

Laboratory Parameters and Clinical Courses in Covid-19 Prognosis: Case Reports

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Abstract

Coronavirus disease 2019 (COVID-19) has spread rapidly around the world since the outbreak in Wuhan, Hubei Province, China in 2019. COVID-19 is an infection caused by the novel coronavirus SARS-CoV-2.

In case 1, there were low white blood cells. In case 2, mild lung involvement was observed on his chest computed tomography. In case 3, high levels of ferritin and procalcitonin in blood parameters were found remarkably. During the COVID-19 pandemic, symptoms and laboratory parameters differ from person to person. Timely diagnosis, isolation and initiation of necessary treatments are necessary to significantly reduce the risk of disease transmission. Several biomarkers have been identified that could potentially assist risk classification models to predict severe and deadly COVID-19.

Keywords: SARS-Cov-2, hematologic parameters, prognosis, COVID-19

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INTRODUCTION

Virus infection with severe acute respiratory syndrome coronavirus-2 (SARS-CoV-2) can be asymptomatic or causes mild to severe symptomatic disease (1,2).

There is an incubation period of 1 to 14 days in the pathogenesis of COVID-19 (3-5).

COVID-19 symptoms include fatigue, mild chills, fever, sore throat, shortness of breath, dry cough, severe respiratory distress, and pulmonary

pneumonia (6,7). Especially in patients with severe disease, fatigue, myalgia or arthralgia, hepatic and renal dysfunction, leukopenia, thrombocytopenia, lymphocytopenia, and high inflammatory biomarkers, as well as respiratory failure, have been identified. Clinical studies have reported elevated liver enzymes in COVID-19 patients (8,9).

CT imaging changes are common in COVID-19 patients. Covid-19 involves bilateral multilobular subsegmental consolidation of the lungs in the early stages followed by multiple mottling and ground-glass opacity (10).

Hypersensitive troponin and higher levels of aspartate aminotransferase and a long with lymphopenia, leukopenia, thrombocytopenia and RNAemia, were observed in the blood laboratory profile of COVID-19 patients (1,11,12).

CASE-1

I am 41-year-old female. I have a history of severe atypical pneumonia about 1.5 years ago.

On November 10, 2020, I felt the first symptom of Covid-19 as fatigue in the evening in Ordu.

On November 11, 2020, I felt tiredness, weakness, mild chills. The real-time reverse transcriptase-polymerase chain reaction (RT-PCR) for SARS-CoV-2, performed by taking nasal and pharyngeal swabs was found to be positive. No lung involvement was observed in my chest X-rays, except for my previous lung sequelae (Figure 1).

Regarding hematologic parameters hematocrit and haemoglobin were normal, White blood cells (WBC) were detected low (WBC: 2.70×10^9 /UL, normal range $4.49-12.6 \times 10^9$ /UL), CRP washigh, (CRP: 6.3 mg/L, normal range 0-5 mg/L), aspartate aminotransferase (AST): 18 U/L (normal range 0-32

U/L), alanine aminotransferase (ALT): 9 U/L (normal range 0-33 U/L), ferritin was within normal limits.

I completed my treatment with favipiravir and anticoagulant drugs. When viewed 10 days after the initiation of treatment (on November 22, 2020), my ALT and AST values increased, AST: 59 U/L (normal range 0-32 U/L), ALT: 91 U/L (normal range 0-33 U/L), CRP was detected as 0.3mg / L (within normal range). On November 28, 2020, my hematological parameters are completely normalized (AST: 16 U/L, ALT: 29 U/L and others)

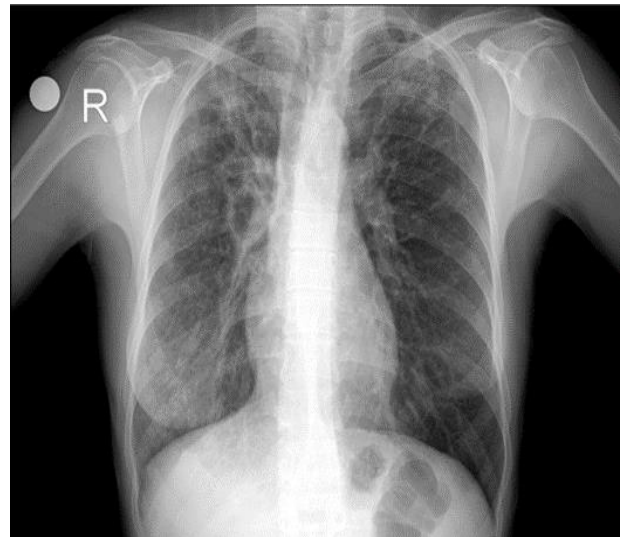


Figure 1. Image of previous lung sequelae

CASE-2

75-year-old my father, who has Parkinson's disease, showed symptoms simultaneously with me. On November 11, 2020, my father had complaints of weakness, tiredness and mild cough. RT-PCR test of my father who went to the hospital for testing was positive. His chest computed tomography (CT) showed mild bilateral ground-glass opacities associated with COVID-19 (Figure 2).



Figure 2. Mild bilateral ground-glass opacities

Despite his chronic illness, my father survived Covid-19 under home surveillance. My father was given only favipiravir and anticoagulant drugs. Fortunately, another problem was not observed.

CASE-3

On November 12, 2020, the PCR test was positive in my husband, a 45-year-old healthy man. My husband's chest CT showed mild bilateral ground-glass opacities associated with COVID-19 (Figure 3).



Figure 3. Mild bilateral ground-glass opacities

At first, only ferritin was detected abnormally in blood parameters. Ferritin:587 μ g/L (normal range 30-400 μ g/L). He was given the same medication (favipiravir and anticoagulant drugs). Despite the

high ferritin level, my husband initially had no symptoms. On November 17, 2020, ferritin was 747 μ g/L, erythrocyte sedimentation rate (ESR):29 (normal range 0-20), CRP was 0.7 mg/L (within normal limit), ALT:28 U/L, AST:23 U/L (within normal limits).

On November 20, 2020, ferritin and CRP were too high, (ferritin:1402 μ g/L, CRP: 73mg/L), ALT:97 U/L (normal range 0-33 U/L), AST:47 U/L (normal range 0-32 U/L).

On November 23, 2020, ferritin and CRP were too high, (ferritin:1517 μ g/L, CRP: 107.3mg/L), ALT:80 U/L (high), AST:39 U/L (high), WBC:11.44x10⁹/UL, PLT (platelet):130x10³/UL (normal range,150-450 x10³/UL).

Meanwhile, symptoms such as fever, mild chill, excessive sweating, myalgia, weakness, fatigue appeared. Temperature: 38.3 °C (100.9 °F). Leukocyte was found to be quite high in urine analysis, leukocyte>204.55 (normal range, 1-4) HPF, erythrocyte:2 (normal range, 1-4) HPF. Infection detected in urine and antibiotic treatment was given (as Ciprofloxacin 500mg).

On November 25, 2020, ferritin:1188 μ g/L, CRP: 108mg/L, ALT:45 U/L, AST:20 U/L. Prokalsitonin:0.495ng/mL (normal range, 0-0.046 ng/mL).

In urine, leukocyte:38 (1-4) HPF, erythrocyte:6 (1-4) HPF determined. There was a significant decrease in leukocyte in urine. The next day, symptoms began to regress.

On November 28, 2020, ferritin:1055 μ g/L, CRP: 24.7mg/L, ALT:52 U/L, AST:35 U/L. In urine, leukocyte:2 (1-4) HPF, erythrocyte:<1.14 (1-4) HPF determined.

On December 06, 2020, ferritin:1011 µg/L, CRP: 20mg/L, ALT:45 U/L, AST:20 U/L. WBC:6.33x10³/UL, PLT:283x10³/UL. In urine, leukocyte<1.14 (1-4) HPF, erythrocyte:<1.14 (1-4) HPF determined.

On December 10, 2020, ferritin:919 µg/L, CRP: 0.5mg/L, ALT:43 U/L, AST:20 U/L.

On December 17, 2020, ferritin:747 µg/L, CRP: 0.5mg/L, ALT:28 U/L, AST:23 U/L, ESR:29 (normal range, 0-20).

DISCUSSION

Despite my (case 1) severe atypical pneumonia sequelaes, my Covid-19 symptoms were mild. No lung involvement was observed in my chest X ray except for my previous lung sequelaes.

Regarding hematologic parameters: hematocrit, haemoglobin, platelet count, D-dimer were normal. White blood cells (WBC) were detected low (WBC:2.70× 10⁹ /L), (normal range: 4.49-12.6 /UL), CRP were detected high level, (CRP: 6.3 mg/L), (normal range: 0-5 mg/L). While WBC is generally expected to increase, WBC was found to be in a decreased value in my hematologic parameters.

Although my father with Parkinson's disease (case 2) had lung involvement, Covid-19 symptoms were mild. Mild cough, weakness and myalgia that started on the 3rd day after possible contact continued for about 2 days.

CT imaging changes are common in Covid-19 patients (10). Although mild lung involvement was found in case 2 and 3, no progress to severe pneumonia was observed.

According to Gutiérrez et al. Lymphopenia (<1000 cells /mL), neutrophilia (> 10,000 cells /mL), high C-reactive protein (> 10 mg / dl), elevated LDH

(> 350 IU / L), D-dimer (> 1 mg / ml), increases in hepatic transaminases, troponin and ferritin were determined as prognostic markers (13).

In case 3, a progressive increase in ferritin was detected after PCR positivity. When ferritin peaked in my husband's blood, his clinical symptoms peaked too.

Case 3 had a partial increase in procalcitonin level, procalcitonin: 0.495ng / mL (normal range: 0-0.046 ng / mL). This increase was supportive of the urinary tract infection detected as secondary infection with Covid-19.

According to literature findings, the presence of SARS-CoV-2 has rarely been shown in urine swabs of COVID-19 patients (8-14). The development of urinary tract infection in our COVID-19 patient (case 3) may be considered as a possible complication of COVID-19. Observing a significant increase in WBCs in severe Covid-19 may indicate a worsening clinical course. According to the literature (13), while the decreases are observed in lymphocytes, monocytes and eosinophils, the increase in WBC appears to be triggered by high neutrophils. Regarding immunological biomarkers, significantly increases were detected for serum ferritin and IL-6 in non-survivors compared to survivors. Therefore, it is recommended that both parameters be used to understand prognosis during hospitalization in COVID-19 patients.

These elevations, together with high CRP, indicate the development of a systemic inflammatory response syndrome, a severe form of the disease (15).

In hospitalized people with respiratory distress, clinicians should consider the WBC count, platelet count, lymphocyte count, serum ferritin level and IL-

6 as potential signs of progression to critical illness. Since procalcitonin is considered to be a marker of secondary bacterial infection commonly observed in non-survivors, it should be measured regularly (16,17).

Clinical symptoms were longer and more severe in case 3, therefore ferritin, CRP, ESR, procalcitonin were useful in clinical surveillance. Laboratory blood parameters differed in case 1 and case 3. Detailed blood tests were not performed for case 2, who was in a different city.

CONCLUSION

During the COVID-19 pandemic, symptoms and laboratory tests differ from person to person. Timely diagnosis, isolation and initiation of necessary treatments are necessary to significantly reduce the risk of disease transmission. Several biomarkers have been identified that could potentially assist risk classification models to predict severe and deadly COVID-19.

As a result, it is thought that it will be useful to examine the WBC count, platelet count, lymphocyte count, serum ferritin level and IL-6 as markers for potential progression to critical disease in Covid-19 patients with respiratory distress.

Ethics Committee Approval: Approval was received for this study from the patient.

Peer-review: Externally peer-reviewed.

Author Contributions: Concept: MAC. Design: MAC., Literature Search: MAC. Data Collection and Processing: MAC. Analysis and/or Interpretation: MAC., Writing: MAC.

Conflict of Interest: No conflict of interest was declared by the author.

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