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A Qualitative Analysis of Neisseria Gonorrhea Disease with Treatment Effect

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Abstract In this research article, we present a treatment epidemic model for Neisseria Gonorrhea disease which is deadly in its transmission. The model dynamics was studied to understand the epidemic phenomenon and recommend strategies for its control. We carried out a global qualitative analysis for this model and studied the stability of the disease-free and endemic equilibrium using Lyapunov function. The disease-free equilibrium was found to be globally stable when $R_0 < 1$ and the endemic equilibrium is globally stable when $R_0 > 1$. To investigate whether treatment has any effect on the infective, we experimented our model on various sets of treatment parameter values. Our simulations showed that an increase in the treatment rate has significant effect on the infectives.

Keywords Next-generation matrix, Lyapunov function, Neisseria Gonorrhea, Basic Reproduction ratio

1. Introduction

Gonorrhea is one of the sexually lethal transmitted diseases (STD), due to the number of complications that it causes in the infected persons [1]. The disease is caused by Neisseria Gonorrhea, a bacterium. Gonorrhea is formed in the warm, moist area of the reproductive tract such as the cervix, uterus and the fallopian tubes in women and in the urethra in both women and men [3]. The history of Gonorrhea goes down to 1792, in Edinburg where the surgeon Benjamin Bell clearly differentiated it from syphilis infection [16]. Gonorrhea has a lot of complications in the infected persons, and a prolong infection can lead to severe eye infections, infertility in both men and woman, ectopic pregnancy, spontaneous abortion, still births and eventually death if untreated [2].

(WHO, 2012) [15], estimated approximately 106 million new cases of gonorrhea among adults globally. This is just an embodiment of the reported cases from Gonorrhea bacterium infection in that year. The incubation period of the disease is approximately 10 days in women and 2 to 5 days in men, but in rare cases, there can be as long as 30 days in which the infective is asymptomatic [4]. The bacterium also grows in the mouth, throat, eyes and anus [6]. First noticeable signs from the infection in men are often a painful sensation during urination, persistent sore throat and pains in the testicles. Women however don't develop overt signs of the disease. When women have the symptoms, they tend to be mild or similar to other infections, making them more difficult to identify. Some of the symptoms women experience are discharge from the vagina, frequent urination, sore throat and uneasy sensation while urinating [5].

Gonorrhea transmission happens through direct contact with exudates from mucous membranes of infected people through unprotected oral, anal or vaginal sex. Infected people without the symptoms are likely to spread the infection to others if condoms and dental dams are not used during sexual intercourse [7].

Gonorrhea treatment can be achieved through a positive rapid diagnostics and nuclei test, followed by the administration of antibiotics in adolescents and adults [10]. However, drug-resistant strains of the bacterium has recently awakened public health bodies on the area of finding an alternative drugs for the treatment of the disease [8]. We however backed that preventive educational programmes should be embarked upon by all stakeholders in order to prevent and control Gonorrhea in the world by means of safer sexual intercourse.

Since discovered, numerous imperative works have been contributed by mathematical and non-mathematical researchers, and these works have helped immensely in the

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area of vaccine development and control intervention strategy to vaccine manufacturers and public health workers at large. Garnett et al [19], examined the sexual behavior of gonorrhea patients in New York, and used it to estimate the parameters of their gonorrhea model. Their model was used to assess the potential impacts of treatment intervention. Kretzschmar et al [9], proposed a stochastic model for gonorrhea which analyze the underlying structure of sexual contact pattern. They compared the benefits of condom use in an age-structured population of sexually active core group. Prabhakararao [11], analyzed a mathematical model of Gonorrhea disease. They ascertained that the spread of the disease involves interaction of the susceptible and the infective. Leung and Gopalsamy [20], formulated a continuous time SIV model for Gonorrhea transmission among homosexuals. They also used a non-standard discretization method to formulate a discrete time model, and they compared the results of their models. Yorke [12], modelled the spread of Gonorrhea in a population that was categorized into n group and used it to further study the asymptotic stability of the model. Kishore and Pattabhiramacharyulu [13], proposed a simple non-linear first order ODE model for Gonorrhea that measure the growth rates of promiscuous and infective in a homosexual population. They further used numerical examples to explain the effect of cure rate and infective rate on the spread and control of the disease.

Besides the mathematical models, an equally outstanding contribution has been achieved by the non-mathematical models. Karnath [17], discusses the symptoms and signs of Neisseria Gonorrhea with regards to the genitourinary and extra-genital, and outlines laboratory diagnosis with recommended treatment measures. Benedek [16], discusses the unsuccessfulness of various experiments in an attempt to infect animals with Gonorrhea infection as well as history of researches on causes and spread of Gonorrhea in humans over the decades. Bala [18], compared and compiled the resistance trends of Neisseria Gonorrhea across various countries of south-East Asia Region by means of surveillance.

In this research article, we consider a mathematical model of Gonorrhea disease using the S, E, I, T, R compartmental model, where $S(t)$, Susceptible, $E(t)$, Exposed, $I(t)$, Infected, $T(t)$, Treatment, $R(t)$, Removed. We however studied the local and the global stabilities, as well as assessing the effect of treatment on the infective.

2. Model Formulation

The model considers five mutually exclusive compartments of $S(t)$, $E(t)$, $I(t)$, $T(t)$, $R(t)$ of a deterministic ordinary differential equation (ODE), in a mixing homogeneous population. The total population at any time (t), denoted by $N(t)$, is the sum of individual populations in each compartment. Thus $N(t) = S(t) + E(t) + I(t) + T(t) + R(t)$. The model maintains the basic intuition of the SEIR model, with the exception of the introduction of treatment compartment, making it five compartments.

The susceptible population $S(t)$ increases at a recruitment rate πN. Recruitment into the exposed class occurs at a rate of β. The population of the infected individual occurring at a rate α, decreases when an infected individual is treated at a rate δ_1. However, given that treatment is only partially effective, and that, a fraction ρ of the treated individuals recover with partial immunity, then a fraction $q = 1 - \rho$ moves to the latent stage of the infection. We further assume that, treatment of latently infected individuals at a rate δ_2 always results in a recovery. Since treatment only provides partial immunity, individuals treated are reinfected at a rate $\frac{\psi_\beta TI}{N}$. The treated population is further decreased by treated individual recovering at a rate γ. Again, natural death occurs at various compartments at a rate, and hence reduces the population further. Treatment here refers to the process of offering the Gonorrhea infected individual with antibiotic drug Ceftriaxone (Rocephin), in combination with either Azithromycin or doxycycline.

The dynamics of the model is displayed in the figure 1.

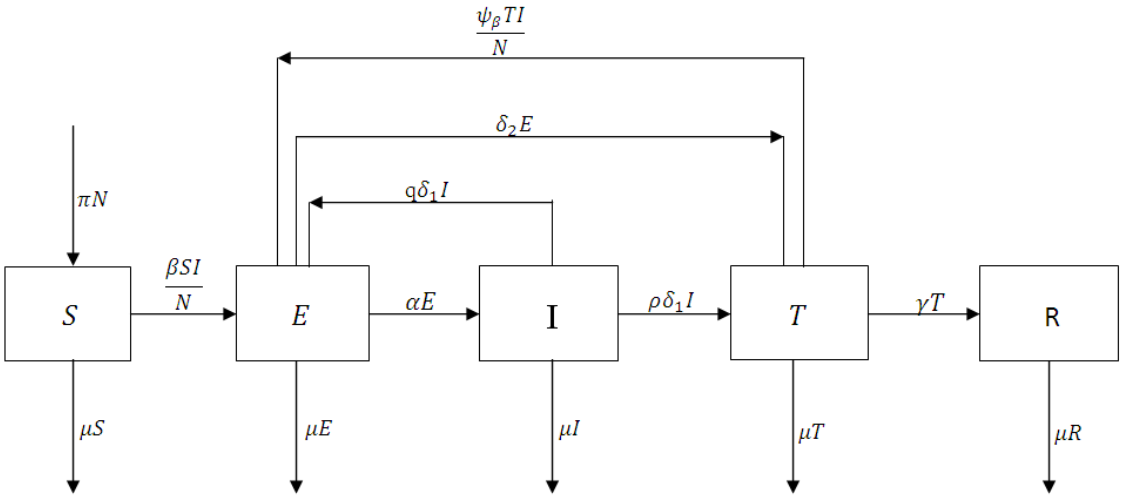

Figure 1. The flowchart showing the dynamics of the model

The non-linear differential equations of the model are given by

$$\frac{dS}{dt} = \pi N - \mu S - \frac{\beta SI}{N} \quad (1)$$

$$\frac{dE}{dt} = \frac{\beta sI}{N} + \frac{\psi_\beta TI}{N} - \mu E - \alpha E - \delta_2 E + q\delta_1 I \quad (2)$$

$$\frac{dI}{dt} = \alpha E - \mu I - q\delta_1 I - \rho\delta_1 I \quad (3)$$

$$\frac{dT}{dt} = \rho\delta_1 T + \delta_2 E - \mu T - \gamma T - \frac{\psi_\beta TI}{N} \quad (4)$$

$$\frac{dR}{dt} = \gamma T - \mu R \quad (5)$$

The above systems are equipped with the initial conditions as follows: $S(0) = S_0,\ E(0) = E_0,\ I(0) = I_0,\ T(0) = T_0$ and $R(0) = R_0$.

The population size $N(t)$ can be determined by $N(t) = S(t) + E(t) + I(t) + T(t) + R(t)$.

Since $R(t) = N(t) - S(t) - E(t) - I(t) - T(t)$, the system (1-5) becomes

$$\frac{dS}{dt} = \pi N - \mu S - \frac{\beta SI}{N} \quad (6)$$

$$\frac{dE}{dt} = \frac{\beta sI}{N} + \frac{\psi_\beta TI}{N} - \mu E - \alpha E - \delta_2 E + q\delta_1 I \quad (7)$$

$$\frac{dI}{dt} = \alpha E - \mu I - q\delta_1 I - \rho\delta_1 I \quad (8)$$

$$\frac{dT}{dt} = \rho\delta_1 T + \delta_2 E - \mu T - \gamma T - \frac{\psi_\beta TI}{N} \quad (9)$$

From Biological point of view, the solution has to be nonnegative. Mathematical properties require that the solution of the system (1)-(5) be studied in a closed set

$$\Gamma = \left\{ \begin{matrix} (S,E,I,T) \in R^4{}_+ | (S+E+I+T) \le \frac{\pi N}{\mu}, \\ S \ge 0, E \ge 0, I \ge 0, T \ge 0 \end{matrix} \right\},$$

The vector field points to the interior of Γ on the part of the boundary when

$(S + E + I + T) = \frac{\pi N}{\mu}$, and is positively invariants. Let Γ^0 and Γ^* represent the boundary and the interior of Γ in R^4 respectively. Then, by direct calculation, it can be shown that the system (6)-(9) has two equilibria in $R^4{}_+$: the disease-free equilibrium $E_0 = (S_0, E_0, I_0, T_0) = \left(\frac{\pi N}{\mu},\ 0,\ 0,\ 0\right) \in \Gamma^0$, and a unique endemic equilibrium $E^* = (S^*, E^*, I^*, T^*) \in \Gamma^*$ where

$$S^* = \frac{\pi N^*}{\left(\mu + \frac{\beta I^*}{N^*}\right)}$$

$$E^* = \frac{I^*(\mu + q\delta_1 + \rho\delta_1)}{\alpha}$$

$$I^* = \frac{\alpha E^*}{(\mu + q\delta_1 + \rho\delta_1)}$$

$$T^* = \frac{\delta_2 E^*}{\left(\mu + \gamma + \frac{\psi_\beta I^*}{N^*} - \rho\delta_1\right)}$$

2.1. The Basic Reproduction Number

The Basic reproduction number or the contact number R_0 is defined as the secondary infection coming from a single infected pathogen that invades a population of only susceptible. If $R_0 \le 1$, it means that E_0 is the only equilibrium in Γ^0. If $R_0 > 1$, the endemic equilibrium exists and is unique in Γ. Our calculation would be based on the next generation matrix, as introduced by Diekmann et al [22], for the R_0. We will use differential equations associated with the exposed (E), infected (I), and treatment (T) compartments given below. We would compute the functions (F) for the rate of new infection term and (V) for the rate of transfer into and out of the exposed, infected, and treatment compartments as indicated in figure 1.

$$\frac{dE}{dt} = \frac{\beta sI}{N} + \frac{\psi_\beta TI}{N} - \mu E - \alpha E - \delta_2 E + q\delta_1 I \quad (10)$$

$$\frac{dI}{dt} = \alpha E - \mu I - q\delta_1 I - \rho\delta_1 I \quad (11)$$

$$\frac{dT}{dt} = \rho\delta_1 T + \delta_2 E - \mu T - \gamma T - \frac{\psi_\beta TI}{N} \quad (12)$$

Hence $f = \begin{bmatrix} \frac{\beta SI}{N} \\ 0 \\ 0 \end{bmatrix}$ and

$$\mathcal{V} = \begin{bmatrix} -\frac{\psi_\beta TI}{N} - q\delta_1 I + \mu E + \alpha E + \delta_2 E \\ -\alpha E + \mu I + q\delta_1 I + \rho\delta_1 I \\ -\rho\delta_1 T - \delta_2 E + \mu T + \gamma T + \frac{\psi_\beta TI}{N} \end{bmatrix}$$

The matrices F and V at $E_0 = (S_0, E_0, I_0, T_0) = \left(\frac{\pi N}{\mu} \quad 0 \quad 0 \quad 0\right)$ are given by

$$F = \begin{bmatrix} 0 & \frac{\beta\pi}{\mu} & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix} \text{ and}$$

$$V = \begin{bmatrix} (\mu + \alpha + \delta_2) & -q\delta_1 & 0 \\ -\alpha & (\mu + q\delta_1 + \rho\delta_1) & 0 \\ -\delta_2 & 0 & (-\rho\delta_1 + \mu + \gamma) \end{bmatrix}$$

Hence the reproduction ratio is given by

$$R_0 = \rho(FV^{-1}) = \begin{bmatrix} \frac{\alpha\beta\pi}{\mu k} & \frac{\beta\pi(\mu+\alpha+\delta_2)}{\mu k} & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix}$$

Where $k = (\mu + \alpha + \delta_2)(\mu + q\delta_1 + \rho\delta_1) + \alpha q\delta_1$

$$R_0 = \frac{\alpha\beta\pi}{\mu(\mu + \alpha + \delta_2)(\mu + q\delta_1 + \rho\delta_1) + \alpha q\delta_1}$$

3. The Disease-free Equilibrium and Its Stability

Theorem 3.1.1: The disease-free equilibrium (E_0) of the system (6)-(9) is locally asymptotically stable in Γ if $R_0 < 1$ and unstable if $R_0 > 1$.

Proof: The disease-free equilibrium has $E_0 = I_0 = T_0 = 0$, hence the solution of the system of equations (6-9) produce a steady state, $E_0 = \left(\frac{\pi N}{\mu}, 0, 0, 0\right)$. The local stability at the point is deduced from the Jacobian of the system (6-9). The Jacobian is given by

$$J(E_0) = \begin{bmatrix} -\left(\mu + \frac{\beta I}{N}\right) & 0 & 0 & 0 \\ \frac{\beta I}{N} & (-\mu - \alpha - \delta_2) & \left(\frac{\beta S}{N} + \frac{\psi_\beta T}{N} + q\delta_1\right) & \frac{\psi_\beta I}{N} \\ 0 & \alpha & (-\mu - q\delta_1 - \rho\delta_1) & 0 \\ 0 & \delta_2 & \frac{-\psi_\beta T}{N} & \left(\rho\delta_1 - \mu - \gamma - \frac{\psi_\beta I}{N}\right) \end{bmatrix}$$

The Jacobian matrix evaluated at $E_0 = \left(\frac{\pi N}{\mu}, 0, 0, 0\right)$ gives

$$J(E_0) = \begin{bmatrix} -\mu & 0 & 0 & 0 \\ 0 & (-\mu - \alpha - \delta_2) & \frac{\beta\pi}{\mu} + q\delta_1 & 0 \\ 0 & \alpha & (-\mu - q\delta_1 - \rho\delta_1) & 0 \\ 0 & \delta_2 & 0 & (\rho\delta_1 - \mu - \gamma) \end{bmatrix}$$

Hence det

$$|E_0 - \lambda I| = \begin{bmatrix} -\mu - \lambda & 0 & 0 & 0 \\ 0 & (-\mu - \alpha - \delta_2) - \lambda & \frac{\beta\pi}{\mu} + q\delta_1 & 0 \\ 0 & \alpha & (-\mu - q\delta_1 - \rho\delta_1) - \lambda & 0 \\ 0 & \delta_2 & 0 & (\rho\delta_1 - \mu - \gamma) - \lambda \end{bmatrix}$$

The characteristic equation of the det $|E_0 - \lambda I|$ is in the form

$$P(\lambda) = (-\mu - \lambda)T(\lambda) = 0 \tag{13}$$

with $T(\lambda)$ being $(\lambda^3 + A_1\lambda^2 + A_2\lambda + A_3)$, where

$$A_1 = \left((-\mu - \alpha - \delta_2) + (-\mu - q\delta_1 - \rho\delta_1) + (\rho\delta_1 - \mu - \gamma)\right)$$

$$A_2 = \left((-\mu - \alpha - \delta_2)(-\mu - q\delta_1 - \rho\delta_1) + (-\mu - \alpha - \delta_2)(\rho\delta_1 - \mu - \gamma) + (-\mu - q\delta_1 - \rho\delta_1)(\rho\delta_1 - \mu - \gamma) - \alpha\left(\frac{\beta\pi}{\mu} + q\delta_1\right)\right)$$

$$A_3 = \left(\left((-\mu - \alpha - \delta_2)(-\mu - q\delta_1 - \rho\delta_1)(\rho\delta_1 - \mu - \gamma)\right) - \left(\alpha\left(\frac{\beta\pi}{\mu} + q\delta_1\right)(\rho\delta_1 - \mu - \gamma)\right)\right)$$

From (13), it is clear that the eigenvalue $\lambda = -\mu$ has a negative real part. The other eigenvalues are all determined by the solution of the characteristic equation

$$\lambda^3 + A_1\lambda^2 + A_2\lambda + A_3 = 0 \tag{14}$$

Hence if $R_0 < 1$, then, according to Hurwitz criterion, the quadratic equation would be with roots with negative real parts and $A_1A_2 > A_3 > 0$. Thus, the local stability of E_0 is asymptotically stable. On the other part, if $R_0 > 1$, then the quadratic equation has only positive roots and the disease-free equilibrium E_0 is unstable.

Theorem 3.1.2: The disease –free equilibrium (E_0) of the system of equations (6)-(9) is globally asymptotically stable in Γ if $R_0 \leq 1$.

Proof: To recognize the global stability of the disease-free equilibrium, we defined a Lyapunov function $L = \alpha\pi v_1 v_2 kS + u_1 u_2 k(\mu(\mu + \alpha + \delta_2)(\mu + q\delta_1 + \rho\delta_1) + \alpha q\delta_1)I$. It follows that $L \geq 0$ along the solution of the system (6)-(9) and $L = 0$ when S and I are zero.

The derivative of L along the solution of (6)-(9) is

$$\frac{dL}{dt} = [\alpha\pi v_1 v_2 kS' + u_1u_2k(\mu(\mu+\alpha+\delta_2)(\mu+q\delta_1+\rho\delta_1)+\alpha q\delta_1)I']\text{s}$$
$$= \left[\alpha\pi v_1 v_2 k\left(\pi N - \mu S - \frac{\beta SI}{N}\right) - u_1u_2k(\mu(\mu+\alpha+\delta_2)(\mu+q\delta_1+\rho\delta_1)+\alpha q\delta_1)(\alpha E - \mu I - q\delta_1 I - \rho\delta_1 I)\right]$$
$$\leq Ik\left[-\frac{\alpha\pi\beta SI}{N} - u_1u_2(\mu(\mu+\alpha+\delta_2)(\mu+q\delta_1+\rho\delta_1)+\alpha q\delta_1)(\mu+q\delta_1+\rho\delta_1)I\right]$$
$$\leq Ik\left[(\mu(\mu+\alpha+\delta_2)(\mu+q\delta_1+\rho\delta_1)+\alpha q\delta_1)\left(\frac{-\alpha\pi\beta S}{N(\mu(\mu+\alpha+\delta_2)(\mu+q\delta_1+\rho\delta_1)+\alpha q\delta_1)} - u_1u_2(\mu+q\delta_1+\rho\delta_1)\right)\right]$$
$$\leq I\left[-\frac{S}{N}R_0 - u_1u_2(\mu+q\delta_1+\rho\delta_1)\right] \text{ using } k = \frac{1}{(\mu(\mu+\alpha+\delta_2)(\mu+q\delta_1+\rho\delta_1)+\alpha q\delta_1)}$$
$$\frac{dL}{dt} \leq I\left[-\frac{S}{N}R_0 - u_1u_2(\mu+q\delta_1+\rho\delta_1)\right] \leq 0 \tag{15}$$

From the inequality (15), $\frac{dL}{dt} = 0$ if and only if $R_0 = -\frac{N}{S}u_1u_2(\mu+q\delta_1+\rho\delta_1)$ and $I = 0$. Thus, by lyapunov-Lasalle's Thereom, all solution that begins in the feasible region where the solutions have biological meaning goes to E_0 as $t \to \infty$. This presupposes that the disease gradually die from the population. Hence, the disease-free equilibrium E_0 is globally asymptotically stable and this proved theorem 3.1.2.

3.1. The Endemic Equilibrium and Its Stability

Theorem 3.2.1 The Endemic equilibrium E^* of the system of equations (6)-(9) is locally stable in Γ if $R_0 > 1$.

Proof: We will linearize the system (6)-(9) around the endemic equilibrium E^*. The matrix of the linearization of the system (6)-(9) at the point $E^* = (S^*, E^*, I^*, T^*)$ is give by

$$J(E^*) = \begin{bmatrix} -\mu - \frac{\beta I}{N} & 0 & 0 & 0 \\ \frac{\beta I}{N} & (-\mu-\alpha-\delta_2) & \left(\frac{\beta S}{N} + \frac{\psi_\beta T}{N} + q\delta_1\right) & \frac{\psi_\beta I}{N} \\ 0 & \alpha & (-\mu - q\delta_1 - \rho\delta_1) & 0 \\ 0 & \delta_2 & -\frac{\psi_\beta T}{N} & \left(\rho\delta_1 - \mu - \gamma - \frac{\psi_\beta I}{N}\right) \end{bmatrix}$$

When the above matrix is evaluated at $J(E^*) = (S^*, E^*, I^*, T^*)$, it produces

$$\begin{bmatrix} -\mu - \frac{\alpha\beta E^*}{N(\mu+q\delta_1+\rho\delta_1)} & 0 & 0 & 0 \\ \frac{\alpha\beta E^*}{N(\mu+q\delta_1+\rho\delta_1)} & (-\mu-\alpha-\delta_2) & \left(\frac{\pi\beta N}{(\mu N+\beta I^*)} + \frac{\psi_\beta\delta_2 E^*}{(\mu N+\gamma N+\psi_\beta I^* - N\rho\delta_1)} + q\delta_1\right) & \left(\frac{\psi_\beta \alpha E^*}{N(\mu+q\delta_1+\rho\delta_1)}\right) \\ 0 & \alpha & (-\mu - q\delta_1 - \rho\delta_1) & 0 \\ 0 & \delta_2 & -\frac{\psi_\beta\delta_2 E^*}{(\mu N+\gamma N+\psi_\beta I^* - N\rho\delta_1)} & \left(\rho\delta_1 - \mu - \gamma - \frac{\psi_\beta \alpha E^*}{N(\mu+q\delta_1+\rho\delta_1)}\right) \end{bmatrix}$$

Hence det $|E^* - \lambda I|$ gives the characteristics equation $(\lambda) = 0$, where

$$P(\lambda) = \lambda^4 + A_1\lambda^3 + A_2\lambda^2 + A_3\lambda + A_4 \tag{16}$$

With

$$A_1 = \left(-\alpha\left(\frac{\pi\beta N}{(\mu N+\beta I^*)} + \frac{\psi_\beta\delta_2 E^*}{(\mu N+\gamma N+\psi_\beta I^* - N\rho\delta_1)} + q\delta_1\right) - \delta_2\left(\frac{\psi_\beta\alpha E^*}{N(\mu+q\delta_1+\rho\delta_1)}\right) + (\mu+\alpha+\delta_2) + \delta_2(-\mu-q\delta_1-\rho\delta_1) + \left(\rho\delta_1 - \mu - \gamma - \frac{\psi_\beta\alpha E^*}{N(\mu+q\delta_1+\rho\delta_1)}\right) + \left(-\mu - \frac{\alpha\beta E^*}{N(\mu+q\delta_1+\rho\delta_1)}\right)\right)$$

$$A_2 = \left(\alpha\left(-\mu - \frac{\alpha\beta E^*}{N(\mu + q\delta_1 + \rho\delta_1)}\right)\left(\frac{\pi\beta N}{(\mu N + \beta I^*)} + \frac{\psi_\beta \delta_2 E^*}{\left(\mu N + \gamma N + \psi_\beta I^* - N\rho\delta_1\right)} + q\delta_1\right)\right.$$
$$+ \delta_2\left(-\mu - \frac{\alpha\beta E^*}{N(\mu + q\delta_1 + \rho\delta_1)}\right)\left(\frac{\psi_\beta \alpha E^*}{N(\mu + q\delta_1 + \rho\delta_1)}\right)$$
$$+ \alpha(-\mu - \alpha - \delta_2)\left(\frac{\pi\beta N}{(\mu N + \beta I^*)} + \frac{\psi_\beta \delta_2 E^*}{\left(\mu N + \gamma N + \psi_\beta I^* - N\rho\delta_1\right)} + q\delta_1\right)$$
$$+ \delta_2(-\mu - \alpha - \delta_2)\left(\frac{\psi_\beta \alpha E^*}{N(\mu + q\delta_1 + \rho\delta_1)}\right) + \alpha\left(\frac{\psi_\beta \alpha E^*}{N(\mu + q\delta_1 + \rho\delta_1)}\right)\left(-\frac{\psi_\beta \delta_2 E^*}{\left(\mu N + \gamma N + \psi_\beta I^* - N\rho\delta_1\right)}\right)$$
$$+ \left(-\mu - \frac{\alpha\beta E^*}{N(\mu + q\delta_1 + \rho\delta_1)}\right)(-\mu - \alpha - \delta_2) + \delta_2(-\mu - q\delta_1 - \rho\delta_1)$$
$$- \alpha\left(\frac{\pi\beta N}{(\mu N + \beta I^*)} + \frac{\psi_\beta \delta_2 E^*}{\left(\mu N + \gamma N + \psi_\beta I^* - N\rho\delta_1\right)} + q\delta_1\right)\left(\rho\delta_1 - \mu - \gamma - \frac{\psi_\beta \alpha E^*}{N(\mu + q\delta_1 + \rho\delta_1)}\right)$$
$$- \delta_2\left(\frac{\psi_\beta \alpha E^*}{N(\mu + q\delta_1 + \rho\delta_1)}\right)(-\mu - q\delta_1 - \rho\delta_1) + (\mu + \alpha + \delta_2)(+\mu + q\delta_1 + \rho\delta_1)$$
$$+ (\mu + \alpha + \delta_2)\left(-\rho\delta_1 + \mu + \gamma + \frac{\psi_\beta \alpha E^*}{N(\mu + q\delta_1 + \rho\delta_1)}\right) + \left(\mu + \frac{\alpha\beta E^*}{N(\mu + q\delta_1 + \rho\delta_1)}\right)(\mu + q\delta_1 + \rho\delta_1)$$
$$\left. + \left(\mu + \frac{\alpha\beta E^*}{N(\mu + q\delta_1 + \rho\delta_1)}\right)\left(-\rho\delta_1 + \mu + \gamma + \frac{\psi_\beta \alpha E^*}{N(\mu + q\delta_1 + \rho\delta_1)}\right)\right)$$

$$A_3 = \left(\alpha\left(-\mu - \frac{\alpha\beta E^*}{N(\mu + q\delta_1 + \rho\delta_1)}\right)(-\mu - \alpha - \delta_2)\left(\frac{\pi\beta N}{(\mu N + \beta I^*)} + \frac{\psi_\beta \delta_2 E^*}{\left(\mu N + \gamma N + \psi_\beta I^* - N\rho\delta_1\right)} + q\delta_1\right)\right.$$
$$+ \left(-\mu - \frac{\alpha\beta E^*}{N(\mu + q\delta_1 + \rho\delta_1)}\right)(-\mu - \alpha - \delta_2)\left(\frac{\psi_\beta \alpha E^*}{N(\mu + q\delta_1 + \rho\delta_1)}\right)$$
$$+ \alpha\left(-\mu - \frac{\alpha\beta E^*}{N(\mu + q\delta_1 + \rho\delta_1)}\right)\left(\frac{\psi_\beta \alpha E^*}{N(\mu + q\delta_1 + \rho\delta_1)}\right)\left(-\frac{\psi_\beta \delta_2 E^*}{\left(\mu N + \gamma N + \psi_\beta I^* - N\rho\delta_1\right)}\right)$$
$$+ \delta_2\left(-\mu - \frac{\alpha\beta E^*}{N(\mu + q\delta_1 + \rho\delta_1)}\right)\left(\frac{\psi_\beta \alpha E^*}{N(\mu + q\delta_1 + \rho\delta_1)}\right)(-\mu - q\delta_1 - \rho\delta_1)$$
$$+ \left(-\mu - \frac{\alpha\beta E^*}{N(\mu + q\delta_1 + \rho\delta_1)}\right)(-\mu - q\delta_1 - \rho\delta_1)\left(\rho\delta_1 - \mu - \gamma - \frac{\psi_\beta \alpha E^*}{N(\mu + q\delta_1 + \rho\delta_1)}\right)$$
$$+ \alpha(-\mu - \alpha - \delta_2)\left(\frac{\pi\beta N}{(\mu N + \beta I^*)} + \frac{\psi_\beta \delta_2 E^*}{\left(\mu N + \gamma N + \psi_\beta I^* - N\rho\delta_1\right)} + q\delta_1\right)\left(\rho\delta_1 - \mu - \gamma\right.$$
$$\left. - \frac{\psi_\beta \alpha E^*}{N(\mu + q\delta_1 + \rho\delta_1)}\right) + \delta_2(-\mu - \alpha - \delta_2)\left(\frac{\psi_\beta \alpha E^*}{N(\mu + q\delta_1 + \rho\delta_1)}\right)(-\mu - q\delta_1 - \rho\delta_1)$$
$$- \left(-\mu - \frac{\alpha\beta E^*}{N(\mu + q\delta_1 + \rho\delta_1)}\right)(-\mu - \alpha - \delta_2)(-\mu - q\delta_1 - \rho\delta_1)$$
$$- \left(-\mu - \frac{\alpha\beta E^*}{N(\mu + q\delta_1 + \rho\delta_1)}\right)(-\mu - \alpha - \delta_2)\left(\rho\delta_1 - \mu - \gamma - \frac{\psi_\beta \alpha E^*}{N(\mu + q\delta_1 + \rho\delta_1)}\right)$$
$$- \alpha\left(-\mu - \frac{\alpha\beta E^*}{N(\mu + q\delta_1 + \rho\delta_1)}\right)\left(\frac{\pi\beta N}{(\mu N + \beta I^*)} + \frac{\psi_\beta \delta_2 E^*}{\left(\mu N + \gamma N + \psi_\beta I^* - N\rho\delta_1\right)} + q\delta_1\right)\left(\rho\delta_1 - \mu - \gamma\right.$$
$$\left. - \frac{\psi_\beta \alpha E^*}{N(\mu + q\delta_1 + \rho\delta_1)}\right) - (-\mu - \alpha - \delta_2)(-\mu - q\delta_1 - \rho\delta_1)\left(\rho\delta_1 - \mu - \gamma - \frac{\psi_\beta \alpha E^*}{N(\mu + q\delta_1 + \rho\delta_1)}\right)$$
$$\left. - \alpha(-\mu - \alpha - \delta_2)\left(\frac{\psi_\beta \alpha E^*}{N(\mu + q\delta_1 + \rho\delta_1)}\right)\left(-\frac{\psi_\beta \delta_2 E^*}{\left(\mu N + \gamma N + \psi_\beta I^* - N\rho\delta_1\right)}\right)\right)$$

$$A_4 = \left(\left(-\mu - \frac{\alpha\beta E^*}{N(\mu + q\delta_1 + \rho\delta_1)}\right)(-\mu - \alpha - \delta_2)(-\mu - q\delta_1 - \rho\delta_1)\left(\rho\delta_1 - \mu - \gamma - \frac{\psi_\beta \alpha E^*}{N(\mu + q\delta_1 + \rho\delta_1)}\right) \right.$$
$$+ \alpha\left(-\mu - \frac{\alpha\beta E^*}{N(\mu + q\delta_1 + \rho\delta_1)}\right)(-\mu - \alpha - \delta_2)\left(\frac{\psi_\beta \alpha E^*}{N(\mu + q\delta_1 + \rho\delta_1)}\right)\left(-\frac{\psi_\beta \delta_2 E^*}{(\mu N + \gamma N + \psi_\beta I^* - N\rho\delta_1)}\right)$$
$$- \alpha\left(-\mu - \frac{\alpha\beta E^*}{N(\mu + q\delta_1 + \rho\delta_1)}\right)(-\mu - \alpha - \delta_2)\left(\frac{\pi\beta N}{(\mu N + \beta I^*)} + \frac{\psi_\beta \delta_2 E^*}{(\mu N + \gamma N + \psi_\beta I^* - N\rho\delta_1)}\right.$$
$$\left. + q\delta_1\right)\left(\rho\delta_1 - \mu - \gamma - \frac{\psi_\beta \alpha E^*}{N(\mu + q\delta_1 + \rho\delta_1)}\right)$$
$$\left. - \delta_2\left(-\mu - \frac{\alpha\beta E^*}{N(\mu + q\delta_1 + \rho\delta_1)}\right)(-\mu - \alpha - \delta_2)\left(\frac{\psi_\beta \alpha E^*}{N(\mu + q\delta_1 + \rho\delta_1)}\right)(-\mu - q\delta_1 - \rho\delta_1)\right)$$

According to Hurwitz criterion [23], the endemic equilibrium E^* is locally asymptotically stable in Γ if $A_i > 0$ for $i = 1, 2, 3, 4$ and $A_1A_2A_3 > {A_3}^2 + {A_1}^2A_4$. Hence if equation (16) satisfy these conditions, then E^* is locally asymptotically stable, otherwise unstable.

Theorem 3.2.2 The endemic equilibrium (E^*) of the system of equations (6)-(9) is globally asymptotically stable in Γ if $R_0 > 1$.

Proof: To see the global stability of the endemic equilibrium, we defined a Lyapunov function $L = k\frac{s}{I}$ [21]. It is easy to verify that $L \geq 0$ along the solution of the system (6)-(9) and $L = 0$ when S and k are zero.

By direct calculation,

$$\frac{dL}{dt} = k\frac{[\dot{S}I - S\dot{I}]}{I^2}$$
$$= \frac{k}{I^2}[\dot{S}I - S\dot{I}]$$
$$\leq \frac{k}{I^2}[\left(\pi N - \mu s - \frac{\beta SI}{N}\right)I$$
$$-S(\alpha E - \mu I - q\delta_1 I - p\delta_1 I)]$$
$$\leq \frac{k}{I^2}[\left(\pi N - \mu s - \frac{\beta SI}{N}\right)I$$
$$-(\alpha SE - \mu SI - q\delta_1 SI - p\delta_1 SI)]$$
$$\leq \frac{k}{I}[\pi N - \mu S - \frac{\beta SI}{N} - \alpha SE + \mu S + q\delta_1 S + p\delta_1 S]$$
$$\leq \tfrac{k}{I}[\pi N - \tfrac{\beta SI}{N} - \alpha SE + q\delta_1 S + p\delta_1 S] \quad \text{setting}$$
$(k = I^2)$ gives
$$\leq I[s\left(\tfrac{-\beta I}{N} - \alpha E + q\delta_1 + p\delta_1\right) + \pi N] \qquad (17)$$

It can be deduced from inequality (17) that when $R_0 > 1$, the derivative $\frac{dL}{dt} = 0$ if and only $I = 0$. Consequently, the largest compact invariant set in $\left\{(S^*, E^*, I^*, T^*) \in \Gamma : \frac{dL}{dt} = 0\right.$ is the singleton $E*$, where $E*$ is the endemic equilibrium. Hence by LaSalle's invariant principle, it implies that E^* is globally asymptotically stable in Γ.

4. Simulation

The system (6)-(9) was simulated with various set of parameter values using Matlab. We have investigated the stability of the disease-free and the endemic equilibrium using linearization approach and Lyapunov functions for both the local and global stability respectively. We have described in figure 4-6, the effect of treatment on the infective; thus reducing the number of infections in the treated class.

To understand the dynamics of the model, our system of equations (6)-(9) were simulated using the parameter values $\psi_\beta = 0.3$, $\delta_1 = 0.6$, $\delta_2 = 0.8$, $\alpha = 0.6$, $\beta = 2.4$, $\pi = 0.8$, $\mu = 0.5$, $\gamma = 0.5$, $N = 1000$, $\rho = 0.3$. The simulated result of figure 1, shows a sudden rise of the infectives in the early stages of the epidemic, and slows down gradually until it reaches its equilibrium. The susceptible however, decreases sharply and meets the infective at 5 and 12 days where they produce equilibrium points, and maintains its equilibrium thereafter.

5. Conclusions

As stated in the introduction, our main purpose is to study the dynamics of deadly Gonorrhea disease in order to understand the epidemic phenomenon and recommend strategies for its control. We have therefore proposed a treatment epidemic model for Neisseria Gonorrhea disease which is deadly when is not properly treated by the infected person, due to it complicated effects in the host. We have carried out a global qualitative analysis for this model and studied the stability of the disease-free and endemic equilibrium. In terms of the basic reproduction number $R_0 = \frac{\alpha\beta\pi}{\mu(\mu+\alpha+\delta_2)(\mu+q\delta_1+\rho\delta_1)+\alpha q\delta_1}$, our results shows that when $R_0 < 1$, the disease-free equilibrium is globally stable and when $R_0 > 1$, the endemic equilibrium is globally stable.

To determine whether treatment has any effect on the infective, we experimented our model on various sets of treatment parameter values. Our simulations showed that an increase in the treatment rate has significant effect on the

infective. Thus it reduces infection in the treated class. Hence we recommend that Gonorrhea treatment should be sought at the early stages of the infection to prevent further complications.

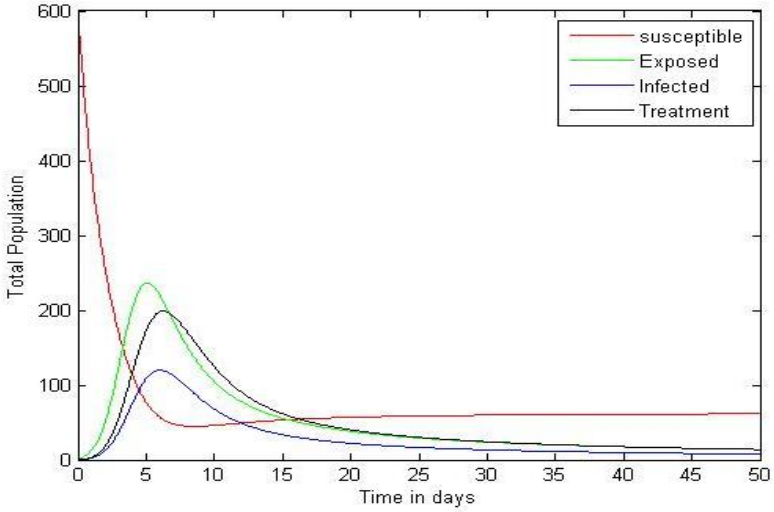

Figure 1. Plot showing the dynamics of the model

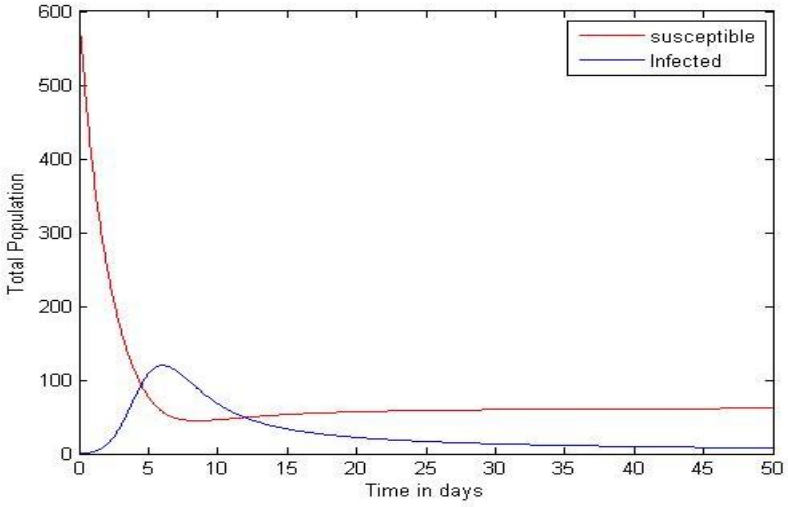

Figure 2. Plot showing the dynamics of the Susceptible and Infected

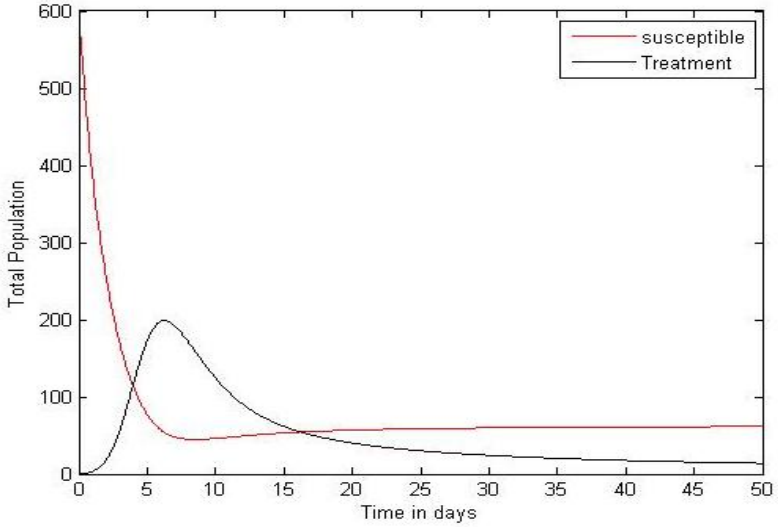

Figure 3. Plot showing the dynamics of the Susceptible and Treatment

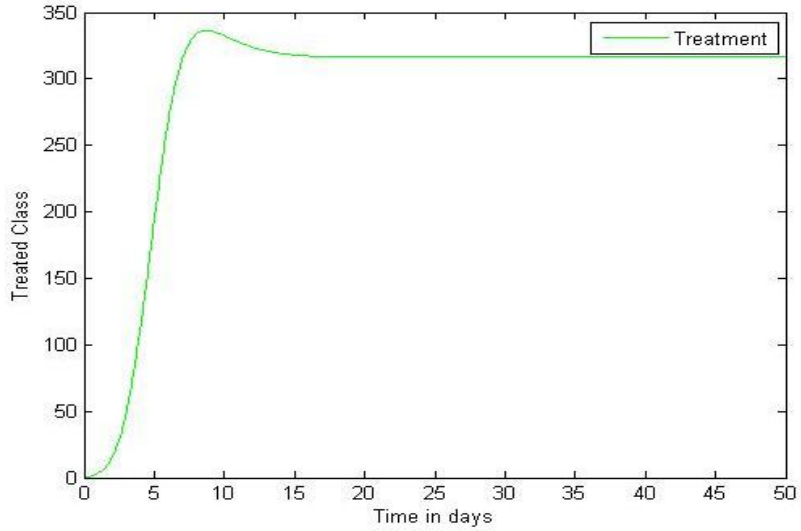

Figure 4. Disease infections in the treated class with a treatment rate $\delta_1 = 0.5$

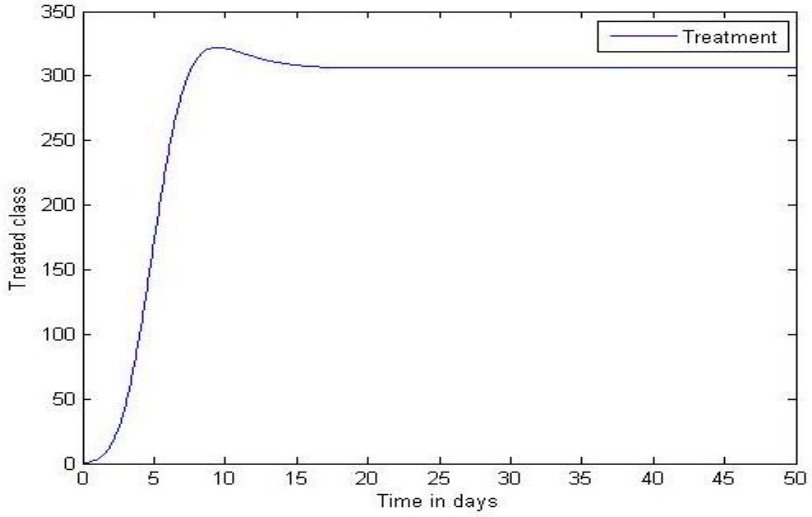

Figure 5. Disease infections in the treated class with a treatment rate $\delta_1 = 0.6$

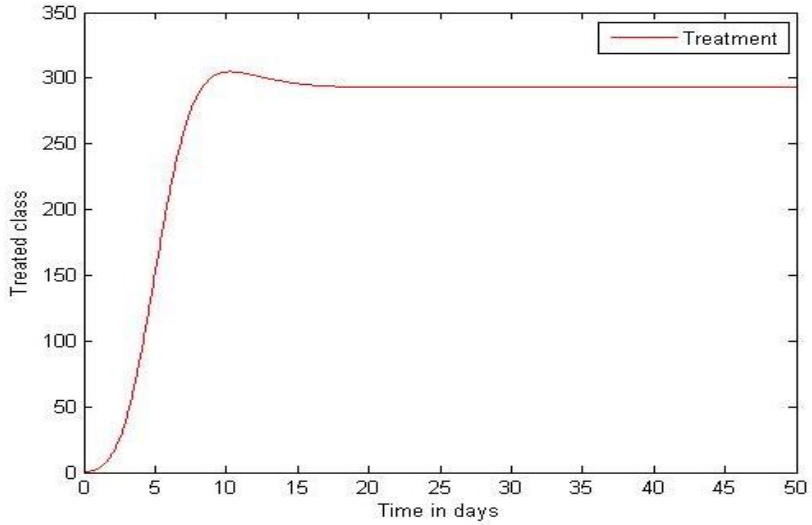

Figure 6. Disease infections in the treated class with a treatment rate $\delta_1 = 0.7$

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