A Mathematical Model for SIS Cholera Epidemic with Quarantine Effect

Deepti Mokati[1], Viqar Hussain Badshah[1], Nirmala Gupta[2]

[1]School of Studies in Mathematics, Vikram University, Ujjain, India

[2]Government Girls P. G. College, Vikram University, Ujjain, India

Email address:
mahe.deepti@gmail.com (D. Mokati)

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Abstract: Cholera was prevalent in the U.S. in the 1800s, before modern water and sewage treatment systems eliminated its spread by contaminated water. Cholera is an acute intestinal infectious disease caused by the bacterium vibrio cholerae. We propose and analyse a mathematical model for cholera considering quarantine. Quarantine plays an important role to control the disease. Our goal is to control the disease through the quarantine even if infected population again becomes suscepted. Determine two equilibrium points of the model: disease-free and endemic. Also basic reproduction number R_q is obtained. Reproduction number plays as a key role for analyzing stability for disease-free and endemic equilibrium points. Stability has been discussed for both equilibrium points using Ruth-Hurwitz criterian. We concluded that the disease-free and endemic equilibria are locally asymptotically stable if $R_q<1$ and $R_q>1$ respectively. Also, Numerical simulations are carried out for the model. From the graphically representation it is more clearly seen that when the disease becomes dies out and when it persistence.

Keywords: SIS, Quarantine, Equilibrium, Stability, Ruth-Hurwitz Criteria, Reproduction Number

1. Introduction

Cholera is an infection of the small intestine by some strains of the bacterium Vibrio cholerae. When the bacteria attaches to the small intestine's walls, human's body begins to secrete large amounts of water that lead to diarrhea and rapid loss of fluids and salts. Contaminated water supplies are the primary source of cholera infection. Uncooked fruits, vegetables, and other foods can also contain the bacteria that cause cholera. Cholera is an ancient disease that continuous to cause epidemic and pandemic infection despite ongoing efforts to limit its spread. Wang J. and Modnak C. have been formulated a cholera mathematical model with control measures which represents a coupling between multiple transmission pathways of cholera and multiple control measures [9]. In spite of the recent progress of medical sciences, cholera still remains as a severe global threat in view of more morbidity or mortality and its currently spreading in countries such as Zimbabwe and other parts of the world. Das P. and Mukherjee D. focused to the role of lytic bacteriophage in the cyclic behavior of cholera outbreaks [4]. Emvudu Y. and Kokomo E. considered a cholera epidemic mathematical model of a closed population in [5]. A nonlinear delayed SIRS cholera mathematical model with immigration for the spread of the disease with carriers in the environment have been proposed and analyzed by Agarwal M. and Verma V [1]. One useful method to control the spread of infectious diseases is to isolate some infectives, in order to reduce transmissions of the infection to susceptibles. Pang Y. et al. discussed the dynamics of a stochastic SIQS epidemic model [8]. The cholera outbreak began in 2010 in Haiti reminds us the importance of cholera prevention, treatment and control that has eliminated the disease from much of the developed world. Chun I. and Fung H. gave a brief introduction of the cholera transmission dynamic models and discussed that how the models can be modified and focused on the model structure, impact of water, sanitation using modeling and model misspecification and parameter uncertainty [2]. Authors proposed a SVR-B cholera model with vaccination [3]. A mathematical model of cholera has been proposed to see the impact of vaccination, therapeutic treatment on the transmission dynamics of

cholera infection in a varying population [6]. Many mathematical models have been proposed by many researchers to investigate the complex epidemic and endemic behavior of cholera. The effects of vaccination on the transmission of cholera models are also studied by many authors. Nirwani N. et al. proposed a SIQR-B cholera epidemic model. They studied and analyzed the effects of quarantine and incidence on the spread of cholera disease [7].

2. The Mathematical Model

We have referred Nirwani N. et al. [7] and modified it by adding transmission rate ωI. Considered an SIS cholera epidemic model with quarantine effect. In the model human population is divided into classes containing susceptible (S), infectious (I), quarantine (Q) and recovered (R) individualsat time t. The pathogen population at time t is given by $B(t)$. Now consider the total number of population at time t is $S+I+Q+R=1$. When there is an adequate contact of a susceptible with an infective, the susceptible becomes infected and leaves the class S. Hence infected individuals enter the class I of infectious people and have a full disease case of an infectious disease. After sometime, infected people leaves the class I. There are two possibilities for the infected peoples: (1) Upon recovery they enter the class R and (2) goes back through an immediate returning path ωI to the susceptible class. There are two ways to recover the infected peoples: Firstly, they can be recovered directly with the transmission rate αI and secondly, they can be recovered through quarantine class Q with transmission rate δI which imposed the disease and then move to class R with transmission rate εQ, otherwise they will be susceptible again. This model is called an SIS model with quarantine effect.

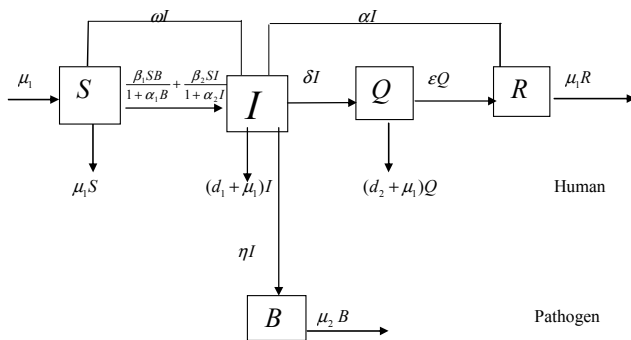The flow of individual is depicted in the following transfer diagram (Figure 1):

Figure 1. *Transfer diagram for SIS cholera model.*

The symbols are used here stands for
μ_1 = Natural human birth and death rate,
β_1, β_2 = Contact rates for the human-environment & human-human
   interactions respectively,
α_1, α_2 = Constant rates,

d_1, d_2 = Disease related death rate constant in I & Q respectively,
α = Recovery rate from the disease,
δ = Transmission rate between compartments I to Q,
ε = Transmission rate between compartments Q to R,
η = Rate of human contribution to the growth of the pathogen,
μ_2 = Death rate of the pathogen in the environment,
ω = Disease transmission rate from compartment I to S.
All parameters are assumed nonnegative.

3. Formulation of the Model

The differential equations corresponding to the transfer diagram are

$$\frac{dS}{dt} = \mu_1 + \omega I - \frac{\beta_1 S B}{1+\alpha_1 B} - \frac{\beta_2 S I}{1+\alpha_2 I} - \mu_1 S$$

$$\frac{dI}{dt} = \frac{\beta_1 S B}{1+\alpha_1 B} + \frac{\beta_2 S I}{1+\alpha_2 I} - (d_1 + \mu_1 + \delta + \alpha + \omega) I$$

$$\frac{dQ}{dt} = \delta I - (\varepsilon + d_2 + \mu_1) Q \qquad (1)$$

$$\frac{dR}{dt} = \alpha I + \varepsilon Q - \mu_1 R$$

$$\frac{dB}{dt} = \eta I - \mu_2 B$$

Since equations first, second, third and fifth of system (1) are independent of the variable R, therefore, the system can be rewritten as

$$\frac{dS}{dt} = \mu_1 + \omega I - \frac{\beta_1 S B}{1+\alpha_1 B} - \frac{\beta_2 S I}{1+\alpha_2 I} - \mu_1 S$$

$$\frac{dI}{dt} = \frac{\beta_1 S B}{1+\alpha_1 B} + \frac{\beta_2 S I}{1+\alpha_2 I} - (d_1 + \mu_1 + \delta + \alpha + \omega) I \qquad (2)$$

$$\frac{dQ}{dt} = \delta I - (\varepsilon + d_2 + \mu_1) Q$$

$$\frac{dB}{dt} = \eta I - \mu_2 B$$

The feasible region of human population D corresponding to the system (2) will be

$$D = \{(S, I, Q) : S \geq 0, I \geq 0, Q \geq 0, S+I+Q \leq 1\}$$

and the feasible region of pathogen Ω will be

$$\Omega = \{B : B \geq 0\}$$

Thus, the proposed model is mathematically well posed and is epidemiologically reasonable, since all of the fractions remain between 0 and 1.

4. Derivation of the Model

4.1. Equilibrium Points

There are two types of equilibrium points: disease-free and endemic. First of all, we determine disease-free equilibrium points.

4.1.1. Disease-free Equilibrium (E^0)

All the equations of the system (2) equate to zero, then the system of equations becomes

$$\mu_1 + \omega I - \frac{\beta_1 SB}{1+\alpha_1 B} - \frac{\beta_2 SI}{1+\alpha_2 I} - \mu_1 S = 0$$

$$\frac{\beta_1 SB}{1+\alpha_1 B} + \frac{\beta_2 SI}{1+\alpha_2 I} - (d_1 + \mu_1 + \delta + \alpha + \omega)I = 0 \qquad (3)$$

$$\delta I - (\varepsilon + d_2 + \mu_1)Q = 0$$

$$\eta I - \mu_2 B = 0$$

Assume that if the disease is not occurring, then $I = 0$.

Now, from equations third and fourth of system (3), we have

$$Q = 0 \text{ and } B = 0.$$

At $I = 0$ and $B = 0$ from equation first of system (3), we get $S = 1$.

Thus, the disease-free equilibrium is $E^0 = (1, 0, 0, 0)$.

4.1.2. Endemic Equilibrium (E^*)

$$\left\{ \left[\frac{\beta_1 \eta}{\mu_2 + \alpha_1 \eta I^*} + \frac{\beta_2}{1+\alpha_2 I^*} \right] \frac{\left[\mu_1 - (d_1 + \mu_1 + \alpha + \delta)I^*\right]}{\mu_1} - (d_1 + \mu_1 + \alpha + \delta + \omega) \right\} I^* = 0.$$

It means that either $I^* = 0$ or

$$\left[\frac{\beta_1 \eta}{\mu_2 + \alpha_1 \eta I^*} + \frac{\beta_2}{1+\alpha_2 I^*} \right] \frac{\left[\mu_1 - (d_1 + \mu_1 + \alpha + \delta)I^*\right]}{\mu_1} - (d_1 + \mu_1 + \alpha + \delta + \omega) = 0.$$

But in case of endemic $I^* \neq 0$, so we have

$$\left[\frac{\beta_1 \eta}{\mu_2 + \alpha_1 \eta I^*} + \frac{\beta_2}{1+\alpha_2 I^*} \right] \frac{\left[\mu_1 - (d_1 + \mu_1 + \alpha + \delta)I^*\right]}{\mu_1} - (d_1 + \mu_1 + \alpha + \delta + \omega) = 0.$$

Or $\dfrac{\mu_1 - (d_1 + \mu_1 + \alpha + \delta)I^*}{\mu_1} = \dfrac{(d_1 + \mu_1 + \alpha + \delta + \omega)}{\left[\dfrac{\beta_1 \eta}{\mu_2 + \alpha_1 \eta I^*} + \dfrac{\beta_2}{1+\alpha_2 I^*} \right]}$.

We assume that $g_1(I^*) = g_2(I^*)$,

where $g_1(I^*) = \dfrac{\mu_1 - (d_1 + \mu_1 + \alpha + \delta)I^*}{\mu_1}$ and $g_2(I^*) = \dfrac{(d_1 + \mu_1 + \alpha + \delta + \omega)}{\left[\dfrac{\beta_1 \eta}{\mu_2 + \alpha_1 \eta I^*} + \dfrac{\beta_2}{1+\alpha_2 I^*} \right]}$.

Here, we find the endemic equilibrium points and discuss the uniqueness of the points. For this, the system (2) can be written as

$$\mu_1 + \omega I^* - \frac{\beta_1 S^* B^*}{1+\alpha_1 B^*} - \frac{\beta_2 S^* I^*}{1+\alpha_2 I^*} - \mu_1 S^* = 0$$

$$\frac{\beta_1 S^* B^*}{1+\alpha_1 B^*} + \frac{\beta_2 S^* I^*}{1+\alpha_2 I^*} - (d_1 + \mu_1 + \alpha + \delta + \omega)I^* = 0 \qquad (4)$$

$$\delta I^* - (d_2 + \mu_1 + \varepsilon)Q^* = 0$$

$$\eta I^* - \mu_2 B^* = 0$$

From equations third and fourth equation of system (4), we get the values of Q^* and B^* such that

$$Q^* = \frac{\delta I^*}{(\varepsilon + \mu_1 + d_2)} \text{ and } B^* = \frac{\eta I^*}{\mu_2}$$

From first and second equation of system (4), we get

$$S^* = \frac{\mu_1 - (d_1 + \mu_1 + \alpha + \delta)I^*}{\mu_1}$$

Again, from second equation of system (4),

$$\left[\frac{\beta_1 \eta}{\mu_2 + \alpha_1 \eta I^*} + \frac{\beta_2}{1+\alpha_2 I^*} \right] S^* I^* - (d_1 + \mu_1 + \alpha + \delta + \omega)I^* = 0 \qquad (5)$$

Substituting the value of S^* in equation (5),

Now, for determining the uniqueness of I^*, we assume that $I = I^*$, then

$$g_1(I) = \frac{\mu_1 - (d_1 + \mu_1 + \alpha + \delta)I}{\mu_1} \text{ and } g_2(I) = \frac{(d_1 + \mu_1 + \alpha + \delta + \omega)}{\left[\dfrac{\beta_1 \eta}{\mu_2 + \alpha_1 \eta I} + \dfrac{\beta_2}{1+\alpha_2 I} \right]}.$$

If $I = 0$, then $g_1(0) = 1$ and $g_2(0) = \dfrac{\mu_2(d_1 + \mu_1 + \alpha + \delta + \omega)}{(\beta_1 \eta + \beta_2 \mu_2)}$.

If $I > 0$ then $g_1(I) < 0$ and $g_2(I) > 0$.

Thus, we see that $g_2(I)$ is increasing function for $I \geq 0$.

Hence, basic reproduction number R_q is given by

$$R_q = \frac{g_1(0)}{g_2(0)}.$$

or

$$R_q = \frac{(\beta_1 \eta + \beta_2 \mu_2)}{\mu_2(d_1 + \mu_1 + \alpha + \delta + \omega)}. \tag{6}$$

When $R_q > 1$, then $g_2(0) < g_1(0) = 1$ that is $g_2(0) < 1$.

Hence, there is a unique solution of I^* to the equation $g_1(I^*) = g_2(I^*)$

Thus, endemic points   are given by

$$E^* = \left(\frac{\mu_1 - (d_1 + \mu_1 + \alpha + \delta) I^*}{\mu_1}, I^*, \frac{\delta I^*}{\varepsilon + \mu_1 + d_2}, \frac{\eta I^*}{\mu_2} \right),$$

where I^* can be determine by $g_1(I^*) = g_2(I^*)$.

4.2. Stability Analysis

In this section, we have analyzed the stability of disease-free and endemic equilibrium by proving the theorem 1 and theorem 2 respectively.

4.2.1. Stability of Disease-free Equilibrium

Theorem 1. If $R_q < 1$, then the disease-free equilibrium is locally asymptotically stable.

Proof. The variational matrix will be

$$J(E^0) = \begin{bmatrix} \frac{-\beta_1 B}{1 + \alpha_1 B} - \frac{\beta_1 I}{1 + \alpha_2 I} - \mu_1 & \frac{-\beta_2 S}{(1 + \alpha_2 I)^2} + \omega & 0 & \frac{-\beta_1 S}{(1 + \alpha_1 B)^2} \\ \frac{\beta_1 B}{1 + \alpha_1 B} + \frac{\beta_1 I}{1 + \alpha_2 I} & \frac{\beta_2 S}{(1 + \alpha_2 I)^2} - (d_1 + \mu_1 + \alpha + \delta + \omega) & 0 & \frac{\beta_1 S}{(1 + \alpha_1 B)^2} \\ 0 & \delta & -(\varepsilon + \mu_1 + d_2) & 0 \\ 0 & \eta & 0 & -\mu_2 \end{bmatrix}.$$

At disease-free equilibrium points, the matrix will be

$$J(E^0) = \begin{bmatrix} -\mu_1 & \omega - \beta_2 & 0 & -\beta_1 \\ 0 & \beta_2 - (d_1 + \mu_1 + \delta + \alpha + \omega) & 0 & \beta_1 \\ 0 & \delta & -(\varepsilon + d_2 + \mu_1) & 0 \\ 0 & \eta & 0 & -\mu_2 \end{bmatrix}.$$

Now, the characteristic equation will be

$$\left| J(E^0) - z I \right| = 0.$$

$$\Rightarrow \begin{vmatrix} -\mu_1 - z & \omega - \beta_2 & 0 & -\beta_1 \\ 0 & \beta_2 - (d_1 + \mu_1 + \delta + \alpha + \omega + z) & 0 & \beta_1 \\ 0 & \delta & -(\varepsilon + d_2 + \mu_1 + z) & 0 \\ 0 & \eta & 0 & -\mu_2 - z \end{vmatrix} = 0.$$

On simplification, we have

$$(\varepsilon + d_2 + \mu_1 + z)(\mu_1 + z)\{ z^2 + (\mu_2 + d_1 + \mu_1 + \delta + \alpha + \omega - \beta_2)z + \mu_2(d_1 + \mu_1 + \delta + \alpha + \omega) - (\mu_2\beta_2 + \beta_1\eta)\} = 0.$$

It is clear that there are four eigen values corresponding to the characteristic equation. First two are given by $z = -\mu_1$ and $z = -(\varepsilon + \mu_1 + d_2)$ which are negative.

where,

Remaining two eigen values are obtaining by the following equation

$$z^2 + (\mu_2 + d_1 + \mu_1 + \delta + \alpha + \omega - \beta_2) z + \mu_2(d_1 + \mu_1 + \delta + \alpha + \omega) - (\mu_2\beta_2 + \beta_1\eta) = 0.$$

which can be written as

$$z^2 + a_1 z + a_2 = 0$$

where,

$$a_1 = (\mu_2 + d_1 + \mu_1 + \delta + \alpha + \omega - \beta_2),$$

$$a_2 = \mu_2(d_1 + \mu_1 + \delta + \alpha + \omega) - (\mu_2\beta_2 + \beta_1\eta) = \mu_2(d_1 + \mu_1 + \delta + \alpha + \omega)\left[1 - \frac{\mu_2\beta_2 + \beta_1\eta}{\mu_2(d_1 + \mu_1 + \delta + \alpha + \omega)}\right]$$

$$= 1 - R_q.$$

where, $R_q = \dfrac{(\beta_1\eta + \beta_2\mu_2)}{\mu_2(d_1 + \mu_1 + \alpha + \delta + \omega)} < 1.$

Hence, it is clearly seen that

$a_1 > 0$, $a_2 > 0$ and $a_1 a_2 > 0$.

Thus, by the Routh-Hurwitz criteria the disease-free equilibrium is locally asymptotically stable if $R_q < 1$.

4.2.2. Stability of Endemic Equilibrium

Theorem 2. If $R_q > 1$, then the endemic equilibrium is locally asymptotically stable.

Proof. The variational matrix will be

$$J(E^*) = \begin{bmatrix} -\dfrac{\beta_1 B^*}{1+\alpha_1 B^*} - \dfrac{\beta_2 I^*}{1+\alpha_2 I^*} - \mu_1 & \omega - \dfrac{\beta_2 S^*}{(1+\alpha_2 I^*)^2} & 0 & \dfrac{-\beta_1 S^*}{(1+\alpha_1 B^*)^2} \\ \dfrac{\beta_1 B^*}{1+\alpha_1 B^*} + \dfrac{\beta_2 I^*}{1+\alpha_2 I^*} & \dfrac{\beta_2 S^*}{(1+\alpha_2 I^*)^2} - (d_1 + \mu_1 + \delta + \alpha + \omega) & 0 & \dfrac{\beta_1 S^*}{(1+\alpha_1 B^*)^2} \\ 0 & \delta & -(\varepsilon + d_2 + \mu_1) & 0 \\ 0 & \eta & 0 & -\mu_2 \end{bmatrix}.$$

This matrix can be written as

$$J(E^*) = \begin{bmatrix} -J_1 - \mu_1 & \omega - J_2 & 0 & -J_3 \\ J_1 & J_2 - (d_1 + \mu_1 + \delta + \alpha + \omega) & 0 & J_3 \\ 0 & \delta & -(\varepsilon + d_2 + \mu_1) & 0 \\ 0 & \eta & 0 & -\mu_2 \end{bmatrix}$$

where,

$J_1 = \dfrac{\beta_1 B^*}{1+\alpha_1 B^*} + \dfrac{\beta_2 I^*}{1+\alpha_2 I^*}$, $J_2 = \dfrac{\beta_2 S^*}{(1+\alpha_2 I^*)^2}$ and $J_3 = \dfrac{\beta_1 S^*}{(1+\alpha_1 B^*)^2}$.

$$\text{Or } J(E^*) = \begin{bmatrix} -J_1 - \mu_1 & \omega - J_2 & 0 & -J_3 \\ J_1 & K & 0 & J_3 \\ 0 & \delta & -L & 0 \\ 0 & \eta & 0 & -\mu_2 \end{bmatrix}$$

where, $K = J_2 - (d_1 + \mu_1 + \alpha + \delta + \omega)$ and $L = (\varepsilon + \mu_1 + d_2)$.

Then the characteristic equation is given by

$$\left| J(E^*) - \lambda I \right| = 0.$$

$$\Rightarrow J(E^*) = \begin{vmatrix} -J_1 - \mu_1 - \lambda & \omega - J_2 & 0 & -J_3 \\ J_1 & K - \lambda & 0 & J_3 \\ 0 & \delta & -(L + \lambda) & 0 \\ 0 & \eta & 0 & -\mu_2 - \lambda \end{vmatrix} = 0.$$

Or $(L+\lambda)[-(J_1 + \mu_1 + \lambda)(K - \lambda)(\mu_2 + \lambda) - (J_1 + \mu_1 + \lambda)J_3\eta - J_1\omega(\mu_2 + \lambda) + J_1 J_2(\mu_2 + \lambda) + J_1 J_3\eta] = 0.$

It means that either $L + \lambda = 0$ or

$[-(J_1 + \mu_1 + \lambda)(K - \lambda)(\mu_2 + \lambda) - (J_1 + \mu_1 + \lambda)J_3\eta - J_1\omega(\mu_2 + \lambda) + J_1 J_2(\mu_2 + \lambda) + J_1 J_3\eta] = 0$. This means that one eigen value $\lambda = -(\varepsilon + \mu_1 + d_2)$ is obtained which is negative and remaining are obtained by the following equation

$$\Rightarrow -(J_1 + \mu_1 + \lambda)(K\mu_2 + K\lambda - \lambda\mu_2 - \lambda^2) - (J_1 + \mu_1 + \lambda)J_3\eta - J_1\omega(\mu_2 + \lambda)$$
$$+ J_1 J_2(\mu_2 + \lambda) + J_1 J_3\eta] = 0.$$

On simplification,

$$\lambda^3 + \lambda^2(-K + \mu_2 + J_1 + \mu_1) + \lambda(-K\mu_2 - K(J_1 + \mu_1) + (J_1 + \mu_1)\mu_2 - J_3\eta - J_1\omega + J_1 J_2)$$
$$+ J_1 J_2\mu_2 + J_1 J_3\eta - J_1\omega\mu_2 - K(J_1 + \mu_1)\mu_2 - (J_1 + \mu_1)J_3\eta = 0.$$

Now, substitute $K = J_2 - (d_1 + \mu_1 + \alpha + \delta + \omega)$ and on solving, we obtain

$$\lambda^3 + (2\mu_1 + \mu_2 + (d_1 + \alpha + \delta + \omega) + J_1 - J_2)\lambda^2 + [J_1 J_2 + (J_1 + \mu_1)(\mu_1 + \mu_2 + d_1 + \alpha + \delta + \omega - J_2)$$
$$- (J_2 - (\mu_1 + d_1 + \alpha + \delta + \omega))\mu_2 - J_3\eta - J_1\omega]\lambda + \mu_2(J_1 + \mu_1)(\mu_1 + d_1 + \alpha + \delta + \omega)$$
$$- \mu_1(J_2\mu_2 + J_3\eta) - J_1\omega\mu_2 = 0.$$

Or $\lambda^3 + a_1\lambda^2 + \lambda a_2 + a_3 = 0$

where,

$$a_1 = 2\mu_1 + (d_1 + \delta + \alpha + \omega) + \mu_2 + J_1 - J_2,$$
$$a_2 = J_1 J_2 + (J_1 + \mu_1)(\mu_1 + \mu_2 + d_1 + \alpha + \delta + \omega - J_2)$$
$$- (J_2 - (\mu_1 + d_1 + \alpha + \delta + \omega))\mu_2 - J_3\eta - J_1\omega,$$
$$a_3 = \mu_2(J_1 + \mu_1)(d_1 + \mu_1 + \delta + \alpha + \omega) - .\mu_1(\mu_2 J_2 + J_3\eta) - J_1\omega\mu_2.$$

It is clearly seen that $a_1 > 0$, $a_2 > 0$, $a_3 > 0$ and $a_1 a_2 - a_3 > 0$.

Hence, by Routh-Hurwitz criteria, the endemic equilibrium
Case I

is locally asymptotically stable.

5. Numerical Analysis and Graphical Representation

In this section, I have analyzed the model numerically and graphically by considering the set of parameters values. From practical point of view, numerical solutions are very important beside analytical system.

$$S(0) = 80000, I(0) = 20000, Q(0) = 40000, R(0) = 50000, B(0) = 200000,$$
$$\mu_1 = 9.13 \times 10^{-5} / day, \beta_1 = 0.00025 / day, \alpha_1 = 5\,days, \beta_2 = 0.00015 / day,$$
$$\alpha_2 = 10\,days, d_1 = 0.015 / day, \alpha = 0.2 / day, \delta = 0.1 / day, \varepsilon = 0.05 / day,$$
$$d_2 = 0.013 / day, \eta = 2\,cells / litre / day / person, \mu_2 = 0.01 / day, \omega = 0.2 / day,$$
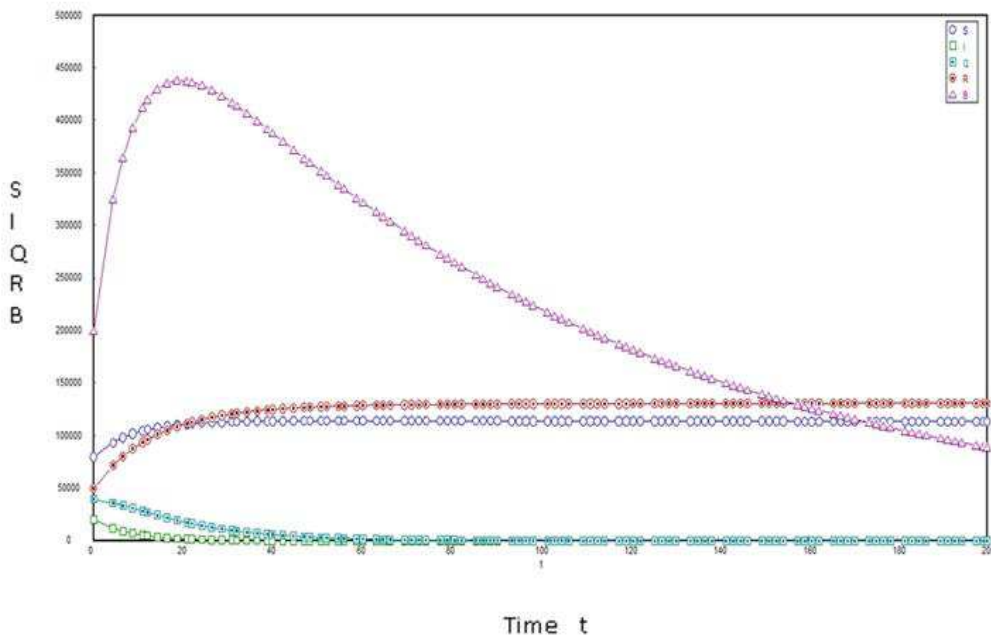$$R_q = 0.097 < 1.$$

Figure 2. *SIS cholera model with Quarantine effect when $R_q < 1$.*

Figure 2 shows that *S(t)* and *R(t)* approaches to its steady state value while *I(t)*, *Q(t)* and *B(t)* approaches zero as time progresses, the disease dies out.
Case II

$$S(0) = 80000, I(0) = 20000, Q(0) = 40000, R(0) = 50000, B(0) = 200000,$$
$$\mu_1 = 9.13 \times 10^{-5} / day, \beta_1 = 0.0025 / day, \alpha_1 = 5\, days, \beta_2 = 0.0015 / day,$$
$$\alpha_2 = 10\, days, d_1 = 0.015 / day, \alpha = 0.2 / day, \delta = 0.1 / day, \varepsilon = 0.05 / day,$$
$$d_2 = 0.013 / day, \eta = 5\, cells / litre / day / person, \mu_2 = 0.03 / day, \omega = 0.05 / day,$$
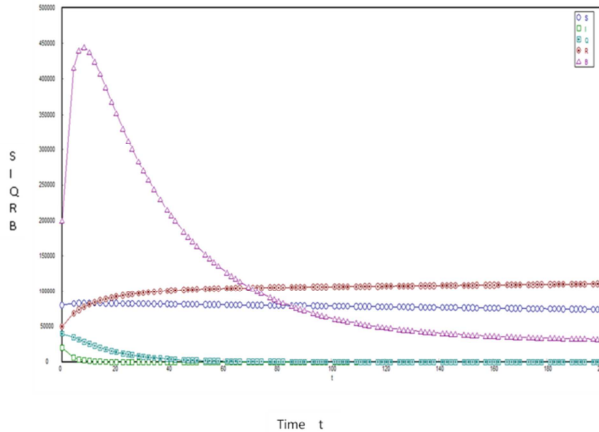$$R_q = 1.14 > 1.$$

Figure 3. *SIS cholera model with Quarantine effect when $R_q > 1$.*

Figure 3 shows that *S(t)* and *R(t)* approaches to its steady state value while *I(t)*, Q(t) and *B(t)* approaches zero as time progresses, the disease becomes endemic.

6. Conclusion

Cholera was prevalent in the U.S. in the 1800s, before modern water and sewage treatment systems eliminated its spread by contaminated water. Cholera outbreaks are still a serious problem in other parts of the world. At least 150,000 cases are reported to the World Health Organization each year. Cholera was first spread in Russia in 1817, after few years in Europe, and from Europe to North America and the rest of the world. In this paper, we have considered an SIQR cholera epidemic model of Nirwani N. et al. [7] and converted into SIS cholera epidemic model with quarantine effect which is a generalized form.

I have found disease-free and endemic equilibria for the model and analyzed the stability criteria for the both equilibria. I have seen that the disease-free equilibria and endemic equilibria arelocally asymptotically stable by Routh - Hurwitz criteria if $R_q < 1$ and $R_q > 1$ respectively. It plays an important role in controlling the disease. Also, numerical simulations are carried out for the model with graphical representation for ordinary differential equation and numerically found that if $R_q < 1$, the disease dies out and if $R_q > 1$, the disease becomes endemic. This can be more clearly seen in the graphs.

References

[1] Agarwal M. and Verma V., *Modeling and Analysis of the Spread of an Infectious Disease Cholera with Environmental Fluctuations*, International Journal of Applications and Applied Mathematics, 7 (1), 406-425, 2012.

[2] Chun I. and Fung H., *Cholera Transmission Dynamic Models for Public Health Practitioners,* Fung Emerging Themes in Epidemiology, 11 (1), 1-11, 2014.

[3] Cui J., Wu Z. and Zhou X., *Mathematical Analysis of a Cholera Model with Vaccination*, Journal of Applied Mathematics, Hindawi Publishing Corporation, Article id. 324767, 1-16, 2014.

[4] Das P. and Mukherjee D., *Qualitative Analysis of a Cholera Bacteriophage Model,* International Scholarly Research Network (ISRN) Biomathematics, Article id 621939, 1-13, 2012.

[5] Emvudu Y. and Kokomo E., *Stability Analysis of Cholera Epidemic Model of a Closed Population*, Journal of Applied Mathematicsand Bioinformatics, 2 (1), 69-97, 2012.

[6] Madubueze C. E., Madubueze S. C., and Ajama S., *Bifurcation and Stability Analysis of the Dynamics of Cholera Model with Controls*, International Journal of Mathematical, Computational, physical, Electrical and Computer Engineering, 9 (11), 633-639, 2015.

[7] Nirwani N., Badshah V. H. and Khandelwal. R, *SIQR Model for Transmission of Cholera*, Advances in Applied Science Research, Pelagia research library, 6 (6), 181-186, 2015.

[8] Pang Y., Han Y. and Li W, *The Threshold of a Stochastic SIQS Epidemic Model*, Advances in Difference Equations, 1, issue 320, 1-15, 2014.

[9] Wang J. and Modnak C., *Modeling Cholera Dynamics with Controls*, Canadian Applied Mathematics Quarterly, 19 (3), 255-273, 2011.