

PROFILING COMORBIDITIES IN COVID-19 PATIENTS: RETROSPECTIVE EVIDENCE FROM THE PANDEMIC

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Abstract

Objectives. This retrospective study aims to characterize demographics, comorbidities, clinical-laboratory features and outcomes of COVID-19 patients admitted to the Infectious Diseases Service during the 2020 pandemic period.

Material and methods. A total of 1001 patients with confirmed COVID-19 (via RT-PCR) admitted between March 9 and December 31, 2020, were included. Demographic, clinical, laboratory, and imaging data were collected from individual medical charts obtained from the hospital Statistics Service. Data were analyzed using SPSS 26.0. Categorical variables were expressed as counts and percentages, and continuous variables as means, *SD*, or *Mdn*. Chi-square tests and multivariate analyses were applied where appropriate.

Results. The study population comprised 65% men and 35% women, with an average (mean) age of 60.4 years (*SD* = 13.9). The most common comorbidity was arterial hypertension (45.3%). The average temperature on admission was 37.2 °C (*SD* = 0.8), and the average PaO₂ was 72.3 mmHg (*SD* = 23.1). Severe lung injury consistent with acute respiratory distress syndrome (ARDS) was observed in 43.9% of patients. Additionally, 15.1% of patients required intubation, and mortality in this subgroup was 100%. C-reactive protein (CRP) values decreased by the eighth day of hospitalization.

Conclusions. In this cohort, men represented the majority of COVID-19 cases, and arterial hypertension was the most prevalent comorbidity. A significant proportion of patients experienced severe respiratory impairment, and CRP levels declined over the first week of hospitalization. These findings provide insights into the clinical characteristics and outcomes of COVID-19 patients in a tertiary hospital setting and highlight the importance of monitoring comorbidities and laboratory markers in disease management.

Keywords: COVID-19; C-reactive protein (CRP); acute respiratory distress syndrome (ARDS); COVID-19 Reporting and Data System (CO-RADS)

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Introduction

Coronavirus disease 2019 (COVID-19), resulting from infection with severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), has triggered a global outbreak and a major public health crisis, resulting in over six million deaths worldwide (Casella et al., 2023).

COVID-19 mainly spreads via respiratory secretions and aerosols released during respiratory actions such as coughing or sneezing, which can settle on the face, including the eyes, nose, and mouth (Zhou et al., 2021).

Many individuals infected with SARS-CoV-2 tend to show only mild or moderate symptoms and typically get better without needing advanced medical intervention (World Health Organization [WHO], 2025).

COVID-19 patients who have comorbidities such as cardiovascular disease, high blood pressure, obesity, chronic lung disease, and diabetes mellitus are associated with poor prognosis and frequently experience adverse outcomes such as pneumonia and severe respiratory failure compared to patients without underlying comorbidities. (Djagaruddin et al., 2021).

Being 65 years or older, male sex, and an elevated Charlson Comorbidity Index (CCI) are identified as significant standalone predictors of death and severe respiratory failure in COVID-19 cases (Sundaram et al., 2022).

An analysis of confirmed COVID-19 cases reported to the CDC between January 22 and May 30, 2020, showed that individuals with underlying medical conditions were hospitalized at a rate nearly six times higher than those without such conditions (45.4% vs. 7.6%) (Aleem et al., 2023).

“While men and women have the same prevalence of COVID-19, men are at higher risk of worse outcomes and death, independent of age” (Jin et al., 2020, para. 4).

The COVID-19 pandemic has placed tremendous pressure on healthcare systems worldwide and, at the same time, has revealed deep-rooted social inequalities and shaped patients’ everyday experiences. While biomedical and clinical research is essential to understand the mechanisms of the disease and its potential long-term organ effects, a full understanding of patient outcomes also requires examining social, economic, and structural factors. Anthropological and public-health research highlights that what is defined as illness, how it is experienced, and who can access care are all influenced by historical social norms, power dynamics, and systemic inequities (Hahn & Schoch-Spana, 2021; Vahedi et al., 2023).

Moreover, the pandemic must be conceptualized as what social theorists term a “total social fact”: a phenomenon that permeates all dimensions of human life, including medical, economic, cultural, psychological, and institutional spheres (Labora González & Fernández-Vilas, 2024).

In this light, the experience of patients with COVID-19 is embedded in broader patterns of social vulnerability, structural violence, and inequality, factors that influence not only infection risk and clinical outcomes but also access to care, psychosocial stress, stigma, and long-term wellbeing. Indeed, qualitative research has documented how, in contexts such as Brazil, the pandemic exacerbated structural violence and deepened gender-based violence, food insecurity, and social marginalization among particularly vulnerable groups (Vahedi et al., 2023).

From a psychological-anthropological perspective, community responses to COVID-19, including adherence to public-health measures, trust in medical institutions, and coping strategies, are profoundly influenced by cultural norms, social trust, historical inequalities, and collective memory (Pillay, 2021).

This calls for a “social medicine” response: one that does not treat COVID-19 solely as a biomedical event, but as a social crisis demanding integrated interventions attentive to equity, social support, and structural determinants of health (Bambra et al., 2020).

Therefore, to study patients affected by COVID-19, especially in a context of limited resources, social stratification, or health disparities, research must go beyond a retrospective analysis of clinical data.

SARS-CoV-2 genetic structure

According to Satarker and Nampoothiri (2020), the SARS-CoV-2 virus possesses "a positive viral RNA genome expressing open reading frames that code for structural and non-structural proteins. The structural proteins include spike (S), nucleocapsid (N), membrane (M), and envelope (E) proteins" (p. 482). The authors further detail that within the first open reading frames (ORF1a/b), "about two-thirds of viral RNA is present that encodes for polyprotein 1a and polyprotein 1b and 1–16 non-structural protein" (p. 484), while the rest of the genome handles the structural and accessory proteins.

Pathophysiology

Novel coronavirus infects host cells by engaging the angiotensin-converting enzyme 2 (ACE2) receptor with its spike glycoprotein, which is commonly found in respiratory epithelial cells and pneumocytes. Its significantly increased ACE2 binding efficiency compared to SARS-CoV may explain the virus's rapid transmission. The presence of protease in lung cells, known as type 2 transmembrane serine protease (TMPRSS2), helps activate the spike protein to enable cell entry, but the restricted presence of ACE2 could be the key factor limiting early viral infection (Zhu et al., 2023).

High concentrations of cytokines such as granulocyte colony-stimulating factor (G-CSF), interferon gamma (IFN- γ), along with interleukins (IL) such as IL-6, IL-7, and IL-10, interferon gamma-induced protein 10 (IP-10), monocyte chemoattractant protein-1 (MCP-1), tumor necrosis factor (TNF), and macrophage inflammatory protein-1 α (MIP-1 α), have been linked to increased COVID-19 severity, suggesting activation of a T-helper type 1 (Th1) and T-helper type 2 (Th2) cell response (Bohn et al., 2020).

Dysfunction in the adaptive immune system alongside excessive activation of the innate inflammatory response may contribute to the development of the cytokine storm observed in COVID-19 (Hu et al., 2020).

SARS-CoV-2 and its impact on the respiratory tract

Filbin (2022) explained:

“Severe coronavirus disease (COVID-19) is characterized by a disruption of barrier function between the pulmonary circulation and alveoli, leading to characteristic alveolar infiltrates, hypoxemia, and in the worst case acute respiratory distress syndrome (ARDS). Endothelial integrity plays an important role in maintaining the pulmonary capillary–alveolar barrier.” (p. 926)

The alveolar microvasculature in COVID-19 patients often displays swelling and blood clots composed of fibrin, platelets, and inflammatory cells. In acute respiratory distress syndrome (ARDS), damage to lung endothelial cells disrupts the endothelial barrier, leading to the accumulation of protein-rich fluid in the alveoli, causing hypoxia and breathing difficulties. Cytokines such as interleukin-1 β trigger inflammatory signals that reduce levels of VE-cadherin, a protein critical for maintaining endothelial junctions, thereby compromising barrier integrity. This disruption intensifies pulmonary inflammation, perpetuating a damaging cycle of barrier breakdown and inflammation (Upadhyaya et al., 2022).

Impact of SARS-CoV-2 on non-respiratory organs

COVID-19 primarily damages the lungs in various ways, and it is also capable of impacting several other bodily systems, including the cardiovascular system, urinary system, endocrine system, central nervous system, and skin (Nadeem et al., 2021).

Ning et al. (2022) explained that COVID-19 can lead to dysfunction in organs outside the lungs through several mechanisms, including direct viral entry into tissues, interference with the renin–angiotensin–aldosterone system (RAAS), immune system imbalance, damage to vascular endothelium, and inflammation-related clotting. While viral attachment via ACE2 receptors and RAS disruption may be distinctive to COVID-19, immune system overactivation with high levels of inflammatory cytokines and impaired microvascular blood flow can also occur in other serious illnesses such as septic shock.

COVID-19 may directly contribute to cardiac injury in infected individuals by targeting ACE2 receptors, which are involved in both acute and chronic cardiovascular effects, including pneumonia and myocardial injury. Since ACE2 functions within the RAAS pathway, this binding further contributes to an increase in circulating ACE2, a molecule linked to the development of cardiovascular disorders (Nadeem et al., 2021).

Johnson et al. (2020) noted that the renal system is also impacted by COVID-19, leading to renal complications, such as acute kidney injury (AKI) observed in a significant proportion of cases.

Clinical manifestations

In COVID-19 patients, the median period of incubation was found to be approximately 5.1 days, and within 11.5 days, symptomatic individuals (97.5%) were able to exhibit symptoms (Lauer et al., 2020).

Patients infected with the SARS-CoV-2 virus present with symptoms ranging in intensity from mild cases to severe forms, including pneumonia, ARDS, sepsis, and septic shock; however, most patients show a favorable clinical progression (Bhat et al., 2021).

Radiologic imaging

Imaging has been essential in diagnosing and managing patients with pneumonia caused by SARS-CoV-2, especially during the pandemic period. Computed tomography (CT) is possibly more sensitive than the other two, chest X-rays (CXR) and standard diagnostic tests for COVID-19. While typical imaging patterns can assist with diagnosis, differential diagnosis can be complex, requiring clinicians and radiologists to interpret findings within the broader clinical context. Imaging, particularly CT and potentially CXR, also provides prognostic insight by assessing the extent of pulmonary changes (Landini et al., 2020).

This retrospective epidemiological study aims to characterize the demographic profile, comorbidities, clinical and laboratory features, and outcomes of patients with COVID-19 admitted to the Infectious Diseases Service at the University Hospital Center “Mother Teresa” in Tirana, Albania, during the 2020 pandemic period. The study specifically investigates the prevalence of comorbidities, severity of respiratory impairment, and key laboratory markers, in relation to patient outcomes.

Material and Methods

Study design and population

This retrospective study included 1001 patients with confirmed COVID-19 admitted to the Infectious Diseases Service between March 9 and December 31, 2020, at the University Hospital Center “Mother Teresa” in Tirana, Albania. COVID-19 positivity was confirmed via real-time reverse transcription polymerase chain reaction (RT-PCR).

Data collection

Demographic, clinical, laboratory, and imaging data were extracted from individual medical charts obtained from the hospital Statistics Service. Charts were reviewed to capture detailed comorbidities, treatments, and clinical complications.

Statistical analysis

All collected data were initially entered into Microsoft Excel and subsequently imported into SPSS (Statistical Package for the Social Sciences), version 26.0, where all statistical analysis was conducted.

The statistical procedures and techniques applied in this study are detailed as follows: For all categorical variables (nominal, including binary/dichotomous and/or ordinal), absolute frequencies (n) and corresponding percentages were calculated. For all numerical variables, measures of central tendency and dispersion were computed. For data that followed a normal distribution, results were expressed as mean, standard deviation (SD) and median (Mdn).

Group differences in discrete variables were analyzed using Chi-square (χ^2) test. P-values less than 0.05 were considered statistically significant.

Outcomes

ARDS is described as “an acute respiratory illness characterized by bilateral chest radiographical opacities with severe hypoxaemia due to non-cardiogenic pulmonary oedema” (Meyer et al., 2021, p. 622).

As defined by the Berlin criteria, ARDS is classified by the extent of hypoxemia using the ratio of arterial oxygen partial pressure to fraction of inspired oxygen (PaO₂/FiO₂ ratio). Mild ARDS presents with a PaO₂ to FiO₂ ratio of 200–300 mm Hg, in contrast to moderate ARDS ranging from 100 to 200 mm Hg, while severe ARDS is indicated by a ratio at or below 100 mm Hg. These classifications also consider whether patients are receiving mechanical ventilation or noninvasive respiratory support, such as continuous positive airway pressure (CPAP) or positive end-expiratory pressure (PEEP) at levels not below 5 cm H₂O (Cascella et al., 2023).

The Dutch Radiological Society, in March 2020, created a nationwide COVID-19 network designed to support the development and broad dissemination of resources related to the pandemic period. The CO-RADS classification system was developed to assess the probability of lung involvement in COVID-19 cases on a five-point scale, where 1 indicates a very low probability and 5 indicates a very high probability (Prokop et al., 2020).

Results

In our study, 65% of the participants were male, and 35% were female.

Although COVID-19 affects men and women at similar rates, men have a higher likelihood of experiencing critical forms of disease and have a higher mortality rate independent of their age (Jin et al., 2020).

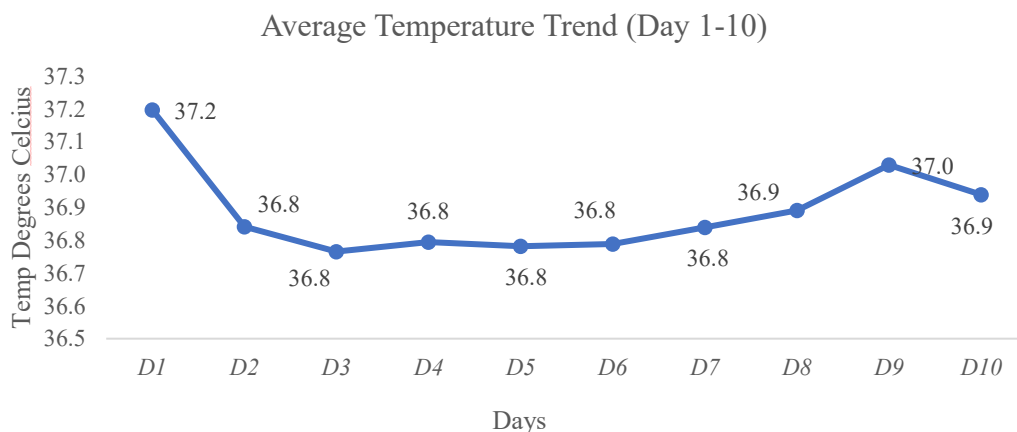
The average age of the population was 60.4 years ($SD = 13.9$, $Mdn = 62$, interquartile range [IQR] = 18).

Multivariate analysis revealed that being 65 years or older, male, and having a higher CCI were all independently associated with an increased risk of mortality and development of severe respiratory failure (Sundaram et al., 2022).

The average temperature was 37.2 °C ($SD = 0.8$, $Mdn = 37$, IQR = 1.2) (Figure 1).

Figure 1

Temperature curve from day 1-10



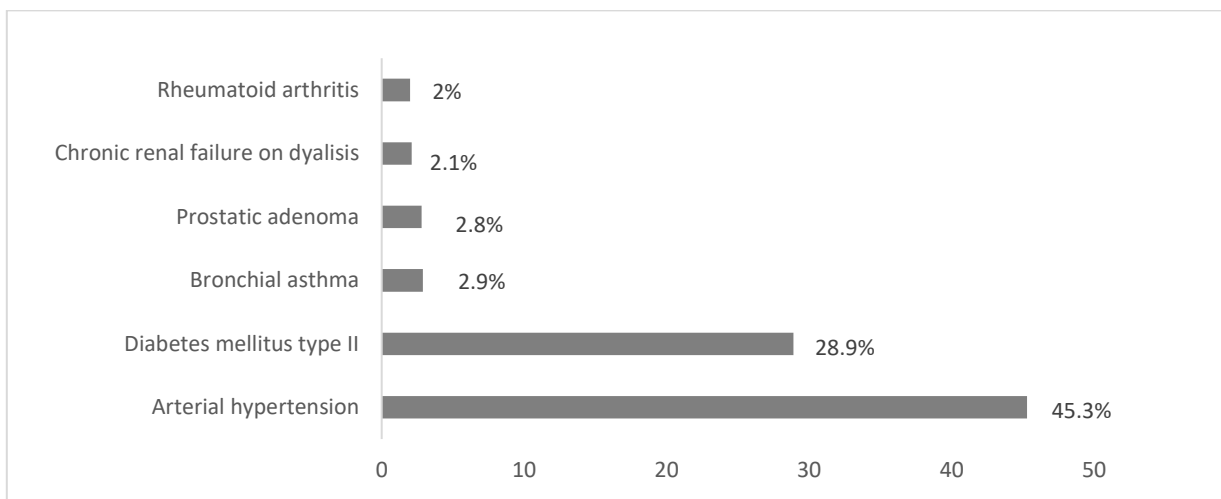
This graphic illustrates a downward trend in the temperature curve from day 1 to day 10.

The most prevalent comorbid disorder was arterial hypertension (45.3%), diabetes mellitus type II came next in frequency (28.9%), bronchial asthma (2.9%), prostatic adenoma (2.8%), chronic renal failure on hemodialysis (2.1%), and rheumatoid arthritis (2%) (Figure 2).

The average systolic and diastolic blood pressures of the population were 126.5 mmHg ($SD = 15.5$, $Mdn = 125$, $IQR = 10$) and 75.6 mmHg ($SD = 9.0$, $Mdn = 72$, $IQR = 10$), respectively.

Figure 2

The most frequent comorbidities according to frequency



According to Figure 2, arterial hypertension is the most prevalent disease, affecting 45.3% of cases compared to other conditions.

In our study, the most prevalent symptoms were difficulty in breathing, dry cough and fatigue which were noted in 77%, 67% and 63.8% of the patients, respectively.

The following table presents the descriptive statistics (M , SD , Mdn , and IQR) for the patients involved in the study (Table 1).

Table 1

Profiles of analysis

Profiles of analysis	M	SD	Mdn	IQR
WBC (K/ μ L)	10.6	7.3	9.3	7.6
Lymphocytes (%)	14.9	10.3	12	11.9
Neutrophils (%)	83.4	12.1	87	7.65
Thrombocytes (K/ μ L)	264.1	109.9	247	130
ESR (mm/h)	30.2	7	30	9.5
CRP1 (mg/dL)	9.2	9.5	7.3	10.85
CRP3 (mg/dL)	5.9	8.7	1.8	7.35

Profiles of analysis	<i>M</i>	<i>SD</i>	<i>Mdn</i>	IQR
LDH (UI/L)	479.1	432.5	384	264.5
Fibrinogen (mg/dL)	664.3	220.6	658	276
Ferritin (ng/mL)	999.2	2050	286.5	689.6
PCT (ng/mL)	0.7	3.3	0.09	0.21
D-Dimer (mg/dL)	362.4	1505.6	0.1	0.84
Albumin (g/dL)	3	1.3	3.2	0.87

Research has demonstrated that COVID-19 significantly impacts both the hematological and hemostatic systems, with thrombocytopenia occurring in 5–21% of patients (Rahman et al., 2021).

Laboratory findings:

General blood tests and clotting function data

Ding et al. (2021) compared inflammatory and blood clotting values at hospital admission between cases of low to intermediate severity COVID-19 and those with critical or high risk of death.

In our population, 45.1% of cases exhibited elevated white blood cell counts (range: 4 - 10 K/ μ L), while 84.9% had decreased lymphocyte levels (lymphopenia < 25%). Additionally, 137 patients (13.7%) showed high levels of neutrophils (neutrophilia > 72%). There were 104 patients (10.4%) with low platelet counts (thrombocytopenia < 150 K/ μ L). Furthermore, 728 patients (72.7%) had elevated fibrinogen levels (> 400 mg/dL), and 86 patients (8.6%) had high levels of D-dimer (> 198 mg/dL).

Other systemic inflammation parameters

In our population, 20.9% of cases exhibited high levels of erythrocyte sedimentation rate (ESR) (> 20 mm/h) and 76.4% showed high levels of C-reactive protein (CRP) (> 0.5 mg/dL). Additionally, 363 patients (36.3%) showed high levels of ferritin (> 275 ng/mL). Also, 13 patients (1.3%) showed high levels of procalcitonin (PCT) (> 0.5 ng/mL), only in patients tested for PCT.

Biochemical indicators

Out of the total patients, 423 (42.3%) exhibited low levels of albumin in their blood (hypoalbuminemia, < 3.5 g/dL). Additionally, 863 patients (86.2%) had elevated levels of lactate dehydrogenase (LDH) (> 220 IU/L). In our study, 527 patients (52.6%) showed high levels of urea, while 184 patients (18.4%) had elevated creatinine levels in their blood (urea > 43 mg/dL and creatinine > 1.11 mg/dL). Furthermore, 606 patients (60.5%) had elevated levels of aspartate aminotransferase (AST), and 345 patients (34.5%) had high levels of alanine aminotransferase (ALT) in their blood (AST > 34 U/L and ALT > 55 U/L). Lastly, 90 patients (9%) exhibited elevated bilirubin levels (> 1.2 mg/dL) (Figure 3).

Figure 3

Laboratory findings

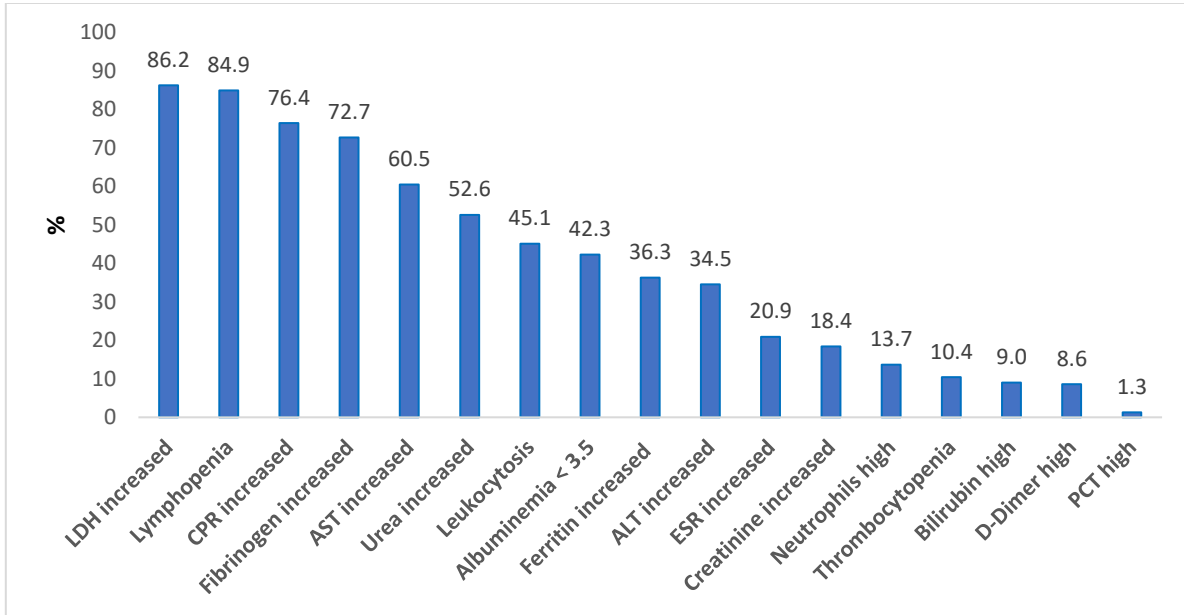


Figure 3 shows laboratory findings, with elevated LDH levels being the most prevalent at 86.2%, compared to other parameters.

The following table presents morbidity by gender along with the corresponding *p*-value, based on a chi-square test assessing the association between gender and morbidity in the study population (Table 2).

Table 2

Morbidity by gender

Comorbidities	Gender		Total, n = 1001	<i>p</i> value
	F, n = 350	M, n = 651		
Arterial hypertension	169 48.3%	284 43.6%	453 45.3%	.158
Atrial fibrillation	9 2.6%	15 2.3%	24 2.4%	.792
Paroxysmal atrial fibrillation	0 0.0%	4 0.6%	4 0.4%	.142
Acute myocardial infarction	1 0.3%	4 0.6%	5 0.5%	.482
Ischemic cardiomyopathy	2 0.6%	3 0.5%	5 0.5%	.813
Hypertensive cardiomyopathy	0	3	3	.203

Comorbidities	Gender		Total, n = 1001	p value
	F, n = 350	M, n = 651		
Unspecified cardiomyopathy	0.0%	0.5%	0.3%	.527
	2	2	4	
Acute coronary syndrome	0.6%	0.3%	0.4%	.813
	2	3	5	
Chronic cardiac failure	0.6%	0.5%	0.5%	.463
	5	6	11	
Mitral valve stenosis	1.4%	0.9%	1.1%	.463
	0	1	1	
Severe aortic stenosis	0.0%	0.2%	0.1%	.463
	0	1	1	
Status post percutaneous transluminal coronary angioplasty (PTCA)	0.0%	0.2%	0.1%	.077
	2	13	15	
Valvuloplasty	0.6%	2.0%	1.5%	.655
	1	1	2	
Dyslipidemia	0.3%	0.2%	0.2%	.346
	1	5	6	
Pacemaker wearer	0.3%	0.8%	0.6%	.172
	1	0	1	
Obesity	0.3%	0.0%	0.1%	.466
	3	9	12	
Type 2 diabetes mellitus	0.9%	1.4%	1.2%	.656
	98	191	289	
Thyroid pathologies	28.0%	29.3%	28.9%	.002
	7	0	5	
Chronic kidney disease	2.0%	0.0%	0.5%	.091
	11	10	21	
Chronic Obstructive Pulmonary Disease (COPD)	3.1%	1.5%	2.1%	.677
	1	3	4	
	0.3%	0.5%	0.4%	

Note. A chi-square test was used to assess the association between gender and morbidity. Overall, no gender differences were found regarding morbidity (with the exception of thyroid diseases, which are significantly more common in women than in men, $p = .002$).

Mortality by gender was analyzed, and their association was assessed using a chi-square test, with the corresponding p-value reported (Table 3).

Table 3*Mortality by gender*

	Gender		Total, n = 1001	p value
	F, n = 350	M, n = 651		
Mortality	55 (15.7)	96 (14.7)	151 (15.1)	.683

Note. A chi-square test was used to assess the association between gender and mortality. No gender difference was found regarding mortality ($p = .683$).

The average PaO₂ was 72.3 mmHg ($SD = 23.1$, $Mdn = 68$, $IQR = 21.9$).

In the studied population, the severe form of ARDS was predominant, constituting 43.9% of instances (439 patients), followed by the moderate form with 31.1% (311 patients), and the mild form with 7.7% (77 patients) (Table 4).

Table 4*Type of ARDS*

Type of ARDS	Frequency	Percentage (%)
Mild	77	7.7
Moderate	311	31.1
Severe	439	43.9
No ARDS	174	17.4
Total	1001	100

Our study showed that 160 patients (16%) received CPAP as part of their respiratory support during hospitalization (Table 5).

Table 5*Mechanical ventilation*

Mechanical ventilation	Frequency	%
CPAP	160	16.0
Intubated	151	15.1

In our population, 151 patients (15.1%) died. The average age of death was 63.3 years ($SD = 13.2$, $Mdn = 65$, $IQR = 16$).

COVID-19 infection in study participants was confirmed via molecular testing using RT-PCR, the most accurate test for finding viral genetic material (Moreira et al., 2021).

The sensitivity of RT-PCR tests is typically greater than that of rapid diagnostic molecular or antigen assays, but it can vary based on the type of sample and the collection process (Bruxvoort et al., 2021).

SARS-CoV-2 infection exhibits imaging results from CT which are similar to those of other viral infection and presents unique uncommon findings observed across different pathological conditions (Prokop et al., 2020).

Imaging results indicate that after chest CT scans, CO-RADS 5 was seen in 95% of participants. Just 15.8% of total cases (n=158) underwent a follow-up chest CT scan, and of those, 142 cases (89.9%) showed improvement.

Discussions

In our study, we observed that male patients were more likely than female patients to experience severe forms of COVID-19 disease, although there was no statistically significant difference.

Other studies have also found that male patients have a higher likelihood than female patients of experiencing critical forms of SARS-CoV-2 based on clinical severity classifications (Jin et al., 2020).

In the course of our research, the average age of the population was 60.4 years ($SD = 13.9$, $Mdn = 62$, $IQR = 18$).

Using multivariable techniques, age ≥ 65 years, male gender, and higher CCI scores were independently associated with increased risk of death and severe respiratory failure among hospitalized COVID-19 patients (Sundaram et al., 2022).

The findings align with previous research indicating that individuals aged 60 and older, as well as those with preexisting health conditions such as excess weight, diabetes, heart and blood vessel conditions, long-term kidney impairment, oncological diseases, persistent respiratory conditions, history of smoking, and organ transplantation, face a significantly elevated risk for COVID-19 related complications. Moreover, the investigation showed that patients with comorbidities had 12 times higher risk of mortality from the disease compared to those without underlying conditions (Aleem et al., 2023).

Lymphopenia is often identified as a frequently observed laboratory finding in most individuals infected with the SARS-CoV-2 virus (Aleem et al., 2023).

Wang et al. (2022) explained that because the presence of SARS-CoV-2 RNA in various immune system components—such as T cells, B cells, and Natural killer (NK) cells, the mechanism behind lymphopenia may involve direct invasion of these cells by the virus, resulting in cell death.

A comprehensive review conducted by Huan and Pranata revealed a significant correlation between lymphopenia and critical COVID-19. Additionally, 20% to 40% of patients were reported to have leukopenia, and 3% to 24% had leukocytosis. Lymphopenia—defined as a lymphocyte count of $\leq 1,100$ cells/ μL —was observed in 30% to 75% of cases (Rahman et al., 2021).

According to a systematic review of 7,613 participants, patients with severe forms of COVID-19 showed reduced platelet levels compared to patients with less severe illness. Not all research supports the role of platelet counts as a prognostic marker for COVID-19 mortality which also corresponds to the results of our study. In this case thrombocytopenia is found in 5–41.7% of patients with COVID-19 and can be mild when the number of thrombocytes ranges from 100–150

$\times 10^9/L$. On the other hand, mild thrombocytopenia has been observed in 58-95% of severe cases of COVID-19, and it was noted that patients with severe forms of disease exhibit platelet counts that are just $23 \times 10^9/L$ to $31 \times 10^9/L$ lower than those observed in non-severe cases. The preservation of platelet counts in these patients is attributed to a pronounced compensatory platelet production response (Wool & Miller, 2020).

According to Li et al. (2023), subjects with significant or critical forms of COVID-19 showed a pronounced increase in neutrophil counts at presentation to the hospital, compared to individuals with mild or moderate disease. Elevated neutrophil levels have been linked to greater disease severity and poorer clinical outcomes. Neutrophils are implicated in the pathogenesis of COVID-19, contributing to hypercytokinemia development and thrombotic complications. Although COVID-19 has been linked to a hypercoagulable condition, the underlying processes remain unclear and are thought to be driven by an exaggerated inflammatory response induced by cytokines.

According to Duca et al. (2022), COVID-19 has been linked to a distinct prothrombotic condition, which contributes to the development of both arterial and venous thromboses. Elevated D-dimer levels have been identified as a significant risk factor for poor clinical outcomes in COVID-19 patients.

A comprehensive meta-analysis, analyzed clinical and pathological features of 8697 subjects with SARS-CoV-2 in China noted laboratory deviations which comprised high CRP levels (65.9 %), along with lymphopenia (47.6 %), which are prevalent among the majority of COVID-19 patients, low leukocyte count ((23.5%), high leukocyte count (9.9%), elevated liver enzymes (26.4%), increased D-dimer levels (20.4%), and fibrinogen level, increased ESR levels (20.4%), high LDH levels, elevated PCT (16.7%), and abnormal kidney test results (10.9%) (Zhu et al., 2020).

Based on laboratory findings (see Figure 3), in our study elevated levels of LDH (86.2%) dominate, followed by lymphopenia (84.9%), and elevated CRP levels (76.4%).

Previous research has indicated that SARS-CoV can infect liver cells, and gastrointestinal symptoms along with higher liver enzyme levels have been identified in 14% to 53% of subjects. Increases in AST and ALT levels have also been related to more critical forms of the illness (Bakkaloglu et al., 2023). Our findings showed an increase in AST 60.5% and ALT 34.5% of cases, higher than in other studies.

Chest CT has demonstrated high sensitivity as an efficient imaging modality for detecting COVID-19 in adult patients. Common radiologic characteristic findings include ground-glass opacities, often accompanied or not by consolidation and air bronchograms, predominantly located in the lower lobes. These findings overlap with those of other viral pneumonias but also exhibit distinct characteristics (Sharif et al., 2022).

Although chest CT is highly sensitive in identifying COVID-19, its use in low-prevalence regions may result in increased false-positive diagnoses. Additionally, radiologists may identify COVID-19-related abnormalities incidentally in patients undergoing CT for other indications (Pezzutti et al., 2021).

In our study, we used CT to diagnose patients with COVID-19, and interpreted using the CO-RADS system.

The Dutch Radiological Society created CO-RADS classification system to provide a standardized approach for CT interpretation in individuals suspected of having COVID-19 (Prokop et al., 2020). Imaging of CT chest is a key component of the diagnostic evaluation in patients with suspected COVID-19 infection (Sharif et al., 2022). The system of CO-RADS was created as a

standardized method to evaluate the likelihood of pulmonary changes and improve consistency in CT scan reporting (Özel et al., 2021).

Özel et al. (2021) demonstrated that the CO-RADS classification is an effective method for diagnosing pneumonia in COVID-19 patients according to CT outcomes, a conclusion that aligns with our study results. The classification of patients' CT scans into CO-RADS categories 2 through 5 was consistent with patterns previously documented in the literature, supporting the system's diagnostic utility.

Prokop et al. (2020) noted that CT scan of chest may appear normal during the initial phase of COVID-19, which may explain why some patients in their study—despite being COVID-19 positive—received CO-RADS scores of low stages from a minimum of one examiner. As such, categories 1 or 2 of CO-RADS score should be interpreted cautiously in the early phase of infection. Additionally, they emphasized that CO-RADS 3 represents an indeterminate category, where CT findings alone are insufficient to confirm or exclude COVID-19.

Compared to other studies, in our study it has been noted that many cases consist of patients who have obtained CO-RADS-5 (950 patients, 94.9%), followed by CO-RADS-1 with 36 patients (3.6%) and then CO-RADS 2 (2 patients, 0.2%), CO-RADS-3 (4 patients, 0.4%), and CO-RADS-4 (8 patients, 0.8%).

Conclusions

Males predominated in the studied population.

Among comorbidities, arterial hypertension was observed in the majority of patients, followed by diabetes mellitus type II and chronic renal failure.

Intubated patients represented 15.1% of the total sample, and mortality in this subgroup was 100%. Patients in need of intensive care unit (ICU) admission at presentation had a worse prognosis.

Regarding ARDS classifications, some of patients did not require supplemental oxygen. The severe form of ARDS predominated compared to others.

Some patients required mechanical ventilation or CPAP.

Additionally, CRP values decreased by the eighth day of hospitalization.

Imaging results indicate that after chest CT scans, the majority of patients were classified as CO-RADS 5. Only a small proportion of cases underwent a follow-up chest CT scan, and only a fraction of them showed improvement.

Furthermore, in our study, patients with impaired renal function were observed, with increased creatinine in blood.

Also, in our population, patients with impaired liver function were identified, with elevated AST and ALT values and subjects with elevated levels of bilirubin in their blood.

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Competing interests

The authors declare no competing interests.

Ethical Approval

The research was approved by the Ethics Committee of the University of Medicine, Faculty of Medicine, Tirana, Albania (no. 11/18.03.2024).

Consent to Participate

Informed written consent was obtained from each participant at the time of recruitment. The subjects were informed that they could withdraw from the study at any stage, and they were assured of confidentiality.

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