment (Gauthier *et al.*, 2006), underreporting of consumed food by the obese due to desirability to eliminate unhealthy food such as fat (Tooze *et al.*, 2007) and poorly educated respondents that may not reflect usual intake.

5. Conclusion

As a result of some complications associated with HIV infection and ARV medication including metabolic and morphological abnormalities that interfere with health status and quality of life of this population, further studies are needed to evaluate the impact of modifiable and preventive programs on morphologic and metabolic complications, their risk factors and risk of future chronic diseases in HIV infected subjects.

Acknowledgments

We thank the Department of Nutrition and Dietetics and also Faculty of Medicine and Health Sciences University Putra Malaysia, Hospital Sungai Buloh, Hospital Selayang, Clinical Research Center (CRC), National Institute of Health (NIH) and Ministry of Health Malaysia for approval to conduct the study. This study was funded by Malaysian AIDS Council (MAC) under the Ministry of Health, Malaysia, year 2006 grant. Special thanks also to Dr. Christopher Lee who accepted my master research project and provided me with the best research environment and support at Hospital Sungai Buloh.

I wish to thank Dr. Adeeba Kamaruzaman, President of the Malaysian AIDS Council (MAC) who assisted me in getting the funding and members of her staff, especially Miss. Ines Yap, Director, Evaluation & Development Division and Mrs. Sivakami Visvalingam Senior Executive, Programme Development Department for their continuous support throughout research. I was so privileged to work with HIV infected people at Hospital Sungai Buloh who willingly participated in this study. Without their patience and cooperation I would not have been able to conduct this research.

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Table 1. Prevalence of Abdominal Obesity According to Waist Circumference and Waist Hip Ratio
(N=334)

Abdominal Obesity	Waist Circumference ^a (n=334)	Waist Hip Ratio [§] (n=334)
Yes	15 (4.5)	122 (36.5)
No	319 (95.5)	212 (63.5)

^a Abdominal obesity: Female WC \geq 88 cm and Male WC \geq 102 cm (WHO, 1998)

[§] Abdominal obesity: Female WHR $>$ 0.85 and Male WHR $>$ 0.9 (WHO, 1998)

Table 2. Population characteristics according to the presence of abdominal obesity (n = 334)

Characteristic	Abdominal Obesity No. (%)		*P-Value
	No (n=212)	Yes (n=122)	
Abdominal Obesity			
Gender			
Male	172 (64.4)	95 (35.6)	
Female	40 (59.7)	27 (40.3)	0.473 ^a
Ethnicity**			
Chinese	129 (59.7)	87 (40.3)	
Malay	63 (75.9)	20 (24.1)	
Indian	20 (57.1)	15 (42.9)	0.024 ^a
Educational Level (Years)			
No Formal Education	4 (57.1)	3 (42.9)	
1-9	123 (62.1)	75 (37.9)	
=>10	85 (65.9)	44 (34.1)	0.725 [§]
Employment Status			
Employed (Full and Part Time)	128 (65.6)	67 (34.4)	
Unemployed	72 (60.0)	48 (40.0)	
Self employed	12 (63.2)	7 (36.8)	0.600 ^a
Monthly Household Income			
RM < 1000	95 (65.1)	51 (34.9)	
RM 1000-2000	66 (64.7)	36 (35.3)	
=> RM 2000	51 (59.3)	35 (40.7)	0.646 ^a
Duration of HIV Infection (Years)			
1-5	98 (62.4)	59 (37.6)	
6-10	74 (60.2)	49 (39.8)	
=>10	36 (75.0)	12 (25.0)	0.182 ^a
Current Supplementation			
No	149 (60.8)	96 (39.2)	
Yes	63 (70.8)	26 (29.2)	0.094 ^a
Ever Drug Abused**			
No	167 (60.1)	111 (39.9)	
Yes	145 (80.4)	11 (19.6)	0.004
CD4 Cell Count at Start of Medication			
< 200 cells/mm ³	145 (62.0)	89 (38.0)	
200-499 cells/mm ³	50 (76.9)	15 (23.1)	
=> 500 cells/mm ³	4 (80.0)	1 (20.0)	0.052 [§]
CD4 Cell Count at Time of Study			
< 200 cells/mm ³	36 (17.0)	22 (72.9)	
200-499 cells/mm ³	112 (52.8)	68 (47.2)	
=> 500 cells/mm ³	64 (30.2)	31 (69.8)	0.674 ^a
Viral Load at Start of Medication			
< 100,000 copies/mL	76 (70.4)	32 (29.6)	
≥ 100,000 copies/mL	62 (62.6)	37 (37.4)	0.238 ^a
Viral Load at Time of Study			
< 50 copies/mL	185 (64.0)	104 (36.0)	
≥ 50 copies/mL	27 (60.0)	18 (40.0)	0.603 ^a
Length of Time on ARV (Months)			
< 6	8 (50.0)	8 (50.0)	
≥ 6	201 (64.0)	113 (36.0)	0.257 ^a
Line of Antiretroviral Regimen			
First Line	110 (67.1)	54 (32.9)	
Changed First Line	101 (60.1)	67 (39.9)	0.188 ^a

Exposure to AZT					
No	103 (62.1)		54 (37.9)		
Yes	105 (65.6)		64 (34.4)		0.514 ^a
Exposure to d4T					
No	108 (62.1)		66 (37.9)		
Yes	100 (65.8)		52 (34.2)		0.486 ^a
Exposure to PI**					
No	188 (66.4)		95 (33.6)		
Yes	23 (46.9)		26 (53.1)		0.009 ^a
BMI at Start of Medication **					
Underweight (BMI < 18.50 Kg/m ²)	56 (82.4)		12 (17.6)		
Normal (BMI 18.5 – 24.99 Kg/m ²)	136 (65.4)		72 (34.6)		
Overweight/Obese (BMI ≥ 18.5–24.99 Kg/m ²)	20 (34.5)		38 (65.5)		0.0001
	Mean; Standard deviation				
Age (Years) ***	40.66;	8.73	44.44;	8.68	0.0001
Number of years of formal education (Years)	7.96;	4.71	7.76;	4.26	0.692
Household Income Per Month (RM)	1,143.07;	1,060.08	1,311.35;	1,570.14	0.293
Duration of HIV Infection (Years)	6.10;	4.14	5.67;	3.48	0.346
CD4 cell count at Start of Medication (cells/mm³) ***	129.42;	119.88	97.73;	99.53	0.015
CD4 cell count at the Time of Study (cells/mm³)	402.02;	222.10	403.21;	220.29	0.963
Viral Load at Start Medication (copies/mL)	269,741.83;	695,459.80	412,087.42;	959,751.22	0.225
Viral Load at the Time of Study (copies/mL)	780.30;	5,894.72	2,491.73;	20,002.51	0.246
Length of Time on ARV (Months)	42.41;	33.77	45.92;	34.12	0.364
Total Cholesterol (mmol/L)	5.63;	1.24	5.75;	1.11	0.385
LDL-cholesterol (mmol/L)	3.41;	1.12	3.47;	1.10	0.646
HDL-cholesterol (mmol/L)***	1.20;	0.37	1.08;	0.33	0.005
Triglycerides (mmol/L)***	2.23;	1.57	2.68;	1.60	0.014
Fasting Plasma Glucose (mmol/L)***	5.34;	1.65	6.28;	2.48	0.0001
BMI at the Start of Medication (Kg/m²)***	20.52;	3.21	23.24;	4.72	0.0001
BMI at the Time of Study (Kg/m²)***	21.02;	3.11	24.04;	3.82	0.0001
Waist Circumference(cm)***	75.83;	7.29	87.17;	8.52	0.0001
Hip Circumference (cm)***	90.32;	6.64	91.95;	7.91	0.044
WHR (ratio)***	83.91;	4.40	94.83;	4.92	0.0001
Body Fat Mass Percentage (%)***	20.65;	6.85	22.83;	7.26	0.006
Body Lean Mass percentage (%)***	79.44;	6.95	77.19;	7.23	0.005
Systolic Blood Pressure (mm Hg)***	126.75;	18.80	132.78;	19.45	0.006
Diastolic Blood Pressure (mm Hg)***	75.10;	11.24	79.03;	11.97	0.003

Statistically significant difference (**Chi-square test for frequencies, ***Independent sample t-test for means), * $p < 0.05$.

^a Chi-square test for frequencies, [§] Fisher's Exact Test.

Table 3. Dietary intake of the population according to the presence of abdominal obesity

(n = 334)

Characteristic	Abdominal obesity				*P-Value
	No (n=212)		Yes (n=122)		
	Mean;		Standard deviation		
Energy (kcal)	1461.06;	416.76	1414.99;	344.34	0.277
Protein (g)	67.45;	24.61	66.33;	24.07	0.688
Carbohydrate (g)	172.27;	46.67	167.33;	36.48	0.284
Total Fat (g)	55.85;	23.53	53.12;	20.25	0.266
Sodium (mg)	1966.35;	883.63	2080.58;	758.59	0.232
Potassium (mg)	1466.82;	1019.66	1302.56;	449.23	0.093
% Energy from Protein	18.39;	3.82	18.61;	4.22	0.629
% Energy from Carbohydrate	48.02;	8.06	48.28;	8.25	0.779
% Energy from Fat, total	33.56;	6.80	32.85;	7.35	0.369

Statistically significant difference (Independent sample t-test for means): * $p < 0.05$.

Table 4. OR (95% CI) for the abdominal obesity from logistic regression analysis for selected variables

Characteristic	Unadjusted OR (95% CI)	P-Value	Adjusted OR (95% CI)	P-Value
Age	1.050 (1.023-1.078)	0.0001*	1.053 (1.012-1.095)	0.010*
Ethnicity				
Chinese	1.00	0.027*	1.00	0.023*
Malay	0.471 (0.266-0.834)	0.010*	0.264 (0.102-0.685)	0.006*
Indian	1.112 (0.540-2.291)	0.773	0.593 (0.188-1.871)	0.372
Ever Abused Drugs				
No				
Yes	0.368 (0.182-0.742)	0.005*	0.610 (0.228-1.637)	0.327
Current Exposure to PI				
No	1.00		1.00	
Yes	2.237 (1.212-4.129)	0.010	2.112 (1.108-3.821)	0.216
Triglycerides	1.192 (1.032-1.378)	0.017*	1.033 (0.854-1.250)	0.738
Fasting Plasma Glucose	1.292 (1.119-1.491)	0.0001*	1.189 (1.014-1.394)	0.033*
BMI at Start of Medication	1.207 (1.128-1.291)	0.0001*	0.986 (0.877-1.109)	0.817
BMI at the Time of Study	1.319 (1.214-1.434)	0.0001*	1.426 (1.215-1.674)	0.0001*
Body Fat Mass (%)	1.044 (1.012-1.078)	0.007*	1.002 (0.936-1.072)	0.962

Adjusted for gender, age, ethnicity, years of education, ever abused drug (yes/no), line of ARV regimen, monthly household income (in Ringgit Malaysia- RM), length of time on ARV (Months), BMI (kg/m²) at start of medication and at the time of the study, CD4 cell count at the time of study and triglyceride, fasting plasma glucose (mmol/L), body fat mass (%), energy intake (kcal), protein intake (g), carbohydrate intake (g), fat intake (g), % energy from protein, % energy from carbohydrate, % energy from total fat.