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FORECAST ANALYSIS FOR USING HYPERTENSION AND HEART DISEASE DATA IN TAMILNADU

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Abstract

In this paper study was to inspect the hypertension and heart disease cases in Tamilnadu between January 1, 2016 and December 31, 2020. Cases of hypertension have been found to be connected to cases of heart disease. The Breusch-Godfrey LM methodology for assessing residual serial auto correlation assumes a VAR model for the error vector ε_t,, and Granger causality tests showed that hypertension might cause heart disease but not the other way around. Cases of hypertension have been found to be linked to cases of heart disease. The model selection is the Akaike Information Criterion (AIC), Schwarz Information Criterion (SIC), Hannan-Quinn Information Criterion (HQ), and the Final Prediction Error (FPE) and stationarity of the data can be used to determine The Augmented Dickey Fuller (ADF) test, Philip Perrons (PP) test, and the Kwiatkowski-Phillip-Schmidt-Shin (KPSS) test were used to test the series. For the ADF and PP tests, respectively.

Key words: hypertension, heart disease, VAR model, Breusch-Godfrey LM methodology,Granger causality tests.

1. Introduction

Instead of being a one-dimensional static measurement, blood pressure is a dynamic, multidimensional, cardiovascular indicator of a person's status. Blood pressure is defined as the force of blood against the artery walls. It's expressed as a ratio of SYSTOLIC pressure (when the heart beats) to DIASTOLIC pressure (when the heart relaxes between beats). (Thomas and colleagues, 2002). High blood pressure is neither a sickness or an illness; rather, it is a risk factor for illnesses that one wants to avoid. Strokes, heart attacks, kidney issues, and other circulatory system problems are among these ailments (blood circulation). High blood pressure is a common and significant modifiable risk factor for heart and renal disease. Hypertension, particularly isolated systolic hypertension, becomes more common as people get older is discussed in Fahey et al.(2004).

There are two types of high blood pressure: namely and high blood pressure affects 90 to 95 percent of people. This disease has no known a etiology, but high blood pressure does have a known cause. This sort of high blood pressure is caused by another illness, and it normally goes away after the underlying issue is managed or cured is discussed in Pickering (1988). The more blood one needs to deliver oxygen and nourishment to the tissues, the more one weighs. Being inactive physically. Smoking momentarily elevates blood pressure. A diet high in salt leads the body to retain water, which raises blood pressure.

Blood pressure can be raised by drinking too much alcohol. High cholesterol, diabetes, sleep apnea, and kidney disease are among chronic illnesses that increase the risk of high blood pressure. Pregnancy can sometimes contribute to high blood pressure. Is discussed in Pickering (1988). The causes of high blood pressure are still a source of controversy. The great majority of people (over 95 percent) do not have an underlying reason. In most persons, multiple variables are likely to contribute to high blood pressure. Genetic factors and lifestyle choices are the leading suspects. Family history, age, and body shape are all genetic influences. Drinking, smoking, work out rate rate, stress, obesity, and a high salt intake are examples of lifestyle and habits.

Changing one's lifestyle, adjusting one's diet, exercising, and quitting smoking are the greatest non-drug treatments for high blood pressure. They reduce the many dangers that can induce high blood pressure or elevate cardiovascular risk levels. According to statistics, increasing activity, dropping weight, reducing alcohol use, and modifying nutrition (cutting salt intake and increasing fruit and vegetable intake) will result in a systolic blood pressure reduction of roughly 4 mmHg on average if these changes are maintained. (Fahey and colleagues, 2004.)

2. Model description

The Vector Autoregressive (VAR) model will be used to model the data. The VAR model, which is a multivariate version of the univariate Autoregressive (AR) model, demonstrates that present and previous values are related. In response, using the VAR model, which was pioneered by Box and Tiao, is one technique to incorporate the influence of one variable on other variables across time (1981). Its primary applications are structural analysis and forecasting. The Vector Autoregressive (VAR) modelling strategy was used in this work as the specific modeling technique. Sims(1980) are proposed the use of the VAR model for time series. The suitable lag length (p), which should be long enough to prevent serial correlation of the residuals, can bebased on established model selection criteria.

2.1. The Vector Autoregressive models.

supposeY_t is a K by 1 vector stochastic process. A p^{th} order VAR model of consistent time series, written as VAR(p), is a process evolve according to

$$Y_t = \Phi_0 + \Phi_0 Y_{t-1} + \Phi_0 Y_{t-2} \cdots \Phi_0 Y_{t-p} + \epsilon_t \qquad \cdots (1)$$

Where,Φ_0 is a k by 1 vector of interrupt parameters, Φ_j is a k by k parameter matrices, with $j = 1,2, \ldots, p$ and ϵ_t is a vector.The general approach to VAR model estimation is to fit the$VAR\ (p)$ with the order $p = 0, \ldots, p_{max}$ and the "value of p should minimize some model selection criteria. Model selection criteria for VAR (p)" models have the form

$$IC(p) = ln\left|\sum(p)\right| + c_T.\varphi(n,p)$$

Where,

$\sum(p) = T^{-1}\hat{\varepsilon}_t\ \hat{\varepsilon}_t'$ is the residual covariance matrix without a df. of correction from the $VAR\ (p)$ model, "c_T is a sequence indexed by the sample size T, and $\varphi(n,p)$ is a penalty function which penalizes large VAR (p) models".The Akaike (AIC), Schwarz-Bayesian (BIC) and Hannan-Quinn (HQ) derived to

$$AIC(p) = ln\left|\sum(p)\right| + \frac{2}{T}pn^2 \qquad \cdots (2)$$

$$BIC(p) = ln\left|\sum(p)\right| + \frac{ln[T]}{T}pn^2 \qquad \cdots (3)$$

$$HQ(p) = ln\left|\sum(p)\right| + \frac{2ln[lnT]}{T}pn^2 \qquad \cdots (4)$$

After a VAR-model has been estimated, it's crucial to examine if the residuals follow the model's assumptions. That is, the error process should be checked for serial correlation and heteroscedasticity, as well as if it is regularly distributed. The LM test devised by Breusch [1978] and Godfrey [1978] is used to check for serial correlation in the residuals of a VAR (p)-model.

2.2. The Breush-Godfrey LM Test VAR models.

For testing residual serial auto correlation, the Breusch-Godfrey LM methodology assumes a VAR model for the error vector"$\varepsilon_t = D_1\varepsilon_{t-1} + \cdots + D_h\varepsilon_{t-h} + v_t$ where v_t is white noise.ε_t is equal to v_t if there is no residual serial correlation". As a result, we test the hypotheses.

$$H_0: D_i = D_1 \ldots . D_h = 0\ against$$
$$H_1: D_j \neq 0 \qquad for\ j = 1,2, \cdots, h$$

The Breusch-Godfrey LM test statistic is given by for any K dimensional VAR model

$$LM_h = t\left(\ k - tr\ (\bar{\Sigma}_R^{-1}\hat{\Sigma}_e)\right) \qquad \cdots (5)$$

2.3. The Granger Causality test for VAR models.

Forecasting is one of the most common applications of VAR models. The VAR model's structure gives information about a variable's or a collection of variables' capacity to forecast other variables. Granger is responsible for the following intuitive notion of a variable's predicting capacity (1969). Granger causality is a good indicator that a VAR is required instead of a univariate model. If a scalar random variable $\{x_t\}$ does not Granger cause $\{y_t\}$, it is said to not Granger cause $\{y_t\}$

$$E\left[y_t/x_{t-1}, y_{t-1}, x_{t-2}, y_{t-2}, \ldots\right] = E\left[y_t/y_{t-1}, y_{t-2}, \cdots\right]$$

Forecasting is a common application of VAR models. The VAR model's structure reveals how well a variable or a collection of variables can forecast other variables. Granger is responsible for the following intuitive sense of a variable's ability to forecast (1969). Granger causality is a good indicator that a VAR is needed instead of a univariate model. If a scalar random variable $\{x_t\}$does not Granger cause $\{y_t\}$, it is said that it is not Granger cause.

$$\begin{bmatrix} x_t \\ y_t \end{bmatrix} = \begin{bmatrix} \phi_{11.1} & \phi_{12.1} \\ \phi_{21.1} & \phi_{22.1} \end{bmatrix}\begin{bmatrix} x_{t-1} \\ y_{t-1} \end{bmatrix} + \begin{bmatrix} \phi_{11.2} & \phi_{12.2} \\ \phi_{21.2} & \phi_{22.2} \end{bmatrix}\begin{bmatrix} x_{t-2} \\ y_{t-2} \end{bmatrix} + \begin{bmatrix} \epsilon_{1,t} \\ \epsilon_{2,t} \end{bmatrix}$$

If $\phi_{21.1} = \phi_{21.2} = 0$, then $\{x_t\}$does not Granger cause $\{y_t\}$in general. If $\{x_t\}$does not Granger cause $\{y_t\}$and $\epsilon_{1,t}$ and $\epsilon_{2,t}$ have no contemporaneous correlation, $\{y_t\}$ is said to be weakly exogenous, and $\{y_t\}$ can be modelled totally independently of $\{x_t\}$. This test can be performed regardless of whether $\{x_t\}$ Granger is the cause of $\{y_t\}$. The coefficients of the past values of X are then tested using an F-test to see if they are all zero. The null hypothesis in this test is that X does not cause Y. Consider the following bivariate VAR (p) model given in scalar form:

$$x_t = \phi_1 + \sum_{i=1}^{p} \phi_{11}^{(i)} x_{i,t-i} + \sum_{i=1}^{p} \phi_{12}^{(i)} x_{i,t-i} + \epsilon_{1t}$$

$$y_t = \phi_1 + \sum_{i=1}^{p} \phi_{21}^{(i)} x_{i,t-i} + \sum_{i=1}^{p} \phi_{22}^{(i)} x_{i,t-i} + \epsilon_{2t}$$

Then an F-test for the joint significance of OLS regression of F-statistic for evaluating the hypothesis is an F-test for Granger causality from x to y. in any regression model is

$$y = \beta_0 + \beta_{1x1} + \cdots \beta_k x_k + \mu \quad \cdots (6)$$

$$F = \frac{(SSE_r - SSE_{ur})/q}{SSE_{ur}/(n-k-1)} \cdots (7)$$

where "SSE_r is the residual sum of squares from the model under H_0 and SSE_u" is (the residualsum of squares for the model in equation (6). Under the null hypothesis the test statistic in equation (7) is F-distributed with$q = df_r - df_{ur}$ and $n - k - 1$ degrees of freedom.)

The Granger causality in a bivariate $VAR\ (p)$ model we drop "$q = p$ variables in a model with $n = T$ observations and $k = 2p$ variables"clear of the constant.

Hence,

$$F = \frac{(SSE_r - SSE_{ur})/p}{SSE_{ur}/(T-2p-1)} \sim F(p, T-2p-1) \quad \cdots (8)$$

2.4. Forecast Error Variances.

The forecast error variance into components due to shocks in the series is referred to as variance decomposition. We may decompose the error variance of the s step-ahead forecast of y_{it} into components accounted for by these innovations since shocks vt is are uncorrelated. Consider a vector MA representation of an orthogonalized VAR with m components.

$$y_t = \sum_{l=0}^{\infty} \psi^*(l) v_{t-1} \quad \cdots (9)$$

The $s$step-ahead forecast for $y_t$is then

$$E_t(y_{t+s}) = \sum_{l=s}^{\infty} \psi^*(l) v_{t+s-l} \quad \cdots (10)$$

Defining the s step-ahead forecast as,

$$\theta_{t+s} = y_{t+s} - E_t(y_{t+s}) \quad \cdots (11)$$

We get,

$$\theta_{t+s} = \sum_{l=s}^{\infty} \psi^*(l) v_{t+s-l} \quad \cdots (12)$$

and its i'th component is given by

$$e_{i,t+s} = \sum_{l=0}^{s-l} \sum_{j=0}^{m} \psi^*{}_{ij}(l) v_{j,t+s-l} = \sum_{j=0}^{m} \sum_{l=0}^{s-1} \psi^*{}_{ij}(l) v_{j,t+s-l} \quad \cdots (13)$$

We now have for the error variance because the shocks are both serially and contemporaneously uncorrelated.

$$V(e_{i,t+s}) = \sum_{l=0}^{s-l} \sum_{j=0}^{m} V\left(\psi^*{}_{ij}(l)\right) v_{j,t+s-l} = \sum_{j=0}^{m} \sum_{l=0}^{s-1} \psi^*{}_{ij}(l)^2 v_{j,t+s-l}$$

All shock components have the same unit variance, which means that

$$V(e_{i,t+s}) = \sum_{j=0}^{m} \left(\sum_{l=0}^{s-1} \psi^*{}_{ij}(l)^2 \right) \quad \cdots (14)$$

$y_j$is error variance is taken into account. By comparing this to the total number of innovation responses, we can gain a sense of how important variable j is innovations are in explaining variance in variable i at various step-ahead estimates, i.e.,

$$R^2_{ij,s} = 100 \frac{\sum_{l=0}^{s-1} \psi^*{}_{ij}(l)^2}{\sum_{k=1}^{m} \sum_{l=0}^{s-1} \psi^*{}_{ij}(l)^2} \cdots (15)$$

(Forecasting the $h - step\ ahead\ of\ a\ VAR\ (p)$ process, $\hat{y}_{t+h|t}$ is given by the formula)

$$E_t[y_{t+h}] = \sum_{j=0}^{h-1} \Phi_1^j \Phi_0 + \Phi_1^j y_t \quad \cdots (16)$$

(Forecasts from higher order VARs can be constructed by direct forward recursion beginning at h = 1, but it is often computed using the deviations from the VAR since it includes no intercept)

$$\tilde{y}_t = \Phi_1 \tilde{y}_{t-1} + \Phi_2 \tilde{y}_{t-2} + \quad \cdots + \Phi_p \tilde{y}_{t-p} + \epsilon_t$$

Using the deviations form *h*-step ahead forecasts is

$$E_t[\tilde{y}_{t+h}] = \Phi_1 E_t[\tilde{y}_{t+h-1}] + \Phi_2 E_t[\tilde{y}_{t+h-2}] + \cdots + \Phi_p E_t[\tilde{y}_{t+h-p}] \cdots (17)$$

(starting at $E_t[\tilde{y}_{t+h-1}]$. Once the forecast of $E_t[\tilde{y}_{t+h}]$has been computed the *h*-step ahead forecast of y_{t+h} ahead is constructed by adding the long run mean)

$$E_t[y_{t+h}] = \mu + E_t[\tilde{y}_{t+h}]$$

3. Results and Discussion.

Two aggregate series of cases, namely hypertension and heart disease cases, are used in the empirical study. Table 1 shows some descriptive data, such as the mean, median, lowest, and maximum values of the instances. According to the table, the minimal number of monthly hypertension cases during the study period is 3 and 0 for cardiac cases. The highest number of instances of hypertension was 717, while the highest number of cases of heart disease was 57. In Tamilnadu, the average monthly number of hypertension cases was 132.92, and the average monthly number of heart disease cases was 9.935. The sample data for the five years contains 60 data points, with the median number of hypertension and heart disease cases being 65 and 6, respectively.

Table – 1: Descriptive Analysis of Hypertension, Heart Disease.

Diseases	Observations	Mean.	Median.	Minimum	Maximum
Hypertension.	60.	133.920	64	03	727
Heart.	60.	09.9330	8	00	56

Table 2 shows an investigation of the amount of hypertension cases every month. The month of January had the greatest average number of cases during the study period (1 January 2016 to 31 December 2020), with 270 cases on average, which could be linked to overindulgence in fatty foods and alcoholic beverages during the holidays. Many people are also too concerned during this time because they are worried about how they will provide for their families.

The month of October has the lowest average number of instances (46.81 number of cases). In terms of the maximum and minimum number of instances, the highest number of hypertension cases (716 cases) happened in February, while the lowest number of cases occurred in October (3 cases).

Table-2:

Month	Mean.	Minimum	Maximum	Median.
JANUARY	270.0	28	711	227
FEBRUARY	215.02	5	707	76
MARCH	106.0	28	269	74
APRIL	60.02	29	97	58
MAY	65.60	29	114	60
JUNE	62.40	11	203	28
JULY	136.80	5	561	32
AUGUST	188.40	6	611	64
SEPTEMBER	120.0	6	283	42
OCTOBER	46.80	2	181	22
NOVEMBER	159.40	11	527	78
DECEMBER	163.20	20	533	83

Table 2 also includes summary information for cases of heart disease, just as it does for cases of hypertension. The month of March had the highest average number of heart disease cases (16.8), while November had the lowest average number of cases (11.2). (ie 4.4 average number of cases). The highest number of heart disease cases (57 cases) occurred in March, and the lowest number (0 instances) occurred in June.

Table-3:

Month	Mean.	Minimun	Maximum	Median.
JANUARY	10.40	3	26	8
FEBRUARY	11.60	0	32	7
MARCH	16.80	2	58	9
APRIL	14.40	0	53	6
MAY	5.3	1	13	4
JUNE	13	0	34	8
JULY	9.20	3	19	8
AUGUST	12.14	4	30	7
SEPTEMBER	11.16	2	26	12
OCTOBER	5.02	5	8	4
NOVEMBER	4.04	0	12	3
DECEMBER	5.18	2	11	4

3.1. Unit root series appears to be stationary result.

Time series plots, the Augmented Dickey Fuller (ADF) test, the Philip Perrons (PP) test, and the Kwiatkowski-Phillip-Schmidt-Shin (KPSS) test were used to determine the series' stationarity. Unit Root Test Results for Hypertension for both the ADF and PP tests Figure -1 shows that, despite the fact that some of the monthly numbers of hypertension cases are extremely high, there is no pattern. The series appears to be stationary as a result of this. Even though there is a considerable increase at the first lag of the PACF, the ACF plots do not slowly decline, which supports this observation. However, until the numerous unit root tests validate these facts, they may only be considered a suspicion. The ADF test, the PP test, and the KPSS test were used to determine stationarity in this study.

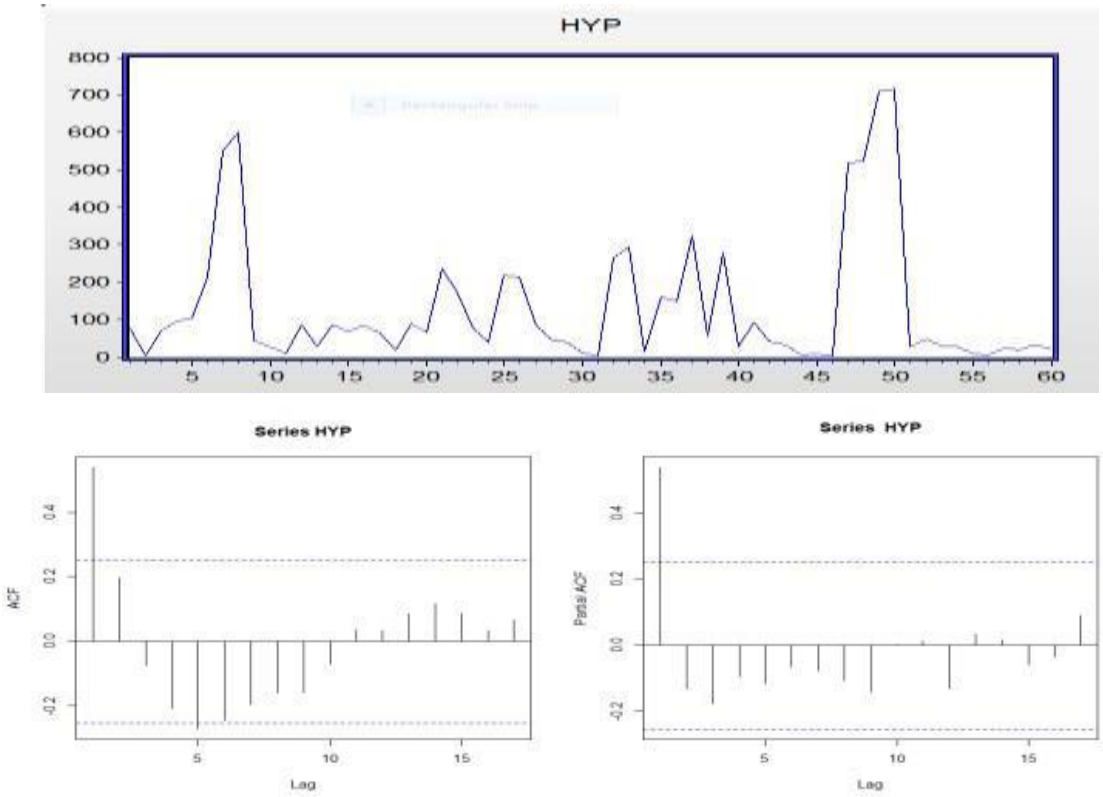

Figure -1: A forecasting the hypertension cases at levels in Tamilnadu district

Whether or not a constant and/or trend is included, or none at all, the hypertension series is stationary. Tables 4 and 5 provide evidence for this claim. As a result, the series does not need to be differentiated. In this instance, the series is said to be integrated of order zero, i.e. $I(0)$.

Table -4: ADF test results for hypertension cases

HYPERTENSION	TEST STATISTIC	P-VALUE
CONSTANT	-4.1020	0.0020
CONSTANT+TREND	-4.0604	0.0118
NONE	-3.1675	0.0020

From Table 4 all the p-values for the ADF tests are less than the conventional significancelevel of 0.05 which is an indication of stationarity per the test of hypotheses provided above.

Table -5: The PP Stationarity test for hypertension.

HYPERTENSION.	Test Statistics	P-s
CONSTANT.	-4.1526	0.0017
CONSTANT+TREND	-4.1098	0.0103
NONE.	-3.2019	0.0018

Table 5 also shows that all of the PP test's p-values are smaller than the traditional significance criterion of 0.05, indicating that the hypotheses are stationarized.

Table -6:KPSS unit root test results for hypertension

Test	Test Statistics.	P-value.
KPSS.	0.063	0.1

Because a p-value greater than 0.05 is an indication of stationarity in the data for the KPSS test, the results of the ADF and the PP tests are confirmed.

Table -7: ADF units root test result for heart diseases cases

HEART DISEASE,	Test Statistic.	P-value.
CONSTANT.	-4.2458	0.0013
CONSTANT+TREND.	-4.4031	0.0045
NONE.	-3.0821	0.0026

Table -8: PP units root test results for heart diseases cases

HEART DISEASES	TEST STATISTIC	P-VALUE
CONSTANT.	-4.2153	0.0014
CONSTANT+TREND.	-4.2973	0.0061
NONE	-2.9628	0.0037

Table -9: KPSS units root test results.

Test	Test Statistic.	P-value.
KPSS	0.3962	0.0788

The heart instances, like the hypertension patients, are stationary at levels, according to Tables 7, 8, and 9.

3.3. Estimation for the VAR model.

The number of delays contained in a VAR model determines its order. The lag length (p) should be calculated to ensure that the residuals are not serially correlated. In diagnostic testing as well as the estimation of VAR models for impulse response analysis and variance decomposition, the lag length is critical (Bhasin, 2004). The inclusion of two lags for the VAR order is supported by four of the selection criteria: the Final Prediction Error (FPE), Akaike Information Criterion (AIC), Schwarz Information Criterion (SC), and Hannan-Quinn Information Criterion (HQ).

The inclusion of two lags is supported by the majority of the criteria, as well as the AIC, which was the key guideline. As a result, the predicted VAR is VAR(2), and the long run association for heart disease and hypertension cases is estimated. Table -11 shows the long run association results for heart disease cases estimated by the VAR (2) model.

Table -10: The long run estimation for result in heart disease

Variable.	Lag.	Estimates	Standard error	t-values	p-values
Heart,	1	0.4084	0.0931	3.0983	0.0023**
Hypertension	1	0.0070	0.0138	0.8401	0.3012
Heart,	2	-0.0923	0.1871	-0.0983	0.2331
Hypertension	2	0.0342	0.0339	3.9330	0.1020**
Constant		2.9840	1.9300	1.2300	0.3891

Table -12 shows the long run association outcomes for hypertension patients predicted by the VAR (2) model.

Table -11: The estimation for the results in hypertension

Variable.	Lag.	Estimates	Standard error	t-values	p-values
Heart,	1	-1.4034	2.0931	-3.098	0.0923
Hypertension	1	0.5060	0.0038	4.841	0.0001***
Heart,	2	1.0973	2.1891	0.983	0.2531
Hypertension	2	-0.0362	0.0439	-0.930	0.6020**
Constant		67.9840	33.9360	2.200	0.0891*

Estimated VAR (2) models are

$$HRT_t = 0.4084\, HRT_{t-1} + 0.0070\, HYPt_{t-1} - 0.0917 HRT_{t-2} + 0.0261\, HYP_{t-2} + 2.4621 \quad \cdots (18)$$

$$HYPt = -1.6614\, HRT_{t-1} + 0.6070\, HYP_{t-1} + 1.200 HRT_{t-2} - 0.0968 HYP_{t-2} + 71.4235 \quad \cdots (19)$$

Equation (18) shows that when the lagged values of heart disease change by one unit, the disease is greatly changed positively by 40%. It may also be deduced from the equation that when the lagged values of hypertension vary by two units, heart illnesses are changed favorably by roughly 3%. When the lagged values are changed by a unit, hypertension is influenced by almost 61 percent, and the constant term is affected by 71 percent, according to equation (19).

3.4. The Granger Causality Test.

When the coefficient of the lagged of the other variable is not zero, the Granger causality test is considered a useful tool for identifying whether one time series is good for predicting. At the standard significance threshold of 5%, the results suggest that hypertension Granger causes heart disease. This indicates that hypertension data from the past can be used to predict future heart disease rates. In the case of this study, however, the opposite is true.

Table -12: presents the result from the Granger causality tests.

NULL HYPOTHESIS	F-STATISTIC	P-VALUE
Hypertension does not Granger-cause Heart Disease	-28.938	0.0005
Heart Disease does not Granger-cause Hypertension	-20.3293	0.7209

At the standard significance threshold of 5%, the results suggest that hypertension Granger causes heart disease. This indicates that hypertension data from the past can be used to predict future heart disease rates. In the case of this study, however, the opposite is true. The preceding test clearly failed to disprove the premise that "heart disease does not induce hypertension."

In the VAR model, the dependent variables are exposed to shocks caused by each of the variables. It aids in determining how the variable reacts when the error terms are given a positive shock of one standard deviation, as well as how the variables react to each other. So a unit shock is administered to the erroneous terms for each variable from each equation independently, and the consequences on the VAR system over time are observed. The shocks on the level of the endogenous variables in the VAR are identified using a conventional decomposition.

4. Conclusion

In Tamilnadu, the forecast for the year 2020 predicts a slight increase in the number of cases of heart disease. The fact that the trend or increase is minor does not mean that the area's stakeholders should let their guard down. Instead, the Ministry of Health should engage with local health workers to give indigenes with extensive education on heart disease risk factors, emphasising hypertension and the importance of reporting to medical facilities to have their blood pressure checked.

For the study period, the highest number of monthly cases of hypertension documented in Tamilnadu was 717, and the highest number of monthly instances of heart disease was 57. The average monthly number of hypertension patients was 132.96, whereas heart disease cases were 9.934. The months of March (16.9 instances) had the highest average number of monthly heart disease cases, while the months of November had the lowest average number of monthly heart disease cases. The months of January were related with the greatest average number of monthly hypertension cases during the study period. The months of October were connected with the fewest monthly average hypertension cases.

This means that as the number of cases of hypertension rises, the number of cases of heart disease rises as well, and vice versa, as the number of cases of hypertension falls, the number of cases of heart disease falls. It was also shown that heart disease is influenced by the immediate past movements of their own cases as well as the previous two hypertension cases. As a result, these lagged factors can be used to forecast future heart disease case numbers. Accordingly, a unit change in the lagged values of hypertension affects heart disease cases positively by about 41%, and two unit changes in the lagged values of hypertension affect cases positively by almost 3%.

According to our findings, hypertension cases can only be significantly explained by their first lag, and at this lag, a unit change in hypertension lagged values can explain around 61 percent of hypertension instances. At the conventional 5% level of significance, it was clear that lags associated with heart disease could not be used to explain hypertension, and this confirms the Granger causality test results, which showed that past heart disease values are not helpful in the prediction of hypertension in Tamilnadu State.

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