



Research Article

Diabetes and COVID-19: Biological profile of type 2 diabetic patients with COVID-19 in Pointe-Noire, Congo

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Abstract

Introduction: COVID-19 is new pneumonia caused by SARS-CoV-2 infection. Several factors of bad prognosis have been implicated including diabetes. Several poor prognostic factors have been associated with the severity of this disease, including diabetes. In Congo, no study to date has investigated the profile of diabetics hospitalized for COVID-19. The aim of this study was to evaluate the biological profile of Congolese diabetic patients admitted to the hospital for COVID-19 in Pointe-Noire.

Materials and methods: This was a prospective study conducted among patients hospitalized for COVID-19 in Pointe-Noire between March and November 2020. Sociodemographic, clinical and biological data, duration of hospitalization, and viral load were investigated. A total of 84 COVID-19 patients have been admitted to Louise Michel Clinic and the Adolph Sice General Public Hospital. Two groups were formed: diabetic patients and non-diabetic patients based on the history of antidiabetic medication or fasting plasma blood glucose levels at admission. Results between the two groups were compared.

Results: Out of 84 COVID-19 patients, 48 were diabetic (mean age: 48.50 ± 11.98 years) versus 36 non-diabetic (mean age: 45.56 ± 8.48 years). Significant increases in fasting blood glucose, D-dimers, white blood cells, low oxygen saturation (SaPO_2), and higher mortality was observed in COVID-19-positive diabetics when compared to non-diabetic patients ($p < 0.02$). However, no significant differences were observed between the two groups in terms of clinical symptoms.

An increased risk of death was associated with higher levels of D-dimers and HbA1c at admission in the diabetic group.

Conclusion: An increase in D-dimer levels and high blood glucose levels at admission increased the risk of death in diabetic patients with COVID-19 in Pointe-Noire.



Introduction

In December 2019, an outbreak of pneumonia due to novel coronavirus 2019, called SARS-CoV-2 (severe acute respiratory syndrome coronavirus 2) was declared in Wuhan, Hubei, China [1]. This *betacoronavirus* causes sometimes severe respiratory pathology named COVID-19 by the World Health Organization (WHO). On 12 March 2020, WHO declared COVID-19 a pandemic [2,3]. Indeed, the USA, India, Brazil, Russia, Italy, and France were the most affected countries in the world. On November 16, 2020, the number of patients suffering from COVID-19 was 55,130,249 and 1,328,881 of them died [2,3]. The mortality rate of COVID-19 varies according to geographical areas. The overall mortality rate is about 2.3%. It reaches 8.0% to 14.8% among patients aged 70 to 79 years [2,3]. In Congo the first case of COVID-19 has been reported on 14 March 2020. The country officially registered 5515 cases and 92 (1.7%) deaths on November 11, 2020 [4]. COVID-19 response in Congo was based on a set of elements including the notion of contact with a suspected/confirmed COVID-19 case, virological test results, and presence of suggestive clinical and radiological signs, and strict compliance with the barrier measures enacted by the government [5,6].

Diabetes is one of the main causes of morbidity and mortality worldwide. This disease is associated with several macrovascular and microvascular complications, which ultimately impact the patient's overall survival [7].

A clinical relationship between diabetes and COVID-19 has been recognized since then [8]. Infections, especially influenza, and pneumonia are often common and more severe in older adults with type 2 diabetes (T2D) [9-11]. Nevertheless, evidence remains controversial as to whether diabetes itself increases susceptibility to COVID-19 severity or whether cardiovascular and renal comorbidities frequently associated with diabetes are the main factors involved [8].

During the SARS-CoV-2 pandemic, a high rate of diabetic patients died from COVID-19. It is with this regard that the authors proposed to study the biological profile of moderate and severe forms of diabetic patients suffering from COVID-19 in Pointe-Noire, to contribute to better patient care in Congo.

Materials and methods

Study population

This was a cross-sectional descriptive study. Data were collected prospectively between March and November 2020. The population of the study included any consenting person admitted for COVID-19 confirmed by RT-PCR test at the Adolphe Sicé General Public Hospital and Louise Michel Clinic in Pointe-Noire. A total of 84 COVID-19 patients were gathered and divided into two comparable groups: 48 diabetic patients (T2D/COVID-19+) and 36 non-diabetic patients (NDT2/COVID-19+). The absence of measurement of SpO₂ at admission was applied as a non-inclusion criterion.

Socio-demographic and clinical data

Clinical data including age (year), gender, active smoking,

the notion of contact with a suspected or confirmed case of COVID-19, diagnostic time (between symptoms appearance and hospitalization dates), length of hospitalization, and medical history of patients were collected using pre-established observation and patient's medical record. At admission, SpO₂ was determined at rest and in-room air using pulse oximeter brand "Bedside monitor, Nihon Kohden Corporation Model BSM3562, Japan". The basic treatment was the combination "Chloroquine-Azithromycin-Aluvia. The number of patients requiring oxygen therapy was noted.

Biological data

A blood sample was collected to determine a standard biological assessment including the following data : complete blood count (CBC), (hemoglobin (g/dl), leukocytes (/mm³), leukocyte formula (/mm³) [neutrophil, eosinophil and basophil polymorphonuclear, lymphocytes and monocytes], platelets (/mm³); CRP (mg/L); sedimentation rate at first hour (VS, mm); renal function (creatinine (mg/l)); hepatic function (transaminases (IU/L)); Blood ionogram (kalemia and natremia, (mmol/l)); fasting blood glucose (g/L); HBA1C (%) and D-dimers (µg/L). All blood sample collection was performed at the elbow folds after a short installation of the withers either in a dry tube or in an EDTA tube. All tests were performed in duplicate with the same kit batch after centrifugation at 4000rpm for 5 min. The automatic Biochemistry analyzer «Cobas C 311 (Roche Diagnostics, HITACHI, Germany)» was used for biochemical assays. A blood Count was performed using the SYSMEX XT Instrument.

Tests RT-PCR du SARS-CoV-2

Nasopharyngeal samples were collected using swabs at patient admission in COVID-19 patient care units. Identification of SARS-CoV-2 genomic material was performed at HDL Molecular Biology Laboratory of Marie Madeleine GOMBES Foundation. We used "NORGEN BIOTEK total RNA purification KIT, CANADA" for extraction according to the recommendation of the manufacturer. Molecular amplification was performed using the thermocycler "MIC qPCR" able to detect 500 copies/ml when using the NORGEN BIOTEK COVID TAQ MAN RTPCR KIT AMPLIFICATION KIT, CANADA (E-RdRP genes).

Ethical approval

This study was conducted by the guidelines of the Declaration of Helsinki and was approved by the Ethics Comity in Health Research (ECRB) of the Marie Madeleine GOMBES Foundation in Pointe-Noire. All patients gave informed consent.

Statistical analysis

Data were analyzed using SPSS 12.0 (SPSS Inc., Chicago, IL, USA). The results are presented in percentage and average ± Standard Deviation. An exact Fischer test was used to compare the categorical variables. Unpaired T-tests and analysis of variance (ANOVA) were used to compare data normally distributed across study groups. The 95% confidence interval (CI) was calculated. P-values below 0.05 were considered significant.



Results

Study population, socio-demographic and clinical characteristics

The study involved 84 hospitalized COVID-19 patients with 66.67% (56) men and 33.33% (28) women aged from 24 to 67 years (47.24 ± 9.95 years). This study population was divided into two groups: 48 type 2 diabetic patients positive for COVID-19 (T2D/COVID19+; Average age: 48.50 ± 11.98 years) and 36 covid-19 positive non-diabetic patients (NDT2/COVID-19+; mean age: 45.56 ± 8.48 years).

The appearance of first symptoms at hospital admission in T2D/COVID19+ patients was 10 days versus 12 days in NDT2/COVID-19 + patients.

The length of hospital stay was similar for both groups (7–9 days). Out of 84 patients, fever, cough, dyspnea, and fatigue were the most common symptoms, while diarrhea, vomiting, and dizziness were rare. However, no statistically significant differences in clinical symptoms were observed between the two groups. All the results are reported in Table 1.

Biological profile

The most common biological abnormalities were: increased sedimentation rate (100%); anemia (23.8%); Increased CRP (95.2%); Increased D-dimers (71.4%); Hyperglycemia (47.6%), hepatic cytolysis (47.6%) basocytosis (52.4%) (Table 2).

Biological profile and mortality

Regarding mortality, a statistically significant difference was observed between the two groups ($p = 0.036$), with 6 (83%) deaths in T2D/COVID19+ compared to two in NDT2/COVID-19+ (27%). Diabetes appears to increase the risk of death in patients with COVID-19 pneumonia based on the following biological

Table 2: Biological profile of diabetic and non-diabetic patients with COVID-19.

Characteristics	DT2/COVID19+ n = 48	NDT2/COVID19+ n = 36	P value
CRP (mg/l)	251.48 ± 118.90	124.98 ± 127.08	0.34
D dimers (µg/L)	4275.83 ± 3200.54	1517.78 ± 2433.30	0.02
Blood glucose (g/L)	2.63 ± 2.45	0.83 ± 0.10	0.07
HBA1C (%)	8.25 ± 2.45	4.77 ± 0.44	0.02
Created (mg/l)	20.70 ± 18.38	15.05 ± 10.29	0.54
Hb (g/ml)	13026 ± 1.92	12.98 ± 2.00	0.53
WBC(M/mm ³)	15315.0 ± 9388.76	8681.11 ± 5779.01	0.41
PNN(M/mm ³)	12937.50 ± 9197.85	6327.56 ± 5403.67	0.42
PNB(M/mm ³)	33.33 ± 36.33	35.44 ± 26.50	0.66
PNE(M/mm ³)	37.17 ± 65.90	29.89 ± 41.83	0.22
Lymphocytes(M/mm ³)	1637.08 ± 620.08	1538.42 ± 861.19	0.4
Monocytes(M/mm ³)	546.93 ± 227.95	715.11 ± 233.13	0.16
Platelets(M/mm ³)	250583.33 ± 145777.82	175222.22 ± 57727.32	0.12
VS (mm/H)	70.08 ± 47.85	62.44 ± 45.86	0.16
GOT(UI/L)	53.35 ± 35.46	55.65 ± 30.62	0.8
GPT(UI/L)	52.65 ± 50.03	49.58 ± 24.90	0.92
Na(mEq/L)	141.83 ± 5.57	141.55 ± 2.65	0.94
K(mEq/L)	4.88 ± 0.59	4.26 ± 1.85	0.013
Cl(mEq/L)	100.80 ± 2.96	100.26 ± 1.85	0.76
SpO ₂ (%)	89.25 ± 4.50	90.89 ± 3.20	0.37
Ct E-gene	21.87 ± 5.19	26.67 ± 6.48	0.07

Unless otherwise indicated, all parameters are expressed as an average ± standard deviation. PNN: Neutrophil polynuclear; PNE: Eosinophilic polynuclear; PNB: Neutrophil polynuclear; CRP: C-reactive protein; HBA1C: Glycated hemoglobin; VS: Sedimentation rate; SpO₂: Partial Oxygen Saturation; K: Kalemia; Cl: Chlorine; Na: Natreemia; GOT: Glutamate oxalic pyruvate; GPT: Glutamate pyruvate transaminases; Ct E-gene: Cut of target E SARSCOV2 gene; DT2COVID-19+: Type 2 diabetic patient with COVID-19; NDT2COVID-19+: COVID-19 patient

Table 1: Socio-demographic and clinical characteristics of patients.

Parameters	DT2/COVID19+	NDT2/COVID19+
	(n = 48)	(n = 36)
Socio-demographic characteristics		
Age (years)	48.50±11.98	45.56±8.48
<50	28	22
≥50	20	12
Sex		
M	30	26
F	18	10
Clinical features		
Hospitalization time (days)	12.00±2.00	10.00±2.0
Fever	97.91	94.4
Cough	79.16	44.44
Dyspnea	81.25	63.88
Fatigue	43.75	44.44
Vomit	18.75	16.66
Dizziness	4.16	2.77
Smoking	4.16	5.55
SPO2	89.25±4.50	90.89±3.20
Notion of contact	43.75	80.55
Diagnostic delay	3	5
Case fatality rate	8.33	2.77

parameters as factors of poor prognosis. The mean values of the biological parameters of patients DT2/COVID-19+ and NDT2/COVID-1+ are reported in Tables 3,4.

Discussion

Because of its pandemic nature, COVID-19 has received great attention compared to other causes of pneumonia around the world. Known history of diabetes has been reported to be independent predictor of morbidity and mortality in SARS-CoV-2 patients [12]. The objective of this work was to determine the biological profile of type 2 diabetic patients with COVID-19 in Pointe-Noire.

In this study, 84 patients with SARS-CoV-2 infection were biologically investigated. Two groups were formed, one diabetic and COVID-19+ patients, the other of the non-diabetic COVID-19+ patients. Clinical and biological data from both groups were compared.

The average age of patients in both groups was substantially superimposable. Several authors have reported during SARS pneumonia or MERS that older age is an independent predictor of mortality [13,14].



Male was predominant in our study compared to female with a sex ratio (M/F) of 2.0. These data match with several studies conducted around the world [15,16]. This male predominance would be due to the high frequency of risk factors for COVID-19 severity such as smoking in the male population compared to the female population [17].

Both groups showed the same general clinical signs such as fever, cough, dyspnea, fatigue, vomiting, and OPS2. Hospitalization time was a bit longer in diabetic COVID-19 patients than in non-diabetic COVID-19 patients. However, a clear difference was observed between the two groups regarding the case fatality rate, which is very high in COVID-19 diabetic patients. These observations have been reported by almost all authors on the subject [18-21].

These results support the hypothesis that diabetes is perceived as an aggravating factor in mortality in cases of SARS viral pneumonia [22,23].

In our study, the biological profile had high blood sugar, D dimers and HbA1c, kalemia, and high viral expression of the SARS-Cov-2 E gene. A statistically significant difference was observed between the two groups on these parameters ($p <$

Table 3: Biological profile of patients who die versus recover from T2D/COVID-19+.

Biological parameters	T2D/COVID-19 n = 48		P value
	Died n = 6	Cured n = 42	
CRP (mg/l)	312.54 ± 108.17	161.25 ± 124.63	0.14
D dimers (µg/L)	7324.00 ± 2227.97	1771.88 ± 2031.07	0.02
Blood glucose (g/L)	4.63 ± 0.46	1.69 ± 0.60	0.001
HBA1C (%)	9.83 ± 0.83	7.75 ± 0.25	0.0001
Creatinine (mg/l)	29.38 ± 26.36	14.81 ± 8.72	0.33
Hb (g/ml)	11.80 ± 2.06	13.56 ± 1.72	0.16
WBC (M/mm ³)	15854.0 ± 9981.91	11415.0 ± 8122.07	0.35
PNN (/ mm ³)	13641.80 ± 10040.26	8999.31 ± 7761.30	0.33
PNB (/ mm ³)	19.80 ± 11.41	38.75 ± 34.90	0.16
PNE (/ mm ³)	45.40 ± 76.50	30.50 ± 50.20	0.38
Lymphocytes (/ mm ³)	1507.80 ± 602.50	1621.99 ± 762.06	0.6
Monocytes (/ mm ³)	630.80 ± 162.98	615.32 ± 263.90	0.72
Platelets (/ mm ³)	315000.00 ± 150258.11	188062.50 ± 95894.71	0.12
VS (mm/H)	85.60 ± 55.32	60.93 ± 43.01	0.21
GOT (UI/L)	55.85 ± 16.20	60.15 ± 19.23	0.02
GPT (UI/L)	61.35 ± 28.71	53.15 ± 8.35	0.005
Na (mEq/L)	140.20 ± 3.56	142.18 ± 4.70	0.35
K (mEq/L)	5.01 ± 0.58	4.49 ± 0.59	0.11
Cl (mEq/L)	99.22 ± 0.97	101.00 ± 8.72	0.33
SpO2 (%)	90.8 ± 6.4	92.6 ± 2.9	0.17
Ct E-gene	17.8 ± 3.1	25.4 ± 5.58	0.004

Unless otherwise indicated. all parameters are expressed as an average ± standard deviation. PNN: Neutrophil polynuclear; PNE: Eosinophilic polynuclear; PNN: Neutrophil polynuclear; CRP: C-reactive protein; HBA1C: Glycated hemoglobin; VS: Sedimentation rate; SpO₂: Partial Oxygen Saturation; K: Kalemia; Cl: Chlorine; Na: Natremia; GOT: Glutamate oxalo pyruvate; GPT: Glutamate pyruvate transaminases; Ct E-gene: Cut of target E SARS-CoV2 gene; DT2COVID19+: Type 2 diabetic patient with COVID19; NDT2COVID19+: COVID19 patient

Table 4: Biological profile of patients who die versus have recovered from NDT2/ COVID-19+

Biological parameters	NDT2/COVID-19 +		P value
	Died n = 2	Cured n = 35	
CRP (mg/l)	112.54 ± 108.17	91.25 ± 124.63	0.84
D dimers (µg/L)	7324.00 ± 2227.97	1771.88 ± 2031.07	0.02
Blood glucose (g/L)	0.83 ± 0.46	0.90 ± 0.30	0.67
HBA1C (%)	4.83 ± 0.83	4.75 ± 0.25	0.87
Created(mg/l)	29.38 ± 26.36	14.81 ± 8.72	0.33
Hb (g/ml)	11.80 ± 2.06	13.56 ± 1.72	0.16
WBC (M/mm ³)	15854.0 ± 9981.91	11415.0 ± 8122.07	0.35
PNN (/ mm ³)	13641.80 ± 10040.26	8999.31 ± 7761.30	0.33
PNB (/ mm ³)	19.80 ± 11.41	38.75 ± 34.90	0.16
PNE (/ mm ³)	45.40 ± 76.50	30.50 ± 50.20	0.38
Lymphocytes (/ mm ³)	1507.80 ± 602.50	1621.99 ± 762.06	0.6
Monocytes (/ mm ³)	630.80 ± 162.98	615.32 ± 263.90	0.72
Platelets (/ mm ³)	315000.00 ± 150258.11	188062.50 ± 95894.71	0.12
VS (mm/H)	85.60 ± 55.32	60.93 ± 43.01	0.21
GOT (UI/L)	55.85 ± 16.20	60.15 ± 19.23	0.85
GPT (UI/L)	61.35 ± 28.71	53.15 ± 8.35	0.74
Na (mEq/L)	140.20 ± 3.56	142.18 ± 4.70	0.35
K (mEq/L)	5.01 ± 0.58	4.49 ± 0.59	0.11
Cl (mEq/L)	99.22 ± 0.97	101.00 ± 8.72	0.33
SpO2 (%)	.8 ± 6.4	90.6 ± 2.9	0.17
Ct E-gene	19.8 ± 3.1	25.4 ± 5.58	0.007

Unless otherwise stated, all parameters are expressed as an average ± standard deviation, PNN: Neutrophil polynuclear; PNE: Eosinophilic polynuclear; PNN: Neutrophil polynuclear; CRP: C-reactive protein; HBA1C: Glycated hemoglobin; VS: Sedimentation rate; SpO₂: Partial Oxygen Saturation; K: Kalemia; Cl: Chlorine; Na: Natremia; GOT: Glutamate oxalo pyruvate; GPT: Glutamate pyruvate transaminases; Ct E-gene: Cut of target E SARS-CoV-2 gene; DT2 COVID-19+: Type 2 diabetic patient with COVID-19; NDT2 COVID-19+: COVID-19 patient.

0.05). In their studies, Guozhe Li, et al. (2020) and D Wang, et al. (2020) reported the same observations as the present study [18,19].” Indeed, the hypersecretion of secondary endogenous glucocorticoids in the context of stress induced by infection or the use of corticosteroids for therapeutic purposes on one hand, and the potential viral damage to the function of pancreatic cells β on the other hand could explain the hyperglycemia and augmentation of HbA1c observed [17,20]. The net elevation of D-dimers ($p < 0.02$) in the diabetic COVID-19 group was also observed by Guozhe Li, et al, [16] Abdelbassat Ketfi, et al. [18]. It is accepted that D-dimers are a marker of fibrinolysis activation and an important prognostic factor in patients with pneumonia or sepsis [20].

Heart damage caused by SARS-CoV-2 infection in diabetic patients would be the basis for the increase in serum potassium rate in our study. The same observations have been reported by other authors in the literature [18,19].

The low Ct levels observed in COVID-19 diabetic patients suggested high expression of the SARS COV2 E gene, which corresponded to a high viremia rate explaining the significant inflammation observed in this group [20].



In terms of lethality, six patients with COVID-19 pneumonia and diabetes died against two in the non-diabetic and COVID-19 patients' group ($P = 0.086$). Our data are in compliance with those obtained by Paquot, et al. (2020); and Cariou, et al. (2021); note that the type 2 diabetic patient was at greater risk of death compared to the non-diabetic patients [18-22].” Several scientific arguments can also explain these results: i) First, there are mechanisms related to general characteristics of T2D including obesity which alters the ventilatory performance of patients [23], ii) There is also the pro-inflammatory character of adipose tissue which would play a role in the severity of SARS-CoV-2 infection in T2D patients [24].

Hyperglycemia also plays a role in the severity of pathology in T2D patients with COVID-19. Indeed, infection of SARS-CoV-2 would induce a reduction in the membrane expression of ACE2 thus leading to a decrease in insulin secretion. This decrease in insulin secretion aggravates insulin resistance reinforcing the hypothesis of a specific SARS-CoV-2 action on insulin-sensitive tissues to develop a severe form of COVID-19. The hypothesis suggests that diabetes is not only a severe form of COVID-19 but COVID-19 infection could itself induce new cases of diabetes [24-26].

In sum, in this work, diabetes was associated with an increased risk of death in COVID-19 patients in Pointe-Noire.

Conclusion

The biological profile of COVID-19 patients with diabetes showed that hyperglycemia, elevated D-Dimers, and high expression of the SARS-CoV-2 virus were factors of poor prognosis during COVID-19 disease in Pointe-Noire. Further studies are needed to establish a general biological profile of these patients throughout the national territory.

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