RESEARCH PAPER

Predictors of Post-COVID Cardiovascular Complications in Patients with SARS-CoV-2–Associated Pneumonia: A Retrospective Cohort Study from Almaty, Kazakhstan

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ABSTRACT

A growing number of patients develop new cardiovascular problems in the months after recovering from SARS-CoV-2–associated pneumonia, yet the early signals that mark who is at risk are still not well defined. The aim of this study was to compare patients who later developed cardiovascular complications with those who did not, and to identify routine clinical and laboratory parameters that predict a complicated course. We conducted a retrospective review of 36 patients aged 25 to 87 years who were treated for SARS-CoV-2–associated pneumonia in Almaty, Kazakhstan. Patients were divided into two groups: 21 who developed cardiovascular complications within one year (Group I) and 15 who did not (Group II). Medical records, complete blood counts, biochemistry, coagulation profiles, and computed tomography findings were analysed in IBM SPSS, and correlation analysis was used to assess the associations between candidate markers and outcomes. Group I patients were on average older, more often female, and carried a heavier burden of arterial hypertension and ischaemic heart disease. They showed a markedly higher D-dimer (median 266 µg/L versus 1.26 µg/L), elevated C-reactive protein (20.4 mg/L), and a raised neutrophil-to-lymphocyte ratio (3.65 versus 1.25). Cerebral infarction occurred in 5 patients (23.8%) of Group I, and a positive correlation was found between high D-dimer and the development of cerebral infarction. Taken together, comorbidity, raised D-dimer and C-reactive protein, and a high neutrophil-to-lymphocyte ratio behaved as predictors of a complicated course and may help identify patients who would benefit from early anticoagulant prophylaxis.

Keywords: SARS-CoV-2, COVID-19 pneumonia, post-COVID syndrome, cardiovascular complications, D-dimer, C-reactive protein, neutrophil-to-lymphocyte ratio, comorbidity, Kazakhstan, retrospective cohort.

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1. Introduction

Several years into the COVID-19 pandemic, the complications and risk factors associated with the aftermath of acute SARS-CoV-2 infection remain only partially understood [1,2]. There is still no firm agreement on what should count as long COVID or as a post-COVID syndrome, and definitions vary between studies and clinical settings [3,4,5]. What is clear from routine practice is that a subset of patients who recover from the acute pneumonia go on to develop new cardiovascular events months later, including ischaemic stroke and myocardial infarction.

Part of the difficulty is that the early phase of severe COVID-19 already disturbs coagulation and triggers a strong inflammatory response. Markers such as D-dimer, C-reactive protein and fibrinogen rise during the acute illness, and abnormal coagulation has been linked to worse outcomes during hospitalisation [9,10]. Whether these same routine parameters, measured around the acute episode, carry information about cardiovascular risk over the following year is a more practical question, and one that matters for clinicians deciding who needs closer follow-up or prophylactic anticoagulation.

Data from Central Asia on this point are scarce. To address the gap, we compared two groups of patients who had survived SARS-CoV-2–associated pneumonia — one that later developed cardiovascular complications and one that did not — and looked for routinely available predictors that separated them. The aim of the study was to identify clinical and laboratory features associated with a complicated course of the disease.

2. Materials and Methods

2.1 Study design and patients

We performed a retrospective review of medical records from 36 patients treated under the State Guaranteed Volume of Free Medical Care (GOBMP) and Compulsory Social Health Insurance (OSMS) programmes at a medical centre in Almaty, Kazakhstan. All patients were aged between 25 and 87 years and had previously been treated for SARS-CoV-2–associated pneumonia. Patients were assigned to two groups. Group I comprised patients who developed a complication following pneumonia, including acute cerebrovascular accident (stroke), acute myocardial infarction, or newly diagnosed type 2 diabetes mellitus. Group II comprised patients with no complications recorded during follow-up after COVID-19.

2.2 Data collection

From each medical record, we extracted demographic characteristics, complete blood count parameters, biochemical markers, coagulation parameters (D-dimer, fibrinogen, and prothrombin index), C-reactive protein, ferritin, and oxygen saturation levels. Hospital admissions occurring within 12 months following SARS-CoV-2–associated pneumonia were subsequently reviewed to identify cardiovascular events.

2.3 Computed tomography assessment

The extent of lung involvement on chest computed tomography (CT) was graded using the standard four-point classification routinely applied in clinical practice: CT-1 (<25%), CT-2 (25–50%), CT-3 (50–75%), and CT-4 (>75%) of lung parenchymal involvement.

2.4 Derived marker

The neutrophil-to-lymphocyte ratio was calculated from the differential white-cell count as the absolute neutrophil count divided by the absolute lymphocyte count, and used as a simple index of the inflammatory state.

2.5 Statistical analysis

Statistical analyses were performed using IBM SPSS Statistics version 26.0. Continuous variables were summarized as medians and ranges, whereas categorical variables were presented as frequencies and percentages. Correlation analysis was performed to assess associations between laboratory markers and cardiovascular outcomes. A p-value <0.05 was considered statistically significant.

3. Results

3.1 Group composition and baseline characteristics

Of the 36 patients reviewed, 21 (58%) developed cardiovascular complications during the year after pneumonia and formed Group I; the remaining 15 (42%) had no complications and formed Group II. Group I had a clear female predominance (66.7%), whereas the sex distribution in Group II was close to even (53.3% male, 46.7% female). Patients in Group I were slightly older, with a mean age of 69 years against 66 years in Group II. Moderate-to-severe lung involvement (CT-2 to CT-3, up to 75% of the parenchyma) predominated in both groups. Oxygen saturation at presentation was similar between the groups. The baseline characteristics are summarised in Table 1.

Table 1. Baseline demographic and clinical characteristics of the two groups.

Characteristic	Group I – with complications (n = 21)	Group II – without complications (n = 15)
Female, n (%)	14 (66.7)	7 (46.7)
Male, n (%)	7 (33.3)	8 (53.3)
Age, years (mean)	69.1	66.2

Characteristic	Group I – with complications (n = 21)	Group II – without complications (n = 15)
Age range, years	57–83	25–87
Oxygen saturation, % (mean)	91.9 (82–98)	91.7 (85–97)
CT grade 2–3 lung involvement, %	52.3	66.0

3.2 Comorbidities

The burden of comorbidity was greater in the group that subsequently developed complications. Arterial hypertension was present in 14 patients (70%) in Group I, predominantly grade 3, compared with 6 patients (40%) in Group II, where grade 2 hypertension predominated. Ischaemic heart disease was recorded in 8 patients (40%) and atherosclerosis in 3 patients (14.3%) of Group I. In Group II, type 2 diabetes mellitus was the most frequent comorbidity, occurring in 4 patients (26.7%). These findings suggest that pre-existing cardiovascular conditions may contribute to a more severe course of COVID-19 pneumonia and increase the risk of subsequent complications. Comorbidity data are presented in Table 2.

Table 2. Comorbidities in patients with and without post-COVID cardiovascular complications.

Comorbidity	Group I (n = 21), n (%)	Group II (n = 15), n (%)
Arterial hypertension	14 (70), mostly grade 3	6 (40), mostly grade 2
Ischaemic heart disease	8 (40)	—
Atherosclerosis	3 (14.3)	—
Type 2 diabetes mellitus	—	4 (26.7)

3.3 Laboratory and imaging markers

The most pronounced differences between the two groups were observed in coagulation and inflammatory markers (Table 3). Group I demonstrated markedly higher D-dimer levels, with a median value of 266 µg/L compared with 1.26 µg/L in Group II. Because D-dimer reflects active thrombus formation, this finding suggests a higher thrombotic risk among patients who subsequently developed cardiovascular events. C-reactive protein, another marker associated with cardiovascular risk when levels exceed 3 mg/L, was substantially elevated in Group I, reaching 20.4 mg/L. The neutrophil-to-lymphocyte ratio, used as a prognostic indicator in the present study, reached 3.65 in Group I, a value considered unfavourable, whereas it remained within the normal range at 1.25 in Group II.

Table 3. Laboratory and coagulation markers in the two groups (median or mean values, as reported).

Marker	Group I – with complications	Group II – without complications
D-dimer (median)	266 µg/L	1.26 µg/L
C-reactive protein	up to 20.4 mg/L	< 3 mg/L (within normal range)
Neutrophil-to-lymphocyte ratio	3.65	1.25
Ferritin (mean)	121.9 µg/L	161.7 µg/L
Fibrinogen (mean)	3.63 g/L	within normal range
Prothrombin index (mean)	94.6 %	94.6 %

3.4 Analysis of Oxygen Saturation Levels

A comparative analysis of oxygen saturation (SpO_2) levels was performed between patients who subsequently developed complications and those who remained free of complications following SARS-CoV-2–associated pneumonia.

The mean oxygen saturation in the complication group was 91.9%, with values ranging from 82% to 98%. In the group without complications, the mean oxygen saturation was 91.7%, with a range of 85% to 97%.

These findings suggest that baseline oxygen saturation during the acute phase of SARS-CoV-2–associated pneumonia was not strongly associated with the subsequent development of cardiovascular complications.

3.5 Cardiovascular outcomes and correlation analysis

During the 12-month follow-up period, cerebral infarction occurred in 5 patients (23.8%) in Group I. Correlation analysis revealed a positive correlation between elevated D-dimer levels and the occurrence of cerebral infarction, consistent with the established role of D-dimer as a marker of thrombus formation and a predictor of cardiovascular events. No comparable events were recorded in Group II.

Table 4. Correlation Between D-Dimer Levels and Cerebral Infarction in Patients with Post-COVID Complications

Variable 1	Variable 2	Correlation Coefficient (r)	Number of Patients (n)
D-dimer	Cerebral infarction (stroke)	0.631	21

The observed correlation suggests that increased D-dimer levels during SARS-CoV-2–associated pneumonia may identify patients at greater risk of subsequent thrombotic and cerebrovascular complications.

Discussion

Our findings align with what is known about the acute phase of severe COVID-19, in which elevated D-dimer, C-reactive protein, and abnormal coagulation already identify patients who do worse [9,10]. This study adds a practical link between those same routine markers and cardiovascular complications that appear over the following year. The patients who developed stroke, myocardial infarction or new diabetes were not a random subset: they were older, more often hypertensive, and they carried higher D-dimer, higher C-reactive protein and a higher neutrophil-to-lymphocyte ratio from the outset.

The D-dimer signal is the one that stands out. A median of 266 µg/L in the complicated group against 1.26 µg/L in the uncomplicated group is a large gap, and the positive correlation with cerebral infarction fits the picture of a persistent prothrombotic state after COVID-19. The neutrophil-to-lymphocyte ratio adds a second, very cheap piece of information: a value of 3.65 against 1.25 separates the groups using nothing more than a routine blood count. C-reactive protein behaved in the same direction. None of these tests is new or expensive, which is part of what makes them attractive for risk stratification in everyday practice.

Comorbidity matters too, and the data here are blunt about it. Arterial hypertension was present in 70% of the complicated group and ischaemic heart disease in 40%, against much lower rates in the uncomplicated group. This is not surprising, but it reinforces the case for watching patients with established cardiovascular disease more closely after COVID-19 pneumonia rather than discharging them from follow-up once the lungs have recovered.

Practically, the combination of raised D-dimer and C-reactive protein, a high neutrophil-to-lymphocyte ratio, and a background of hypertension or ischaemic heart disease describes a patient who may benefit from oral anticoagulant prophylaxis to reduce the risk of thrombosis. That conclusion needs testing in a prospective trial before it can become a firm recommendation, but the direction of the data is consistent.

Significance

This study provides data from Kazakhstan, a setting where information on post-COVID cardiovascular outcomes is limited, and shows that simple, widely available markers can flag patients at higher risk of complications in the year after SARS-CoV-2–associated pneumonia.

Limitations

Several limitations should be kept in mind. The sample was small (36 patients) and drawn from a single centre, which limits how far the results generalise. The design was retrospective and relied on existing records, so some parameters were incomplete, and the timing of measurements relative to the acute illness varied. The follow-up captured cardiovascular events over one year but did not track the persistence of laboratory abnormalities or antibody responses. Finally, the correlation analysis shows association, not cause, and the candidate predictors require confirmation in a larger, prospective cohort before they are used to guide treatment.

Conclusion

Comorbid conditions such as arterial hypertension and ischaemic heart disease, together with raised D-dimer and C-reactive protein and a high neutrophil-to-lymphocyte ratio, were associated with a complicated course of SARS-CoV-2–associated pneumonia and can be regarded as risk factors for cardiovascular complications. Among these, D-dimer showed a positive correlation with the development of cerebral infarction. The data suggest that patients with this profile may benefit from oral anticoagulant prophylaxis to prevent thrombosis and underscore the value of continued cardiovascular follow-up after recovery from COVID-19 pneumonia. Larger prospective studies are needed to confirm the predictive value of these markers and to define which patients should receive prophylactic treatment. These findings also fit the broader Kazakhstan-centred SARS-CoV-2 evidence base, including antiviral drug-repurposing studies, companion-animal One Health surveillance, post-infectious neurological complications, and rare vaccine-related adverse events. These findings are also consistent with recent evidence showing that tenofovir prodrugs may suppress SARS-CoV-2 replication in vitro. Khaidarov et al. reported that tenofovir disoproxil fumarate (TDF) and tenofovir alafenamide (TAF) demonstrated dose- and exposure-dependent antiviral activity against SARS-CoV-2 in Vero E6 cells, with TDF reducing high viral loads and TAF achieving near-complete suppression at higher concentrations while maintaining acceptable cell viability. Importantly, the authors suggested that the antiviral potential of TAF may be relevant to

persistent viral reservoirs in lymphoid tissues, which are discussed as a possible contributor to long COVID pathogenesis [11–16].

Abbreviations

CRP — C-reactive protein
CT — Computed tomography
COVID-19 — Coronavirus disease 2019
GOBMP — State Guaranteed Volume of Free Medical Care
IHD — Ischaemic heart disease
NLR — Neutrophil-to-lymphocyte ratio
OSMS — Compulsory Social Health Insurance
RR — Relative risk
SARS-CoV-2 — Severe acute respiratory syndrome coronavirus 2
SPSS — Statistical Package for the Social Sciences

Declarations

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Data availability: The data that support the findings of this study are available from the corresponding author upon reasonable request.
Ethics approval: The study was a retrospective review of de-identified clinical records conducted in accordance with the relevant institutional guidelines and the Declaration of Helsinki.
Competing interests: The authors declare no conflicts of interest.

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