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Clinical Characteristics of COVID-19 in Patients with Type 2 Diabetes

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Abstract

A total of 74 COVID-19 diabetic patients were enrolled in the study. Twenty-seven patients (36.5%) were severely ill, and ten patients (13.5%) died. Severe patients had higher blood glucose, serum amyloid A (SAA), C reactive protein, and interleukin 6 levels than non-severe patients ($P < 0.05$). Severe patients had lower levels of albumin, cholesterol, high density lipoprotein, small and dense low density lipoprotein, and CD4 T lymphocyte counts than non-severe patients ($P < 0.05$). Reduced CD4 21 + T lymphocyte counts (OR = 0.988, 95 percent confidence interval [95 percent CI] 0.979–0.997) and elevated SAA levels (OR = 1.029, 95 percent CI 1.002–1.058) were found as risk variables for COVID- 19 severity with diabetes in a logistic regression analysis ($P < 0.05$).

Keywords: COVID-19, Diabetes, Hyperglycemia, Dislipidemia, CD4+ T lymphocyte , follow up symptoms.

Introduction

Type 2 diabetes is a chronic condition that develops when the pancreas is unable to produce enough insulin or when the body's tissues are unable to adequately utilize insulin. Insulin is a hormone that aids the utilization of sugar (glucose) for energy by the body's cells. It also aids in the storage of excess sugar in the body's muscle, fat, and liver cells [1]. Coronavirus disease 2019 (COVID-19) is a newly recognized viral disease caused by the severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) virus, which was recently declared a pandemic. The number of people who have died as a result of COVID-19 has risen as of April 21, 42 2020, 2397 people had been confirmed worldwide, with 162,956 instances dying. China has diagnosed 84,250 cases in total, including 4642 deaths. Previous research has outlined the overall clinical characteristics and epidemiological findings of COVID-19 individuals, and certain clinical observations have revealed that the status of certain individuals is progressively deteriorating [2]. Diabetic people are more susceptible to infection and have a poor prognosis due to various innate immune and metabolic problems. Pneumonia, caused by a variety of

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infections, has become an increasingly common cause of death in diabetics. Clinical aspects of COVID-19 in diabetics, particularly severe cases, have sparked widespread concern [3].

Type 2 diabetes: follow up: Self-control is a skill that may be learned. the glycosuria could be a sign of poor glucose control. It is necessary to investigate the knowledge of diabetes, diabetes medication, and diabetes management practices. The cornerstones of successful diabetes control include a proper diet and lifestyle strategies such as regular physical activity. Assessing these can assist in identifying motivational issues and highlighting knowledge gaps. Also, Cardiovascular disease will claim the lives of 80% of diabetics. Not only this but also is to look for symptoms and indicators of coronary heart disease and peripheral vascular disease. The management must be personalized for each individual. A younger diabetic will require more strict risk factor management. Cardiac disease might start with erectile dysfunction. Moreover, Problems with psychosocial functioning, in order to screen for depression, it's crucial to ask about the patient's coping with the illness and mood, just as it is with any long-term ailment [4-7].

The overall case-fatality rate (CFR) of COVID-19 was 2.3 percent in a retrospective cohort study from China, but CFR was considerably higher among individuals with diabetes (7.3 percent). Preexisting diabetes individuals accounted for 31% of COVID-19 cases that died. However, there are few papers describing the clinical characteristics of COVID-19 individuals with type 2 diabetes or clinical research examining risk factors for COVID-19 severity in diabetic populations [8]. This study looked at the clinical characteristics of COVID-19 patients with type 2 diabetes who were hospitalized at Zhongnan Hospital of Wuhan University in Wuhan, China, and specifically looked at risk variables linked to COVID-19 severity in diabetics.

Material and Methods

Participants and study design

This single-center retrospective observational study was performed at Zhongnan Hospital of Wuhan University in Wuhan, China, which is a designated hospital for COVID-19. We analyzed 74 COVID-19 patients with type 2 diabetes who were either treated and discharged or died 70 during hospitalization from January 3 to April 14, 2020. The Chinese National Health Committee confirmed all of the patients based on the COVID-19 diagnosis and treatment guidelines (version 5). The institutional ethics board of Wuhan University's Zhongnan Hospital gave their approval to this study (No.2020042K) [9]. A 78-member team of trained physicians from Wuhan University's Zhongnan Hospital analyzed the computerized medical data of these patients. Patients' demographics, exposure histories, comorbidities, diabetic medicines, signs and symptoms, laboratory exams, and therapies were all part of the information gathered (antibiotics, corticosteroid, respiratory supports, kidney replacement

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therapy and ECMO). Patients who met one of the following criteria were classified as having severe COVID-19: (a) respiratory distress with a respiratory frequency of less than 30 per minute; (b) pulse oximeter oxygen saturation of less than 93 percent at rest; and (c) oxygenation index (artery partial pressure of oxygen/inspired oxy88 gen fraction, PaO2/FiO2) of less than 300 mm Hg [10].

Statistical analysis Continuous variables were characterized using median (interquartile range [IQR]) values, whereas categorical variables were reported as frequency rates and percentages. When the data were regularly distributed, the means for continuous variables were compared using independent group t-tests; otherwise, the Mann-Whitney test was utilized. The 2 test was used to compare proportions for categorical variables. Statistical significance was defined as a 2-sided P b 0.05. The risk factors linked to severity were investigated using a logistic regression analysis. On a stratified analysis odds ratio (OR) provided with 95 percent confidence intervals, we assessed interactions in the model that were significant (95 percent CI). All statistical analyses were performed with SPSS, version 22.0 (IBM Corp) for Windows [11].

Results

COVID-19 type 2 diabetes patients' clinical features. This study included 74 Type 2 diabetes patients who have been confirmed as COVID-19 patients. 36 (48.6%) of the patients were male and 38 (51.4%) were female, with a median age of 62 years (interquartile range [IQR] 58–81). COVID-19 was infected in communities by the majority of patients (71/74, or 95.9%) [12]. Apart from diabetes, 55 (74.3%) of the 74 patients had 109 concomitant chronic conditions, the three most prevalent of which were hypertension (47.3%), coronary heart disease (17.6%), and secondary pulmonary tuberculosis (17.6%). (16.2 percent). Table 1 shows the patients' symptoms at the start of treatment. Fever was the most prevalent 113 symptoms, with 77.0 percent reporting it, followed by respiratory illness 114 (70.3 percent reporting cough, chest tightness, or dyspnea), gastrointestinal 115 symptoms (28.4 percent reporting nausea, diarrhea, and abdominal discomfort), and weariness [13]. (24.3 percent). As shown in Table 1, 27 (36.5%) of the 74 patients were severe cases, and 10 (13.5%) of the patients died. 18 (66.7%) of the severe patients, with a median age of 72 (58–81) years, were male. Males made up 18 (38.3%) of the non-severe cases, with a median age of 61 (54–67) years. Men over the age of 50 were more likely to have severe COVID-19 symptoms (P <0.05) [14]. Twenty-two severe patients (81.5%) had pre-existing chronic conditions in addition to diabetes, compared to 124 33 (70.2%) non-severe patients. The prevalence of coronary heart disease was 29.6% in the severe group and 10.6% in the non-severe group (P < 0.05), showing that type 2 diabetes individuals with coronary heart disease are more likely to have severe COVID-19. There were no significant variations in hypertension prevalence between those with severe and those without severe hypertension (42.6 percent vs. 55.6 percent, P >0.05). In total, 58 patients

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(78.4%) were overweight, with no significant differences between the severe and non-severe groups [14].

Table 1
Demographics and clinical characteristics of 74 COVID-19 patients with type 2 diabetes.

	All patients (n = 74)	Diseases severity		
		Non-severe patients (n = 47)	Severe patients (n = 27)	P value
Age, years	62(56–72)	61(54–67)	72(58–81)	0.012
Sex				
Male	36(48.6%)	18(38.3%)	18(66.7%)	0.019
Female	38(51.4%)	29(61.7%)	9(33.3%)	
BMI, kg/m^2	24.54 (22.27–27.55)	24.66 (21.72–27.69)	24.54 (22.64–26.99)	0.961
BMI < 18.5	0	0	0	0.495
18.5 ≤ BMI < 23	16(21.6%)	9(19.1%)	7(25.9%)	
BMI ≥ 23	58(78.4%)	38(80.9%)	20(74.1%)	
Exposure history				
Community infections	71(95.9%)	47(100%)	24(88.9%)	0.012
Hospital infections	3(4.1%)	0	3(11.1%)	
Comorbidity	55(74.3%)	33(70.2%)	22(81.5%)	0.285
Hypertension	35(47.3%)	20(42.6%)	15(55.6%)	0.281
Coronary heart disease	13(17.6%)	5(10.6%)	8(29.6%)	0.042
Stroke	2(2.7%)	0	2(0.4%)	0.042
Secondary pulmonary tuberculosis	12(16.2%)	6(12.8%)	6(22.2%)	0.295
Tumor	7(9.5%)	5(10.6%)	2(7.4%)	0.642
Cholelithiasis	10(13.5%)	4(8.5%)	6(22.2%)	0.104
Symptoms during hospitalization				
Fever	57(77.0%)	34(72.3%)	23(85.2%)	0.206
Respiratory symptoms	52(70.3%)	29(61.7%)	23(85.2%)	0.033
Gastrointestinal symptoms	21(28.4%)	10(21.3%)	11(40.7%)	0.074
Fatigue	18(24.3%)	12(25.5%)	6(22.2%)	0.749
Headache	4(5.4%)	3(6.4%)	1(3.7%)	0.614
Treatments				
Noninvasive mechanical ventilation	7(9.5%)	0	7(25.9)	<0.01
Invasive mechanical ventilation	11(14.9%)	0	11(40.7%)	<0.01
ECMO	2(2.7%)	0	2(7.4%)	0.042
CRRT	3(4.1%)	0	3(11.1%)	0.012
Glucocorticoid therapy	33(44.6%)	8(17.0%)	25(92.6%)	<0.01
Antibiotics therapy	59(79.7%)	32(68.1%)	27(100%)	<0.01
Anti-fungal therapy	9(12.2%)	1(2.1%)	8(29.6%)	<0.01
Prognosis				
Recovery	54(73.0%)	43(91.5%)	11(40.7%)	<0.01
Dead	10(13.5%)	0	10(37.0%)	<0.01
In hospital	10(13.5%)	4(8.5%)	6(22.2%)	0.156
Diabetic management				
Insulin injection	43(58.1%)	24(51.1%)	19(70.4%)	0.105
Metformin	25(33.8%)	21(44.7%)	4(14.8%)	<0.01
α-Glucosidase inhibitors	37(50%)	23(48.9%)	14(51.9%)	0.809
Others	20(27.0%)	12(25.5%)	8(29.6%)	0.702

Abbreviations: *ECMO* Extracorporeal Membrane Oxygenation, *CRRT* Continuous Renal Replacement Therapy.
Data are n (%), n/N (%) and median (IQR).
P < 0.05 was considered statistically significant between severe and non-severe subgroups.

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Laboratory findings and risk factors for severity. Fasting blood glucose (FBG), serum amyloid A (SAA), C-reactive protein (CRP), interleukin 6 (IL-6), absolute numbers of neutrophils, alanine aminotransferase, aspertate aminiotransferase, creatinine, serum cystatin C, eGFR (estimating glomerular filtration rate using the CKD-EPI cystatin and creatinine 2012 equation), 2-microglobulin, and Severe patients had higher levels of creatine kinase isoenzyme (CK-MB), high sensitivity troponin I (hsTnI), lactate dehydrogenase, and D-dimer than non-severe patients ($P < 0.05$). The homes of 57 patients were got to visit and their house dust was tested for allergens of dust mite, cockroach and cat; the results are expressed as micrograms per gram of sieved dust for allergens of cat and dust mite and as units per gram for allergens of cockroach (Figure) [11]. For patients with asthma and control subjects, the percentage of homes with high levels of each allergen (and, in particular, the Mlergen cockroach) was very similar. There was a cat in the house for none of the patients or control subjects, and only two houses had >8 ug Fel d I/gin dust. Some patients did not have dust samples taken because they did not agree to visits or it was not safe to visit their neighborhoods [15]. Dust was also available at Eggleston Hospital from the homes of 26 patients seen for asthma in the pediatric pulmonary clinic. None of those samples ($n = 85$) had significant cockroach allergen (0/85 >2 units Bla g II per gram of dust). Albumin, cholesterol, high density lipoprotein (HDL), small and dense low density lipoprotein (sd-LDL), and CD4 levels, on the other hand, are all factors to consider. The number of lymphocytes in severe patients was significantly lower than in non-severe instances ($P < 0.05$). Furthermore, in routine urine tests, severe patients had larger percentages of positive protein, glucose, and ketone than non-severe patients ($P < 0.05$) [16]. Furthermore, there were no significant differences between the two groups in hemoglobin, platelet count, glycated hemoglobin (HbA1c), triglyceride, low density lipoprotein (LDL), free fatty acid (FFA), uric acid, crea150 tine kinase, erythrocyte sedimentation rate, NK cell count, CD8+ T lymphocyte count, ACE, and serum antibodies for SARS-CoV-2 ($P > 0.05$. Between severe and non-severe patients, the duration of infectious virus replication (defined as conversion from positive to negative swabs) was also similar ($P > 0.05$). (Table 2) [17].

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Table 2
Laboratory results of COVID-19 patients with type 2 diabetes.

	All patients (n = 74)	Diseases severity		
		Non-severe patients (n = 47)	Severe patients (n = 27)	*P* value
Complete blood cell count, $\times 10^9$/L				
Leukocytes	5.66(4.74–7.67)	5.26(4.34–6.17)	7.35(5.41–10.22)	<0.01
Neutrophils	3.53(2.89–6.21)	3.09(2.47–4.02)	6.21(3.44–8.52)	<0.01
Lymphocytes	1.12(0.70–1.71)	1.42(1.04–1.89)	0.7(0.42–0.94)	<0.01
Eosinophils	0.06(0.03–0.10)	0.08(0.04–0.13)	0.03(0.01–0.06)	<0.01
Hemoglobin, g/L	122(114–132)	123(118–133)	120(102—131)	0.056
Platelet count, $\times 10^9$/L	186(148–255)	318.3(239.3–388.6)	172(133–248)	0.985
Diabetic indexes				
HbA1c, %(mmol/mol)	8.7(72)(7.7[61]-10.2[88])	8.8(73)(8.2[66]-10.2[88])	8.7(72)(7.4[57]-10.5[91])	0.925
Blood glucose, mmol/L	8.2(6.7–11.0)	7.35(5.91–10.65)	9.67(7.72–12.88)	0.044
Blood lipids				
Triglyceride, mmol/L	1.26(0.93–2.19)	1.50(0.93–2.31)	1.18(0.89–1.77)	0.134
Cholesterol, mmol/L	4.21(3.64–4.97)	4.25(3.71–5.75)	4.01(2.95–4.59)	0.019
HDL, mmol/L	1.03(0.86–1.25)	1.08(0.96–1.28)	0.92(0.74–1.20)	0.021
LDL, mmol/L	2.60(1.97–3.25)	2.76(2.16–3.31)	2.1(1.63–3.13)	0.055
sd-LDL, mmol/L	0.90(0.55–1.22)	1.00(0.62–1.26)	0.80(0.35–0.95)	0.014
FFA, μmol/L	122.4(474.6–4228.2)	131.2(479.4–956.3)	122.4(339.8–4228.2)	0.367
Liver and renal function				
Albumin, g/L	35.0(31.2–39.9)	38.5(34.6–41.1)	31.3(29.9–33.4)	<0.01
Alanine aminotransferase, U/L	30(15–49)	24(15–43)	44(22–53)	0.029
Aspartate aminotransferase, U/L	26(17–43)	20(16–31)	39(26–56)	<0.01
Creatinine, μmol/L	60.2(49.5–76.3)	59.1(46.0–69.5)	67.8(53.9–87.7)	0.028
Cystatin C, mg/L	0.98(0.80–1.25)	0.86(0.78–1.14)	1.21(0.95–1.36)	<0.01
eGFR, mL/min/1.73m^2	88.00 (68.00–104.50)	81.00 (67.00–105.00)	89.00 (69.75–104.75)	<0.01
CK-MB fraction, U/L	10(8–14)	10(7–11)	15(11—23)	0.011
Lactate dehydrogenase, U/L	196(159–272)	176(150–224)	281(214–478)	<0.01
hsTnI, pg/mL	5.7(2.2–22.7)	2.7(1.2–5.3)	16.5(7.2–109.1)	<0.01
Inflammatory biomarkers				
Procalcitonin, ng/mL	0.05(0.05–0.24)	0.05(0.05–0.055)	0.19(0.06–0.61)	0.012
CRP, mg/L	15.0(3.8–56.3)	8.7(2.6–28.6)	51.8(20.0–99.6)	<0.01
ESR, mm/h	26(15–50)	24(10–46)	31(17–63)	0.173
IgE, kU/L	39.6(23.7–150.2)	30.7(21.6–45.1)	198.0(132.7–264.5)	<0.01
IL-6, pg/mL	11.71(3.56–57.17)	4.41(2.66–13.00)	51.95(11.93–91.51)	<0.01
SAA, mg/L	31.16(8.08–106.02)	10.84(5.99–55.15)	106.05(52.05–167.62)	<0.01
SOD, kU/L	74.0 (160.3–224.9)	90.1 (171.3–224.9)	74.0 (128.7–170.3)	<0.01
C1q, mg/L	123.9 (185.6–313.7)	127.5 (193.0–266.1)	123.9 (177.4–313.7)	0.109
Lymphocyte subsets, /μL				
NK cell count	198(72,385)	232(96,484)	103(49,322)	0.059
B lymphocyte count	187(79,270)	207(150,300)	91(55,229)	0.036
T lymphocyte count	837(396,1178)	1070(812,1307)	394(201,619)	0.01
CD4+ lymphocyte count	494(240,715)	598(512,870)	234(74,319)	<0.01
CD8+ lymphocyte count	315(146,438)	361(295,486)	133(77,231)	0.446
D-dimer, ng/mL,	352(163–968)	242(114–424)	827(368–1865)	<0.01
ACE, U/L	18(15–37)	36(16–38)	16(14–22)	0.057
Routine urine test				
Proteinuria	29/62(46.8%)	12/41(29.3%)	17/21(81.0%)	<0.01
Urine glucose	39/62(62.9%)	20/41(48.8%)	19/21(90.5%)	<0.01
Urine ketone	15/62(24.2%)	5/41(12.2%)	10/21(47.6%)	<0.01
Duration of virus replication, day	10.0(5.0–19.5)	8.0(4.0–19.7)	10.0(6.0–19.5)	0.745
Antibodies of SARS-CoV-2, AU/mL				
IgG antibody	21.99(14.20–36.73)	21.52(13.56–33.98)	27.12(18.40–47.12)	0.965
IgM antibody	1.85(0.73–2.65)	1.87(0.80–2.67)	1.83(0.50–2.61)	0.652

Diabetes patients with COVID-19 have a higher risk of developing the disease. Reduced CD4+ T lymphocyte counts (OR = 0.988, 95 percent confidence interval [95 percent CI] 0.979–0.997) and elevated SAA levels (OR = 1.029, 95 percent CI 1.002–1.058) at admission were identified as risk variables for severity of COVID-19 with type 2 diabetes in a logistic regression analysis [18].

Table 3
Logistic regression modelling evaluating risk factors for severity of COVID-19 patients with type 2 diabetes.

Item	β	S.E.	Wald	df	P value	OR	95% CI
SAA	0.029	0.014	4.289	1	0.038	1.029	1.002–1.058
CD4+ lymphocyte count	−0.012	0.004	7.311	1	0.007	0.988	0.979–0.997

Abbreviations: *SAA* serum amyloid A.
$P < 0.05$ was considered statistically significant.

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Discussion

In this single-center retrospective research of 74 COVID-19 type 2 diabetes patients, 36.5 percent (27/74) were severe cases, with a 13.5 percent (10/74) fatality rate. Patients who were older and male were more likely to acquire a severe form of COVID-19 (median age 72 (58–81) years, 66.7 percent male). According to the previous research, the 99 patients of COVID-19 admitted to Jinyintan Hospital in Wuhan 9 were mostly male (68.0 percent), with a median age of 55.5 years, a 23 percent ICU requirement, and an 11 percent fatality rate. In another study, 138 COVID-19 case series10 revealed a 26 percent (36/138) ICU admission rate with a 4.3 percent (6/138) fatality rate. In comparison to their findings, our investigation of COVID-19 patients with type 2 diabetes found a higher likelihood of ICU hospitalization and even mortality than the total COVID-19 infection population. It's still unclear whether type 2 diabetes-related hyperglycemia and dyslipidemia play a role in COVID-19 aggravation. Despite the fact that the two groups had similar HbA1c levels, the level of FBG in severe patients was 9.67 (7.72–12.88) mmol/L, which was significantly higher than the level in non-severe cases (7.35 [5.91–10.65] mmol/L, $P = 0.044$). Furthermore, the severe group had considerably higher positive rates of glucose and ketone in urine tests than the non-severe group (90.5 percent vs 47.6 percent, respectively). suggesting that COVID-19 degradation is linked to acute hyperglycemia. Despite the fact that our study failed to confirm hyperglycemia as a risk factor for severe infections, a multi-center study of 7337 cases in Wuhan11 found that individuals with well-controlled blood glucose (BG, glycemic variability within 3.9–10.0 mmol/L) had significantly lower mortality than those with poorly controlled blood glucose (BG, glycemic variability within 3.9–10.0 mmol/L). Severe patients had higher levels of hypoalbuminemia and dislipidemia than non-severe patients due to lower levels of albumin, cholesterol, HDL, and sd-LDL ($P < 0.05$). Although no significant differences in triglyceride, LDL, or FFA levels were discovered, there were downtrends between two groups. Previous research has shown that some foods, such as amino acids and lipids, can influence the immune system by initiating, interacting, differentiating, and expressing immune cells' functional expression. Furthermore, energy or protein deficiency were major risk factors 200 for intestinal dysbacteriosis. Some nutrients, such as amino acids and lipids, have been demonstrated to affect the immune system by starting, interacting, differentiating, and expressing the functional expression of immune cells in the past. 12 In addition, a lack of energy or protein was a strong risk factor for intestinal dysbacteriosis in 200 people. In severe patients, higher neutrophil counts, alanine aminotransferase, aspertate aminiotransferase, creatinine, serum cystatin C, eGFR, 2-microglobulin, CK-MB, hsTnI, and lower lymphocyte counts reflected more secondary infections and impaired liver, kidney, and heart functions, according to other studies. Obesity or hypertension did not play a significant influence in predicting severe and non-severe COVID-19 patients in this investigation. Simonnet et al. 18 found that obesity and a high BMI

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were linked to disease severity and the necessity for invasive mechanical ventilation in a prior investigation [19].

Conclusion in conclusion, when diabetic patients were infected with SARS-CoV-2, they had a greater risk of developing more severe diseases than the general population the COVID-19 patients with diabetes who had severe cases were mostly older males. Hyperglycemia and dyslipidemia may be linked to virus susceptibility and immune system damage. In COVID-19 patients with diabetes, aggressive treatment such as better blood glucose management and nutrition delivery should be considered, especially when these patients had lower CD4 + T lymphocyte counts and higher SAA levels.

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