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New Onset Diabetes and Its Incidence in Severe COVID 19 Disease A Single Centre Study From Kashmir

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Introduction

Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), the novel coronavirus that causes coronavirus disease 2019 (COVID-19), was first reported in Wuhan, China, in December 2019 and has spread worldwide. SARS-CoV-2 is a positive-stranded RNA virus that is enclosed by a protein containing lipid bilayer with a single-stranded RNA genome; SARS-CoV-2 has 82% homology with human SARS-CoV, which causes severe acute respiratory syndrome.SARS-CoV-2, virus binds to angiotensin-converting enzyme 2 (ACE2) receptors, which are expressed in key metabolic organs and tissues, including pancreatic beta cells, adipose tissue, the small intestine, and the kidneys. Thus, it is believed that SARS-CoV-2 may cause pleiotropic alterations of glucose metabolism that could complicate the pathophysiology of pre-existing diabetes or lead to new mechanisms of disease.

Many studies have made observations that provide support for the hypothesis of a potential diabetogenic effect of Covid-19; in addition it is well-recognized that stress response associated with severe illness have diabetogenic effect. However, whether the alterations of glucose metabolism that occur with a sudden onset in severe COVIOD-19 persist or remit when the infection resolves is unclear. How frequent is the phenomenon of new-onset diabetes, and is it classic type 1 or type 2 diabetes or a new type of diabetes.

Key words: COVID 19; Prediabetes; Diabetes; Pneumonia.

Aims and objectives

The aim of this study was to find out incidence of first time detected diabetes and blood glucose abnormalities in COVID 19 patients with severe disease at presentation to emergency.

Materials and Methods

The study was conducted in emergency unit of internal medicine GMC Srinagar.309 patients admitted during period of 4 months September to December 2020 with severe covid pneumonia were investigated and inquired for diabetes and were grouped into three categories on the basis of blood sugar .Random Blood glucose less than 140 was considered normal between 140-200 as pre diabetic and more than 200 as diabetes. Only patients with bilateral pneumonia and hypoxemia with SPO2 of less than 90% at room air and later confirmed as RT PCR positive for Covid 19 were included. The analysis was done using Software SPSS 23.

Results

In this study of 309 patients (table 2) the number of males was 207(67%) and Females 103(33%).The minimum age was 17yrs and maximum 90 years with a mean of 52.21 years (Table 1)

Out of 309 patients 240(77.6) patients had no underlying co morbidity .Hypertension and diabetes was already present in 26(8.41%) patients COPD was present in 4 (1.29%) patients .COPD and hypertension was pre-existing in 7(2.2%) patients and COPD with Diabetes was pre-existing in 5(1.6%) patients .Isolated diabetes was pre-existing in 11(3.5%) patients and isolated hypertension was seen in 6(1.94%) patients and other illness included chronic kidney disease, coronary artery disease, chronic liver disease ,Malignancy was pre-existing in 27(2.9%) patients (Table 3)

The minimum Random blood sugar was 134 and maximum 712mg/dl and the mean blood sugar Random was 270mg/dl (Table 4)

Out of 309 patients 42 (13.59%) had already pre-existing diabetes and 29(9.38%) had diabetes which was first time detected,208 patients (67.3%) had normal blood sugar and 30 patients(9.7%) has impaired blood sugar falling in pre diabetic range at presentation to Emergency room (Table 5 and 6)

Table 1: showing maximum, minimum and mean age of patients

	No. of Patients	Minimum age	Maximum age	Mean	Std. Deviation
AGE	309	17	90	52.21	17.698

Table 2: showing the gender variable of participating patients.

Gender	Frequency	Percent	Valid Percent	Cumulative Percent
Male	207	67	67	67
Female	102	33	33	100
Total	309	100	100	

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Table 3: showing various co morbidities in admitted patients

Co morbidities	Frequency	Percent	Valid Percent
None	240	77.6	78
Diabetes/Hypertension	26	8.41	8.4
COPD	4	1.29	1.3
COPD/ Hypertension	7	2.26	2.2
COPD/ Diabetes	5	1.61	1.6
Other disease	9	2.91	2.9
Diabetes	11	3.55	3.6
Hypertension	6	1.94	1.9
Total	103	100	100

Table 4: showing minimum, maximum and mean blood sugar random.

	Number	Minimum	Maximum
Blood sugar random	309	76	712
Valid Number	309		

Table 5: showing patients with known diabetes and no history of diabetes at admission.

	Frequency	Percent
No.history of diabetes	267	86.4
Known diabetic	42	13.59
Total	309	100

Table 6: Showing random blood glucose level at admission at categorising 20th patients into normal pre diabetes and diabetes.

	Frequency	Percent
BG<140	208	67.3
BG>140<200	30	9.7
1st time detected BG>200	29	9.3
Known diabetic BG>200	42	13.6
Total	309	100

Discussion

There is bi-directional relationship between coronavirus disease-19 (COVID-19) and diabetes[1]in addition to precipitating new-onset diabetes, COVID-19 may also unmask previously undiagnosed diabetes by causing pleiotropic alterations in glucose metabolism[2] .Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), the virus that causes Covid-19, binds to angiotensin-converting enzyme 2 (ACE2) receptors, which are expressed in key metabolic organs and tissues, including pancreatic beta cells, adipose tissue, the small intestine, and the kidneys[3] Thus, SARS-CoV-2 may cause pleiotropic alterations of glucose metabolism that could complicate the pathophysiology of pre-existing diabetes or lead to new mechanisms of disease. There are also several precedents for a viral cause of ketosis-prone diabetes, including other coronaviruses that bind to ACE2 receptors[4]. There have been reports of COVID-19 induced severe metabolic decompensation of pre-existing or new-onset diabetes such as diabetic ketoacidosis (DKA) and hyperglycaemic hyperosmolar state (HHS) [4-9].

Infection with SARS COV 2 can lead to increased levels of inflammatory mediators in the blood, including lipopolysaccharide[10,11]. Poor glycaemic control predicts an increased need for medications and hospitalizations, and increased mortality[12,13]. Several mechanisms have been proposed by which virally induced inflammation and increases insulin resistance[14].

Multiple studies have been conducted to find the prevalence of diabetes in COVID 19. In six studies, the prevalence of diabetes was ≤10% that it was 128 diabetes patients in 2333 patients with COVID-19 included Wang (15/242)[15] Guan (81/1099)[16] , Wan (12/135)[17] , Hui (2/41)[18], Yang (9/710)[19], and Chen (9/106)[20]. Fourteen studies report prevalence in 10.1–20% that it was 216 diabetes patients in 1559 patients with COVID-19 included Shi (10/81) [21], Zhao (4/37)[22] , Hu (47/323)[23] , Zhang (17/140)[24] , Zhou (36/191)[25] , Wang (54/339)[26], Wang (14/138)[27] , Wu (22/201)[28] , and Liu (12/109)[29] . And three studies report prevalence above 20% that it was 171 diabetes patients in 404 patients with COVID-19 included Xu (147/355)[30] , Bhatraju (14/24) [31], and Li (10/25 death) [32]The results of meta-analysis on 18 studies has 14.5% of the subjects with diabetes, in which there was no publication bias (t = 1.06 P = 0.304).

In our study incidence of diabetes was very high(22.9%) 42 of 309 patients (13.6%) patients had already diabetes and were on treatment at the time of admission to hospital, 29 of 309 (9.3%) patients had new onset diabetes which was diagnosed at time of admission and 30 out of 309 patients (9.7%) had impaired blood sugar at presentation.

Conclusion

The high incidence of new onset diabetes in our population with severe COVID 19 disease could be due to multiple factors.

1. Only patients with severe COVID 19 Pneumonia were enrolled.

2. There could be high incidence of undiagnosed diabetes in our population.

3. Our population may have some genetic predisposition to diabetes which is activated by COVID 19

References

1. Papadokostaki E, Tentolouris N, Liberopoulos E. COVID-19 and diabetes: what does the clinician need to know? Prim Care Diabetes. 2020;14(5):558-563.doi: 10.1016/j.pcd.2020.06.010.
2. Li H, Tian S, Chen T, Cui Z, Shi N, Zhong X, et al. Newly diagnosed diabetes is associated with a higher risk of mortality than known diabetes in hospitalized patients with COVID-19.Diabetes Obes. Metab. 2020;22(10):1897-1906.doi:10.1111/dom.14099.
3. Hamming I, Timens W, Bulthuis ML, Lely AT, Navis G, van Goor H. Tissue distribution of ACE2 protein, the functional receptor for SARS coronavirus: a first step in understanding SARS pathogenesis. J Pathol.2004;203(2):631-637.doi:10.1002/path.1570.
4. Yang J-K, Lin S-S, Ji X-J, Guo L-M. Binding of SARS coronavirus to its receptor damages islets and causes acute diabetes. Acta Diabetol.2010; 47(3):193-199.doi: 10.1007/s00592-009-0109-4.
5. YJ Chee, SJH Ng, E Yeoh. Diabetic ketoacidosis precipitated by Covid-19 in a patient with newly diagnosed diabetes mellitus. Diabetes Res Clin Pract. 2020;164:108166.doi:10.1016/j.diabres.2020.108166.
6. C Sardu, N D'Onofrio, ML Balestrieri, M Barbieri, MR Rizzo, V Messina, et al. Outcomes in patients with hyperglycemia affected by COVID-19: can we do more on glycemic control? Diabetes Care. 2020;43(7):1408-1415.doi: 10.2337/dc20-0723.
7. J Li, X Wang, J Chen, X Zuo, H Zhang, A Deng. COVID-19 infection may cause ketosis and ketoacidosis. Diabetes Obes Metab. 2020;22(10):1935-1941.doi: 10.1111/dom.14057.
8. M Apicella, MC Campopiano, M Mantuano, L Mazoni, A Coppelli, SD Prato. COVID-19 in people with diabetes: understanding the reasons for worse outcomes. The lancetDiabetes & Endocrinology. 2020;8(9).782-792.doi:10.1016/s2213-8587(20)30238-2.
9. PK Reddy, MS Kuchay, Y Mehta, SK Mishra. Diabetic ketoacidosis precipitated by COVID-19: a report of two cases and review of literature. Diabetes Metab Syndr.2020;14(5).1459-1462. doi:10.1016/j.dsx.2020.07.050.
10. Nansen A, Christensen J P, Marker O, Thomsen A R. Sensitization to lipopolysaccharide in mice with asymptomatic viral infection: role of T cell-dependent production of interferon-gamma. J Infect Dis.1997;176(1):151-157.doi: 10.1086/514017.
11. Neu U and Mainou B A. Virus interactions with bacteria: partners in the infectious dance. PLoS Pathog.2020;16(2):e1008234.doi: 10.1371/journal.ppat.1008234.
12. Zhu L, ZG She, X Cheng , JJ Qin, XJ Zhang, J Cai, et al. Association of blood glucose control and outcomes in patients with COVID-19 and pre-existing type 2 diabetes. Cell Metab. 2020;31(6):1068-1077.e3.doi: 10.1016/j.cmet.2020.04.021.
13. J A Critchley, I M Carey, T Harris, S DeWilde, FJ Hosking, DG Cook. Glycemic control and risk of infections among people with type 1 or type 2 diabetes in a large primary care cohort study. Diabetes Care. 2018;41(10):2127-2135.doi: 10.2337/dc18-0287.
14. M Šestan, S Marinović, I Kavazović, Đ Cekinović , S Wueest, T T Wensveen, et al. Virus-induced interferon-γ causes insulin resistance in skeletal muscle and derails glycemic control in obesity. Immunity.2018.17;49(1):164-177.e6.doi: 10.1016/j.immuni.2018.05.005.
15. J Zhou, J Sun, Z Cao, W Wang, K Huang, F Zheng, et al. Epidemiological and clinical features of 201 COVID-19 patients in Changsha city, Hunan, China. Medicine (Baltimore). 2020; 99(34): e21824.doi:10.1097/MD.0000000000021824.
16. Guan W-J, Ni Z-Y, Hu Y, Liang W-H, Ou C-Q, He J-X, et al. Clinical characteristics of coronavirus disease 2019 in China. N Engl J Med.2020; 382:1708-1720.doi:10.1056/NEJMoa2002032.
17. Wan S, Xiang Y, Fang W, Zheng Y, Li B, Hu Y, et al. Clinical Features and Treatment of COVID-19 Patients in Northeast Chongqing. J Med Virol. 2020;92(7):797-806.doi: 10.1002/jmv.25783.
18. Hui H, Zhang Y, Yang X, Wang X, He B, Li L, et al. Clinical and radiographic features of cardiac injury in patients with 2019 novel coronavirus pneumonia. medRxiv; 2020.doi:10.1101/2020.02.24.20027052.
19. Yang X, Yu Y, Xu J, Shu H, Liu H, Wu Y, et al. Clinical course and outcomes of critically ill patients with SARS-CoV-2 pneumonia in Wuhan, China: a single-centered, retrospective, observational study. Lancet Respir Med. 2020;8(5):475-481.doi:10.1016/S2213-2600(20)30079-5.
20. Chen X, Hu W, Ling J, Mo P, Zhang Y, Jiang Q, et al. Hypertension and diabetes delay the viral clearance in COVID-19 patients. medRxiv. 2020.doi:10.1101/2020.03.22.20040774.
21. Shi H, Han X, Jiang N, Cao Y, Alwalid O, Gu J, et al. Radiological findings from 81 patients with COVID-19 pneumonia in Wuhan, China: a descriptive study. Lancet Infect Dis. 2020;20(4):425-434.doi:10.1016/S1473-3099(20)30086-4.
22. Zhao S, Ling K, Yan H, Zhong L, Peng X, Yao S, et al. Anesthetic management of patients with COVID 19 infections during emergency procedures. J Cardiothorac Vasc Anesth. 2020;34(5):1125-1131. doi:10.1053/j.jvca.2020.02.039.
23. Hu L, Chen S, Fu Y, Gao Z, Long H, Ren H-W, et al. Risk factors associated with clinical outcomes in 323 COVID-19 patients in Wuhan, China. medRxiv. 2020. doi:10.1101/2020.03.25.20037721.
24. Zhang J-J, Dong X, Cao Y-Y, Yuan Y-D, Yang Y-B, Yan Y-Q, et al. Clinical characteristics of 140 patients infected with SARS-CoV-2 in Wuhan, China. Allergy. 2020;75(7):1730-1741.doi: 10.1111/all.14238.
25. Zhou F, Yu T, Du R, Fan G, Liu Y, Liu Z, et al. Clinical course and risk factors for mortality of adult inpatients with COVID-19 in Wuhan, China: a retrospective cohort study. Lancet. 2020;395(10229):1054-1062.doi: 10.1016/S0140-6736(20)30566-3.
26. Wang L, He W, Yu X, Hu D, Bao M, Liu H, et al. Coronavirus Disease 2019 in elderly patients: characteristics and prognostic factors based on 4-week follow-up. J Infect. 2020;80(6):639-645.doi: 10.1016/j.jinf.2020.03.019.
27. Wang D, Hu B, Hu C, Zhu F, Liu X, Zhang J, et al. Clinical characteristics of 138 hospitalized patients with 2019 novel coronavirus–infected pneumonia in Wuhan, China. JAMA. 2020;323(11):1061-1069.doi: 10.1001/jama.2020.1585.
28. Wu C, Chen X, Cai Y, Zhou X, Xu S, Huang H, et al. Risk factors associated with acute respiratory distress syndrome and death in patients with coronavirus disease 2019 pneumonia in Wuhan, China. JAMA Intern Med. 2020; 180(7): 1–11.doi:10.1001/jamainternmed.2020.0994.
29. Liu Y, Sun W, Chen L, Wang Y, Zhang L, Yu L. Clinical characteristics and

progression of 2019 novel coronavirus-infected patients concurrent acute respiratory distress syndrome. medRxiv; 2020.doi:10.1001/jamainternmed.2020.0994.

30. Xu S, Fu L, Fei J, Xiang H-X, Xiang Y, Tan Z-X, et al. Acute kidney injury at early stage as a negative prognostic indicator of patients with COVID-19: a hospital-based retrospective analysis. medRxiv. 2020.doi :10.1101/2020.03.24.20042408.
31. Bhatraju PK, Ghassemieh BJ, Nichols M, Kim R, Jerome KR, Nalla AK, et al. Covid-19 in critically ill patients in the Seattle region—case series. N Engl J Med. 2020;382(21):2012-2022.doi: 10.1056/NEJMoa2004500.
32. Li X, Wang L, Yan S, Yang F, Xiang L, Zhu J, et al. Clinical characteristics of 25 death cases with COVID-19: a retrospective review of medical records in a single medical center, Wuhan, China. Int J Infect Dis. 2020;94:128-132.doi: 10.1016/j.ijid.2020.03.053.