Modeling the Spread of Corona Virus with Lockdown and Quarantine Effect

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Abstract - A nonlinear mathematical model to study the effect of transmission dynamics of COVID-19 virus in a population with variable size structure is proposed and analyzed. The model divides the total human population into five subclasses namely, susceptibles, self-protected susceptibles, infectives, quarantined infectives and recovered population including a class representing cumulative density of corona virus in the environmental reservoir. The model exhibits two equilibria namely, the disease-free and the endemic equilibrium. Analysis of the model reveals that the global dynamics of the spread of the COVID-19 infectious disease is completely determined by the basic reproduction number R_0. If $R_0 \geq 1$ the endemic equilibrium is locally asymptotically stable and is globally asymptotically stable under certain conditions showing that the disease becomes endemic. It is found that the infective population can be decreased if the individuals from susceptible population lockdown themselves and do not come in direct contact with viral density deposited on surfaces/objects or airborne droplets accumulated in the environmental reservoir. However, if higher number of individuals from infective class is quarantined at home or hospital, the spread of the disease can further be slowed down. It is also found that the improving the diagnosis rate of COVID-19 is very beneficial to control the spread of COVID-19. Numerical analysis of the model is also performed to investigate the influence of certain key parameters on the spread of the disease and to support the analytical results.

Keywords: COVID-19, lockdown, asymptomatic infectives, symptomatic infectives, Quarantine, stability analysis

I. INTRODUCTION

In the past two decades, many cases of common cold (flu) are due to different corona viruses. These corona viruses have left their impact at large scale (mainly: SARS in 2002 and MERS- in 2012) in different part of the world. Corona virus disease 2019 (COVID-19) is an infectious disease raise up by severe rapid respiratory syndrome corona virus 2 (SARS-CoV-2). It has taken all over world in its grip just within three months. The situation is becoming worse in country like USA, Brazil, Italy, Spain, France, Germany, India etc. According to WHO, most of people who are infected with COVID-19 virus experience mild to moderate respiratory illness and recover without needing special treatment. Older people or who have underlying medical problems are more severe to develop the illness. Common symptoms of COVID-19 are dry cough, fever, tiredness, sore throat, aches, and shortness of breath. In general, many times it is possible to have infection without any symptoms. Also, in case of current pandemic COVID-19, according to New York times [31], some individuals who are infected with the corona virus can spread it even though they have no symptoms. People who are healthy or have mild symptoms should keep themselves in self-quarantine and contact COVID-19 information line for guidance on testing and referral. However, till now there is no any specific treatment available for COVID-19. Though some vaccine is available but no single vaccine is effective at all the strains as COVID-19 changing its strain in every country. So, it is predicted that the number of COVID-19 infections may still increase. Therefore, at present, the key question is how to prevent/control the spread of COVID-19. In case of COVID-19, social distancing has emerged as one of the most broadly adopted intervention strategies (e.g., self isolation, social distancing, quarantine infected individuals, promoting social consensus on self-

protection like wearing a face mask in the public area, washing hands regularly etc.) to reduce the infection risk/transmission rate and control the spread of COVID-19 through the reduction of social contacts. More importantly, at this stage of the outbreak, it is important to understand transmission dynamics of the COVID-19 and deployment of different control strategies such as self isolation of susceptible, quarantine of infected individuals.

Lockdown, self isolation and quarantine are the important measures by which further spread of the disease could be stop. Different governments are actively restricting the movement of people by imposing lockdown, which may be known as one of the largest quarantine in history. Except lockdown, different governments are also adopting various steps and imposing different types of intervention strategies, for instance, social distancing, washing hands for at least 20 seconds, wearing masks on public places, tracing close contacts.

Mathematical models have a long history of application to help humans to understand how the dynamics of a disease spread in a population, for example in dengue [1,2,18], tuberculosis[23, 26], HIV [3, 23, 24] and many more. These models[1-3,7,18,22-24,26] try to accommodate various essential factors in the spread of a disease, such as the presence of a disease vector, the phenomenon of relapse and reinfection, symptomatic and asymptomatic cases, analysis of the success of interventions with limited costs, and others. The transmission potential of a disease is often measured in terms of the basic reproduction numbers. Since COVID-19is recent pandemic and has rapidly spread in many countries across the world, few mathematical studies have been conducted[4-6,8,9,12,14,15,17,25,26,28-30] to capture the transmission mechanism and the effect of preventive measure. In particular, Yang et al. [4] proposed a mathematical model for COVID-19 incorporating multiple transmission pathways, including both human-to-human and environment-to-human transmission routes. The authors employed a bilinear incidence rate based on the law of mass action and fitted the model with the data of Wuhan city of China and estimated the reproduction number. Ngonghala et al. [5] developed a mathematical model of COVID-19 pandemic in US (particularly, in New York) for assessing the population-level impact of the mitigation strategies. The authors performed the rigorous analysis of the model and the impacts of non-pharmaceutical intervention strategies, social distancing, quarantine, contact-tracing, isolation, face mask, etc. Legesse et. Al [13]. found the optimal control strategies for the transmission risk of COVID-19 and shows that that comprehensive impacts of prevention, intensive medical care and surface disinfection strategies outperform in reducing the disease epidemic with optimum implementation cost. Garba et al. [28] proposed a compartmental model to analyze the dynamics of COVID-19 in South Africa. The model system in [28] was used to estimate the effect of mitigation strategies and various control. The results of this particular study was twofold: (i) the disease may die out if control measures are implemented early and for a sustainable period of time (ii) effectiveness of self-isolation reduces the number of cases.

Several compartmental models of COVID-19 outbreak in India, have also been studied [12, 25, 29, 30]. Khajanchi et al. [25] proposed a compartmental model with quarantine for the transmission dynamics of COVID-19and calibrated the mode lwith daily and cumulative cases for the four provinces of India. The authors have performed a detailed theoretical analysis in terms of the basic reproduction number and predicted the cumulative cases. Moreover, the study suggests that quarantine, unreported and reported individuals as well as intervention policies like social distancing, lockdown, and media effect can play an important role in controlling the transmission of COVID-19. Sarkar et al. [12] proposed a mathematical model that predicts the dynamics of COVID-19 in India along with its 17 provinces. Their findings revealed the fact that the contact rate between susceptible and infected individuals could be reduced by a strict isolation imposed for susceptible individuals. Moreover the numerical evaluations of the model system [12] suggested the complete elimination of COVID-19 via suitable combination of contact tracing and restrictive social distancing. Further the authors also indicated that the accurate course of epidemic largely depends on how and when precautionary measures, isolation, and quarantine are enforced. In this direction, Sardar et al. [30] also considered a mathematical model on to analyze the impact of social distancing and lockdown. The authors have done a detailed analysis and validated the model with the data of India and its five different states. In particular, Sarita et al. formulated a COVID-19 model to analyze the role of intervention strategies and lockdown and found that after removal of lockdown fully or partially the endemic level would be high.

The aim of this paper is to provide a qualitative study of the dynamics of COVID-19 vis-a-vis its impact on human population. A basic compartmental model, which subdivides a given population into a number of mutually exclusive sub-populations is designed and qualitatively analyzed. Our main contribution related with considering the class of Lock-down in model. This new class, as compiled to any compartmental model, implies a number of

analysis about absence of disease and endemic equilibrium point, which is also consider in this work. This paper is organized as follows. In section 2, we present our model and assumptions, and conduct a detailed mathematical analysis and the non-negativity and boundedness. Section 3 represents calculation of basic reproduction number ; Section 4 discusses the dynamics of the model system including local and global stability of disease-free and endemic equilibrium; Section 5 presents sensitivity analysis; Section 6 demonstrates numerical simulation and discussion; and Section 7 concludes the paper.

II. MATHEMATICAL MODEL

Consider a population of size $N(t)$ at time t with constant immigration of susceptibles at a rate Λ. The population size $N(t)$ is divided into five subclasses of Susceptibles $X(t)$, population in Lockdown situation L(t), Symptomatic infectives $I_s(t)$, Asymptomatic infectives $I_a(t)$ and $Q(t)$ Quarantine class . Population $L(t)$ are the people who living in the area where the lockdown is applied. Symptomatic infectives $I_s(t)$ are the infectives who are infectious with strong infectivity and shows the symptom of corona virus infection. Asymptomatic infectives $I_a(t)$ are infected by corona virus but are asymptomatic it means the system of corona virus are not appear in them. Quarantine class Q(t) consists of individuals who are infected and diagnosed but have been quarantined. They are isolated and do not contribute to infection spread with natural mortality rate d in all the classes human population and a separate class $V(t)$ of cumulative density of corona virus in environmental reservoir. Susceptibles become infected via proper contact with symptomatic infective, asymptomatic infective and with the virus in the environmental reservoir by the contact rate β_1, β_2 and β_3 respectively. l is the rate of transfer of susceptible to the lockdown class. η is the fraction of new infectives who will join the symptomatic infectives and the remaining portion 1-η of new infectives will join the asymptomatic infective class. ϕ is the rate of movement of symptomatic infectives who will join the quarantine class after being diagnosed. ψ is the rate of movement of asymptomatic infectives who will join the quarantine class after being randomly diagnosed. Some asymptomatic infectives shows the symptoms after certain time hence μ is the rate of transfer of asymptomatic infectives to the symptomatic infectives.

It is further assumed that ξ is the rate of movement of quarantined people to the susceptible class after being recoverd from COVID-19. Some of the asymptomatic infectives will recover without quarantine and again increase the susceptible population by the rate ρ. The constant α denotes the disease- induced death rate of infectives with are without being in quarantined class. The growth of viral density V (t) in the environmental reservoir is assumed to be directly proportional to the asymptomatic and symptomatic infectives where γ is the rate of increase of V. The constant γ_0 is the rate by which viral density declines due to control/ preventive measures like mass sanitization in the environment.

With the above assumptions and considerations, the dynamics of the disease is assumed to be governed by the following system of nonlinear ordinary differential equations,

$$\frac{dS(t)}{dt} = \Lambda - \beta_1 S(t) I_s(t) - \beta_2 S(t) I_a(t) - \beta_3 S(t) V(t) - (l + d) S(t) + \xi Q(t) + \delta L(t) + \rho I_a(t) \tag{2.1}$$

$$\frac{dL(t)}{dt} = lS(t) - (\delta + d) L(t) \tag{2.2}$$

$$\frac{dI_s(t)}{dt} = \eta\{\beta_1 I_s(t) + \beta_2 I_a(t) + \beta_3 V(t)\} S(t) - (\phi + \alpha + d) I_s(t) + \mu I_a(t) \tag{2.3}$$

$$\frac{dI_a(t)}{dt} = (1 - \eta)\{\beta_1 I_s(t) + \beta_2 I_a(t) + \beta_3 V(t)\} S(t) - (\psi + \mu + \rho + \alpha + d) I_a(t) \tag{2.4}$$

$$\frac{dQ(t)}{dt} = \phi I_s(t) + \psi I_a(t) - (\xi + d + \alpha) Q(t) \tag{2.5}$$

$$\frac{dV(t)}{dt} = \gamma I_s(t) + \gamma I_a(t) - \gamma_0 V(t) \tag{2.6}$$

$$S(0) = S_0 > 0,\ L(0) = L_0 > 0,\ I_s(0) = I_{s0} \geq 0,\ I_a(0) = I_{a0} \geq 0,\ Q(0) = Q_0 \geq 0,\ V(0) = V_0 \geq 0$$

2. 1 Non-negativity and boundedness of solutions

It is important to show that all the population variables are nonnegative for all $t \geq 0$, which implies that any trajectory which starts with positive initial condition will remain positive for $t \geq 0$. It is an important feature of an epidemiological model. From equation (2.1), we have

$$\frac{dS(t)}{dt} = -dS(t)$$

Integrating the above inequality and using initial condition, we obtain

$$S(t) = S(0)e^{-dt} > 0$$

Thus $S(t) > 0$. Similarly, one can show that all the variables are non-negative for all $t > 0$.

III. COMPUTATION OF BASIC REPRODUCTION NUMBER

The transmission potential of a disease is often measured in terms of the basic reproduction numbers. The basic reproduction number (R_0) is described as the expected number of secondary infections appearing from a single infectious individual throughout his/her whole infectious period, in the entire susceptible population [10, 11]. In the study of epidemiology, the fundamental concept of reproduction number (R_0) is one of the most valuable ideas that the mathematical thinking has conveyed to epidemic theory [11]. In an epidemic disease, it could be determined that which control measures (intervention strategies) would be most helpful for suppressing R_0 below one and which may also provide important advice for public health initiatives. More importantly, the R_0 is also called a controlled reproduction number when it depends on the control strategies, it is computed for mathematical models including control strategies [16]. We calculate R_0 by closely following the approach in Van den Driessche and Watmough [20, 21]. We first compute the new infectious matrix F and transfer matrix W [19], according to formula

$$[F - W] = \begin{bmatrix} \frac{\partial(dI_s/dt)}{\partial I_s} & \frac{\partial(dI_s/dt)}{\partial I_a} & \frac{\partial(dI_s/dt)}{\partial V} \\ \frac{\partial(dI_a/dt)}{\partial I_s} & \frac{\partial(dI_a/dt)}{\partial I_a} & \frac{\partial(dI_a/dt)}{\partial V} \\ \frac{\partial(dV/dt)}{\partial I_s} & \frac{\partial(dV/dt)}{\partial I_a} & \frac{\partial(dV/dt)}{\partial V} \end{bmatrix} \tag{3.1}$$

To calculate F and W, we only consider equations (2.3), (2.4) and (2.6), which correspond to the groups (I_s, I_a, V) capable of transmitting the disease. The non-negative matrix F, corresponding to new infections in the population at disease-free equilibrium is,

$$F = \begin{bmatrix} \eta\beta_1 S_0 & \eta\beta_2 S_0 & \eta\beta_3 S_0 \\ (1-\eta)\beta_1 S_0 & (1-\eta)\beta_2 S_0 & (1-\eta)\beta_3 S_0 \\ 0 & 0 & 0 \end{bmatrix} \tag{3.2}$$

The non-singular matrix W, corresponding to the transfer of individuals into and out of compartment is,

$$W = \begin{bmatrix} a & -\mu & 0 \\ 0 & b & 0 \\ -\gamma & -\gamma & \gamma_0 \end{bmatrix} \tag{3.3}$$

W^{-1} is given by $$W^{-1} = \begin{bmatrix} \frac{1}{a} & \frac{\mu}{ab} & 0 \\ 0 & \frac{1}{b} & 0 \\ \frac{\gamma}{a\gamma_0} & \frac{\gamma(a+\mu)}{ab\gamma_0} & \frac{1}{\gamma_0} \end{bmatrix} \tag{3.4}$$

Where,

$a = (\phi + \alpha + d)$, $b = (\psi + \mu + \rho + \alpha + d)$

FW^{-1} is the next generation matrix of the system (2.1)-(2.6). It follows that the spectral radius of matrix FW^{-1} is

$$FW^{-1} = \begin{bmatrix} \eta\beta_1 S_0 & \eta\beta_2 S_0 & \eta\beta_3 S_0 \\ (1-\eta)\beta_1 S_0 & (1-\eta)\beta_2 S_0 & (1-\eta)\beta_3 S_0 \\ 0 & 0 & 0 \end{bmatrix} \begin{bmatrix} \frac{1}{a} & \frac{\mu}{ab} & 0 \\ 0 & \frac{1}{b} & 0 \\ \frac{\gamma}{a\gamma_0} & \frac{\gamma(a+\mu)}{ab\gamma_0} & \frac{1}{\gamma_0} \end{bmatrix}$$

$$FW^{-1} = \begin{bmatrix} \left[\beta_1 + \frac{\beta_3\gamma}{\gamma_0}\right]\frac{\eta S_0}{a} & \left[\frac{\beta_1\mu}{a} + \beta_2 + \frac{\beta_3\gamma(a+\mu)}{a\gamma_0}\right]\frac{\eta S_0}{b} & \frac{\eta\beta_3 S_0}{\gamma_0} \\ \left[\beta_1 + \frac{\beta_3\gamma}{\gamma_0}\right]\frac{(1-\eta)S_0}{a} & \left[\frac{\beta_1\mu}{a} + \beta_2 + \frac{\beta_3\gamma(a+\mu)}{a\gamma_0}\right]\frac{(1-\eta)S_0}{b} & \frac{(1-\eta)\beta_3 S_0}{\gamma_0} \\ 0 & 0 & 0 \end{bmatrix}$$

$$\lambda^2 - \{\eta A + (1-\eta)B\lambda = 0$$

$$\lambda^2 - \left[\left\{\beta_1 + \frac{\beta_3\gamma}{\gamma_0}\right\}\frac{\eta S_0}{a} + \left\{\frac{\beta_1\mu}{a} + \beta_2 + \frac{\beta_3\gamma(a+\mu)}{a\gamma_0}\right\}\frac{(1-\eta)S_0}{b}\right]\lambda = 0$$

$$R_0 = \left[\beta_1 + \frac{\beta_3\gamma}{\gamma_0}\right]\frac{\eta S_0}{a} + \left[\frac{\beta_1\mu}{a} + \beta_2 + \frac{\beta_3\gamma(a+\mu)}{a\gamma_0}\right]\frac{(1-\eta)S_0}{ab}$$

According to van den Driessche and watmough [13,14], the basic reproduction number(on putting value of a, b) of the system (2.1)-(2.6) is

$$R_0 = \left[\beta_1 + \frac{\beta_3\gamma}{\gamma_0}\right]\frac{\eta}{(\phi+\alpha+d)}\frac{\Lambda(\mu+d)}{d(\mu+l+d)} + \left[\frac{\beta_1\mu}{(\phi+\alpha+d)} + \beta_2 + \frac{\beta_3\gamma(\phi+\alpha+d+\mu)}{\gamma_0(\phi+\alpha+d)}\right]\frac{(1-\eta)}{(\psi+\mu+\rho+\alpha+d)}\frac{\Lambda(\mu+d)}{d(\mu+l+d)} \quad (3.5)$$

From the above expression of R_0 we can see that as the lock down rate of susceptible increases reproduction rate decreases if l will be sufficiently large, reproduction rate will less then one. Also with the increase of quarantine rate ϕ and ψ of symptomatic and asymptomatic infectives respectively, reproduction rate decreases as ϕ and ψ are only in the denominator.

If R_0<1, then on average an infected individual produces less than one infected individual over the course of its infectious period and infection cannot grow. Conversely, if R_0 >1 then on average an infected individual produces more than one new infection and the disease can invade the population.

IV. EQUILIBRIA AND STABILITY ANALYSIS OF THE MODEL

4.1 Equilibria of the model

The model (2.1)-(2.6) has two non-negative equilibria namely,

(i) $E_0\left(\frac{\Lambda(\mu+d)}{d(\mu+l+d)}, \frac{\Lambda l}{d(\mu+l+d)}, 0, 0, 0\right)$ the disease-free equilibrium, which exists without any condition.

(ii) $E^*(S^*, L^*, I_s^*, I_a^*, Q^*, V^*)$, the endemic equilibrium. The equilibrium values of different variables are given as,

$$I_s^* = kI_a^*, \quad Q^* = mI_a^*, \quad V^* = nI_a^*, \quad S^* = \frac{(\psi+\mu+\rho+\alpha+d)}{(1-\eta)(\beta_1 k+\beta_2+\beta_3 n)}$$

$$L^* = \frac{l(\psi+\mu+\rho+\alpha+d)}{(1-\eta)(\beta_1 k+\beta_2+\beta_3 n)(\delta+d)}, \quad I_a^* = \frac{\Lambda+\delta L^*-(l+d)S^*}{\{(\beta_1 k+\beta_2+\beta_3 n)S^*-\xi m-\rho\}}$$

endemic equilibrium will exist only if $\Lambda+\delta L^* > (l+d)S^*$ and $(\beta_1 k+\beta_2+\beta_3 n)S^* > \xi m+\rho$

Where $n = \frac{\gamma(1+k)}{\gamma_0}$, $m = \frac{\psi+\phi k}{\xi+\alpha+d}$, $k = \frac{(1-\eta)(\phi+\alpha+d)}{\eta(\psi+\mu+\rho+\alpha+d)+(1-\eta)\mu}$

4.2 Local stability of the equilibria

To determine the local stability of E_0, the following variational matrix of the system (2.1) – (2.6) is computed about E_0 as,

$$J(E_0)=\begin{bmatrix} -(l+d) & \delta & -\beta_1 S_0 & -\beta_2 S_0+\rho & \xi & -\beta_3 S_0 \\ l & -(\delta+d) & 0 & 0 & 0 & 0 \\ 0 & 0 & -n_1 & \eta\beta_2 S_0+\mu & 0 & \eta\beta_3 S_0 \\ 0 & 0 & (1-\eta)\beta_1 S_0 & -n_2 & 0 & (1-\eta)\beta_3 S_0 \\ 0 & 0 & \phi & \psi & -(\xi+\alpha+d) & 0 \\ 0 & 0 & \gamma & \gamma & 0 & -\gamma_0 \end{bmatrix}$$

Where $n_1=-\eta\beta_1 S_0+(\phi+\alpha+d)$, $n_2=-(1-\eta)\beta_2 S_0+(\psi+\mu+\rho+\alpha+d)$

three roots of the characteristic equation are - $(l+d)$, - $(\delta+d)$ $-(\xi+\alpha+d)$. The other three roots of the determined by the equation

$$f(\lambda)=\lambda^3+a_1\lambda^2+a_2\lambda+a_3=0 \qquad (4.1)$$

where,

$$a_1=n_1+n_2+\gamma_0 \quad , \qquad a_2=n_1n_2+n_1\gamma_0+n_2\gamma_0-(\eta\beta_2 S_0+\mu)(1-\eta)\beta_1 S_0-(\eta\beta_2 S_0+\mu)(1-\eta)\beta_3 S_0$$

$$a_3=n_1n_2\gamma_0-\gamma_0(\eta\beta_1 S_0+\mu)(1-\eta)\beta_1 S_0-\gamma(\eta\beta_1 S_0+\mu)(1-\eta)\beta_3 S_0-\eta\beta_3 S_0(1-\eta)\beta_1 S_0\gamma$$
$$-\eta\beta_3 S_0 n_2\gamma-n_1\gamma(1-\eta)\beta_3 S_0$$

$$a_3=\{-\eta\beta_1 S^*+(\phi+\alpha+d\}\{-(1-\eta)\beta_2 S^*+(\psi+\mu+\rho+\alpha+d)\}\gamma_0-\gamma_0(\eta\beta_2 S^*+\mu)(1-\eta)\beta_1 S^*$$
$$-\gamma(\eta\beta_2 S^*+\mu)(1-\eta)\beta_3 S^*-\eta\beta_3 S^*(1-\eta)\beta_1 S^*\gamma-\eta\beta_3 S^*\{-(1-\eta)\beta_2 S^*+(\psi+\mu+\rho+\alpha+d)\}\gamma$$
$$-\{-\eta\beta_1 S^*+(\phi+\alpha+d\}\gamma(1-\eta)\beta_3 S^*>0$$

$$=-(1-\eta)\beta_2 S^*(\phi+\alpha+d)\gamma_0-\eta\beta_1 S^*(\psi+\mu+\rho+\alpha+d)\gamma_0+\gamma_0(\phi+\alpha+d)(\psi+\mu+\rho+\alpha+d)$$
$$-\gamma_0\mu(1-\eta)\beta_1 S^*-\gamma\mu(1-\eta)\beta_3 S^*-\eta\beta_3 S^*(\psi+\mu+\rho+\alpha+d)\gamma-(\phi+\alpha+d)\gamma(1-\eta)\beta_3 S^*>0$$

$$\gamma_0(\phi+\alpha+d)(\psi+\mu+\rho+\alpha+d)>(1-\eta)\beta_2 S^*(\phi+\alpha+d)\gamma_0+\eta\beta_1 S^*(\psi+\mu+\rho+\alpha+d)\gamma_0$$
$$+\gamma_0\mu(1-\eta)\beta_1 S^*-\gamma\mu(1-\eta)\beta_3 S^*+\eta\beta_3 S^*(\psi+\mu+\rho+\alpha+d)\gamma+(\phi+\alpha+d)\gamma(1-\eta)\beta_3 S^*$$

$$1>\frac{(1-\eta)\beta_2 S_0(\phi+\alpha+d)\gamma_0}{\gamma_0(\phi+\alpha+d)(\psi+\mu+\rho+\alpha+d)}+\frac{\eta\beta_1 S_0(\psi+\mu+\alpha+d)\gamma_0}{\gamma_0(\phi+\alpha+d)(\psi+\mu+\rho+\alpha+d)}$$
$$+\frac{\gamma_0\mu(1-\eta)\beta_1 S_0-\gamma\mu(1-\eta)\beta_3 S^*}{\gamma_0(\phi+\alpha+d)(\psi+\mu+\rho+\alpha+d)}+\frac{\eta\beta_3 S_0(\psi+\mu+\alpha+d)\gamma}{\gamma_0(\phi+\alpha+d)(\psi+\mu+\rho+\alpha+d)}$$
$$+\frac{(\phi+\alpha+d)\gamma(1-\eta)\beta_3 S_0}{\gamma_0(\phi+\alpha+d)(\psi+\mu+\rho+\alpha+d)}$$

$$1>\frac{(1-\eta)\beta_2 S_0}{(\psi+\mu+\rho+\alpha+d)}+\frac{\eta\beta_1 S_0}{(\phi+\alpha+d)}$$
$$+\frac{\mu(1-\eta)\beta_1 S_0}{(\phi+\alpha+d)(\psi+\mu+\rho+\alpha+d)}+\frac{\eta\beta_3 S_0\gamma}{\gamma_0(\phi+\alpha+d)}+\frac{(\mu+\phi+\alpha+d)\gamma(1-\eta)\beta_3 S_0}{\gamma_0(\phi+\alpha+d)(\psi+\mu+\rho+\alpha+d)}$$

$$1 > \left[\beta_1 + \frac{\beta_3\gamma}{\gamma_0}\right]\frac{\eta}{(\phi+\alpha+d)}\frac{\Lambda(\mu+d)}{d(\mu+l+d)} + \left[\frac{\beta_1\mu}{(\phi+\alpha+d)} + \beta_2 + \frac{\beta_3\gamma(\phi+\alpha+d+\mu)}{(\phi+\alpha+d)\gamma_0}\right]\frac{(1-\eta)}{(\psi+\mu+\rho+\alpha+d)}\frac{\Lambda(\mu+d)}{d(\mu+l+d)} = R_0$$

$1 > R_0$

Thus, from equation (3.5) it is noted that $a_3 > 0$.

We can see a_1, a_2, a_3 are all positive.Using the Routh- Hurwitz stability criteria, it can be shown that the eigen values of matrix $J(E_0)$ have negative real parts. If $R_0 > 1$, then $a_3 > 0$ thus $J(E_0)$ has at least one eigen value with positive real part. Hence, disease free equilibrium E_0 of the (2.1)-(2.6) is locally asymptotically stable if $R_0 < 1$. Therefore, the disease dies out i.e. infection does not persist in the population and under this condition the equilibrium E^* does not exist. It is unstable for $R_0 > 1$ and then E^* exists and the disease always persists in the population. Now the variational matrix corresponding to E^* is given by,

$$J(E^*) = \begin{bmatrix} -(D_1+l+d) & \delta & -\beta_1 S^* & -(\beta_2 S^* - \rho) & \xi & -\beta_3 S \\ l & -(\delta+d) & 0 & 0 & 0 & 0 \\ \eta D_1 & 0 & -D_2 & D_3 & 0 & D_4 \\ (1-\eta)D_1 & 0 & D_5 & -D_6 & 0 & D_7 \\ 0 & 0 & \phi & \psi & -D_8 & 0 \\ 0 & 0 & \gamma & \gamma & 0 & -\gamma_0 \end{bmatrix}$$

Where,

$$D_1 = \beta_1 I_s^* + \beta_2 I_a^* + \beta_3 V^*, \quad D_2 = -\eta\beta_1 S^* + (\phi+\alpha+d), \quad D_3 = \eta\beta_2 S^* + \mu$$

$$D_4 = \eta\beta_3 S^* \quad D_5 = (1-\eta)\beta_1 S^*, \quad D_6 = -(1-\eta)\beta_2 S^* + (\psi+\mu+\rho+\alpha+d) \quad D_7 = (1-\eta)\beta_3 S^*, \quad D_8 = (\xi+\alpha+d)$$

The characteristic equation corresponding to $M(E^*)$ is given by

$$f(\lambda) = \left(\lambda^6 + b_1\lambda^5 + b_2\lambda^4 + b_3\lambda^3 + b_4\lambda^2 + b_5\lambda + b_6\right) = 0 \qquad (4.2)$$

$$b_1 = (d+l+D_1) + (\delta+d) + D_2 + D_6 + D_8 + \gamma_0$$

$$b_2 = ((D_1+l+d)(d+D_2+D_6+D_8+\gamma_0)) + (\delta+d)(D_2+D_6+D_8+\gamma_0) + D_2(D_6+D_8+\gamma_0) + D_6(D_8+\gamma_0) + D_8\gamma_0 + (D_1+d)\delta - \beta_3 S^*\gamma - D_3D_5 + \beta_1 S^*\eta D_1$$

$$b_3 = ((D_1+l+d)(\delta+d)(D_2+D_6+D_8+\gamma_0)) + (D_1+l+d)D_2(D_6+D_8+\gamma_0) + (D_1+l+d)D_6(D_8+\gamma_0) + (D_1+l+d)D_8\gamma_0 + (\delta+d)D_2(D_6+D_8+\gamma_0) + (\delta+d)D_6(D_8+\gamma_0) + (\delta+d)D_8\gamma_0 + D_2D_6(D_8+\gamma_0) + D_6D_8\gamma_0 + D_2D_8\gamma_0 - D_4D_5\gamma - D_2D_8\gamma - D_3D_8\gamma - D_3D_5\gamma_0 - D_4D_6\gamma - \xi\psi(1-\eta)D_1 + \beta_3 S^*\gamma D_1 - (\beta_3 S^*\gamma + D_3D_5)(D_1+l+d+\delta+d+D_7) - \delta l(D_2+D_6+D_7+\gamma_0) + \beta_1 S^*(1-\eta)D_1D_3 - \xi\phi\eta D_1 + \beta_1 S^*\eta D_1(\delta+d+D_6+D_7+\gamma_0) + \beta_1 S^*(1-\eta)D_1$$

$$
\begin{aligned}
b_4 = {} & \left((D_1 + l + d)(\delta + d)D_2(D_6 + D_8 + \gamma_0)\right) + (D_1 + l + d)(\delta + d)D_6(D_8 + \gamma_0) + (D_1 + l + d)(\delta + d)D_8\gamma_0 \\
& + (D_1 + l + d)D_2D_6(D_8 + \gamma_0) + (D_1 + l + d)D_6D_8\gamma_0 + (D_1 + l + d)D_2D_8\gamma_0 + (\delta + d)D_2D_6(D_8 + \gamma_0) \\
& + (\delta + d)D_6D_8\gamma_0 + (\delta + d)D_2D_8\gamma_0 + D_2D_6D_8\gamma_0 - (\beta_3S^*\gamma + D_3D_5)((D_1 + l + d)(\delta + d + D_7) + (\delta + d)D_7) \\
& - (D_4D_5\gamma + D_2D_8\gamma + D_3D_8\gamma + D_3D_5\gamma_0 + D_4D_6\gamma)(D_1 + l + d + \delta + d + D_7) + \delta l\gamma D_8 + \delta lD_3D_5 + \delta l\gamma D_4 \\
& - \delta l\{D_2(D_6 + D_7 + \gamma_0) + D_6(D_7 + \gamma_0) + D_7\gamma_0\} + \beta_1S^*\eta D_1\{(\delta + d)(D_6 + D_7 + \gamma_0) + D_6(D_7 + \gamma_0) + D_7\gamma_0\} \\
& - \beta_1S^*\eta D_1D_8\gamma + \beta_1S^*(1 - \eta)D_1D_3(\delta + d + D_7 + \gamma_0) + \beta_1S^*(1 - \eta)D_1D_4\gamma + (\beta_2S^* - \rho)\eta D_1D_5(\delta + d + D_7 + \gamma_0) \\
& + (\beta_2S^* - \rho)\eta D_1D_8\gamma + (\beta_2S^* - \rho)(1 - \eta)D_1D_2(\delta + d + D_7 + \gamma_0) - (\beta_2S^* - \rho)(1 - \eta)D_1D_4\gamma - \xi\eta D_1D_5\psi \\
& - \xi\phi\eta D_1(\delta + d + D_6 + \gamma_0) - \xi\psi(1 - \eta)D_1(\delta + d + D_2 + \gamma_0) - \xi\phi(1 - \eta)D_1D_3 + \beta_3S^*\gamma\eta D_1(\delta + d + D_6 + D_5 + D_7) \\
& + \beta_3S^*\gamma(1 - \eta)D_1(\delta + d + D_2 + D_7) + \beta_3S^*\gamma(1 - \eta)D_1D_3
\end{aligned}
$$

$$
\begin{aligned}
b_5 = {} & \left((D_1 + l + d)(\delta + d)D_2D_6(D_8 + \gamma_0)\right) + (D_1 + l + d)(\delta + d)D_2D_8\gamma_0 + (D_1 + l + d)(\delta + d)D_6D_8\gamma_0 \\
& + (\delta + d)D_2D_6D_8\gamma_0 - (\beta_3S^*\gamma + D_3D_5)(D_1 + l + d)(\delta + d)D_7 - \delta l\{D_2D_6(D_7 + \gamma_0) + D_2D_7\gamma_0 + D_6D_7\gamma_0\} \\
& - (D_4D_5\gamma + D_2D_8\gamma + D_3D_8\gamma + D_3D_5\gamma_0 + D_4D_6\gamma)((D_1 + l + d)(\delta + d + D_7) + (\delta + d)D_7) + \delta l\gamma D_8(D_2 + D_7) \\
& + \delta lD_3D_5(\gamma_0 + D_7) + \delta lD_3D_8\gamma + \delta lD_4D_5\gamma + \delta lD_4\gamma(D_6 + D_7) + \beta_1S^*(1 - \eta)D_1D_4\gamma(\delta + d + D_7) \\
& + \beta_1S^*\eta D_1\{(\delta + d)D_6(D_7 + \gamma_0) + D_6D_7\gamma_0 + (\delta + d)D_7\gamma_0\} - \beta_1S^*\eta D_1D_8\gamma(\delta + d + D_7) \\
& + \beta_1S^*(1 - \eta)D_1D_3((\delta + d)(D_7 + \gamma_0) + D_7\gamma_0) + (\beta_2S^* - \rho)\eta D_1D_5\{D_7(\delta + d + \gamma_0) + (\delta + d)\gamma_0\} \\
& + (\beta_2S^* - \rho)\eta D_1D_8\gamma(\delta + d + D_7) + (\beta_2S^* - \rho)(1 - \eta)D_1D_2\{D_7(\delta + d + \gamma_0) + (\delta + d)\gamma_0\} \\
& - (\beta_2S^* - \rho)(1 - \eta)D_1D_4\gamma(\delta + d + D_7) - \xi\eta D_1D_5\psi(\delta + d + \gamma_0) - \xi\phi\eta D_1\{(\delta + d)(D_6 + \gamma_0) + D_6\gamma_0\} \\
& + \xi\eta D_1D_8\gamma(\phi - \psi) - \xi\psi(1 - \eta)D_1\{(\delta + d)(D_2 + \gamma_0) + D_2\gamma_0\} - \xi\phi(1 - \eta)D_1D_3(\delta + d + \gamma_0) \\
& - \xi\gamma(1 - \eta)D_1D_4(\phi - \psi) + \beta_3S^*\gamma\eta D_1\{(\delta + d)(D_6 + D_5 + D_7) + D_7(D_6 + D_5)\} \\
& + \beta_3S^*\gamma(1 - \eta)D_1\{(\delta + d)(D_2 + D_7) + D_2D_7\} + \beta_3S^*\gamma(1 - \eta)D_1D_3(\delta + d + D_7)
\end{aligned}
$$

$$
\begin{aligned}
b_6 = {} & (D_1 + l + d)(\delta + d)D_2D_6D_8\gamma_0 - (D_1 + l + d)(\delta + d)D_7(D_4D_5\gamma + D_2D_8\gamma + D_3D_8\gamma + D_3D_5\gamma_0 \\
& + D_4D_6\gamma - \delta lD_2D_6D_7\gamma_0 + \delta lD_2D_6D_7\gamma + \delta lD_3D_5D_7\gamma_0 + \delta lD_3D_8D_7\gamma + \delta lD_4D_5D_7\gamma + \delta lD_4D_6D_7\gamma \\
& + \beta_1S^*\eta D_1(\delta + d)D_6D_7\gamma_0 - \beta_1S^*\eta D_1(\delta + d)D_8D_7\gamma + \beta_1S^*(1 - \eta)D_1(\delta + d)D_3D_7\gamma_0 \\
& + \beta_1S^*(1 - \eta)D_1(\delta + d)D_4D_7\gamma + (\beta_2S^* - \rho)\eta D_1(\delta + d)D_5D_7\gamma_0 + (\beta_2S^* - \rho)\eta D_1(\delta + d)D_8D_7\gamma \\
& + (\beta_2S^* - \rho)(1 - \eta)D_1(\delta + d)D_2D_7\gamma_0 - (\beta_2S^* - \rho)(1 - \eta)D_1(\delta + d)D_4D_7\gamma - \xi\psi\eta D_1D_5(\delta + d)\gamma_0 \\
& - \xi\phi\eta D_1D_6(\delta + d)\gamma_0 + \xi(\phi - \psi)\eta D_1D_8(\delta + d)\gamma_0 - \xi\psi(1 - \eta)D_1D_2(\delta + d)\gamma_0 \\
& - \xi\phi(1 - \eta)D_1D_3(\delta + d)\gamma_0 - \xi(\phi - \psi)(1 - \eta)D_1D_4(\delta + d)\gamma + \beta_3S^*\eta D_1(\delta + d)(D_6 + D_7)\gamma D_7 \\
& + \beta_3S^*(1 - \eta)D_1(\delta + d)D_2\gamma D_7 + \beta_3S^*(1 - \eta)D_1(\delta + d)D_3\gamma D_7
\end{aligned}
$$

Therefore, $b_i > 0$ for i=1,2,3,4,5,6. Thus by Routh-Hurwith criteria, E^* is locally asymptotically stable as if the remaining conditions

$$
\begin{vmatrix} b_1 & 1 \\ b_3 & b_2 \end{vmatrix} > 0, \quad \begin{vmatrix} b_1 & 1 & 0 \\ b_3 & b_2 & b_1 \\ 0 & b_4 & b_3 \end{vmatrix} > 0, \quad \begin{vmatrix} b_1 & 1 & 0 & 0 \\ b_3 & b_2 & b_1 & 1 \\ b_5 & b_4 & b_3 & b_2 \\ 0 & 0 & b_5 & b_4 \end{vmatrix} > 0, \quad \begin{vmatrix} b_1 & 1 & 0 & 0 & 0 \\ b_3 & b_2 & b_1 & 1 & 0 \\ b_5 & b_4 & b_3 & b_2 & b_1 \\ 0 & b_6 & b_5 & b_4 & b_3 \\ 0 & 0 & 0 & b_6 & b_5 \end{vmatrix} > 0,
$$

are satisfied.

4.3. Global Stability of the endemic Equilibrium

To show the global stability [3, 9] behavior of E^*, we need the bounds of dependent variables involved. For this, we find the region of attraction stated in the form of following lemma, stated below

Lemma 1: The region

$$\Omega = \left\{(N,E,I,Q,V);\ 0 < S(t) \le \overline{N};\ 0 \le L(t) \le \overline{L};\ 0 \le I_s(t) + I_a(t) \le \overline{I};\ 0 \le Q(t) \le \overline{Q};\ 0 \le V(t) \le \overline{V};\right\} \tag{4.3}$$

is a region of attraction for the system (2.1)-(2.6).

where, $\overline{S} = \dfrac{\Lambda}{d}$, $\overline{L} = \dfrac{\Lambda}{d(\delta + d)}$, $\overline{I} = (\overline{I}_s + \overline{I}_a) = \dfrac{\beta_3 \overline{S}\,\overline{V}}{\sigma - \beta \overline{S}}$, $\overline{Q} = \dfrac{\zeta\, \overline{I}}{(\xi + d + \alpha)}$, $\overline{V} = \dfrac{2\gamma\Lambda}{\gamma_0 d}$,

here, $\beta = \max(\beta_1, \beta_2)$, $\zeta = \max(\phi, \psi)$, $\sigma = \min\left((\phi + \alpha + d), (\psi + \rho + \alpha + d)\right)$

Theorem 1. If the endemic equilibrium E^* exists, then it is globally asymptotically stable provided the following sufficient conditions are satisfied in Ω

$$\left\{\frac{\beta_1 S^*\left(\eta\beta_2 S^* + \mu\right)}{\eta \overline{A}} + \frac{\beta_2 S^*\left(\beta_2 S^* - \rho\right)}{\overline{A}}\right\}^2 < \frac{1}{4} D_3 D_4 \frac{\beta_1 S^*\left(\beta_2 S^* - \rho\right)}{\eta(1-\eta)\,\overline{A}^2} \tag{4.4}$$

$$\frac{15\xi^2}{4(l+d)D_5} < \min\left\{\frac{\beta_1 S^* D_3 D_5}{3\phi^2 \eta \overline{A}}, \frac{\left(\beta_2 S^* - \rho\right) D_4 D_5}{3\psi^2 (1-\eta)\,\overline{A}}\right\} \tag{4.5}$$

$$5(\delta + lk_1)^2 < 4(l+d)(\delta + d)k_1 \tag{4.6}$$

Where $\overline{A} = \beta_1 \overline{I}_s + \beta_2 \overline{I}_a + \beta_3 \overline{V}$, $D_3 = \left\{\eta\beta_1 S^* - (\phi + \alpha + d)\right\}$,

$D_4 = \left\{(1-\eta)\beta_2 S^* - (\psi + \mu + \rho + \alpha + d)\right\}$, $D_5 = (\xi + \alpha + d)$

Proof. Consider the following positive definite function about E^*,

$$P = \frac{1}{2}\left(S - S^*\right)^2 + \frac{1}{2}k_1\left(L - L^*\right)^2 + \frac{1}{2}k_2\left(I_s - I_s^*\right)^2 + \frac{1}{2}k_3\left(I_a - I_a^*\right)^2 + \frac{1}{2}k_4\left(Q - Q^*\right)^2 + \frac{1}{2}k_5\left(V - V^*\right)^2$$

Differentiating P with respect to t, we get

$$\frac{dP}{dt} = \left(S - S^*\right)\frac{dS}{dt} + k_1\left(L - L^*\right)\frac{dL}{dt} + k_2\left(I_s - I_s^*\right)\frac{dI_s}{dt} + k_3\left(I_a - I_a^*\right)\frac{dI_a}{dt} + k_4\left(Q - Q^*\right)\frac{dQ}{dt} + k_5\left(V - V^*\right)\frac{dV}{dt}$$

Using equations (2.1)-(2.6) and simplifying, we get

$$\frac{dP}{dt} = -(\beta_1 I_s + \beta_2 I_a + \beta_3 V + l + d)(S - S^*)^2 - k_1(\delta + d)(L - L^*)^2 - k_2\{\eta\beta_1 S^* - (\phi + \alpha + d)\}(I_s - I_s^*)^2$$
$$- k_3\{(1-\eta)\beta_2 S^* - (\psi + \mu + \rho + \alpha + d)\}(I_a - I_a^*)^2 - k_4(\xi + \alpha + d)(Q - Q^*)^2 - k_5\gamma_0(V - V^*)^2$$
$$+ (\delta + lk_1)(S - S^*)(L - L^*) + (-\beta_1 S^* + \eta(\beta_1 I_s + \beta_2 I_a + \beta_3 V)k_2)(S - S^*)(I_s - I_s^*)$$
$$+ (-\beta_1 S^* + (1-\eta)(\beta_1 I_s + \beta_2 I_a + \beta_3 V)k_3 + \rho)(S - S^*)(I_a - I_a^*) + \xi(S - S^*)(Q - Q^*)$$
$$- \beta_3 S^*(S - S^*)(V - V^*) + (k_2(\eta\beta_2 S^* + \mu) + (1-\eta)\beta_2 S^* k_3)(I_s - I_s^*)(I_a - I_a^*)$$
$$+ (\eta\beta_3 S^* k_2 + \gamma k_5)(I_s - I_s^*)(V - V^*) + \phi k_4(I_s - I_s^*)(Q - Q^*) + \psi k_4(I_a - I_a^*)(Q - Q^*)$$
$$+ ((1-\eta)\beta_3 S^* k_2 + \gamma k_5)(I_a - I_a^*)(V - V^*)$$

$$\frac{dP}{dt} = -\frac{1}{2}a_{11}(S - S^*)^2 + a_{12}(S - S^*)(L - L^*) - \frac{1}{2}a_{22}(L - L^*)^2$$
$$- \frac{1}{2}a_{11}(S - S^*)^2 + a_{13}(S - S^*)(I_s - I_s^*) - \frac{1}{2}a_{33}(I_s - I_s^*)^2$$
$$- \frac{1}{2}a_{11}(S - S^*)^2 + a_{14}(S - S^*)(I_a - I_a^*) - \frac{1}{2}a_{44}(I_a - I_a^*)^2$$
$$- \frac{1}{2}a_{11}(S - S^*)^2 + a_{15}(S - S^*)(Q - Q^*) - \frac{1}{2}a_{535}(Q - Q^*)^2$$
$$- \frac{1}{2}a_{11}(S - S^*)^2 + a_{16}(S - S^*)(V - V^*) - \frac{1}{2}a_{66}(V - V^*)^2$$
$$- \frac{1}{2}a_{33}(I_s - I_s^*)^2 + a_{34}(I_s - I_s^*)(I_a - I_a^*) - \frac{1}{2}a_{44}(I_a - I_a^*)^2$$
$$- \frac{1}{2}a_{33}(I_s - I_s^*)^2 + a_{35}(I_s - I_s^*)(L - L^*) - \frac{1}{2}a_{55}(L - L^*)^2$$
$$- \frac{1}{2}a_{33}(I_s - I_s^*)^2 + a_{36}(I_s - I_s^*)(V - V^*) - \frac{1}{2}a_{66}(V - V^*)^2$$
$$- \frac{1}{2}a_{44}(I_a - I_a^*)^2 + a_{45}(I_a - I_a^*)(Q - Q^*) - \frac{1}{2}a_{55}(Q - Q^*)^2$$
$$- \frac{1}{2}a_{44}(I_a - I_a^*)^2 + a_{46}(Y - Y^*)(V - V^*) - \frac{1}{2}a_{66}(V - V^*)^2$$

$$a_{11} = \frac{2}{5}(\beta_1 I_s + \beta_2 I_a + \beta_3 V + l + d),\ a_{22} = 2k_1(\delta + d),\ a_{33} = \frac{1}{2}k_2\{\eta\beta_1 S^* - (\phi + \alpha + d)\},$$
$$a_{44} = \frac{1}{2}k_3\{(1-\eta)\beta_2 S^* - (\psi + \mu + \rho + \alpha + d)\},\ a_{55} = \frac{2}{3}k_4(\xi + \alpha + d), a_{66} = \frac{2}{3}k_5\gamma_0,\ a_{12} = (\delta + lk_1),$$
$$a_{13} = (-\beta_1 S^* + \eta(\beta_1 I_s + \beta_2 I_a + \beta_3 V + l + d)k_2),\ a_{46} = ((1-\eta)\beta_3 S^* k_3 + \gamma k_5)$$
$$a_{14} = (-\beta_1 S^* + (1-\eta)(\beta_1 I_s + \beta_2 I_a + \beta_3 V + l + d)k_3),\ a_{15} = \xi,\ a_{16} = -\beta_3 S^*$$
$$a_{34} = (k_2(\eta\beta_2 S^* + \mu) + (1-\eta)\beta_2 S^* k_3),\ a_{36} = (\eta\beta_3 S^* k_2 + \gamma k_5),\ a_{35} = \phi k_4,\ a_{45} = \psi k_4,$$

Now for *dP*/*dt* to be negative definite, the following conditions must be satisfied,

$$\left\{\frac{\beta_1 S^*\left(\eta\beta_2 S^* + \mu\right)}{\eta\overline{A}} + \frac{\beta_2 S^*\left(\beta_2 S^* - \rho\right)}{\overline{A}}\right\}^2 < \frac{1}{4} D_3 D_4 \frac{\beta_1 S^*\left(\beta_2 S^* - \rho\right)}{\eta(1-\eta)\,\overline{A}^2}$$

$$\frac{15\xi^2}{4(l+d)D_5} < k_4 < \min\left\{\frac{\beta_1 S^* D_3 D_5}{3\phi^2\eta\overline{A}}, \frac{\left(\beta_2 S^* - \rho\right)D_4 D_5}{3\psi^2(1-\eta)\,\overline{A}}\right\}$$

$$5(\delta + lk_1)^2 < 4(l+d)(\delta+d)k_1$$

Where $\overline{A} = \beta_1\bar{I}_s + \beta_2\bar{I}_a + \beta_3\overline{V}$, $D_3 = \left\{\eta\beta_1 S^* - (\phi+\alpha+d)\right\}$,

$D_4 = \left\{(1-\eta)\beta_2 S^* - (\psi+\mu+\rho+\alpha+d)\right\}$, $D_5 = (\xi+\alpha+d)$

And $k_2 = \frac{\beta_1 S^*}{\eta\overline{A}}$, $k_3 = \frac{\left(\beta_2 S^* - \rho\right)}{(1-\eta)\,\overline{A}}$ $\frac{15\left(\beta_3 S^*\right)^2}{4(l+d)\lambda_0} \le k_5$

Under these conditions, dP/dt will be negative definite showing that P is a Liapunov function with respect to E^* whose domain contains Ω.

V. SENSITIVITY ANALYSIS

Sensitivity indices allow us to measure the relative change in a variable when a parameter changes. The derivatives are the rate of change of predictions with respect to parameter. This work adopts the normalized forward sensitivity index to conduct the sensitivity analysis [15, 27, 29]. The normalized forward sensitivity index of a variable with respect to a parameter is the ratio of relative change in the parameter. When variable is differentiable function of the parameter, the sensitivity index may be alternatively defined using partial derivative. For instance, the normalized forward sensitivity index is

$$Y_M^{R_0} = \frac{\partial R_0}{\partial M} \times \frac{M}{R_0}$$

The parameter values displayed in below table are taken as the baseline and they are used to evaluate the sensitivity indices of some parameters which are responsible for the transmission dynamics of COVID-19 infectious disease to four places of decimal in relation to the effective reproduction number R_0, using equation (3.5) , the result of which is presented in table 1 below

$$\frac{\partial R_0}{\partial\beta_1}\frac{\beta_1}{R_0} = \frac{\beta_1}{U}\left[\eta + \frac{\mu}{\left(\psi+\mu+\rho+\alpha+d\right)}\right] \quad , \quad \frac{\partial R_0}{\partial\beta_2}\frac{\beta_2}{R_0} = \frac{(1-\eta)\beta_2(\phi+\alpha+d)}{U\left(\psi+\mu+\rho+\alpha+d\right)}$$

$$\frac{\partial R_0}{\partial\beta_3}\frac{\beta_3}{R_0} = \frac{\beta_3\gamma}{U\gamma_0}\left[\eta + \frac{(1-\eta)(\phi+\mu+\alpha+d)}{\left(\psi+\mu+\rho+\alpha+d\right)}\right] \quad , \quad \frac{\partial R_0}{\partial l}\frac{l}{R_0} = -\frac{l}{\left(l+\delta+d\right)}$$

$$\frac{\partial R_0}{\partial \phi}\frac{\phi}{R_0} = -\frac{\phi}{d(l+\delta+d)U}\left(\beta_1 + \frac{\beta_3\gamma}{\gamma_0}\right)\left[\eta + \frac{(1-\eta)\mu}{(\psi+\mu+\rho+\alpha+d)}\right]$$

$$\frac{\partial R_0}{\partial \psi}\frac{\psi}{R_0} = -\frac{\psi(1-\eta)}{(\psi+\mu+\rho+\alpha+d)^2 U}\left(\beta_1\mu + \beta_2(\phi+\alpha+d) + \frac{\beta_3\gamma(\phi+\alpha+d+\mu)}{\gamma_0}\right)$$

$$\frac{\partial R_0}{\partial \gamma}\frac{\gamma}{R_0} = \frac{\beta_3\gamma}{U\gamma_0}\left[\eta + \frac{(1-\eta)(\phi+\mu+\alpha+d)}{(\psi+\mu+\rho+\alpha+d)}\right], \quad \frac{\partial R_0}{\partial \gamma_0}\frac{\gamma_0}{R_0} = -\frac{\beta_3\gamma}{U\gamma_0}\left[\eta + \frac{(1-\eta)(\phi+\mu+\alpha+d)}{(\psi+\mu+\rho+\alpha+d)}\right]$$

Where,

$$U = \left(\beta_1 + \frac{\beta_3\gamma}{\gamma_0}\right)\eta + \frac{(1-\eta)}{(\psi+\mu+\rho+\alpha+d)}\left(\beta_1\mu + \beta_2(\phi+\alpha+d) + \frac{\beta_3\gamma(\phi+\alpha+d+\mu)}{\gamma_0}\right)$$

Table 1. Sensitivity index and indices Table

Parameter Symbol	Sensitivity indices
β_1	$1.75\text{x}10^{-7}$
β_2	$4.46\text{x}10^{-9}$
β_3	1.369861
l	-704188
ϕ	-502.3532
ψ	$-6.7\text{x}10^{-3}$
γ	1.369861
γ_0	-1.369861

From table we can see that the positive indices i.e. β_1, β_2, β_3 and γ show that they have great impact on expanding the disease in the population if their valve increases R_0 increases, it means the number of secondary infections increases in the population. Moreover, to make sure that $R_0 < 1$, we need to decrease the values of the effective contact rates (β_1, β_2 and β_3). Further the parameter l, ϕ, ψ and γ_0 for which the sensitivity indices is negative, shows that if these parameter will increase the basic reproduction number will decrease, which minimize the disease in the population. Thus as the rate of lockdown susceptible l increases the disease decreases but it is not possible for long time so test rate of the infective people should increase so that after detecting COVID-19 positive more and more infective will quarantine soon and ϕ and ψ increase which decrease the reproduction rate. It shows that the improving the diagnosis rate of COVID-19 is very beneficial to control the spread of COVID-19. As γ_0, the rate of elimination of corona virus density, increases R_0 decreases. Thus proper sanitization is also very helpful to reduce the disease up to a level.

VI. NUMERICAL SIMULATION AND DISCUSSION

To see the dynamical behavior of the model system, the system (2.1)-(2.6) is integrated numerically by fourth order Runge-Kutta method using the following set of parameters values:

$\Lambda = 1,\ d = 0.000038,\ \alpha = 0.001,\ \beta_1 = 0.1,\ \beta_2 = 0.3,\ \beta_3 = 0.5,\ \mu = 0.7,\ l = 0.5,\ \xi = 0.003,$
$\eta = 0.3,\ \delta = 0.21,\ \rho = 0.005,\ \phi = 0.01,\ \psi = 0.005,\ \gamma = 2\text{x}10^{-7},\ \gamma_0 = 10^{-13},\ \rho = 0.005,$

with initial values $S(0) = 155,\ L(0) = 0,\ I_s(0) = 0,\ I_a(0) = 0,\ Q(0) = 0$ and $V(0) = 1$.

The results of numerical simulation are displayed graphically in figs. (1 - 7). In fig. 1, the variation of asymptomatic infective population $I_a(t)$ with time t is shown for different values of β_2, the rate of transmission of susceptibles to infective class through direct contact with asymptomatic infectives present in the population. It is seen that asymptomatic infective population increases with increase in the value of β_2. This implies that if a person not known about his infection due to no symptom will spread the disease more fast because he will go every where without taking any precaution. In figs. 2 the variation of symptomatic infective population $I_s(t)$ with time t is shown for different values of β_1, the rate of transmission of susceptibles to infective class through direct contact with symptomatic infectives present in the population. It is seen that symptomatic infective population increases with increase in the value of β_1. In figs. (3 - 4), the variation of asymptomatic infective population $I_a(t)$ and symptomatic infective population $I_s(t)$ respectively is shown with time t for different values of l, the lockdown rate of the susceptibles. It is noted that with increase in the lockdown rate of susceptible, asymptomatic and symptomatic infective population decreases. This indicates that the individual should keep isolated himself as much as possible during the covid period and he should go out side the house only if there is very urgent work. Fig (4-7) the variation of asymptomatic infective , symptomatic infectives and quarantine population with time t for different value of ϕ, the rate of transfer of the symptomatic infectives to the quarantined class. It is found that as the value of ϕ increase infective populations decrease and quarantine population increase. This indicates that if rate of transfer of infectives into quarantined class increases, the population in quarantined class who are either isolated at home or hospital increases. Since this increased population of quarantined individuals is isolated, it does not contribute to viral transmission further and hence, the spread of the disease can be lowered. It means as the testing rate of infectives should increase as much as possible so that a person can know about their infection at early stage and can quarantine at home or hospital.

From the above discussion, it follows that if more and more susceptible individuals either lockdown themselves by following the COVID-19 guidelines or quarantined at home or hospital, the spread of the disease can be controlled. It is also observed that if the testing rate of COVID-19 will increase, more and more infectives will quarantine soon and will not take part in spreading the disease which helps the epidemic under control.

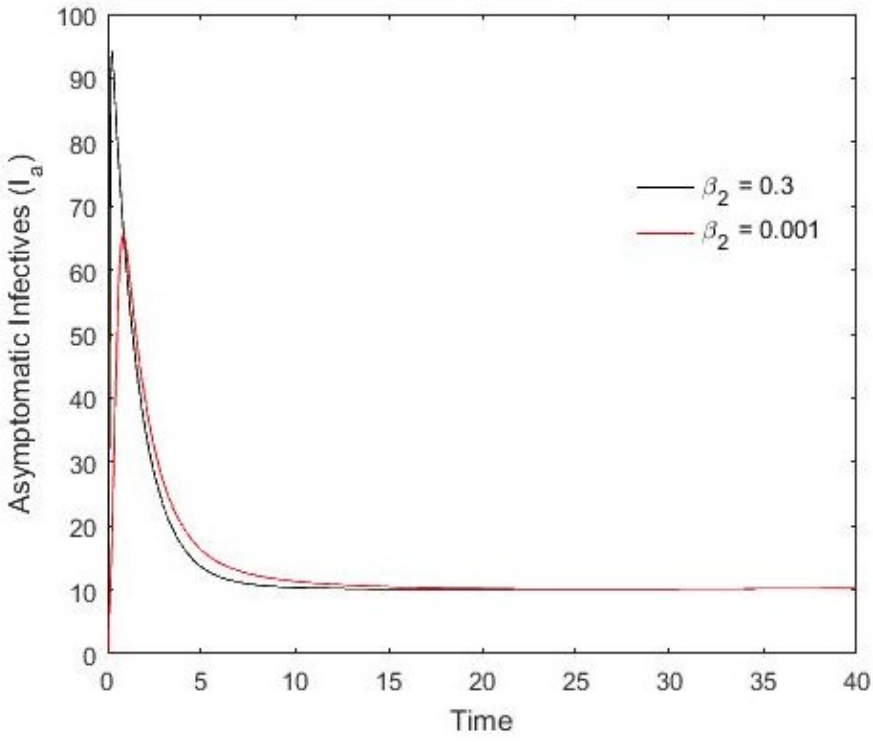

Figure 1. Variation of asymptomatic infective population for different value of β_2

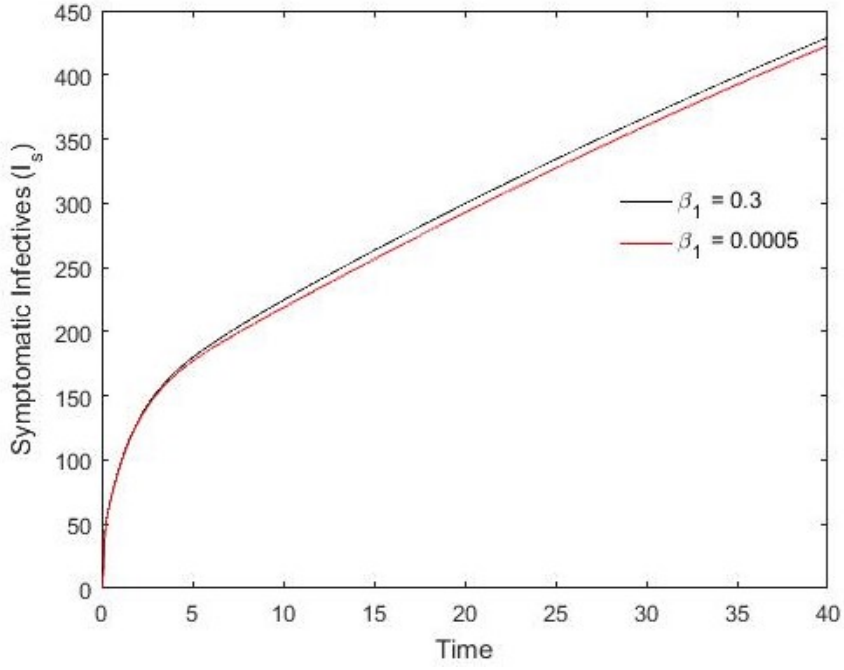

Figure 2. Variation of symptomatic infective population for different value of β_1

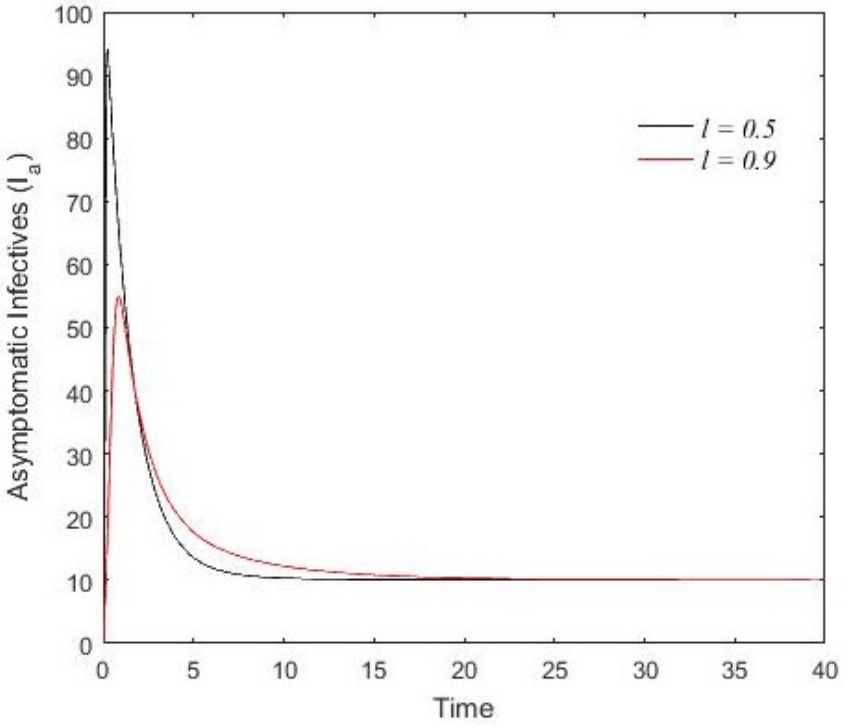

Figure 3. Variation of asymptomatic infective population for different value of l

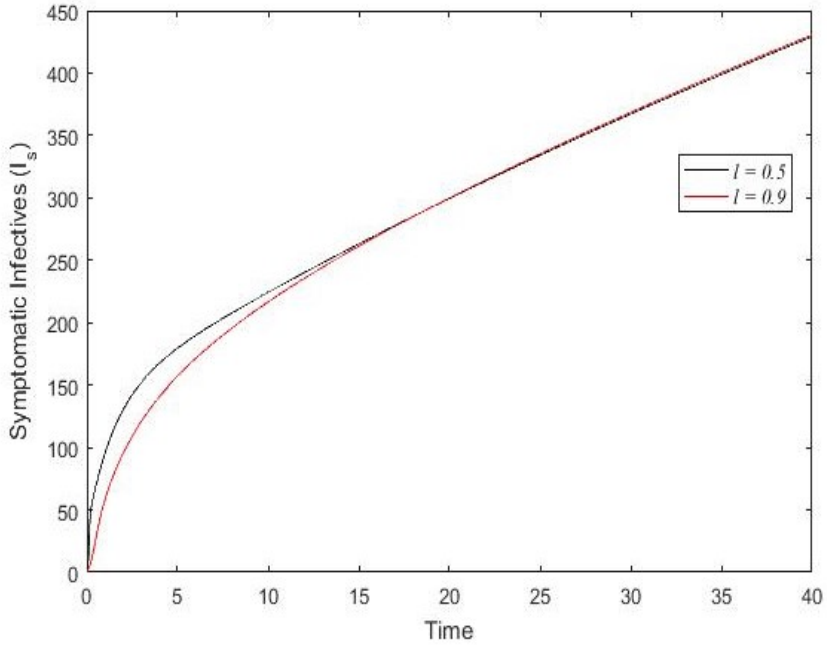

Figure 4. Variation of symptomatic infective population for different value of l

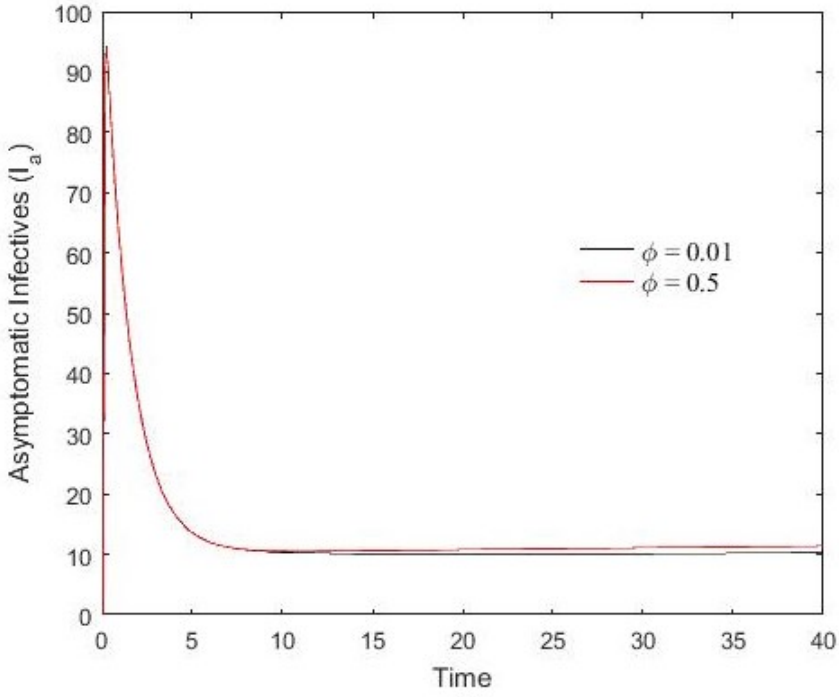

Figure 5. Variation of asymptomatic infective population for different value of ϕ

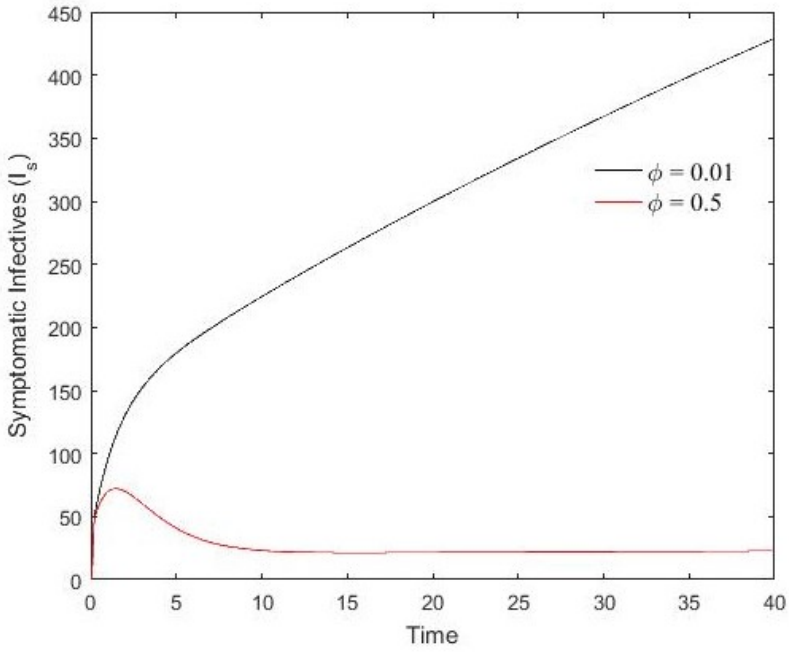

Figure 6. Variation of symptomatic infective population for different value of ϕ

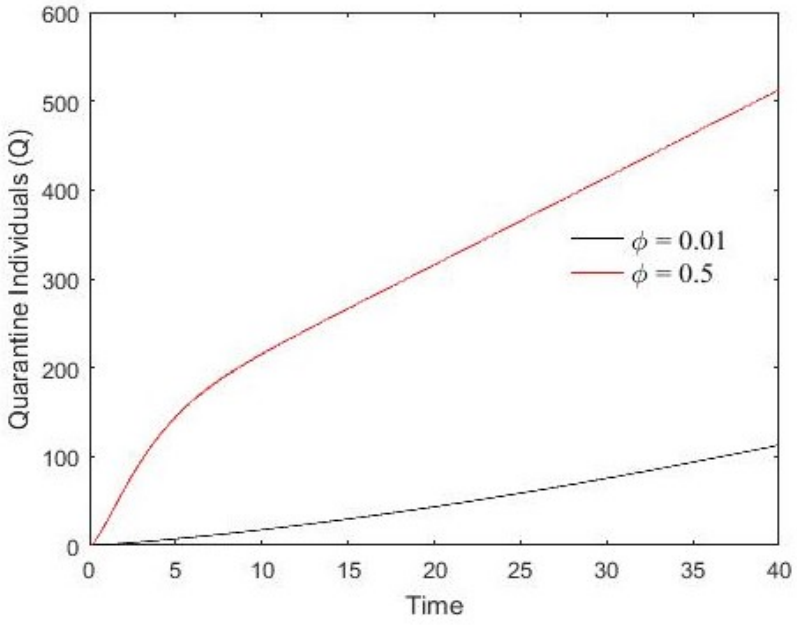

Figure 7. Variation of quarantine population for different value of ϕ

VII. CONCLUSION

In this paper, a nonlinear mathematical model has been proposed and analyzed to study the effect of self-protection and quarantine strategy on the spread of corona virus in a population with variable size structure. The analysis of the proposed model has been done using stability theory of differential equations and computer simulations. The model exhibits two equilibria namely, the disease-free and the endemic equilibrium. The local and global stability results of these equilibria have been established. It is found that if the individuals from susceptible population lockdown themselves, the infective population can be decreased. This decrease is further affected if the individuals from infective populations will tested at early stage and quarantine themselves. Moreover, if higher numbers of individuals from infective classes are quarantined at home or hospital, the spread of the disease can be slowed down.

Finally from the analysis, it may be concluded that lock down help to reduce the disease up to a level. The roll of asymptomatic infectives are very crucial in spreading the disease because they don't show any symptoms and there screening rate is very low. Hence they can go any where and spread the disease easily. So random screening should increase in the public places. It is also found that if the virus density will decrease by sanitization, isolation or by other means the then infectives does not reach the certain threshold, the epidemic can be kept under control.

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