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Research Article

Conformable Fractional-Order Modeling and Analysis of HIV/AIDS Transmission Dynamics

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The mathematical model of the dynamics of HIV/AIDS infection transmission is developed by adding the set of infected but noninfectious persons, using a conformable fractional derivative in the Liouville–Caputo sense. Some fixed point theorems are applied to this model to investigate the existence and uniqueness of the solutions. It is determined what the system's fundamental reproduction number R_0 is. The disease-free equilibrium displays the model's stability and the local stability around the equilibrium. The study also examined the effects of different biological features on the system through numerical simulations using the Adams–Moulton approach. Additionally, varied values of fractional orders are simulated numerically, demonstrating that the results generated by the conformable fractional derivative-based model are more physiologically plausible than integer-order derivatives.

1. Introduction

HIV/AIDS has been a global epidemic since the late 20th century, infecting millions of people around the world [1]. HIV is an infectious disease that targets the body's defenses, specifically targeting CD^+4 T-cells, which act as a bodyguard against any viral infection [2]. Body fluids are the primary vector for HIV transmission. Also, unprotected sexual contact, sharing of needles and syringes among drug injectors, and mother-to-child transmission during delivery or nursing are common routes of transmission. Therefore, if we cannot manage HIV effectively, it can gradually destroy the body's immunity, making it difficult for the body to resist infections and diseases. As this virus has negative impacts on public health, healthcare systems, and communities, and with advances in medical research and treatment, a large number of researchers have sought and put in much effort to manage HIV and limit its spread and allowing individuals infected with HIV to live a better life and be healthier and reduce the risk of infecting others. Classical differential equations use integer-order derivatives to explain population dynamics. However, the fractional differential equations (FDEs) extend this idea to fractional-order derivatives, allowing a more accurate depiction of complex systems as the propagation of infectious illnesses. In recent decades, FDEs have gained much attention due to their use in modeling a wide range of phenomena in various disciplines, including biology, chemistry, economics, engineering, physics, and others [3]. FDEs are among the most important tools used to build these mathematical models. It has been found that they give more accurate results than those that can be obtained using differential equations in the integer order. The researchers point out in [4] that the model can predict the course of infection and the effectiveness of

different treatment strategies. It can also be used to understand the disease's mechanisms and identify potential targets for new therapies [5, 6]. Many fractional differential operators have been used in so-called mathematical epidemiology to model many infectious diseases as mathematical models of infectious diseases are important sources for understanding and interpreting the dynamics of those diseases such as coronaries, polycystic ovarian syndrome, tuberculosis, immunodeficiency, influenza, cancer, and hepatitis [7–17]. FDEs are crucial in understanding the dynamics of HIV/AIDS transmission. Many mathematical models consisting of FDEs and fractional differential operators have emerged to describe the dynamics of HIV/AIDS transmission, control its spread, and understand its transmission mechanisms between people (see [18]). Mustapha et al. [19] established a stochastic HIV/AIDS model with protection awareness. Their paper revealed the impact of protection awareness on the control of AIDS. In [20], the authors studied how the human immunodeficiency virus (HIV) and acquired immune deficiency syndrome (AIDS) spread. Hassani et al. [21], investigated the fractional mathematical model of the HIV/AIDS spread optimization technique by using generalized shifted Jacobi polynomials. In [22], the authors presented a new mathematical model that examines the interaction between the immune system and cancer cells using Caputo and Caputo–Fabrizio derivatives. The model shows a reduction in the number of cancer cells for both derivatives, with a higher reduction observed for the Caputo–Fabrizio derivative. In [23], the chaotic behavior of a fractional-order HIV-1 model involving interactions between cancer cells, healthy CD4+T lymphocytes, and virus-infected $CD^{+}4-T$ lymphocytes was investigated. Here, we discuss some literature about the study's subject. In [24], the authors discussed a modeling approach to study the role of prostitution in HIV transmission. It considers a fractional-order HIV epidemic model and analyzes the spread of HIV through prostitution. An HIV infection model of $CD^{+}4-T$ cells was discussed using the Atangana–Baleanu fractional derivative, and the existence and uniqueness of solutions for the model were analyzed in [25]. Javidi et al. [26] investigated a fractional-order HIV/AIDS (FOHA) epidemic system with treatment and solved the system obtained by using the fourth-order Runge–Kutta method. Ding et al. [27] discussed the conditions that must be met for a general fractional optimal control to be optimal control of the HIV-immune system with memory whose FD is described in the Caputo sense. Carla et al. [28] studied the FD model for HIV and TB coinfection, and they considered vertical HIV transmission and treatment for both ailments. They also analyzed the numerical results of the suggested model for various FD order values. Also, Carla et al. [29] studied the FD order model for the three phases of HIV epidemics with drug resistance. The model includes $CD^{+}4-T$ cells, CTLs, macrophages, and pathogen populations, and they simulated the model for different values of the FD. A fractional-order nonlinear mathematical model is presented for analyzing and monitoring the propagation of HIV/AIDS by Zahra et al. [30]. A population fractional model for transmitting human immunodeficiency virus (HIV) was presented in [31]; the authors assumed a homogenous mixing population and considered the availability of anti-HIV preventive vaccines. Hikal and Zahra in [32] investigated the fractional model of HIV/AIDS that includes treatment and time delay. They also presented the numerical simulations of the model by a finite difference method for a fractional system. Liu et al. [33] introduced the fractional-order HIV/AIDS infection model with the Beddington–DeAngelis functional response rate. The fractional-order HIV/AIDS model of analyzing and monitoring the propagation was considered by Zafar et al. [34]. They also investigated the numerical simulations and the impact of the system parameter on the spread of the disease using the Adams–Bashforth–Moulton algorithm and Grunwald–Letnikov method. FDs were used by Arshad et al. [35] to analyze the HIV infection model. Their analysis focused primarily on the degree of T-cell depletion from viral cytopathicity. Numerical simulations employing the Caputo derivative's finite difference approximation were used to demonstrate the analytical conclusions. Cristiana et al. [36] proposed a Caputo fractional-order SICA epidemiological model for HIV/AIDS transmission. The model extended an integer-order HIV/AIDS model, incorporating memory effects, long-range interactions, and hereditary properties through fractional differentiation. The local and uniform asymptotic stability of the disease-free and endemic equilibrium points was analyzed. A mathematical model for the transmission dynamics of HIV/AIDS with fractional order and fractal-fractional order has been studied in [1]. The model was evaluated for its basic properties, equilibria, and reproductive number.

A fractional conformable derivative (FCD) shares many characteristics with a classical derivative. It has been used to model several physical and biological problems. The author in [37] used a mathematical operator called the conformable derivative in the sense of the Liouville–Caputo derivative (LC) to investigate measles infection.

Motivated by the works mentioned above, we expand the HIV transmission model proposed in [36, 38] by adding the set of individuals who are infected but not infectious under FCD in the LC sense. We first verify the existence and uniqueness of the model solution. Then, we discuss the fundamental characteristics of the model, such as the disease-free equilibrium and basic reproduction number. Additionally, the model is numerically solved using the Adams–Moulton technique. Lastly, the graphical effects of different parameters are shown for various values of fractional order. This study aims to investigate the behavior of the HIV/AIDS virus infection model under different ϱ values and to analyze the effect of various factors on the disease dynamics.

This work used fractional-order conformable derivatives in the Liouville–Caputo sense to suggest a novel model of HIV/AIDS transmission. This allowed the model to represent the intricate dynamics involved in developing the disease. The fractional-order method enabled more physiologically accurate simulations than the conventional integer-order models. We conducted mathematical analysis and obtained analytical formulas describing the basic reproduction number R_0 to ensure the model is well-posed.

The fractional differential equations were solved by numerical simulations using Adams–Moulton techniques. The study clarified the implications of numerous biological parameters, such as viral load and infection age, through extensive simulations. The paper built upon earlier HIV modeling research by considering infection characteristics. All things considered, this work presented an improved modeling formalism for analyzing HIV/AIDS dynamics, which may have consequences for assessment.

This research paper is presented as follows. Section 2 provides some fundamental topics related to fractional calculus. The model structure and its associated results are given in Section 3. In Section 4, we prove the existence and uniqueness of the results by using fixed point theory and nonlinear analysis. In Section 5, we calculate disease-free equilibrium and R_0. Section 6 shows active numerical sketches for the HIV model solution by asymptomatic transporters.

2. Preliminaries

Here are some fundamental definitions that will be used in this study.

Definition 1 (see [39]). The Riemann–Liouville FD of order $q > 0$ can be defined as

$$ {}^{\mathrm{RL}}D^{q}_{a,t}\xi = \frac{1}{\Gamma(n-q)}\frac{d^n}{dt^n}\int_a^t (t-s)^{n-q-1}\xi(s)ds, \tag{1} $$

where Γ is the gamma function.

Definition 2 (see [39]). The Liouville–Caputo FD of order q may be expressed as follows:

$$ {}^{\mathrm{LC}}D^{q}_{a,t}\xi = \frac{1}{\Gamma(n-q)}\int_a^t (t-s)^{n-q-1}\frac{d^n}{ds^n}\xi(s)ds, \quad q \in (n-1, n], q > 0. \tag{2} $$

Definition 3 (see [40]). The expression:

$$ {}^{\varrho}D^{q}_{a,t}\xi = \lim_{\tau \longrightarrow 0}\frac{\xi\left(t+\tau t^{1-q}\right)-\xi}{\tau}, \tag{3} $$

where $q, t > 0$, is called FCD of order q.

Remark 4 (see [40]). The most crucial feature of the FCD that links it with classical derivatives is

$$ {}^{\varrho}D^{q}_{a,t}(\xi) = (t-a)^{1-q}\frac{d}{dt}\xi. \tag{4} $$

Definition 5 (see [41]). Let $\xi \in C^{n}_{q,\varrho}([a,b]), \mathrm{Re}(\varrho) \geq 0$ and $n = \lceil \varrho \rceil + 1$.

Then, the FCD in the sense of LC is defined as

$$ \begin{aligned} {}^{\varrho}_{c}D^{q}_{a,t}\xi &= \frac{1}{\Gamma(n-\varrho)}\int_a^t \left(\frac{(t-a)^q-(s-a)^q}{q}\right)^{n-\varrho-1}\frac{D^{n}_{a,t}}{(s-a)^{1-q}}\xi(s)ds \\ &= {}^{n-\varrho}_{c}I^{q}_{a,t}\xi\left({}^{\mathrm{n}}_{c}D^{q}_{a,t}\xi\right). \end{aligned} \tag{5} $$

Definition 6 (see [41]). Let $\xi \in C^{n}_{q,\varrho}([a,b]), \mathrm{Re}(\varrho) \geq 0$ and $n = \lceil \varrho \rceil + 1$.

Then, the fractional conformable integral in the sense of LC is defined as

$$ {}^{\varrho}\mathscr{I}^{q}_{a,t}\xi = \frac{1}{\Gamma(\varrho)}\int_a^t \left(\frac{(t-a)^q-(s-a)^q}{q}\right)^{\varrho-1}\frac{\xi(s)}{(s-a)^{1-q}}ds. \tag{6} $$

3. The Model

The FCD model is first formulated using integer-order derivatives. To further explore the model at hand, we will examine the scenario in which the recruitment rate remains constant, but the people size varies. The cumulative density of the human population, denoted as $\mathscr{N}$, may be further categorized into several groups. These groups include the susceptible population, represented as S, and the latent population, denoted as E. The latent population refers to individuals who are infected with HIV but are not yet able to transmit the virus to others. Additionally, a group of HIV-infected individuals can transmit the infection but do not exhibit any clinical signs of AIDS. In this representation, the symptomatic stage of HIV infection is denoted as I. In contrast, those taking antiretroviral therapy (ART) with a lower viral load are denoted as $\mathscr{C}$. Additionally, individuals with HIV and AIDS are depicted as $\mathscr{A}$. The susceptible population enters the latent class at a rate of μ or the infectious class at a rate of $(1-\mu)$ after effective contact with individuals in the I and $\mathscr{A}$ classes, taking into account their immune strength. Latent individuals transition to the I class at rate σ after the latent period. Untreated infectious individuals develop AIDS at rate τ_2, initiating ART treatment

at rate θ, with treatment failure returning them to I class at rate λ. Infected individuals experience AIDS-related mortality at rate d_1. We assume higher infectiousness for AIDS patients ($\nu_A \geq 1$) than infectious individuals (ν_C), with recruitment rate ρ and background mortality rate d for all classes. We adopt baseline numeric values for parameters presented in Table 1.

In light of the preceding discussion and hypotheses, the proposed compartmental model for HIV/AIDS can be expressed by the following differential equation system:

$$\begin{aligned}
\mathcal{S}' &= \rho - \iota\left(I + \nu_c\mathscr{C} + \nu_A\mathscr{A}\right)S - dS,\\
E' &= \iota\mu\left(I + \nu_c\mathscr{C} + \nu_A\mathscr{A}\right)S - (\sigma + d)E,\\
I' &= (1-\mu)\iota\left(I + \nu_c\mathscr{C} + \nu_A\mathscr{A}\right)S + \sigma E - \left(\tau_1 + \tau_2 + d\right)I + \lambda\mathscr{C} + \theta\mathscr{A},\\
\mathscr{C}' &= \tau_1 I - (\lambda + d)\mathscr{C},\\
\mathscr{A}' &= \tau_2 I - \left(d + \theta + d_1\right)\mathscr{A}.
\end{aligned} \tag{7}$$

The HIV/AIDS model (7) is now transformed into fractional order. Fractional-order differential systems are preferred over conventional ones because they can help reduce mistakes caused by overlooked parameters in real-world application modeling. The model (7) with FCD of order q in the sense of LC is

$$\begin{aligned}
{}^{\varrho}D^q_{0,t}S &= \rho - \iota\left(I + \nu_c\mathscr{C} + \nu_A\mathscr{A}\right)S - dS,\\
{}^{\varrho}D^q_{0,t}E &= \iota\mu\left(I + \nu_c\mathscr{C} + \nu_A\mathscr{A}\right)S - (\sigma + d)E,\\
{}^{\varrho}D^q_{0,t}I &= (1-\mu)\iota\left(I + \nu_c\mathscr{C} + \nu_A\mathscr{A}\right)S + \sigma E - \left(\tau_1 + \tau_2 + d\right)I + \lambda\mathscr{C} + \theta\mathscr{A},\\
{}^{\varrho}D^q_{0,t}\mathscr{C} &= \tau_1 I - (\lambda + d)\mathscr{C},\\
{}^{\varrho}D^q_{0,t}\mathscr{A} &= \tau_2 I - \left(d + \theta + d_1\right)\mathscr{A},
\end{aligned} \tag{8}$$

where $S(t_0) = S_0, E(t_0) = E_0, I(t_0) = I_0, \mathscr{C}(t_0) = \mathscr{C}_0$, and $\mathscr{A}(t_0) = \mathscr{A}_0$ are initial conditions.

4. Existence and Uniqueness Results

For model (8), it will be shown here that the solution exists and is unique with FCD of order q in the LC sense.

Suppose the real-valued function $B(\mathbb{I})$ is continuous and contains the sup norm property. It is clear that $(B(\mathbb{I}), \|\cdot\|)$ is a Banach space. Let $\mathbb{I} = [0, b]$ and $P = B(\mathbb{I}) \times B(\mathbb{I}) \times B(\mathbb{I}) \times B(\mathbb{I}) \times B(\mathbb{I})$ with norm $\|(S, E, I, \mathscr{C}, \mathscr{A})\| = \|S\| + \|E\| + \|I\| + \|\mathscr{C}\| + \|\mathscr{A}\|$, $\|S\| = \sup_{t\in\mathbb{I}}|S|$, $\|E\| = \sup_{t\in\mathbb{I}}|E|$, $\|I\| = \sup_{t\in\mathbb{I}}|I|$, $\|\mathscr{C}\| = \sup_{t\in\mathbb{I}}|\mathscr{C}|$, $\|\mathscr{A}\| = \sup_{t\in\mathbb{I}}|\mathscr{A}|$.

At first, we put

$$\begin{aligned}
\xi_1(t, S) &= \rho - \iota\left(I + \nu_c\mathscr{C} + \nu_A\mathscr{A}\right)S - dS,\\
\xi_2(t, E) &= \iota\mu\left(I + \nu_c\mathscr{C} + \nu_A\mathscr{A}\right)S - (\sigma + d)E,\\
\xi_3(t, I) &= (1-\mu)\iota\left(I + \nu_c\mathscr{C} + \nu_A\mathscr{A}\right)S + \sigma E - \left(\tau_1 + \tau_2 + d\right)I\\
&\quad + \lambda\mathscr{C} + \theta\mathscr{A},\\
\xi_4(t, \mathscr{C}) &= \tau_1 I - (\lambda + d)\mathscr{C},\\
\xi_5(t, \mathscr{A}) &= \tau_2 I - \left(d + \theta + d_1\right)\mathscr{A}.
\end{aligned} \tag{9}$$

Using the fractional integral operator on both sides of model (8), we get

Table 1: Details of the model's parameters (8).

Parameters	Description	Value	Source
ρ	Recruitment rate	2.1	[36]
ι	Contact rate	0.1	[1]
d_1	Death rate due to AIDS	0.1	[1]
d	Natural mortality rate	0.014	[1]
μ	Fraction of susceptible enter to E	0.1	[1]
ν_c, ν_A	Relative infectiousness rates	0.0150, 1.30	[36]
σ	Convert from class E to class I	0.0150	[1]
τ_1	Treatment rate	1	[36]
τ_2	Convert from I to $\mathscr{A}$ class	0.1	[36]
θ	Flow $\mathscr{A}$ to I due to appropriate care	0.33	[36]
λ	Treatment failure rate	0.090	[36]

$$\begin{aligned}
S - S(t_0) &= {}^{\varrho}\mathscr{I}_{0,t}^{q}\left[\rho - \iota\left(I + \nu_c\mathscr{C} + \nu_A\mathscr{A}\right)S - dS\right],\\
E - E(t_0) &= {}^{\varrho}\mathscr{I}_{0,t}^{q}\left[\iota\mu\left(I + \nu_c\mathscr{C} + \nu_A\mathscr{A}\right)S - (\sigma + d)E\right],\\
I - I(t_0) &= {}^{\varrho}\mathscr{I}_{0,t}^{q}\left[(1-\mu)\iota\left(I + \nu_c\mathscr{C} + \nu_A\mathscr{A}\right)S + \sigma E\right.\\
&\qquad \left. - \left(\tau_1 + \tau_2 + d\right)I + \lambda\mathscr{C} + \theta\mathscr{A}\right],\\
\mathscr{C} - C(t_0) &= {}^{\varrho}\mathscr{I}_{0,t}^{q}\left[\tau_1 I - (\lambda + d)\mathscr{C}\right],\\
\mathscr{A} - \mathscr{A}(t_0) &= {}^{\varrho}\mathscr{I}_{0,t}^{q}\left[\tau_2 I - \left(d + \theta + d_1\right)\mathscr{A}\right],
\end{aligned} \tag{10}$$

which implies

$$\begin{aligned}
S - S(t_0) &= \frac{1}{\Gamma(\varrho)}\int_a^t \left(\frac{(t-a)^q - (s-a)^q}{q}\right)^{\varrho-1} \frac{\xi_1(s, S(s))}{(s-a)^{1-q}}\,ds,\\
E - E(t_0) &= \frac{1}{\Gamma(\varrho)}\int_a^t \left(\frac{(t-a)^q - (s-a)^q}{q}\right)^{\varrho-1} \frac{\xi_2(s, E(s))}{(s-a)^{1-q}}\,ds,\\
I - I(t_0) &= \frac{1}{\Gamma(\varrho)}\int_a^t \left(\frac{(t-a)^q - (s-a)^q}{q}\right)^{\varrho-1} \frac{\xi_3(s, I(s))}{(s-a)^{1-q}}\,ds,\\
\mathscr{C} - \mathscr{C}(t_0) &= \frac{1}{\Gamma(\varrho)}\int_a^t \left(\frac{(t-a)^q - (s-a)^q}{q}\right)^{\varrho-1} \frac{\xi_4(s, \mathscr{C}(s))}{(s-a)^{1-q}}\,ds,\\
\mathscr{A} - \mathscr{A}(t_0) &= \frac{1}{\Gamma(\varrho)}\int_a^t \left(\frac{(t-a)^q - (s-a)^q}{q}\right)^{\varrho-1} \frac{\xi_5(s, \mathscr{A}(s))}{(s-a)^{1-q}}\,ds.
\end{aligned} \tag{11}$$

Let $S, E, I, \mathscr{C}, \mathscr{A}$, $S^*, E^*, I^*, \mathscr{C}^*, \mathscr{A}^* \in C(\mathbb{I}, R)$ and let $S \le \Psi_1, E \le \Psi_2, I \le \Psi_3, \mathscr{C} \le \Psi_4$, and $\mathscr{A} \le \Psi_5$, where $\Psi_1, \Psi_2, \Psi_3, \Psi_4, \Psi_5 \in R^+$.

The symbols $\xi_1, \xi_2, \xi_3, \xi_4$, and ξ_5 have to hold for the Lipschitz condition only if $S, E, I, \mathscr{C}$, and $\mathscr{A}$ possess an upper bound. Suppose that S and S^* are couple functions, and we have

$$\begin{aligned}
&\left\|\xi_1(t, S) - \xi_1(t, S^*)\right\|\\
&\quad = \left\|\left(\rho - \iota\left(I + \nu_c\mathscr{C} + \nu_A\mathscr{A}\right)S - dS\right) - \left(\rho - \iota\left(I + \nu_c\mathscr{C} + \nu_A\mathscr{A}\right)S^* - dS^*\right)\right\|,\\
&\quad = \left|\iota\left(I + \nu_c\mathscr{C} + \nu_A\mathscr{A}\right) + d\right|\left\|S - S^* - dS^*\right\|.
\end{aligned} \tag{12}$$

Take ω_1 as

$$\omega_1 = \left|\iota\left(\Psi_3 + \nu_c\Psi_4 + \nu_A\Psi_5\right) + d\right|. \tag{13}$$

Proceeding in the same way as before, we obtain

$$\begin{aligned}
\left\|\xi_1(t,E) - \xi_1(t,E^*)\right\| &= \left\|\iota\mu\left(I + \nu_c\mathscr{C} + \nu_A\mathscr{A}\right)S - (\sigma + d)E - \left(\iota\mu\left(I + \nu_c\mathscr{C} + \nu_A\mathscr{A}\right)S - (\sigma + d)E^*\right)\right\|,\\
\left\|\xi_1(t,I) - \xi_1(t,I^*)\right\| &= \left\|(1-\mu)\iota\left(I + \nu_c\mathscr{C} + \nu_A\mathscr{A}\right)S + \sigma E - \left(\tau_1 + \tau_2 + d\right)I + \lambda\mathscr{C} + \theta\mathscr{A}\right.\\
&\quad \left. - \left((1-\mu)\iota\left(I^* + \nu_c\mathscr{C} + \nu_A\mathscr{A}\right)S + \sigma E^* - \left(\tau_1 + \tau_2 + d\right)I^* + \lambda\mathscr{C} + \theta\mathscr{A}\right)\right\|,\\
\left\|\xi_1(t,\mathscr{C}) - \xi_1(t,\mathscr{C}^*))\right\| &= \left\|\left(\tau_1 I + (\lambda + d)\mathscr{C}\right) - \left(\tau_1 I + (\lambda + d)\mathscr{C}*\right)\right\|,\\
\left\|\xi_1(t,\mathscr{A}) - \xi_1(t,\mathscr{A}^*))\right\| &= \left\|\left(\tau_2 I + \left(d + \theta + d_1\right)\mathscr{A}\right)\right) - \left(\tau_2 I + \left(d + \theta + d_1\right)\mathscr{A}^*\right)\right\|.
\end{aligned} \tag{14}$$

Therefore,

$$\begin{aligned}
\left\|\xi_1(t,S) - \xi_1(t,S^*)\right\| &\le \omega_1\left\|S - S^*\right\|,\\
\left\|\xi_2(t,E) - \xi_2(t,E^*)\right\| &\le \omega_2\left\|E - E^*\right\|,\\
\left\|\xi_3(t,I) - \xi_3(t,I^*)\right\| &\le \omega_3\left\|I - I^*\right\|,\\
\left\|\xi_4(t,\mathscr{C}) - \xi_4(t,\mathscr{C}^*)\right\| &\le \omega_4\left\|\mathscr{C} - \mathscr{C}^*\right\|,\\
\left\|\xi_5(t,\mathscr{A}) - \xi_5(t,\mathscr{A}^*)\right\| &\le \omega_5\left\|\mathscr{A} - \mathscr{A}^*\right\|,
\end{aligned} \tag{15}$$

where $\omega_2 = |\sigma + d|$, $\omega_3 = |(1-\mu)\iota\Psi_1|, \omega_4 = |\lambda + d|, \omega_5 = |d + \theta + d_1|$. This means that the Lipschitz condition has been done for all four functions. Let us now take the expressions iteratively. Indeed, (10) yields

$$\begin{aligned}
S_n - S(t_0) &= \frac{1}{\Gamma(\varrho)}\int_a^t \left(\frac{(t-a)^q - (s-a)^q}{q}\right)^{\varrho-1}\frac{\xi_1\left(s, S_{n-1}(s)\right)}{(s-a)^{1-q}}ds,\\
E_n - E(t_0) &= \frac{1}{\Gamma(\varrho)}\int_a^t \left(\frac{(t-a)^q - (s-a)^q}{q}\right)^{\varrho-1}\frac{\xi_2\left(s, E_{n-1}(s)\right)}{(s-a)^{1-q}}ds,\\
I_n - I(t_0) &= \frac{1}{\Gamma(\varrho)}\int_a^t \left(\frac{(t-a)^q - (s-a)^q}{q}\right)^{\varrho-1}\frac{\xi_3\left(s, I_{n-1}(s)\right)}{(s-a)^{1-q}}ds,\\
\mathscr{C}_n - \mathscr{C}(t_0) &= \frac{1}{\Gamma(\varrho)}\int_a^t \left(\frac{(t-a)^q - (s-a)^q}{q}\right)^{\varrho-1}\frac{\xi_4\left(s, \mathscr{C}_{n-1}(s)\right)}{(s-a)^{1-q}}ds,\\
\mathscr{A}_n - \mathscr{A}(t_0) &= \frac{1}{\Gamma(\varrho)}\int_a^t \left(\frac{(t-a)^q - (s-a)^q}{q}\right)^{\varrho-1}\frac{\xi_5\left(s, \mathscr{A}_{n-1}(s)\right)}{(s-a)^{1-q}}ds,
\end{aligned} \tag{16}$$

with $S(t_0) = S_0, E(t_0) = E_0, I(t_0) = I_0, \mathscr{C}(t_0) = \mathscr{C}_0$, and $\mathscr{A}(t_0) = \mathscr{A}_0$.

When the difference between the following terms is taken, we obtain

$$\begin{aligned}
\Upsilon_{S,n} &= S_n - S_{n-1} = \frac{1}{\Gamma(\varrho)}\int_a^t \left(\frac{(t-a)^q-(s-a)^q}{q}\right)^{\varrho-1} \frac{(\xi_1(s,S_{n-1}(s))-\xi_1(s,S_{n-2}(s)))}{(s-a)^{1-q}}ds,\\
\Upsilon_{E,n} &= E_n - E_{n-1}(0) = \frac{1}{\Gamma(\varrho)}\int_a^t \left(\frac{(t-a)^q-(s-a)^q}{q}\right)^{\varrho-1} \frac{(\xi_2(s,E_{n-1}(s))-\xi_2(s,E_{n-2}(s)))}{(s-a)^{1-q}}ds,\\
\Upsilon_{E,n} &= I_n - I_{n-1}(0) = \frac{1}{\Gamma(\varrho)}\int_a^t \left(\frac{(t-a)^q-(s-a)^q}{q}\right)^{\varrho-1} \frac{(\xi_3(s,I_{n-1}(s))-\xi_4(s,I_{n-2}(s)))}{(s-a)^{1-q}}ds,\\
\Upsilon_{\mathscr{C},n} &= \mathscr{C}_n - \mathscr{C}_{n-1} = \frac{1}{\Gamma(\varrho)}\int_a^t \left(\frac{(t-a)^q-(s-a)^q}{q}\right)^{\varrho-1} \frac{(\xi_4(s,\mathscr{C}_{n-1}(s))-\xi_3(s,\mathscr{C}_{n-2}(s)))}{(s-a)^{1-q}}ds,\\
\Upsilon_{\mathscr{A},n} &= \mathscr{A}_n - \mathscr{A}_{n-1} = \frac{1}{\Gamma(\varrho)}\int_a^t \left(\frac{(t-a)^q-(s-a)^q}{q}\right)^{\varrho-1} \frac{(\xi_5(s,\mathscr{A}_{n-1}(s))-\xi_2(s,\mathscr{A}_{n-2}(s)))}{(s-a)^{1-q}}ds.
\end{aligned} \tag{17}$$

It is important to note that $S_n = \sum_{i=1}^n \Upsilon_{S,i}, E_n = \sum_{i=1}^n \Upsilon_{E,i}$, $I_n = \sum_{i=1}^n \Upsilon_{I,i}\, \mathscr{C}_n = \sum_{i=1}^n \Upsilon_{\mathscr{C},i}, \mathscr{A}_n = \sum_{i=1}^n \Upsilon_{\mathscr{A},i}$. Also, by using (15)–(17) and fact that $\Upsilon_{S,n-1} = S_{n-1} - S_{n-2}$, $\Upsilon_{E,n-1} = E_{n-1} - E_{n-1}$, $\Upsilon_{I,n-1} = I_{n-1} - I_{n-1}$, $\Upsilon_{\mathscr{C},n-1} = \mathscr{C}_{n-1} - \mathscr{C}_{n-2}$, $\Upsilon_{\mathscr{A},n-1} = \mathscr{A}_{n-1} - \mathscr{A}_{n-2}$, we have

$$\begin{aligned}
\|\Upsilon_{S,n}\| &\le \frac{1}{\Gamma(\varrho)}\omega_1 \int_a^t \left(\frac{(t-a)^q-(s-a)^q}{q}\right)^{\varrho-1} \frac{\|\Upsilon_{S,n-1}\|}{(s-a)^{1-q}}ds,\\
\|\Upsilon_{E,n}\| &\le \frac{1}{\Gamma(\varrho)}\omega_2 \int_a^t \left(\frac{(t-a)^q-(s-a)^q}{q}\right)^{\varrho-1} \frac{\|\Upsilon_{E,n-1}\|}{(s-a)^{1-q}}ds,\\
\|\Upsilon_{I,n}\| &\le \frac{1}{\Gamma(\varrho)}\omega_3 \int_a^t \left(\frac{(t-a)^q-(s-a)^q}{q}\right)^{\varrho-1} \frac{\|\Upsilon_{I,n-1}\|}{(s-a)^{1-q}}ds,\\
\|\Upsilon_{\mathscr{C},n}\| &\le \frac{1}{\Gamma(\varrho)}\omega_4 \int_a^t \left(\frac{(t-a)^q-(s-a)^q}{q}\right)^{\varrho-1} \frac{\|\Upsilon_{\mathscr{C},n-1}\|}{(s-a)^{1-q}}ds,\\
\|\Upsilon_{\mathscr{A},n}\| &\le \frac{1}{\Gamma(\varrho)}\omega_5 \int_a^t \left(\frac{(t-a)^q-(s-a)^q}{q}\right)^{\varrho-1} \frac{\|\Upsilon_{\mathscr{A},n-1}\|}{(s-a)^{1-q}}ds.
\end{aligned} \tag{18}$$

The following theorem will prove the existence of the solution and its uniqueness.

Theorem 7. *Suppose that*

$$\frac{1}{\Gamma(\varrho+1)}\omega_j < 1, \quad i = 1, 2, 3, 4, 5. \tag{19}$$

Then, for every $t \in [0, b]$, a unique solution exists for model (8).

Proof. It is clear that functions $S, E, I, \mathscr{C}$, and $\mathscr{A}$ are bounded. Additionally, from (14) and (15), the symbols ξ_1, ξ_2, ξ_3, ξ_5, and ξ_4 satisfy the Lipchitz condition. Taking (18) along with a recursive hypothesis, we get

$$\begin{aligned}
\|\Upsilon_{S,n}\| &\le \|S_0\|\left(\frac{1}{\Gamma\varrho+1}\right)\omega_1\Big)^n,\\
\|\Upsilon_{E,n}\| &\le \|E_0\|\left(\frac{1}{\Gamma\varrho+1}\right)\omega_2\Big)^n,\\
\|\Upsilon_{I,n}\| &\le \|I_0\|\left(\frac{1}{\Gamma\varrho+1}\right)\omega_3\Big)^n,\\
\|\Upsilon_{\mathscr{C},n}\| &\le \|\mathscr{C}_0\|\left(\frac{1}{\Gamma\varrho+1}\right)\omega_4\Big)^n,\\
\|\Upsilon_{\mathscr{A},n}\| &\le \|\mathscr{A}_0\|\left(\frac{1}{\Gamma\varrho+1}\right)\omega_5\Big)^n.
\end{aligned} \tag{20}$$

As a result, it is evident that $\|\Upsilon_{S,n}\| \longrightarrow 0, \|\Upsilon_{E,n}\| \longrightarrow 0$, $\|\Upsilon_{I,n}\| \longrightarrow 0, \|\Upsilon_{\mathscr{C},n}\| \longrightarrow 0, \|\Upsilon_{\mathscr{A},n}\| \longrightarrow 0$, as, $n \longrightarrow \infty$. More over, from (20) and imposing the triangle, we obtain

$$\begin{aligned}
\|S_{n+k} - S_n\| &\le \sum_{j=n+1}^{n+k} r_1^j = \frac{r_1^{n+1} - r_1^{n+k+1}}{1-r_1},\\
\|E_{n+k} - E_n\| &\le \sum_{j=n+1}^{n+k} r_4^j = \frac{r_4^{n+1} - r_4^{n+k+1}}{1-r_4},\\
\|I_{n+k} - I_n\| &\le \sum_{j=n+1}^{n+k} r_2^j = \frac{r_2^{n+1} - r_2^{n+k+1}}{1-r_2},\\
\|\mathscr{C}_{n+k} - \mathscr{C}_n\| &\le \sum_{j=n+1}^{n+k} r_3^j = \frac{r_3^{n+1} - r_3^{n+k+1}}{1-r_3},\\
\|\mathscr{A}_{n+k} - \mathscr{A}_n\| &\le \sum_{j=n+1}^{n+k} r_2^j = \frac{r_2^{n+1} - r_2^{n+k+1}}{1-r_2}.
\end{aligned} \tag{21}$$

According to (19), $r_i := 1/\Gamma\varrho + 1)\omega_i < 1$. Thus, S_n, E_n, I_n, $\mathscr{C}_n$, and $\mathscr{A}_n$ are Cauchy sequences in the Banach space $B(\mathbb{I})$. This proves that it is uniformly convergent (see [42]). The

limit of (16) as $n \longrightarrow \infty$ confirms the unique solution of these sequences and satisfies model (8). This guarantees the existence of a unique solution for model (8) according to (19). □

5. Disease-Free Equilibrium (DFE)

To calculate the equilibrium point of model (8), we make the model's left side (8) equal to zero as follows:

$$\begin{aligned}
\rho - \iota\left(I + \nu_c\mathscr{C} + \nu_A\mathscr{A}\right)S - dS &= 0,\\
\iota\mu\left(I + \nu_c\mathscr{C} + \nu_A\mathscr{A}\right)S - (\sigma + d)E &= 0,\\
(1-\mu)\iota\left(I + \nu_c\mathscr{C} + \nu_A\mathscr{A}\right)S + \sigma E - \left(\tau_1 + \tau_2 + d\right)I + \lambda\mathscr{C} + \theta\mathscr{A} &= 0,\\
\tau_1 I + (\lambda + d)\mathscr{C} &= 0,\\
\tau_2 I + \left(d + \theta + d_1\right)\mathscr{A} &= 0.
\end{aligned} \tag{22}$$

By solving the above HIV/AIDS model regarding no disease condition, we find the DFE of the model as follows: $E_0 = (S, E, I, \mathscr{C}, \mathscr{A}) = (\rho/d, 0,0,0,0)$.

Theorem 8. *The DFE E_0 ought to achieve $Re(\Psi_j) < 0$, $j = 1, \ldots, 5$ for being locally asymptotically stable (LAS), when Ψ the eigenvalue of the Jacobian matrix calculated at each equilibrium point.*

Proof. To prove the LAS of the DFE point in our model (8), it is necessary to establish that all eigenvalues of the Jacobian matrix J, which is derived from model (8) of equations and evaluated at the DFE $J(E_0)$ point, satisfy a specific requirement.

The next analysis aims to determine the J matrix at the DFE for model (3). Therefore, the Jacobian matrix is denoted by $J(E_0)$:

$$J_{E_0} = \begin{bmatrix} -\rho & 0 & -\iota S^* & -\iota S^*\nu_c & \iota S^*\nu_A \\ 0 & -h_1 & \iota\mu S^* & \mu\iota S^*\nu_c & \iota S^*\nu_A\mu \\ 0 & \sigma & \iota S^*(1-\mu) - h_2 & \lambda + (\mu-1)\iota S^*\nu_c & \theta + (\mu-1)\iota S^*\nu_A \\ 0 & 0 & \tau_1 & -h_3 & 0 \\ 0 & 0 & \tau_2 & 0 & -h_4 \end{bmatrix}, \tag{23}$$

where $S^* = \rho/d, h_1 = (\sigma + d), h_2 = (\tau_1 + \tau_2 + d), h_3 = (\lambda + d), h_4 = (d + \theta + d_1)$. The characteristic polynomial $P(\Psi)$ for $J(E_0)$ is

$$-(d + \Psi)\left(\Psi^4 + B\Psi^3 + H\Psi^2 + M\Psi + N\right) = 0. \tag{24}$$

To simplify, we will symbolize the following:

$$\begin{aligned}
B &= h_1 + h_2 + h_3 + h_4 - \iota\frac{\rho}{d} + \iota\frac{\rho}{d}\mu,\\
H &= h_3h_3 - h_2\left(h_3 + h_4\right) - \sigma\iota\mu\frac{\rho}{d} - \iota h_3\frac{\rho}{d}\\
&\quad + \iota h_3\frac{\rho}{d}\mu - \iota h_4\frac{\rho}{d} + \iota h_3\frac{\rho}{d}\mu\\
&\quad + h_1\left(-h_2 + h_4 + h_4 + \iota(1-\mu)\frac{\rho}{d}\right) - \lambda\tau_1 - \iota\frac{\rho}{d}\nu_c\tau_1\\
&\quad + \iota\frac{\rho}{d}\nu_c\tau_1\mu\left(\theta_\iota(\mu-1)\frac{\rho}{d}\nu_A\right)\tau_2,
\end{aligned}$$

$$
\begin{aligned}
M = {} & -h_2h_3h_4 - \sigma\iota\mu h_3\frac{\rho}{d} - \sigma\iota\mu h_4\frac{\rho}{d} - \iota^2 h_3^2\left(\frac{\rho}{d}\right)^2 h_4^2\mu \\
& - \lambda h_4\tau_1 - \sigma\iota\mu\frac{\rho}{d}\nu_c - \iota h_4\frac{\rho}{d}\nu_c\tau_1 - \iota h_4\frac{\rho}{d}\nu_c\tau_1\mu \\
& - \theta h_3\tau_2 - \sigma\iota\mu\frac{\rho}{d}\nu_A\tau_2 - \iota h_3\frac{\rho}{d}\nu_A\tau_2 - \iota^2 h_1^2\nu_A^2\left(\frac{\rho}{d}\right)^2\tau_2^2\mu \\
& - h_1\left(h_2\left(h_3 + h_4\right)\right) - h_3\left(h_4 + \iota(\mu - 1)\frac{\rho}{d}\right) + \lambda\tau_1 - \iota(1-\mu)\frac{\rho}{d}\left(h_4 + \nu_c\tau_1\right) + \theta\tau_2\Big), \\
Q = {} & h_1\Big(h_4\Big(-h_2h_3 - \lambda\tau_1 + \iota(\mu - 1)\frac{\rho}{d}\left(h_3 + \nu_c\tau_1\right)\Big) \\
& - h_3\left(\theta - \iota(\mu - 1)\right)\frac{\rho}{d}\nu_A\Big)\tau_2\Big) - \sigma\iota\mu)\frac{\rho}{d}\left(h_4\nu_c\tau_1 + h_3\left(h_4 + \nu_A\tau_2\right)\right).
\end{aligned}
\tag{25}
$$

The eigenvalues of $J(E_0)$ are the roots of $P(\Psi)$. Hence, we put

$$-(d + \Psi)\left(\Psi^4 + G\Psi^3 + H\Psi^2 + M\Psi + Q\right) = 0. \tag{26}$$

It follows that $\Psi = -d$ and

$$\Psi^4 + G\Psi^3 + H\Psi^2 + M\Psi + Q = 0. \tag{27}$$

Let us make

$$P(\Psi) = \Psi^4 + G\Psi^3 + H\Psi^2 + M\Psi + Q. \tag{28}$$

To prove that all roots of the polynomial (27) satisfy $|\arg(\Psi_i)| > \gamma\pi/2$, we use the fractional Ruth–Hurwitz criterion. The discriminant $D(p)$ of the polynomial $P(\Psi)$ is given as

$$
\begin{aligned}
D(p) = {} & G^2H^2M^2 - 4H^3M^2 - 4G^3M^3 + 18\mathrm{GHM}^3 \\
& - 27M^4 - 4G^2H^3Q + 16H^4Q + 18G^3\mathrm{HMQ} \\
& - 80GH^2\mathrm{MQ} - 6G^2M^2Q + 144\mathrm{HM}^2Q - 27G^4Q^2 \\
& + 144G^2\mathrm{HQ}^2 - 128M^2Q^2 - 192G\mathrm{MQ}^2 + 256\mathrm{Q}^3.
\end{aligned}
\tag{29}
$$

If the subsequent conditions are satisfied, the solution to polynomial (28) that satisfies $|\arg(\Psi_i)| > \gamma\pi/2$ is correct.

(1) If $D(p) > 0, G \geq 0, H \geq 0, M \geq 0, Q > 0, \gamma < 1/2$, then the solution to polynomial (28) satisfies $|\arg(\Psi_i)| > \gamma\pi/2$ which indicates E_0 is (LAS).

(2) If $D(p) > 0, G < 0, H < 0, M < 0, \gamma > 1/2$, then the solution to polynomial (28) does not satisfy $|\arg(\Psi_i)| > \gamma\pi/2$ which indicates E_0 is unstable.

(3) If $D(p) > 0, G \geq 0, H \geq 0, M \geq 0, \mathrm{GHM} - M^2 = G^2Q$, then the solution to polynomial (28) satisfies $|\arg(\Psi_i)| > \gamma\pi/2$, for $\gamma \in (0,1)$.

(4) If $D(p) < 0, G \geq 0, H \geq 0, M \geq 0, Q > 0, \gamma > 1/2$, then the solution to polynomial (28) does not satisfy $|\arg(\Psi_i)| > \gamma\pi/2$.

(5) If $D(p) < 0, G < 0, H < 0, M < 0, \gamma > 1/2$, then the solution to polynomial (28) does not satisfy $|\arg(\Psi_i)| > \gamma\pi/2$ which indicates E_0 is unstable.

(6) If $D(p) < 0, G \geq 0, H \geq 0, M \geq 0, \mathrm{GHM} - M^2 = G^2Q$, then the solution to polynomial (28) satisfies $|\arg(\Psi_i)| > \gamma\pi/2$, for $\gamma \in (0,1)$. The necessary condition that the solution to polynomial (28) satisfies $|\arg(\Psi_i)| > \gamma\pi/2$ is $Q > 0$ and $Q > 0$, if and only if $R_0 < 1$. □

5.1. Basic Reproduction Number (R_0). R_0 is the number of infected cases due to the transmission of infection from a previously injured person. It can be calculated as explained by Driessche in [43]. By using the relation $R_0 = \lambda(\mathrm{FV}^{-1})$, where λ the spectral radius of the second-generation operator, F and V are the matrices for the new disease class and for the rest of the transitional terms, respectively. The matrices F and V connected to model (8) are given by

$$
\begin{aligned}
E &= \iota\mu\left(I + \nu_c\mathscr{C} + \nu_A\mathscr{A}\right)S - (\sigma + d)E, \\
I &= (1-\mu)\iota\left(I + \nu_c\mathscr{C} + \nu_A\mathscr{A}\right)S + \sigma E \\
&\quad - \left(\tau_1 + \tau_2 + d\right)I + \lambda\mathscr{C} + \theta\mathscr{A}, \\
\mathscr{C} &= \tau_1 I - (\lambda + d)\mathscr{C}, \\
\mathscr{A} &= \tau_2 I - \left(d + \theta + d_1\right)\mathscr{A}.
\end{aligned}
\tag{30}
$$

Set $h_1 = (\sigma + d), h_2 = (\tau_1 + \tau_2 + d), h_3 = (\lambda + d), h_4 = (d + \theta + d_1)$,

$$f = \begin{bmatrix} \iota\mu\left(I + \nu_c\mathscr{C} + \nu_A\mathscr{A}\right)S \\ (1-\mu)\iota\left(I + \nu_c\mathscr{C} + \nu_A\mathscr{A}\right)S \\ 0 \\ 0 \end{bmatrix},$$

$$v = \begin{bmatrix} h_1 E \\ h_2 I - \sigma E - \lambda\mathscr{C} - \theta\mathscr{A} \\ h_3\mathscr{C} - \tau_1 I \\ h_4\mathscr{A} - \tau_2 I \end{bmatrix}, \tag{31}$$

$$F = \begin{bmatrix} 0 & \iota\mu S^* & \iota\mu\nu_c S^* & \iota\mu\nu_A S^* \\ 0 & (1-\mu)\iota S^* & (1-\mu)\iota\nu_A S^* & (1-\mu)\iota\nu_A S^* \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix},$$

and

$$V = \begin{bmatrix} h_1 & 0 & 0 & 0 \\ \sigma & h_2 & -\lambda & -\theta \\ 0 & -\tau_1 & h_3 & 0 \\ 0 & -\tau_2 & 0 & h_4 \end{bmatrix}, \tag{32}$$

and we obtain $R_0 = \lambda(\mathrm{FV}^{-1}) = \rho\iota(\mu\sigma + (1-\mu)h_1)(h_4\nu_c\tau_1 + h_3(h_4 + \nu_A\tau_2))/dh_1(h_4(h_3h_2 - \lambda\tau_1 - h_3\tau_2))$, which is greater than one; by using (Theorem 2, [43]), we can say that the disease can infect the population.

6. Discussion and Numerical Results

This section discusses the numerical simulations for model (8) utilizing the Adams-Moulton approach, an iterative method proposed in [44–46] that yields approximate solutions of fractional-order ordinary differential equations to provide results of simulations for state variables in the model (8). The Cauchy ODE by the LC of the order ϱ has been considered as the following:

$$\begin{aligned} &{}^{c}_{\varrho}D_{0,t}y = f(t, y), \quad \varrho > 0, t \in [0, \mathbb{B}], \\ &y^{(j)}(0) = y^{(j)}_{(0)}, \end{aligned} \tag{33}$$

where $j = 0,1,2,\ldots,\lceil\varrho\rceil - 1$. The mentioned Cauchy initial value problem can be turned into a Volterra integral equation of the second kind as follows:

$$y = \sum_{j=0}^{n-1} y^{(j)}_{(0)}\frac{t^j}{j!} + \frac{1}{\Gamma(\varrho)}\int_0^t (t-\zeta)^{\varrho-1} f(\zeta, y(\zeta))d\zeta, \quad \varrho \in (n-1, n]. \tag{34}$$

To get the repeated approach, we suppose the constant time step size $\Delta t = \mathbb{B}/N, t_j = j\Delta t, j = 0, 1, 2, \ldots, N$ where N is the number of times of integration in the interval $[0, \mathbb{B}]$. Estimating the preceding equation in terms of fractions by taking the differential operator into account the conformable derivative of order q, we get the Adams–Moulton technique [37] for the fractional conformable derivative of order q with LC of order ϱ:

$$y_{n+1} = y(0) + \frac{(\Delta t)^\varrho}{\varrho\Gamma(\varrho)}\sum_{j=0}^{n}\left[(n+1-j)^\varrho - (n-j)^\varrho\right]D^q_{0,t}f\left(t_j, y_j\right), \quad j \in [0, n]. \tag{35}$$

By using the fractional conformable derivative of order q, we get

$$D^q_{0,t}f\left(t_j, y\left(t_j\right)\right) = \frac{1}{t_j^{q-1}}\frac{d}{dt}f\left(t_j, y\left(t_j\right)\right), \quad q > 0. \tag{36}$$

Applying the iterative process described in (35), the HIV model (8) with the fractional conformable derivative of order q with (LC) operator of order β can be expressed as follows:

$$S_{n+1} = S(0) + \frac{(\Delta t)^\varrho}{\varrho\Gamma(\varrho)}\sum_{j=0}^{n}\left[(n+1-j)^\varrho - (n-j)^\varrho\right]D^q_{0,t}f_1\left(t_j, S_j, E_j, I_j, \mathscr{C}_j, \mathscr{A}_j\right),$$

$$E_{n+1} = E(0) + \frac{(\Delta t)^\varrho}{\varrho\Gamma(\varrho)}\sum_{j=0}^{n}\left[(n+1-j)^\varrho - (n-j)^\varrho\right]D^q_{0,t}f_2\left(t_j, S_j, E_j, I_j, \mathscr{C}_j, \mathscr{A}_j\right),$$

$$I_{n+1} = I(0) + \frac{(\Delta t)^\varrho}{\varrho\Gamma(\varrho)}\sum_{j=0}^{n}\left[(n+1-j)^\varrho - (n-j)^\varrho\right]D^q_{0,t}f_3\left(t_j, S_j, E_j, I_j, \mathscr{C}_j, \mathscr{A}_j\right),$$

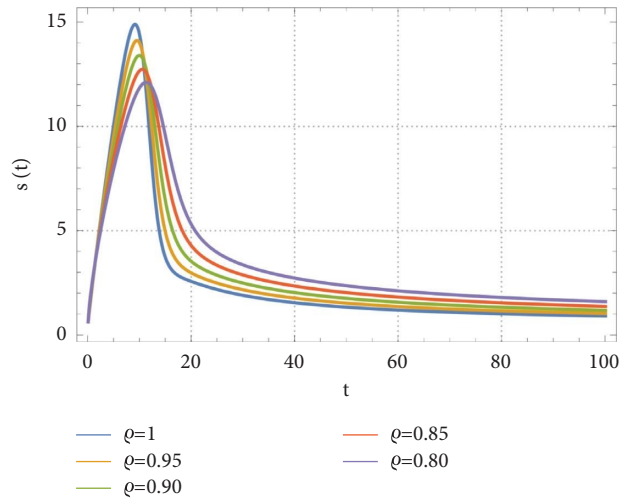

Figure 1: The approximate solution of S of the considered model (8) for a fractional order $q = 0.95$ of FCD and different fractional orders of LC.

$$\begin{aligned}\mathscr{C}_{n+1} &= \mathscr{C}(0) + \frac{(\Delta t)^{\varrho}}{\varrho\Gamma(\varrho)}\sum_{j=0}^{n}\left[(n+1-j)^{\varrho} - (n-j)^{\varrho}\right]D_{0,t}^{q} f_4\left(t_j, S_j, E_j, I_j, \mathscr{C}_j, \mathscr{A}_j\right),\\ \mathscr{A}_{n+1} &= \mathscr{A}(0) + \frac{(\Delta t)^{\varrho}}{\varrho\Gamma(\varrho)}\sum_{j=0}^{n}\left[(n+1-j)^{\varrho} - (n-j)^{\varrho}\right]D_{0,t}^{q} f_5\left(t_j, S_j, E_j, I_j, \mathscr{C}_j, \mathscr{A}_j\right),\end{aligned} \tag{37}$$

where

$$\begin{aligned}f_1\left(t_j, S_j, E_j, \mathscr{C}_j, I_j, \mathscr{A}_j\right) &= \frac{1}{t_j^{q-1}}\left[\rho - \iota\left(I + \nu_c\mathscr{C} + \nu_A\mathscr{A}\right)S - dS\right],\\ f_2\left(t_j, S_j, E_j, \mathscr{C}_j, I_j, \mathscr{A}_j\right) &= \frac{1}{t_j^{q-1}}\left[\iota\mu\left(I + \nu_c\mathscr{C} + \nu_A\mathscr{A}\right)S - (\sigma + d)E\right],\\ f_3\left(t_j, S_j, E_j, \mathscr{C}_j, I_j, \mathscr{A}_j\right) &= \frac{1}{t_j^{q-1}}\left[(1-\mu)\iota\left(I + \nu_c\mathscr{C} + \nu_A\mathscr{A}\right)S + \sigma E\right.\\ &\quad \left. - (\tau_1 + \tau_2 + d)I + \lambda\mathscr{C} + \theta\mathscr{A}\right],\\ f_4\left(t_j, S_j, E_j, \mathscr{C}_j, I_j, \mathscr{A}_j\right) &= \frac{1}{t_j^{q-1}}\left[\tau_1 I + (\lambda + d)\mathscr{C}\right],\\ f_5\left(t_j, S_j, E_j, \mathscr{C}_j, I_j, \mathscr{A}_j\right) &= \frac{1}{t_j^{q-1}}\left[\tau_2 I + (d + \theta + d_1)\mathscr{A}\right].\end{aligned} \tag{38}$$

Throughout simulations, Δt is the magnitude of the time step which is equal to 10^{-3}. The time interval is taken from 0 to 100 and the initial conditions are assumed as 0.6256, 0.03, 0.0222, 0, 0.0522, while the parameter values are given as shown in Table 1.

Our simulations are based on continuous model tracking, where the orders of the derivative are considered at different values.

Figure 1 describes how the susceptible population changes over time; we note that the susceptible cases continue to increase in the first days until 14.88 when $\varrho = 1$, 14.08 when $\varrho = 0.95$, 13.6 when $\varrho = 0.90$, 12.68 when $\varrho = 0.85$, and 11.6 when $\varrho = 0.80$ (FCD) while keeping the q value constant. After fifteen days, the susceptible cases begin to decrease, and as a result of this decrease, there is an increase in $E, I, \mathscr{A}$ as in Figures 3–6. Similarly, in Figure 2, the susceptible cases continue to increase

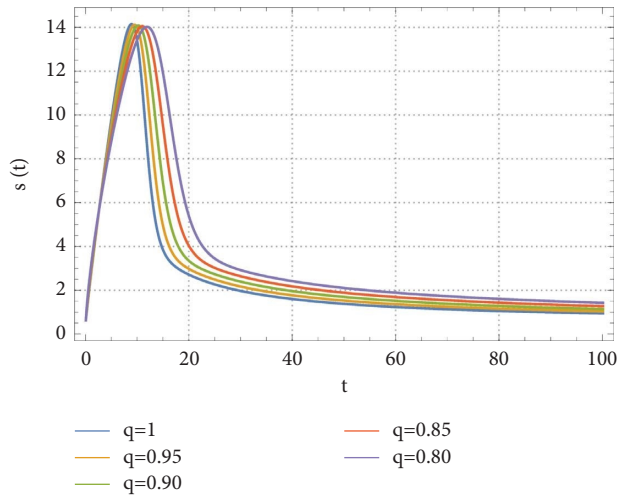

Figure 2: The approximate solution of S of the considered model (8) for different fractional orders of FCD and a fractional order $\varrho = 0.95$ of LC.

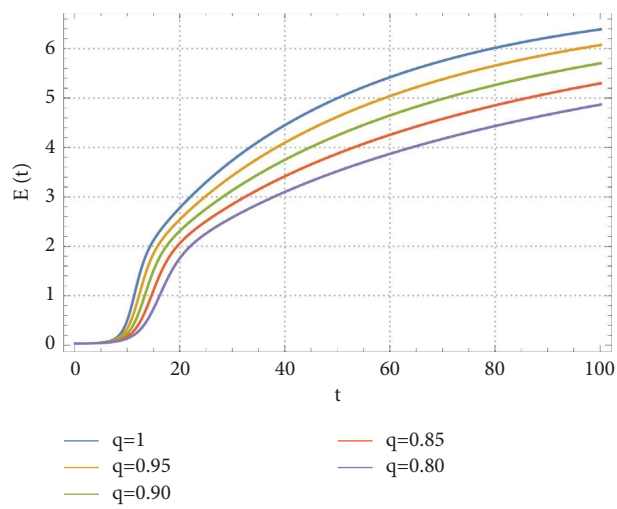

Figure 3: The approximate solution of E of the considered model (8) for different fractional orders of FCD and a fractional order $\varrho = 0.95$ of LC.

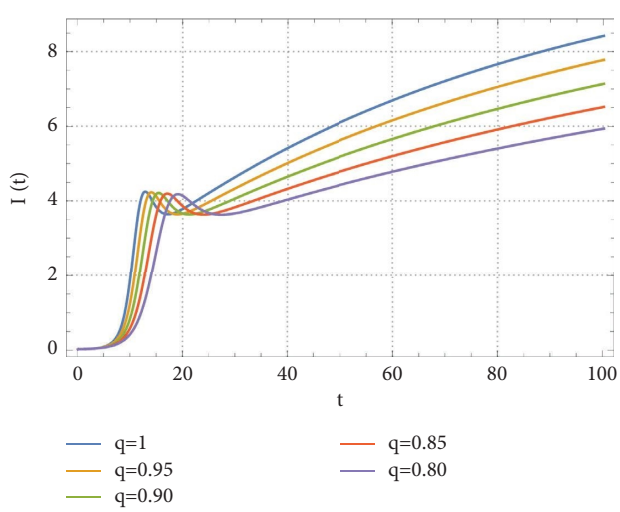

Figure 4: The approximate solution of I of the considered model (8) for different fractional orders of FCD and a fractional order $\varrho = 0.95$ of LC.

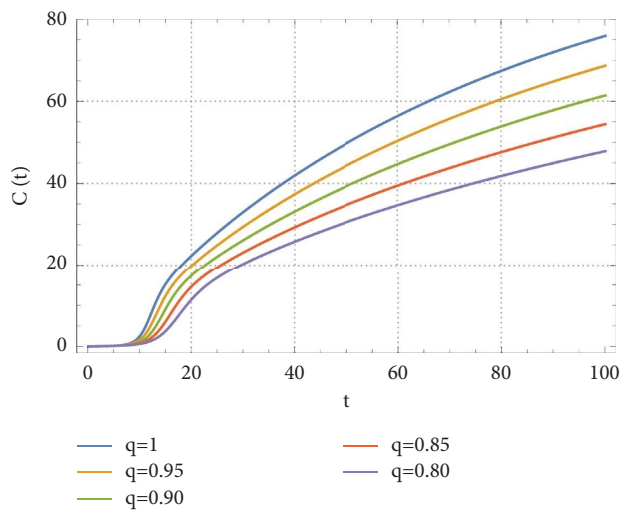

Figure 5: The approximate solution of $\mathscr{C}$ of the considered model (8) for different fractional orders of FCD and a fractional order $\varrho = 0.95$ of LC.

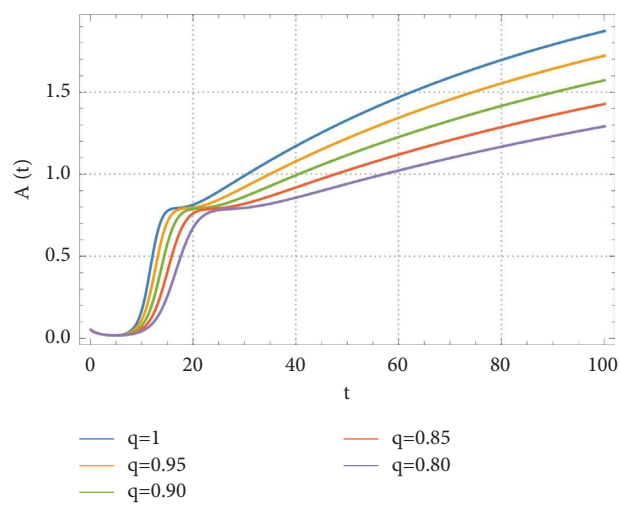

Figure 6: The approximate solution of $\mathscr{A}$ of the considered model (8) for different fractional orders of FCD and a fractional order $\varrho = 0.95$ of LC.

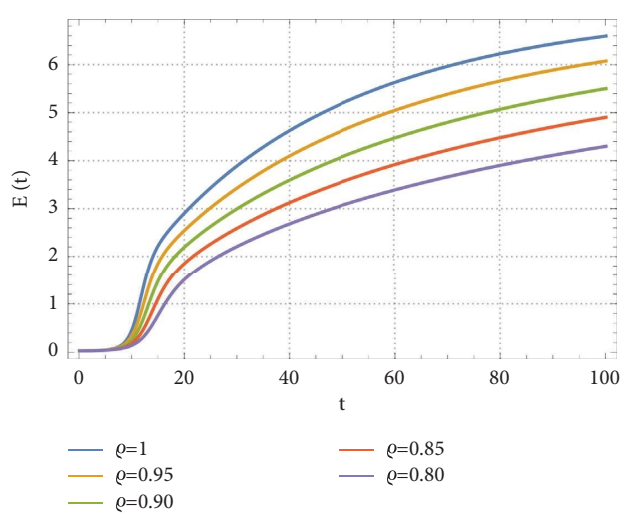

Figure 7: The approximate solution of E of the considered model (8) for a fractional order $q = 0.95$ of FCD and different fractional orders of LC.

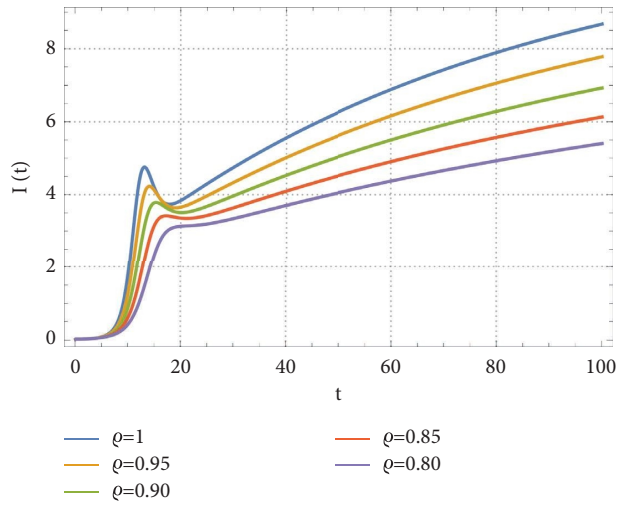

Figure 8: The approximate solution of I of the considered model (8) for a fractional order $q = 0.95$ of FCD and different fractional orders of LC.

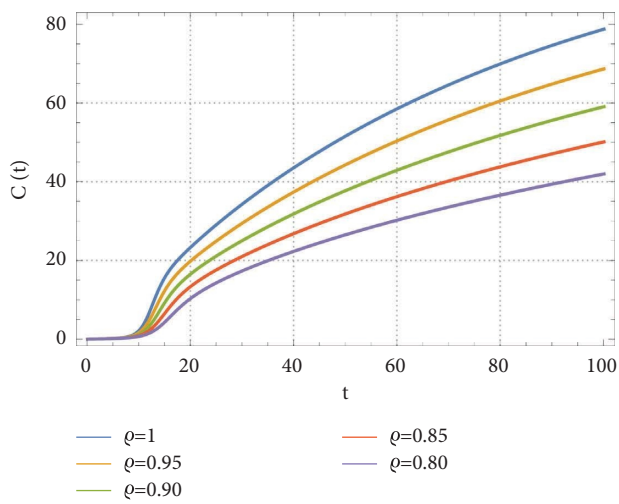

Figure 9: The approximate solution of $\mathscr{C}$ of the considered model (8) for a fractional order $q = 0.95$ of FCD and different fractional orders of LC.

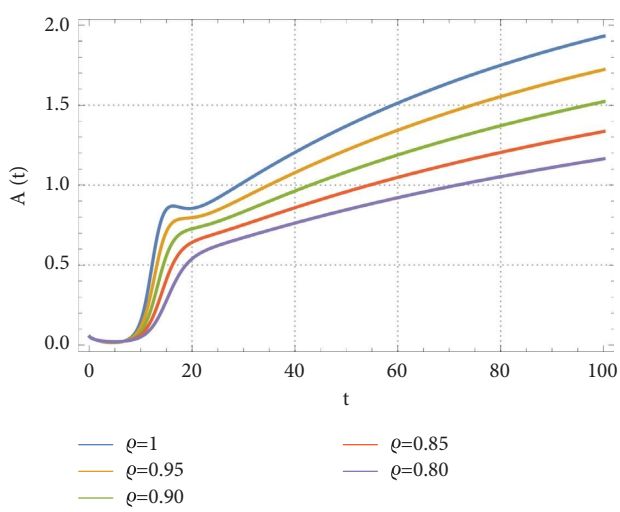

Figure 10: The approximate solution of $\mathscr{A}$ of the considered model (8) for a fractional order $q = 0.95$ of FCD and different fractional orders of LC.

in the first days until 14 for all values of (q) while keeping $\varrho = 0.95$. Following that, it decreases, and, as a result of this decrease, there is an increase in $E, I, \mathscr{A}$ as we can see in Figures 7–10. We note that the approximate method gives us the same results with little difference that is barely noticeable when we use different values for a local derivative $q \in (0, 1]$. In contrast, we note that the increase and decrease in the curves of the categories representing the model come according to the increase or decrease in values of (iterated or fractionalizing index) $\varrho \in (0,1]$. Finally, we note that the values of q do not significantly affect, unlike those caused by the values of ϱ.

7. Conclusion

In this study, the fractional conformable derivative (FCD) of order q in the fractional Liouville–Caputo (LC) sense of order ϱ is applied to develop the model of the transmission dynamics of HIV/AIDS infection. A new fractional HIV/AIDS infection model is presented, with people divided into five classes. Fixed point theorems have been used to investigate the existence and uniqueness of the solutions for the proposed model. The model's basic reproduction number R_0 has been determined. The stability of the model and local stability around the equilibrium in the disease-free case were presented. Using numerical simulations with the aid of the Adams–Moulton approach, the study also investigated the effects of numerous biological characteristics on the system. Numerical simulations for various fractional order values have been carried out, showing that results produced with the FCD-based model are more physiologically reasonable than with models of integer-order cases. Further research can explore applying the conformable fractional derivative-based model to other infectious diseases beyond HIV/AIDS to assess its effectiveness in capturing the dynamics of different diseases. Future studies can focus on refining the model by incorporating more detailed biological characteristics and considering the interactions between different subpopulations, such as high-risk groups or individuals with varying immunity levels.

Data Availability

No underlying data were collected or produced in this work.

Conflicts of Interest

The authors declare that there are no conflicts of interest.

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