



A Glance at Coronavirus in General Population of Thi-Qar and Al- Muthanna Provinces

ARTICLE INFO

Article Type

Original Research

Authors

Ahmed Saeed H.¹ MSc,
Mohammed Ali Jassim M.¹ PhD,
Mahmood M.M.^{*2} PhD,
Saad Hanfoosh H.¹ MSc

How to cite this article

Ahmed Saeed H, Mohammed Ali Jassim M, Mahmood M M, Saad Hanfoosh H. A Glance at Coronavirus in General Population of Thi-Qar and Al- Muthanna Provinces. Health Education and Health Promotion. 2022;10(3):587-591.

ABSTRACT

Aims This research aimed to investigate the spread of COVID-19 infection among suspected adults and assess the relationship between tobacco smoking, diabetes, and high blood pressure comorbidities, and their association with susceptibility to acquiring the infection with COVID-19.

Material & Methods This study was carried put on 214 suspected cases of CoV-2, among male and female adults (age ≥ 20 years) in 2021, and tested for CoV-2 infection by RT-PCR. Plus the IgG/IgM Rapid Test Cassette, along with CoV-2-related symptoms. Information on whether the subjects smoke or suffer from diabetes and high blood pressure has also been supplemented.

Findings According to the results, the age group of 61-80 was the most affected group among the COVID-19 patients by 45%. In addition, the proportion of males infected with COVID-19 was higher across all age groups (64%) than females (36%). A significant difference was observed between the patients concerning age and gender ($p < 0.05$). The percentage of smoker patients versus non-smokers was 47% vs 53%. The proportion of diabetics to nondiabetics was 43 % vs 57 %. As well as the ratio of hypertensive to normotensive COVID-19 patients was 62 % vs 38 %. There was a significant difference between smoker and non-smoker, diabetic and non-diabetic, and hypertensive and non-hypertensive patients in terms of affected by COVID-19 ($p < 0.05$).

Conclusion Age, gender, smoking, diabetes, and hypertension are some risk factors for Coronavirus infection. Gender and age are associated with infection rates and their consequences. Also, the likelihood of COVID-19 infection is likely to be lowered in half for active smokers. Patients with diabetes and hypertension especially those who use ACE2-increasing medications are at a higher risk of developing a severe COVID-19 infection.

Keywords SARS-CoV-2; Diabetes Mellitus; Hypertension; Smoking

¹College of Dentistry, Al-Muthanna University, Al-Muthanna, Iraq

²Department of Biology, College of Science, Mustansiriyah University, Baghdad, Iraq

*Correspondence

Address: Department of Biology, College of Science, Mustansiriyah University, Baghdad, Iraq. Postal Code: 10011

Phone: +96 (47) 703904461

Fax: -

majidmahmood93@yahoo.com

Article History

Received: May 31, 2022

Accepted: August 14, 2022

ePublished: : August 22, 2022

CITATION LINKS

[1] Current smoking and COVID-19 risk: results ... [2] Inhibition of viral infection by using ... [3] Clinical characteristics of coronavirus ... [4] Active smoking is not associated with severity ... [5] Tracing of some salivary immune ... [6] The impact of the COVID-19 epidemic on the ... [7] Role of angiotensin-converting ... [8] Are patients with hypertension and diabetes ... [9] Comorbidities in SARS-CoV-2 patients ... [10] Association between blood pressure control ... [11] The influence of gender on COVID-19 ... [12] Case characteristics, resource use ... [13] Sex-specific SARS-CoV-2 mortality: among ... [14] Higher severity and mortality in male patients ... [15] Impact of sex and gender on ... [16] ACE2 and TMPRSS2 variants and expression ... [17] The lethal sex gap ... [18] A deep convolutional neural network based ... [19] Smoking and COVID-19: a scoping ... [20] Smoking and the risk of COVID-19 ... [21] Smoking prevalence and COVID-19 ... [22] Is there a smoker's paradox ... [23] The effect of smoking on COVID-19 ... [24] Co-morbidity and blood group type risk ... [25] Interplay between EBERS and P27 tumor ... [26] COVID-19 and hypertension: risks and ... [27] COVID-19: angiotensin-converting enzyme 2 ... [28] ACE2 protein expression in lung tissues of ... [29] Circulating ACE2 activity predicts mortality and disease severity ...

Introduction

Accurate assessments of the spread of COVID-19 in the population are required to assess and improve disease prevention strategies and testing techniques, calculate seasonal effects, predict future disease spread, assess mobility and travel risks, and identify vaccine needs [1]. The SARS-CoV-2 caused the COVID-19 pandemic is expanding worldwide, making prognostic indications a global challenge. Some people may acquire symptomatic COVID-19 infections, which can lead to progressive lung involvement, respiratory failure, and systemic consequences. People who have long-term medical conditions such as lung cancer, and asthma, are more likely to suffer major risks (COPD) [2, 3].

To date, the importance of smoking among epidemiological risk factors is unknown. Even though smoking has been related to a deterioration of COVID-19 disease prognosis, this result is concerning [4, 5]. Diabetic people have a higher risk of viral infection. While the early study focused on persons with type 2 diabetes, new studies have revealed that patients with type 1 diabetes are also at high risk of COVID-19 infection. Indicating the syndromic character of the disease, the rationale for diabetics' poor prognosis is unquestionably multifaceted. Age, gender, race, and comorbidities, including hypertension, cardiovascular disease, and pro-coagulant conditions contribute to negative outcomes. Finally, extreme acute respiratory syndrome coronavirus 2 infections can worsen the cause for people with diabetes since it has immediate detrimental impacts on -cell activity and may precipitate acute metabolic complications. Diabetic patients can develop diabetic ketoacidosis as a result of these changes in -cell structure [6, 7].

SARS-CoV-2 attaches ACE2, which is expressed by the intestine, kidney, and blood vessel epithelial cells. Angiotensin-converting enzyme inhibitors and angiotensin II type-I receptor antagonists have been associated with considerably greater ACE2 expression in individuals with type 1 or type 2 diabetes [8]. Consequently, the use of ACE2-stimulating medications to treat diabetes and hypertension has been linked to an increased risk of severe COVID-19. Aging, diabetes, and other comorbidities have also been linked to increased morbidity and mortality. Possible underlying mechanisms of the diabetes-COVID-19 association include chronic inflammation, increased coagulation function, and compromised immunological function by SARS-CoV-2 [9].

Elderly COVID-19 patients with asthma, cardiovascular disease, diabetes mellitus, and chronic respiratory illness had a greater death rate. The renin-angiotensin-aldosterone RAS may help coronaviruses like SARSCoV2 enter target cells, especially in the lungs [10]. As a result, it's been suggested that angiotensin receptor blockers ARBs

and angiotensin transforming enzyme inhibitors ACE inhibitors, both of which cause ACE2 expression, may affect SARSCoV2 infection susceptibility and intensity. Patients with inadequate blood pressure management had poorer COVID-19 results, most likely as a result of advanced atherosclerosis and organ damage. In this case, certain patients may need to maintain a higher level of social isolation to reduce COVID-19's impact [9, 10]. So, more knowledge about the new pandemic of COVID-19 is required to make applicable strategies for the prevention of disease spread out and death by this virus. In this regard, this research aimed to investigate the spread of COVID-19 infection among suspected adults and assess the relationship between tobacco smoking, diabetes, and high blood pressure comorbidities, and their association with susceptibility to acquiring the infection with COVID-19.

Material and Methods

This study was carried out in the Iraqi governorates of Thi-Qar and Al-Muthanna on 214 suspected cases of CoV-2 among adults of both genders (age \geq 20 years) from early 2021 until April 2021, who tested for CoV-2 infection by RT-PCR. Plus the IgG/IgM Rapid Test Cassette, along with CoV-2-related symptoms. Out of the subjects, 100 individuals were confirmed to be infected, whose ages ranged were 20-80 years with an average of 31.54-62.59.

The research tool was a researcher-made questionnaire to investigate chronic conditions such as diabetes, hypertension, and smoking among the subjects. Nasopharyngeal swabs were collected and analyzed using oligonucleotide primers and probes to identify COVID-19 patients with positive SARS-CoV-2 nucleic acid. The entire laboratory guidelines for COVID-19 identification and diagnosis were created following the manufacturer's specifications (bioPerfectus technologies). The OnSite COVID-19 IgG/IgM rapid test is a single-use lateral flow immunoassay designed to detect and differentiate anti-SARS-CoV-2 IgG and IgM antibodies in human blood. The OnSite COVID-19 IgG/IgM Rapid Test is designed to help distinguish people who have developed an adaptive immune response to SARS-CoV-2, signaling recent or previous infection. The entire diagnosis and identification procedure was carried out following the production company's specifications (CTK Biotech).

Data were analyzed using Pearson Chi-square tests through SPSS 23 software and the significance level was determined to be less than 0.05 ($p < 0.05$).

Findings

The average age of the subjects was 31.54-62.59. The age group of 20-40 had the maximum frequency, and the least frequent age group was the age group of 20-40 (Table 1).

Table 1) Distribution of COVID-19 patients according to their age ranges

Age (Year)	N	Mean±SD	Range		p.
			Min	Max	
20-40	23	31.54±9.93	20	40	0.03
41-60	32	47.13±11.36	41	60	
61-80	45	62.59±7.19	61	80	

The results showed that the frequency of male patients with "COVID-19" was higher in all age groups (64%) than in females (36%). In addition, the male-to-female ratio in the age ranges of 20-40, 41-60, and 61-80 were as follows 2.29, 1.91, and 1.5, respectively. There was a significant difference between the subjects in terms of age groups ($p < 0.05$; Table 2).

Table 2) Distribution of the subjects according to their gender

Gender		COVID-19 patients			p.
		20-40	41-60	61-80	
Male	N	16	21	27	0.02
	%	69.57	65.63	60	
Female	N	7	11	18	
	%	30.43	34.37	40	
Total	N	23	32	45	
	%	100	100	100	
Male/female ratio		2.29	1.91	1.5	

SARS-CoV-2 patients were classified based on smoking and comorbidities such as diabetes and hypertension (Table 3). There were 43% of diabetics vs. non-diabetics subjects (57%). The hypertensive and non-hypertensive people were 62% and 38%, respectively. There were significant differences between smokers and non-smokers, diabetic and non-diabetic, and hypertensive and non-hypertensive patients with COVID-19 infection ($p < 0.05$; Table 3).

Table 3) Classification of the subjects according to comorbidities

Individuals		Number	%Ratio	p-value
Smoker	Smoking	47	1.13	0.05
	Non-smoking	53		
Diabetes Mellitus	Normal	43	1.33	0.043
	Patients	57		
Hypertension	Normal	62	16.63	0.01
	Patients	38		

Discussion

This study aimed to investigate the spread of COVID-19 infection among suspected adults and assess the relationship between tobacco smoking, diabetes, and hypertension comorbidities, and their association with susceptibility to acquiring the infection with COVID-19 in Thi-Qar and Al-Muthanna, Iraq.

Age and gender-disaggregated data are needed for a detailed investigation of the COVID-19 epidemic. To study age and gender infection disparities, we investigated the proportions of men and women infected with SARS-Cov-2 by demographic characteristics. This study supports the notion that gender and age play a significant role in COVID-19 infection. Based on the results, males in the age

range of +60 were more contaminated with COVID-19 than women. The importance of age on COVID-19 contraction has been recognized in several studies [11-12]. With aging, prolonged activation of innate and adaptive immune mechanisms raises, resulting in a decrease in immune system activity and increased susceptibility to pathogens and chronic diseases [13]. Generally, male-to-female ratios on COVID-19 vary by age group due to a different range of internal biological gender and age physiology and environmental factors sociality, habits, job, and lifestyle. The results are following the findings of Jin *et al.* [14], Gebhard *et al.* [15], Asselta *et al.* [16], and Márquez *et al.* [17]. This difference was most likely caused by men's proclivity for congregating outside. Based on the studies, females have a greater immunological response to viral infection than males due to higher natural and adaptive immune systems. The immune system is controlled by sex hormones; estrogens stimulate both innate and adaptive immune responses, resulting in a better and faster response to pathogens [14, 15]. Androgens weaken the immune system, which explains why men are more prone to infectious diseases. In women, X-chromosome inactivation generates an imbalance of immune response genes such as CD40L, TLR7, and ACE2, which protect against illnesses such as hypertension, heart disease, and acute respiratory distress syndrome. Having these diseases at the same time is the main risk factor for a worse outcome in COVID-19 [16, 18]. According to the findings of Marquez, young people have higher ACE2 expression than older people, and women have higher ACE2 expression than men [17]. The presence of overexpressed X-linked genes may become more significant as people age. Furthermore, vitamin D deficiency in older men is not properly evaluated, whereas vitamin D deficiency in women in their forties and fifties is often assumed to aid the menopausal process and avoid osteoporosis due to a shortage of estrogen. Vitamin D deficiency may decrease inflammation and ARIA. Many experts believe men in their 60s may be more susceptible to viral infections due to lower vitamin D levels than women their age [19].

The results of this study showed a significant difference between smoker and non-smoker patients in terms of being infected by the Coronavirus. Cigarette smoking's influence on COVID-19 susceptibility is currently being debated. Some studies indicated that current smokers are underrepresented among COVID-19 patients, suggesting a smoker's paradox, and are protected from significant COVID-19 consequences [20]. Smokers who have several comorbidities are more prone to the COVID-19 virus and have a bad prognosis for both the virus and their comorbidities [21].

The negative association between the prevalence of confirmed COVID-19 cases and the level of smoking

in the adult population, as reported by Usman [22], and Reddy [23], may explain the novel coronavirus's unusual infection mechanisms. According to the findings of Badedi *et al.* [24], aside from the potential role of squamous cell carcinoma, there are several biological reasons why smoking may protect against COVID-19. Nicotine has an anti-inflammatory effect, smokers have a lower immune response which lessens the probability of a cytokine storm in COVID-19, and greater nitric oxide in the respiratory tract may inhibit SARS-CoV-2 from entering cells [24]. Reddy *et al.*, [23] showed that smoking was linked to a significantly higher risk of COVID-19 patients experiencing symptoms, as well as a higher symptom burden, suggesting that smoking affects disease severity. The ACE2 is elevated in the lung in association with smoking, which increases the risk of SARS-CoV-2 infection. However, a recent meta-analysis revealed inconsistent findings of overexpression in epithelial cells and downregulation in alveolar type 2 cells [25].

The results of our study showed a higher risk of being infected by the Coronavirus in diabetic patients. The mechanism behind this difference in effect between diabetics and non-diabetics is unknown. As previously reported, DM raises COVID-19 complications and the risk of COVID-19-related mortality [26]. Diabetes patients are more likely than people without diabetes to experience severe COVID-19 symptoms and complications because hyperglycemia makes it easier for the virus to enter cells since both ACE2 and the virus require glucose to survive [27].

The results of this research showed a significant difference in infection by Corona virus between hypertensive and no hypertensive patients. However, a variety of factors may theoretically confound any potential connection between hypertension and extreme COVID-19. The first factor is age; high COVID-19 and hypertension are common among the elderly. Instead of hypertension, underlying arterial endothelial dysfunction and/or organ damage enhance the chance of severe COVID-19 [28]. Furthermore, Because SARSCoV2 utilizes ACE2 to enter and infect cells, high-ACE2 cells were thought to be more susceptible to infection. ARB and ACE inhibitors enhance ACE2 on cell membranes. Anxiety surrounds the use of "RAS inhibitors" because SARSCoV2 needs ACE2 receptors to enter cells [29].

Conclusion

The results of this research showed that Gender and age are associated with infection rates and their consequences. Also, the likelihood of COVID-19 infection is likely to be lowered in half for active smokers. Patients with diabetes or hypertension especially those who use ACE2-increasing medications are at a higher risk of developing a

severe COVID-19 infection, and they should be assessed for ACE2-modulating medications including ACE inhibitors and ARBs. Because of the vast variety of potential risks, physicians may take a systematic approach to patient care, and biomarkers may offer valuable prognostic details. Overall, multidisciplinary COVID-19 management will help patients, especially those with risk factors like hypertension, achieve the best possible results.

Acknowledgments: None declared.

Ethical Permissions: None declared.

Conflicts of Interests: None declared.

Authors' Contributions: Ahmed Saeed H (First Author), Introduction Writer/Main Researcher/Discussion Writer (25%); Mohammed Ali Jassim M (Second Author), Methodologist/Statistical Analyst (25%); Mahmood MM (Third Author), Methodologist/Main Researcher (25%); Saad Hanfoosh H (Third Author), Assistant Researcher (25%)

Funding/Support: None declared.

References

- 1- Hopkinson NS, Rossi N, El-Sayed Moustafa J, Laverty AA, Quint JK, Freidin M, et al. Current smoking and COVID-19 risk: results from a population symptom app in over 2.4 million people. *Thorax*. 2020;76(7):714-22.
- 2- Al-garawyi AMA, Hussein TA, Jassim MMA. Inhibition of viral infection by using of natural herbal remedies as alternative treatment. *Syst Rev Pharm*. 2020;6(11):416-49.
- 3- Guan WJ, Ni ZY, Hu Y, Liang WH, Ou CQ, He JX, et al. Clinical characteristics of coronavirus disease 2019 in China. *N Engl J Med*. 2020;382:1708-20.
- 4- Lippi G, Henry BM. Active smoking is not associated with severity of coronavirus disease 2019 (COVID-19). *Eur J Intern Med*. 2020;75:107-8.
- 5- Apicella M, Campopiano MC, Mantuano M, Mazoni L, Coppelli A, Del Prato S. COVID-19 in people with diabetes: understanding the reasons for worse outcomes. *Lancet Diabetes Endocrinol*. 2020;8(9):782-92.
- 6- Qaysar Musa S, Mohammed Alijassim M, Mohammed Mahmood M. Tracing of some salivary immune elements in Iraqi SARS-2 patients. *Arch Razi Inst*. 2022;77(5):1561-5.
- 7- Ni W, Yang X, Yang D, Bao J, Li R, Xiao Y, et al. Role of angiotensin-converting enzyme 2 (ACE2) in COVID-19. *Crit Care*. 2020;24:422.
- 8- Fang L, Karakiulakis G, Roth M. Are patients with hypertension and diabetes mellitus at increased risk for COVID-19 infection?. *Lancet Respir Med*. 2022;8(4):e21.
- 9- Ng WH, Tipih T, Makoah NA, Vermeulen JG, Goedhals D, Sempa JB, et al. Comorbidities in SARS-CoV-2 patients: a systematic review and meta-analysis. *mBio*. 2021;12(1):e03647-20.
- 10- Sheppard JP, Nicholson BD, Lee J, McGagh D, Sherlock J, Koshiaris C, et al. Association between blood pressure control and coronavirus disease 2019 outcomes in 45 418 symptomatic patients with hypertension: an observational cohort study. *Hypertension*. 2021;77(3):846-55.
- 11- Doerre A, Doblhammer G. The influence of gender on COVID-19 infections and mortality in Germany: Insights from age- and gender-specific modeling of contact rates,

infections, and deaths in the early phase of the pandemic. *PLoS One*. 2022;17(5):e0268119.

12- Karagiannidis C, Mostert C, Hentschker C, Voshaar T, Malzahn J, Schillinger G, et al. Case characteristics, resource use, and outcomes of 10 021 patients with covid-19 admitted to 920 German hospitals: an observational study. *Lancet Respir Med*. 2020;8(9):853-62.

13- La Vignera S, Cannarella R, Condorelli RA, Torre F, Aversa A, Calogero AE. Sex-specific SARS-CoV-2 mortality: among hormone-modulated ACE2 expression, risk of venous thromboembolism and hypovitaminosis D. *Int J Mol Sci*. 2020;21(8):2948.

14- Jin JM, Bai P, He W, Liu S, Wu F, Liu XF, et al. Higher severity and mortality in male patients with COVID-19 independent of age and susceptibility. *medRxiv*. 2020;8(152).

15- Gebhard C, Regitz-Zagrosek V, Neuhauser HK, Morgan R, Klein SL. Impact of sex and gender on COVID-19 outcomes in Europe. *Biol Sex Differ*. 2020;11(29).

16- Asselta R, Paraboschi EM, Mantovani A, Duga S. ACE2 and TMPRSS2 variants and expression as candidates to sex and country differences in COVID-19 severity in Italy. *Aging*. 2020;12(11):10087-98.

17- Márquez EJ, Trowbridge J, Kuchel GA, Banchereau J, Ucar D. The lethal sex gap: COVID-19. *Immun Ageing*. 2022;17:13.

18- Sharifi O, Mokhtarzade M, Beirami BA. A deep convolutional neural network based on local binary patterns of Gabor features for classification of hyperspectral images. *Proceeding of International Conference on Machine Vision and Image Processing (MVIP)*, 18-20 February 2020, Iran. Piscataway: IEEE; 2020.

19- Haddad C, Bou Malhab S, Sacre H, Salameh P. Smoking and COVID-19: a scoping review. *Tob Use Insights*. 2021;14.

20- Israel A, Feldhamer E, Lahad A, Levin-Zamir D, Lavie G.

Smoking and the risk of COVID-19 in a large observational population study. *Medrxiv*. 2020 June.

21- Tsigaris P, Teixeira da Silva JA. Smoking prevalence and COVID-19 in Europe. *Nicotine Tob Res*. 2020;22(9):1646-9.

22- Usman MS, Siddiqi TJ, Khan MS, Patel UK, Shahid I, Ahmed J, et al. Is there a smoker's paradox in COVID-19?. *BMJ Evid Based Med*. 2020;26(6):279-84.

23- Reddy RK, Charles WN, Sklavounos A, Dutt A, Seed PT, Khajuria A. The effect of smoking on COVID-19 severity: a systematic review and meta-analysis. *J Med Virol*. 2021;93(2):1045-56.

24- Badedi M, Makrami A, Alnami A. Co-morbidity and blood group type risk in coronavirus disease 2019 patients: a case-control study. *J Infect Public Health*. 2021;14(4):550-4.

25- Jassim MMA, Mahmood MM, Ali SHM, Kamal MS. Interplay between EBERS and P27 tumor suppressor proteins in molecular transformation of nasopharyngeal and sinonasal carcinomas. *Indian J Public Health Res Dev*. 2019;10(6):894-900.

26- Clark CE, McDonagh ST, McManus RJ, Martin U. COVID-19 and hypertension: risks and management. A scientific statement on behalf of the British and Irish hypertension society. *J Hum Hypertens*. 2021;35(4):304-7.

27- Beyerstedt S, Casaro EB, Rangel ÉB. COVID-19: angiotensin-converting enzyme 2 (ACE2) expression and tissue susceptibility to SARS-CoV-2 infection. *Eur J Clin Microbiol Infect Dis*. 2021;40(5):905-19.

28- Gheware A, Ray A, Rana D, Bajpai P, Nambirajan A, Arulselvi S, et al. ACE2 protein expression in lung tissues of severe COVID-19 infection. *Sci Rep*. 2022;12:4058.

29- Fagyas M, Fejes Z, Sütő R, Nagy Z, Székely B, Pócsi M. Circulating ACE2 activity predicts mortality and disease severity in hospitalized COVID-19 patients. *Int J Infect Dis*. 2022;115:8-16.