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Risk Factors for Mortality by Novel Corona Virus Disease, in Mexico: A Cross-Sectional Study

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Abstract

The emergence of the Severe Acute Respiratory Syndrome Coronavirus 2 (SARS-CoV-2) infection epidemic in China alerted countries to the possibility of spread of the epidemic and Mexico was no exception. With a slow start, to date there were 120,102 confirmed cases.The objective was to analyze the effect of underlying pathologies in the presence of Coronovaris Infectious Disease -19 (COVID-19) on the possibility of dying in the Mexican population. Ancross-sectional study was conducted using the open database of the National Epidemiological Surveillance System (NESS) of the General Directorate of Epidemiology (GDE) to analyze the mortality of patients infected with SARS-CoV-2 and with disease due to COVID-19.Logistic regression models were generated for co-morbidities (diabetes, obstructive pulmonary disease, chronic, asthma, immunosuppression, hypertension, cardiovascular disease, chronic kidney disease, obesity, and smoking) and death, and differences in proportions were calculated between having the different co-morbidities. morbidities and die.The sample was 120,102 records, of which 14,053 (11.7%) had died and there were statistically significant differences (P <.05) between having co-morbidities and dying. All co-morbidities had ORs greater than 1, except asthma.Age and gender showed a confounding effect. Having a comorbidity and having COVID-19, increases the possibility of dying in the Mexican population.

Keywords: *Coronavirus; diabetes; hypertension; mortality*

1. INTRODUCTION

In December 2019, cases of unknown cause pneumonia are reported in Wuhan, Hubei Province, China [1]. As of June 8, 2020, the World Health Organization (WHO) reports almost 7 million infected and just over 400,000 deaths in 216 affected countries worldwide [2].

The causative virus, when identified, was called 2019- novel Coronavirus and later, the WHO called it Severe Acute Respiratory Syndrome Coronavirus 2 (SARS-CoV-2) [3].

Coronaviruses belong to the *Coronaviridae* family in the *Nidovirales* order. Corona represents crown-like spikes on the outer surface of the virus; thus, it was named as a coronavirus. Coronaviruses are small (65–125 nm in diameter) and contain a single-stranded RNA as a nucleic material, size ranging from 26 to 32 kbs in length [4].

The zoonotic origin of SARS-CoV-2 has not been confirmed, however sequential analysis suggests bats as their reservoir; DNA recombination was found to be involved in the spine glycoprotein which classifies SARS-CoV, thus being the reason for cross-transmission between species and rapid infection. According to phylogenetic branches, SAR-CoV-2 is close to SARS.CoV of bats [4].

The virus was identified and characterized by Zhu et al, and also, confirmed that SARS-CoV-2, uses the same cell entry receptor, Angiotensin-Converting Enzyme 2 (ACE2), as SARS-CoV, which is highly expressed in airway epithelial cells [5].

It has been reported that the virus remains until the second week after the attack in mild cases and in severe cases it remains in the body for up to three weeks. The permanence of the virus was longer in men and in those over 60 years of age; in feces from 17 to 31 days, in respiratory tract from 13 to 29 days in plasma from 11 to 21 days [6].

According to Phan [7], he reviewed the SARS-CoV-2 genome, recovered from patients from China, the USA, Australia, Japan, France, Singapore, England, Taiwan, South Korea, Belgium, Germany and Vietnam and its alignment sequence of nucleotides revealed 93 mutations, eight of which corresponded to the surface spine glycoprotein and this is the target of neutralizing antibodies [8] SARS-CoV-2 enters mucous membranes mainly nose and larynx and up to the lungs ; the initial symptoms are fever and cough [9], it passes into the circulation, causing viremia; it attacks target organs that express ACE2, such as the lungs, kidney, heart, and gastrointestinal tract [10,11]. It is speculated that B lymphocytes could decrease by affecting the production of antibodies [12], and the inflammation factors, mainly Interleukin-6, increase contributing to the worsening of the disease, 7 - 14 days after the attack [13] and the phase Clinic is divided into three stages: viremia, acute and recovery phases; if the patient is older or has immunocompromise and is accompanied by other diseases such as diabetes, hypertension, the immune system does not control the virus in the acute phase and becomes a critical patient [13].

Therefore, the objective was to analyze the evolution of the spread of COVID-19, as well as its mortality and comorbidities and its effect on death, in the Mexican population until June 8, 2020.

2. METHODS

A cross-sectional study was designed with the data included in the NESS/GDE database published on June 8, 2020 [14].

The study was approved by the Ethics Committee of the Celaya-Salvatierra Campus of the University of Guanajuato, with an expedited review and not requiring informed consent because only the database was worked and no personal data was collected, with the registry CBCCS- 05130042020.

The data included in the database are: age, gender, date of onset of symptoms, date of death if it occurred, presence of diabetes, COPD, asthma, immunosuppression, hypertension, cardiovascular disease, chronic kidney disease, obesity, smoking, Real Time-Polymerase Chain Reaction (RT-PCR) result for SARS-CoV-2.

In Mexico, a suspicious case was considered to be one with cough, fever, dyspnea, contact with a case and/or having traveled to a country affected by the pandemic.

A confirmed case was one that was a suspicious case and that the RT-PCR test was positive for SARS-CoV-2.

Confirmed cases in which the presence of comorbidities was unknown were eliminated from the analysis for underlying pathologies, but not from the initial analysis.

Descriptive statistics were used for all variables. Z was used for two proportions and a P value to check sex differences between the deceased and the non-deceased, and Student's Ttest and P value for two means to check differences in average age.

To analyze the co-morbidities with COVID-19 mortality, Z was used for two proportions and a P value.

Logistic regression models were generated between each co-morbidity and mortality from COVID-19. These models were adjusted for age and gender, and the Likelihood Odds Ratio Test (LRT) was used to compare the models.

In all cases the value of $\alpha = 0.05$. Statistical analysis was performed in STATA 13.0 ® (Stata Corp. College Station, TX, USA)

3. RESULTS

Table 1. *Distribution by sociodemographic characteristics by deaths of confirmed cases of COVID-19, Mexico (n=120,102)*

Variable	Deaths (n=14,053) n %	Non-deaths (n=196,049) n %		
Gender Male Female	 9,324 66.35 4,729 33.65	 57,801 54.50 48,248 45,50	 Z= 26.59	 *P* = .0000 1
Age (years) Range Mean ± S	 0 to 103 60.36 ± 14.23	 0 to 120 44.13 ± 15.37	 t=118.65 df 120,460	 *P* = .0000 1

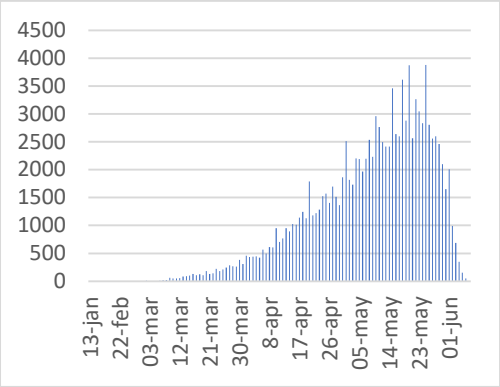
S standard deviation

Source: NESS/GDE [14]

The public database of confirmed cases of COVID-19 in Mexico published on June 8, 2020 [14], includes 120,102 people, of which 14,053 had died, with a specific mortality rate of 11.70%.

Table 1 shows the distribution of confirmed cases of COVID-19, by gender, age, and death. In both the deceased and the non-deceased, men predominated and the differences are significant (P <.05): The average age was higher among the deceased and the difference in the average age is significant (P <.05).

Figure 1 shows the curve of confirmed cases per day; the highest number of cases is between May 14 and 24, 2020; the date corresponds to the start of clinical data and the decrease in cases in late May and early June, could be an artifact due to the delay in the delivery of RT-PCR results and their report to NESS/GDE.



Source: NESS/GDE [14]

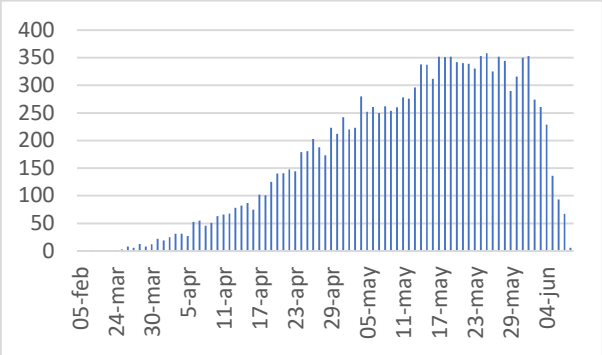
Figure 1. *Confirmed cases of COVID-19 per day (n=120,102)*

Figure 2 shows the distribution of COVID-19 cases that died, and the highest number of deaths have been from May 14 to June 1, 2020. The decrease in deaths after June 1, could be an artifact due to the delay of notifications to NESS/GDE.

With the data from the public database of the NESS/GDE from June 8, 2020 [14], they were divided into two groups; one for the deceased and the rest for the undead, but all confirmed as cases of COVID-19.

For analysis of the effect of co-morbidities on COVID-19 mortality, records were removed from the database where it was unknown if the patients had each condition.

Table 2 shows the cases eliminated by illness and by deceased and non-deceased. The proportion of records deleted in almost all cases were less than 1%.



Source: NESS/GDE [14]

Figure 2. *Deaths attributed to COVID-19 per day (n = 14,953)*

Table 2. *Distribution of registries eliminated by death and by co-morbidities (n = 120,102)*

	Deaths (n %0	**Non-deaths (n %)**
Diabetes	112 0.80	498 0.47
COPD	116 0.83	451 0.43
Asthma	113 0.08	461 0.04
Immunosuppression	121 0.86	506 0.48
Hypertension	107 0.76	482 0.45
Cardiovascular disease	128 0.09	474 0.04
Chronic kidney disease	117 0.08	474 0.04
Obesity	154 1.10	481 0.45
Smoking	120 0.85	509 0.48

Source: NESS/GDE [14]

Table 3 shows the distribution of co-morbidities and deaths, as well as z for two proportions, finding that for all co-morbidities, significant differences were found (P <.05) between deceased and non-deceased, showing the importance of these co-morbidities on COVID-19 mortality.

Table 3. *Distribution of deaths by co-morbidities in confirmed cases of COVID-19 in Mexico (n=120,102)*

Co-morbidities	**Deaths n %**	**Non-deaths n %**	**Z**	**P-value**
Diabetes Yes No	5,244 37.62 8,697 62.38	15,087 14.29 90,464 85.71	68.93	.00001
COPD Yes	739 5.30	1,540	31.1	.0000

No	13,198 94.70	1.46 104,058 98.54	5	1
Asthma Yes No	309 2.22 13,631 97.78	3,108 2.94 102,480 97.06	-4.80	.00001
Immunosuppression Yes No	418 3.00 13,514 97.00	1,395 1.32 104,148 98.68	15.25	.00001
Hypertension Yes No	5,917 42.43 8,029 57.57	18,635 17.65 86,932 82.35	68.08	.00001
Cardiovascular disease Yes No	791 5.68 13,134 94.32	2,215 2.10 103,360	25.35	.00001
Chronic kidney disease Yes No	978 7.02 12,958 92.98	1,755 1.66 103,820 98.34	39.80	.00001
Obesity Yes No	3,650 26.26 10,249 73.74	20,658 19.57 84,910 80.43	18.42	.00001
Smoking Yes No	1,297 9.31 12,636 90.69	8,504 8.06 97,036 91.04	5.05	.00001

Source: NESS/GDE [14]

Table 4. *Crude logistic regression models adjusted for age and gender between co-morbidities and deaths from COVID-19 in Mexico.*

	OR (CI95%)	**OR (CI95%) adjusted by age**	**OR (CI95%) adjusted by gender**
Diabetes	3.62 (3.45 - 3.76)	1.97 (1.89 – 2.05)*	3.63 (3.50 – 3.78)*
COPD	3.78 (3.46 – 4.14)	1.37 (1.24 – 1.51)*	3.87 (3.54 – 4.24)*
Asthma	0.7h5 (0.66 – 0.84)	0.92 (0.81 – 1.04)*	0.80 (0.71 – 0.90)*
Immunosuppression	2.31 (2.07 – 2.58)	1.80 (1.60 – 2.04)*	2.39 (2.14 – 2.67)*
Hypertension	3.44 (3.31 – 3.57)	1.60 (1.53 – 1.66)*	3.49 (3.36 – 3.62)*
Heart disease	2.81 (2.59 – 3.05)	1.29 (1.18 – 1.42)*	2.81 (2.58 – 3.05)*
Chronic kidney disease	4.46 (4.12 – 4.84)	3.00 (2.75 – 3.28)*	4.47 (4.12 – 4.84)*
Obesity	1.46 (1.41 – 1.52)	1.51 (1.45 – 1.58)*	1.49 (1.43 – 1.56)*
Smoking	1.17 (1.10 – 1.25)	1.18 (1.11 – 1.26)*	1.02 (1.02 - 1.03)*

** Likelihood Ratio Test P< .05 OR Odds Ratio*

Source: NESS/GDE [14]

Logistic regression models show a significant effect of diabetes, COPD, immunosuppression, hypertension, cardiovascular disease, chronic kidney disease, and obesity with ORs greater than 1. In all comorbidities, age and gender acted as confounders with P <.05, comparing the models. Separate mention is the asthma that seemed to have a preventive effect of dying, but disappears when adjusting for age (Table 4).

4. Discussion

The sample of 120,102 confirmed to have COVID-19 in the Mexican population was dominated by men, both in those who died (66.35%) and in those who did not die (54.50%). The average age was higher among the deceased (60.36 ± 14.23 years) than in the non-deceased (4.13 ± 15.37 years) (*P* <.05) (Table 1). This confirms that the age of 60 or more years becomes a risk factor for dying from COVID-19 [15], also confirmed by Zhou et al [16], because reported 191 infected by COVID, had increase risk to death with higher age.

In Mexico, the specific mortality rate was 11.70%. In Italy, as of June 3, 2020, the specific mortality rate per case 13.8% and for those over 60 years of age was much higher than the rest of the ages of those infected with SARS-CoV-2 [15]. European countries show an increase in case-specific mortality for all countries, with Italy and the Netherlands having the highest rates [17].

In New York, Petrilli, et al [18], reported as risk factors for a more severe SARS-CoV-2 infection that requires hospitalization were being male, age over 44 years, heart failure, chronic kidney disease and BMI> 40 kg/m2 and for a more severe infection it was heart failure, chronic kidney disease and obesity. Older age, hypertension can develop severe COVID-19 and patients male, with heart disease and hyperglycemia have more risk to death [19],

In Mexican population, asthma does have preventive effect on death by COVID-19 (Table3). The same result was reported by Padilla et al, in Mexican population with data from May 2020, on asthma and death by COVID-19 [20]. Although, according to the United States Center for Disease Control and Prevention, risk factors are age greater than 65 years, chronic lung disease, immune suppression from any source, serious heart disease, severe obesity, chronic kidney disease, and severe asthma [21].

5. Conclusion

The spread of COVID-19 continues to manifest itself in Mexico, with the increase of new cases every day. The role of co-morbidities and age on mortality from COVID-19 is verified. An important fact is the protective effect of asthma on COVID-19 mortality in this sample of the Mexican population.

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