

The Impact COVID-19 Pandemic on Coronary Heart Disease Deaths: Using Bayesian Lorenz Curve and Gini-Index Distribution

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Abstract: Aim: The aim to investigate and assess role of COVID-19 on Coronary Heart Disease (CHD) mortality using Bayesian Lorenz Curve and associated Gini Index

Statistical Method: Bayesian estimation was applied to analyze CHD mortality rates, focusing on both gender and age group differences.

Application: A total of 341,467 patients were treated during 2-year period from 2020 to 2021 during COVID-19 in Turkey. 195,413 females and 146,054 males were diagnosed and 155,211 deaths where 88,824 were males and 66,387 were females with CHD, and hence were studied to evaluate whether female gender was an independent predictor for poor prognosis.

Results: Mortality rates increase with age for females compared to males. The model suggests that males have higher risks or proportions across all groups compared to females, particularly in older age categories. The Lorenz curves for both genders show that a significant portion of deaths is concentrated in a relatively small subset of age groups, particularly older adults. The Gini Index regarding mortality for males is found to be 0.123 compared to value of 0.384 associated with female's age distribution. Meanwhile, the Gini Index regarding morbidity for males (0.146) and females (0.394) are very similar, suggesting that the patterns of inequality in morbidity distribution are comparable across genders.

Conclusion: The study highlights the effectiveness of empirical Bayesian techniques in estimating CHD-related COVID-19 mortality rates across Turkey. It suggests that such statistical methods can help allocate resources more efficiently to high-risk areas and to ensure fair resource distribution and healthcare interventions.

Keywords: Bayesian, Markov Chain and Monte Carlo (MCMC), Morbidity, Mortality, COVID-19, Lorenz Curve, Gini index.

INTRODUCTION

COVID-19 has caused a global pandemic unprecedented in a century [1]. Though primarily a respiratory illness, cardiovascular risk factors predict adverse outcomes [2-5]. Coronary heart disease (CHD) is a pathological inflammatory process characterized by atherosclerotic accumulation in the epicardial arteries, and it is the most leading cause of mortality and morbidity worldwide [3-7]. It has been known for more than half a century that the risk factors for CHD include hypertension, increased levels of low-density lipoprotein cholesterol (LDL-C), type-2 diabetes and

smoking. CHD diseases have long been a significant public health concern, ranking among the leading causes of morbidity and mortality globally. More recently study [4]-supported study found that COVID-19 infection significantly increased the risk of heart attack, stroke, and death for up to three years among unvaccinated individuals infected during the early pandemic. The research, which included people with and without CHD, confirms previous findings of a higher risk of cardiovascular events after infection and is the first to suggest that this risk may persist for several years. With over 1 billion people worldwide having had COVID-19, the potential long-term impact on global heart health is substantial [1]. The increased risk of heart attacks, strokes, and other cardiovascular issues following infection underscores the need for ongoing research and preventive strategies.

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Previous reported studies on COVID-19 revealed its strong link to cardiovascular disease (CVD), particularly during the acute phase of infection. Among hospitalized COVID-19 patients, CVD (especially CHD) and related risk factors like hypertension and diabetes were prevalent in 10-30% of cases [10-13]. These conditions were associated with increased in-hospital mortality. Moreover, the presence of CVD and its risk factors correlated with increased in-hospital mortality among COVID-19 patients (Hillser *et al.* 2024). Additionally, acute CVD manifestations, including acute coronary syndrome, arrhythmias, heart failure, and thrombosis, were frequently observed in hospitalized patients.

Most recently the study [9] highlighted the potential long-term cardiovascular risks of COVID-19, emphasizing the need for CHD prevention strategies, especially for those with severe infections. It also suggests a possible genetic link, as patients with blood types A, B, or AB faced more than double the risk of heart attack or stroke after hospitalization, while those with type O appeared to have a lower risk of severe COVID-19. Further research is needed to confirm these findings and develop effective prevention strategies. Understanding the demographic disparities in mortality rates, particularly across genders and age groups, is essential for creating equitable and effective public health strategies [10-13]. Evidence has consistently shown that male patients experience higher mortality rates and more severe outcomes compared to females, with this disparity being most pronounced among older age groups [5,8,10]. Gender-based disparities in immune response, hormonal influences, and comorbidities have been posited as contributing factors to the observed male predominance in mortality [2-6]. Similarly, age-specific disparities are linked to the increasing vulnerability of older adults due to immune senescence and the presence of comorbid conditions [5,8, 10]. In Turkey, these patterns mirror the significant strain placed on healthcare systems during the pandemic, making it imperative to analyze gender and age-based differences in respiratory disease-related mortality.

This study employs a Bayesian estimation approach to model age-specific and gender-specific mortality rates for respiratory diseases during the pandemic. Bayesian methods are particularly suited for analyzing mortality rates due to their ability to incorporate prior knowledge and provide robust estimates even with complex data structures ([13-15]. Furthermore, Lorenz curves and Gini indices are utilized to quantify and visualize the inequality in mortality distribution across

age groups [14-18]. COVID-19 pandemic affects the CHD with various mechanisms [2-7].

The aim of this study is to investigate and assess role of COVID-19 on CHD mortality using Bayesian Lorenz Curve and associated Gini Index. In addition the Lorenz curve and Gini index implemented to measure gender as an independent predictor of mortality for the COVID-19 patients.

SUBJECT AND METHODS

This study proposes a Bayesian model based on the Beta distribution to estimate respiratory diseases-related mortality rates across different age groups during the COVID-19 pandemic. The methodology combines probabilistic modeling of age-group-specific mortality rates using the Beta-Binomial framework, followed by an analysis of inequality in mortality rates using the Lorenz curve and Gini index distribution.

Let N_{ij} and X_{ij} be total cases of COVID-19 among Turkish people and the number of deaths of COVID-19 respectively, where in the $(i,j)^{th}$ cell, i indicates sex factor being $i=1$ for male and $i=2$ for female, and j specifies age groups. Let us assume that X_{ij} is a binomial random variable with index N_{ij} and parameters λ_{ij} . Under the multiplicative model, $\lambda_{ij} = \theta_i \varphi_j (i=1,2; j=1,2,...,13)$. Where φ_j is a parameter represents proportion of number of cases in age group j , and θ_i is a parameter represent the proportion of death among the age group. Our objective is to obtain maximum likelihood estimates (MLE's) of the parameter θ_i , φ_j . Assuming the X_{ij} is independent, the log likelihood of X_{ij} is given by

$$= \sum_i \sum_j \ln (N_{ij} C_{X_{ij}}) + \sum_i \sum_j X_{ij} \ln \theta_i + \sum_i \sum_j X_{ij} \ln \varphi_j + \sum_i \sum_j (N_{ij} - X_{ij}) \ln (1 - \theta_i \varphi_j) \quad (1)$$

Which it readily follows that the MLE's should satisfy

$$\hat{\theta}_i = \frac{X_{i.}}{\sum_j \{(N_{ij} - X_{ij}) \varphi_j\} / (1 - \hat{\theta}_i \hat{\varphi}_j)} \quad (2)$$

$$\hat{\varphi}_j = \frac{X_{.j}}{\sum_i \{(N_{ij} - X_{ij}) \theta_i\} / (1 - \hat{\theta}_i \hat{\varphi}_j)} \quad (3)$$

$$\text{Where, } X_{i.} = \sum_j X_{ij}, X_{.j} = \sum_i X_{ij}.$$

Equations (2) and (3) can be solved iteratively. Initial values of the parameters $\hat{\theta}_i$ and $\hat{\varphi}_j$ may be set to

$$\hat{\theta}_i = \frac{X_{i.}}{N_{i.}}, \quad \hat{\varphi}_j = \frac{X_{.j}}{N_{.j}} \quad \text{where, } N_{i.} = \sum_j N_{ij}, \quad N_{.j} = \sum_i N_{ij}.$$

In order to further simplify the calculation Poisson approximation to the binomial set-up can be used. This is justified since population size N_{ij} for each $(i, j)^{th}$ cell is sufficiently large. Under the Poisson approximation, the X_{ij} 's are independent Poisson random variables with the parameter $N_{ij}\lambda_{ij}$, so that the likelihood of the sample is

$$= \sum_i \sum_j \left[-N_{ij}\theta_i\varphi_j + X_{ij} \ln N_{ij} + \ln \theta_i + \ln \varphi_j - \ln(X_{ij}) \right] \quad (4)$$

From (4), we obtain the MLE's of θ_i and φ_j as

$$\hat{\theta}_i = \frac{X_{i.}}{\sum_j N_{ij} \hat{\varphi}_j}, \quad \hat{\varphi}_j = \frac{X_{.j}}{\sum_i N_{ij} \hat{\theta}_i}$$

Which again have to be solved iteratively. Convenient initial values may be $\hat{\varphi}_j = X_{.j} / N_{.j}$

and $\hat{\theta}_i = X_{i.} / N_{i.}$

A **Bayesian model** based on the **Beta distribution** is often used to estimate probabilities. This makes it particularly useful in modeling scenarios where the parameter of interest is a proportion. In cases where the parameter of interest, θ , represents a probability, the **Beta distribution** is an appropriate prior due to its flexibility and conjugacy with the **Binomial likelihood** (13-16)

The number of deaths, y_i for each population group is modeled using a Binomial distribution:

$$y_i \sim \text{Binomial}(n_i, \theta_i),$$

where n_i is the total population of the i -th group and θ_i is the mortality rate.

The Binomial likelihood function is expressed as:

$$P(y_i | \theta_i, n_i) = \binom{n_i}{y_i} \theta_i^{y_i} (1 - \theta_i)^{n_i - y_i}$$

The prior distribution for θ_i , the probability of death due to respiratory diseases, is modeled using the Beta distribution: $\theta_i \sim \text{Beta}(\alpha_i, \beta_i)$ where

α_i : Prior successes, reflecting prior knowledge about deaths due to respiratory diseases.

β_i : Prior failures, reflecting prior knowledge about survivors in the population.

Using Bayes' theorem, the posterior distribution for θ_i is proportional to the product of the prior and the likelihood [17-18]:

$$P(\theta_i | y_i, n_i) = \text{Beta}(\alpha_i + y_i, \beta_i + n_i - y_i)$$

This posterior distribution reflects updated beliefs about age-specific mortality rates after observing the data.

Lorenz Curve and Gini Index

Lorenz curve is a widely used and practical method for modelling mortality rates of a disease [14-20]. The Lorenz curve is used to assess the inequality in respiratory disease-related mortality rates across age groups in this study. The cumulative proportion of deaths and the cumulative proportion of the population are calculated:

$$L(k) = \frac{\sum_{i=1}^k y_i}{\sum_{i=1}^I y_i}, \quad P(k) = \frac{\sum_{i=1}^k n_i}{\sum_{i=1}^I n_i}$$

where $L(k)$ is the cumulative proportion of deaths up to the k -th age group, and $P(k)$ is the cumulative proportion of the population [17-18].

The Gini index is derived from the Lorenz curve to quantify inequality in mortality rates:

$$G = 1 - 2 \int_0^1 L(P) dP$$

In practice, this is approximated using numerical integration or the trapezoidal rule based on discrete data points:

$$G = 1 - \sum_{k=1}^I (L_k + L_{k-1})(P_k + P_{k-1})$$

where L_k and P_k are the Lorenz curve values and population proportions at the k -th age group (Lee 2012).

- $G=0$ indicates perfect equality (all age groups have identical mortality rates),
- $G=1$ indicates maximum inequality.

Sathar and Jeevanand [19-20] have discussed the Bayesian estimation of the Lorenz curve and Gini-index of the Pareto and exponential distributions respectively.

Lorenz curve is a widely used and practical method for modelling mortality rates of a disease [13-15]. The Lorenz curve is used to visualize the inequality in COVID-19 deaths relative to incidence across different exposure levels (age groups). The exposure levels are ordered from the lowest to highest fatality risk. If COVID-19 risk is evenly distributed across all exposure levels, the Lorenz curve follows a straight diagonal line, indicating that age is not a significant factor. However, the more the Lorenz curve deviates from this diagonal, the greater the inequality in COVID-19 incidence across age groups.

This variation is quantified using the Gini Index [16-20], which ranges from 0 to 1. Higher values indicate greater disparity in COVID-19 incidence across age groups, while lower values suggest a more uniform distribution.

The variation depicted using a Lorenz curve can be summarized using the Gini index [16-20]. The value of the index is between 0 and 1, with larger values reflecting greater variability in CHD incidence over exposure levels (age groups) and smaller values reflect greater uniformity. One realization of the index is expressed mathematically as

$$Gini = \sum_{i=1}^k \left| \frac{x_{i-1}}{x_i} - \frac{y_{i-1}}{y_i} \right| = \sum (x_{i-1}y_i - x_iy_{i-1})$$

Where, (x_i, y_i) represent the coordinates of the points in the Lorenz curve, $x_i = \sum_{j \leq i} n_j / N$ and $y_i = \sum_{j \leq i} a_j / A$. n_i denotes the incidence in the i th (age group) exposure level ($i = 1, \dots, k$) and N denotes the total incidence ($N = \sum_i n_i$). A and a_i denote,

respectively, the total number of CHD deaths and number of deaths in the i th exposure level, $A = \sum_i a_i$.

Subjects and Statistical Analysis

The data obtained from University Teaching and Research Hospital, Ministry of Health State Hospitals and Turkish Statistical Institute. The patient's physicians collected data from the clinical records at the time of the patient's hospital discharge according to predefined criteria for each data point. These records have been coded and registered at the hospital. With the described database all patients presenting CHD with COVID-19 were identified. COVID-19 was defined for this study according to World Health Organization criteria, International Classification Diseases. Data included age, population size, number of cases and death, death rate due to coronary heart diseases and incidence of respiratory diseases. Electrocardiogram, echocardiography, cardiac biomarkers, and coronary angiography are essential diagnostic tools. Untangling the mechanisms of coronary involvement in COVID-19 ensures timely diagnosis and appropriate treatment.

The independent samples t-test was performed to test the significant differences between two means of a continuous variable. Bayesian model implemented using WinBUGS.

RESULTS

Table 1 shows the percentage of death due to CHD diseases and incidence of treated among male and female patients during 2020 and 2021 years. A total 465,375 males and 457,759 females were diagnosed

Table 1: Deaths, and Incidence Rates of Coronary Heart Diseases (CHD) among Turkish Males and Females with Respect to Age Groups

Age groups	2020 Death Rates		2021 Death Rates		2020 Incidence Rates		2021 Incidence Rates	
	Males	Females	Males	Females	Males	Females	Males	Females
0-14	232 (0.24)	208(0.24)	306(0.32)	255 (0.27)	580 (0.25)	520 (0.24)	840 (0.36)	688 (0.27)
15-24	223(0.22)	134(0.15)	233(0.24)	235 (0.24)	558(0.24)	335 (0.15)	233 (0.10)	634 (0.25)
25-34	467(0.25)	266(0.26)	570 (0.60)	333 (0.25)	1167(0.50)	665 (0.30)	582 (0.25)	899 (0.35)
35-44	1853 (1.94)	771(0.87)	286 (2.16)	912 (0.97)	4633 (1.99)	1928 (0.87)	715 (0.31)	2462 (0.97)
45-54	6198 (6.55)	2374(2.67)	6155 (6.43)	2403 (2.55)	15495 (6.66)	5935 (2.67)	15388 (6.61)	6488 (2.55)
55-64	14828 (15.67)	6315(7.11)	14800 (15.46)	6496 (6.89)	37070 (15.94)	15863 (7.13)	37000 (15.89)	17539 (6.88)
65-74	22947 (24.25)	14929(16.81)	23719 (24.78)	15948(16.93)	53368 (22.95)	37322(16.80)	59298 (25.47)	43059(16.90)
75-84	27422 (28.99)	29700(33.44)	27412 (28.64)	31192 (33.02)	68555 (29.48)	74250 (33.41)	68530 (29.43)	84218 (33.05)
≥85	20446 (21.61)	34156(38.45)	20444 (21.36)	36599 (38.36)	51115 (21.98)	85390 (37.52)	51110 (21.956)	98817 (38.78)
Total	94.616	88.826	95.703	94.930	232.541	222.208	232.831	235.551

Table 2: Summaries of the Posterior Distribution of the Coronary Heart Diseases Deaths Model

Nodes	Males						Females					
	Mean	SE	SD	Median	Lower 2.5	Upper 97.5	Mean	SE	SD	Median	Lower 2.5	Upper 97.5
α	0.807	0.282	0.005	0.363	0.774	1.446	0.704	0.251	0.005	0.316	0.672	1.296
β	4.585	2.111	0.036	1.327	4.294	9.417	4.243	2.025	0.040	1.151	3.945	9.041
θ_1	0.038	0.045	0.001	0.000	0.023	0.159	0.156	0.097	0.001	0.027	0.137	0.400
θ_2	0.120	0.062	0.001	0.030	0.110	0.271	0.148	0.091	0.001	0.026	0.130	0.375
θ_3	0.107	0.042	0.000	0.042	0.102	0.204	0.041	0.050	0.001	0.000	0.023	0.180
θ_4	0.075	0.023	0.000	0.037	0.073	0.126	0.062	0.038	0.000	0.011	0.054	0.156
θ_5	0.093	0.022	0.000	0.054	0.091	0.140	0.062	0.038	0.000	0.011	0.055	0.155
θ_6	0.072	0.015	0.000	0.046	0.071	0.105	0.109	0.034	0.000	0.053	0.106	0.184
θ_7	0.062	0.014	0.000	0.039	0.061	0.092	0.091	0.025	0.000	0.049	0.088	0.145
θ_8	0.102	0.016	0.000	0.073	0.101	0.135	0.108	0.026	0.000	0.064	0.106	0.164
θ_9	0.105	0.017	0.000	0.075	0.104	0.141	0.088	0.021	0.000	0.052	0.086	0.135
θ_{10}	0.129	0.020	0.000	0.093	0.128	0.170	0.086	0.019	0.000	0.053	0.085	0.129
θ_{11}	0.131	0.028	0.000	0.083	0.130	0.190	0.132	0.032	0.000	0.078	0.129	0.201
θ_{12}	0.098	0.030	0.000	0.048	0.095	0.165	0.063	0.024	0.000	0.024	0.060	0.118
θ_{13}	0.181	0.062	0.001	0.082	0.174	0.326	0.040	0.024	0.000	0.007	0.035	0.100

and treated, also, total 188,319 males and 183,756 females were died during 2 years. When compared to female patients, male patients were significantly older; the mean age of females was 54.80 ± 14.36 years, whereas that of the males was 57.50 ± 15.14 years ($p < 0.001$).

This Bayesian model estimates the respiratory diseases mortality rate (θ_i) for each age group (Table 2). We modelled the CHD deaths in the i -th age group. Age group was as 5-24, 25-34, 35-44, 45-54, 55-64, 65-74, 75-84, 85+ years. The mean age of females was 50.75 ± 14.97 years, whereas that of the males was 53.50 ± 15.91 years ($p < 0.001$).

y_i : observed number of CHD in the i -th age group n_i : Total population in the i -th age group

θ_i : The probability of a person in the i -th age group dying from COVID-19

The likelihood function is modeled as a Binomial distribution: $y_i \sim \text{Binomial}(n_i, \theta_i)$

For θ_i , we used a Beta distribution; $\theta_i \sim \text{Beta}(\alpha, \beta)$

The Beta distribution is appropriate for modelling probabilities (values between 0 and 1).

Informative priors ($\alpha=2$, $\beta=2$) were used as the starting point for θ_i . These values are constants and do

not have variability ($SE=0$, $SD=0$). A square root transformation was applied to the posterior statistic to stabilize variance and reduce skewness. This preprocessing step ensures more robust parameter estimation and mitigates the influence of outliers, particularly in age groups with disproportionately high or low mortality rates.

Figure 1 shows the Lorenz Curve of Death Rates for the years of 2020 and 2021, to examine the comparison between males and females across different age groups. The blue line represents the cumulative share of deaths for males, while the red line represents females. Both curves deviate from the line of equality, reflecting inequality in the distribution of deaths across age groups. It also deviates moderately from the line of perfect equality. The Gini indices for males (0.122) and females (0.384) are very similar, suggesting that the patterns of inequality in mortality distribution are comparable across genders.

Figure 2 is designed to present the Lorenz Curve of incidence rates for males and females over the years 2020 and 2021, for the examination of comparison between the cumulative share of incidences among different age groups. The Gini Index regarding morbidity for males (0.146) and females (0.394) are very similar, suggesting that the patterns of inequality in mortality distribution are comparable across genders.

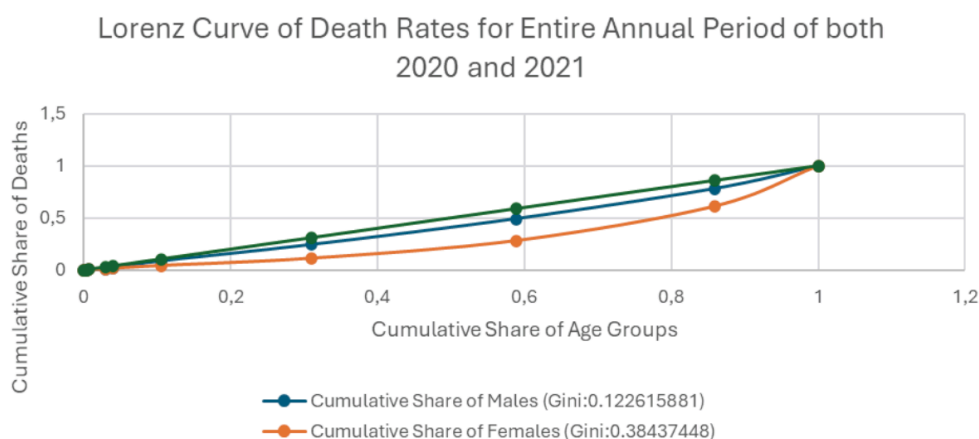


Figure 1: The Lorenz curve and associated Gini indices for Coronary Heart Disease (CHD) mortality among male and female patients.

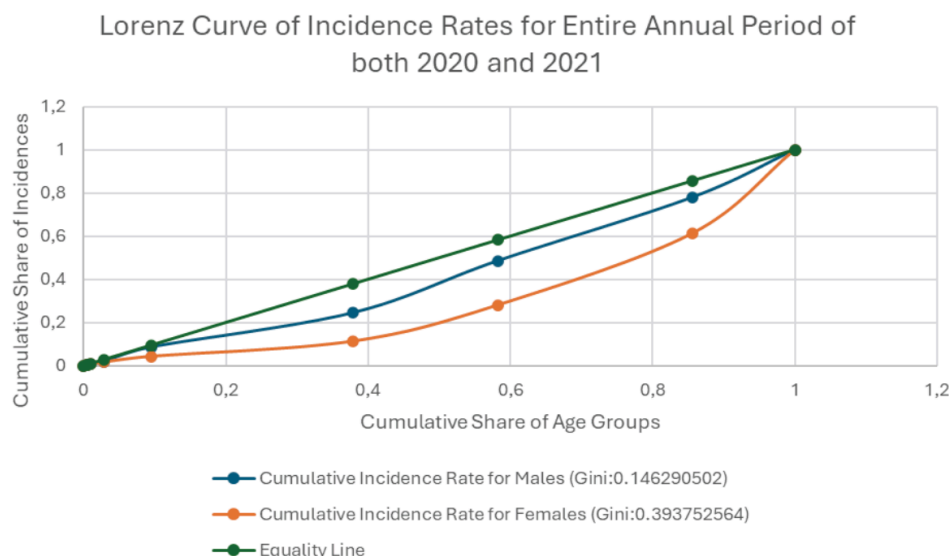


Figure 2: The Lorenz curve and associated Gini indices for Congestive Heart Disease (CHD) Incidence Rates among male and female patients.

Therefore, the incidence rates of female group are more concentrated in certain age groups, whereas the incidence rates of male group are more equally spread among different ages.

DISCUSSION

By employing Bayesian statistical methods, the study provides more flexible and robust modeling of mortality rates compared to traditional methods. This methodology ensures reliable estimates, especially in complex datasets with demographic variations. This approach can serve as a reference for future research on demographic disparities.

CHD issues are significant public health concerns influenced by occupation, employment, and economic stress [11-13]. Research highlights the link between economic hardship, unemployment, and mental health

decline [10-13]. Disaster mental health studies show that people often experience psychological distress after crises, leading to negative emotions like fear, loneliness, and despair, as well as symptoms such as anxiety and depression [11-13]. The COVID-19 pandemic has directly impacted mental health, altering personal behaviors and habits, with past epidemics also showing similar patterns of stress-induced behavioral changes [10-13].

The current study discusses (Figure 1) the distribution of heart disease mortality rates among males and females using the Gini coefficient and Lorenz curve. A higher Gini coefficient indicates greater inequality, meaning female mortality rates are more concentrated in specific age groups, while male deaths are more evenly spread. The Lorenz curve for females deviates more from the equality line, confirming this disparity. This suggests age-specific vulnerabilities for

females, whereas the more uniform distribution among males may indicate evenly spread risk factors across different ages. The current study results are consistent with the previous reported studies [2-8],

The current study revealed the distribution of CHD incidence rates among males and females using the Gini coefficient and Lorenz curve (Figure 2). The female incidence curve deviates more from the equality line, indicating a more uneven spread of cases across age groups, while the male curve remains closer, suggesting a more uniform distribution. Similar to mortality patterns, females have a higher Gini coefficient for both incidence and death rates. However, Gini values for incidence rates are higher, meaning disease cases are more unequally distributed than deaths. This disparity may stem from biological factors, healthcare access, behavior, or environmental and occupational risks. Empirical Bayesian models help adjust for natural variability in mortality rates while accounting for small population countries [15-20].

CONCLUSION

The study highlights the effectiveness of empirical Bayesian techniques in estimating CHD-related COVID-19 mortality rates across Turkey. It suggests that such statistical methods can help allocate resources more efficiently to high-risk areas. Researchers are encouraged to use Bayesian methods to identify high-risk groups and apply Lorenz insights to ensure fair resource distribution and healthcare interventions.

ACKNOWLEDGEMENTS

The authors would like to thank the Istanbul Medipol University for their support

ETHICAL APPROVAL (HELSINKI DECLARATION) AND CONSENT TO PARTICIPATE

The Ethics Committee Approval given by the Istanbul Medipol University, Institutional Review Board in accordance with the principles of the Helsinki Declaration of 1964 (IRB# 10840098-604.01.01-E.14180 and IRB# E-10840098-772.02-1411).

AVAILABILITY OF DATA AND MATERIALS

The datasets used and/or analyzed during the current study are available from the corresponding author on reasonable request.

COMPETING INTERESTS

No conflict of interest was declared by the authors.

FINANCIAL DISCLOSURE

The authors declared that this study has received no financial support

FUNDING

No funding was received for this work.

AUTHORS' CONTRIBUTIONS

AB, MHS, ZK and ZN contributed to conception, design, organized study, collected data, performed statistical analysis and wrote the first draft of the article, and contributed to the interpretation of the data and writing, revised critically and approved final version of manuscript. All authors approved the final.

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Received on 11-03-2025

Accepted on 08-04-2025

Published on 03-05-2025

<https://doi.org/10.6000/1929-6029.2025.14.26>© 2025 Bener *et al.*

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