©IDOSR PUBLICATIONS International Digital Organization for Scientific Research

IDOSR JOURNAL OF APPLIED SCIENCES 11(2):71-76, 2026 ISSN: 2550-7931

https://doi.org/10.59298/IDOSRJAS/2026/1127176 IDOSRJAS11200

Antiretroviral-Induced Metabolic Dysregulation: Pathways and Long-Term Cardiovascular Risk in HIV

Ernest Nsubuga

Department of Clinical Pharmacy Kampala International University Uganda
Email: ernest.nsubuga@studwc.kiu.ac.ug

ABSTRACT

The introduction of combination antiretroviral therapy has transformed human immunodeficiency virus infection from a fatal disease into a chronic, manageable condition, resulting in a substantial increase in life expectancy among affected individuals. However, this epidemiological success had been accompanied by a growing burden of metabolic complications and cardiovascular disease. The purpose of this review was to critically examine the molecular and biochemical pathways underlying antiretroviral-induced metabolic dysregulation and to elucidate their contribution to long-term cardiovascular risk in people living with HIV. This narrative review was developed through comprehensive analysis and synthesis of current experimental, translational, and clinical literature on antiretroviral therapy and cardiometabolic outcomes. Evidence indicated that mitochondrial dysfunction, oxidative stress, adipocyte impairment, altered insulin signaling, dyslipidemia, chronic immune activation, and endothelial dysfunction collectively mediate atherogenic processes in treated HIV populations. Specific antiretroviral classes exerted differential metabolic effects, further modulating cardiovascular risk profiles. Longitudinal cohort data demonstrated increased incidence of myocardial infarction, subclinical atherosclerosis, and arterial stiffness in this population. Early risk stratification and mechanistically informed therapeutic strategies are therefore essential. Integrated cardiometabolic monitoring and individualized antiretroviral selection were critical to reducing long-term cardiovascular morbidity in HIV.

Keywords: HIV, antiretroviral therapy, Metabolic dysregulation, Cardiovascular disease, Mitochondrial dysfunction.

INTRODUCTION

The advent of combination antiretroviral therapy represents one of the most significant achievements in modern medicine [1, 2]. Once uniformly fatal, human immunodeficiency virus infection has evolved into a chronic condition characterized by near-normal life expectancy in individuals with sustained viral suppression. This therapeutic success has shifted the clinical landscape from opportunistic infections and acquired immunodeficiency syndrome related mortality to chronic non communicable diseases. Among these, cardiovascular disease has emerged as a leading cause of morbidity and mortality in people living with HIV [3].

The increased cardiovascular burden in treated HIV populations is multifactorial. Persistent immune activation, chronic inflammation, and traditional cardiometabolic risk factors contribute substantially [4, 5]. However, compelling evidence demonstrates that antiretroviral therapy itself plays a pivotal role in the development of metabolic abnormalities that accelerate vascular pathology. Dyslipidemia, insulin resistance, altered fat distribution, hepatic steatosis, mitochondrial dysfunction, and endothelial injury are increasingly recognized consequences of long-term exposure to certain antiretroviral agents.

Despite advances in pharmacological refinement, metabolic dysregulation remains a significant therapeutic challenge. The biochemical mechanisms linking antiretroviral therapy to cardiovascular disease are complex and

involve perturbations in cellular energy metabolism, adipocyte biology, inflammatory signaling, and vascular homeostasis [6-10]. A comprehensive understanding of these pathways is essential for optimizing treatment strategies and minimizing long-term risk. This review examines the molecular and biochemical mechanisms underlying antiretroviral-induced metabolic dysregulation, explores their pathophysiological progression to cardiovascular disease, evaluates current clinical evidence on long-term risk, and discusses emerging therapeutic and preventive strategies. The purpose is to integrate mechanistic insights with clinical data to inform evidence-based management of cardiometabolic complications in HIV.

Overview of Antiretroviral Therapy and Metabolic Complications

Combination antiretroviral therapy typically includes agents from multiple classes, including nucleoside reverse transcriptase inhibitors, non-nucleoside reverse transcriptase inhibitors, protease inhibitors, integrase strand transfer inhibitors, and entry inhibitors [7, 8]. These regimens effectively suppress viral replication and restore immune function. However, their metabolic consequences vary by class and individual drug.

Historically, protease inhibitors were strongly associated with dyslipidemia and insulin resistance. Elevated triglycerides, increased low-density lipoprotein cholesterol, and reduced high-density lipoprotein cholesterol were frequently observed [9]. Nucleoside reverse transcriptase inhibitors, particularly earlier agents, were linked to mitochondrial toxicity and lipodystrophy. More recent integrase inhibitors demonstrate improved metabolic profiles, yet emerging data suggest potential associations with weight gain and adiposity. The metabolic phenotype in treated HIV encompasses dyslipidemia, impaired glucose tolerance, visceral adiposity, hepatic steatosis, and altered fat redistribution [10, 11,12-16]. Lipodystrophy, characterized by peripheral lipoatrophy and central fat accumulation, reflects adipocyte dysfunction and altered lipid handling. These metabolic disturbances not only compromise quality of life but also create a pro-atherogenic milieu.

Importantly, metabolic complications may develop even in virally suppressed individuals, underscoring the independent contribution of antiretroviral agents. As treatment duration extends over decades, cumulative exposure amplifies the risk of metabolic derangements and subsequent cardiovascular sequelae.

Molecular and Biochemical Pathways of Antiretroviral-Induced Metabolic Dysregulation

Antiretroviral-induced metabolic dysregulation arises from multiple intersecting biochemical pathways.

Mitochondrial Dysfunction and Oxidative Stress: Certain nucleoside reverse transcriptase inhibitors inhibit mitochondrial DNA polymerase gamma, impairing mitochondrial DNA replication [12, 17-20]. This leads to reduced oxidative phosphorylation, diminished adenosine triphosphate production, and increased reactive oxygen species generation. Oxidative stress damages cellular lipids, proteins, and nucleic acids, promoting insulin resistance and endothelial injury.

Adipocyte Dysfunction and Adipokine Imbalance: Protease inhibitors and some integrase inhibitors alter adipocyte differentiation by interfering with peroxisome proliferator activated receptor gamma signaling. Impaired adipogenesis and enhanced lipolysis result in ectopic lipid deposition and elevated circulating free fatty acids. Dysregulated secretion of adipokines such as adiponectin and leptin further exacerbates insulin resistance and systemic inflammation [13].

Insulin Signaling Disruption: Protease inhibitors directly inhibit glucose transporter type 4 activity in adipose tissue and skeletal muscle, reducing glucose uptake [14, 15, 21-27]. Concurrently, inflammatory cytokines such as tumor necrosis factor alpha and interleukin 6 activate serine kinases that impair insulin receptor substrate phosphorylation. These mechanisms converge to produce peripheral insulin resistance.

Hepatic Lipid Metabolism Alterations: Antiretroviral agents influence hepatic lipid synthesis by activating sterol regulatory element binding proteins, enhancing very low density lipoprotein production. Increased hepatic triglyceride synthesis and reduced lipid clearance contribute to dyslipidemia and non alcoholic fatty liver disease.

Chronic Immune Activation and Endothelial Dysfunction: Even with viral suppression, residual immune activation persists. Antiretroviral therapy may not fully normalize inflammatory pathways. Elevated C-reactive protein, soluble CD14, and other markers reflect ongoing inflammation that damages vascular endothelium [16, 17, 28-33]. Reduced nitric oxide bioavailability impairs vasodilation and promotes vascular stiffness.

Collectively, these molecular perturbations establish a pro-inflammatory, pro-oxidative, and pro-atherogenic metabolic environment.

Pathophysiological Progression to Cardiovascular Disease

The transition from metabolic dysregulation to overt cardiovascular disease involves a complex cascade of vascular events. Dyslipidemia promotes low-density lipoprotein oxidation and deposition within the arterial intima [18]. Oxidized lipoproteins stimulate macrophage recruitment and foam cell formation, initiating atherosclerotic plaque

development. Insulin resistance further enhances endothelial dysfunction by reducing nitric oxide synthesis and increasing endothelin-mediated vasoconstriction.

Chronic inflammation amplifies plaque instability. Activated immune cells secrete matrix metalloproteinases that degrade fibrous caps, predisposing plaques to rupture [19,34-40]. Additionally, prothrombotic alterations, including elevated fibrinogen and platelet activation, increase the likelihood of acute coronary events. Arterial stiffness and vascular remodeling are frequently observed in treated HIV populations. Structural changes in the vascular wall result from sustained oxidative stress and inflammatory signaling. These alterations elevate systolic blood pressure and left ventricular workload, compounding cardiovascular risk.

Importantly, cardiovascular pathology in HIV often manifests at younger ages compared with the general population [20,41-44]. Subclinical atherosclerosis, measured by carotid intima media thickness and coronary artery calcium scoring, is more prevalent among individuals receiving long term therapy. Thus, antiretroviral induced metabolic abnormalities accelerate classical atherogenic pathways, bridging cellular dysfunction and clinical cardiovascular disease.

Clinical Evidence and Long Term Cardiovascular Risk

Large cohort studies have consistently demonstrated increased cardiovascular event rates in people living with HIV compared with uninfected controls. While traditional risk factors such as smoking and hypertension contribute, antiretroviral exposure independently modifies risk. Protease inhibitor-based regimens have been associated with higher rates of myocardial infarction in longitudinal analyses. Certain nucleoside reverse transcriptase inhibitors have also been implicated in elevated cardiovascular risk, possibly through endothelial and inflammatory mechanisms [21,45-48]. Newer agents exhibit improved safety profiles, yet weight gain and emerging metabolic effects warrant continued surveillance.

Risk stratification remains challenging. Conventional cardiovascular risk calculators may underestimate risk in HIV populations because they do not account for chronic inflammation or therapy-related metabolic disturbances [22,48]. Biomarkers such as high-sensitivity C-reactive protein, interleukin-6, D-dimer, and markers of immune activation provide additional prognostic information. Importantly, duration of therapy and cumulative drug exposure correlate with metabolic burden. As individuals initiate treatment earlier and remain on therapy lifelong, understanding long-term cardiovascular implications becomes increasingly critical.

The clinical evidence underscores the necessity of integrating cardiometabolic monitoring into routine HIV care, including lipid profiling, glucose assessment, body composition evaluation, and vascular imaging when appropriate.

Therapeutic Strategies, Risk Mitigation, and Future Directions

Mitigating cardiovascular risk in treated HIV requires a multifaceted approach. Optimization of antiretroviral regimens is fundamental. Selecting agents with favorable metabolic profiles can reduce dyslipidemia and insulin resistance. Switching from protease inhibitor based regimens to alternative classes may improve lipid parameters in selected patients [23]. Lifestyle interventions, including dietary modification, regular physical activity, and smoking cessation, remain cornerstones of cardiovascular prevention. Pharmacological management with statins, antihypertensive agents, and insulin sensitizers should be implemented according to established guidelines, with attention to potential drug interactions [24].

Emerging research explores targeted anti inflammatory therapies to reduce residual immune activation. Modulation of adipokine signaling and mitochondrial protective strategies represent promising investigational avenues [25]. Precision medicine approaches incorporating genetic susceptibility and biomarker profiling may enable individualized cardiovascular risk prediction.

Future research should prioritize long-term randomized studies comparing metabolic outcomes across contemporary regimens and identifying mechanistic biomarkers that predict cardiovascular events. Translational integration between molecular science and clinical cardiology is essential.

CONCLUSION

The transformation of HIV into a chronic manageable disease has revealed the unintended metabolic consequences of long-term antiretroviral therapy. Mitochondrial dysfunction, oxidative stress, adipocyte impairment, insulin resistance, hepatic lipid alterations, chronic inflammation, and endothelial injury constitute interconnected biochemical pathways that promote atherosclerosis and cardiovascular disease. Clinical evidence confirms that treated HIV populations experience elevated rates of myocardial infarction, vascular stiffness, and subclinical atherosclerosis, reflecting the cumulative impact of therapy-related and traditional risk factors. Understanding the molecular underpinnings of antiretroviral-induced metabolic dysregulation provides a foundation for rational therapeutic decision-making. Careful selection of antiretroviral regimens, early identification of metabolic abnormalities, and aggressive management of modifiable risk factors are essential components of comprehensive

HIV care. As life expectancy continues to improve, attention must shift toward preserving long-term cardiometabolic health. Strategic integration of mechanistic research, longitudinal clinical surveillance, and individualized therapeutic optimization is imperative to reduce cardiovascular morbidity in people living with HIV. Routine incorporation of comprehensive cardiometabolic risk assessment and individualized antiretroviral selection should become a standard of care in long-term HIV management.

REFERENCES

1. Weichseldorfer, M., Reitz, M., Latinovic, O.S.: Past HIV-1 Medications and the Current Status of Combined Antiretroviral Therapy Options for HIV-1 Patients. Pharmaceutics. 13, (2021). https://doi.org/10.3390/pharmaceutics13111798
2. Alum, E.U., Uti, D.E., Ugwu, O.P.-C., Alum, B.N.: Toward a cure - Advancing HIV/AIDs treatment modalities beyond antiretroviral therapy: A Review. Medicine (Baltimore). 103, e38768 (2024). https://doi.org/10.1097/MD.0000000000038768
3. Obeagu, E., P.C., U., Aja, P., Okon, M.: HIV Infection and Cardiovascular Diseases: The obnoxious duo. Newport International Journal of Research in Medical Sciences, 3(2): 95-99. (2023)
4. Esser, N., Paquot, N., Scheen, A.J.: Inflammatory markers and cardiometabolic diseases. Acta Clinica Belgica. 70, 193–199 (2015). https://doi.org/10.1179/2295333715Y.0000000004
5. Mitroulis, I., Hajishengallis, G., Chavakis, T.: Bone marrow inflammatory memory in cardiometabolic disease and inflammatory comorbidities. Cardiovasc Res. 119, 2801–2812 (2023). https://doi.org/10.1093/cvr/cvad003
6. Mohan, J., Ghazi, T., Chuturgoon, A.A.: A Critical Review of the Biochemical Mechanisms and Epigenetic Modifications in HIV- and Antiretroviral-Induced Metabolic Syndrome. International Journal of Molecular Sciences. 22, (2021). https://doi.org/10.3390/ijms222112020
7. Vanangamudi, M., Kurup, S., Namasivayam, V.: Non-nucleoside reverse transcriptase inhibitors (NNRTIs): a brief overview of clinically approved drugs and combination regimens. Current Opinion in Pharmacology. 54, 179–187 (2020). https://doi.org/10.1016/j.coph.2020.10.009
8. Mbuagbaw, L., Mursleen, S., Irlam, J.H., Spaulding, A.B., Rutherford, G.W., Siegfried, N.: Efavirenz or nevirapine in three-drug combination therapy with two nucleoside or nucleotide-reverse transcriptase inhibitors for initial treatment of HIV infection in antiretroviral-naïve individuals - Mbuagbaw, L - 2016 | Cochrane Library.
9. Langsted, A., Jensen, A.M.R., Varbo, A., Nordestgaard, B.G.: Low High-Density Lipoprotein Cholesterol to Monitor Long-Term Average Increased Triglycerides. J Clin Endocrinol Metab. 105, e1657–e1666 (2020). https://doi.org/10.1210/clinem/dgz265
10. Koethe, J.R.: Adipose Tissue in HIV Infection. Comprehensive Physiology. 7, 1339–1357 (2017). https://doi.org/10.1002/j.2040-4603.2017.tb00775.x
11. Lagathu, C., Béréziat, V., Gorwood, J., Fellahi, S., Bastard, J.-P., Vigouroux, C., Boccara, F., Capeau, J.: Metabolic complications affecting adipose tissue, lipid and glucose metabolism associated with HIV antiretroviral treatment. Expert Opinion on Drug Safety. 18, 829–840 (2019). https://doi.org/10.1080/14740338.2019.1644317
12. Lutu, M.R., Nzuza, S., Mofo Mato, P.E., Govender, K., Gumede, L.M., Kumalo, S.I., Mlambo, N.N., Hurchund, R., Oroma Owira, P.M.: DNA polymerase-γ hypothesis in nucleoside reverse transcriptase-induced mitochondrial toxicity revisited: A potentially protective role for citrus fruit-derived naringenin? European Journal of Pharmacology. 852, 159–166 (2019). https://doi.org/10.1016/j.ejphar.2019.03.017
13. Unamuno, X., Gómez-Ambrosi, J., Rodríguez, A., Becerril, S., Frühbeck, G., Catalán, V.: Adipokine dysregulation and adipose tissue inflammation in human obesity. European Journal of Clinical Investigation. 48, e12997 (2018). https://doi.org/10.1111/eci.12997
14. Chadt, A., Al-Hasani, H.: Glucose transporters in adipose tissue, liver, and skeletal muscle in metabolic health and disease. Pflugers Arch - Eur J Physiol. 472, 1273–1298 (2020). https://doi.org/10.1007/s00424-020-02417-x
15. Ueda, M., Hayashibara, K., Ashida, H.: Propolis extract promotes translocation of glucose transporter 4 and glucose uptake through both PI3K- and AMPK-dependent pathways in skeletal muscle. BioFactors. 39, 457–466 (2013). https://doi.org/10.1002/biof.1085

16. Ansar, W., Ghosh, S.: Inflammation and Inflammatory Diseases, Markers, and Mediators: Role of CRP in Some Inflammatory Diseases. In: Ansar, W. and Ghosh, S. (eds.) Biology of C Reactive Protein in Health and Disease. pp. 67–107. Springer India, New Delhi (2016)
17. Germolec, D.R., Shipkowski, K.A., Frawley, R.P., Evans, E.: Markers of Inflammation. In: DeWitt, J.C., Rockwell, C.E., and Bowman, C.C. (eds.) Immunotoxicity Testing: Methods and Protocols. pp. 57–79. Springer, New York, NY (2018)
18. Khatana, C., Saini, N.K., Chakrabarti, S., Saini, V., Sharma, A., Saini, R.V., Saini, A.K.: Mechanistic Insights into the Oxidized Low-Density Lipoprotein-Induced Atherosclerosis. Oxidative Medicine and Cellular Longevity. 2020, 5245308 (2020). https://doi.org/10.1155/2020/5245308
19. Hansson, G.K., Libby, P., Tabas, I.: Inflammation and plaque vulnerability. Journal of Internal Medicine. 278, 483–493 (2015). https://doi.org/10.1111/joim.12406
20. Hudson, J.A., Majonga, E.D., Ferrand, R.A., Perel, P., Alam, S.R., Shah, A.S.V.: Association of HIV Infection With Cardiovascular Pathology Based on Advanced Cardiovascular Imaging: A Systematic Review. JAMA. 328, 951–962 (2022). https://doi.org/10.1001/jama.2022.15078
21. Chen, Y.-F., Stampley, J.E., Irving, B.A., Dugas, T.R.: Chronic Nucleoside Reverse Transcriptase Inhibitors Disrupt Mitochondrial Homeostasis and Promote Premature Endothelial Senescence. Toxicol Sci. 172, 445–456 (2019). https://doi.org/10.1093/toxsci/kfz203
22. Triant, V.A., Perez, J., Regan, S., Massaro, J.M., Meigs, J.B., Grinspoon, S.K., D'Agostino, R.B.: Cardiovascular Risk Prediction Functions Underestimate Risk in HIV Infection. Circulation. 137, 2203–2214 (2018). https://doi.org/10.1161/CIRCULATIONAHA.117.028975
23. Martini, S., Pisaturo, M., Russo, A., Palamone, M.G., Russo, M.T., Zollo, V., Maggi, P., Coppola, N.: Evaluation of Lipid Profile and Intima Media Thickness in Antiretroviral-Experienced HIV-Infected Patients Treated with Protease Inhibitor-Based Regimens versus Protease Inhibitor-Sparing Regimens. Pathogens. 12, (2023). https://doi.org/10.3390/pathogens12070925
24. Alum, E.U.: Role of phytochemicals in cardiovascular disease management: Insights into mechanisms, efficacy, and clinical application. Phytomedicine Plus. 5, 100695 (2025). https://doi.org/10.1016/j.phyplu.2024.100695
25. Zheng, Y., Yang, N., Pang, Y., Gong, Y., Yang, H., Ding, W., Yang, H.: Mitochondria-associated regulation in adipose tissues and potential reagents for obesity intervention. Front. Endocrinol. 14, (2023). https://doi.org/10.3389/fendo.2023.1132342
26. Alum EU, Ugwu OPC, Obeagu EI, Aja PM, Okon MB. HIV infection and cardiovascular diseases: the obnoxious duos. Newport Int J Res Med Sci. 2023;3(2):95–9.
27. Alum EU, Ugwu OPC, Obeagu EI, Okon MB. Curtailing HIV/AIDS spread: impact of religious leaders. Newport Int J Res Med Sci. 2023;3(2):28–31.
28. Obeagu EI, Malot S, Obeagu GU, Ugwu OPC. HIV resistance in patients with sickle cell anaemia. Newport Int J Sci Exp Sci. 2023;3(2).
29. Alum EU, Obeagu EI, Ugwu OPC, Egba SI, Uti DE, Ukaidi CUA. Confronting dual challenges: substance abuse and HIV/AIDS. Elite J HIV. 2024;2(5):1–8.
30. Alum EU, Uti DE, Ugwu OPC, Alum BN. Toward a cure—advancing HIV/AIDS treatment modalities beyond antiretroviral therapy: a review. Medicine. 2024;103(27):e38768.
31. Obeagu EI, Obeagu GU, Obiezu J, Ezeonwumelu C, Ogunnaya FU, Alum EU, et al. Hematologic support in HIV patients: blood transfusion strategies and immunological considerations. Newport Int J Biol Appl Sci. 2023;3(3):9–15.
32. Obeagu EI, Obeagu GU, Alum EU, Ugwu OPC. Advancements in immune augmentation strategies for HIV patients. IAA J Biol Sci. 2023;11(1):1–11.
33. Obeagu EI, Obeagu GU, Odo EO, Ugwu OPC, Alum EU. Revolutionizing HIV prevention in Africa: landmark innovations that transformed the fight. IAA J Appl Sci. 2024;11(1):1–12.
34. Obeagu EI, Obeagu GU, Odo EO, Ugwu OPC, Alum EU. Combatting stigma: essential steps in halting HIV spread. IAA J Appl Sci. 2024;11(1):22–9.
35. Obeagu EI, Obeagu GU, Odo EO, Ugwu OPC, Alum EU. Disaster fallout: impact of natural calamities on HIV control. IAA J Appl Sci. 2024;11(1):13–21.
36. Obeagu EI, Ugwu OPC, Alum EU. Nutritional approaches for enhancing immune competence in HIV-positive individuals: a comprehensive review. IDOSR J Appl Sci. 2024;9(1):40–50.
37. Obeagu EI, Obeagu GU, Alum EU, Ugwu OPC. Stigma associated with HIV: a review. Newport Int J Public Health Pharm. 2023.

38. Alum EU, Ugwu OPC, Obeagu EI, Okon MB. Reducing HIV infection rate in women: a catalyst to reducing HIV infection pervasiveness in Africa. Int J Innov Appl Res. 2023;11(10):1–6.
39. Alum EU, Obeagu EI, Ugwu OPC, Aja PM, Ugwu CN, Okon MB. Inclusion of nutritional counseling and mental health services in HIV/AIDS management: a paradigm shift. Medicine. 2023;102(41):e35673.
40. Madu CV, Alum EU, Aloh HE, Ugwu OPC, Obeagu EI, Uti DE, et al. The price of progress: assessing the financial costs of HIV/AIDS management in East Africa. Medicine. 2025;104(18):e42300.
41. Ezenwaji CO, Alum EU, Ugwu OPC. Bridging the gap: telemedicine as a solution for HIV care inequities in rural and vulnerable communities. Int J Equity Health. 2025;24(1):205.
42. Swase DT, Dangana RS, Mbina SA, Mounmbegna PEP, Aja PM, Ugwu OPC, et al. Nanoparticles in HIV treatment for improved drug delivery, clinical translation, and future direction. Discov Nano. 2026;21(6). doi:10.1186/s11671-026-04167-7
43. Swase DT, Ugwu OPC, Chebet J, Mounmbegna PEP, Mbina SA, Aja PM, et al. Molecular, immunological and oncogenic mechanisms of cervical cancer mediated by HPV/HIV co-infection, clinical implication and management. Infect Agents Cancer. 2026. doi:10.1186/s13027-025-00729-0
44. Obeagu EI, Obeagu GU, Alum EU, Ugwu OPC. Comprehensive review of antiretroviral therapy effects on red blood cells in HIV patients. INOSR Exp Sci. 2023;12(3):63–72.
45. Obeagu EI, Obeagu GU, Alum EU, Ugwu OPC. Persistent immune activation and chronic inflammation: unraveling their impact on anemia in HIV infection. INOSR Exp Sci. 2023;12(3):73–84.
46.Obeagu EI, Obeagu GU, Alum EU, Ugwu OPC. Anemia as a prognostic marker for disease progression in HIV infection. IAA J Biol Sci. 2023;11(1):33–44.
47. Obeagu EI, Obeagu GU, Alum EU, Ugwu OPC. Understanding the impact of HIV-associated bone marrow alterations on erythropoiesis. INOSR Sci Res. 2023;10(1):1–11.
48. Obeagu EI, Nwosu DC, Alum EU, Ugwu OPC. Adverse drug reactions in HIV/AIDS patients on highly active antiretroviral therapy: a review of prevalence. Newport Int J Sci Exp Sci. 2023.

CITE AS: Ernest Nsubuga (2026). Antiretroviral-Induced Metabolic Dysregulation: Pathways and Long-Term Cardiovascular Risk in HIV. IDOSR JOURNAL OF APPLIED SCIENCES 11(2):71-76. https://doi.org/10.59298/IDOSRJAS/2026/1127176