

Clinical Characteristics and Risk Factors of Dyslipidemia in AIDS Patients Treated with ART

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Abstract

Background: The incidence of dyslipidemia in patients with AIDS and antiviral therapy was relatively high. Patients with AIDS and antiviral therapy with dyslipidemia have a high risk of vascular disease emergencies, which seriously threatened the life and health of patients, and the pathogenesis and drug safety of have attracted much attention. **Objective:** To explore the prevalence and risk factors of dyslipidemia in AIDS patients treated with antiviral therapy, so as to provide basis for improving the safety of antiviral drugs, avoiding the occurrence of vascular disease emergencies, and ensuring the long-term health of patients. **Methods:** Total cholesterol (TC) and triglyceride (TG) were measured in 161 patients with AIDS after antiviral therapy for 1, 2 and 3 years. TC ≥ 5.72 mmol/l or TG ≥ 2.58 mmol/l were included in the observation group, and TC < 5.72 mmol/l or TG < 2.58 mmol/l were included in the control group. The blood pressure, arterial plaque, CD4⁺T, CD8⁺T, viral load (HIVL), leukocyte (WBC), hemoglobin (HB), platelet (PLT), bilirubin (TBIL), alanine aminotransferase (ALT), aspartate aminotransferase (AST), blood creatinine (BCR), TC, TG and blood glucose (BG) levels of the two groups were compared, and the influencing factors were calculated by Logistic regression. **Re-**

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sults: The dyslipidemia rate of AIDS patients treated with antiviral therapy was 46.6% (75/161) for one year, 60.2% (97/161) for two years and 76.4% (123/161) for three years. The longer the course of therapy, the higher the rate of dyslipidemia ($\chi^2 = 30.168$, $P < 0.01$) and the average rate of dyslipidemia in patients who have been treated was 61.1% (295/483) for three years. Male was higher than female for the characteristics of patients with dyslipidemia (83.0% vs 68.5%, $\chi^2 = 4.628$, $P < 0.05$). Patients with hypertension were higher than without hypertension (93.7% vs 65.3%; $\chi^2 = 17.087$, $P < 0.05$). Patients with arteriosclerosis were higher than without arteriosclerosis (97.4% vs 69.7%; $\chi^2 = 12.633$, $P < 0.05$). The levels of AST and BG in dyslipidemia patients were higher than those in normal lipidemia patients. HIVL levels are lower than the normal lipid group. Logistic analysis showed that gender, hypertension and arteriosclerosis were the influencing factors of dyslipidemia in AIDS antiviral therapy. Among AIDS patients treated with antiviral therapy, patients with dyslipidemia have 5.2 times higher for hypertension, 4.4 times higher for arteriosclerosis, and 0.5 times lower for female. **Conclusion:** Patients receiving antiretroviral therapy for HIV/AIDS have a high prevalence of dyslipidemia; the prevalence is particularly high among male patients, those hypertension, and those with arteriosclerosis, and increases with the duration of treatment. For HIV/AIDS antiretroviral therapy, it is essential to actively control patients' pressure and lipid levels, prevent the occurrence of vascular events, and ensure their long-term health.

Keywords

AIDS, Antiviral Therapy, Dyslipidemia, Clinical Characteristics, Risk Factors

1. Introduction

AIDS patients who have received Anti Retrovirus Therapy (ART) for a long time have a relatively high rate of abnormal blood lipids, which leads to a high risk of vascular events such as coronary heart disease, myocardial infarction, arterial dissection, cerebral hemorrhage and cerebral thrombosis and the combination of chronic vascular diseases has become one of the main causes of death for AIDS patients [1]. A number of research results on the burden of chronic diseases such as dyslipidemia caused by long-term anti retrovirus therapy (ART) in patients with AIDS showed at the Conference on Retroviruses and Opportunistic Infections (CROI, 2021) that dyslipidemia seriously threatened the life and health of patients with AIDS [2]. ARTDA refers to a clinical syndrome with increased total cholesterol (TC) or (and) triglycerides (TG) during ART, which may cause potential damage to the health of patients. At present, it is believed that HIV infection, ART and ARTDA are all related, but its occurrence and development mechanism has not been fully explored [3]. Lipids are transported to tissues through apolipo-

proteins, and enter cells for metabolism through lipoprotein receptor-mediated endocytosis. It is generally believed that the imbalance of metabolism in the synthesis and utilization of lipids can lead to lipid disorders, and play a critical role in the occurrence and development of chronic vascular diseases [4]. ART requires lifelong treatment, dyslipidemia could lead to inflammatory lesions in the vascular wall of small and medium-sized arteries, fibrosis, endothelial hyperplasia, thrombosis, vascular deformation and narrowing or aneurysm formation, endanger the lives of patients, and the prevention and early warning mechanism of ARTDA has attracted much attention [5]. TC and TG were measured in 161 patients treated with ART for 1, 2 and 3 years respectively to explore the clinical characteristics and risk factors of dyslipidemia in patients treated with ART. TC $\geq$ 5.72 mmol/l or TG $\geq$ 2.58 mmol/l were included in the observation group. TC $<$ 5.72 mmol/l or TG $<$ 2.58 mmol/l were included in the control group. The blood pressure, arterial plaque, CD4⁺T, CD8⁺T, viral load (HIVL), leukocyte (WBC), hemoglobin (HB), platelet (PLT), bilirubin (TBIL), alanine aminotransferase (ALT), aspartate aminotransferase (AST), blood creatinine (BCR), TC, TG and blood glucose (BG) levels of the two groups were compared, and the influencing factors were calculated by Logistic regression. The results are as follows.

2. Materials and Methods

2.1. Materials

2.1.1. Instruments

1) HIV load analyzer was completed with Versant 440 molecular system. 2) CD4⁺T and CD8⁺T was counted with BD FACS Cali bur GH0156 flow cytometer. 3) Blood cell analysis was completed with Mindray BC-5000 automatic blood cell analyzer. 4) The analysis of liver and kidney function, glucose and lipid metabolism function was completed with HITACHI 7600-020 Automatic Analyzer. 5) Aloka- α 10 imaging system with probe frequency of 7.5 MHz for carotid ultrasound examination.

2.1.2. Reagents

1) Human Regulatory T cell Staining Kit-PE Foxp3 PCH101、FITC CD4, provided by Wuhan Antgene Biotechnology Co., Ltd. 2) Becton Dickinson and company BD Biosciences was produced according to YZB/USA 3550-2013 standard, and the packaging specification was 20 L. Provided by BD Medical Devices (Shanghai) Co., Ltd.

2.1.3. Blood Collection Method

Avoid high-fat diet, overnight food and alcohol consumption at dinner the day before blood sampling, fasting for $\geq$ 10 hours after dinner, and getting up in the morning the next day free-time activities for $\geq$ 30 minutes before blood sampling.

1) Took 10 ml of peripheral venous blood with ETDA-K2 anticoagulant tube for HIV load test specimen, and immediately reversed and mixed it for 6 - 8 times

after blood sampling. Centrifuged for 10 minutes (3000 rpm/min) within 6 hours, separated the plasma, divided it into 4 parts, and stored it at -80°C for examination. 2) Took 2 ml of peripheral venous blood from fresh EDTA anticoagulant tubes, and immediately reversed and mixed for 6 - 8 times after blood drawing, set it at 20°C and send it for examination within 8 h for CD4 cell and CD8 cell count test specimens. 3) Used EDTA anticoagulation tube to collect 2 ml of peripheral venous blood, place it at 20°C , and send it for examination within 3 hours for Blood cell analysis sample. 4) 2 ml of peripheral venous blood was collected by ordinary serum tube, placed at 20°C , and sent for examination within 3 hours for analysis of liver and kidney function, glucose and lipid metabolism function. 5) For patients with abnormal increase of cholesterol or triglyceride, repeated the test twice within a week, and took the mean value of the three tests as the observation value.

2.1.4. Pharmaceutical Preparation

1) Tenofovir (TDF), produced by TIGR Pharmaceutical Responsible Co., Ltd., approval number: NMPN H20050803. 2) Lopinavir/ritonavir (LPV/r), produced by Abb Vie China, approval number: H20130693. 3) Lamivudine (3TC), produced by Fujian Sosunter Pharmaceutical Co., Ltd., approval No.: NMPN H20113025. 4) Efavirenz (EVP), produced by Merck sharp & Dohme Pty. Ltd, approval number: 12310H305. 5) Netobimin (NVP), produced by Boehringer Ingelheim Ellas A. E., approval No.: H20110483. 6) Rilpivirine (RPV), produced by Janssen-Cilag SpA, with registration certificate No. H20171075.

2.2. Method

2.2.1. Grouped Objects

Patients who received HAART in Youjiang District People's Hospital of Baise City from January 2009 to June 2019 were approved by Hospital ethical committees (No. KF-100.2039/91, KF-01-144/01, AND H-KF-01-144/01) and institutional review boards, and written informed consent was obtained from all participants. The subjects were those who met the following conditions: 1) Patients have a history of contact with HIV carriers or AIDS patients, or have a history of unsafe sex, intravenous drugs, blood transfusion or blood products. 2) According to the diagnostic criteria of Treatment Guidelines for the Use of Antiretroviral Agents in HIV-Infected Adults and Adolescents: An Update [6], patients with an epidemiological history, HIV antibody positive in the preliminary screening test, and confirmed that the patients with HIV antibody positive in the test were diagnosed as AIDS infected persons or patients with AIDS. 3) There are indications for anti-HIV therapy and no contraindications, and patients receive HAART regimen by clinical evaluation. 4) Patients with severe opportunistic infection or acute attack of previous chronic diseases should be stabilized before ART. 5) Patients with other infectious diseases, immune diseases, cortisol drugs used within half a year or who cannot adhere to continuous medication for ART for other reasons should be excluded. 6) ART according to the scheme in 1.2.3 and measure the levels of

total cholesterol (TC) and triglyceride (TG) 1 year, 2 and 3 years after ART. 7) Grouping method: according to different blood lipid levels, 123 patients with cholesterol ≥ 5.72 mmol/l or triglyceride ≥ 2.58 mmol/l were included in the observation group, and 38 patients with cholesterol < 5.72 mmol/l or triglyceride < 2.58 mmol/l were included in the control group. Blood pressure, arterial plaque, CD4⁺T, CD8⁺T, viral load (HIVL), white blood cell (WBC), hemoglobin (HB), platelet (PLT), total bilirubin (TBIL), alanine aminotransferase (ALT), aspartate aminotransferase (AST), blood creatinine (BCR), TC, TG and blood glucose (BG) levels were compared between the two groups after 3 years of therapy.

2.2.2. Carotid Ultrasound Examination

Aloka- $\alpha 10$ imaging system with probe frequency of 7.5 MHz. The participant takes the supine position, extends head slightly backward, turns to the opposite side of the participant, and fully exposes neck. The common carotid artery were examined laterally from the medial end of the clavicle, and then the common carotid artery (CCA), internal carotid artery (ICA), external carotid artery (ECA), vertebral artery (VA) were displayed longitudinally along the outer edge of the sternocleidomastoid muscle. Judgment criteria: I. measure the inner diameter and inner and middle membrane thickness of CCA, ECA and ICA.

2.2.3. Medication Regimen

The benchmark regimen of HAART is 3TC/TDF/EVP which according to Guidelines for the use of antiretroviral agents in HIV-1-infected adults and adolescents (2016) [6]. The patients maintain 3TC/TDF in the baseline protocol, change EVP to LPV/r when relatively severe intolerable drug reactions occur during treatment, for patients with mother infant block, and patients with neuropsychiatric symptoms; change EVP to NVP when CD4 count decrease (≤ 400 μ l for male, ≤ 250 μ l for female) for the first time; and change EVP to RPV when there are patients who are treated with methadone, and the dosage of methadone should be appropriately reduced.

2.2.4. Observation Index and Classification Definition

HIVL normal value range is undetectable or HIVL < 50 (copy/ml), normal range of CD4⁺T is 450 - 1440 (μ l), normal range of CD8⁺T is 320 - 1250 (μ l), normal range of WBC is $(4 - 10) \times 10^9$ /l, normal range of Hb is (110 - 150 for female, 130 - 160 for male) g/l, normal range of PLT is $(100 - 300) \times 10^9$ /l, normal range of TBIL is 1.7 - 17.1 μ mol/l, normal range of latent jaundice is 17.1 - 34.2 μ mol/L, normal range of mild jaundice is 34.2 - 170 μ mol/l, normal value range of ALT is (5 - 40 u/l for male, 5 - 35 u/l for female), normal value range of AST is (0 - 40) u/l, normal range of BCR is (44 - 97) μ mol/l, normal range of TC is (2.85 - 5.71) mmol/l, normal range of TG is (0.45 - 2.56) μ mol/l. For BG, which normal value range of fasting blood glucose is 3.9 - 6.1 mmol/l, normal range of blood glucose 2 hours after meal (or random) is < 7.8 mmol/l, and the range of 6.1 - 7.0 mmol/l is impaired fasting blood glucose, the range of 7.8 - 11.1 mmol/l is impaired blood glucose 2 hours after meal (or random). Fasting blood glucose ≥ 7.0

mmol/l, 2 hours after meal (or random) blood glucose > 11.1 mmol/l is diabetes or diabetes has been diagnosed. Body mass index (BMI) ≥ 25.0 kg/m² [weight (kg)/height (m)²] is obesity. Mean systolic blood pressure ≥ 140 mmHg or mean diastolic blood pressure ≥ 90 mmHg or hypertension has been diagnosed is Hypertension. Carotid artery intima-media thickness (IMT) ≥ 1.2 mm is arteriosclerosis.

2.2.5. Statistical Analysis

The data were processed by SPSS 22.0 software. The counting data were calculated according to the number of observation cases. 161 patients were observed for 3 times, a total of 483 cases. The intensity of clinical features was expressed by percentage (%), and χ^2 test was used for comparison between groups; Normal distribution mean and standard deviation of measurement data ($\bar{x} \pm s$) indicates that the two samples are compared with independent sample t-test, and paired data t-test is used before and after treatment; The mean and standard deviation of non-normal distribution are expressed by (M) and quartile (Q1, Q3), and the two groups are compared by rank sum test. The influencing factors of dependent variables were analyzed by Logistic regression. $P < 0.05$ means the difference is statistically significant.

3. Results

3.1. Dyslipidemia Rate and Continuous Treatment Time of AIDS Patients Treated with ART

161 patients treated with ART, the abnormal blood lipid rate was 46.6% (75/161) after one year of therapy, 60.2% (97/161) after two years of therapy, 76.4% (123/161) after three years of therapy, and the average abnormal blood lipid rate for three years of therapy was 61.1% (295/483). The longer the course of therapy, the higher the rate of dyslipidemia in AIDS patients with ART (46.6% vs 60.2% vs 76.4%, $\chi^2 = 30.168$, $P = 0.000$).

3.2. Dyslipidemia and Gender, Age, Therapeutic Regimen and Metabolism of AIDS Patients Treated with ART

In AIDS patients treated with ART, the rate of dyslipidemia in male was higher than that in female (83.0% vs 68.5%, $\chi^2 = 4.628$, $P < 0.05$). There was no significant difference in the abnormal rate of blood lipids in different age (87.5% vs 76.0% vs 75.0% vs 76.9%; $\chi^2 = 1.185$, $P > 0.05$). There was no significant difference in the rate of dyslipidemia between different therapeutic regimen (77.3% vs 70.0% vs 84.6% vs 50.0%; $\chi^2 = 3.089$, $P > 0.05$) that patients with hypertension was higher than without hypertension (93.7% vs 65.3%; $\chi^2 = 17.087$, $P < 0.05$). Patients with diabetes was higher than without diabetes (90.0% vs 73.3%; $\chi^2 = 3.783$, $P > 0.05$), patients with arteriosclerosis was higher than without arteriosclerosis (97.4% vs 69.7%; $\chi^2 = 12.633$, $P < 0.05$). There was no significant difference in the abnormal blood lipid rate between patients with obesity group and without obesity (87.5% vs 73.6%; $\chi^2 = 2.730$, $P > 0.05$). The dyslipidemia rate of male, hypertensive and

arteriosclerotic ART patients treated with ART was high ($P < 0.05$). As seen in **Table 1**.

Table 1. Dyslipidemia and gender, age, medication regimen and metabolism of AIDS patients treated with ART.

Observation Index	Grading	Abnormal number of blood lipids	Observation Group (%)	Control Group (%)	χ^2	P
Gender	male	88	73 (83.0)	15 (17.0)	4.628	0.031
	female	73	50 (68.5)	23 (31.5)		
Age (year)	<25	8	7 (87.5)	1 (12.5)	0.599	0.897
	25-	104	79 (76.0)	25 (24.0)		
	50-	36	27 (75.0)	9 (25.0)		
	65-	13	10 (76.9)	3 (23.1)		
Medication Regimen	3TC/TDF/EVP	132	102 (77.3)	30 (22.7)	3.089	0.378
	3TC/TDF/LPV	10	7 (70.0)	3 (30.0)		
	3TC/TDF/NVP	13	11 (84.6)	2 (15.4)		
	3TC/TDF/RPV	6	3 (50.0)	3 (50.0)		
Metabolic characteristics	obesity	32	28 (87.5)	4 (12.5)	2.730	0.098
	hypertension	63	59 (93.7)	4 (6.3)	17.087	0.000
	Diabetes	30	27 (90.0)	3 (10.0)	3.783	0.052
	arteriosclerosis	39	38 (97.4)	1 (2.6)	12.633	0.000

3.3. Dyslipidemia and HIVL, CD4⁺T and CD8⁺T Levels in AIDS Patients Treated with ART

Comparison between observation group and control group: HIVL were 0 (0, 60) vs 40.00 (0, 100) [copy/ml, $Z = 3.082$, $P < 0.05$], CD4⁺T were 556.54 ± 278.60 vs 586.74 ± 277.60 (μl , $t = 1.265$, $P > 0.05$) and CD8⁺T were 794.72 ± 470.57 vs 769.45 ± 557.84 (μl , $t = 0.926$, $P > 0.05$). The level of HIVL in the observation group was lower than that in the control group, and the difference was statistically significant ($Z = 3.082$, $P < 0.05$). The levels of CD4⁺T and CD8⁺T had no statistically significant difference ($t = 1.265$, $P > 0.05$ and $t = 0.926$, $P > 0.05$) was compared with the control group and suggested that dyslipidemia in patients with ART has an impact on HIVL, but has little impact on the levels of CD4⁺T and CD8⁺T. As seen in **Table 2**.

Table 2. Dyslipidemia and HIVL, CD4⁺T and CD8⁺T levels in AIDS patients treated with ART.

Observation Index	Observation Group (123)	Control Group (38)	t/Z	P
HIVL [M (Q1, Q3), copy/ml]	0 (0, 60)	40.00 (0, 100)	3.082	0.002
CD4 ⁺ T ($\bar{x} \pm s$, μl)	556.54 ± 278.60	586.74 ± 277.60	1.265	0.585
CD8 ⁺ T ($\bar{x} \pm s$, μl)	794.72 ± 470.57	769.45 ± 557.84	0.926	0.277

3.4. Dyslipidemia, Bone Marrow, Liver and Kidney Functions and Blood Glucose Levels in Patients with AIDS after ART

The levels of AST, BG, TC and TG in the observation group were higher than those in the control group ($P < 0.01$). There was no significant difference in WBC, Hb, PLT, TBIL, ALT and BCR levels between the observation group and the control group ($P > 0.05$). Patients with dyslipidemia had high glucose and lipid levels, mild liver function damage, and bone marrow and renal function were not affected. As seen in **Table 3**.

Table 3. Dyslipidemia, bone marrow, liver and kidney functions and blood glucose levels in patients with AIDS treated with ART.

Observation Index	Observation Group (123)	Control Group (38)	T	P
WBC ($\bar{x} \pm s$, $10^9/l$)	6.53 ± 1.65	6.14 ± 1.53	1.304	0.194
Hb ($\bar{x} \pm s$, g/l)	135.93 ± 22.73	137.19 ± 20.55	0.306	0.760
PLT ($\bar{x} \pm s$, $10^9/l$)	222.25 ± 67.16	201.19 ± 63.46	1.711	0.089
TBIL ($\bar{x} \pm s$, $\mu\text{mol/l}$)	12.43 ± 9.81	11.50 ± 4.67	0.560	0.576
ALT ($\bar{x} \pm s$, u/l)	38.10 ± 22.40	36.46 ± 27.93	0.372	0.750
AST ($\bar{x} \pm s$, u/l)	35.43 ± 13.55	26.60 ± 3.85	3.962	0.000
BCR ($\bar{x} \pm s$, $\mu\text{mol/l}$)	73.53 ± 14.50	72.32 ± 17.55	0.427	0.670
BG ($\bar{x} \pm s$, mmol/l)	6.98 ± 1.21	6.40 ± 0.74	2.814	0.006
TC ($\bar{x} \pm s$, mmol/l)	5.55 ± 1.23	4.69 ± 0.72	4.055	0.000
TG ($\bar{x} \pm s$, mmol/l)	3.65 ± 2.56	1.39 ± 0.31	5.426	0.000

3.5. Logistic Regression Analysis of Dyslipidemia in AIDS Patients Treated with ART

Logistic regression analysis was carried out for the abnormal blood lipid level of patients with AIDS ART as the dependent variable, and gender, age, medication regimen, CD4⁺T, CD8⁺T, HIVL, WBC, PLT, Hb, BCR, obesity, hypertension, diabetes and arteriosclerosis as the independent variables. The results showed that gender, hypertension and arteriosclerosis were the influencing factors of abnormal blood lipid. Compared with the normal blood lipid group, patients in the dyslipidemia group had 5.2 times higher hypertension, 4.4 times higher arteriosclerosis, and 0.5 times lower for female. The age, medication regimen, CD4⁺T, CD8⁺T, HIVL, WBC, PLT, Hb, BCR, obesity and diabetes were not the influencing factors of dyslipidemia. As seen in **Table 4**.

Table 4. Logistic regression analysis of dyslipidemia in patients with aids after ART.

various factors	β	S_x	Wald χ^2	P	OR	95% CI
Gender (F)	-0.647	0.204	10.027	0.002	0.524	(0.351 $\pm$ 0.781)
Patient Age	0.050	0.159	0.098	0.754	1.051	(0.769 $\pm$ 1.437)

Continued

Medication Regimen	0.035	0.130	0.074	0.786	1.036	(0.803 ± 1.337)
CD4 ⁺ T (μl)	0.034	0.192	0.031	0.860	1.034	(0.710 ± 1.506)
CD8 ⁺ T (μl)	-0.053	0.215	0.060	0.806	0.949	(0.623 ± 1.445)
HIVL(copy/ml)	-0.037	0.107	0.119	0.730	0.964	(0.781 ± 1.190)
WBC (10 ⁹ /l)	0.984	0.594	2.746	0.098	2.676	(0.835 ± 8.572)
PLT (10 ⁹ /l)	-0.137	0.244	0.315	0.575	0.872	(0.540 ± 1.407)
HB (g/l)	0.162	0.259	0.390	0.533	1.175	(0.708 ± 1.952)
BCR (μmol/l)	-0.179	0.389	0.211	0.646	0.836	(0.390 ± 1.792)
obesity	-0.415	0.629	0.435	0.510	0.660	(0.192 ± 2.268)
hypertension	1.639	0.443	13.669	0.000	5.151	(2.160 ± 12.283)
Diabetes	-1.114	0.786	2.009	0.156	0.328	(0.070 ± 1.532)
arteriosclerosis	1.473	0.604	5.956	0.015	4.364	(1.337 ± 14.249)

4. Discussion

Lipid material (LM) is an ester and its derivatives generated by the action of fatty acids and alcohols and is a compound insoluble in water and soluble in fat soluble solvents. Very low density lipoprotein cholesterol, low density lipoprotein cholesterol, medium density lipoprotein cholesterol, high density lipoprotein cholesterol, etc. in the blood are lipoproteins that are non-covalently bound by LM and proteins, and are the transportation mode of lipids in the body [7]. LM widely exists in human tissue cells, is not only a nutrient, but also a membrane structure of tissue cells and a component of nerve myelin sheath and has important physiological functions and is indispensable in maintaining life activities [8]. LM is a nutrient which has very limited storage and synthesis in the body and depends on the continuous and balanced acquisition from food every day. All lipid substances in the body restrict each other and maintain the balance of lipid metabolism [9]. The balance of lipid metabolism plays a very important role in life and health. The lipid disorder caused by the metabolic imbalance of lipid substances in digestion, absorption, transport, transformation, storage and other factors can lead to the occurrence and development of atherosclerosis and other diseases, resulting in vascular events that endanger life [10]. Dyslipidemia is caused by congenital or acquired factors of the body, caused by the imbalance of digestion, absorption and excretion. The quality and quantity of LM metabolites in blood and other tissues and organs are abnormal, LM molecules such as CH and TG enter the inner wall of blood vessels and deposit on the wall of blood vessels, narrowing the vascular lumen and promoting atherosclerosis of blood vessels leads to the pathological manifestations that appear play an important role in chronic vascular diseases [11]. When genetics, diet, disease and other factors cause the intake of CH and TG to be greater than consumption, the proportion of LM components in the

body is unbalanced, and patients have lipid metabolism disorders, produce clinical symptoms, induce chronic vascular diseases, and affect health [12]. In patients with dyslipidemia, lipid components such as CH and TG invade the inner wall of blood vessels, which pathological manifestation is that a layer of lipid like millet porridge is deposited on the tube wall, the elasticity of the tube wall decreases, the lumen narrows, and local ischemia and necrosis occur [13]. The evolution process of the disease is the accumulation of lipids and compound sugars, local hemorrhage, thrombosis, fibrous tissue hyperplasia and calcinosis, gradual degeneration and calcification of the middle layer of the artery, systemic blood vessels can be involved, often involving elastic and large and medium-sized muscular arteries, arterial stenosis and obstruction, ischemic necrosis of tissues or organs, vascular dysfunction, and clinical symptoms of hypoxia and ischemia of the blood supply tissue [14].

Studies have shown that most patients with AIDS are accompanied by lipid disorders and chronic vascular damage which mainly manifested as abnormal increase of TC and TG, accompanied by inflammatory lesions in the vascular wall of small and medium-sized arteries, fibrosis, vascular endothelial hyperplasia, thrombosis, vascular deformation and narrowing or aneurysm formation, which are closely related to the infection damage of patients and the lipid abnormalities caused by ART. Due to the ART time of patients with AIDS is relatively long, the potential vascular damage mechanism and the prevention and control early warning have attracted much attention [15]. The clinical application of antiviral program could effectively inhibit virus replication, reduce the infectivity of the virus, promote the immune reconstruction function of patients and increase patient compliance and long-term standardized treatment that the virus discretion can be controlled below the low detection line, the quality of life of patients can be improved, and the life expectancy can be extended, and AIDS gradually changes from a terminal disease to a preventable and treatable chronic disease [16]. However, some adverse side effects caused by long-term use of antiviral therapy are worrisome. According to the data of the CROI (2021), patients receiving antiviral therapy are prone to dyslipidemia, and the incidence of vascular disease and heart damage is relatively high [4]. Many investigations have shown that the incidence of dyslipidemia in patients with AIDS after ART for 1 year is more than 40%, and the incidence of dyslipidemia in patients after treatment for 3 years is more than 65%, and there is a high incidence of atherosclerosis, arrhythmia, heart failure, embolism, sudden cardiac death and other complications [5].

Basic research shown that patients with dyslipidemia and vascular disease caused by AIDS infection or ART, individual genetic genes, living habits, virus infection, antiviral drugs and other factors jointly participate in the process of lipid damage vascular disease and the important feature of lipid damage in patients with AIDS was mainly manifested in lipoprotein regulation disorder [17]. Life science research has been confirmed that Proprotein invertase *Bacillus subtilis* protease (PCSK9), lipoprotein receptors (LDLR, VLDLR and scavenger receptors, etc.),

sterol regulatory element connexins (SREBPs), adaptor proteins (ARH), ATP binding cassette transporter g (ABCG) and cholesterol 7-hydroxylase (CYP7A1) played an important role in the process of lipid damage vascular disease and the main mechanism was related to lipoprotein receptor related binding proteins, sterol regulatory element connexins, and Lipoprotein lipase was related to functional disorders such as inhibition of apolipoprotein degradation [18].

The data of this study showed that the longer the course of treatment, the higher the rate of dyslipidemia in AIDS patients treated with ART ($P < 0.05$) and the rate of dyslipidemia was 46.6% (75/161) in patients treated with ART for 1 year, 60.2% (97/161) in patients treated with ART for 2 years, and 76.4% (123/161) in patients treated with Lipoprotein lipase for 3 years. The epidemic characteristics of dyslipidemia in patients treated with Lipoprotein lipase were as follows: the male was higher than the female ($P < 0.05$), hypertension was higher than non without hypertension ($P < 0.05$), arteriosclerosis was higher than without arteriosclerosis ($P < 0.05$). Logistic analysis showed that gender, course of treatment, hypertension and arteriosclerosis were the influencing factors of dyslipidemia in AIDS ART. Among AIDS patients treated with ART, hypertension in patients with dyslipidemia was 5.2 times higher, arteriosclerosis was 4.4 times higher, and female was 0.5 times lower. The results of this study showed that AIDS patients treated with ART have a high rate of dyslipidemia, and male, hypertension and arteriosclerosis have a high rate of dyslipidemia. The longer the course of treatment, the higher the rate of dyslipidemia.

We believe that for antiretroviral therapy for HIV/AIDS, the risk factors for vascular disease in patients should be actively controlled, the occurrence of events should be prevented and controlled, and the long-term health of patients should be ensured.

Author Contributions

Da Huang and Jindu Li are in charge of the design of the research project, the research work and the thesis writing. Case screening was completed by Caihua Zou. Data collection, collation and thesis writing by Ye Liang and Tianzi Li completed. Specimen collection was completed by Huiquan Lu.

The order of authors in this paper was jointly discussed and determined by all authors before submission, and will not be changed after submission. If change is absolutely necessary, an institutional certificate and a written statement of no objection to the authorship signed by all authors must be presented. The authors of this paper simultaneously meet the following four:

- 1) Participated in the selection and design of the thesis topic, or participated in data analysis and interpretation;
- 2) Drafted revised key theories or other major content in the thesis;
- 3) Able to revise the manuscript according to the editorial board's comments, answer academic questions, and ultimately agree to publication of the paper;
- 4) In addition to being responsible for their own research contributions, agreed

to be responsible for the integrity of all aspects of the research work. Da Huang, Jindu Li: data collection, organization, statistical analysis, plotting, and paper writing; Huiqing Lu, Caihua Zou, Ye Liang, Tianzi Li, etc. were in conceptualization, resources, data management, software, formal analysis, funding acquisition, investigation, visualization, methodology, writing the original draft, project administration, writing review and editing, made rigorous revisions to the final manuscript. This manuscript has been revised by all authors and unanimously agreed to be submitted.

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Conflicts of Interest

There is no conflict of interest between the funding and the conclusion of the paper.

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