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Analysis of clinical features and pulmonary CT features of coronavirus disease 2019 (COVID-19) patients with diabetes mellitus

Yimin Yan[1, 2], Fang Yang[2], Xinxin Zhu[2], Min Wang[2], Zhibing Sun[1], Tao Zhao[1], Xiaohong Yang[3], Yi Zou[1]

[1]*Department of Endocrinology, Xiaogan Hospital Affiliated to Wuhan University of Science and Technology, The Central Hospital of Xiaogan, Xiaogan, Hubei, China*
[2]*Medical College of Wuhan University of Science and Technology, Wuhan, China*
[3]*Department of Gynaecological Endocrinology, Xiaogan Hospital Affiliated to Wuhan University of Science and Technology, The Central Hospital of Xiaogan, Xiaogan, Hubei, China*

Abstract

Introduction: The objective of this paper was to investigate the clinical features and pulmonary CT imaging features of COVID-19 patients with diabetes mellitus.

Material and methods: From January 16, 2020 to March 28, 2020, among the 568 cases of COVID-19 patients diagnosed in Xiaogan Central Hospital, 64 cases of COVID-19 patients with diabetes were selected as the diabetic group, and 64 cases of COVID-19 patients with age and gender matching without diabetes were selected as the non-diabetic group, and their clinical data and pulmonary CT characteristics were retrospectively analysed.

Results: Compared with the non-diabetic group, the proportion of patients in the diabetic group with chronic underlying disease was higher, and they were in more a serious condition at admission. Inflammation index and characteristics of glycolipid metabolism results showed that COVID-19 patients with diabetes mellitus were more likely to have elevated inflammatory markers and hypercoagulability, accompanied by hypoproteinaemia and glucose and lipid metabolism disorders. Treatment and clinic outcome results showed that the time of nucleic acid turning negative in the diabetic group was significantly longer than that in the non-diabetic group. Radiological data showed that COVID-19 combined with diabetes prolonged the time of detoxification in patients.

Conclusion: COVID-19 patients with diabetes mellitus and chronic hypertension are associated with increased inflammatory markers and disorders of glucose and lipid metabolism. These patients tend to develop serious diseases, especially the rapid progression of CT lesions in the lungs of patients with a wide range of involvement, and prolonged absorption and detoxification time. (**Endokrynol Pol 2020; 71 (5): 367–375)**

Key words: *COVID-19; diabetes; CT image; detoxification time*

Introduction

Coronaviruses are enveloped RNA viruses that are widely distributed in humans, other mammals, and birds, and which cause respiratory, intestinal, liver, and neurological diseases [1–2]. At present, six kinds of coronaviruses are known to cause human diseases [3]. In view of the high prevalence and wide distribution of coronaviruses, the huge genetic diversity, and frequent recombination of their genomes, as well as the increasing human-animal interface activities that induce frequent cross-species infections and occasional spillovers, new coronaviruses may appear periodically in human beings [4–5]. In December 2019, the outbreak of severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) was first reported in Wuhan, China, which had a huge impact on China and the world. The disease caused by SARS-CoV-2 was named as coronavirus disease 2019 (COVID-19) [6]. In clinical practice, we have observed many severe or critically COVID-19 patients with typical clinical manifestations of shock, including cold extremities and weak peripheral pulse. Even in the absence of obvious hypotension, many patients present with severe metabolic acidosis, suggesting the possibility of microcirculatory dysfunction [7]. Furthermore, in addition to severe lung injury, some patients also have impaired liver and kidney functions [8], which suggests that COVID-19 may be associated with chronic underlying diseases.

As a representative of chronic basic diseases, diabetes is a disease involving multiple metabolic disorders, characterised by high blood glucose concentration and

Dr. Xiaohong Yang, Department of Gynaecological Endocrinology, Xiaogan Hospital Affiliated to Wuhan University of Science and Technology, No.6 Plaza Street, Xiaogan, 432000, Hubei, China, tel: (+86) 712 234 86 33; e-mail: y285163361@163.com
Prof. Yi Zou, Department of Endocrinology, Xiaogan Hospital Affiliated to Wuhan University of Science and Technology, No. 6 Plaza Street, Xiaogan, 432000, Hubei, China, tel: (+86) 712 234 86 33; e-mail: zouyixg331@163.com

inhibition of glucose oxidation, resulting in increased lipid metabolism, which in turn causes in hyperlipidaemia, hyperinsulinaemia, hypercoagulable symptoms, and other clinical symptoms [9]. At the same time, studies have shown that 71.4% of non-survivors of COVID-19 met the significant disseminated intravascular coagulation grade (≥ 5, meeting the standards of the International Society on Thrombosis and Haemostasis) and showed a significant correlation between abnormal coagulation results and poor prognosis at the later stage of COVID-19 disease [10]. An association between COVID-19 and diabetes has been suggested, but there is currently very limited research on the association between COVID-19 and diabetes. Therefore, the focus of this present study was to explore the correlation between COVID-19 and diabetes mellitus in terms of clinical features and pulmonary CT features. In addition, the clinical indexes, inflammatory indexes, glucose and lipid metabolism, and detoxification time of patients with COVID-19 combined with diabetes were studied. This article aims to make up for the limitations of the study of COVID-19 patients with diabetes, and at the same time provide a certain theoretical basis and clinical guidance for the treatment of COVID-19 patients with diabetes.

Material and methods

General data collection

The patients' epidemiological data, medical history, contact history, symptoms and signs, laboratory examination, complications, clinical outcomes, CT imaging data, and treatment plan were extracted from electronic medical records. The date of onset of the disease was the date of the first symptom. The team analysed all the data, and it was double checked by two doctors. Nucleic acid detection was carried out by Xiaogan Central Hospital. At the time of admission, throat swab specimens were obtained from the patient's upper respiratory tract and stored in a virus transport medium. Total RNA was extracted within two hours using the RNA separation kit of respiratory tract samples. Suspected cases were selected according to the diagnostic criteria for COVID-19 pneumonia [11]:

1. Epidemiological history:
   - — travel or residence history in Wuhan or other areas with continuous transmission of local cases within 14 days before the onset of the COVID-19 disease;
   - — contact with patients with fever or respiratory symptoms from Wuhan or other areas where local cases continued to spread within 14 days before the onset of the COVID-19 disease;
   - — clusters of COVID-19 disease or epidemiological association with COVID-19 infection.
2. Clinical manifestations:
   - — fever;
   - — with the above-mentioned imaging characteristics of pneumonia;
   - — normal or decreased total number of white blood cells in the early stage of COVID-19 disease, or reduced lymphocyte count. Suspected cases could be diagnosed if any one of them had an epidemiological history and conformed to any two of the clinical manifestations.

Inclusion criteria for patients: 1 — suspected cases of COVID-19 pneumonia; 2 — COVID-19 nucleic acid RT-PCR detection of sputum, throat swabs, lower respiratory tract secretions, and other specimens were positive; 3 — diabetes was diagnosed according to the 1999 World Health Organisation (WHO) diagnostic criteria for diabetes [12]. This study was approved by the ethics committee of Xiaogan Central Hospital (ethics no.: XGLY2020-03-29) and conformed to the declaration of Helsinki.

Detection methods

After admission, fasting venous blood was collected in the morning and sent to the laboratory for blood routine examination and biochemical examination; the operation was carried out according to our laboratory instructions.

CT image collection

Two experienced physicians were employed to review the films, and they conducted quantitative accounting according to the distribution, location, size, morphology, edge, density, and pulmonary manifestations of the lesions.

Statistical analysis

Classification variables were expressed as frequency and percentage, continuous variables were expressed as average, and quantitative data of non-normal distribution were calculated by quartile. Chi-square test and Fisher exact test were used in the two groups of data, and t test or Mann-Whitney U test were used to analyse continuous variables. SPSS21.0 software was used for all statistical analysis. $P < 0.05$ was considered statistically significant.

Results

From January 16, 2020 to March 28, 2020, among the 568 cases of COVID-19 (data not shown) patients diagnosed in Xiaogan Central Hospital, 64 cases of COVID-19 patients with diabetes were selected as the diabetic group (hereinafter referred to as C-DM), and 64 cases of COVID-19 patients with age and gender matching without diabetes were selected as the non-diabetic group (hereinafter referred to as Non-C-DM) to study the clinical characteristics of patients with COVID-19 combined with diabetes mellitus – 128 cases in total. Clinical characteristics of C-DM and Non-C-DM cases were summarised in Table 1. In detail, among the 128 COVID-19 patients, the median age was 58 years [IQR (50-70)]; there were 74 males (57.81%) and 54 females (42.19%). The most common symptoms were fever (85.16%) and cough (71.09%); followed by dyspnoea (34.38%), fatigue (31.25%), and expectoration (25.78%); meanwhile diarrhoea (7.03%), myalgia (5.47%), dizziness (2.34%), sore throat (2.34%), nausea (1.56%), and conjunctival congestion (0.78%) were relatively rare. The vast majority of patients had a clear contact history, including Wuhan tourism history (23.44%), Wuhan residential history (10.16%), and contact history with confirmed patients (21.88%); the proportion of patients with non-clear contact history was 44.53%. Hypertension (39.06%) was the most common chronic disease, followed by cardiovascular disease (8.59%) and cerebrovascular disease (2.34%).

Table 1. *Clinical characteristics of patients with diabetes (C-DM) and without diabetes (Non-C-DM)*

Characteristics	All patients (n = 568)	Non-C-DM (n = 504)	C-DM (n = 64)	p value
Age (years) Median (IQR)	52 (43–63)	52 (42–62)	58 (50–70)	0.004
Age groups (years) n (%)				
≤ 39	107 (18.84)	101 (20.04)	6 (9.38)	0.040
40–49	119 (20.95)	111 (22.02)	8 (12.5)	0.078
50–59	158 (27.82)	137 (0.27)	21 (32.81)	0.344
60–69	80 (14.08)	67 (13.29)	13 (20.31)	0.128
≥ 70	104 (18.31)	88 (17.46)	16 (25.00)	0.142
Gender n (%)				
Male	309 (54.40)	272 (53.97)	37 (57.81)	0.561
Female	259 (45.60)	232 (46.03)	27 (42.19)	0.561
BMI [kg/m²]				
≤ 18.5	21 (3.70)	19 (3.77)	2 (3.13)	1.000
18.5 < BMI ≤ 24	288 (50.70)	265 (52.58)	23 (35.94)	0.012
24 < BMI ≤ 28	193 (33.98)	167 (33.13)	26 (40.63)	0.233
28 < BMI ≤ 32	52 (9.15)	42 (8.33)	10 (15.63)	0.057
> 32	8 (1.41)	7 (1.39)	1 (1.56)	1.000
Exposure history n (%)				
History of residence in Wuhan	60 (10.56)	56 (11.11)	4 (6.25)	0.329
Wuhan tourism history	126 (22.18)	115 (22.82)	11 (17.19)	0.307
Contact history with confirmed patients	139 (24.47)	122 (24.21)	17 (26.56)	0.680
Denied a clear contact history	243 (42.78)	211 (41.87)	32 (50.00)	0.215
Comorbidities n (%)				
Smoking	34 (5.99)	28 (5.56)	6 (9.38)	0.225
Hypertension	139 (24.47)	107 (21.23)	32 (50.00)	0.000
Cardiovascular disease	31 (5.46)	23 (4.56)	8 (12.50)	0.008
Cerebrovascular disease	14 (2.46)	11 (2.18)	3 (4.69)	0.430
Chronic pulmonary disease	23 (4.05)	20 (3.97)	3 (4.69)	1.000
Chronic kidney diseases	4 (0.70)	3 (0.60)	1 (1.56)	0.938
Chronic liver disease	22 (3.87)	22 (4.37)	0 (0)	N/A
Rheumatic immune diseases	6 (1.06)	6 (1.19)	0 (0)	N/A
Malignancies	17 (2.99)	12 (2.38)	5 (7.81)	0.016
Clinical symptoms n (%)				
Fever	507 (89.26)	453 (89.88)	54 (84.38)	0.180
Cough	392 (69.01)	339 (67.26)	53 (82.81)	0.011
Expectoration	144 (25.35)	124 (24.60)	20 (31.25)	0.250
Dyspnoea	202 (35.56)	175 (34.72)	27 (42.19)	0.240
Conjunctival congestion	1 (0.18)	0 (0)	1 (1.56)	N/A
Pharyngalgia	30 (5.28)	27 (5.36)	3 (4.69)	1.000
Dizziness	8 (1.41)	7 (1.39)	1 (1.56)	1.000
Myalgia	28 (4.93)	25 (4.96)	3 (4.69)	1.000

→

Table 1. *Clinical characteristics of patients with diabetes (C-DM) and without diabetes (Non-C-DM)*

Characteristics	All patients (n = 568)	Non-C-DM (n = 504)	C-DM (n = 64)	p value
Fatigue	185 (32.57)	164 (32.54)	21 (32.81)	0.965
Nausea or vomiting	14 (2.46)	12 (2.38)	2 (3.13)	1.000
Diarrhoea	28 (4.93)	24 (4.76)	4 (6.25)	0.832
Temperature ≥ 37.3°C	147 (25.88)	130 (25.79)	17 (26.56)	0.895
$SpO_2 \leq 93\%$	90 (15.85)	69 (13.69)	21 (32.81)	0.000
HR > 100	89 (15.67)	73 (14.48)	16 (25.0)	0.029

IQR — interquartile range; BMI — body mass index; SpO_2 — oxygen saturation; HR — heart rate

As shown in Table 1, compared with the Non-C-DM group, the number of patients with chronic underlying diseases complicated with hypertension in the C-DM group increased significantly (50.00% *vs.* 28.13%, $p < 0.05$). Cough symptoms were more common in the C-DM group (82.81% *vs.* 59.38%, $p < 0.05$). In addition, patients in the C-DM group were more likely to have decreased oxygen saturation and tachyarrhythmia at admission, in which oxygen saturation ≤ 93% (32.81% *vs.* 9.38%, $p < 0.05$) and heart rate > 100 bpm (25.00% *vs.* 10.94%, $p < 0.05$), indicating that patients in the C-DM group were in more a serious condition at admission.

Inflammation index and characteristics of glycolipid metabolism of C-DM and Non-C-DM cases are summarised in Table 2. In particular, in comparison

Table 2. *Laboratory test of patients with diabetes (C-DM) and without diabetes (Non-C-DM)*

	Normal range	All patients (n = 128)	Non-C-DM (n = 64)	C-DM (n = 64)	p value
Blood routine					
White blood cell count [$\times 10^9$/L]	3.5–9.5	5.07 (3.76–6.74)	4.64 (3.40–5.97)	5.65 (4.44–7.37)	0.006
Red blood cell count [$\times 10^{12}$/L]	3.92–5.61	4.39 (3.97–4.76)	4.33 (3.83–4.71)	4.47 (3.99–4.77)	0.563
Haemoglobin	115–150	137 (118–148)	138.00 (118.00–152.00)	135.50 (117.75–146.00)	0.335
Neutrophil [$\times 10^9$/L]	1.5–6.3	3.45 (2.43–4.94)	3.08 (2.03–4.10)	4.15 (2.75–5.86)	0.003
Lymphocyte [$\times 10^9$/L]	1.1–3.2	0.95 (0.70–1.38)	0.98 (0.73–1.42)	0.93 (0.64–1.32)	0.255
Platelets [$\times 10^9$/L]	125–350	166 (124–222)	153.00 (120.00–207.00)	187 (130.75–267.75)	0.053
Blood coagulation					
Active partial thrombin time (APTT)	23–45	31.00 (28.20–33.70)	31.85 (29.25–34.10)	30.50 (27.75–33.45)	0.140
PT	9–14	12.70 (11.90–13.70)	13.00 (12.23–13.78)	12.60 (11.75–13.65)	0.259
D-dimer (SDD)	0–1	0.30 (0.25–0.48)	0.28 (0.23–0.36)	0.34 (0.26–0.62)	0.007
TP	65–85	67.20 (63.70–72.63)	66.90 (63.70–72.45)	67.75 (63.73–72.73)	0.864
ALB	40–55	38.00 (35.18–40.73)	38.95(37.13–41.15)	36.80(34.83–39.63)	0.007
ALT	7–40	20.00 (13.00–33.25)	19.00 (12.75–34.25)	21.00 (14.00–32.25)	0.639
AST	13–35	24.00 (18.75–34.25)	24.50 (19.75–32.25)	24.00 (16.75–41.00)	0.888
Total bilirubin	0–23	12.05 (9.48–16.35)	12.50 (9.50–15.98)	11.95 (9.18–16.38)	0.884
Urea nitrogen	2.6–7.5	4.55 (3.30–5.83)	4.60 (3.50–5.60)	4.40 (3.10–6.45)	0.691
Creatinine	41–73	70.35 (60.15–85.25)	71.80 (62.98–86.78)	67.40 (56.03–84.18)	0.168
eGFR		94.74 (81.58–109.55)	93.55 (79.69–105.75)	102.58 (84.49–115.63)	0.076
LDH	120–250	245.50 (196.75–324.25)	226.50 (194.75–280.25)	271.50 (210.50–347.0)	0.019
ALP	40–150	69.00 (59.00–85.00)	69.00 (60.00–84.25)	67.00 (57.75–87.25)	0.924
FPG	3.89–6.11	6.19 (5.50–9.26)	5.58 (5.19–6.00)	8.85 (6.51–11.16)	0.000
TC	2.9–5.17	3.58 (2.93–4,18)	3.53 (2.93–4.13)	3.62 (2.95–4.25)	0.708
TG	0.23–1.7	1.33 (1.00–1.86)	1.11 (0.94–1.60)	1.54 (1.12–2.29)	0.000

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Table 2. *Laboratory test of patients with diabetes (C-DM) and without diabetes (Non-C-DM)*

	Normal range	All patients (n = 128)	Non-C-DM (n = 64)	C-DM (n = 64)	p value
Four items of chest pain					
CKMB	0–4	2.48 (1.70–3.70)	2.20 (1.74–3.11)	2.60 (1.66–3.85)	0.414
BNP	0–125	227.00 (93.50–740.75)	247.00 (158.50–652.50)	191.50 (54.75–820.50)	0.296
cTnI	0.04–0.5	0.06 (0.03–0.08)	0.06 (0.04–0.09)	0.06 (0.03–0.08)	0.670
Infection-related indicators					
PCT	0–0.5	0.17 (0.12–0.29)	0.14 (0.11–0.28)	0.19 (0.14–0.29)	0.096
CRP	0–3	19.61 (4.61–45.40)	15.90 (3.27–35.19)	24.43 (6.47–68.05)	0.029
ESR	0–20	39.50 (25.00–74.75)	29.00 (21.50–60.00)	55.00 (38.50–80.00)	0.045
Blood gas analysis					
pH	7.35–7.45	7.43 (7.40–7.47)	7.43 (7.42–7.47)	7.44 (7.40–7.46)	0.557
PO_2	83–108	80.15 (67.15–99.75)	84.40 (68.18–105.00)	74.75 (66.10–90.45)	0.793
PCO_2	35–45	38.05 (32.55–41.73)	39.25 (36.03–42.45)	36.30 (30.80–40.75)	0.166
LAC	0.5–1.6	1.55 (1.10–2.00)	1.20 (0.09–1.68)	1.80 (1.30–2.90)	0.112

APTT — active partial thrombin time; PT — prothrombin time; TP — total protein; ALB — albumin propagated; ALT — alanine aminotransferase; AST — glutamates transaminase; eGFR — estimated glomerular filtration rate; LDH — lactate dehydrogenase; ALP — alkaline phosphatase; FPG — fasting plasma glucose; TC — total cholesterol; TG — triglyceride; CKMB — creatinase myocardial band; BNP — N-terminal brain natriuretic peptide precursor; cTnI — cardiac troponin; PCT — procalcitonin; CRP — C-reactive protein; ESR — Erythrocyte sedimentation rate; PO_2 — oxygen partial pressure; PCO_2 — partial pressure of carbon dioxide LAC — lactic acid

to the Non-C-DM group, C-DM group white blood cells [5.65 IQR (4.44–7.37) *vs.* 4.64 (3.40–5.97)], PCT [0.19 IQR (0.14–0.29) *vs.* 0.14 (0.11–0.28)], CRP (24.43 IQR (6.47–68.05) *vs.* 15.90 (3.27–35.19)], ESR [55.00 IQR (38.50–80.00) *vs.* 29.00 (21.50–60.00)], SDD [0.34 IQR (0.26–0.62) *vs.* 0.28 (0.23–0.36)], FPG [8.85 IQR (6.51–11.16) *vs.* 5.58 (5.19–6.00)], and TG [3.62 IQR (2.95–4.25) *vs.* 3.53 (2.93–4.13)] increased, and albumin [36.80 IQR (34.83–39.63) *vs.* 38.95 (37.13–41.15)] decreased. These results suggested that COVID-19 patients with diabetes mellitus are more likely to have elevated inflammatory markers and hypercoagulability, accompanied by hypoproteinaemia and glucose and lipid metabolism disorders.

Treatment and clinic outcome of C-DM and Non-C-DM cases are summarised in Table 3. In detail, compared with the Non-C-DM group, the proportion of C-DM-group patients with respiratory failure (37.50% *vs.* 7.81%), requiring oxygen therapy (48.44% *vs.* 31.25%), and a non-invasive ventilator (17.19% *vs.* 3.13%) were significantly higher. Meanwhile, the C-DM group critical illness rate (28.13% *vs.* 3.13%) and the mortality rate (15.63% *vs.* 3.13%) were also significantly higher than those in the Non-C-DM group. The median length of stay for all patients was 20 days, IQR (14–25). The length of stay in the C-DM group [22 IQR (16–27) *vs.* 17 (13–23)] was also relatively long, and the time of nucleic acid turning negative in the C-DM group was significantly greater than that in Non-C-DM group [18 IQR (15–22) *vs.* 23 (18–30)].

Radiological data of C-DM and Non-C-DM cases are summarised in Table 4. Specifically, according to the distribution characteristics and the range of involvement of COVID-19 lung CT, we counted the imaging characteristics of lung CT in the first week of admission. Among all the patients, the proportion of lesions involving both lungs was 79.69%, the proportion of lesion distribution simultaneously involving peripheral and central areas was 50.78%, the proportion of lesion size of > 3 cm was 78.91%, the proportion of three or more lesions was 77.34%, and the proportion of lesion edge blur was 83.59%. In terms of lung CT lesion involvement location, compared with the Non-C-DM group (54.69% *vs.* 28.13%), the proportion of peripheral and central involvement was higher the C-DM group (62.50% *vs.* 39.06%). In terms of lung CT lesion morphology, the Non-C-DM group was mostly affected by plaque (57.81% *vs.* 25.00%), while the lung lobes of C-DM group were often affected (32.81% *vs.* 12.50%). More importantly, we calculated the proportion of pulmonary CT progression at the first week and found that the C-DM group had significantly higher progession than the Non-C-DM group (92.19% *vs.* 60.94%, $p < 0.001$), while the time of pulmonary CT absorption was significantly greater in the C-DM group than in the non-diabetic group [26 IQR (16–34) *vs.* 19 (15–23)], $p < 0.05$ (Fig. 1). This proved our hypothesis that COVID-19 combined with diabetes might prolong the time of detoxification in patients.

Table 3. *Treatment and clinical outcome of patients with diabetes (C-DM) and without diabetes (Non-C-DM)*

	Number of cases (n = 128)	Non-C-DM (n = 64)	C-DM (n = 64)	p value
Oxygen n (%)	51 (39.84)	20 (31.25)	31 (48.44)	0.047
Non-invasive ventilator n (%)	13 (10.16)	2 (3.13)	11 (17.19)	0.019
Invasive ventilator n (%)	7 (5.47)	1 (1.56)	6 (9.38)	0.120
Complications n (%)				
Respiratory failure	29 (22.66)	5 (7.81)	24 (37.50)	0.000
Heart failure	5 (3.91)	2 (3.13)	3 (4.69)	1.000
Septic shock	3 (2.34)	1 (1.56)	2 (3.13)	1.000
Clinical classification n (%)				
Ordinary	80 (62.50)	52 (81.25)	28 (43.75)	0.000
Heavy	28 (21.88)	10 (15.63)	18 (28.13)	0.087
Critical type	20 (15.63)	2 (3.13)	18 (28.13)	0.000
Clinical outcome n (%)				
Discharge	116 (90.63)	62 (96.88)	54 (84.38)	0.015
Death	12 (9.38)	2 (3.13)	10 (15.63)	0.034
Length of stay	20 (14–25)	17 (13–23)	22 (16–27)	0.029

Table 4. *Radiological data of patients with diabetes (C-DM) and without diabetes (Non-C-DM)*

		Number of cases (n = 128)	Non-C-DM (n = 64)	C-DM (n = 64)	p value
Lesion distribution	Left lung	8 (6.25)	7 (10.94)	1 (1.56)	0.068
	Right lung	8 (6.25)	4 (6.25)	4 (6.25)	1.000
	Double lung	102 (79.69)	49 (76.56)	53 (82.81)	0.380
Lesion location	Periphery	53 (41.41)	35 (54.69)	18 (28.13)	0.002
	Periphery and centre	65 (50.78)	25 (39.06)	40 (62.50)	0.008
Lesion size [cm]	< 1	7 (5.47)	6 (9.38)	1 (1.56)	0.120
	1~3	10 (7.81)	7 (10.94)	3 (4.69)	0.323
	> 3	101 (78.91)	47 (73.44)	54 (81.38)	0.129
Lesion form	Patch	53 (41.41)	37 (57.81)	16 (25.00)	0.000
	Lung segment	36 (28.13)	15 (23.44)	21 (32.81)	0.238
	Lobe	29 (22.66)	8 (12.50)	21 (32.81)	0.006
Number of lesions	1	13 (10.16)	8 (12.50)	5 (7.81)	0.380
	2	6 (4.69)	4 (6.25)	2 (3.13)	0.676
	3 or more	99 (77.34)	48 (75.00)	51 (79.69)	0.526
Lesion margin	Clear	10 (7.81)	6 (9.38)	4 (6.25)	0.742
	Vague	107 (83.59)	53 (82.81)	54 (81.38)	0.811
Lesion density	Ground glass	48 (37.50)	24 (37.50)	24 (37.50)	1.000
	Substantiality	3 (2.34)	2 (3.13)	1 (1.56)	1.000
	Mixed type	68 (53.13)	35 (54.69)	33 (51.56)	0.723
Extrapulmonary manifestations	Mediastinal lymphadenopathy	0	0	0	N/A
	Pneumothorax	0	0	0	N/A
	Pleural effusion	8 (6.25)	4 (6.25)	4 (6.25)	1.000

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Table 4. *Radiological data of patients with diabetes (C-DM) and without diabetes (Non-C-DM)*

	Number of cases (n = 128)	Non-C-DM (n = 64)	C-DM (n = 64)	p value
CT progress in the first week	98 (76.56)	39 (60.94)	59 (92.19)	0.000
CT absorption time	21 (16–29)	19 (15–23)	26 (16–34)	0.001

CT — computed tomography; N/A — not available

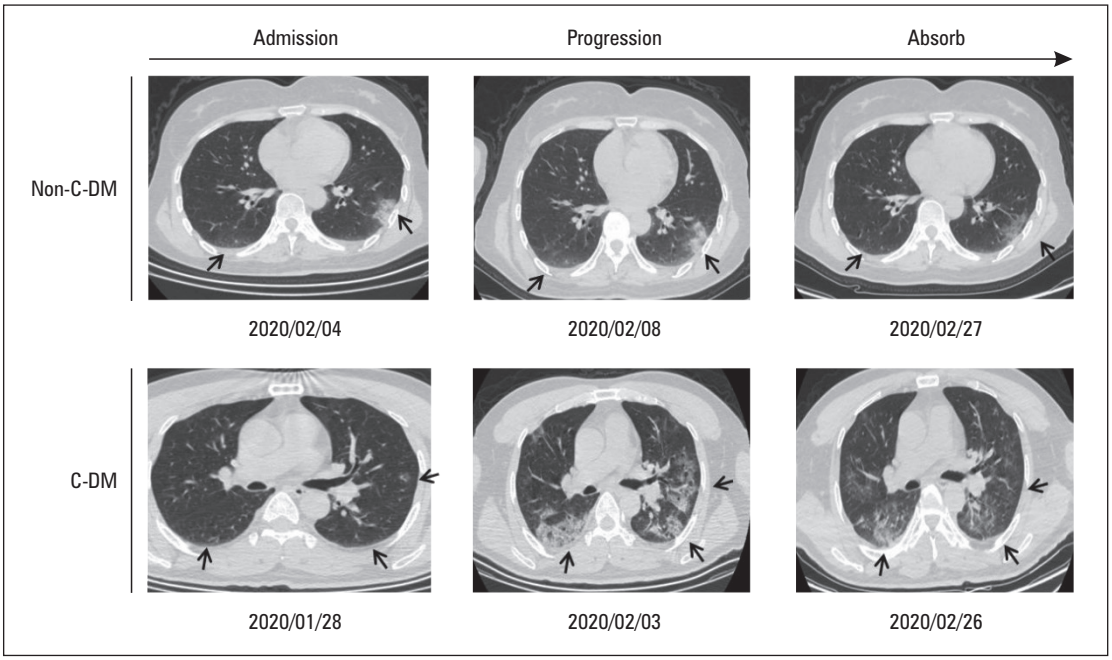

Figure 1. *CT imaging of progression and absorption of pulmonary lesions in patients with diabetes (C-DM) and without diabetes (Non-C-DM)*

Discussion

Coronaviruses are mostly round or oval in shape. They are named as coronaviruses because of their crown like appearance under an electron microscope. They can be divided into four genera: α, β, γ, and δ. COVID-19 belongs to the β genus, with a diameter of 60–140 nm. Coronaviruses are mainly caused by binding with receptors in patients [13]. Lu et al. found that the receptor binding domain (RBD) of COVID-19 was similar to that of SARS CoV, through the construction of a homologous structure model. Hoffmann et al. proved that the receptor of SARS CoV — angiotensin converting enzyme 2 (ACE2) was also the cellular receptor of COVID-19, and it needed the participation of cytoprotease TMPRSS2 to complete the invasion [14, 15]. The COVID-19 mainly destroyed alveoli and deep bronchial epithelial cells, and its pathological features were mainly inflammatory infiltration. These pathological features are consistent with our statistical laboratory examination results, clinical features, and CT imaging features.

According to the general situation of patients, we could observe that patients with COVID-19 complicated with diabetes mellitus were more likely to have unstable vital signs, decreased oxygen saturation, rapid heart rate, etc. The laboratory test results showed that patients with COVID-19 complicated with diabetes mellitus were more likely to have inflammatory reactions and hypercoagulable blood, and most of them were in severe condition at the time of admission. Previous studies also suggested that after COVID-19 virus invasion of patients, the results of laboratory examination were mostly showed the decrease of peripheral lymphocytes and the passive activation of the immune system [16]. This was consistent with the statistical results in this present study. SARS-related studies have shown that blood glucose fluctuations play a positive role in promoting the generation of inflammatory storms and the poor prognosis of clinical outcomes [17]. Patients with diabetes mellitus complicated with COVID-19 can be hyperglycaemic due to improper diet, irregular use of hypoglycaemic

drugs, and glucocorticoid drugs and other factors that affect blood sugar. Hyperglycaemia can further reduce lymphocytes, thereby causing overactivation of the immune system, while excessive activation and improper activation of the epidemic free system can cause inflammatory storm, which is often the reason for severe clinical symptoms [18]. Some studies also suggest that blood glucose can be increased after COVID-19 virus invasion, and high blood glucose level is a high risk factor for disease progression. People with diabetes are more likely to be infected with COVID-19 virus than people without diabetes because of their immune deficiency. When the virus invades, it mainly acts on the complement system. Complement immunity is one of the main mechanisms of humoral immunity. The complement system activates and mediates the production of antibodies at the time of the virus invading the body. However, in diabetic patients with immunodeficiency, the response to complement activation is decreased, and glycosylation can affect the expression of receptors in the complement activation system, resulting in diabetic patients being more susceptible to infection, and they are prone to change from mild patients to severe patients, with a higher mortality rate [19]. This was also consistent with the clinical outcome of this study that patients with COVID-19 complicated with diabetes mellitus were more likely to develop respiratory failure and have a higher mortality rate than those without diabetes mellitus.

Compared with Non-C-DM, C-DM patients presented more lung segment and lobe infections on CT imaging. The area around the lesion was blurred and the boundaries were unclear. Lesions were found in the centre and around the lobes of both lungs, with a wide range, indicating severe lesions in C-DM patients. Studies found that COVID-19 mainly destroyed alveoli and deep bronchial epithelial cells, and its pathological features were mainly inflammatory infiltration. Under electron microscopy, tracheal epithelial cells were swollen, local cilia disappeared, and diffuse alveolar destruction was observed. COVID-19 caused exudation of high protein fluid, formation of a clear membrane, obvious monocyte infiltration, bronchiole were filled with cell debris, and there was apparent alveolar collapse with haemorrhage [20, 21]. The pathological characteristics were similar to those caused by SARS and Mars coronavirus, but the degree of fibrosis and consolidation was weaker than that caused by SARS, which was consistent with the characteristics of our CT imaging statistics in this study. Most of the CT imaging of COVID-19 patients are cumulative double lung, with lesions distributed in and around the centre of both lungs, and the lesion range is more than 3 cm.

According to the early autopsy results, the deep airway of the deceased patient was blocked by a large number of sputum plugs, which was consistent with the clinical manifestations [22]. Early studies confirmed that respiratory failure and circulatory failure were the main causes of death in COVID-19 patients [23]. The statistical results of this study showed that the proportion of C-DM patients complicated with respiratory failure during hospitalisation was higher than that of Non-C-DM and was statistically significant. In addition, it could also be found that C-DM patients had more severe CT imaging manifestations and a wide range of lesions, consistent with the clinical outcome.

Conclusion

To sum up, we reported 128 confirmed patients with COVID-19 infection and explored the effect of diabetes on COVID-19 patients based on clinical indicators and CT imaging features. We have provided a theoretical basis and clinical guidance for the treatment of patients with COVID-19 complicated with diabetes mellitus.

Authors' contributions

Y.Y and F.Y. contributed equally to the work.

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